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GENERAL ARTICLE

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ADOLESCENT CARE

NORMAL ADOLESCENT DEVELOPMENT

***Swati Y Bhave**
****Sangeeta Yadav**

Abstract: *Normal adolescent development is the foundation stone for healthy adulthood. Early, mid- and late adolescence are the three phases in adolescent development. Early adolescence (10-13 yrs) is characterized by concrete thinking, growth spurt and the beginning of sexual maturation. In mid-adolescence (14-15 yrs) the physical changes are complete, abstract thinking begins and the adolescents develop a strong sense of identity and are very much under peer influence although family still remain important. There is an increased tendency for experimenting and risk taking. In late adolescence (16-19 yrs) physical and sexual maturity is complete, identity is significantly developed and abstract thinking is well developed.*

Keywords: *Normal adolescent development, Stages, Early, Middle, Late.*

Adolescence is the transitional period during which an individual matures from childhood into an adult with physical, psychosocial and reproductive maturity.[1] WHO defines adolescence to be between the ages of 10 and 19 years of age.

Puberty signifies the beginning of biological growth during adolescence that results in individuals who are sexually dimorphic.[2] Initially, there is gonadal maturation with acquisition of secondary sexual characteristics and associated growth spurt leading to fertility and final height.[3] The biological changes occurring during puberty are sexual maturation, increases in height and weight, i.e., adolescent growth spurt, completion of skeletal growth, increase in skeletal mass and changes in body composition. During puberty the succession of these events are consistent among adolescents. However, there are likely to be deviations in the age of onset, duration and the tempo of these events between and within individuals. Physical changes represent just a fraction of the developmental processes that adolescents experience. Biological, psychosocial and cognitive changes that begin during puberty and continue throughout adolescence directly affect not only the physical status, but also influence their final personality as an adult in society.[1]

The onset of puberty corresponds to a skeletal (biological) age of approximately 11 years in girls and 13 years in boys.[4] Girls enter and complete each stage of puberty earlier than boys, but the variation is significant even amongst the same gender and ethnic background in the timing and tempo of puberty. The pubertal growth consists of a phase of acceleration, followed by a phase of deceleration and the eventual cessation of growth with the closure of epiphyses. Therefore, we observe that the adolescents of same chronological age can vary greatly in physical appearance. This leads to early and late bloomers and results in significant variation in physical characteristics in children of the same age and can cause profound emotional and psychosocial issues. Boys, who are late bloomers, may feel physically inferior to their peers who mature earlier. This results in low self-esteem and a sense of dissatisfaction that can lead to the use of anabolic steroids and other supplements. Their physical immaturity can also lead to their being subjected to sexual abuse. Boys who mature early are physically stronger than their peers and hence are more likely to be looked upon as natural leaders but if they have aggressive tendencies it can lead to aggression and bullying behaviors. In females, however, early maturation results in a woman's body with child's mind that may lead to sexual abuse and exploitation. Late blooming girls have poor body image and self-esteem, sexual experimentation and possibly, disturbed or disordered eating behaviors. Both early and later bloomers are at increased risk of engaging in other unhealthy behaviors such as smoking, alcohol consumption and early sexual intercourse. Gonadal steroid hormones (primarily estradiol in both genders) enhance bone mineral accrual and affect adult height by promoting epiphyseal fusion through direct effects on the growth plate.[5]

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Clinicians need to evaluate the stages of puberty in their adolescent clients and the well accepted standard reference is use of sexual maturity rating (SMR), also known as Tanner staging. Sexual maturation correlates remarkably well with linear growth, changes in weight, and body composition and hormonal changes.[1]

Brain growth and maturity

The brain undergoes tremendous changes during adolescence though final maturity is achieved only by 25 years of age, as per the research with functional brain imaging. The unused connections in the thinking and processing part of the child's brain are 'pruned' away.[6] At the same time, other connections are strengthened. This is the brain's way of becoming more efficient. This pruning process begins in the back of the brain.[7] The prefrontal cortex is the last to develop.[8] Hence, decision making and understanding of one's actions is poorly developed in adolescents and they depend on amygdala which is associated with emotions, impulses, aggression and instinctive behavior. This leads to high risk taking behaviors.

Normal adolescent development

Psychosocial and cognitive changes are best understood when divided into three periods: Early adolescence, middle adolescence and late adolescence. Each of these phases of development is marked by the development of new emotional, cognitive and social skills[1] (Table I).

Cognitive development

The cognitive development includes the ability to reason effectively, solve problems, think abstractly and plan for the future.[1] New capacities allow adolescents to engage in introspection and problem solving through critical thinking, creative thinking and mature decision making that was previously beyond their cognitive capacity.[1] Hence life skill education is very important to empower teens.

Early adolescence

The cognitive abilities are dominated by concrete thinking, egocentrism and impulsive behavior. Hence they lack skills necessary to comprehend risky behaviour and futuristic implications and also find it difficult to change behavior.

Middle adolescence

The abstract reasoning skills begin to emerge among most teens during middle adolescence. Adolescents will often regress to concrete thinking skills when faced with overwhelming emotions or stressful situations. They consider themselves invincible and love experimentation and high risk behaviour - smoking, alcohol consumption, using street drugs, engaging in risky sexual activities, weapon carrying, drinking and driving and various violent behaviours.

Late adolescence

The expansion of abstract reasoning skills continues to occur during late adolescence, which assists teens in developing an ability to comprehend how current health behaviors affect long-term health status. Older adolescents are able to manage increasingly sophisticated social situations, are able to suppress impulsive behaviors and are less affected by peer pressure.

Table I. Summary of psychosocial processes and the substages of adolescent development.[1]

Substage	Cognitively related	Emotionally related	Socially related
Early adolescence	Concrete thinking, early moral concepts	Adjustment to a new body image, adaptation to emerging sexuality	Strong peer effect
Middle adolescence	Emergence of abstract thinking, expansion of verbal abilities and conventional morality, adjustment to increased school demands	Establishment of emotional separation from parents	Increased health risk behavior, sexual interests in peers, early vocational plans
Late adolescence	Development of abstract, complex thinking, emergence of post conventional morality	Establishment of a personal sense of identity, further separation from parents	Increased impulse control, emerging social autonomy, establishment of vocational capability

Sense of identity

Development of the 'possible self', that is IDENTITY formation - how adolescents see themselves right now.[9,10] This has traditionally been thought of as the central task of adolescence.[11] This is well developed by late adolescence as biological growth and development complete and body image issues are less common. Identity includes the following two concepts.

Self-concept: This includes beliefs about one's attributes (physical, emotional and psychological), roles and goals (career and interests), values and beliefs (religious, political).

Self-esteem: involves evaluating how one feels about one's self-concept. 'Global' self-esteem refers to how much we like or approve of our perceived selves as a whole. 'Specific' self-esteem refers to how much we feel about certain parts of ourselves (physical, social etc.). Self-esteem develops uniquely for each adolescent and results in different trajectories of self-esteem possible over the course of development.[12]

Moral development

The before adolescent's cognitive and psycho-social development lays the groundwork for moral reasoning honesty and prosocial behaviors such as helping, volunteerism or caring for others.[2]

Emotional development

Emotional development during adolescence involves establishing a realistic and coherent sense of identity in the context of relating to others and learning to cope with stress and manage emotions.[12]

Emotional skills necessary for understanding emotions and having good interpersonal skills are very important. Emotional intelligence involves self-awareness, relationship skills - the ability to get along well with other people and to make friends.[13] The most important skills that adolescents may develop as part of their emotional development are recognizing and managing emotions, developing empathy and cooperative spirit and learning to resolve conflict constructively.

In early adolescence the emotions are intense and labile and can lead to impulsive actions. In middle adolescence, peer groups become more important than family and their influence with regard to making lifestyle behavior peaks, leading to conflicts. In late adolescence economic and emotional dependence upon family is markedly decreased and conflict over personal issues, such as lifestyle also decreases. Relationships with a single individual become more influential than those with a group of peers as a stronger sense of personal identity emerges. Emotional autonomy leads to increasing detachment from family. The bulk of physical growth and development is completed during this stage; however the body image issue may continue to be a source of much concern.

Social development

Adolescents experience dramatic biological changes related to puberty which can significantly affect psychosocial development. An increased awareness of sexuality and a heightened preoccupation with body image are fundamental psychosocial tasks during adolescence. Dramatic changes in body shape and size can cause a great deal of ambivalence among adolescents, especially among females, leading to the development of poor body image and eating disturbances or disorders if not addressed by family or health care professionals. A perceived delay in sexual maturation and biological development, especially among males, may lead to the development of poor body image and lowered self-esteem.[1] The social development of adolescents is best considered in the contexts in which it occurs; that is, relating to peers, family, school, work and community.

Peer influence

This is a dominant psychosocial issue especially during early adolescence. Peer groups serve a number of important functions throughout adolescence which includes providing a temporary reference point for a developing sense of identity, developing moral judgment and values through identification with peers, providing information about the world outside of the family and about themselves. It serves as powerful reinforcement during adolescence as source of popularity, status, prestige and acceptance.

Younger adolescents typically have at least one primary peer group with whom they identify whose members are usually similar in many respects and preference for same gender. During middle adolescence, peer groups tend to be more gender mixed. Less conformity and more tolerance of individual differences in appearance, beliefs and feelings are typical. By late adolescence, peer groups have often been replaced by more intimate relationships, such as one-on-one friendships and romances that have grown in importance.[10]

Family relationships

Regardless of family form, a strong sense of bonding closeness and attachment to family have been found to be

associated with better emotional development, better school performance and engagement in fewer high-risk activities, such as drug use. The level of parental supervision and monitoring necessary to promote healthy adolescent development can differ depending on the characteristics of the adolescent's peer and neighbourhood environments. During adolescence, parent–adolescent conflict occurs and this appears to be a necessary part of gaining independence from parents while learning new ways of staying connected to them.

School

School is a prominent part of adolescent life, where the individuals relate to and develop relationships with their peers and develop key cognitive skills. School also gives safety and stability. Adolescent perception of teacher fairness has also been found to be associated with positive adolescent development.

Community

Communities that are rich in resources provide support and opportunity for adolescent .and impact positive development. These include factors such as the socioeconomic characteristics of one's neighborhood, the types of resources available, the service systems within the community (including schools), religious organizations, the media and the people who live in the community.

Behavioral development

Experimentation helps them to fine-tune their development. Risk taking is an important way that shapes their identities, helps them try out their new decision-making skills and develop realistic assessments of themselves, other people and the world around them. They sometimes overestimate their capacities to handle new situations, especially under peer influence and this can cause adverse short and long term health effects.

Summary

Adolescence is not a period of 'normative disturbance'. In fact, the majority of teenagers are able to cope up with the challenges related to this period without developing any significant social, emotional or behavioral difficulties. However, adolescence is inherently said to be a period of difficulty as the healthy adolescent development is more about the avoidance of problems than about the growth of competencies in the day to day life. During the transition from childhood to adolescence, individuals develop more abstract characterizations of themselves and self-concepts become more differentiated and better organized. Though there is little evidence that psychological difficulties stem directly from hormonal changes at puberty, it is likely that the bodily changes of adolescence do play a role in the development of psychosocial disorders like depression and disordered eating behaviors. Pubertal maturation impacts the family relationships and vice versa. Since the last decade better understanding of the adolescent changes in the brain growth has highlighted the significant impact on the risk taking behaviors, psychological and mental health issues. Adolescents are creative, energetic and challenging because of the tremendous biological, psychosocial and cognitive growth and development. The interventions need to be tailored according to the level of development of an individual adolescent.[10]

Points to Remember

- ***Puberty is a dynamic period of development with rapid changes in body size, shape, and composition, which are sexually dimorphic.***
- ***Normal adolescent development includes cognitive skills, emotional maturity, self-identity and social development.***
- ***Health professionals should always give adequate time to an adolescent for determining his/her degree of biological maturity and level of cognitive development.***
- ***All stake holders dealing with adolescents should impart WHO life skills to empower the adolescents, teachers in schools and colleges and parents.***
- ***For adolescents to develop optimally, resilience and positive environment act as protective factors to reduce negative peer influence and risk taking behavior. A supportive environment in homes, community and teaching institutions is vital.***

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CLIPPINGS

Effect of Delayed Cord Clamping on Neurodevelopment at 4 Years of Age - A Randomized Clinical Trial.

Prevention of iron deficiency in infancy may promote neurodevelopment. Delayed umbilical cord clamping (CC) prevents iron deficiency at 4 to 6 months of age, but long-term effects after 12 months of age have not been reported. The effects of delayed CC compared with early CC on neurodevelopment at 4 years of age was studied.

Children who were included in randomized clinical trial conducted from April 16, 2008, through May 21, 2010, at a Swedish county hospital in the original study (n = 382) as full-term infants born after a low-risk pregnancy were invited to return for follow-up at 4 years of age. Wechsler Preschool and Primary Scale of Intelligence (WPPSI-III) and Movement Assessment Battery for Children (Movement ABC) were assessed by a blinded psychologist. Between April 11, 2012, and August 13, 2013, parents recorded their child's development using the Ages and Stages Questionnaire, Third Edition (ASQ) and behavior using the Strengths and Difficulties Questionnaire. All data were analyzed by intention to treat.

Delayed CC improved the adjusted mean differences (AMDs) in the ASQ personal-social (AMD, 2.8; 95% CI, 0.8-4.7) and fine-motor (AMD, 2.1; 95% CI, 0.2-4.0) domains and the Strengths and Difficulties Questionnaire prosocial subscale (AMD, 0.5; 95% CI, >0.0-0.9). Fewer children in the delayed-CC group had results below the cutoff in the ASQ fine-motor domain (11.0% vs 3.7%; *P* = .02) and the Movement ABC bicycle-trail task (12.9% vs 3.8%; *P* = .02). Boys who received delayed CC had significantly higher AMDs in the WPPSI-III processing-speed quotient (AMD, 4.2; 95% CI, 0.8-7.6; *P* = .02), Movement ABC bicycle-trail task (AMD, 0.8; 95% CI, 0.1-1.5; *P* = .03), and fine-motor (AMD, 4.7; 95% CI, 1.0-8.4; *P* = .01) and personal-social (AMD, 4.9; 95% CI, 1.6-8.3; *P* = .004) domains of the ASQ. No differences were found in WPPSI-III scores between groups.

Delayed CC compared with early CC improved scores in the fine-motor and social domains at 4 years of age, especially in boys, indicating that optimizing the time to CC may affect neurodevelopment in a low-risk population of children born in a high-income country.

Andersson O, Lindquist B, Lindgren M, Stjernqvist K, Domellöf M, Hellström-Westas L. Effect of Delayed Cord Clamping on Neurodevelopment at 4 Years of Age - A Randomized Clinical Trial. JAMA Pediatr. Published online May 26, 2015. doi:10.1001/jamapediatrics.2015.0358.

ADOLESCENT CARE

COMMON MEDICAL PROBLEMS IN ADOLESCENTS

***Chitra Dinakar**
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Abstract: *Medical problems in adolescence encompass a spectrum of disorders which would require a unique age appropriate approach including counseling. Disorders like hypertension and diabetes could reflect an early appearance of adult onset disease. Nutritional anemia and malnutrition with a childhood onset may persist or get aggravated in this age group. Acne and dysmenorrhea are puberty related adolescent onset disorders. A few of the commonly encountered problems in adolescents like hypertension, dysmenorrhoea, acne and nutritional anemia are discussed in this article.*

Keywords: *Adolescent, Hypertension, Dysmenorrhoea, Acne, Anemia*

Adolescent hypertension

Studies in India put the prevalence at 5-6% for hypertension and 10-12% for prehypertension among adolescent school children.[1] Hypertension even in adults is an under diagnosed silent killer disease. Routine blood pressure (BP) monitoring and plotting on nomogram charts is the key to diagnosis.

Starting at a BP of 115/75 mm of Hg the risk for coronary vascular disease doubles for every rise in BP of 20/10 mm of Hg.[2] There is a tracking of hypertension from childhood through adolescence and adulthood. One third of obese individuals could have hypertension.[3] Important causal factors include excess bodyweight, excess dietary sodium, reduced physical activity, inadequate intake of fruits, vegetables, and potassium and excess alcohol intake. In India the combination of low birth weight, increasing childhood obesity and life style changes predispose to metabolic syndrome X (hypertension, insulin resistance and hyperlipidemia). Control of hypertension reduces cardiovascular and renal morbidity and mortality.

The 4th report on diagnosis, evaluation and treatment of high blood pressure in children and adolescents highlights the standard management guidelines.[4] The same has been ratified by an Indian expert committee of the paediatric nephrology group which recommends that all children > 3 years including adolescents should get their blood pressure (BP) checked on every health visit.[5]

Definition and classification

Hypertension: Average systolic and/or diastolic blood pressure that is more than 95th percentile for gender, age and height on three or more separate occasions.

Prehypertension: defined as systolic or diastolic blood pressure between the 90th and 95th percentile. Adolescents having blood pressure >120/80 mm Hg, but below the 95th percentile are also included in this category.

Stage 1 hypertension: Systolic or diastolic blood pressure values exceeding the 95th percentile and up to 5 mm above the 99th percentile. Blood pressures in this range should be rechecked at least twice in the next 1-3 weeks, or sooner if symptomatic, before the patient is diagnosed to have sustained hypertension.

Stage 2 hypertension: Systolic or diastolic blood pressure values 5 mm or more above the 99th percentile. The presence of stage 2 hypertension should be confirmed on a repeat measurement, at the same visit. These patients require further evaluation within one week or immediately if they are symptomatic.

Other important aspects

Mercury manometers are the recommended devices to check BP. Oscillometric automated devices are not recommended as values can be falsely lower. The child should be sitting quietly for at least 5 minutes prior to BP check and avoid checking immediately after food/activity/drugs. Cuff should cover approximately 1/2 to 2/3 length of the upper arm and length of the bladder should be adequate to go around 80%-100% of arm circumference.

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Age, sex, height centile and BP charts are required for interpreting and classification of BP. Weight, BMI and detailed history including HEADDSSS (Home, Education and Employment, Activities, Drugs, Depression, Safety, Sexuality and Suicidality), family history and physical examination are essential for complete diagnosis and identifying secondary causes such as renal disease, coarctation of aorta, etc. Investigations are indicated to assess etiology, co-morbid conditions and target organ damage (Table I).

Non pharmacological measures include lifestyle changes (Table II). Drug therapy is indicated in hypertensive urgency/emergency, target organ damage (brain/heart/kidney/eye), co-morbid diabetes mellitus/renal disease, secondary hypertension and hypertension not responding to non-pharmacological measures (after 6 months trial). Target BP is <95th centile. However in the presence of co-morbid conditions like renal disease/ diabetes mellitus, target is <90th centile. Control of BP is more important than choice of drugs. Drugs commonly used include amlodipine, enalapril and losartan.

The young person with hypertension is the one most likely to know less about it, have no fear of consequences, be most likely to abuse alcohol/tobacco and stop drugs without informing the physician. Hence, there is a need to monitor for adherence, BP control, life style changes and drug side effects.

Dysmenorrhoea

Dysmenorrhea means painful cramping pain accompanying menstruation. The prevalence of this very common and distressing condition in adolescent girls in an Indian setting was found to be 80 %.[6] Roughly half of those affected had severe disabling symptoms affecting daily activities. There seems to be reluctance on the part of both adolescents and their parents in bringing the symptoms to the doctor's attention as suffering through the symptoms is considered acceptable and unavoidable. With targeted history to assess both symptoms and severity of the same, needless morbidity can be avoided. Dysmenorrhea is the leading cause of time lost from work and school.[7]

Table I. Evaluation of adolescent hypertension

Etiology	History: Family history, life style, clues to secondary causes Examination: Anthropometry, anemia, pulses, signs of renal/vascular disease, head to toe exam for secondary causes Investigations: Complete blood count, blood urea, creatinine, electrolytes, urine routine, renal ultrasound for all. Other investigations as clinically directed
Co-morbid conditions	Diabetes/ abnormal glucose tolerance test (GTT), hyperlipidemia, renal disease, drug abuse, obstructive sleep apnea
Target organ effects	Fundus examination Echocardiography Albuminuria and renal function tests - Renal disease as a consequence of hypertension occurs late

Table II. Life style modifications for control of hypertension

Weight loss	Reduce to BMI <85th centile
Dietary approach to stop hypertension (DASH)	Consume a diet rich in fruits and vegetables (8–10 servings/d), low-fat dairy products (2–3 servings/d), and reduced saturated fat and cholesterol
Low sodium	Ideally 1.5 g/d of sodium corresponding to 3.8 g/d of sodiumchloride
High potassium	4.7 g /day (present in most fruits and vegetables)
Physical activity	Ideal 60 min/day. Minimum 30 min/day
Drug use/abuse	Avoid smoking and alcohol
Media use	Less than 2 hours/day of all media like television/video games/mobile screen/computer

Symptoms associated with pain include gastrointestinal (anorexia, vomiting, constipation, diarrhoea), psychological (irritability, fatigue, depression, inability to concentrate on work) and other nonspecific symptoms (breast tenderness, frequency of micturition, profuse sweating, giddiness, fainting). Medication to relieve pain is extremely rewarding. There is no consistent co-relation between severity of symptoms and general state of health of adolescents, putting all girls at equal risk.[8]

The two clinically relevant types of dysmenorrhoea are (a) Primary: There is no associated pelvic pathology and pain is attributed to biochemical derangement. Increased levels of prostaglandins lead to the distressing symptoms,(b) Secondary: There is associated organic pelvic pathology like fibroids, endometriosis, pelvic inflammatory disease or uterine/vaginal structural anomalies.

The hallmark of typical primary dysmenorrhea is pain which starts with ovulatory cycles (6-12 months after onset of menarche), is most intense on the first day of the cycle with gradual waning. The pain is usually spasmodic and cramping and involves hypogastrium, back and inner thighs. It is also associated with other generalized symptoms. Differentiating the more common primary from secondary dysmenorrhea will obviate need for further investigations (Table III).

Management includes counselling on handling the distressing symptoms, reassurance on absence of serious organic cause and medical management which is effective in over 80% of those affected. Drugs used are paracetamol, mefenemic acid, indomethacin, naproxen and antispasmodics like buscopan. Choice of drug, dose and duration are directed to severity of symptoms and individual response. In non-responders a short 3 month course of oral contraceptives could be helpful. Adolescents not responding to treatment will require referral to a gynaecologist for further evaluation and therapy.

Acne vulgaris

Acne vulgaris (common acne) is an inflammation of the pilo-sebaceous unit. It affects adolescents of both sexes in the age group 10-19 years.[9] The sites of predilection are face, neck, upper arms, trunk and buttocks. The lesions of acne are a complex interaction between hormones (androgens) and bacteria (Propionibacterium acnes). Hormonal activity during normal puberty and the menstrual cycle predisposes to the formation of acne due to increase in sebum production. Polycystic ovary disease with the associated hyperandrogenic state is a risk factor in a subset of adolescents and may be a clue to the diagnosis. Emotional stress, comedonic cosmetics, occlusion or pressure on skin (phone, sports equipment) and tight fitting clothes (fashion accessories) are often important unrecognized exacerbating factors. Some common drugs exacerbating the problem in adolescents are oral contraceptives, topical or systemic steroids, lithium and hydantoin.

The three common types of skin lesions of acne vulgaris are (a) Comedones:Open (black heads) or closed (white heads) obstructive lesions which are first to appear, (b) Papules/Papulopustules:With or without inflammation (red). Size varies from 1-2 mm micro papules to large 5 mm nodules and (c) Nodules/Nodulo-ulcerative lesions/ Cysts:1-4cms in diameter. Nodules form from repeated ruptures and re-encapsulations with inflammation and abscess formation. They all associated with post inflammatory discoloration (red, violaceous, grey, brown).

Table III. Primary and secondary dysmenorrhoea - features

Features	Primary	Secondary
Onset	Within 2 years of menarche	Anytime
Pain characteristic	Cramping, spasmodic, typical locations	Generalized pelvic discomfort due to congestion.
Relation to menses	Usually 1st and 2nd days	Usually premenstrual onset and continues
Associated symptoms	Nausea, tiredness, fatigue, headache, vomiting	Dyspareunia, infertility, menorrhagia etc.
Pelvic findings	Normal	Suggestive of etiology
Management	Reassurance, Paracetamol, NSAID, Antispasmodic, Oral contraceptives	Investigate and treat cause

Table IV. Medical management of acne vulgaris

Mild Acne (1/4th of face involved, few papules / pustules but no nodules/ scarring)	**Moderate Acne** (½ of face involved, few to several papules/ pustules/ nodules and few scars)	**Severe Acne** (3/4th or more face involved with many papules, pustules, nodules and extensive scarring)
1. Benzoylperoxide (BP) gel (2%, 5% or 10%) is the first-line treatment.	1. Comedonal: Topical retinoid +/- topical antibiotic combination. If inadequate response, increase strength or change type of topical retinoid.	1.Initial treatment of oral antibiotic and topical retinoid 2. Referral to a dermatologist
2. Topical antibiotics (Clindamycin & erythromycin) along with benzoyl peroxide gel if response is low.	2. Inflammatory mixed: Step I Benzoyl peroxide gel (BP) or topical antibiotic combination +/-Topical retinoid +/- oral antibiotic (Tetracycline, doxycycline, erythromycin, minocycline)	Isotretinoin 0.5 to 2.0 mg/kg in 3 divided doses with food for 15-20 weeks, very effective for severe or moderate acne refractory to other treatments. Improvement typically after one to two months of use.
3.Topical retinoid (Tretinoin) are effective in comedonal and papulo pustular acne but detailed instructions for gradual increase in concentration should be given as 0.025% cream or 0.01% gel, 0.05% cream or 0.025% gel 0.1% cream	Or Topical Retinoid + oral antibiotic (Minocycline 50-100mg bid tapered to 50mg once daily as acne lessens) Step II If inadequate response, increase strength or change type of topical retinoid. Consider oral contraceptives and/or spironolactone in females who have premenstrual or menstrual flare.	Isotretinoin is teratogenic and pregnancy must be avoided during treatment. Both tetracycline and isotretinoin cause pseudotumor cerebri and therefore combination should be avoided. *Monitoring blood lipids is required as 25% patients experience increased plasma triglycerides.
Continue at lowest effective maintenance dose	Refer to a dermatologist.	

Pitted, depressed or hypertrophic scars may follow all types but are especially common in cases of nodulo-cystic acne. Round nodules may coalesce to form linear mounds or sinus tracts. Besides scarring, acne may give rise to major psychological side effects such as reduced self-esteem and depression.[10] Medical management of acne includes an evaluation of types of lesions, extent of involvement and severity of the inflammation / infection (Table IV).[11]

The pediatrician should take care to give clear instructions on topical applications. Benzoyl peroxide and retinoids need to be applied only at night times and washed away in the morning as they can cause pigmentation with sun exposure. Retinoids can cause scaling and peeling, especially in dry weather and hence can be used for 1-2 hours initially, in lower strength and gradually increased for overnight use. The response time can be 2-4 months. Expectations on rapid response should be put in perspective and counselling for same is required. Treatment is generally for several months, cyclically, depending on need. It is necessary to follow up regularly once in 2-4 weeks for treatment modification based on clinical evaluation and side effects of treatment. Application of advertised products and quick fixes are to be avoided. Drinking adequate fluids, eating a balanced diet, adequate exercise and sleep should be stressed.

Counselling for body image concerns and tips for coping strategies are an integral part of treatment in adolescents. Surgical measures and other cosmetic therapies include comedone extraction, injecting corticosteroids into inflamed comedones, drainage by lancing, light therapy and laser surgery to reduce the scars of acne.[12] Long term follow up studies are necessary to assess the efficacy of some of these procedures.

Table V. Age group and hemoglobin (g/dL) to classify anemia (WHO)

Age group	No Anemia	Mild Anemia	Moderate anemia	Severe anemia
10-11 yrs	≥11.5	11 -11.4	8 -10.9	<8
12- 14 yrs	≥12	11 – 11.9	8- 10.9	<8
15 yrs -19 yrs (girl, not pregnant)	≥12	11 – 11.9	8- 10.9	<8
Pregnant girl	≥11.0	10 -10.9	7 – 9.9	<7
15 -19 yrs (boy)	≥13	11 -12.9	8 -10.9	<8

Nutritional anemia

As per the WHO global data on anemia, India has a prevalence of 74%, classifying it as a major public health problem.[13] The commonest cause of anemia continues to be nutritional iron deficiency. The consequences of anemia in a young population are, decreased work capacity due to fatigue, increased maternal morbidity/mortality, increased low birth weight and cognitive impairment with learning difficulty. Anemia is classified as mild, moderate and severe based on Hb levels at different ages (Table V).

Almost 58% of pregnant women in India are anemic and it is estimated that anemia is the underlying cause for 20%-40% of maternal deaths. India contributes to about 80% of the maternal deaths due to anemia in South Asia.[14] The prevalence of anemia among girls (Hb <12 g %) and boys (Hb <13 g %) is alarmingly high at 56%-68 % (Fig.1) and 30% respectively as per National Family Health Survey-3 (NFHS-3) and the National Nutrition Monitoring Bureau Survey (NNMBS), 2006.

The factors contributing to development of anemia in the adolescent include:

(i) Changing lifestyle, temperament, newly acquired freedom, irregular meal timings, peer pressure regarding food choices, increased consumption of junk foods.

(ii) Gender bias with reduced availability of nutritious food to the adolescent girl child

(iii) Rapid growth spurt with hormonal changes and mismatched diet (eg. testosterone which affects erythropoiesis, peaks during adolescence).[15]

(iv) In adolescent boys, rapidly increasing muscle mass requires higher amount of iron.

(v) In adolescent girls, menstrual losses, increased demand for iron due to growth spurt and teenage pregnancy.

(vi) Fear and anxiety of weight gain leading to decrease in proper diet in the girl child.[16]

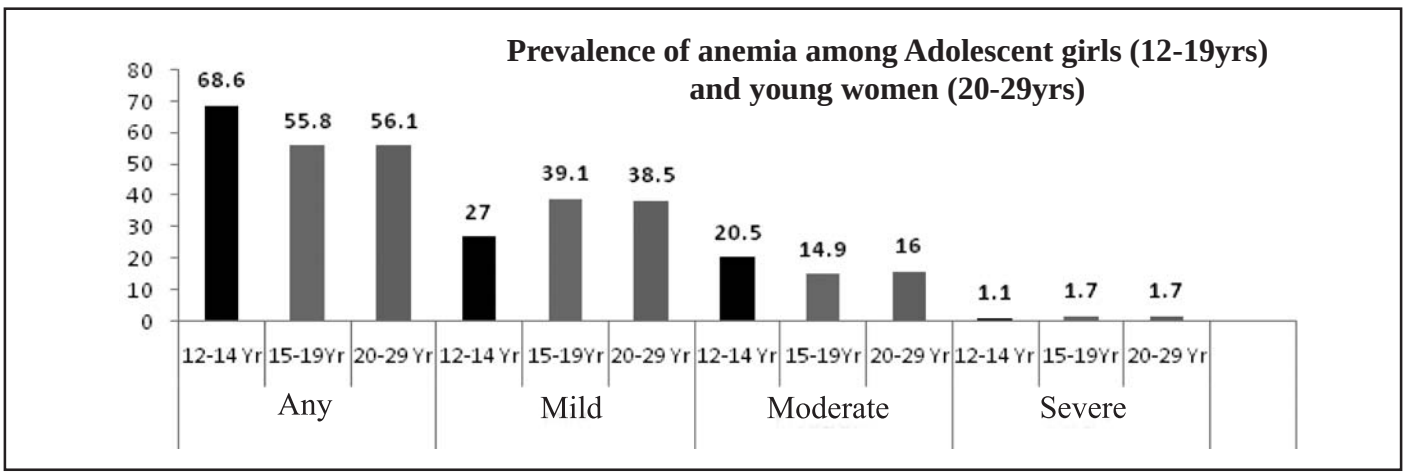

Fig.1. NFHS-3 and the National Nutrition Monitoring Bureau Survey (NNMBS), 2006.

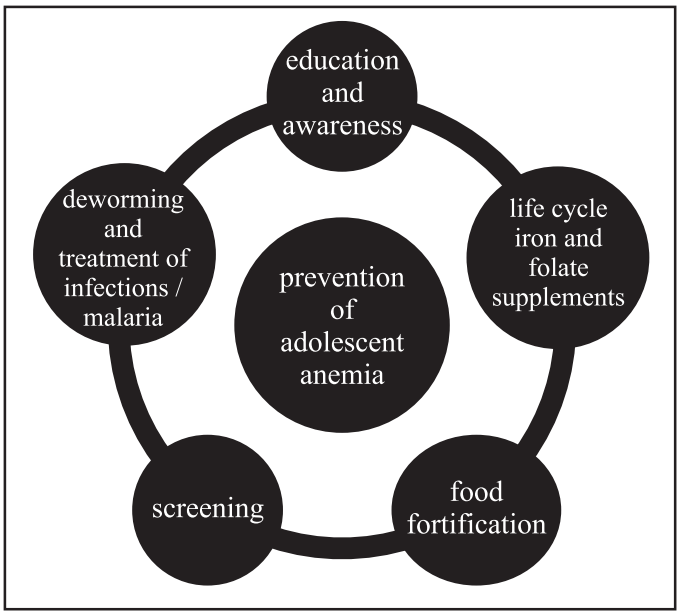

Fig.2. Prevention of adolescent anemia

(vii) Reluctance to visit doctors and poor compliance to treatment in both sexes

(viii) Traditional food habits which may lead to iron deficiency e.g. predominantly vegetarian foods poor in haem iron, inadequate intake of raw fruits and vegetables (vitamin C present in them is an enhancer of absorption) and consuming tea/coffee/cocoa drink along with meals (caffeine, tannin and phenolic compounds interfere with iron absorption).

Iron rich foods include animal protein, green leafy vegetables, sprouts, jaggery and fermented cereals. Consuming these as a snack between meals will further enhance absorption. Phytates present in cereals and legumes can reduce absorption. Several preventive steps help in management of adolescent anemia. Screening of adolescents with Hb estimation (clinical examination may miss mild/moderate anemia), awareness programs fostering healthy dietary habits and reduced consumption of junk food, increased consumption of foods rich in iron, weekly iron and folate supplementation, fortification of commonly consumed food eg. atta with iron, and bi annual deworming of all adolescents can contribute to a significant decrease in anemia (Fig.2).

Additionally, prevention of adolescent pregnancies by delaying age of marriage and providing family planning strategies, reducing the total number of pregnancies and spacing of births can improve the state of anemia in young women.

Treatment is directed to severity and includes dietary interventions in addition to therapeutic iron and follow up for normalization of Hb and prevention of relapse (Table VI). [17]

Blood loss due to parasitic infestations or menstruation and continued poor intake of iron rich foods can result in poor response or recurrence. A thorough history and physical examination will help to detect other nutritional deficiency anemias like megaloblastic anemia, haemolytic anemia and leukemia. Parenteral iron is used in selected cases only when oral iron is not tolerated or rapid increase in Hb is desired (in pregnancy). Blood transfusion is reserved for Hb less than 4 gm/dL and those adolescents with Hb 4–6 gm/dL with complications such as dehydration, shock, impaired consciousness, heart failure, deep and laboured breathing or very high malarial parasitaemia (>10% of RBC). Packed cells 10 ml/kg over 3-4 hours are preferred.

If packed cells are not available, whole blood 20 ml/kg over 3-4 hours with diuretics may be used with close monitoring for volume overload. Oral therapeutic iron is indicated following transfusion with Hb evaluation on follow up. All adolescents who have successfully completed treatment with recovery of Hb need to continue with weekly supplementation of iron and folate (Box 1).[17]

Table VI. Management of adolescent anemia based on Hb levels

Level of Hb in gm/dl	Treatment	Followup	Referral
Mild (10 – 11.9)	60 mg of elemental iron daily for 3 months	Every month clinically. Repeat Hb after 3 months to confirm >12 Hb	In case of no response clinically or non-normalization of Hb at 3 months, refer
Moderate (8 – 9.9)	60 mg of elemental iron daily for 3 months	Every month clinically. Repeat Hb after 3 months to confirm >12 Hb	In case of no response clinically or non-normalization of Hb at 3 months, refer
Severe < 8	Refer urgently		Needs additional evaluation for causes

Box 1. Weekly iron and folic acid supplementation (WIFS); A New National Health Program[17]

- Weekly iron (100 mg) and folic Acid (500mcg) to adolescent boys and girls 52 weeks a year are administered in schools, every Monday. Out of school adolescents also need to be supplemented. Biannual de-worming (Albendazole 400mg) is done, six months apart, for control of worm infestation. Information and counselling are given for improving dietary intake and for taking actions for prevention of intestinal worm infestation.

In summary, adolescent anemia needs to be tackled using a multipronged approach of prevention, screening, treatment and follow-up. Physicians need to address this scourge keeping in mind the life cycle implications and intergenerational impact of unaddressed anemia.

Points to Remember

- ***Hypertention, dysmenorrhen, acne vulgaris and anemia are the commonly encountered medical problems in adolescents.***
- ***Routine blood pressure monitoring and plotting on nomogram is the key to diagnosis of hypertension.***
- ***Life style modification is an important component in the management of adolescent hypertension.***
- ***Dysmenorrhea is commonly present in adolescent girls and they respond well to medical management.***
- ***Acne vulgaris affects both adolescent boys and girls and requires prolonged topical therapy and dermatologist opinion in moderate to severe cases.***
- ***Nutritional iron deficiency anemia is highly prevalent among adolescent girls. Weekly iron and folate supplementation, biannual deworming and improvement in nutrition and they prevention strategies of the new national health program.***

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ADOLESCENT CARE

ADOLESCENT OBESITY

***Karthik Kumar B**
****Hemchand K Prasad**

Abstract: *Adolescent obesity is a common clinical problem faced by a pediatrician. Annual measurement of waist circumference (abnormal: ≥90th percentile) and Body Mass Index (BMI) (abnormal: > adult equivalent of 23 for overweight and 27 kg/m^2 for obesity) is mandatory in all adolescents. Though all overweight and obese adolescents must be screened for metabolic risk factors; endocrine screening is at the clinician's discretion. A physician treating an obese adolescent must have a low threshold to investigate, high threshold to medicate and a very high threshold to refer to a surgeon. A high index of suspicion is needed to look for polycystic ovary syndrome in girls. A cafeteria of medications are available for metabolic consequences arising from obesity, which may be useful as a temporary resort. Combination of therapy dietary changes, physical activity and lifestyle modifications is the key for sustained long term success.*

Keywords: *Adolescent obesity, Polycystic ovary syndrome (PCOS), Metformin, Metabolic Syndrome.*

Adolescent obesity is not a cosmetic problem, less likely an endocrine problem, more likely a metabolic problem and almost always a life style related issue. Adolescent obesity results in development of cardio-metabolic complications, both, in adolescence and in adulthood.[1,2,3] It is the responsibility of the primary pediatrician to recognise adolescent obesity prior to the development of cardio metabolic complications. The purpose of this article is to help the paediatrician to assess an adolescent who presents with obesity in his office practice, evaluate him/her and initiate remedial measures in an evidence based manner.

Clinical history

History taking in an obese adolescent should be methodical. One must be able to ascertain the risk factors for commonly presenting nutritional obesity pertaining to:

- Dietary recall (3 day/ recall and common danger foods he/she would consume)
- Physical activity
- Screen time (time spent on television, mobiles, computers and laptop).

Detailed history may help in identifying a specific underlying etiology (Table I).

Table I. Historic details to identifying etiology

Details in history	Respected etiology
Lethargy, poor growth and pubertal delay	Hypothyroidism
Short stature, headaches	Cushing's disease
Headaches	Pseudotumor cerebri and prolactinoma
Headaches, hyperphagia and focal neurological deficit	Intracranial tumor
Poor vision, hearing deficit and developmental delay	Bardet Biedl syndrome
Drug intake	Chronic steroid therapy and anti epileptic drugs like sodium valproate

Symptoms also point towards the presence of complications and comorbidities (Table II).

Table II. Complications/ comorbidities in obesity

Symptoms	Co-morbid condition
Snoring/ nocturnal sweating/ enuresis/ sleep disturbance and day time sleepiness	Obstructive sleep apnea syndrome (OSAS)
Recurrent abdominal pain	Non-alcoholic steato hepatitis [Non alcoholic fatty liver disease (NAFLD)]
Sudden weight loss without exercise	Development of type 2 diabetes mellitus
Recurrent hip pain	Slipped capital femoral epiphyses
Anxiety or school avoidance	Psychological affect because of obesity

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Table III. Quick guide to evaluation of obesity in adolescents in office practice

	Boys					Girls				
Age (in years)	Over weight[a] (BMI in kg/m^2)	Obese[a] (BMI in kg/m^2)	Abnormal waist circum ference[b] (in cm)	Hyper tension[c] at height 50th percentile	Hyper tension[c] at height 97th percentile	Over weight[a] (BMI in kg/m^2)	Obese[a] (BMI in kg/m^2)	Abnormal waist circum ference[b] (in cm)	Hyper tension[c] at height 50th percentile	Hyper tension[c] at height 97th percentile
11	18.7	21.5	82.3	17/76	121/78	19.3	23.0	82.1	117/75	120/77
12	19.5	22.6	85.8	120/76	123/79	20.2	24.1	85.5	119/76	122/78
13	20.2	23.4	88.8	122/77	126/79	21.1	25.2	88.3	121/77	124/79
14	20.8	24.2	91.2	125/78	128/80	21.8	25.9	90.3	122/78	125/80
15	21.4	24.9	93.2	127/79	131/81	22.3	26.3	91.6	123/79	127/81
16	22.0	25.5	94.9	130/80	134/82	22.6	26.5	92.5	124/80	128/82
17	22.6	26.0	96.3	132/82	136/84	22.9	26.7	93.3	125/80	128/82
18	23.2	26.6				23.2	26.8			

Legends: a - The classification of overweight and obese based on prescriptive approach BMI classification of the IAP 2015 charts6 linking to adult cardiovascular risks in South Asians.[6]

b - Cut-offs based on pan Indian waist circumference cut-offs7 (2014). Although the paper recommends 70th percentile, the cut-offs provided here are 90th percentile to classify as metabolic syndrome.

c - Cut off for defining hypertension based on NHLBI references and cut-offs provided for adolescents at 50th and 97th percentile of height. For BP at other height percentiles, the reader is requested to refer original article.[12]

A family history of any of the following is an indication to screen for dyslipidemia.[4]

- Either parent, grandparent, aunt, uncle or sibling aged < 55 years (males) and < 65 years (females) having any of these: myocardial infarction, stroke, angina, coronary artery by-pass, stent, angioplasty or sudden cardiac death

- Parents with total cholesterol > 240 mg/dL or known lipid abnormalities in parents.

Clinical examination

A methodical clinical examination is pivotal in an obese adolescent. As an initial measure, one needs to concentrate on the anthropometry and establish the presence and degree of obesity. A diagnosis of obesity is made from the growth chart. Height (in cm) is measured by a calibrated stadiometer and weight (in kg) is measured on an electronic digital weighing scale and Body mass index (BMI) is calculated using the formula BMI = Weight (in kg)/ Height (in m^2). Interpretation of BMI to diagnose obesity is an area of controversy. Conventional definitions of overweight and obesity include BMI $\geq$ 85th percentile and BMI $\geq$ 95th percentile, respectively.[5] However, childhood obesity is known to track on to adulthood and extensive evidence is available on the cardiometabolic risk in obese adolescents. Asians are prone for metabolic risk at lower BMI. Hence, the Indian Academy of Pediatrics (IAP) in 2015 came out with charts and cut-offs linked to adult cut-offs of 23 and 27 kg/m^2 as "overweight" and "obesity" (shown in Table III).[6]

Waist circumference is the other parameter found to be useful in clinical settings. It must be measured with a stretch resistant tape, applied horizontally just above the upper lateral border of right ileum at the end of normal expiration and recorded to the nearest 0.1 cm. It is interpreted as abnormal, based on cut-offs shown in Table III.[7]

Subsequently, one must look for markers of an underlying genetic or endocrine cause and also subtle or overt markers of evolving metabolic complications. The red flag signs that one must NEVER miss include:

- Short stature
- Growth velocity < 25th percentile
- Hypoplastic genitalia
- Extra digits
- Severe hypertension (BP > 99th percentile)
- Focal neurological deficit

Assessment of Tanner's stage is mandatory in all adolescents, more so in obese adolescents. Obese children have a tendency to enter into puberty early. Also, delayed puberty in an adolescent (No breast stage 2 by 13.5 years in females or pre-pubertal testes > 14 years in males) may be a pointer to Bardet Biedl syndrome, hypothyroidism or hypopituitarism. Blood pressure measurement is mandatory in all obese adolescents. Hypertension in obesity is multifactorial (altered renal sodium handling, increased systemic vascular resistance and activation of the sympathetic nervous system).

One must examine the adolescent for features of complications of obesity like insulin resistance (acanthosis nigricans, skin tags and furunculosis); tonsillar enlargement and typical facies of obstructive sleep apnea syndrome (OSAS); hepatomegaly (fatty liver in non-alcoholic fatty liver disease - NAFLD); abnormal gait (slipped capital femoral epiphyses); papilledema and VI cranial nerve palsy (pseudotumor cerebri). Examine all adolescents for pubertal gynecomastia (resulting from increased estrogen levels from aromatase in adipose tissue). One must look for features of an underlying endocrinopathy like goitre and delayed relaxation of reflexes (hypothyroidism); abdominal striae and truncal obesity (Cushing's disease); midline defects and cherubic facies (GH deficiency); short 3rd and 4th metacarpals (pseudohypoparathyroidism). Never forget to look for pointers to genetic obesity: Hypoplastic genitalia (undescended testes, micropenis, hypoplastic labia) and typical phenotypic features of Prader Willi syndrome; optic fundus features of retinitis pigmentosa and polydactyly (Bardet Biedl syndrome).

In menstruating adolescent girls, one must always look for features of polycystic ovary syndrome (PCOS). Examine the adolescent girl for clinical hyper androgenism and insulin resistance. Hyperandrogenism in adolescent girls is assessed by Ferriman Gallwey score[8] (Fig. 1). Assess hair growth in the **9** androgen dependant areas shown in the Fig.1 by direct comparison or visual comparison by the adolescent girl. Add up the score and get the total score (Score >8 is abnormal). In extreme cases of PCOS, there may be signs of virilisation such as clitoromegaly, temporal balding, voice change, acne and seborrhea.

Evaluation

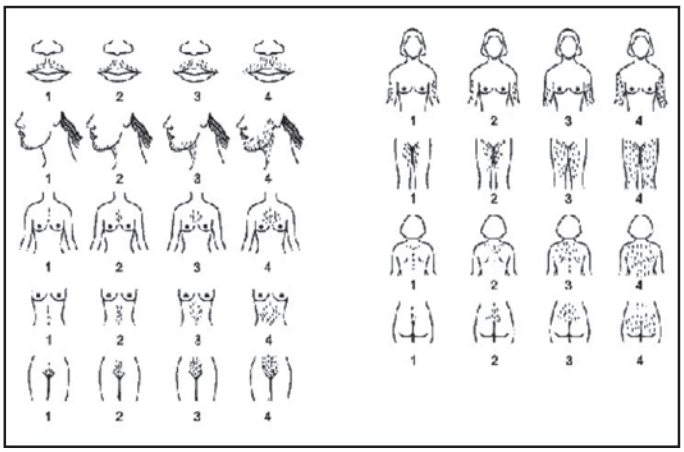


Fig.1. Ferriman Gallwey Score to assess hirsuitism in adolescent girls

Evaluation of an obese adolescent is a 3 step process (Fig. 2a)

Step-1 - Screen for underlying endocrine abnormality (if needed)

Step-2 - Baseline screen for cardio-metabolic risks in all overweight and obese adolescents

Step-3 - Screen for specific metabolic abnormalities as per the clinical judgement and baseline screen.

Step-1: The endocrine society recommends endocrine work-up of obesity, only, if the height velocity is attenuated with respect to the family or pubertal status OR child has markers of specific endocrine dysfunction. Commonly recommended screening tests include, a fasting thyroid profile (Free T4 and TSH) and overnight dexamethasone suppression test (ONDST) (Cushing's disease screen). In the ONDST, a dose of oral dexamethasone is given at 0.3 mg/m^2 (maximum dose: 1 mg) at bedtime and serum cortisol is estimated at 8 am next day morning. Serum cortisol > 5µ/dL is interpreted as positive screen and warrants specialist referral. One must remember that obese adolescents and adolescent girls with PCOS have a marginally raised TSH between 6 and

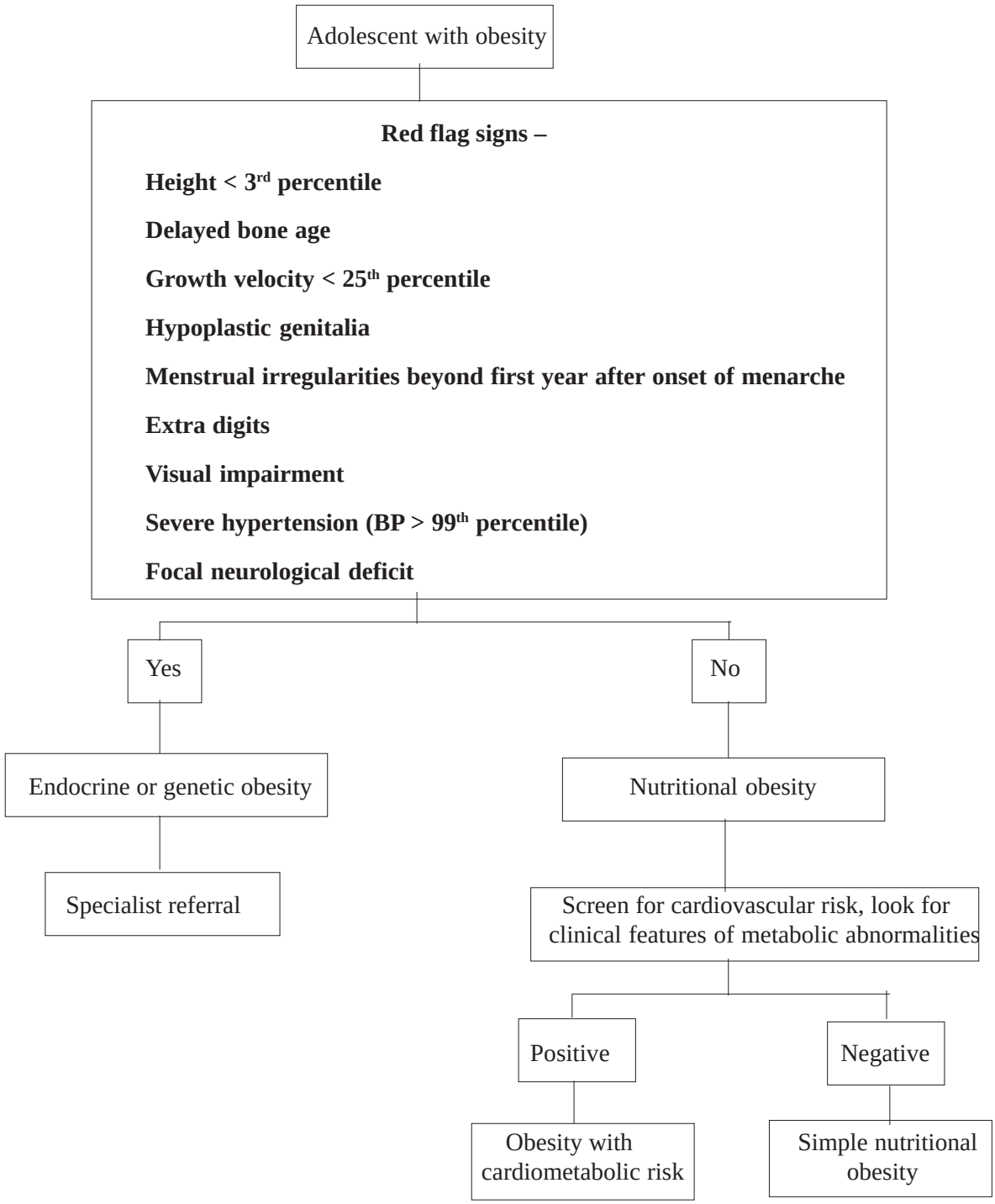


Fig. 2a. Approach to adolescent obesity

10 mIU/mL. This is an effect of the obesity (TSH release mediated by leptin) and not the cause of obesity. Growth hormone deficiency can rarely present as adolescent obesity. Performance of GH stimulation test may yield a false positive result. Bone age provides the vital clue in such cases. Delay of bone age > 2 years is a pointer to a likely GH deficiency.

Step-2: All overweight and obese adolescents must have their fasting blood sugar, liver function test and fasting lipid profile estimated. These laboratory reports are interpreted as indicated in Table IV. Diagnosis of metabolic syndrome in adolescents is an area where there is no consensus.[9,10,11] A practical definition of adolescent metabolic syndrome is the presence of three out of the

Table IV. Important laboratory cut-offs for screening tests in obesity evaluation

	Acceptable	Borderline	Abnormal
Total cholesterol	<170 mg/dL	170-199 mg/dL[d]	≥200 mg/dL[e]
LDL cholesterol	<110 mg/dL	110-129 mg/dL[d]	≥130 mg/dL[e]
Triglycerides			
0-9 years	<75mg/dL	75-99 mg/dL[d]	≥100 mg/dL[e]
10-19 years	<90 mg/dL	90-129 mg/dL[d]	≥130 mg/dL[e]
HDL cholesterol	>45 mg/dL	40-45 mg/dL[f]	< 40 mg/dL[g]
Fasting blood sugar	<100 mg/dL	100-125mg/dL[a]	>125 mg/dL[c]
Post prandial blood sugar	<140 mg/dL	140-200mg/dL[b]	>200 mg/dL[c]

a - Impaired fasting glucose, b - Impaired glucose tolerance, c - Overt diabetes (as per the WHO criteria)

d - Above the 75th percentile, e - Above the 90th percentile, f - Below the 25th percentile, g - Below the 10th percentile

following five criteria:

1. Abdominal obesity (waist circumference >90th percentile)

2. Hyper-triglyceridemia ≥ 110 mg/dL[12]

3. High density lipoprotein (HDL) cholesterol ≤ 40 mg/dL[12]

4. Blood pressure ≥ 90th percentile according to age, gender and height[13]

5. Fasting blood glucose ≥ 100 mg/dL

Step-3: Adolescents, classified to have metabolic syndrome, or, with even one of the five criteria positive or clinical features of insulin resistance, need to undergo a detailed evaluation of the metabolic profile. A formal glucose tolerance test (GTT) is mandatory in such cases. The child should consume a normal carbohydrate diet for 3 days, followed by taking fasting samples for blood sugar and insulin. This is followed by administration of 1.75 gm/kg of oral glucose and taking samples for blood sugar and serum insulin at 2 hours.

HOMA (Homeostatic model for assessment of insulin resistance) is the most useful index to assess insulin resistance in clinical practice. It is calculated as: HOMA = (Fasting glucose in mg/dL x fasting serum insulin in μU/ml)/405. HOMA is considered abnormal if ≥ 2.5 before puberty or ≥4.5 during puberty. One must remember that HOMA is superior to isolated glucose and insulin levels to assess insulin resistance.[14]

Type 2 diabetes mellitus: A diagnosis of type 2 DM is made if WHO criteria are satisfied (Table IV). Once a diagnosis of type-2 DM is established, it is important that:

a) Type 1 diabetes is ruled out - By testing C-peptide levels (to assess endogenous insulin secretion) and autoimmunity (anti GAD antibodies).

b) Complications are ruled out - By screening for nephropathy (urine albumin creatinine ratio) and indirect ophthalmoscopy and fundus examination for retinopathy screen.

Adolescents with suspected NAFLD[15] should undergo ultrasound evaluation. Ultrasound abdomen may show bright liver when >30% hepatocytes have steatosis. Liver biopsy to look for necroinflammation and peri portal tract fibrosis, is, of academic interest. Authorities also recommend ruling out viral infections (hepatitis B and hepatitis C) auto immune hepatitis and drug induced hepatitis prior to labeling as NAFLD.

PCOS

The adult criteria[16,17] to diagnose PCOS include NIH criteria, Rotterdam criteria and Androgen excess society criteria, which, when applied in adolescents can lead to over-diagnosis. Hence, most centres adopt the Sultan and Paris criteria[18] which warrants 4 out of 5 criteria to be present to make a diagnosis of PCOS:

a) Oligomenorrhea or amenorrhea

b) Clinical hyperandrogenism

c) Biochemical hyperandrogenism

d) Clinical or biochemical features of hyperinsulinemia

e) Polycystic ovaries on ultrasound.

To make a diagnosis of PCOS, common causes of secondary PCOS including hypothyroidism, non-classical congenital adrenal hyperplasia, prolactinoma, adrenal tumor and Cushing's disease must be ruled out. Hence, screening tests include: Free T4 and TSH, 17 OH Progesterone (screen positive if > 2 ng/mL), serum prolactin and ONDST. Subsequently their hormonal profile is assessed in the follicular phase (day 5 after stoppage of periods for convenience). LH/FSH ratio > 1 in the follicular phase, total testosterone levels > 60 ng/dL support the diagnosis of PCOS.

Ultrasound is a less useful tool in adolescents[19] because, variable ovarian volumes are present in growing adolescents, polycystic ovaries may be seen in normal girls and trans vaginal sonography is not feasible. An increased ovarian volume and presence of >10 follicles without a dominant follicle of >10mm supports a diagnosis of PCOS.

Other rare orthopedic complications one must look for include: Blount's disease, slipped capital femoral epiphyses and flat feet by appropriate X-rays. DEXA scan may be done to compute the body fat percentage, for academic interest.

Management

Diet and exercise: All obese adolescents must have advice provided on dietary modifications, exercise regimen to be followed and life-style modifications to be made. There are no uniform guidelines on these aspects applicable for all adolescents across socio economic strata. These measures need to be adopted are to be tailor made for the socio cultural aspects of the family. A useful measure that could be adopted would be asking the mother to maintain a food diary for 3 days and adding up the caloric intake of food consumed versus the recommended caloric intake for the age. Foods that the child is specifically interested which are unhealthy should be avoided. Consistency of the regimen during weekends must be emphasised. In families with more than one child, everyone must adhere to the advice given. The stress that is placed on extra schooling and extra-curricular activities should be shared with specific time for exercises. Abnormal laboratory tests often serve as an ultimatum for families to be more strict with the diet. Marking of BMI on the growth chart and waist circumference shown on the percentile charts may have an impact on the family. Eating outside often, binge eating and eating in parties must be restrained. Physical activity must be tagged with the specific interest of the adolescent: aerobics and sports activities can become a part of the daily schedule of the adolescent as per his interest. Caloric intake should be matched with calories burnt in specific activities. Institutional placement may not be logically feasible in our country. The list is exhaustive, easier written than practiced. Constant motivation and adherence is the key to success.

Metabolic syndrome (Fig. 2b): Key factor in the process for underlying metabolic syndrome is insulin. Hence, adolescents with severe, fasting and post prandial hyperinsulinemia with strong family history of type 2 DM, dyslipidemia, impaired fasting glucose or impaired glucose tolerance may be a candidate for metformin therapy. Sustained release forms of metformin (extended release) are preferred in adolescents. It is begun on a dose of 250 mg once a day and slowly escalated over a period of 4 weeks to 500 mg twice a day. Increased bowel movements, nausea, vomiting and reduced appetite are common side effects that may need dosage reduction. Metformin causes improvement in anthropometry and insulin sensitivity. However, one must remember the following caveats to metformin therapy:

a) If given as a first line measure, adolescents tend to have unrealistic expectations and do not adhere to dietary and exercise regimens. Hence, metformin is started as a measure after ensuring some weight loss.

b) Metformin has a doubtful effect on lipids.

c) Metformin reduces sub-cutaneous fat, not the visceral fat that is metabolically active.

Metformin is given as a rescue measure for 1 year and slowly withdrawn after the metabolic parameters improve.

Type 2 Diabetes mellitus (Fig. 2c): Management depends on the mode of presentation and HbA1C. Diet and exercise remain the key to control of type 2 DM (in contrast to type 1 DM who consume normal diet).

NAFLD (Fig. 2d): Diet and exercise are the cornerstone in management of NAFLD. Thiazolidinediones like rosiglitazone and pioglitazone are not FDA approved in adolescents.

Dyslipidemia (Fig. 2e): Adolescents with abnormal lipid levels must be initiated on regimen of diet and exercise, as with other metabolic complications. Statin preparations approved for usage in adolescents include:

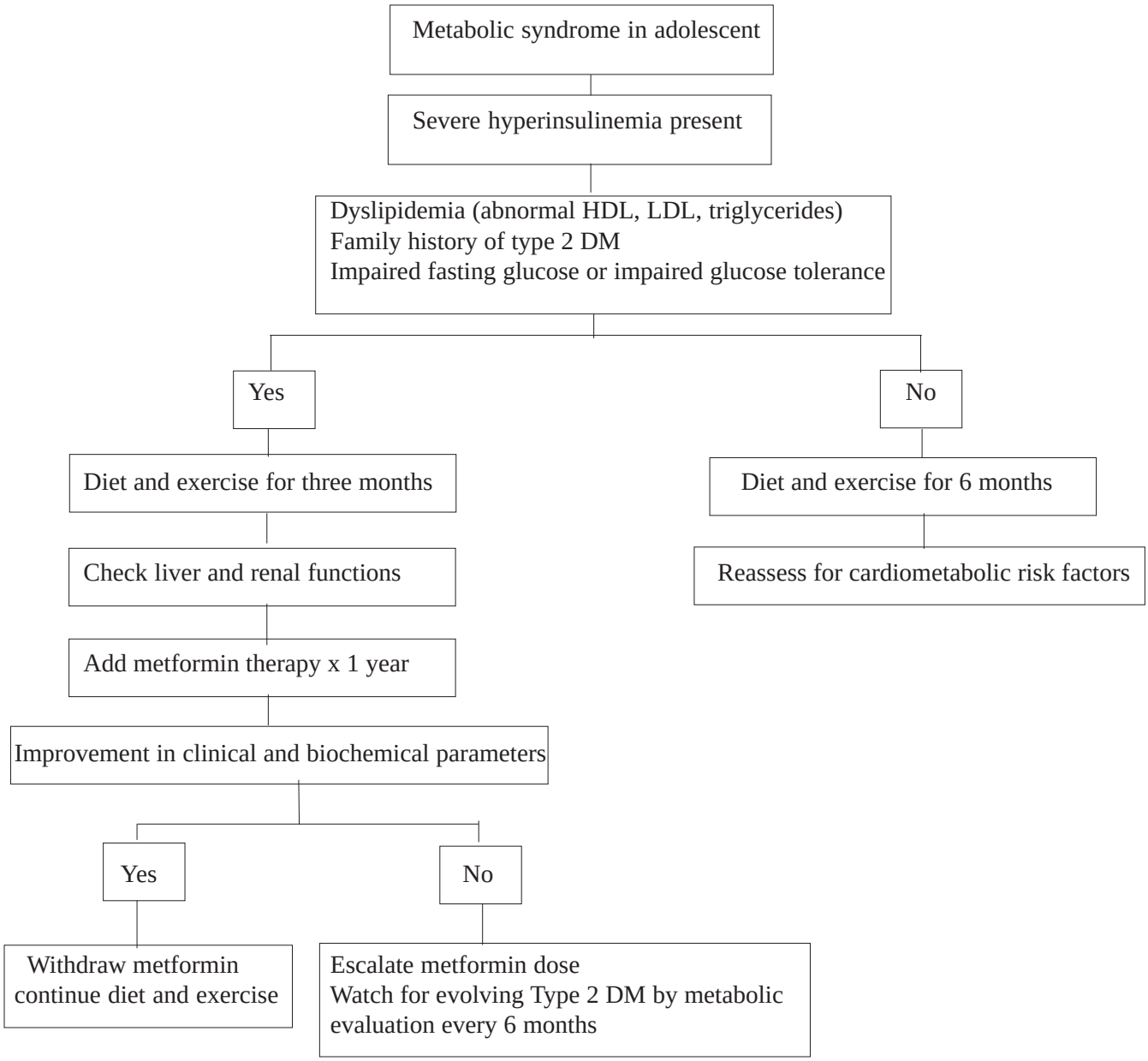


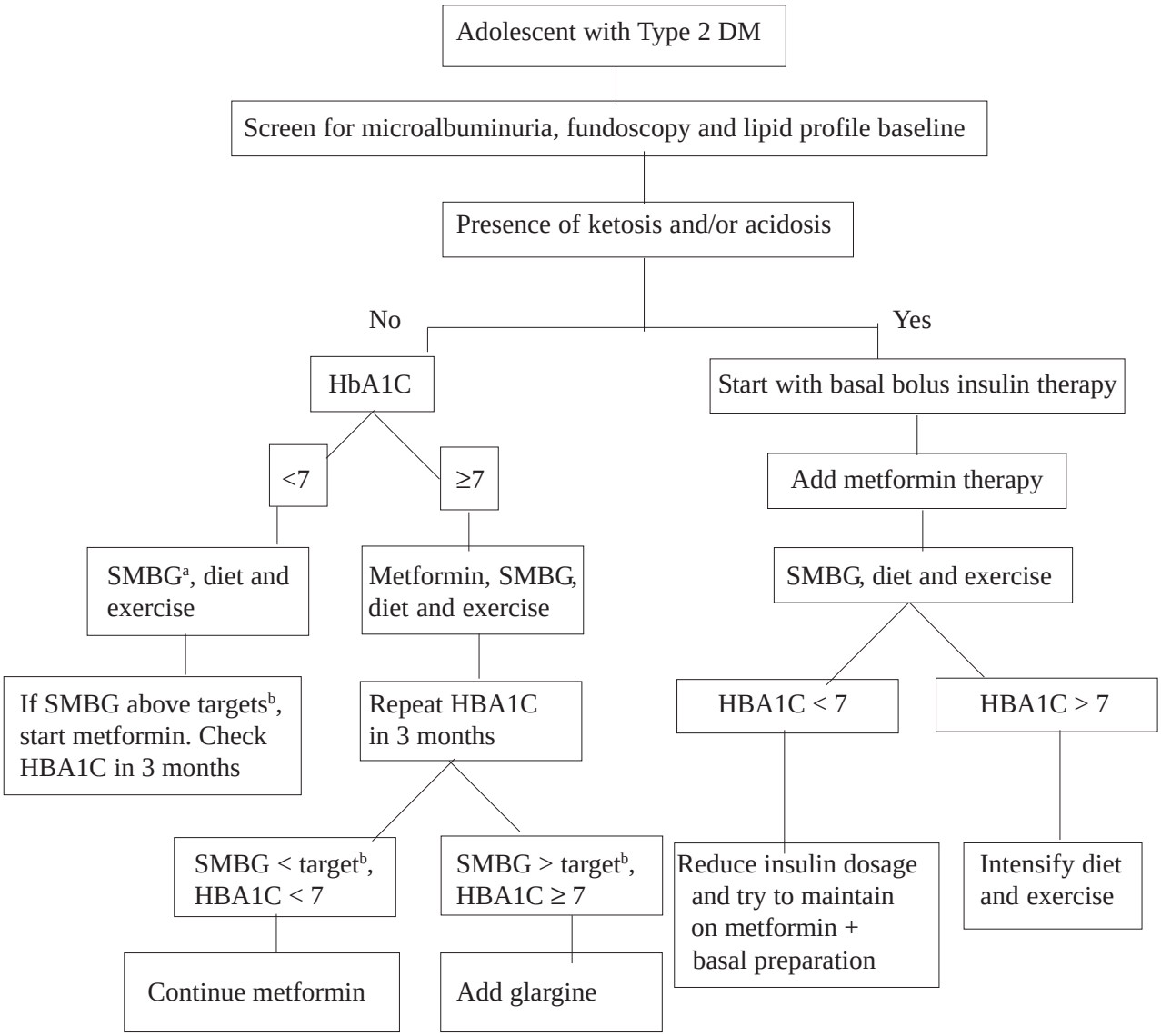
Fig. 2b. Approach to metabolic syndrome in adolescents

pravastatin, simvastatin, fluvastatin, atorvastatin and lovastatin. Elevations in triglyceride levels > 500 mg/dL would warrant therapy with fibrates and niacin.

Hypertension (Fig. 2f): Beta blockers and diuretics are not recommended in the adolescents owing to their diabetogenic potential. Calcium channel blockers and angiotensin converting enzyme inhibitors are preferred drugs.

Pubertal gynaecomastia: This condition needs reassurance only, most of the times. Diet and exercise remain the keystone in management. Gynecomastia that persists despite weight loss, causes significant distress to the adolescent and is painful, may, respond to a short course of tamoxifen therapy at 10 mg/day twice a day for 3 months.

Adolescent PCOS: The motive of therapy is to control symptoms of androgen excess and reduce the long term complications. Management of adolescent PCOS is symptomatic (Fig 2g), i.e., it depends on the predominant presenting symptom. The keystone includes exercise and weight loss. Medications act as a rescue. Oral contraceptive (OC) pills act by reducing the androgen production from the ovary and adrenal gland and reduces the free testosterone (by increasing the sex hormone binding globulin). Conventional OC pills like ethinyl estradiol /

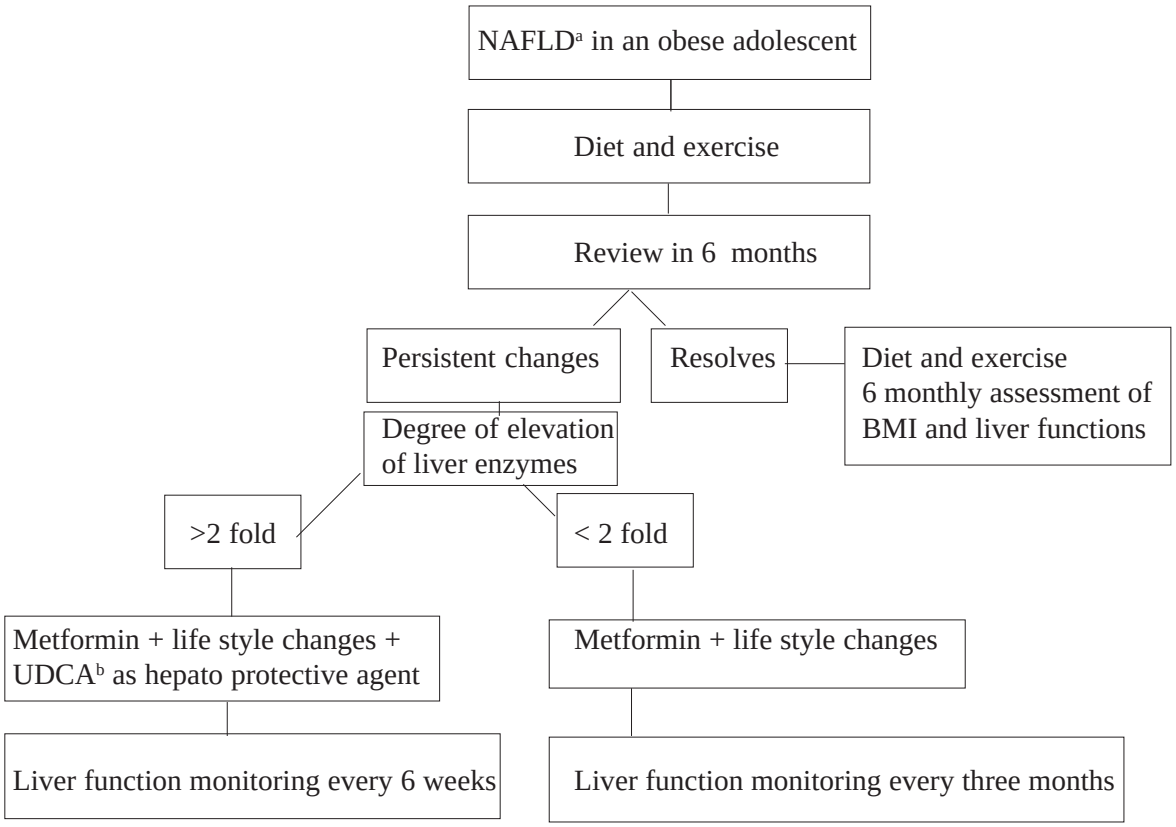


Legend: a - Self monitoring of blood glucose, b - Fasting blood glucose > 130 mg/dL, 2 hours post prandial > 180 mg/dL after the largest meal of the day

Fig. 2c. Approach to type 2 diabetes in adolescents

levonorgestrel can be used or newer ones like drospirenone and ethinyl estradiol (Yasmin) or Cyproterone and ethinyl estradiol (Ginnette) which contain anti androgenic progestins are ideal. OC pill therapy once initiated must be continued for 2-3 years till improvement of the metabolic abnormality. Anti androgens like aldactone (that act on androgen receptor) or finasteride that act on cutaneous 5á reductase can be used. They are specially useful in adolescents who are highly image conscious. Metformin acts by multiple mechanisms: improves the insulin sensitivity, reduces the androgen production from the ovaries and facilitates weight loss.

It should be remembered that drugs take 3-6 months to have a clinical effect because one hair cycle lasts for 3 months. Hence therapy should not be labelled as a failure in a short duration. Cosmetic therapy in an image conscious adolescent includes: depilation measures that remove the hair shaft - shaving or epilation measures that remove the hair bulb - like laser therapy. Always history of photosensitivity should be elicited and adequate normal androgen levels should be ensured prior to referral to a laser therapist. One must remember that laser therapy is not permanent.



Legend: a - NAFLD - non alcoholic fatty liver disease, b - UDCA - Ursodeoxycholic acid

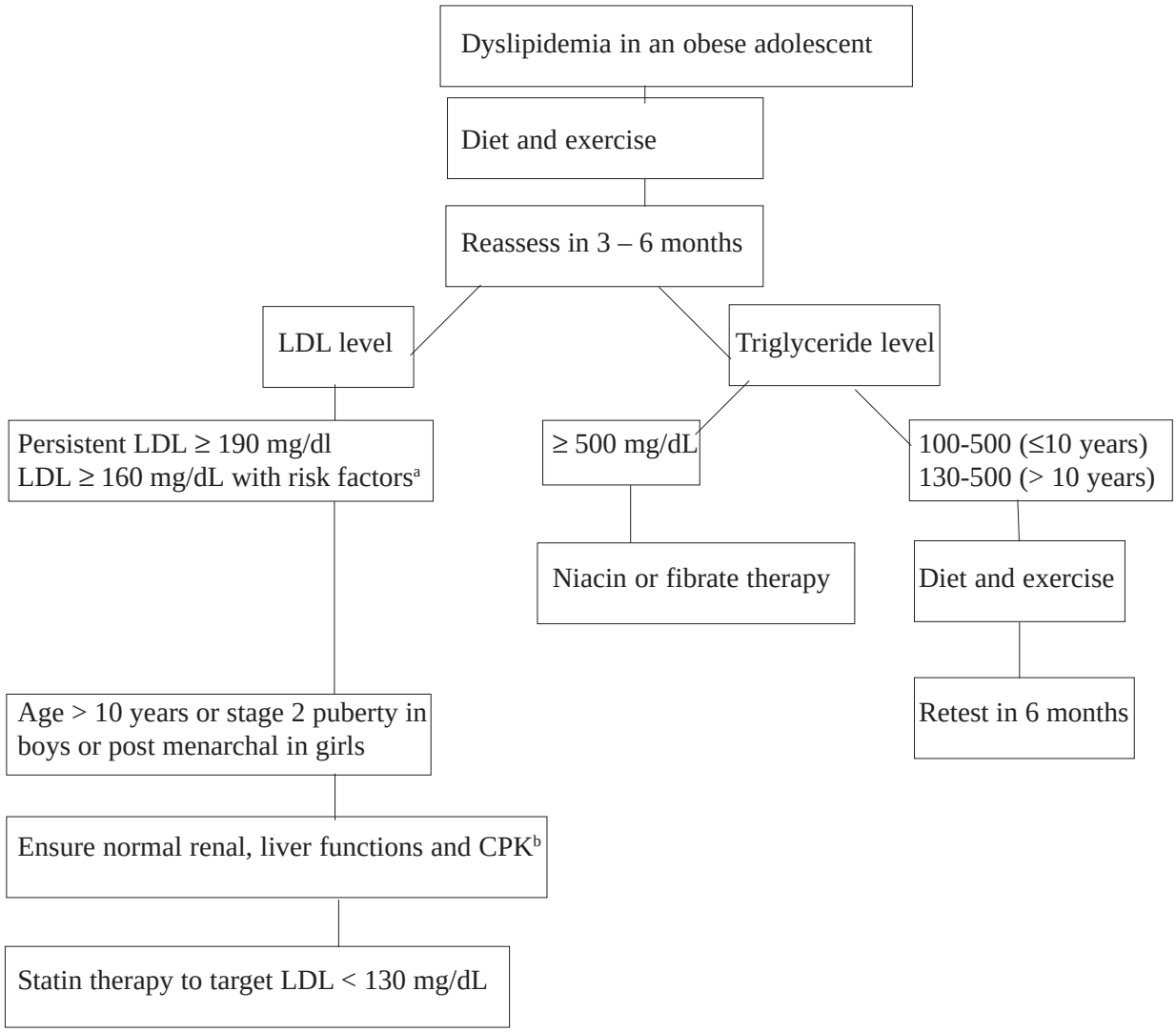
Fig. 2d. Approach to NAFLD in obese adolescents

Surgical referral: Bariatric surgery for adolescents is a last extreme resort done after completion of growth (near final height), completion of puberty (Tanner stage 4 or 5), bone age >13 years in girls and >15 years in boys.[20] They need 3 months pre-operative preparation to maintain adherence of diet and exercise regimen post surgery. BMI ≥ 35 and severe complications (type 2 diabetes mellitus, severe obstructive sleep apnoea syndrome, severe hypertension on drugs) or BMI ≥ 40 and mild complications (severe insulin resistance, glucose intolerance and hypertension) may be referred to a bariatric surgeon. There is no role for a bariatric surgery in an adolescent with an eating disorder, Prader-Willi syndrome or one who cannot adhere to a dietary regimen, post surgery.

In a nutshell, management of adolescent obesity requires a multidisciplinary approach involving a pediatrician, nutritionist, endocrinologist, gynaecologist and a psychologist.

Points to Remember

- ***A non-nutritional cause must be considered in obese adolescents with short stature, delayed bone age, growth velocity < 25th percentile, hypoplastic genitalia, extra digits, severe hypertension (Blood Pressure > 99th percentile) and focal neurological deficit.***
- ***The penile length must be measured and compared to age specific norms. Buried penis is the most likely, but, not the only cause for small penile length in an adolescent male.***
- ***Obese adolescents may have a marginal elevation in TSH mediated by leptin. This warrants only diet and exercise and not thyroxine replacement.***
- ***Sudden weight loss without exercise is a danger sign. It may indicate the decompensation of Type 2 diabetes in an obese adolescent.***



Legend: a - Either parent, grandparent, aunt, uncle or sibling aged < 55 years (males) and < 65 years (females) having any of these: Myocardial infarction, stroke, angina, coronary artery by-pass, stent, angioplasty or sudden cardiac death or parents with total cholesterol > 240 mg/dl or known lipid abnormalities in parents. b – Creatine phospho kinase

Fig. 2e. Approach to dyslipidemia in adolescents

- ***Although there are specific indications for drug therapy in adolescents with metabolic complications, diet and exercise are the main modes of therapy for all obese adolescents. Metformin is not a substitute for diet and exercise in adolescent metabolic syndrome.***
- ***Investigate PCOS only in the follicular phase of the cycle. Cosmetic laser therapy must be embarked upon after attaining biochemical control of androgen levels.***

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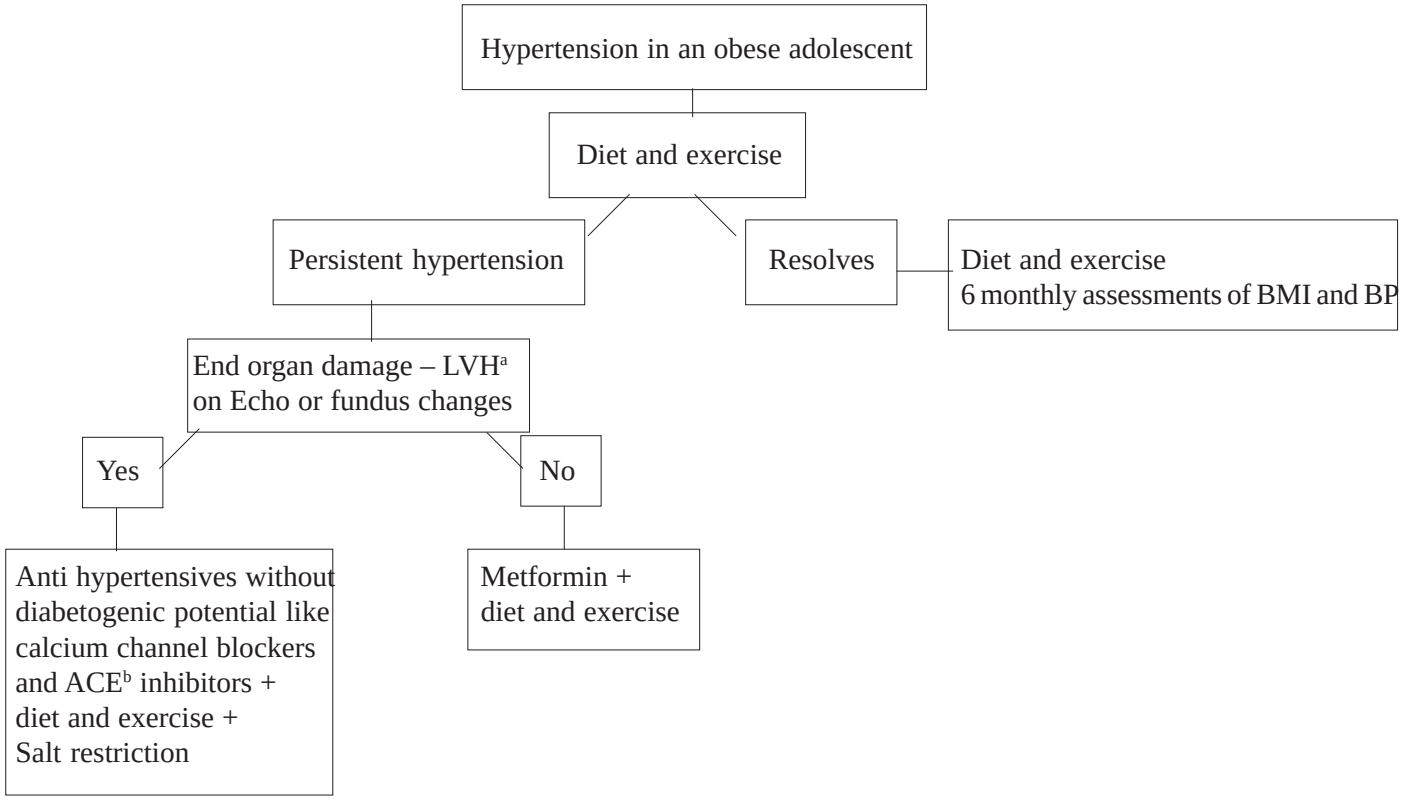

(Legend: a – Left ventricular hypertrophy, b – Angiotensin converting enzyme inhibitors)

Fig. 2f. Approach to hypertension in an obese adolescent

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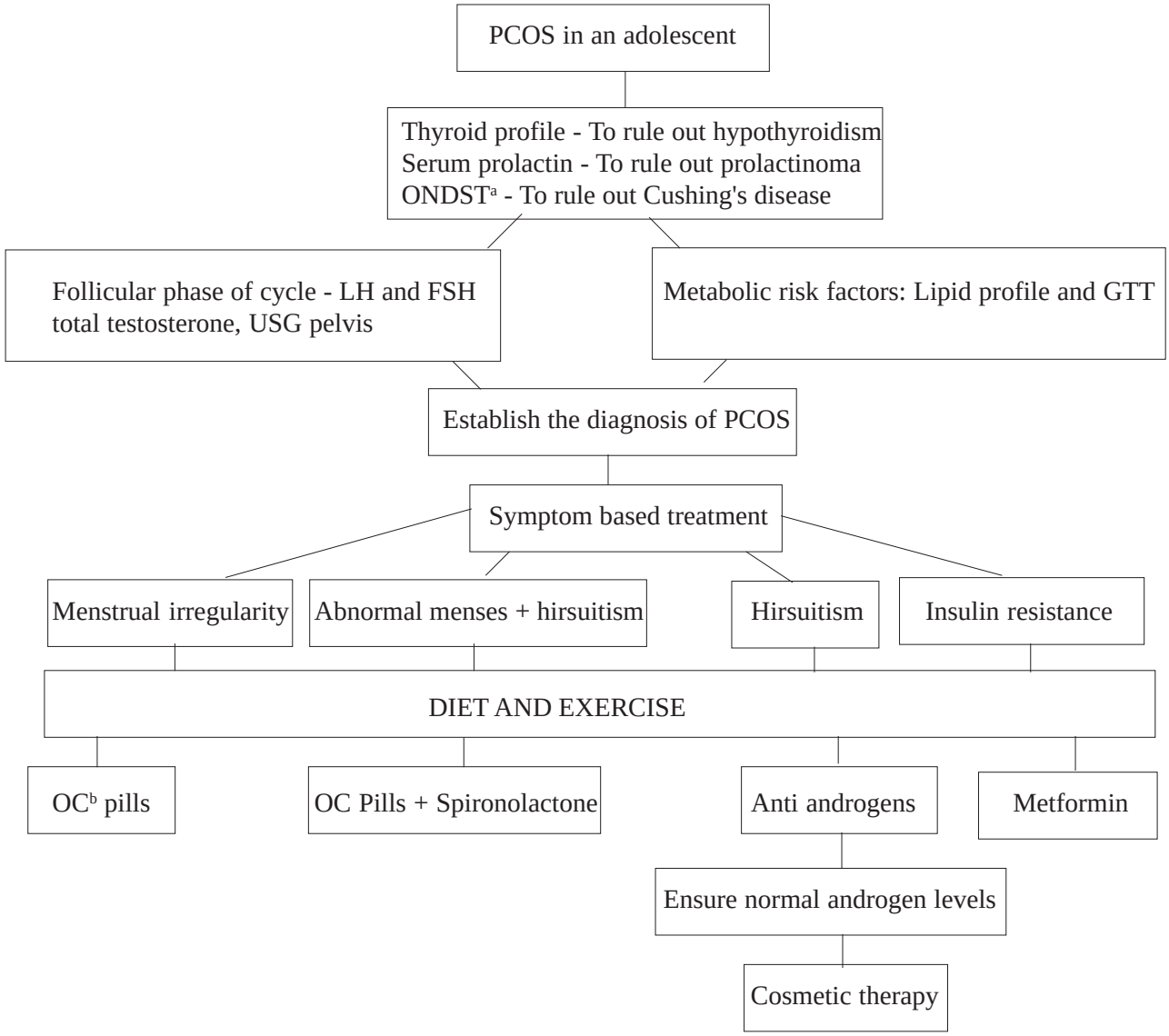

(Legend: a - Overnight dexamethasone suppression test, b - Oral contraceptive pills)

Fig. 2g. Approach to an adolescent with PCOS

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ADOLESCENT CARE

ADOLESCENT SEXUALITY

Chandrika Rao

Abstract: *Adolescent sexuality is an important issue that is encountered while dealing with the problems of adolescents. The HEEADSSS (home, education, eating habits, activities, depression, substance use, sexuality and safety) approach gives us an idea of the general problems involved, when an adolescent (he/she) constantly feels uncomfortable about the changes in the body and also when the natural interest towards the opposite sex, expected during the stage, is not evinced. Hence adolescents may harbor the idea that there is a deviation in their sexual orientation and may imagine themselves to be either gay or lesbian. One should address the problems associated with this stage such as sexual abuse, STD, pregnancy and other infections also.*

Keywords: *Adolescence, Sexuality.*

Sexuality has been a controversial topic from the time of Aristotle's ideas to Sigmund Freud's era and more so in the modern era. The adolescent development and formation of their identity is a transition phase for the adolescents, parents and society. In the 21st century, adolescent sexual behaviour is being discussed openly, emphasizing the importance of sexuality, sexual behaviours and relationships with its risks.

The World Health Organization and other international health agencies also identify adolescent-friendly health services as a worldwide priority,[1,2] with 70% of more than 1 billion youth (10 to 19 years) living in developing countries.[3] The important role that the health providers play in ensuring a positive sexual attitude is being recognized and stressed on now.

Definition

Sexuality is often considered only as the act of sex and often the discussion is limited by that concept. However, human sexuality is more than the act of sex and involves the person's concept of his or her own body image, sexual identity, role at home and society, personal feelings and self-esteem.[4]

Anatomic sex: This refers to the anatomic sexual reproductive organs. Anatomic sex is only one component of sexuality. *Gender identity:* This refers to the feeling within the person as being either masculine or feminine. Gender identity and anatomic sex sometimes do not match. For example, a person can be born as a boy but feels and behaves like a girl. This is sometimes referred to as transgender. *Sexual orientation:* This refers to the sexual attraction one feels towards another person. One may be attracted to people of the opposite sex (heterosexual or straight), the same sex (homosexual, gay, or lesbian), or both (bisexual). Sexual orientation is influenced by many factors, including anatomic sex, gender identity and the society. It is common for adolescents to feel confused about their sexual orientation, which is normal. These feelings may change as the person matures or may persist. It is important for adolescents and adults to be comfortable with all aspects of sexuality (anatomic sex, gender identity and sexual orientation).

Adolescent sexual development

There are many theories regarding adolescent sexuality.[5] The popular ones are listed below:

1. Freud's psychoanalytic theory states that the stages of psychosexual development are genetically determined. He opined that physiological changes are affected by emotional changes, and negative emotions like anxiety, depression, tension have greater impact .He also stressed on the role of self-image in the development of sexuality.
2. Anna Freud's theory: She emphasized on id, the ego and the superego. She opined that adolescent conflicts arise due to poor formation of id and ego of the individual.
3. Erikson's theory: He opined that the sense of personal identity is more important than sexuality. Individual efforts, peer relationships and value system contribute to a stable identity. The adolescent should form his own identity and not mimic an elder's or peer's

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identity as it will lead to confusion and dissatisfaction in adult life.

4. According to Piaget, cognitive development through adolescence involves movement from concrete to abstract thinking and a decrease in egocentric thought.

5. The social constructionist perspective examines the role of power and culture in the development of adolescent sexuality. In a practical sense, this explains why some girls may believe that sex is necessary to maintain relationships.

6. Developmental feminist perspective: Gender norms in society influence gender identity formation. Adolescent girls may learn to consider themselves as objects of desire and focus on what others expect out of them than being assertive about what they need. Society having double standards in viewing the same male and female sexual behaviour differently indicates that premarital sex also influences adolescent sexual development. Adolescents are often judged for their sexual behaviour by their gender. Elder siblings also influence sexual attitudes as per this theory.

7. Social learning and the sexual self-concept: Peer pressure plays an important role. The sexual experiences in childhood and adolescence, family roles and media are responsible for forming a self-concept and learning. Research has shown boys to experience lower sexual self-esteem and higher sexual anxiety. They also experience ambivalent feelings as society expects them to be dominant. Girls in age group of 14-17 years demonstrate high self-esteem and decreased anxiety. Researchers state that these behaviours are not impulsive but have been affected by self-concept and experiences.[6]

Adolescent sexual development

Early adolescence in age groups of 10-13 years show increased interest in opposite sex, excess interest in sexual feelings and may masturbate. Girls achieve menarche and males experience nocturnal emissions. In middle adolescence in age groups of 14-16 year old, puberty is often completed. They explore relationships with other gender and like to spend more time with same or opposite gender friends. In late adolescence (approximately 17 to 19 years old) goals are established. Adolescents begin to understand consequences of sexual behaviour, begin to understand pregnancy and sexually transmitted diseases. They explore relationships, understand their own sexual orientation. Adolescents may be sexually active and begin long term relationships. They understand the role of media. The oedipal complex (a child's attraction to the parent of the opposite sex) is common during the adolescent years.[7] Parents can acknowledge the adolescent's physical changes and gradually lay down ground rules to not cross parent-child boundaries.

Sexual orientation begins during puberty. Sexual orientation and gender identity are not a choice and appear to be established by early childhood. Feelings of homosexuality or lesbianism, transgender may emerge to discontinue later or may continue. These are influenced by both biological and environmental influences. In the DSM V edition, transgender people are diagnosed under 'Gender dysphoria' which communicates the distress that can result from "a marked incongruence between one's experienced/expressed gender and assigned gender." The term 'Gender identity disorder' is being replaced with the term 'Gender dysphoria'.[8]

Adolescent assessment

The HEADDSSS questionnaire is usually used to assess adolescent sexual behaviour and obtaining a comprehensive, confidential and developmentally appropriate adolescent psychosocial history allows for the discovery of strengths and assets as well as risks. Non-judgemental attitude, privacy and confidentiality should be ensured. Resource material and reading material should be freely available.

Role of a pediatrician

Pediatricians should have offices which are adolescent friendly and welcoming to youth. This includes having supportive office staff members who do not discriminate and do not behave differently with sexual minorities. They should encourage parents to talk to their teen about sexual behaviour in a non-judgemental way. Parents should be counselled that the adolescent interest in one's own body may be natural. It does not indicate that their child is involved in sexual activity. It is necessary to discuss abstinence, contraception, consent and substance abuse with the adolescent and life skill training to adolescent to say 'no'. During communication use inclusive words as gay, lesbian, bisexual, or transgender. This opens avenues for adolescent to communicate. For transgender youth, pediatricians should provide the opportunity to acknowledge and affirm their feelings of gender dysphoria. Adolescent physicians should refer transgender youth to a qualified mental health professional who will assist with the dysphoria, educate them and help them in their transition.[9] The impact of sexuality in adolescence: a) pregnancy, b) contraception, c) sexually transmitted

diseases, d) dating violence, e) sexual abuse should be discussed with the adolescent . These are discussed below.

Adolescent pregnancy

About 16 million women between 15-19 years old give birth each year, about 11% of all births worldwide. Half of all adolescent pregnancy occur in just seven countries: Bangladesh, Brazil, the Democratic Republic of the Congo, Ethiopia, India, Nigeria and the United states.[10] In India, adolescent pregnancy is high in rural areas due to early marriage. Data shows that teen pregnancy in India is 62 out of every 1,000 women. Although they are married, pregnancy is usually not a choice but occurs due to inadequate knowledge about contraception, societal pressure to bear children once married and very often rights violation.[11] The age of the mother is determined by the verified date when the pregnancy ends, not by the estimated date of conception. Adolescent pregnancy is harmful for both mother and baby. Adolescent pregnancy may result in problems like post-partum hemorrhage, anemia, hypertension, depression, premature and low birth weight babies and increased number of neonatal complications.

Role of pediatrician

The pediatrician plays an important role along with the obstetrician in antenatal care of an adolescent pregnant girl and can educate, counsel the teen and the parents and ensure good outcome of mother and child. Counselling on complete antenatal care, psychological support to family, immunisation, diet, supplementation of iron and folic acid tablets and advice to abstain from drugs and alcohol and ensuring delivery in hospital and care of newborn are the necessary things which a paediatrician has to do.

Contraception

The various methods which can be recommended are as follows-

1. Natural methods: Abstinence during fertile phase and withdrawal (Coitus interruptus). Periodic abstinence may not be reliable for adolescents to follow as impulsive behaviour dominates in this period.
2. Barrier contraceptives: Male condoms, spermicidal agents, use of diaphragm or cervical cap in the vagina, use of hormones which alter the cervical mucus and prevent entry of sperm into the cervical canal (today sponge with spermicidal cream). Barrier methods are best for adolescents while- condoms prevent pregnancy and infection.
3. Intrauterine contraceptive devices (IUCDs): COPPER T-may not be suitable in nulliparous women. However it is a long term, coital independent method which a sexually active adolescent may choose if no contraindications are present.
4. Suppression of spermatogenesis: Gossypol is the drug of choice.
5. Suppression of ovulation with hormones: Estrogen and progesterone pills can be prescribed in adolescents. Third generation combined oral contraceptive pills (COCs) have few side effects. Ultra low dose combined oral contraception pill (COCs) with 15 microgram ethinylestradiol have fewer side effects. Some may prefer three monthly injection of depo provera at dose of 150 microgrm IM, implants or skin patches.
6. Interceptive agents (post-coital contraception): I pill, 2 tablets as soon as possible within 48-72 hours.

Sexually transmitted infections (STI)

Infections can spread through sexual intercourse, anal sex, oral sex, and using fingers, other body parts or sex toys that have come in contact with another person's genitals or body fluids. Abstinence is the only100% effective method to prevent STD's. Adolescents who have early sexual intercourse, multiple sexual partners, unprotected sex, homosexuality, poverty, drugs or high risk sex behaviour are at greater risk of getting STD.

Some of the most important causes STIs of are Chlamydia, gonorrheae, HPV, Herpes simplex virus and the treatment is given in the Table I.[12]

Human papillomavirus: This may be a silent infection. HPV 6, 16 and 18 can cause condylomata acuminate. This can be treated with local application of podophyllin 25% in alcohol for 6 hours daily or 25% trichloracetic acid plus fluorouracil to cause sloughing in 3-7 days and can be repeated weekly but contraindicated if adolescent is pregnant. Diathermy, laser ablation and surgical excisions are other available treatment options. HPV causes cervical cancer in women, penile cancer in men and anal or oropharyngeal cancer in either sex and can be prevented by vaccine.

HIV - Recommendations are that HIV screening should be routinely done in sexually active adolescents. Posters and pamphlets are useful in dissemination of information in the pediatrician's clinic.

d) Dating violence

Girls under age 18 are twice more likely to be beaten by their child's father than women over age of eighteen. 10% to 30% of teenagers have experienced violence in a dating relationship in different populations studied.

Table I. STIs and treatment

Syndrome	Organisms/Diagnoses	Treatment[a]
Urethritis and cervicitis	Neisseria gonorrhoeae, Chlamydia trachomatis Other causes - Mycoplasma genitalium, Ureaplasma urealyticum, Trichomonas vaginalis Herpes simplex virus (HSV)	Ceftriaxone, 250 mg, IM, in a single dose[b] or Cefixime, 400 mg, orally, in a single dose[b] Plus either Azithromycin, 1 g, orally, in a single dose or Doxycycline, 100 mg, orally, twice a day for 7 days
	HSV-primary infection	Acyclovir, 400 mg, orally, 3 times/day for 7-10 days or Acyclovir, 200 mg, orally, 5 times/day for 7-10 days or Famciclovir 250 mg, orally, 3 times/day for 7-10 days or Valacyclovir 1g, orally, twice daily for 7-10 days
Adolescent vulvovaginitis	T vaginalis	Metronidazole, 2g, orally, in a single dose or Tinidazole, 2g, orally, in a single dose
	Bacterial vaginosis	Metronidazole, 500 mg, orally, twice daily for 7 days or Metronidazole gel 0.75%, 1 full applicator (5 g), intravaginally, once a day for 5 days or Clindamycin cream 2%, 1 full applicator (5 g), intravaginally at bedtime, for 7 days
	Candida species	Ketoconazole (400 mg/day) or itraconazole (50-100 mg/day) or fluconazole (100 mg/wk) for 6 weeks, and clotrimazole (500-mg vaginal suppositories once per wk)
	HSV-primary infection	Acyclovir, 400 mg, orally, 3 times/day for 7-10 days or Famcyclovir, 250 mg, orally, 3 times/day for 7-10 days or Valcyclovir, 1g, orally twice/day for 7-10 days
Pelvic inflammatory disease (PID)	N gonorrhoeae, C trachomatis, anaerobes, coliform bacteria, and Streptococcus species	**Mild: Ceftriaxone** 250 mg IM in a single dose Plus **Doxycycline** 100 mg orally twice a day for 14 days With or without **Metronidazole** 500 mg orally twice a day for 14 days **Severe: Cefotetan** 2 g IV every 12 hours or **Cefoxitin** 2 g IV every 6 hours Plus **Doxycycline** 100 mg orally or IV every 12 hours

Syndrome	Organisms/Diagnoses	Treatment[a]
Syphilis	Treponema pallidum	Preferred Therapy:Benzathine penicillin G 2.4 million units IM for 1 dose Alternative Therapy (For penicillin-allergic patients): • Doxycycline 100 mg PO BID for 14 days, or • Ceftriaxone 1g IM or IV daily for 10-14 days, or • Azithromycin 2g PO for 1 dose
Genital ulcer disease	T pallidum	Same as for syphilis
	HSV-primary infection	See prepubertal vaginitis
	Haemophilus ducreyi (chancroid)	Azithromycin, 1 g, orally, in a single dose or Ceftriaxone, 250 mg, IM, in a single dose or Ciprofloxacin, 500 mg, orally, twice daily for 3 days[c] or Erythromycin base, 500 mg, orally, 3 times/day for 7 days
	Klebsiella granulomatis (granuloma inguinale [Donovanosis])[d]	Doxycycline, 100 mg, orally, twice a day for at least 3 weeks and until all lesions have healed completely or Azithromycin, 1 g, orally, once/wk for at least 3 weeks and until all lesions have healed completely or Ciprofloxacin, 750 mg, orally, twice a day for at least 3 weeks and until all lesions have healed completely or Erythromycin base, 500 mg, orally, 4 times/day for at least 3 weeks and until all lesions have healed completely or Trimethoprim-sulfamethoxazole, 1 double-strength (160 g/800 mg) tablet, orally, twice a day for at least 3 weeks and until all lesions have healed completely
Sexually acquired epididymitis	C trachomatis, N gonorrhoeae	Ceftriaxone, 250 mg, IM, in a single dose Plus Doxycycline, 100 mg, orally, twice daily for 10 days
	Enteric organisms (for patients allergic to cephalosporins and/or tetracycline)	Levofloxacin, 500 mg, orally, once daily for 10 days or Ofloxacin, 300 mg, orally, twice a day for 10 days
Gonococcal infections of the pharynx	N gonorrhoeae	Ceftriaxone, 250 mg, IM, in a single dose
Anogenital warts	Human papillomavirus	Patient-applied: Podofilox 0.5% solution or gel or Imiquimod 5% cream

Syndrome	Organisms/Diagnoses	Treatment[a]
		or Sinecatechins 15% ointment Provider-administered: Cryotherapy or Podophyllin resin 10%–25% or Trichloroacetic acidor Bichloroacetic acid or Surgical removal
Vulvovaginal Candidiasis		Intravaginal agents: Butoconazole, 2% cream, 5 g, intravaginally, for 3 days[a,b] or Butoconazole, 2% cream (sustained release), 5g, single dose intravaginal application for 1day or Clotrimazole, 1% cream, 5 g, intravaginally, for 7-14 days[a,b] or Clotrimazole 2% cream, 5 g, intravaginally, for 3 days[a,b] or Miconazole, 2% cream, 5 g, intravaginally, for 7days[a,b] or Miconazole, 4% cream, 5 g, intravaginally, for 3 days[a,b] or Miconazole, 100-mg vaginal suppository, 1 suppository for 7 days[a, b] or Miconazole, 200-mg vaginal suppository, 1 suppository for 3 days[a, b] or Miconazole, 1200 mg vaginal suppository, 1 suppository for 1 day[a,b] or Nystatin, 100 000-U vaginal tablet, 1 tablet for 14 days or Tioconazole, 6.5% ointment, 5 g, intravaginally, in a single application[a,b] or Terconazole, 0.4% cream, 5g, intravaginally,for 7 days[a] or Terconazole, 0.8% cream, 5g, intravaginally,for 3 days[a] or Terconazole, 80-mg vaginal suppository, 1 suppository for 3 days[a] Oral agent: Fluconazole, 150-mg oral tablet, 1 tablet in single dose

e) Sexual abuse

Multiple studies have indicated a strong link between early childhood sexual abuse and subsequent teenage pregnancy in industrialized countries. Up to 70% of women who gave birth in their teens were molested as young girls; by contrast, 25% of women who did not give birth as teens were molested.[13]

In India, sexual intercourse between a minor and an adult is not considered consensual under the law because a minor is believed to lack the maturity and competence to make an informed decision to engage in fully consensual sex with an adult. In India, sex with a minor is therefore considered statutory rape.

Legal aspects

In India legal age for consensual sex is 18 years under the Criminal Law (Amendment) Act, 2013. Marriage of a female less than 18 years of age or a male of less than 21 years of age is illegal. Marriage is voidable and not void. Marriage will become valid if no steps are taken by such 'child' seeking declaration of marriage as void, (Hindu Marriage Act, 1955). Lesbian, gay, bisexual and transgender people in India face danger of being imprisoned up to a lifetime because of their sexual orientation. Homosexual intercourse is a criminal offence under Section 377 of the Indian Penal Code since 1860. The Supreme Court of India overturned the decision of the lower court in 2013 and upheld the primacy of section 377. The Medical Termination of Pregnancy (MTP) Act of India clearly states the conditions under which a pregnancy can be ended or aborted, the persons who are qualified to conduct the abortion and the place of implementation.[14] One of the qualification is pregnancies in unmarried girls under the age of eighteen with the consent of a guardian. (Medical Termination of Pregnancy, 1971).

Points to Remember

- ***Sexuality is influenced by adolescents over body language, sexual identify, role at home and society, personal feeling and self-esteem.***
- ***Feelings of homosexuality, transgender may emerge to discontinue later or may continue.***
- ***HEADDSSS questionnaire is usually used to assess the adolescent behaviours.***
- ***Pediatricians should have an adolescent friendly clinic to address to sexuality and related assess like adolescent pregnancy, STDs, sexual abuse, etc.***

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ADOLESCENT CARE

POOR SCHOOL PERFORMANCE IN ADOLESCENCE

***Preeti M Galagali**
****** Luiz N**

Abstract: *Poor school performance in adolescence is a common problem encountered in clinical practice. It may present for the first time in adolescence or may be a part of the continuum since childhood. It can be associated with short and long term physical and mental morbidity and even mortality. Multiple factors may contribute to its occurrence in adolescence. A thorough clinical evaluation is essential to make a precise diagnosis and plan further management. A multidisciplinary team involving pediatricians, teaching fraternity and mental health professionals is required for appropriate management.*

Keywords: *Poor school performance, Specific learning disability, Attention deficit hyperactivity disorder.*

Scholastic underachievement is a common problem presenting to adolescent health care professionals. Most parents and teachers are distressed if their wards perform poorly in academics. Academic failure is one of the most common causes of suicide and school drop out in adolescence. It can also be a pointer towards emerging mental disorder in this age group.

Academic challenges in adolescence are immense.[1] One of these challenges is an increase in academic input in terms of volume and complexity of subject matter. A high level cerebral processing, increased working memory and meta cognitive abilities are required to cope with these demands. There is also an increase in academic output, i.e., efficient retrieval of stored information is required for sailing through scholastic tests and exams. To meet these challenges, physical, emotional and psychosocial well being of adolescents is essential and so is an encouraging home and school environment. Academic achievement in adolescence is an important determinant of high self- esteem, peer and family approval and is said to have long term impact on future career prospects.

The prevalence of *poor school performance* (PSP) in India varies from 5% to 15%.[2,3,4,5] The most common causes include poor home environment, inefficient study strategies and learning disability.[2,3,4,6] In India, rote learning is the norm and there is an intensely competitive examination system. High parental expectations also increase the academic stress on adolescents.

Definition

The precise definition of PSP is controversial. A practical one that can be used in clinical practice is, "adolescents are said to be performing poorly in academics if they fail in one or more subject or class or if their marks are below 10th percentile in a particular class or subject or if they are identified by the parent or teacher as 'difficult' to teach".[7]

Causes

Causes of poor academic performance are usually multifactorial. They can be broadly divided into factors in school, family or the adolescent. These are enumerated in the Table I. PSP in childhood can continue in adolescence or may present for the first time in this age group. This spectrum of presentation is elucidated in Fig.1. Disorders that present early in life like mild *attention deficit hyperactivity disorder* (ADHD), *specific learning disability* (SLD), low intellectual ability, autism and cognitive defects due to prematurity or low birth weight may be compensated in childhood due to relatively low academic load but may present for the first time in adolescence as poor school performance when the load increases. The causes of PSP are briefly described below.

Family factors

A stable, nurturing and stimulating home environment helps the adolescents to perform to their potential. Low socioeconomic status is the strongest predictor of poor school performance at all ages. Research has shown that in adolescence, appropriate parental expectations, authoritative parenting style, parental involvement in school activities and a high investment by parents in social

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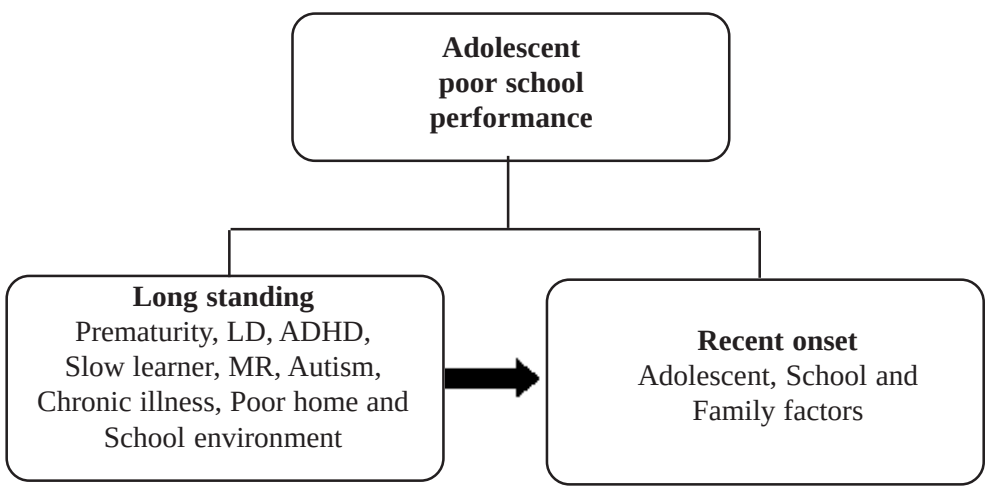
Table I. Causes of poor school performance in adolescence

Family factors	School factors	Adolescent factors
Low socio economic status Low maternal education Illiteracy Marital discord Divorce Single parent Authoritarian or permissive parenting Punitive parenting High parental expectations Violence	Change of school Change in medium of instruction Poor teaching methodology Crowded classrooms High student teacher ratio Overexpectations of teachers Abusive teachers Bullying Disruptive peer group	Poor study skills Learning disability Low intellectual ability Autism Spectrum Disorder ADHD, oppositional defiant disorder, conduct disorder Prematurity Low birth weight Poor motivation Speech/ language impairment Anemia Hearing impairment Visual impairment Mental disorders Abuse Chronic diseases Head injury

capital in terms of encouraging creative arts, hobbies and sports play an important role in improving scholastic performance.[8] Parental role modeling of a healthy lifestyle by following a balanced diet, adequate physical activity, sleep and time management and appropriate use of life skills also enhance the adolescent's school performance.

School factors

The school should provide an encouraging atmosphere that develops the all round personality of the students. Teachers should accept the fact that not all adolescents are 'hardwired' to excel in academics. They should avoid comparisons and rebuking students in public. Emotional abuse hurts their selfesteem and may be responsible for school refusal, drop out and even suicide. Uncomfortable class rooms with poor and boring teaching methodologies can result in low academic performance.[6] Peer groups that give importance to academics help the individual members to excel and those who do not, have members who perform poorly. In adolescence, where a lot of importance is given to selfimage and 'fitting in' with peer group, some gifted students may perform badly on



Note: Due to increased academic load, mild LD, ADHD can present as recent onset PSP in adolescence

Fig. 1. Clinical presentation of poor school performance in adolescence

purpose lest they may be branded as 'nerds' by their friends.[8] Academic institutions should have strict rules against bullying and 'ragging', both of which can result in fall in academic performance amongst adolescents.

Adolescent factors

Adolescents with low 'intrinsic' motivation may do poorly in academics.[8] Right from childhood the feeling of 'joy in learning', faith in one's abilities, putting in hard work and use of efficient study strategies has to be instilled by both parents and teachers. Overzealous emphasis on academics can result in 'burn out' and academic failure. Poor time management with excessive and inappropriate media usage can also result in the same. Adolescent sexuality issues like 'love affairs' and promiscuity can also result in distractions from academics and poor performance.

Mental disorders such as depression, anxiety, substance addiction, bipolar disorders, conduct disorders and schizophrenia may clinically present with a fall in academic performance as the only symptom. In 60 to 80% cases, childhood ADHD persists into adolescence when the hyperactivity decreases but inattention with poor peer and social relationships persists. Co-morbid mental disorders and emotional problems are commonly seen in adolescence with SLD, ADHD, autism and low intellectual ability.

Anemia, usually nutritional is the most common medical problem present in Indian adolescents with an overall prevalence of 45%. Anemia affects both physical and mental growth and is an important contributor to poor academic performance in adolescence. Adolescents with chronic disorders such as diabetes mellitus, asthma, epilepsy, juvenile rheumatoid arthritis and hypothyroidism may have problems with adherence and compliance to medication. This may result in poor control of the disease, school absenteeism and poor performance. Also, in diseases like epilepsy, side effects of medications like phenobarbitone may contribute to cognitive defects. Genetic disorders like fragile X and Klinefelter Syndrome may present with impaired mental ability and PSP for the first time in adolescence.[9] Head injuries, obstructive sleep apnea syndrome, visual and hearing impairment can also result in PSP.

Impact and consequences

PSP can lead to immediate and long term repercussions (Box 1). The adolescent can clinically present with these problems in the backdrop of PSP.[1]

Box 1. Impact of poor school performance in adolescence

- Psychosomatic Problems
- Low selfesteem
- Stress in the adolescent and parents
- Aggressive behavior
- Anxiety
- Depression
- Suicide
- School absenteeism
- School drop out
- Drug abuse
- Sexual promiscuity
- Clinical approach

A thorough clinical evaluation is essential to delineate the causes of poor academic performance, to identify strengths and to plan further management. A clinical approach is outlined below:

History taking

A detailed history is taken from the parents and adolescent regarding:

- Presenting complaints like aches and pains (psychosomatic disorder) or change in school performance and behavior
- Onset and duration of learning difficulty namely problems in completing notes, concentration, spelling mistakes, memory lapses, number of failures in a class or a subject
- Chronic diseases like diabetes mellitus, asthma, etc. Current status and disease severity along with adherence and compliance to medication is assessed
- Symptoms suggestive of obstructive sleep apnea syndrome (OSAS), hypothyroidism, anemia and head injury and meningitis in the past
- Birth and developmental history including history of prematurity, low birth weight, delayed milestones are suggestive of SLD, ADHD, autism in childhood.[10] Family history of SLD, ADHD, autism and mental disorder is also taken as these are heritable.

Table II. HEEADSSS psychosocial history in PSP in adolescence

Item	Key points
Home	Relationship with parents and family members, type of parenting, marital discord, abuse, drug use, mental disorder
Education	Details of scholastic problems, study habits, ambition, any change in academic performance, peer group, relationship with teachers, bullying
Eating habits	Caloric and green leafy vegetable intake, body image concerns
Activities	Hobbies, type and duration of media usage, time spent with peers, sleep, any recent loss of interest in activities
Depression	Any change in mood, behaviour and interest, duration of such change, suicide ideation or attempt
Substance Use	Attitude towards drug use, drug use amongst peers, type and frequency of drug use
Sexuality	Details regarding sexual health, intimate partners, sexual encounters, pregnancy, abortion, abuse
Safety	Indulgence in violent acts, run away behaviour

- Psychosocial history is taken from parents and adolescent. History is taken in privacy maintaining confidentiality. One of the practical methods of eliciting a psychosocial history is by using the HEEADSSS tool. HEEADSSS is an acronym that stands for home, education, eating habits, activities, depression, substance use, sexuality and safety.[11] Important points to be noted under various headings are given in Table II. Apart from identifying weaknesses and various adolescent, family and school factors contributing to PSP, HEEADSSS is an excellent tool to delineate strengths of the adolescents like non academic achievements that can be used in strength based counseling for motivating an improvement in academic performance.

A report from the teacher regarding current school performance and behavior and changes if any in the recent past is also beneficial in diagnosis and management.

Examination

Physical, systemic and mental status examinations are essential. On physical examination, signs of undernutrition, nutrient deficiency, anemia, goiter, visual and hearing impairment, stigmata of Fragile X syndrome (large testis) and Klinefelter syndrome (small testes) are carefully looked for. Systemic and mental status examinations are done for clinical assessment of chronic diseases and mental disorders. An informal academic assessment can be done by reviewing current and previous classwork notebooks, exam papers and report cards that may give pointers to long term learning problems like SLD. A brief assessment of reading, writing, analytical and mathematical ability and skills can be carried out by giving age appropriate tasks and short assignments.

Investigations

Investigations vary according to the provisional diagnosis that is made after history and examination. Hemogram, thyroid function tests, audiometry, refraction and karyotyping may be required in few cases. DSM 5 (Diagnostic and Statistical Manual 5) criteria, Connor's scale for ADHD and Beck's inventory for depression are examples of clinically useful screening and diagnostic tools for mental disorders. Psychoeducational testing that includes standardized tests for IQ and learning disability assessments are required in most cases of PSP.[12] These tests should be carried out by a trained clinical psychologist. A simple algorithmic approach to a case of PSP in adolescence is shown in Fig.2.

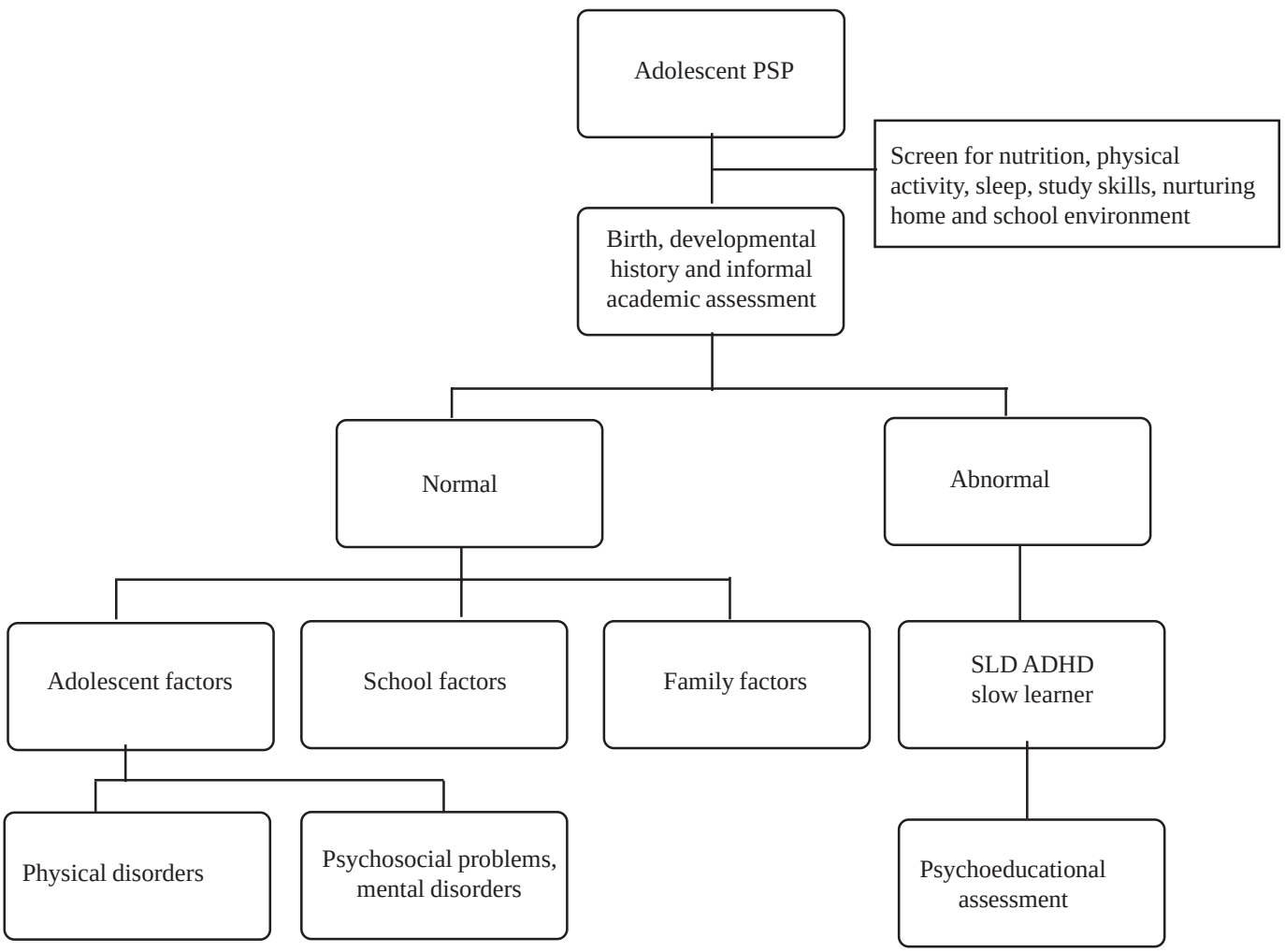
Management

Management of a case of PSP in adolescence requires a multidisciplinary approach and team work. The team leader is usually the pediatrician/adolescent specialist who

shares a good rapport with the adolescent and family. The pediatrician makes the provisional diagnosis, does appropriate investigations and referrals and finally coordinates with other team members. According to the final diagnosis the team members may vary but essentially include a psychologist, the class teacher and in most cases a special educator and psychiatrist.

The treating pediatrician must inform the adolescent and parents about the different factors contributing to PSP and discuss with them various treatment options. The pediatrician should help them to set realistic, scholastic and career goals. Multiple counseling sessions may be required. At each session, strengths of the adolescent like achievements in sports, music and dance are encouraged and reinforced.[10] Medical problems like anemia and chronic disorders should be managed appropriately emphasizing on adherence and compliance issues. Mental disorders like depression, anxiety and ADHD may need psychotherapy in the form of cognitive behavior therapy and a few cases may require specific drugs like fluoxetine (selective serotonin reuptake inhibitors) and methylphenidate respectively.

Adolescents, parents and teachers are counseled about the importance of ensuring age appropriate nutrition, physical activity, sleep, study skills and a nurturing school and family environment. A few important study skills that



Note: Multiple factors may coexist contributing to PSP

Fig. 2. Algorithm approach to PSP in adolescence

can be discussed with the adolescent include time management, active listening skills, methodology of taking notes, use of mnemonics, mind maps, flash cards and repeated rehearsals for improving memory abilities and efficient exam taking techniques.[13]

An individualised education plan (IEP) may be charted out for adolescents with SLD and ADHD by a remedial educator. Many of these students after certification can avail of special privileges by state and central boards in the form of extra time for completion of exams, subject exemption, use of a calculator and scribe for exams. Some of them may not be able to cope with regular schooling and may opt to join the National Institute of Open Schooling (NIOS). Under NIOS scheme, a wide array of academic and vocational subjects is offered and the students can complete their secondary education at their own pace with minimal stress.[14]

Pediatricians can play a major role in managing cases of PSP in adolescence as they enjoy the trust and faith of both the adolescent and parents. They can screen for causes, give initial counseling and treatment for disorders contributing to PSP and make appropriate referrals and assist mental health professionals in formulating a case-specific management plan. They can also assess response to treatment during follow up sessions. Hence, pediatricians can ensure that the adolescents perform to their potential that assists them in attaining success in their respective careers as adults.

Points to Remember

- ***Poor school performance in adolescence may indicate an emerging mental disorder.***
- ***Multiple factors in the family, school or intrapersonal may contribute to PSP in the adolescent.***
- ***Detailed clinical evaluation is the key to appropriate management.***
- ***A multidisciplinary team that essentially includes a pediatrician and mental health professional should manage cases of PSP in adolescence.***
- ***Pediatricians should emphasise the importance of adequate nutrition, sleep, physical activity, nurturing home and school environment and encouraging non-academic strengths in all cases of PSP apart from assisting in formulating an individualized treatment plan.***

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ADOLESCENT CARE

ADOLESCENT ANXIETY AND DEPRESSION

***Nair MKC**

Abstract: *Behavioural, emotional and mental health problems are widely prevalence among adolescents in India. Primary care physicians or pediatricians need to recognize and manage a majority of anxiety and depression problems among adolescents due to limited availability of psychiatrists or clinical psychologists. Anxiety disorders may be generalized anxiety disorders, phobias, panic disorders, obsessive compulsive disorders, post traumatic stress disorders, etc. Depression in adolescent may be difficult to identify due to the normal behavioural variations associated with hormonal changes in this age group. It can manifest as low self-esteem, difficulty in establishing autonomy and suicidal ideation. Depression may be mistaken for attention deficit hyperactivity disorder or may present with physical ailments. In the majority of children with depression, both psychotherapy and medication are required.*

Keywords: *Anxiety, Depression, Selective serotonin reuptake inhibitors, Adolescents.*

The mental health of child, adolescent and youth is probably a continuum from a child development perspective – normal and abnormal, evolution of problems, causative and risk factors and ultimately the outcome. Any retrospective search for cause, is likely to be biased. It is said that many of the adult psychiatric disorders have their onset in adolescence. Mental health issues of adolescents have to be addressed in the primary care set-up itself, because there are far too many children and adolescents with behavioural, emotional and mental health problems in the community and only limited number of psychiatrists, clinical psychologists and trained counsellors are available. Yet in India, trends in adolescent health in priority mental health areas have uniformly been static or adverse, in contrast to gains made in other countries.[1]

Hence, what we need in India, is a team approach with a referral line starting with parents/teachers/ community health workers who may suspect a problem, which may be diagnosed and managed at the primary care setting itself. Referral services to higher centres are needed only for a few selected cases, primarily to confirm the diagnosis, rule out co-morbid conditions and chalk out a management strategy, implementation of which can be done by the primary care team, if only they could suspect and diagnose early. We need to remember that although DSM-V/ICD 10 Criteria is the gold standard for diagnosing mental health disorders including anxiety disorders and depression, what is more important, for a primary care physician is to remember that the symptom complex per se do not make a diagnosis, unless it is (i) more than explainable by the apparent cause (ii) have significant bodily symptoms and (iii) symptoms are severe enough to cause impairment in daily functions.

Anxiety Disorders

Anxiety disorders are a group of mental health disorders, characterized by excessive feelings of anxiety and fear, the anxiety being worry about future events and fear, a reaction to current events causing physical symptoms such as a racing heart and shakiness. Fear and anxiety are in the same continuum, fear is the reaction to a present danger, an adaptive and evolutionary refined process and anxiety is the response to a potential threat. Anxiety is a disproportionately intense, chronic and potentially irreversible reaction to an imagined threat, operated through brain-body-emotion-cognitive-changes and their interaction with the environment.[2] The anxiety disorders may be grouped as follows:-

Generalized anxiety disorder (GAD): GAD is a common, chronic disorder characterized by long-lasting anxiety that is not focused on any one object or situation. Those suffering from generalized anxiety disorder experience non-specific persistent fear and worry, and become overly concerned with everyday matters. In children GAD may be associated with headache, restlessness, abdominal pain

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and heart palpitations. Typically it begins around 8 to 9 years of age. If a child has GAD, they may worry about anything, even if it is seemingly minor. They long for attention, approval and encouragement from others.

Phobias: Phobic disorders, includes all cases in which fear and anxiety are triggered by a specific stimulus or situation. Sufferers typically anticipate terrifying consequences from encountering the object of their fear. Sufferers understand that their fear is not proportional to the actual potential danger but still are overwhelmed by the fear. School phobia is a common anxiety disorder in children, which in some cases can be a type of separation anxiety, with no obvious cause. School phobia may also be a form of social phobia, also known as social anxiety.

Panic disorder: With panic disorder, a person suffers from brief attacks of intense terror and apprehension, often marked by trembling, shaking, confusion, dizziness, nausea, and/or difficulty in breathing. These panic attacks are fear or discomfort that abruptly arise and peak in less than ten minutes and can last for several hours. Attacks can be triggered by stress, fear, or even exercise; the specific cause is not always apparent.

Social anxiety disorder (SAD): SAD also known as social phobia describes an intense fear and avoidance of negative public scrutiny, public embarrassment, humiliation, or social interaction.

This fear can be specific to particular social situations (such as public speaking) or, more typically, is experienced in most (or all) social interactions.

Obsessive-compulsive disorder (OCD): OCD is a type of anxiety disorder primarily characterized by repetitive obsession (distressing, persistent and intrusive thoughts or images) and compulsion (urges to perform specific acts or rituals).

Post-traumatic stress disorder (PTSD): PTSD is an anxiety disorder that results from a traumatic experience. Adolescents would normally feel upset and anxious with any unusual event, but when there is (i) a history of real or perceived catastrophic trauma like death of parents, (ii) an intrusive recollection of the traumatic event, (iii) with autonomic arousal symptoms like sweating and palpitation and all resulting in, (iv) avoidance of the situation, it is called a post traumatic stress syndrome.

Separation anxiety disorder (SepAD): SepAD is the feeling of excessive and inappropriate levels of anxiety over being separated from a person or place. Separation anxiety is a normal part of development in babies or children, and it is only when this feeling is excessive or inappropriate that it can be considered as a disorder.

Situational anxiety: Situational anxiety is caused by new situations or changing events. It can also be caused by various events that make that particular individual uncomfortable. Often, an individual will experience panic attacks or extreme anxiety in specific situations.

Although anxiety disorders are among the most common and functionally impairing mental health disorders to occur in adolescence, there is paucity of comprehensive data on adolescent anxiety disorders in India.[3] Among the juvenile age group globally the reported prevalence of anxiety disorders vary from 6.9% to 27% which is more than the most often seen morbidity of mood disorders (6.4%), disruptive disorders (6.4%) and substance abuse (5.3%).

The reported prevalence of generalized anxiety disorder vary from 0.2 to 5.8%, social anxiety disorder vary 1.6 to 12.8%, panic disorder 0.2% to 10% of those attending child psychiatry clinics and separation anxiety disorder (4.1%).[4] The common symptoms among anxiety disorders in adolescents observed in India are: (i) anxious mood (12.6%), cognitive symptoms (9.9%) and physical symptoms (9.2%). The predominant symptoms among various sub types anxious mood; (i) panic disorder (32%), (ii) generalized anxiety disorder (12.2%), (iii) separation anxiety disorder (5.3%) and social anxiety disorder (1%).[5]

To screen for anxiety disorders among adolescents in primary care settings many self rating measures exists, a recent meta-analysis has shown that the most commonly used one to evaluate anxiety disorder symptoms is "Screen for Child Anxiety Related Emotional Disorders (SCARED)". SCARED, a self-rated questionnaire has 41 items under the five subscales of panic / somatic, generalized anxiety, separation anxiety, social phobia and school phobia. Adolescents are asked to rate the frequency with which they experience each symptom using a 3 point likert scale (0=almost never, 1=sometimes and 2=often). As against the original cut-off score of 31, a recent community study in Kerala has suggested a cut-off score of 21 for screening anxiety disorders among adolescents with better diagnostic accuracy properties.[6]

Depression

Historically, children were not considered candidates for depression. Today, childhood depression is widely recognized and health professionals see depression as a serious condition affecting both adolescents and young

children.[7] Because adolescents are already moody and unpredictable due to other changes and pressures in their lives, parents must know how to differentiate between the normal struggles of adolescent growth and serious emotional problems. One of the factors that make depression so difficult to diagnose in adolescents is the common behavior change that are normally associated with the hormonal changes of this period.[8] It has only been in recent years that the medical community has acknowledged childhood depression and viewed it as a condition, which requires intervention.

Self-esteem: One of the chief differences between adult and adolescent depression is that depression in adolescents usually involves more social and interpersonal difficulties, which directly leads to self-esteem problems. The inability to relate positively in social situations may lead to low self-esteem which leads to depression. The depression then leads to further inability to relate with others or be fully accepted in social groups which then adds to the feelings of low self-esteem.[9]

Autonomy: Another factor associated with adolescent depression and negative behaviors is difficulty in establishing autonomy in the adolescent's relationship with parents. Adolescent depression is seen in higher frequency in families where the children have difficulty establishing their own identity because of negative communication patterns and other dysfunctional family attributes.[10]

Suicidal ideation: Adolescents are also more likely to idealize suicide as a solution to feelings of helplessness. Adolescents may also socially isolate themselves when depressed out of feelings of guilt. Dramatic behaviors such as aggression and an obsession or fascination with death often accompany their depression.

Depressive disorders, which include major depressive disorder (unipolar depression), dysthymic disorder (chronic, mild depression), and bipolar disorder (manic-depression), can have far reaching effects on the functioning and adjustment of young people. Among both children and adolescents, depressive disorders confer an increased risk for illness and interpersonal and psychosocial difficulties that persist long after the depressive episode is resolved; in adolescents there is also an increased risk for substance abuse and suicidal behavior.[11,12,13]

Diagnosis

Symptoms of major depressive disorder common to adults, children and adolescents are: (i) persistent sad or irritable mood, (ii) loss of interest in activities once enjoyed, (iii) significant change in appetite or body weight, (iv) difficulty in sleeping or oversleeping, (v) psychomotor agitation or retardation, (vi) loss of energy, (vii) feelings of worthlessness or inappropriate guilt, (viii) difficulty concentrating and (ix) recurrent thoughts of death or suicide.[14] Five or more of these symptoms must persist for 2 or more weeks before a diagnosis of major depression is indicated. Depression may often be seen in physical ailments such as digestive problem, sleep disorders or persistent boredom (vegetative symptoms). Lamarine, considers that in children, depression may often be mistaken for other conditions such as attention deficit disorder, aggressiveness, physical illness, sleep and eating disorders and hyperactivity. Although depression in children may be confused with attention deficit hyperactivity disorder (ADHD), ADHD must begin before the age of 7.[15,16] According to Fritz, about 5% of adolescents suffer from depression symptoms such as persistent sadness, falling academic performance and a lack of interest in previously enjoyable tasks. In order to be considered major depression, symptoms such as suicidal thoughts, lack of appetite and loss of interest in social activities must continue for a period of at least two weeks.[17]

Community diagnosis, however usually rely on a formal testing using Beck's depression inventory (BDI) or Children's depression rating scale (Revised). Information provided by collaterals, including parents, teachers and community advisors should also be taken into account. Beck's Depression Inventory (BDI), is a mood measuring device developed by Dr.Aaron T.Beck (US). The device detects the presence of depression and accurately rates its severity. The multiple choice questionnaire has 21 groups of statement. The score ranges from 0 to 3 for each statement. The total score is 63.

The questionnaire is scored by adding up the score for each of the 21 items and obtaining the total. A score 21 and above suggest moderate depression needing individual cognitive behavior therapy and score 31 and above suggest severe depression needing medication in addition to psychotherapy.

Management

There are two main avenues to treatment: psychotherapy and medication. Often, both may be required. The majority of mild depression in adolescents respond to supportive psychotherapy with active listening, advice and encouragement. Issues of alcohol and substance abuse may have to be addressed by referral to relevant agencies. Formal family therapy may be required to deal

with specific problems or issues. Co-morbidity is not unusual in adolescents and possible pathology, including anxiety, obsessive-compulsive disorder, learning disability or attention deficit hyperactive disorder, should be searched for and treated, if present.

For the more serious and persistent depression, particularly those with vegetative symptoms or suicidal ideation, medication is essential and may be life-saving. Adolescents because of the common side effects, including sedation and anti-cholinergic action, generally poorly tolerate traditional antidepressant drugs. This leads to poor compliance. The advent of selective serotonin reuptake inhibitors (SSRIs) has largely put these worries to rest. SSRIs are well tolerated by adolescents because of their fairly rapid action and low tendency to cause side effects.

Low toxicity also makes them particularly helpful in an impulsive patient population. It is important that an adequate time period be given to allow the medication to work (four to six weeks) and that adequate doses are used (Table I).

Acute phase: The drug is continued for 4 - 6 weeks.

- Always target symptoms desired, the side effects, dose schedule and delayed onset of antidepressant action should be discussed.Look for side effects and if response is inadequate, increase the dose or change the drug.

Continuation phase: Continue the same dose for 6 - 12 months along with psychological methods.

Maintenance: This is to prevent recurrence of depression in the following situations which includes multiple severe episode, family history of bipolar disorder or recurrent depressive disorder, co morbid psychotic symptoms, stressful/ non supportive environment and residual symptoms.

While the recovery rate from a single episode of major depression in children and adolescents is quite high,[19] episodes are likely to recur.[20] In addition, youth with dysthymic disorder are at risk for developing major depression.[21] Prompt identification and treatment of depression can reduce its duration and severity and associated functional impairment.

In summary, mood disorders, particularly depression, are increasingly being recognized among adolescents. The adolescent may not look depressed always, instead may try to cover-up depression by showing over activity. The pediatrician must ask for evidence of a persistent feeling of (i) worthlessness, (ii) hopelessness, (iii) helplessness, (iv) no future at all and (v) suicidal ideation. In depression, the bio-psycho-social model denotes that there are neurotransmitters involved and hence drug therapy is of prime importance. Cognition or the thought process as such, primarily affects mood and hence the primary defect is in the thought process called cognitive error-over generalization, minimization of positive and maximization of negative attributes, necessitating cognitive behavior therapy by trained clinical psychologists.

The pediatrician must ensure the support of family, teachers and friends to maintain positive results of therapy and consult a psychiatrist, if no response at all after 6 weeks drug therapy, which may be extended for 6 months to one year.

Points to Remember

- ***Many of the mental health problems in adolescents can be effectively managed at the primary care setting itself.***
- ***Anxiety disorders are the most common and functionally impairing mental health disorders in adolescents.***
- ***Anxiety disorders are characterized by worry about future and current events and fear causing fast heart rate and tremors.***
- ***Depression in adolescents manifests with problems in establishing self-esteem and autonomy and occurrence of suicidal ideas.***
- ***Both psychotherapy and pharmacotherapy will be required in majority of adolescents with depression.***
- ***Support of family, teenagers and friends is essential to sustain the good results of treatment.***
- ***In case of no response in six weeks or whenever the primary pediatrician feels the need, psychiatrist has to be consulted.***

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NEWS AND NOTES

World Down Syndrome Congress (WDSC) 2015, Chennai

Date: 19-08-2015 to 21-08-2015

Enquiries to: Dr. Surekha Ramachandran Email: rekharami@gmail.co

Website link: http://wdsc2015.com

2nd Primer in Pediatric Nephrology for Asia, Singapore

Date: 19-08-2015 to 22-08-2015

Email: ckc_enquiries@nuhs.edu.sg

Website links: http://nuh.com.sg/ktp-nucmi/events/event-home.html

ADOLESCENT CARE

PARENTING AN ADOLESCENT

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Abstract: *Family connectedness during early adolescence is an important protective factor and is aimed at early adolescence. This delays sexual debut, reduces violence, delinquency, substance abuse, drunken driving, depression and suicide in future. World Health Organization recommends a five dimensional approach to parenting that is being included in intervention programs to help parents guide their adolescents. Three parenting styles determine the outcomes in adolescents as they reflect the demandingness and responsiveness in parents. Highly responsive and highly demanding parents seem to win the adolescents in compliance.*

Keywords: *Parenting, Adolescence, Issues*

Parents are defined to encompass "all those who provide significant and/or primary care for adolescents, over a significant period of the adolescent's life, without being paid as an employee," including biological parents, foster parents, adoptive parents, grandparents, other relatives and fictive kin such as godparents.[1]

Indian adolescents live with their parents until they move out either for education or for employment. In spite of physically staying away from the parental shelter most adolescents are being in touch with their parents in a regular manner. How and for what the parents interact with their adolescents determines the connection between them.

Psychosocially, an adolescent is in the process of searching for his identity keeping in mind the expectations of his parents, other family members, school and the social setting in which he lives. The parents are in the process of admitting the fact that their adolescent may not need their inputs soon in all aspects of life. It is easy for the parents to rejoice and accept independent walking by their toddler as an important milestone; but is very difficult to acknowledge the independent decision making skills by their adolescent as a significant appreciable milestone. Not only does it cause concern in the parents but also makes them realize that they have to let go of their role as parents soon.

World Health Organization has gathered and analyzed significant information from existing research and experience obtained from various intervention programs conducted in many countries to help parents recognize their role in preventing adolescents from indulging in high-risk health behaviours. In 1997, WHO, UNFPA and UNICEF jointly issued a technical report on 'Programming for adolescent health and development' where the following five areas were proposed as major themes of intervention that could be carried out to promote healthy development of this population (Box 1).[1]

Box 1. Major theme of intervention

- Creating a safe and supportive environment
- Providing accurate information
- Building skills
- Providing counsel
- Improving health services

Thus the home comprising the family members including parents is central to program interventions to prevent adolescent ill health. Family and parents provide support and love, promote moral development and a sense of responsibility, provide role models and education about culture, set expectations, negotiate for services and opportunities and filter out or counteract harmful or inconsistent impact from the social environment.[1]

Family connection as a protective factor

By definition, factors are called 'protective' if they discourage one or more behaviours that might lead to negative health outcomes (e.g., having sex with many partners) or encourage behaviours that might prevent a negative health outcome (e.g., using condoms and contraception).[2] Factors that are associated with increasing the likelihood of negative consequences of behaviour are called risk factors. Family connectedness, school

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connectedness, presence of a trustworthy adult and spirituality are the major protective factors in the lives of adolescents. Thus, parents play a key role in three of the four protective factors and contribute to a large extent in enjoying the fourth protective factor.

Literature reveals that parents are the most significant adults in the life of adolescents. Information to create awareness on aspects like growing up, mood variations, substance use and sexuality are better received if offered by parents during early adolescence. Boys and girls between 10 and 14 are called 'very young adolescents' who are connected with the parents and are still receptive as they are in the process of understanding themselves. As adolescents of this group learn to think about their identity they begin to question their parents about the relevance of each instruction given by their parents. Though this perturbs the parents, it gives them an opportunity to place the facts and explain the consequences of the various actions that could be performed by the emotionally charged adolescent.

The five dimensions[1] of parenting which have influence on adolescents are the following.

a. Connection: A positive and stable emotional bond between the parent and the adolescent established over the entire lifespan of the child conveys love, affection, compassion and warmth. Early childhood attachment helps in forming secure relationships.

b. Behaviour control: Structured approach to supervise, monitor, limit, regulate the behaviour of adolescents, with negotiation and guidance to promote health in the adolescents; conveying that the well being of their children is of foremost importance, helps in regulating behaviour.

c. Respect for individuality: Allowing the adolescent to search for his or her identity resulting in self-worth and self-concept: for example seeking information about substances and then taking a decision to stay away from using them is a healthier way. This takes time but with guidance the process can be augmented.

d. Modelling of appropriate behaviour: Children and adolescents imbibe values, ethics and etiquette practised by their parents and significant adults. Appropriate role modelling promotes healthy behaviours in adolescents.

e. Provision and protection: Parents may not have resources to provide all material comforts and information to their adolescent, but they can promote access to resources by empowering their children. Protection cannot be offered all through but skills to protect oneself from harm can be imparted by the parents, e.g. empowering with skills to say "no" to sexual exploitation.

Parents should be given information about adolescent health and development and should be provided opportunities to equip themselves with communication skills to transform the knowledge to enlighten the adolescents in a useful manner.

Styles of parenting[3]

Depending on the parental expectations and responsiveness to their children, Diana Baumrind classified parenting styles into four types, a) Authoritarian: Highly demanding and less responsive, b) Permissive / Indulgent: Highly responsive and not demanding, but over indulging, c) Authoritative: Highly demanding and highly responsive with role modelling and d) Neglectful: Not demanding and not responsive and not available. Table I highlights the additional characteristics of each of the parenting style. Each parenting style promotes specific psychosocial developmental aspects in the child. Highly responsive parent will create an immense sense of well being and trust in the child. Promoting higher responsiveness in parents is being currently thought to improve treatment outcomes by enhancing adherence.[4]

Positive parenting and adolescent behaviour

Numerous studies have made us understand that adolescents who have parents who practice authoritative style of parenting evolve as well-adjusted, well-informed and capable adolescents who seem to have the skills to handle challenges independently using effective problem solving skills. Parental willingness to provide autonomy since early childhood for handling age appropriate challenges with an assurance of guidance in the event of necessity promotes emancipation in the adolescent. Parental responsiveness like being available to share strong emotions that emerge within the family setting or outside contribute to the acquisition of emotional regulation skills in the adolescents. Availability of an active listener especially the parent enhances expression of emotions with a resultant reduction in issues such as depression, suicidal ideation, violence, use of weapons, delinquency, school dropout, substance use and drunken driving. Thus positive reinforcement of an accepted behaviour with inputs to understand the negative consequences of a behaviour that cannot be accepted paves the way to better activities during adolescence.

Adolescents feel less burdened and relieved when they are able to share their emotions and experiences with their parents; especially if the parents are capable of appropriate empathy and compassion. Adolescents do not look for readymade solutions but regard parents as a source of major support in the events of crises.

Table I. Characteristic feature of the four parenting styles[5]

Feature/ Parenting Style	Authoritarian	Permissive / Indulgent	Authoritative	Neglectful
Demandingness / Expectations	High	Nil	High	Nil
Responsiveness	Less	High	High	Nil
Style of communication	Aggressive	Passive/submissive	Assertive	Poor/ limited to few words
Expression of love	Care is considered as love	Well expressed	Well expressed	Rarely expressed
Demonstrative love	Not expressed	Well expressed	Well expressed	Nil
Time spent with children	Less	High	High	Minimal
Value inculcation	Through instruction, "Do as I say, and not do as I do"	Left to child as, "Whatever you do is right for you!"	"We are value-loaded and follow what we practice" Explanations are provided for each expectation	No attempt at value inculcation is made
Insistence on discipline	High with psychological control like black mailing, name calling and sometimes physical abuse	Nil	Firm and high with explanation and negotiation and differential reinforcement like encouragement for positive behaviours and ignoring negative behaviours	Nil
Role modelling	Minimal	Nil, entire household is child centred	Very high	Nil
Compassion	Nil	High	Very high	Nil
Freedom of speech and expression	Nil	Total freedom, child will not know how to make use of the freedom, gets confused and anxious	Freedom of expression is given, with lot of inputs from parents on the right and the wrong. Guidance on modification is offered with compassion.	As there is not much of contact, expression is determined by the children who feel not guided.
Moralistic	Highly moralistic	Willing to relax all moral values to see happiness in children	High, but not judgmental. Guidance is offered always	Not applicable

Feature/ Parenting Style	Authoritarian	Permissive / Indulgent	Authoritative	Neglectful
Opinionated	Yes	Nil	Not opinionated but explains possible opinions that can be formed by certain behaviours	Not applicable
Care of children	Care is complete, all acts of service done in a methodical but in an impersonal manner. Attempt to self care is not encouraged. Dependency is seen as obedience and subordination.	Very good with personal attention to begin with; later with scare that the child might scold the parents for lapses	Very good with personal attention, involving children in inculcation of habits of self care very early in life, generation of independence and preparation of the children to survive without the support of parents is practiced	Care is not adequate and children usually fend for themselves with breach in self care and hygiene.
Appreciation and encouragement	Rarely offered	Offered more than necessary as the parents are scared at consequences if not offered	Offered in right doses at right occasions.	Not offered as the parents are rarely aware of accomplishments
Criticism	Liberal and frequent	Nil	Yes but with explanations and ways to rectify	Rarely take note of deviance in behaviours or actions.
Outcome in children	Submissive or rebellious, lack of self-control, self-esteem and self-confidence as they have been controlled always. Performance anxiety is high and accomplishments are less	Self-driven, anxious at outcomes as they have not been exposed to challenges appropriately with guidance from parents; self-esteem low with reduced confidence at new activities	Self-motivated, self-disciplined and self-confident, children with high self-esteem and frequent success in many tasks.	Feel that they lack direction or may latch on to people to receive direction which may be right or wrong.
Children's perception about the parents	Strict, controlling parents who gave no space for growth	Ineffective parents who offered no guidance and direction	Excellent parents who were available always but at the same time encouraged independence; offered help whenever necessary	Non-available parents who were totally away in mind, body and actions.

Parenting and sexuality[6–8]

New HIV infections in young people between 15 and 25 years have been the motivating factor to conduct researches on the role of parents in empowering the adolescents before they turn 15 about matters related to sex. In most parts of the world sex is considered a taboo subject and rarely do parents give information to their children. Open communication by parents about matters relating to sex, especially when an adolescent shows curiosity to know more about marriage, relationships, child birth and related issues, have been found to delay sexual debut, are related to an increased use of contraception and promote more stable relationships with reduction in promiscuity. Equipping parents to handle this challenge using an effective communication style is the major agenda for health care professionals.

Parenting and life style in adolescents

Life style factors such as physical activity, nutritional intake, sleep routine, hygiene and hobbies are influenced by the parents' inputs either as suggestions or as role models. Parents who observe authoritarian style insist on certain practices with firmness amounting to aggression. Few adolescents might perceive this as an intrusion into their privacy and might rebel actively by arguing with the parents or passively by non-cooperation. Parents who are permissive would allow the adolescent to take decisions and thus indirectly promote ill health. Authoritative parents establish a routine with appropriate explanation and negotiation.

Role of health care professionals

Health care professionals can play a major role in the following ways.

- Making parents comfortable with the growth and development during adolescence
- Equipping the parents with the aspects of the psychosocial developmental stage in which the adolescents are expected to evolve
- Enriching the parents with strategies to assist in the exploration of identity by their adolescent son or daughter
- Enabling the parents to allow space for the adolescent to develop autonomy and permit individualization
- Empowering the parents with essential communication tools to establish effective connection with their adolescents
- Enhancing the existing monitoring and guidance principles practised by the parents that would help in early detection of deviations in behaviour
- Expanding the horizon of the parents by including the peer group of their adolescents in a non-judgmental manner
- Exposing the parents to the benefits of sharing information on sexuality with their adolescents and thus ascertaining the value of open parent–adolescent communication on delicate topics as a successful method to prevent high risk health behaviours in the next generation.

Points to Remember

- Educate the parents on adolescent growth and development.
- Empower parents on effective communication skills.
- Enlighten the parents to include taboo subjects like sexuality, substance use in their discussions with adolescents.
- Ensure the inculcation of authoritative parenting style by all parents since the first meeting.

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ADOLESCENT CARE

ADOLESCENT COUNSELING

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Abstract: *Adolescent counseling is a sensitive and skillful task needing knowledge and practical training in various theories approaches to the process. Pediatricians as primary mental health caretakers should take the responsibility towards shaping the attitudes, emotional health and responsible behavior of teenagers. Adolescent mental health is a neglected topic in India in spite of exponential rise in risk taking behaviors among teens. Adolescent counseling spreads over important areas of teen's life including life skills, scholastics, prevention of substance abuse, safety, sexual abuse, responsible sexual behaviors, career guidance and premarital issues. Ethics and adolescent friendly approach is all that is needed.*

Keywords: *Adolescent, Counseling, Skills.*

Counseling psychology is one of the largest subfields in psychology. It is centered on offering therapy and aiding clients who suffer from psychological distress and mental illness. According to The Society of Counseling Psychology, the goal of counseling psychology is to improve personal functioning by focusing on social, emotional, educational, health, developmental, family and work-related issues. Some amount of depression, thought distractions, anxiety, phobias, tendency to experiment and even blues (sadness) are normal for teenagers. Intervention is needed only when these troubles augment to cause emotional disturbance and hinder the mental growth and potential of the individual. The purpose of intervention counseling is prevention, remediation, learning new skills (behavior modification), growth and personality development.

Various thinkers, theories, languages and cultures have given different meanings to the word 'counseling'. Like the terms 'personality' or 'intelligence', many scholars have defined counseling in various ways. However, all the psychologists agree on the ultimate purpose or aim of counseling i.e. helping the individuals to overcome future problems. The Oxford dictionary defines mentions counseling as "the provision of professional assistance and guidance in resolving personal or psychological problems".

Perez (1965) gave a popular definition of counseling as an interactive process conjoining the counselee who needs assistance and the counselor who is trained and educated to offer this assistance. This interactive process needs to be initiated, facilitated and maintained by the counselor through feelings of spontaneity, warmth, tolerance, respect and sincerity. Carl Rogers in his book "Counseling and Psychotherapy", has defined counseling as a process consisting of a definitely structured permissive relationship which allows the client to gain an understanding of self to a degree which enables the client to take positive steps in the light of his/her new orientation. Rogers also adds that in the process of counseling, the structure of the self is relaxed in the safety of the client's relationship with the therapist and previously denied experiences are perceived and then integrated into an altered self.

Most of the definitions proposed by renowned scholars maintain that counseling is a process which involves bringing about sequential changes over a period of time, leading to a set goal. Over the years, due to exponential growth of various old and new stressors, the scope of counseling has evolved into multiple fields of human life and at all ages (Fig.1).

Counseling, guidance and psychotherapy are terminologies which are used synonymously by many, although their meanings differ practically (Table I).

For the benefit of practitioners, researchers and theoreticians, the Western Inter-State Commission for Higher Education (WICHE) led by Parker (1974) has proposed a 3 dimensional model for the functions or roles of a counselor. This further elaborates and specifies the scope of counseling in the modern world (Fig.2).

Mental health professionals employ various theories of counseling in dealing with their clients with an approach that keeps the "best benefit of client's growth in focus"

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Fig.1. The scope of counseling

(eclectic approach). The choice and combination of the theories lies with the professional only and depends on the case at hand. Although different concepts are used either singly or in combination, many professionals follow selective theories only.

Special issues in counseling adolescents

Many adolescents are at times difficult to counsel. The obvious reasons are unwillingness for therapy, unfriendly attitudes of the therapist, non-acceptance of problem leading to resistance, tendency to blame the caretakers, social stigma, poor compliance leading to drop-outs, peer influence and a feeling of being victimised by parents or teachers. However, the positive points about adolescent counseling cannot be ignored. Adolescents are quite receptive to the process provided the counselor exhibits skillful and adolescent friendly interview. Further, because adolescence is a period of storm and stress with multiple happenings, adolescents are looking for someone

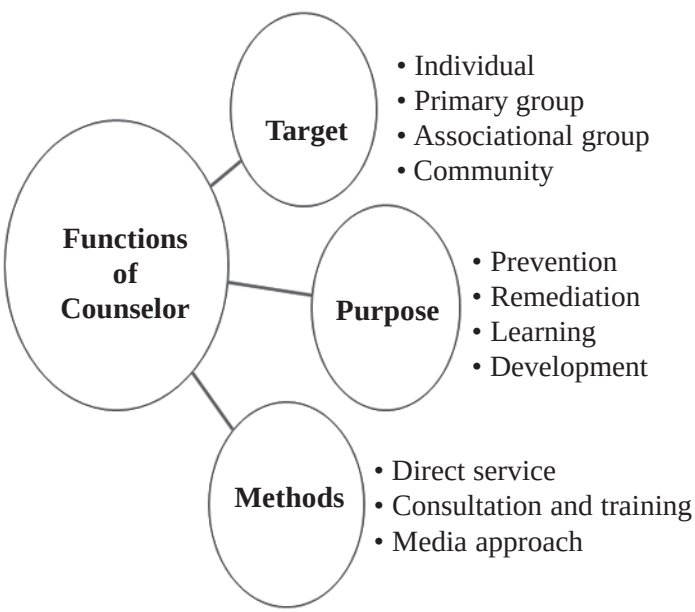

Fig.2. Three dimensional model for counselor's role proposed by WICHE

Table I. Comparison between guidance, counseling and psychotherapy

	Guidance	Counseling	Psychotherapy
Aim	Making choices/decisions in future for otherwise normal people in difficulties.	Focus on the present problem in a disturbed individual who is capable of changing into a "fully functioning individual".	Restructuring of personality with focus on the root causes/conflicts originating due to past experiences or mental trauma in mentally disordered individuals.
Role of the director or professional	As guide, information provider, supervisor or assistant for short term only (1-3 sessions).	Purely mental with communicator's and facilitator's role for dealing with the deranged triad of thinking, feeling and behaving. May take 1-15 sessions.	Long term intervention as trained and sometimes dominant authority in exploration and resolution of conflicts or unconscious processes that resulted in severe emotional disturbances in the client/counselee.
Problem in the patient or client	Confusion in choices and unclear vocational ideas or concepts.	Stress related situation/crisis which could be due to intrapersonal or interpersonal disturbances.	Severe mental health disorder (psychosis, severe anxiety, phobias, severe depression, etc.) usually needing pharmacotherapy and/or electro convulsive therapy (ECT) in addition.
Process	Quantitative analysis, problem solving techniques and listing of possibilities using information pool.	Qualitative as well as quantitative tools and specialty skills which may be cognitive, emotive or behavioral.	Mostly qualitative, directive and interpretative due to severity of disorders. Psychoanalysis including dream therapy or cognitive behavior therapy in conjunction with (ECT) and drugs.
Objectives	Educational or vocational or career improvement.	Positive self esteem, capacity building and autonomy for becoming a fully functional and responsible human.	Empowerment for adjustment, functionality and improved mental health.

who will understand them and be with them unconditionally and with respect. Frontalization of brain and pruning (unused neuronal connections are chopped off) occur during adolescence which contributes to the development of adult personality. For a counselor and caretaker, adolescence should be looked upon as a period when basic life philosophies take shape and interventions done during this period will have a long term benefit, provided the counselor is adolescent friendly. The basic principles and the skills used in counseling are shown in Fig.3.

The counselor should be interested in helping adolescents, have perceptual sensitivity, normally adjusted in his/her personal life, genuine and well trained with congruence and good emotional control. A lot of importance is given by Carl Rogers to the term "Acceptance". He defined acceptance as "a warm regard for the client as a person of unconditional self worth and of value under any condition, behavior or feelings." Acceptance implies helping an individual and not controlling him/her.

A counselor should follow work ethics like anonymity, confidentiality and record keepings. Advice giving, lecturing, excessive questioning, storytelling, etc should be avoided.

The effective communication skills Fig.3, 4, 5 that work wonders with an adolescent client are:

1. Non-verbal tools: These account for nearly 80% of total interaction and include eye contact, smile, unfolded arms with flexed knees, minimum body movements and safe physical proximity.

2. Active listening: This process which aims at understanding the feelings and emotions along with spoken words and includes means to minimize ambient distractions, facilitating responses, paraphrasing or reflecting the feelings and words for the adolescent and

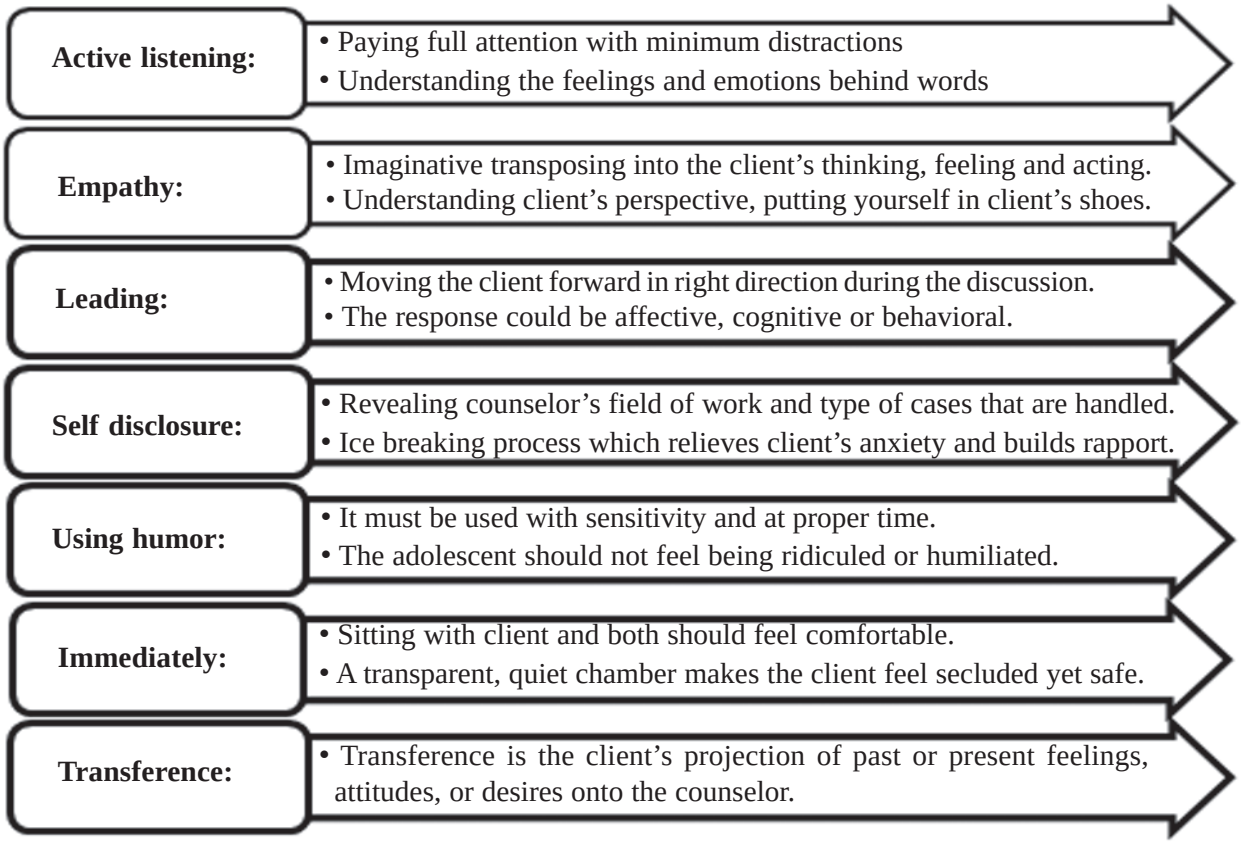

Fig.3. Principles and basic skills of counseling

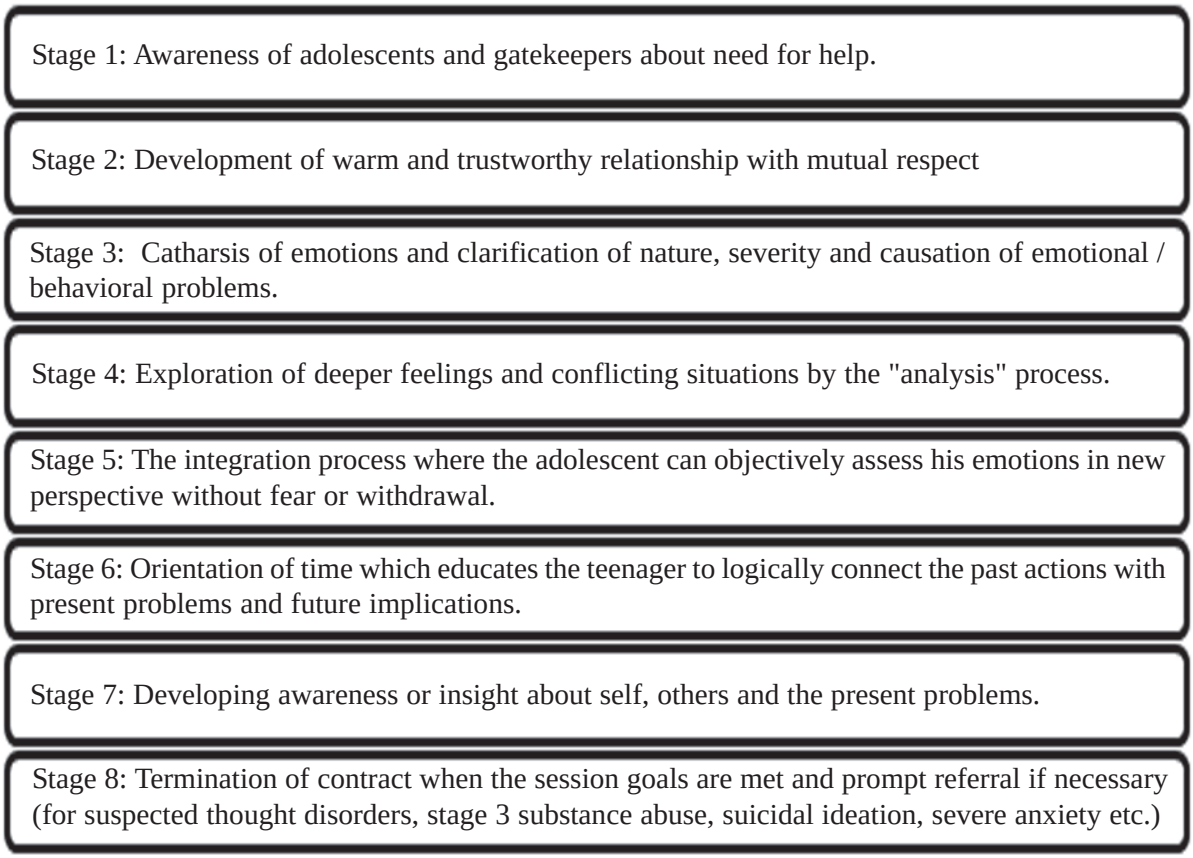

Fig.4. The counseling process

summarizing the key points. Silence at certain moments is a very effective tool that aids in ventilation of clouded emotions, gives breathing space for restructuring words, putting thoughts sequentially and understanding the exact flow of emotions. Silence can thus be paradoxically called the most useful tool in interpersonal communication.

3. Verbal tools: A right choice of words and questions in a soft tone which facilitate communication must be learnt for proper counseling. Open ended questions (Can you describe your feelings?, what do you think about your relationship with parents?, What is it that you don't like about your school?, What are the ways a teenager can enjoy life?), yield descriptive answers unlike parallel closed ended questions (Did you feel sad?, Is your relationship with your parents fine?, Do you like your school?, Do you enjoy life?), which yield yes or no responses and are not helpful. The steps is counseling process is shown in Fig.4.

Role of pediatrician has extended from school health check-up to imparting family life education (or premarital

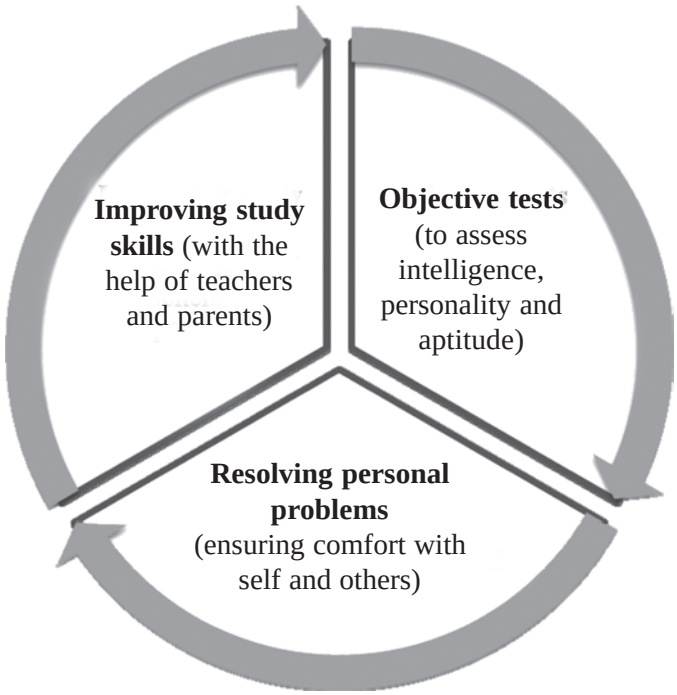

Fig.5. Areas of educational counseling

1. Self awareness: recognition of our character, strength, weakness, likes and dislikes. Aprerequisite for effective communication.
2. Empathy: Imaging what is life for another person. It helps us understand and accept others and develops a nurturing attitude.
3. Interpersonal relations: It helps us develop and nurture supportive networks and also enables to end sore relationships constructively.
4. Effective communication: Capacity to express ourselves both verbally and non-verbally in culturally appropriate ways and situations.
5. Critical thinking: Ability to analyze information and experiences in an objective manner. Helps to develop right attitudes and behaviors.
6. Creative thinking: It enables us to explore available options, view consequences of actions and respond adaptively and flexibly.
7. Decision making: It involves defining the problem, considering consequences, family values and preferences to make right decisions.
8. Problem solving: Involves identifying needs and attitudes, detailed definition of problem, brainstorming and evaluation of various solutions.
9. Coping with emotions: Identifying effects of emotions on self and others. Understanding the triad of thinking, feeling and behaving.
10. Coping with stress: Identifying the stressors, time management, positive thinking, behavior modifications and relaxation techniques.

Fig.6. Ten basic life skills (WHO)

counseling for college graduates) and helping teacher counselors to improve scholastic performance in children. Life skills education is also an important tool for improving mental health.

Life skills education (LSE) is a didactic, psycho-educational, active-directive and eclectic approach of counseling adolescents. World health organization (WHO) defines life skills as the abilities for adaptive and positive behavior that enable individuals to deal effectively with the demands and challenges of everyday life while UNICEF defines life skills based education as being a behavior change approach, designed to a balance of knowledge, attitude and skills. Life skills are usually used in combination to deal with real life situations (Fig.5).

The goals of LSE are 1) Curative (pertaining to present problems) and 2) Preventive (to avoid future problems). LSE is one of the useful tools for imparting sexuality education, teaching responsible behaviors and adolescent counseling individually or in groups (Fig.6).

Adolescent counseling by a well-trained professional is the need of the hour and pediatricians, as primary mental health caretakers of children and adolescents, need to take this responsibility.

Points to Remember

- ***Adolescent counseling is the need of the hour.***
- ***Special skills must be learnt beforehand.***
- ***Pediatricians are the key persons in early detection, management and timely referrals for mental health problems in adolescents.***

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CLIPPINGS

Alcohol Use in Films and Adolescent Alcohol Use

A cross-sectional study with 5163 15-year-olds from the Avon Longitudinal Study of parents and children in United Kingdom was done to investigate whether exposure to alcohol use in films (AUFs) is associated with early alcohol use, binge drinking, and alcohol-related problems in British adolescents. After adjustment, adolescents with the highest exposure to AUFs were 1.2 (95% confidence interval [CI]: 1.1–1.3) times more likely to have tried alcohol compared with those least exposed and 1.7 (95% CI: 1.5–2.0) times more likely to binge drink. They were 2.4 (95% CI: 1.9–3.1) times more likely to drink weekly and 2.0 (95% CI: 1.7–2.4) times more likely to have alcohol-related problems than those least exposed.

Exposure to AUFs is associated with higher risk of alcohol use and alcohol-related problems in UK adolescents and support the argument that a review of film-rating categories and alcohol ratings for all films may help reduce problem-related alcohol consumption in young people.

Waylen A, Leary S, Ness A, Sargent J. Alcohol Use in Films and Adolescent Alcohol Use Pediatrics. Published online April 13, 2015(doi: 10.1542/peds.2014-2978).

GENERAL ARTICLE

FLUOROSIS AND ASSOCIATED HEALTH ISSUES

***Susheela AK**

Abstract: *In this update the types of fluorosis affecting different tissues / organs / systems in the body, their characteristics, how fluorosis can be suspected from the symptoms and how it can be confirmed based on the diagnostic procedures are discussed. After early diagnosis, complete recovery from fluorosis, is achieved by withdrawal of fluoride consumption through diet modification. Diet counselling to promote intake of nutrients, vitamins and antioxidants has been highlighted. Drugs are less effective in the recovery process. This article also deals with associated health problems due to fluoride toxicity such as anemia in pregnancy despite iron and folic acid supplementation and anemia in adolescent girls. This article also details the approaches for addressing fluorosis in children and the commonalities with iodine deficiency disorders (IDD).*

Keywords: *Fluorosis, Diagnosis, Recovery.*

Fluorosis, was detected in 1930's both in cattle and in human beings in Nellore district, then in Madras Presidency and was reported in the Indian Medical Gazette.[1] At that time the disease did not draw the attention of health administrators and policy makers, as during the Second World War and post independent era, the priorities in health sector were different. However, the publications made significant impact on the disease characteristics in the western world. The contributions of Amarjit Singh and Jolly from Punjab were classics in the understanding of the disease.[2,3,4] Until the 1970s, the two major entities of the disease recognized and reported were dental and skeletal fluorosis and for a good number of professionals and scientists, fluorosis meant only dental and skeletal fluorosis. But the nation marched forward to find new scientific dimensions, and thus other health problems that are of immense significance for addressing the disease were identified.

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As research activities gained momentum, it was inconceivable that an environmental toxin, a powerful, corrosive chemical 'fluoride', upon entering the body would selectively attack only the teeth and bones. The concept that fluoride being a negatively charged anion (F^{-}) would be reacting with positively charged cations such as calcium (Ca^{++}) is acceptable; but positively charged cations are present in abundance in soft tissues as well. The major breakthrough in the understanding of soft tissue involvement in fluorosis led on to a third entity, "non-skeletal fluorosis"[5], which was considered to be the beginning of a new era. "Non-skeletal fluorosis" and its clinical manifestations led to diagnosis of fluorosis at very early onset and complete recovery from fluorosis became a reality. Unfortunately, the early manifestations are still "non-specific".

This article deals not only with classical dental, skeletal and non-skeletal fluorosis but also the recent developments in the understanding of associated health problems due to fluoride toxicity in new born, infants, children and pregnant women as more information in greater detail is available in recent years.[6,7,8]

Fluorosis and its manifestations

Dental fluorosis (DF): This affects children during development of teeth when the mother has consumed or inhaled fluoride through food, drinking water, drugs, dental products and / or industrial emission. DF may start from intra-uterine life when tooth germ erupts. When the disease sets in, it disfigures the matrix molecules of the teeth. The permanent teeth in children would then develop discoloration. The discoloration on the enamel surface is horizontal, away from the gums and bilaterally symmetrical. It is not reversible, but masking the discoloration of teeth during adolescence is done as it causes social and cosmetic problems.

Skeletal fluorosis (SkF): This afflicts the bones and major joints (excluding joints in the hand and feet) of young and old, men and women without discrimination. In advanced stages of skeletal fluorosis, it is not reversible. Severe pain in joints and rigidity or stiffness in joints would incapacitate an individual. Patients of skeletal fluorosis may also get paralysis.

Non-Skeletal Fluorosis (NSkF): This is the earliest manifestation of fluorosis. The clinical manifestations of non-skeletal fluorosis are seldom understood. Fluorosis may not be suspected based on the history and the symptoms. The manifestations that may overlap with other diseases are quite non-specific. Yet another possibility is that the health complaints are considered as non-specific and ignored. The elicitation of proper history and conduct of diagnostic tests for confirmation of the disease are of prime importance.

The questionnaire which can be used for screening fluorosis is given in Box 1.

While dealing with a suspected case of fluorosis the name of the state, district or village where the patient is/ was residing and source of water used for drinking, cooking and duration of stay, provides valuable information.

Whenever fluorosis is suspected it is necessary to confirm the diagnosis.

Diagnostic procedure for fluorosis

The diagnostic procedure for fluorosis needs the following tests done:

(1) Fluoride content of (a) drinking water (b) serum (c) urine.

(2) X-ray of forearm antero - posterior (AP)

(3) Baseline hemoglobin (would be used for impact assessment during the recovery process)

Precautions to be taken while estimating fluoride levels in blood, urine and water source

1. The drinking water, urine and serum samples for fluoride testing is collected in plastic bottles /vials and not in glass bottles. Fluoride would bind to silica in glass, resulting in erroneous results.

2. Spot samples of urine and blood: 30 ml sample volume for water and urine; 1 ml serum.

Box 1. Proforma for screening for fluorosis

A. Non-skeletal manifestations with focus on Gastro-intestinal system (tick ✓)

1. Nausea / Loss of appetite:	☐	4. Constipation:	☐
2. Flatulence	☐	5. Diarrhea (intermittent):	☐
3. Pain in the abdomen:	☐	6. Headache:	☐

B. Other Non-skeletal manifestations.

1. Polyuria	☐	4. Muscle weakness:	☐
2. Polydipsia:	☐	5. Unenthusiastic and prefers to sleep whole day:	☐
3. Fatigue (extreme tiredness):	☐	6. Allergic reaction on the skin (which may appear/ disappear)	☐

C. Skeletal manifestations

1. Pain in major joints	☐
2. Stiffness in the major joints with pain	☐
3. Immovable joints with pain	☐

D. Dental Fluorosis:

1. Discoloration on enamel surface (away from the gums) seen, irrespective of age ☐

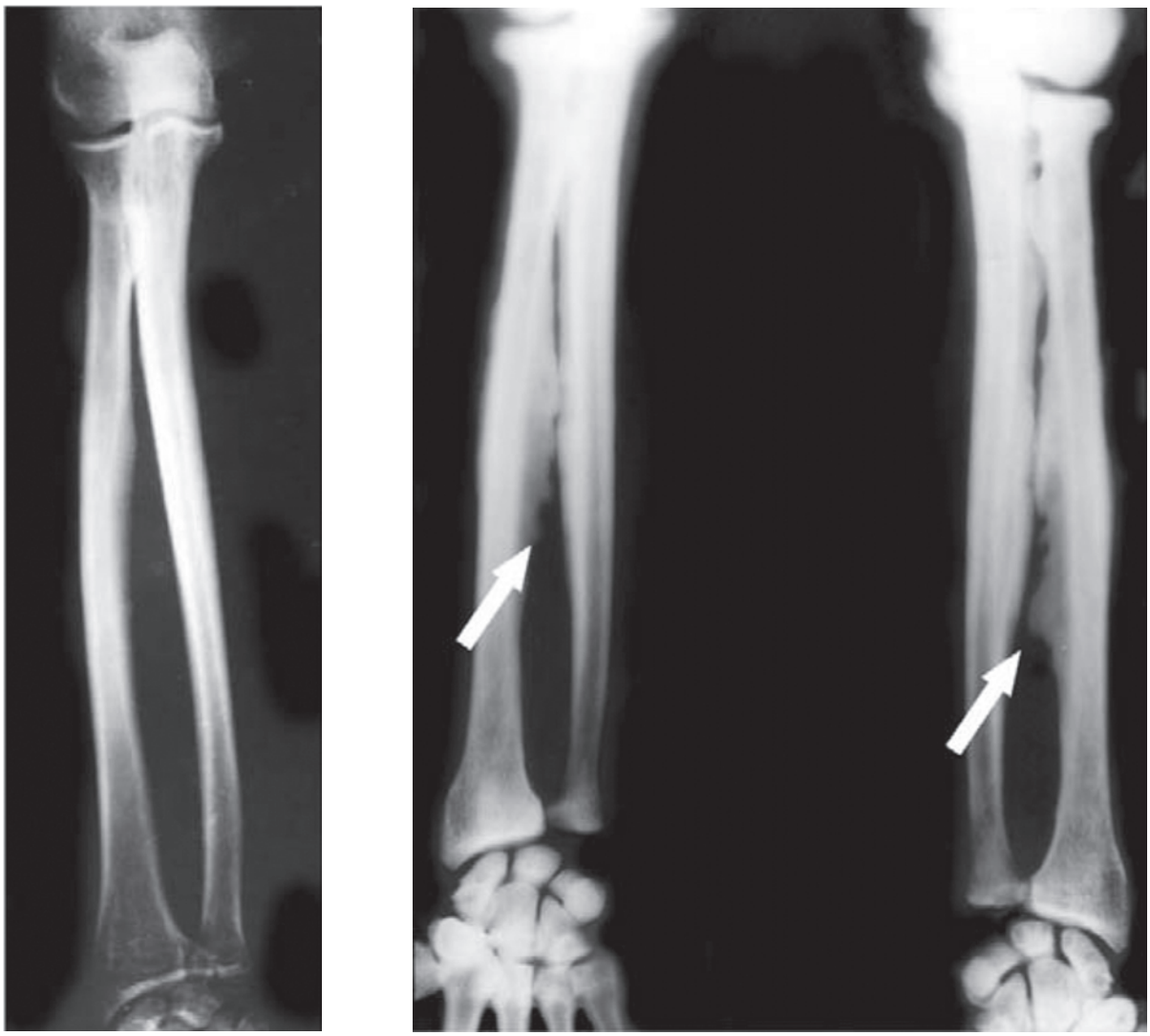
3. For assessing whether interosseous membrane covering the radius and ulna is calcified or not, an X-ray of forearm bones AP is required.

Evaluation of test results

Normal reference range for fluoride in:

- Urine: 0.1 - 1.0 mg/L
- Serum: 0.02 - 0.05 mg/:L
- Drinking water: 1.0 mg/L is the upper limit the body may tolerate; less the better as fluoride is injurious to health (Bureau of Indian Standards, 2012).[9]
- X-ray forearm AP (Fig.1): While looking at the X-ray forearm it is necessary to look for the interosseous membrane calcification. It has to be noted that the calcified interosseous membrane may be mistaken for "inflammatory reaction of the membrane" and may not be attributed any importance while reporting.

The diagnosis is confirmed on the basis of the test reports of high levels of F^- in body fluids and interosseous membrane calcification. It is possible that the drinking water F^- is within safe limits. Then it can be inferred that the source of fluoride may be food, beverages, dental products, industrial emission, chewing habit of foaming substances, viz. churans, supari or tobacco and use of black



Normal fore-arm x-ray **Fluorosed fore-arm x-ray**

(Fluorosed fore-arm x-ray radiographs, revealing calcified interosseous membrane (arrows) sticking out as thorns)

Fig. 1. Radiograph of normal and fluorosed fore-arm x-ray

rock salt as a spice (with 157 ppm F^-).[10] The markets are flooded with ready-to-cook gravy with F^- containing spices, cocktail party snacks, soft drink preparation such as jaljeera which are high in fluoride due to addition of black rock salt (CaF_2). Fluorosis afflicting a number of urban elite has been traced to consumption of high F^- salt through a variety of food items, beverages, black tea, lemon tea etc.

Management of fluorosis

The best option for the patient is to undergo counselling for diet editing for withdrawal of all fluoride sources from use/consumption. Simultaneously the patient should undergo diet counselling for promotion of intake of nutrients (essential nutrients, micronutrients, vitamins and antioxidants) through dairy products, vegetables and fruits. The concept of consumption of fruits and vegetables has considerable variation from family to family and may not be rewarding when left to the decision of the family. Diet editing and diet counselling are integral parts of the recovery process.

The regular hospital dieticians have an extremely important role and should be updated to understand the implications for offering an effective counselling for the patients of fluorosis to recover within a matter of 10 to 15 days. The issue that may confront the treating physician, is that the patient shall not be happy, if he or she does not receive a prescription for medicines. The doctor may at such times prescribe calcium and vitamin C tablets for a few days, until the dietary regime is standardized and the patient is comfortable with adequate intake of nutrients through dietary sources. The patient may be counselled that with dietary option the recovery will be in 10 to 15 days while it may take longer with tablets alone.

The recovery needs to be monitored and patient should be informed to come for review after 4 weeks post-intervention for assessing the impact of the interventions by re-testing i) urine fluoride and ii) hemoglobin along with re-assessing health complaints. Reduction in urine fluoride levels and rise in hemoglobin with disappearance of symptoms of non-skeletal fluorosis to a large extent would be the first impact on the health of the patient after interventions are practised. The need for the patient to be brought for follow up has to be re-emphasized including reassessment after 3 months and / or 6 months. There can be a recurrence of symptoms if there is contamination of drinking and cooking water with high fluoride due to industrial effluents. The ideal management for such patients would be to procure a RO water filter system so that even if industrial effluents are discharged into the ground contaminating the drinking water source, they are not affected by the fluorides.

The research and development activities in the field of fluorosis have enriched the nation with a wealth of additional information and contributed immensely for the welfare of the society. (Fig.2) highlights the four discoveries with focus on fluoride action and the strategy for prevention and control of the public health problems.

The major reasons for the delay in rectifying the important health problems confronting pregnant women and infants include the wide publicity and promotion of fluoride as an essential element for good teeth and prevention of dental caries in children and the gross unawareness among the medical fraternity and health administrators of the necessity to develop infrastructure for testing fluoride in body fluids, to diagnose fluorosis and associated health problems. The role of fluoride from that of an essential trace element to that of a toxic,

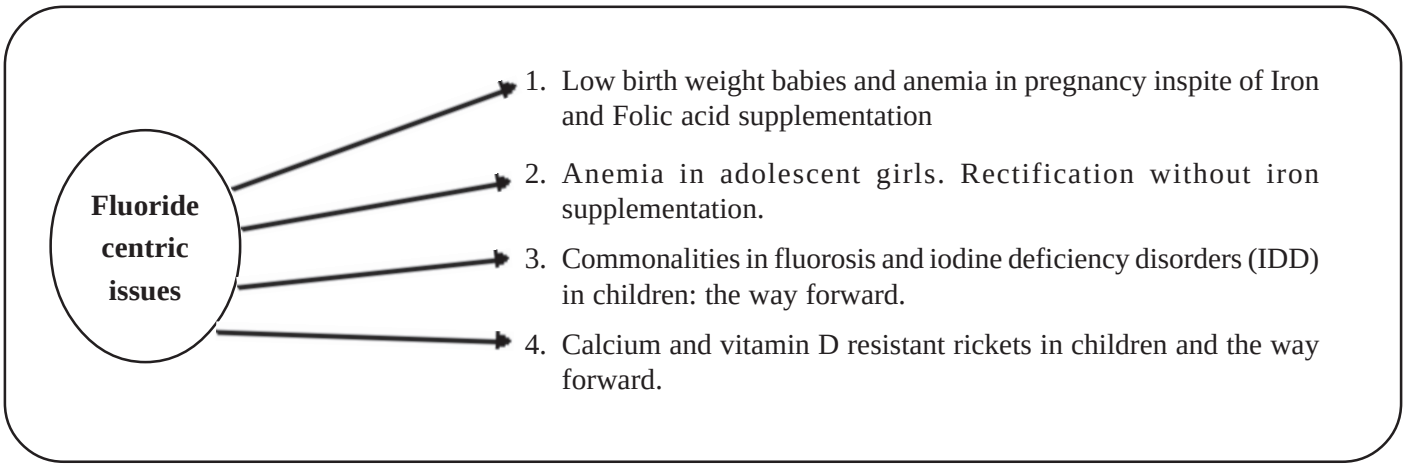


Fig. 2. Discoveries with focus on fluoride action

corrosive, poisonous element will be a major change. In the national norms for "Recommended Dietary Allowances"[11], there is no guideline for fluoride. More and more institutes in the health sector, starting from district hospitals to teaching hospitals are beginning to understand the importance of development of human resource, skills and infrastructure to address fluorosis and associated health problems. The use of fluoridated dental products, in the name of prevention of dental caries, based on the information available is unethical, unscientific and an out-dated concept.

Action of fluoride in the biological system

While addressing the associated problems, it may be meaningful to provide the details on how fluoride acts essentially causing anemia in antenatal women resulting in low birth weight babies.

Fluoride consumption through food, water and use of dental products essentially destroys the gastro-intestinal mucosa (Figs.3-5). Mucus production by the goblet cells is considerably reduced and microvilli of the mucosa fall off which are the most damaging effects of fluoride consumption. The function of the microvilli is to absorb nutrients from the diet, including orally administered iron and folic acid provided to pregnant women.[12] If there is scanty mucus production, the individual would be constipated. The wide publicity that is 'on' in the country for correction of anemia, designates the condition as under-nutrition and/or malnutrition. It may be more appropriate now to add the terminology "non-absorption of nutrients" for the reasons cited above.

Secondly fluoride ingestion destroys the probiotics, (the good bacteria) in the intestine which produce vitamin B12, an essential ingredient for hemoglobin biosynthesis.[13,14]

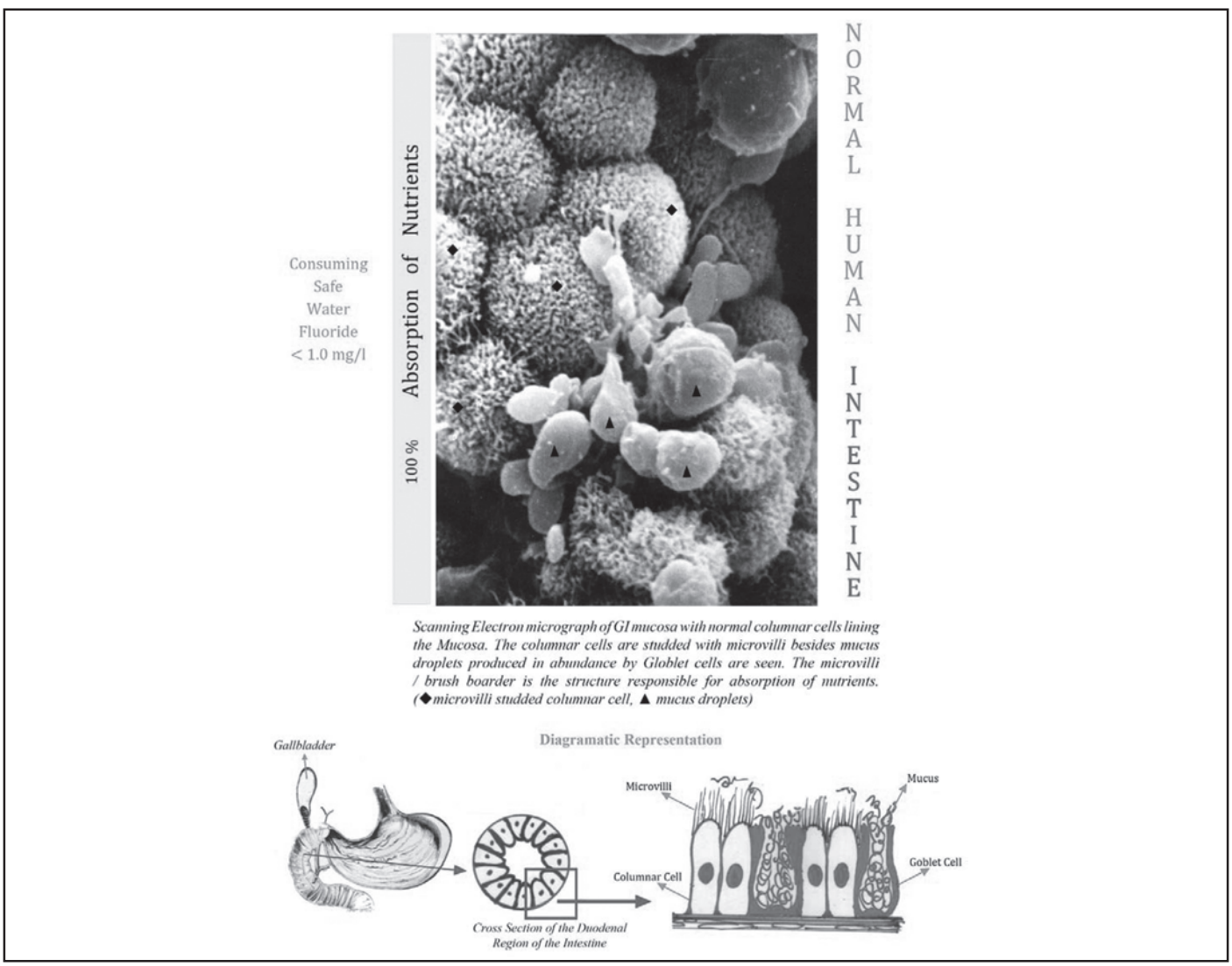


Fig. 3. Normal human intestine

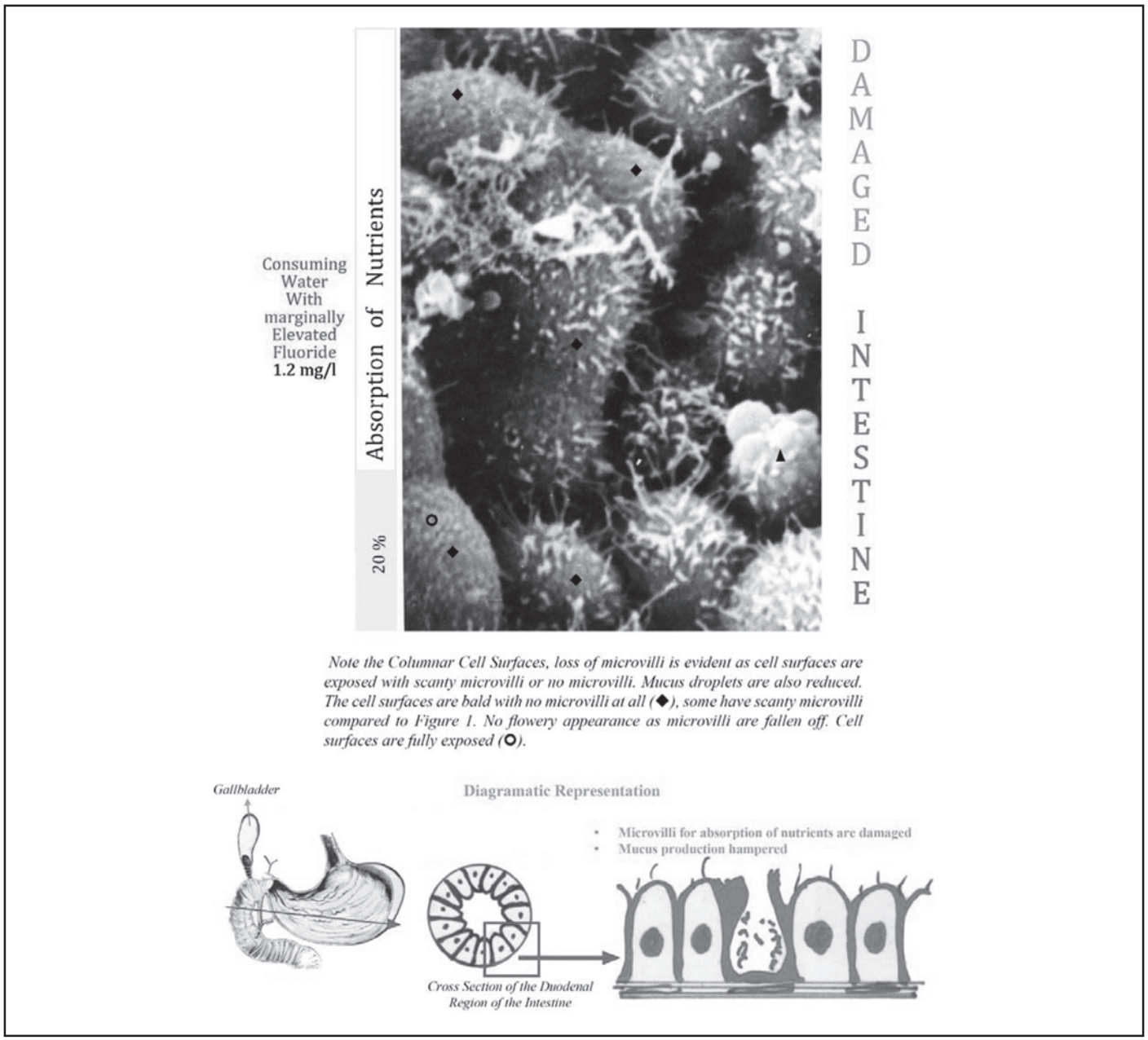


Fig. 4. Damaged intestine

Thirdly, fluoride ingestion deranges the structure and function of the thyroid gland, leading to reduction in thyroid hormone production, resulting in inadequate stimuli on erythropoietic tissues to produce erythrocytes. A high percentage of erythrocytes produced in an environment high in fluoride, are abnormal with crenations known as echinocytes. The echinocytes do not survive the normal RBC life span of 120-130 days but get phagocytosed and eliminated from blood stream.[15] Less number of erythrocytes result in low hemoglobin. The 3 major reasons for low hemoglobin production can be reversed to normal by mere withdrawal of fluoride i.e. diet editing and simultaneous diet counselling for promotion of consumption of nutrients for correction of the damages caused to the system.

There is an urgent need for the doctors handling the antenatal clinics to be sensitized on the possibility of fluorosis as a cause for anemia in pregnant women and the need to test all anemic pregnant women for fluorosis by testing 30 mL of urine collected in plastic bottles for fluoride content . All pregnant women with high urine fluoride > 1.0 mg/L are instructed to bring a sample of their drinking and cooking water in a plastic bottle (30 mL) collected from the source of the water, and the fluoride content in the water is tested. All anemic pregnant women

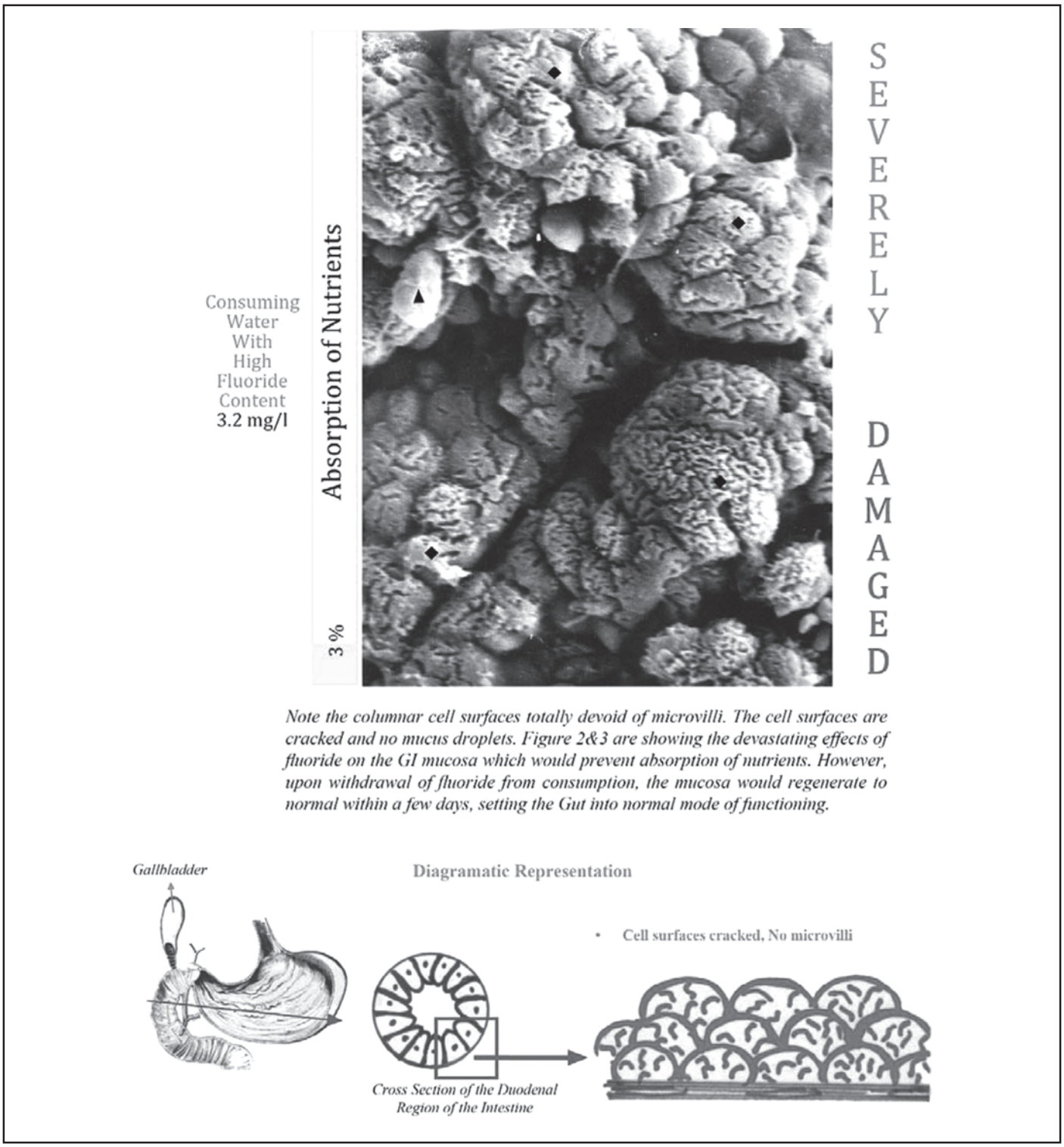


Fig. 5. Severely damaged intestine

with Hb< 12. g/dL; urine F^->1.0 mg/L and drinking water F^- > 1.0 mg/L are to be introduced to diet editing and diet counselling. If the drinking water is contaminated with F^-, they have to shift to an existing safe source of drinking water in the neighbourhood. This is considered as the best option particularly for pregnant women.

Studies have shown that iron and folic acid at the levels currently in use (100 mg iron and 500µg folic acid) are effective in reducing the risk of low birth weight babies, provided there is no increase in fluoride content in the anemic antenatal women. Studies have shown that with monitoring of urinary F^-, appropriate interventions and

dietary modifications Hb level had improved and the frequency of low birth weight babies had come down.[16] There is no short-cut way for increasing hemoglobin in pregnancy and improving birth weight of babies other than promoting non-toxic food and safe water for consumption with iron and folic acid supplementation. For the first time the nation reported fluoride as a high risk factor in production of haemoglobin.[16,17,18] The concept was validated in other anemic individuals as well. A recent review on perinatal health in India[19] emphasises that "good maternal nutrition, prevention and management of anemia and high quality antenatal care will reduce the incidence of complications and there by improve chances of survival of the mother, the foetus and the new born infants". The most essential factor missing in the above review is total elimination of consumption of fluoride by the pregnant women so that the results emerging within a span of 37- 40 weeks from the date of commencement of the programme in ANCs across the country, would be highly rewarding.

The efforts to rectify anemia in school children with withdrawal of fluoride consumption and monitoring of urine fluoride levels followed by adequate intake of nutrients through diet editing and counselling have improved the Hb levels in them and opened out very valuable and sustainable path to follow.[20,21]

Commonalities in fluorosis and iodine deficiency disorders (IDD): The way forward.

Children living in fluoride endemic regions in the country whether in Assam, Andhra Pradesh, Bihar, Gujarat, Punjab, Haryana, Delhi, Rajasthan, Karnataka, Tamil Nadu and/or West Bengal would present with health problems such as bone deformities, short stature/ cretinism, mental retardation / low IQ, the reason may be due to consumption of fluoride in excess (> 1.0 mg/L) or it may be due to deficiency of iodine in diet. The excess fluoride and/or iodine deficiency may commence during intrauterine life when the pregnant women are the victims, and the children born shall reveal the above mentioned derangements (2005).[22]

Therefore when fluoride toxicity / fluorosis is suspected in children, besides testing fluoride in urine, iodine estimation is considered necessary. In a study conducted on children in endemic areas of Delhi state with focus on children with dental fluorosis, the thyroid hormone profile led to the conclusion that testing of drinking water and body fluids for fluoride content along with FT_3 , FT_4 and TSH in children living in endemic areas with and without dental fluorosis is desirable for recognizing thyroid hormone derangements.The primary cause of iodine deficiency disorders (IDD) may not always be due to iodine deficiency as it might be due to fluoride ingestion in excess. Prevention and control of fluorosis and IDD require an integrated approach for diagnosis and patient management.

If children have the deformities due to consumption of excess fluoride, it can be corrected to a large extent by diet editing and diet counselling for withdrawal of fluoride and adequate intake of all nutrients.

Calcium and Vitamin D resistant rickets in children: Protocol for correction

Assessment of serum fluoride and its association with calcium and Vitamin D resistant rickets in infants and children is the most recent revelation.[23] Children living in fluoride endemic areas in the country have overtly visible bone deformities suggestive of rickets. Infants and children are brought to the out patient department with respiratory distress and X-ray radiographs reveal that they suffer from rickets. Certain percentage of infants and children do not respond to mega dose of calcium and vitamin D and they are labelled as vitamin D resistant rickets. Assessing the serum fluoride levels and introducing diet editing and counselling with adequate calcium and vitamin D through dietary sources, as an alternate treatment option for children is recommended, if found resistant to mega-dose of calcium and vitamin D treatment.[24]

In conclusion it will be of immense value and significance both for the treating physician and the patients to consider the following: (i) Sensitize the medical fraternity to fluorosis with associated health problems and introducing the testing for fluorosis for routine patient care services. (ii) Infants and children attending pediatric OPDs with bone deformities, under-nourished with anemia and growth retardation may be considered for testing fluoride in body fluids, drinking water and thyroid hormone profile. With this approach, it may be possible to address the health problems in an effective manner than what has been experienced in the past. (iii) Finally the newly emerged scientific information in addressing anemia in pregnant women, adolescent girls and low birth weight babies due to fluoride toxicity is adding a new chapter in medical history.

Points to Remember

- ***Non-skeletal fluorosis is the earliest manifestation of fluorosis and requires a high index of suspicion for diagnosis.***

- ***Testing fluoride in body fluids and drinking water is necessary for diagnosis and management.***
- ***Fluorosis, IDD and rickets have commonalities in clinical manifestations.***

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GENERAL ARTICLE

MANAGEMENT OF STAPHYLOCOCCAL INFECTIONS-FROM OUTPATIENT DEPARTMENT TO INTENSIVE CARE UNITS

Vijayalakshmi Balakrishnan

Abstract: *Staphylococcal infections are commonly seen both in community acquired and hospital acquired infections. They can present as a simple skin infection as well as a lethal septic shock. An increasing incidence of resistant staphylococcal infections both from the community and in the hospitals is being seen. Infection control and isolation measures are very important to prevent hospital outbreaks.*

Keywords: *Staphylococcus aureus, MRSA, Septic shock, Hand washing.*

"Real knowledge is to know the extent of one's ignorance"

This is a quote by the 5th century Chinese philosopher Confucius. It can be easily applied to the extent of understanding we have towards Staphylococcus aureus (SA), which is probably the one most studied species. Staphylococcus, meaning "A cluster of berries" was identified in 1881 in pus by a surgeon, Sir Alexander Ogston. Later Rosenbach isolated S. aureus and albus in pure cultures. Now we differentiate the virulent Staphylococcus aureus and the less virulent Staphylococci by the catalase and coagulase tests which are characteristically positive in the former. For the sake of ease, we will club the large number of coagulase/ catalase negative Staphylococci as CONS in the ongoing discussion.

The ingenuity of this pathogen lies in the fact that it can remain both as a coloniser establishing a symbiotic relationship and can become a virulent invasive life threatening infection. S. aures can be a community acquired or a hospital acquired infection. It can exert its effects both by local invasion and production of toxins which act distally also. It can be said with conviction that no part of the human body has escaped this pathogen.

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Clinical syndromes

SA infection can present as a systemic septicemic illness or as a focal infection. Table. I illustrates the common focal presentations. Patients with septicemic illness often present with septic shock and multi- organ failure. The onset is usually as a short febrile illness with focal signs and symptoms (cough, painful erythematous lesions in the skin, post-operative site oozing, etc) progressing rapidly to shock and end organ damage. Mortality is extremely high without prompt treatment.

Table I illustrates quite a number of suppurative infections. SA is a very virulent pathogen with a tendency for invasion and tissue destruction. It has evolved several mechanisms to evade host immunity. Certain skin infections like furuncles are pathognomonic of SA. Tropical pyomyositis is a destructive myositis caused by SA.[1] The child usually presents with indolent fever and pain in certain muscle groups like quadriceps or iliopsoas after some vague injury. If there is no prompt initiation of antibiotics, there can be significant destruction of muscle and fascia requiring surgical debridement.

SA can also cause certain invasive syndromes quite distant from the point of infection. These are caused by release of toxins which either diffuse through the tissue, blood stream or activate immune system locally to exert distal effects. Table II describes the common toxin mediated syndromes. Toxic shock syndrome (TSS) is a classic example where the toxin is a superantigen, usually released in the gut, vagina or skin.[2] It activates the T lymphocytes without antigen presentation through the mucosal or skin dentritic cells causing a catastrophic cytokine storm leading to an acute circulatory failure. The primary site of infection may not even be clinically apparent at times. Blood cultures may be negative and toxin typing enables diagnosis. Staphylococcal scalded- skin syndrome (SSSS) is an exfoliative skin disorder usually seen in newborns, often in neonatal units and infants.[3] In the generalised form, there is extensive skin exfoliation through out the body. Mucosa are characteristically not involved, helping us to differentiate from toxic epidermal necrolysis (TEN) syndrome. Blood and skin swab cultures are usually negative. Nasal swabs of the caretakers are often positive for SA. We also have a localised SSSS, where

Table I. Organ specific syndromes with staphylococcus and treatment

System Involved	Clinical condition	Treatment
Nervous system	Meningitis, brain abscess, subdural empyemas, CSF shunt infections, spinal osteomyelitis, spondylodiscitis	High dose cloxacillin or vancomycin Evacuation of pus
Skin/Soft tissue	**Primary pyodermas** Impetigo*, folliculitis, furuncle, carbuncle **Soft tissue infections** Erysipelas/Cellulitis*, necrotising fasciitis*, suppurative lymphadenitis/ lymphangitis*, pyomyositis	Topical agents- 2% mupirocin or fusidic acid ointments Systemic antibiotics occasionally All the drugs Avoid rifampicin monotherapy Drainage of any purulent collection
Bone and Joint	Osteomyelitis, septic arthritis, implant related infections, spinal osteomylitis, spondylodiscitis	All the drugs. Antibiotics are required in high doses for 6- 8 weeks. Removal of implant
Eye/ ENT	Bacterial conjuctivitis/ keratitis, endopthalmitis, otitis externa,Sinusitis**, sialadenitis/parotitis,neck abscesses**, suppurative thyroiditis	Topical moxifloxacin/ sulfacetamide drops. Debridement/ antibiotics All the antibiotics Evacuation of pus
Respiratory tract	Tracheobronchitis, pnuemonia, lung abscess, empyema thoracis	Cloxacillin, cefazolinvancomycin, clindamycin, cotrimoxazole, Drainage of pus
Cardiovascular system	Infective endocarditis Suppurative thrombophlebitis	Cloxacillin, cefazolin, vancomycin Daptomycin for MRSA Right sided endocarditis Rifampicin is added on for prosthetic implant infections
Genito- urinary system	Acute pyelonephritis, pyonephrosis / peri nephric abscesses, tubo ovarian abscesses, endometritis / septic abortions	Cloxacillin, cefazolin, Vancomycin, teicoplanin, co trimoxazole. Drainage of pus
Abdomen	Intra abdominal abscessesCholangitis/ Liver abscess after interventions	All the drugs. Drainage of pus

*Streptococcal pyogenes is the commonest cause

**Amoxicillin- Clavulonate is used more for associated anaerobic cover

there are bullous lesions just around the wound and the wound culture is invariably positive for SA. Here, the exfoliotoxin diffuses around the wound and cleaves the desmoglein in the skin causing blisters.

Staphylococcal pulmonary infections are usually complicated pneumonias presenting in the post influenza scenario or due to toxins like PVL (Table II) from the community. Toxin mediated pneumonias are usually hemorrhagic with a very low blood culture yield and a turbulant course.[4] SA can present as lung abscess or empyema thoracis. SA is also the commonest cause of gram positive hospital acquired pneumonia.

SA is implicated in post- operative wound infection. It also causes infections secondary to invasive procedures like guided biopsies, cyst drainage, etc. Staphylococci have a predilection for foreign bodies. It is a major cause of intravenous cathether related infections. Both SA and CONS cause implant related infections in orthopedics and

Table II. Toxin mediated SA syndromes

Toxin mediated Syndrome	Toxin released	Clinical presentation	Management	Important features
Staphylococcal scalded-skin syndrome (SSSS)	Exfoliotoxin A, B	Generalised- fever, lethargy, generalised skin scalding Localised -Blisters around wound	Supportive care and clindamycincloxacillin or cefazolin	Mortality <5% in children, >50% in adults
Toxic shock syndrome (TSS)	TSST1, Enterotoxin B,C	Fever, diffuse morbilliform rash, anasarca, hypotension. Evolves to MODS	Supportive care clindamycincloxacillin/ cefazolin	Superantigen, anergy towards toxin, high mortality
Food poisoning	Enterotoxins	2- 6 hrs after food intake, nausea, vomiting, abdominal pain	Supportive care,	Pre-formed toxin
Necrotising pulmonary and skin/ soft tissue infection	Panton Valentine Leukocidin (PVL) toxin	Hemorraghic pneumonia, progressive furunculosis or fascitis	Supportive care vancomycin or linezolid, clindamycin	Community acquired MRSA

cardiology. They form a layer on the surface of the foreign body with a polysaccharide proteineceous material, platelets and fibrin called 'Biofilm'.[5] Once a biofilm is formed, it is extremely difficult to eradicate the bug even with prolonged antibiotics and removal of the implant becomes the only option.

SA causes osteomyelitis and septic arthritis in children often resulting from an obscure skin infection. The presentation is usually subacute with low grade fever, local pain and swelling. Blood cultures may be negative and tissue cultures are needed for diagnosis. If inadequately treated, chronic osteomyelitis is the inevitable sequelae.

Management of staphylococcal diseases

Specific diagnosis is mandatory in any disease management and cultures enable the same. If there is a definite uncomplicated focal source like a furuncle, localised cellulitis, etc, empirical treatment is cost effective. However, if the patient comes with severe sepsis or has risk factors for resistantce, always ensure that cultures are taken before antibiotics. If there is pus anywhere (soft tissue abscess, liver abscess, etc) drainage is mandatory. Antibiotics do not penetrate pus filled cavities and therapeutic drainage is the primary treatment. Gram stain of pus shows gram positive cocci in clusters. Blood culture is mandatory in any sick patient. The oxacillin sensitivity of the staphylococcal growth helps us to choose the antibiotic.

Oxacillin resistant SA is called MRSA and is generally resistant to cloxacillin, cephalosporins, beta- latam/ lactamase inhibitors, carbapenems. They should be treated with vancomycin, teicoplanin, linezolid, co- trimoxazole, daptomycin or clindamycin. Table.III summarises all the anti- staphylococcal antibiotics. Oxacillin sensitive strains are called MSSA and are sensitive to cloxacillin and cefazolin. Amoxicillin - clavulanate, most of the third generation cephalosporins, betalactam lactamase inhibitors, carbapenems have reasonable activity against MSSA. Hence, separate antibiotic need not be added in non septicemic mixed infections (intra- abdominal collections). However, if there is a pure culture growth of MSSA in blood, neurologic or cardiac infections, always give cloxacillin or cefazolin as there is enough evidence now that first line agents are much superior.[6]

CONS are adapted to colonise in human body and are usually contaminants when drawing blood cultures or collecting urine in a wailing child. However, they are implicated in subacute endocarditis, foreign body related infections and central line related infections. They are considered methicillin resistant till the sensitivity proves otherwise.

Almost all of the community acquired and a significant amount of post- operative wound infections in our country are caused by MSSA. Gram negative organisms are implicated more in hospital acquired

Table III. Dosage of antibiotics and major side effects

Drug	Dose	Side effects
Cloxacillin	50-200 mg/kg in 3-4 divided doses	Skin rash, local thrombophlebitis
Cefazolin	25-100 mg/kg in 3-4 divided doses	Skin rash, diarrhoea
Clindamycin	15-25 mg/kg 6 to 8 hourly	Diarrhoea, clostridium difficle colitis
Trimethoprim/ sulphamethoxazole	Trimethoprim 8-15 mg/kg in 3-4 divided doses	Skin reaction, myelosuppresion, hyperkalemic renal failure
Linezolid	10 mg/kg every 8 hourly	Diarrhoea, vomiting, headache,anemia, thrombocytopenia
Teicoplanin	10 mg/kg BD 3 doses followed by every 24 hours	Skin rash
Vancomycin	10-20 mg/kg every 6 to 8 hourly	Nephrotoxicity, anaphylactoid reaction, ototoxicity
Daptomycin	4-6 mg/kg OD	Myopathy, peripheral neuropathy
Ciprofloxacin	10-20 mg/ kg twice a day	Cartilage destruction
Rifampicin	10 mgs/ kg	Abdominal cramps, skin rashhepatotoxicity

infections in India.[7] There have been reports of increasing rates of MRSA infections in the recent literature. A survey across 15 tertiary care hospitals in India from 2008- 2009 showed 41% MRSA among 26310 SA isolates.[8] MRSA infections occur as clusters in hospitals, especially in post-operative patients. They also occur as health-care associated infections in diabetics, children on immunosuppressants, transplant recipients, etc. Occasionally, we do see community acquired MRSA infections in children with skin and soft tissue infections. A survey conducted in slums in North India in 2005 showed SA nasal colonisation was 52.3% and MRSA rates were 3.89% in school children between 5- 15 years.[9]

The empirical choice of anti- staphylococcal antibiotic in hospital acquired infections depends upon the local incidence of MRSA. A rough guide is, if more than 10% of the gram positive infections are caused by MRSA, empirical treatment should include MRSA cover. There is no drug to curb the staphylococcal toxins once released. However, the toxin release can be inhibited by certain antibiotics like clindamycin, linezolid and daptomycin.

Apart from drugs listed in Table III several other agents used are extensively in western world like quinupristin- dalfopristin, telavacin, fusidic acid. Rifampicin is used as add on therapy for foreign body related SA infections, especially in prosthetic valve endocarditis and orthopedic implant infections. This is because of the ability of rifampicin to penetrate the biofilm but if it is used as a single agent, resistance develops immediately to it. There are several topical agents used for skin/ soft tissue infections. 2% Mupirocin and 2% Fusidic acid ointments are used for impetigo, furunculosis and burns wound infection, post- operative wound infections etc.

Appropriate choice of anti SA agent

Table.III may give the appearance of abundance in our arsenal against SA. On the contrary in real life scenario we actually do not get many options. In septic shock, we need a parenteral agent which is bactericidal, gets concentrated in blood immediately with the first dose, persists in adequate concentration despite acidosis and hypotension, and has adequate penetration into all possible sources. Table IV gives the issues while choosing an antibiotic in septic shock. It is prudent to start on Gram negative bacterial cover also when the patient is in shock and the source is unclear. Deescalate with the appropriate emerging clinical picture and results of gram stain or cultures. The loading dose of the antibiotic need not be

Table IV. Issues faced with parenteral antibiotics in severe sepsis

Antibiotic	Issues faced
Cloxacillin	Thrombophlebitis
Cefazolin	Dose adjustment needed in renal failureDoes not penetrate the blood brain barrier
Vancomycin	Nephrotoxic. Dose adjustment and trough level monitoring mandatory in renal failure
Clindamycin	Does not get concentrated in bloodDoes not penetrate blood brain barrier
Teicoplanin	Extensively protein bound, takes a long time to maintain bactericidal concentration in bloodDoes not cross blood brain barrier
Linezolid	Bacteriostatic agent
Daptomycin	Inactivated by surfactant, cannot be used in pneumonia

adjusted for creatinine clearance. After the initial resuscitation further antibiotics and their doses depend on the source of sepsis, hypotension, renal and liver injury and the drug interactions with the other essential drugs. Table.I gives the antibiotics to be used in specific syndromes based on pharmacokinetics.

Reiterating the point again, in any purulent infection, pus drainage is mandatory and antibiotics are only adjuvants. If it is a localised skin abscess, isolated drainage would do. Systemic antibiotics are added in purulent infections of viscera or body cavity after evacuation of pus for 48 hrs to 2 weeks depending on the drainage possible. In bone and joint infections, SA often becomes relatively dormant (becomes a small colony variant or forms a biofilm) and hence prolonged course of antibiotics, anywhere between 6 to 12 weeks is needed. If an implant becomes infected, removal of implant is followed by antibiotics.[10] CONS are also important causes of implant related infection and treatment modalities are essentially the same.

Both SA and CONS can cause infective endocarditis (IE). CONS cause a subacute IE while SA is associated with an acute fulminant illness with high mortality. Most of the SA endocarditis occurs in the post-operative setting and often re- exploration and removal of prosthetic valve along with high dose parenteral drugs are needed.[10] Right sided endocarditis with MRSA and CONS is often seen in intra- venous drug abusers. Treatment is antibiotics with or without surgery for 4 to 6 weeks. Intravenous catheter related infections are often caused by SA.[11] Removal of line followed by antibiotic for 1-2 weeks is the treatment of choice.

Staphylococcal pneumonias are usually treated with cloxacillin or cefazolin when MSSA and vancomycin or linezolid when MRSA is suspected.[12] Clindamycin is used in necrotising pneumonias. Drainage of the pus through needle, tube or open- thoracostomy is mandatory for empyema thoracis.

Antibiotic prophylaxis

Peri-surgical prophylaxis covering SA is given to decrease post- operative wound infection. Cefazolin/ Cloxacillin or cefuroxime is the usual antibiotic of choice. When the MRSA incidence is high, some hospitals use vancomycin or teicoplanin as prophylaxis though there is not enough literature to support it.[13] Peri-surgical prophylaxis is a single dose at incision followed by an added dose if the surgery extends for over 6 hours. Antibiotics are continued for 48 hours in most cardio thoracic and neurosurgical centres though there is not much evidence either way.[14] Continuing antibiotics beyond 48 hours increases the rate of resistant bugs in the hospital.

Infection control

As described earlier SA colonises in the skin and nasopharygeal mucosa and is usually spread by touch.[15] Since colonisation precedes infection, decolonisation should theoretically decrease the rate of infection. Topical chlorhexidine body washes and intranasal 2% mupirocin ointment are given for a week to decolonise MRSA.[16] Isolation in a single room with adequate contact precautions including aprons, gloves and handwashing significantly prevents transmission[17] Hand washing remains the single most important means to prevent transmission.[18]

Conclusion

Staphylococcal infections have a wide spectrum. Management of the same includes identification of the species with drug sensitivity, starting an appropriate antibiotic at the earliest, source reduction and adjusting the antibiotics with the pharmacokinetics and dynamics along with prompt resuscitation of the patient. Good infection control practices, hand washing, contact screening, isolation and decolonization will help in preventing MRSA transmission significantly.

Points to Remember

- ***Staphylococcus aureus gets colonised in skin and nasopharyngeal mucosa and is spreads by touching.***
- ***Staphylococcus aureus can produce toxins which can exert its effects quite distant from the foci of infection.***
- ***Methicillin resistant Staphylococcus aureus rates are increasing in the hospital.***
- ***Prompt initiation of antibiotics and draining of pus are needed to treat infections.***
- ***Hospital cross transmission can be prevented by adequate hand washing and isolation of infected patients.***

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DRUG PROFILE

ANTI-MALARIALS

Jeeson C Unni

Abstract: *Treatment options for malaria, especially falciparum malaria, is continuously changing due to the rapid development of resistance to individual drugs given as monotherapy. Artemisinin-based combination therapies (ACTs) are presently considered the drug of choice for uncomplicated falciparum malaria and though choloquine is still the standard therapy for chloroquine sensitive vivax malaria, ACTs are increasingly being considered for the treatment of non-falciparum malaria. Artemisinin resistance is also being reported of late and much research is necessary to develop novel drugs and drug combinations to work around these emerging scenarios so as to achieve and maintain malaria control with the ultimate aim of malaria elimination.*

Keywords: *Malaria, Treatment, Artemisinin-based combination therapies (ACTs), Chloroquine.*

Malaria is a major public health problem of developing countries. India had 8.7 lakh malaria infections in 2013 but is on track to reduce cases by 50-75% to between 4.35 and 2.2 lakh by 2015 according to the World Malaria Report 2014.[1] This is deemed possible with large-scale use of WHO-recommended strategies, currently available diagnostic tools, strong national commitment and coordinated efforts with partners. We pediatricians need to be aware of issues involved in the move toward elimination of malaria. This article on anti-malarials is an attempt in that direction.

Status of drug resistance in India

Chloroquine (CQ) resistant P. falciparum malaria was first reported in 1973 from Assam[2]; followed soon after from other states.[3,4,5] By 2004, the National Vector Borne Disease Control Program suggested use of sulphadoxine - pyrimethamine (SP) due to high treatment failure to CQ in 44 districts of 18 states in India.[6] Resistance to SP combination at various levels has also been reported in the districts of seven North Eastern States. It has been seen that the introduction of a single new drug leads to rapid development of resistance. To overcome this, WHO has recommended Artemisinin based combination therapy (ACT) for the treatment of uncomplicated falciparum malaria.[5] Though there are reports of emerging choroquine resistant P. vivax[7], in India the drug is still effective for treating P. vivax malaria.[8]

Rationale for combination therapy

It was recognised that the rapid onset of resistance to an antimalarial occurred when it is used as a monotherapy. Combination therapy entails the simultaneous administration of two or more schizontocidal drugs with independent mode of action. Rapid clearance of parasitemia, high killing rate (decreases parasite load by 10,000 fold per cycle as against others which reduce at 100 to 1000 fold) and resolution of symptoms; rapid drug elimination preventing drug residue from providing a selective filter for resistant parasites; lack of serious adverse effects and absence of significant resistance till date make artemisinin and its derivatives an essential component of such combination therapy; hence known as artemisinin based combination therapy (ACT). As a result of combination therapy the artemisinin component is protected from resistance by the partner medicine provided is efficacious and partner medicine is in turn protected by the artemisinin derivative. Artemisinin also has the advantage of reducing gametocyte carriage and thus transmission of malaria which is particularly important in malaria control. Residual parasites are taken care by other drug in combination.

ACTs and other combination therapies being studied

If artemisinin are combined with other rapidly eliminated antimalarials like tetracycline or clindamycin a 7 days course of treatment is required. This long course invariably results in poor adherence. But when combined with slowly eliminated antimalarials like SP, mefloquine (MQ) or lumifantrine shorter courses of treatment (3 days) will be effective and also ensure adherence.

The recommended dose of artemether-lumefantrine provides reliable efficacy in most patients with

* IAP Drug Formulary
Cochin

uncomplicated malaria. However, therapeutic efficacy was lowest in young children from Asia and young underweight a higher dose regimen should be assessed in these groups children from Africa.[9]

Artemisinin-naphthoquine is a new combination developed in China, which is being marketed as a one-day treatment. Although shorter treatment courses may improve adherence, the WHO recommends at least three days of the short-acting artemisinin component to eliminate 90% P. falciparum parasites in the bloodstream, before leaving the longer-acting partner drug to clear the remaining parasites.[10] Future trials should be adequately powered to demonstrate non-inferiority.

ACT is at least equivalent to chloroquine in effectively treating non-falciparum malaria. These findings may facilitate development of simplified protocols for treating all forms of malaria with ACT.[11] Although there is a lack of direct evidence comparing artemether with artesunate, artemether is probably less effective than artesunate in preventing deaths from severe malaria. In circumstances where artesunate is not available, artemether is an alternative to quinine.[12] Rapid Plasmodium falciparum clearance is achieved with artesunate-amodiaquine (ASAQ) combination.[13] Clindamycin plus quinine is an alternative non-artemisinin-based combination recommended by World Health Organization to reduce cost of therapy but studies on efficacy of this combination is inconclusive.[14]

Treatment of severe malaria

The evidence clearly supports the superiority of parenteral artesunate over quinine for the treatment of severe malaria in both adults and children and in different regions of the world.[15]

Cost effectiveness of treatment and chemo-prophylaxis[16]

In areas with both Plasmodium falciparum and Plasmodium vivax transmission, artemether-lumefantrine and dihydroartemisinin-piperaquine, respectively, are currently the most cost-effective treatment options. Treatment of severe malaria with artesunate is more cost effective compared with treatment with quinine. For patients who live more than 6 hours away from an appropriate healthcare facility, pre-referral treatment proved to be more cost-effective compared with no pre-referral intervention. Cost-effectiveness of intermittent preventive treatment in pregnant women (IPTp) was dependent an clinical attendance. IPT in infants with sulphadoxine-pyrimethamine (SP) is cost effective in sites with high malaria transmission. IPT in children with artesunate (AS + SP), amodiaquine (AQ) + SPQ or SP alone is a cost effective and safe intervention for reducing the burden of malaria in children in areas with markedly seasonal malaria transmission. Although there is a need for it, little is known about the cost-effectiveness of current approaches to malaria therapy in nonendemic countries and the cost-effectiveness of antimalarial chemoprophylaxis.

The only significant adverse effect from clinical trials with artemisinin is rare type I hypersensitivity reaction manifested by urticaria. The following ACTs are currently, available in our country

The essential anti-malarial drugs for children

Essential medicines list for children and adolescents in India created by Indian Academy of Pediatrics in collaboration with WHO mentions the following anti-malarials[17]

Antimalarial medicines

a) For curative treatment: Amodiaquine - may be used alone or in combination with artesunate 50 mg; Artemether (severe malaria); Artesunate in combination with either amodiaquine, mefloquine or sulfadoxine + pyrimethamine; Chloroquine; Mefloquine; Primaquine; Quinine - severe malaria, in combination with doxycycline: Doxycycline

b) For prophylaxis: Chloroquine; Doxycycline; Mefloquine; Proguanil.

The guidelines were arrived upon considering the likely susceptibility of the infecting parasites and the pharmacokinetic and pharmacodynamic properties of the drugs. The gametocytocidal properties of the drug, which help reduce transmission, were also taken into consideration. In this provisional list, it is emphasized that medicines for the treatment of P. falciparum malaria cases should be used in combination. All fixed drug combinations are not yet licensed for children and studies to develop formulations and dosage forms appropriate for children are warranted.

The IAP recommended treatment of choloroquine sensitive malaria is given in Table I.[18] Chloroquine should not be given in empty stomach and when the child has high fever. Bring down the temperature first. If vomiting occurs within 45 minutes of a dose of chloroquine that particular dose is to be repeated after taking care of vomiting by giving domperidone or ondansetron. If the

Table I. Treatment of chloroquine-sensitive malaria

Malaria	Recommended treatment
P. vivax	Chloroquine 10 mg base/kg stat followed by 5mg/kg at 6, 24 and 48 hours. Or Chloroquine 10mg base/kg stat followed by 10mg/kg at 24 hours and 5mg/kg at 48 hours (total dose 25mg base/kg). For prevention of relapse:** Vivax malaria, Primaquine :0.25 mg/kg OD x 14 days.** Falciparum malaria, a single dose of Primaquine (0.75mg/kg) for gameto-cytocidal action.

second dose is vomited, treat as severe complicated malaria. All children, except infants, with P vivax malaria must to given follow up primaquine for 14 days after G6-PD deficiency screening. For borderline G6PD deficiency, primaquine may be given weekly at a dose of 0.6-0.8 mg/kg weekly for 6 weeks.

The age wise dosage schedule for chloroquine-sensitive P. vivax as per guidelines for diagnosis and treatment of malaria in India 2014 of the National Institute of Malaria Research and National Vector Borne Disease Control Programme is given in Table II & III.[19]

Treatment of falciparum malaria[18]

Only artemisinin based combination therapy may be initiated for falciparum malaria as recommended by WHO. The regimens that are effective include artesunate 4mg/kg once daily x 3 days and a single administration of SP as 25mg/kg of sulfadoxine and 1.25 mg/kg of pyrimethamine on day 1;

OR Artesunate as above and Mefloquine 25mg/kg in two (15 + 10) divided doses on day 2 and day 3;

OR combination Tab (Artemether 20 mg + Lumefantrine 120mg) at a dose of 1 BD x 3 days (six dose regimen) [5-14 kg 1 tab stat, 1 Tab again after 8 hours and then 1BD x 2 days; 15 to 24 kg, 25-35 kg, >35 kg may use the same schedule with 2, 3 and 4 tabs, respectively].

The advantages of using lumefantrine in the combination are that it is a new drug, its absorption increases in presence of milk and it is only marketed in combination with artemisinin.

Table II. Chloroquine for P. vivax

Age (years)	Number of tablets		
	Day 1 (10 mg/kg)	Day 2 (10 mg/kg)	Day 3 (5 mg/kg)
<1	1/2	1/2	1/4
1-4	1	1	1/2
5-8	2	2	1
9-14	3	3	1 1/2
>15	4	4	2

Table III. Primaquine for P. vivax (Daily dosage for 14 days)

Age (years)	Daily dosage (in mg base)
<1	Nil
1-4	2.5
5-8	5
9-14	10
>15	15
The strength of the tablet available is 2.5, 7.5 and 15 mg. Number of tablets should be given accordingly	

The National Institute of Malaria Research and National Vector Borne Disease Control Programme recommends artesunate (4 mg/kg body weight) daily for 3 days and sulfadoxine (25 mg/kg body weight) - pyrimethamine (1.25 mg/kg body weight) [AS+SP] on Day 0 for treatment of P falciparum in all parts of India except in the northeastern states. The dosage schedule of AS+SP for different age groups is given in Table IV.

In the Northeastern states (Arunachal Pradesh, Asom, Manipur, Meghalaya, Mizoram, Nagaland and Tripura), due to the recent reports of late treatment failures to the current combination of AS+SP in P. falciparum malaria, the presently recommended ACT in national drug policy is fixed dose combination (FDC) of Artemether-lumefantrine (AL). The dosage schedule of AL for different age groups is given in Table V.

The other fixed dose combinations registered for marketing in India are artesunate-amodiaquine, artesunate-mefloquine and arterolane-piperaquine (for adults only) and can be used for treatment of uncomplicated P. falciparum or mixed infections.

Table IV. Dosage schedule of AS+SP and PQ for P. falciparum malaria

Age group (years)	1st day		2nd day		3rd day
	AS	SP	AS	PQ	AS
0–1	1 (25 mg)	1 (250+12.5mg)	1 (25 mg)	Nil	1 (25 mg)
1–4	1 (50 mg)	1 (500+25 mg)	1 (50 mg)	1 (7.5 mgbase each)	1 (50 mg)
5–8	1 (100 mg)	1 (750+37.5 mg)	1 (100 mg)	2 (7.5 mgbase each)	1 (100 mg)
9–14	1 (150 mg)	2 (500+25 mgeach)	1 (150 mg)	4 (7.5 mgbase each)	1 (150 mg)
15 & above	1 (200 mg)	2 (750+37.5 mgeach)	1 (200 mg)	6 (7.5 mgbase each)	1 (200 mg)
Figures in parentheses indicate doses and outside the parentheses number of tablets					

Table V. Dosage schedule of AL in northeastern states

Co-formulated tablet AL	Total dose of AL (twice daily for 3 days)	No. of tablets in the packing	Administration (twice daily for3 days) tablets
5–14kg (>5/12 - <3yr)	20 mg/120 mg	6	1
15–24 kg (>3 to <9yr)	40 mg/240 mg	12	2
25–34 kg (>9 - <14yr)	60 mg/360 mg	18	3
>34kg (14yrs & more)	80 mg/480 mg	24	4
Not recommended during the first trimester of pregnancy and for children weighing < 5kg			

Treatment of mixed infections

Mixed infections with P. falciparum should be treated as falciparum malaria. Since AS+SP is not effective in vivax malaria, other ACT should be used. However, anti-relapse treatment with primaquine can be given for 14 days, if indicated.

Treatment of severe malaria

Severe malaria is a medical emergency. The child is extremely sick and aggressive pharmacological and supportive therapy needs to be urgently instituted to prevent mortality or at least reduce probability of death to 15-20%; mortality is 100% if untreated. All other issues in malaria therapy, therefore, are secondary. IV infusions of anti-malarials must be administered urgently to immediately achieve therapeutic serum concentrations of the drug. IV quinine or artemisinin derivatives to be given irrespective of chloroquine resistance status and irrespective of the infecting species.[19]

Artesunate: 2.4 mg/kg I.V. or I.M. given on admission (time=0), then at 12 hours and 24 hours, then once a day (Care should be taken to dilute artesunate powder in 5% Sodium bi-carbonate provided in the pack).

Quinine: 20 mg quinine salt/kg on admission (i.v. infusion in 5% dextrose/dextrose saline over a period of 4 hours) followed by maintenance dose of 10 mg/kg 8 hourly; infusion rate should not exceed 5 mg/kg per hour. Loading dose of 20 mg/kg should not be given, if the patient has already received quinine. NEVER GIVE BOLUS INJECTION OF QUININE. If parenteral quinine therapy needs to be continued beyond 48 hours, dose should be reduced to 7 mg/kg 8 hourly.

Artemether: 3.2 mg/kg i.m. given on admission then 1.6 mg/kg per day. Alpha, beta-arteether: 150 mg daily I.M. for 3 days in adults only (not recommended for children).

IV preferred over IM preparations. Parenteral treatment should be given for minimum of 24 hours once started. After clinical improvement, full course of ACT is to be given even if initially treated with IV quinine. Severe P vivax malaria must be given

primaquine for 14 days as per guidelines when and if primaquine can be tolerated.

Treatment of multidrug resistant P. falciparum (resistant to both chloroquine and sulfadoxine-pyrimethamine).[18]

Such cases may be treated with alternative ACT or Quinine with Tetracycline or Doxycycline or Clindamycin for 7 days. Doxycycline is contraindicated in pregnancy, lactation and in children up to 8 years. Treatment failure with chloroquine in P. vivax malaria is rare in India.

Quinine, 10mg salt/kg/dose 3 times daily for 7 days.

+

Tetracycline (above 8 years) 4mg/kg/dose 4 times daily for 7 days

OR

Doxycycline (above 8 years) 3.5mg/kg once a day for 7 days

OR

Clindamycin 20mg/kg/day in 2 divided doses for 7 days.

In case of cinchonism,
Quinine, 10mg salt/kg/dose 3 times daily for 3-5 days

+

Tetracycline (above 8 years) 4mg/kg/dose 4 times daily for 7 days

OR

Doxycycline (above 8 years) 3.5mg/kg once a day for 7 days

OR

Clindamycin 20mg/kg/day in 2 divided doses for 7 days.

A single dose of primaquine above 1 year age (0.75mg/kg) is given for gametocytocidal action.

OR

Artemether lumefantrine combination

Chemoprophylaxis[19]

Chemoprophylaxis is recommended for travellers, migrant labourers and military personnel exposed to malaria in highly endemic areas. Use of personal protection measures like insecticide-treated bed nets should be encouraged for pregnant women and other vulnerable populations.

Short-term chemoprophylaxis (less than 6 weeks) - Doxycycline: 100 mg daily in adults and 1.5 mg/kg body weight for children more than 8 years old. The drug should be started 2 days before travel and continued for 4 weeks after leaving the malarious area. Doxycycline is contraindicated in pregnant lactating women and children less than 8 years.

Long-term chemoprophylaxis (more than 6 weeks) - Mefloquine: 5 mg/kg body weight (up to 250 mg) weekly and should be administered two weeks before, during and four weeks after leaving the area. Mefloquine is contraindicated in cases with history of convulsions, neuropsychiatric problems and cardiac conditions

Conclusion

The article highlights the evolving scenario of drug therapy of malaria with emphasis on the issues pertaining to our country.

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CLIPPINGS

Tdap Vaccine Effectiveness in Adolescents During the 2012 Washington State Pertussis Epidemic.

Cellular pertussis vaccines replaced whole-cell vaccines for the 5-dose childhood vaccination series in 1997 in US. A sixth dose of pertussis-containing vaccine, tetanus toxoid, reduced diphtheria toxoid, and acellular pertussis, adsorbed (Tdap), was recommended in 2005 for adolescents and adults. Studies examining Tdap vaccine effectiveness (VE) among adolescents who have received all acellular vaccines are limited.

A matched case-control study to assess Tdap VE and duration of protection during the 2012 pertussis epidemic in Washington among adolescents born during 1993–2000 was done. All pertussis cases reported from January 1 through June 30, 2012, in 7 counties were included; 3 controls were matched by primary provider clinic and birth year to each case. Vaccination histories were obtained through medical records, the state immunization registry and parent interviews. Participants were classified by type of pertussis vaccine received on the basis of birth year: a mix of whole-cell and acellular vaccines (1993–1997) or all a cellular vaccines (1998–2000). It was found among adolescents who received all acellular vaccines (450 cases, 1246 controls), overall Tdap VE was 63.9% (95% confidence interval [CI]: 50% to 74%). VE within 1 year of vaccination was 73% (95% CI: 60% to 82%). At 2 to 4 years postvaccination, VE declined to 34% (95% CI: "0.03% to 58%).

Tdap protection wanes within 2 to 4 years. Lack of long-term protection after vaccination is likely contributing to increases in pertussis among adolescents.

Acosta AM, DeBolt C, Tasslimi A, Lewis M, Stewart LK, Misegades LK, Messonnier NE, Clark TA, Martin SW, Patel M. Tdap Vaccine Effectiveness in Adolescents During the 2012 Washington State Pertussis Epidemic. Pediatrics 2015; 135:6 1130-1132.

DERMATOLOGY

CHILDHOOD PSYCHOCUTANEOUS DISORDERS - AN OVERVIEW

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Abstract: *The prevalence of psychosomatic disorders among children with dermatological problems is high but frequently unreported because of difficulties in diagnosing and treating this patient group. Psychiatric and psychological factors may play different roles in the pathogenic mechanism of some skin diseases. The mainstay of diagnosis and treatment is the differentiation between skin disorders associated with psychiatric illness and those of a purely psychiatric nature. Dermatologists and Psychiatrists should be aware of this pathology and work together as a team to resolve difficult cases, especially in children. This article highlights the psychocutaneous diseases most frequently seen in pediatric population.*

Keywords: *Psychocutanious disorders, Factitial dermatitis, Psychodermatology.*

Skin lesions in children with psychologic problems on psychiatric condition "call for attention" and highlight an auto aggressive behaviour induced by depression, anxiety and compulsive disorders. Moreover, the patient denies having produced the lesions and family members, particularly parents, may seek multiple medical consultations before accepting the diagnosis of dermatitis artefacta and the need for psychiatric assessment. Team work between dermatology and psychiatry is the most important step in long and very difficult road of treatment for psychocutaneous disorders in children. There are no national data regarding the occurrence of these disorders in children; this is a report made by dermatologists based on clinical information. This paper seeks to draw attention to the distinctive aspects of skin lesions induced by psychiatric disorders and to emphasize the importance of an interdisciplinary approach. With close collaboration between dermatologists, psychiatrists and pediatricians, a national or international report could be done in the future.

Current knowledge about the multiple and various facets of psychodermatology is of significant importance. Clarification of psychocutaneous disorders is given in Box 1.[1,2]

Box.1. Clarification of psychocutaneous disorders

I. Dermatoses of primary psychiatric disorders

- Dermatitis artefacta
- Dermatitis para artefacta: Skin picking syndrome (epidermotillomania, neurotic excoriations); acne excoriata; pseudo knuckle pads; morsicatio buccarum; factitiouscheilitis; onychophagia; onychotillomania, onychotemnomania; trichotillomania, trichotemnomania, trichoteiromania
- Delusions of parasitosis
- Somatoform disorders (glossodynia)
- Body dysmorphic disorders (dysmorphophobia).

II. Psychosomatic dermatoses (dermatoses with a multifactorial basis and a psychiatric component)

- Psoriasis
- Atopic dermatitis
- Acne excoriate
- Chronic forms of urticaria
- Lichen simplex chronicus
- Hyperhidrosis.

III. Psychosomatic diseases

These are secondary psychiatric disorders due to disfiguring dermatoses and are associated with depression, anxiety and delusional symptoms.

Some of the above skin lesions are frequently encounted in pediatric dermatology practice are more frequently diagnosed. Clinicians caring for children should

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be able to recognize the following common factitial dermatoses and know when to ask for psychiatric intervention.

Dermatitis artefacta

Dermatitis artefacta (also known as factitial dermatitis or pathomimia) is characterized by self-inflicted skin lesions that raise suspicion because of their curious morphology, location, lack of therapeutic response and long evolution. The highest prevalence of dermatitis artefacta is between adolescence and early adulthood[3] and the condition is seldom reported in small children. It is very important to differentiate dermatitis artefacta, where self-inflicted injuries are unconscious, from:

I. Dermatitis para artefacta, with semiconscious/ admitted self-induced lesions.

II. malingering with induced and simulated disorders for the purpose of material gain.

III. Special forms, such as Gardner Diamond syndrome, Munchausen syndrome and Munchausen-by-proxy syndrome.[4]

Dermatitis para artefacta (Skin picking syndrome)

This entity has been described in the medical literature under various synonyms, i.e., epidermotillomania, neurotic excoriations, emotional excoriations, nervous scratching artefact, dermatillomania and para-artificial excoriations.

Acne excoriata

Acne excoriata is considered by several authors to be the same entity as skin picking syndrome,[2] although there is ongoing discussion with regards to the identification of these entities.

Neurotic excoriations

Neurotic excoriations are seen in patients with impaired impulse control and more frequently observed in females, although cases in children have been reported. The skin lesions result from self-injury in order to reduce emotional tension. The prevalence is not known, although it is estimated that 2% of dermatology patients suffer from this disease.[5] The clinical picture is quite characteristic, with excoriations and superficial erosions, sometimes covered by crusts, scars and hyperpigmentation, and most often produced by a sharp instrument. Cutaneous lesions result from the intense impulse of harming the skin. Psychiatric examination and treatment is mandatory because neurotic excoriations can be the first sign of an extensive list of psychiatric disorders, including depression, anxiety, body obsessive-compulsive disorder, body dysmorphic disorder, borderline personality disorder and hypochondriasis.[6] It is very important for the child that family members accept the psychiatric nature of this skin disease and undertake psychiatric treatment.

Trichotillomania, trichotemnomania, trichoteiromania

From the dermatology point of view, trichotillomania is a nonscarring alopecia induced by repetitive self-pulling of the hair. On clinical examination, different hair lengths are noticed, with broken hairs, new growth, no desquamation and a negative pull test. Psychiatric criteria include existence of nervousness associated with pulling the hair and relief afterward as a result of reducing tension, depression and anger. Trichotillomania should be differentiated from obsessive-compulsive disorder where pulling the hair is an obsessive impulse. It commonly affects the scalp, but other hairy regions can be affected, including the eyebrows, eyelashes and even the pubic hair. In small children, trichotillomania is mainly diagnosed on the scalp.[5] In trichotemnomania, the hair is intentionally cut off and so the disorder is part of a malingering syndrome. Trichoteiromania is characterized by breaking the hairs by rubbing or scratching the scalp in a repetitive way.

Pseudoknuckle pads

Pseudoknuckle pads are hypertrophic lesions localized to the dorsal aspects of the finger joints, more often unilaterally, caused by habitual chewing, rubbing, or sucking the fingers and is the expression of a psychiatric disorder, e.g., mental retardation, bulimia nervosa (self-induced vomiting in girls), or obsessive behaviour.

Factitious cheilitis

Factitious cheilitis is a common observation in pediatric dermatology patients with compulsive disorders. The main mechanism is licking the lips, with or without biting, followed by development of an irritant contact dermatitis and a characteristic clinical picture.

Onychophagia, onychotillomania, onychotemnomania

Onychophagia is nail biting or chewing with swallowing of nail fragments and is often diagnosed in children. In younger children, onychophagia can be associated with thumb-sucking or secondary irritant

dermatitis, infection, inflammation and even malformation of the digits. It results from stressful situations and does not require a psychiatric evaluation in all situations. Onychotillomania refers to a self-induced nail disease brought on by chronic traumatization of the nail, also involving the paronychia and cuticle, with a variable degree of severity. Onychotemnomania is the result of cutting the nails too short, with secondary trauma to the nails.

Psychodermatology is an expression of the interaction between skin and mind. It is of paramount importance for the clinician to establish an appropriate physician-patient-family relationship in order to diagnose and treat factitial skin diseases. Clues to the clinical diagnosis include bizarre, linear, or geometric features on accessible parts of the body, ambiguous history of lesions that are done by the patient for public eye. Skin injuries can be found on locations easily accessible for self-injury, i.e., the face, trunk and extremities.

Points to Remember

- ***The prevalence of psychosomatic disorders among children with dermatological problems is high***
- ***They are frequently unreported because of difficulties in diagnosing and treating***
- ***Psychiatric and psychological factors may play different roles in the pathogenic mechanism of some skin diseases.***
- ***The mainstay of diagnosis and treatment is the differentiation between skin disorders associated with psychiatric illness and those of a purely psychiatric nature.***
- ***Dermatologists and psychiatrists should be aware of this pathology and work together as a team to resolve difficult cases, especially in children.***

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NEWS AND NOTES

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SURGERY

ANTENATAL DIAGNOSIS AND MANAGEMENT OF UROLOGIC ANOMALIES

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Abstract: *With advancing techniques and widespread availability of sonography, more genito-urinary anomalies are being picked up antenatally. This has substantially added to the anxiety of the propspectice parents and a spate of questions to the clinicians. This article is intended to clarify the antenatal and post-natal issues involved in the management of antenatally detected urologic anomalies in a lucid and practical manner.*

Keywords: *Hydronephrosis, Genito-urinary anomalies, Antenatal Diagnosis.*

The human fetus has for centuries remained a medical recluse in an opaque womb. However, in the 21st century, with lot of advancements in radio-diagnostic techniques, the accurate delineation of the normal and abnormal fetal anatomy has become a reality. Today, almost all congenital malformations occurring in these unborn 'patients' can be diagnosed antenatally. The exact pathology of the anomaly involved, its natural course, complications and treatment options help in prognosticating to the parents better.

Anomalies of the genitourinary system are common, accounting for 14-40% of anomalies detected on prenatal sonography.[1] Prenatal assessment with ultrasonography provides excellent imaging of fluid-filled structures (hydronephrosis, renal cysts, dilated bladder) and renal parenchyma which helps in generation of a differential diagnosis, identification of associated anomalies and assessment of the prenatal and postnatal risks.[2] The common genito urinary pathologies that can be detected on the ultrasound antenatally include hydronephrosis(HDN), cystic renal disease, renal agenesis, bladder and cloacal exstrophy and prune belly syndrome. Of these, hydronephrosis is the most common pathology observed. The focus of this article is mainly towards the antenatal diagnosis and management of hydronephrosis. The causes for antenatal hydronephrosis[3] are given in Table I.

Table I. Antenatal hydronephrosis – causes and their frequency

Etiology	Relative incidence
Transient Hydronephrosis	41-88%
PUJ-obstruction pelvic ureteric junction	10-30%
Vesico-ureteric reflux	10-20%
Vesico-ureteric junction obstruction, megaureter	5-10%
Multi-cystic dysplastic kidney	4-6%
Duplex kidney+/- ureterocele	2-7%
Posterior urethral valves	1-2%
Others: Prune belly syndrome, urethral atresia, urogenital sinus, exstrophy, tumors, etc.	Uncommon

Depending on diagnostic criteria and gestation, the prevalence of antenatally detected hydronephrosis (ANH) ranges from 0.6 to 5.4%.[4,5] The condition is bilateral in 17-54% and additional abnormalities are occasionally associated.[6] It is important to distinguish infants with significant illness that require long-term follow-up or surgery, from those with transient hydronephrosis and minimum need for invasive investigations.[7] ANH is said to be present if the antero-posterior diameter (APD) of renal pelvis is ≥4 mm in second trimester and ≥7 mm in the third trimester.[8]

Importance of diagnosis of antenatal hydronephrosis

Children with antenatally detected HDN are at a higher risk of having a postnatal pathology which increased

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with the increasing severity of the antenatal HDN. Mild ANH carries 11.9% risk for postnatal pathology, while moderate and severe ANH have a considerable risk of pathology, 45.1% and 88.3% respectively6. However, with the current level of knowledge of the natural history of antenatal HDN, a significant majority may not require any intervention, but it is the responsibility of the concerned clinician to counsel the expectant parents about the generally good prognosis for the vast majority of these fetuses.

Grading of antenatal HDN

Fetal hydronephrosis has been classified several ways. The grading provided by Society of Fetal Urology[9] (SFU) is based on the pattern of renal sinus splitting and is probably the most acceptable. The classification is based on renal pelvic dilation, number of calyces visualized and degree of parenchymal atrophy (Table II).

Table II: SFU grading of ANH

Grade 0	Intact central renal complex (renal pelvis)
Grade 1	Mild splitting of central renal complex
Grade 2	Dilatation of pelvis and major calyces; minor calyces non-dilated.
Grade 3	A markedly split pelvis with uniformly dilated minor calyces, but normal renalparenchyma
Grade 4	Characteristics of grade III with thinning of renal parenchyma

Other systems of classification have utilized the antero-posterior diameter (APD) of renal pelvis for classification of ANH (Table III).

Table III. Classification of ANH based on renal pelvic APD[6]

ANH Classification	APD (mm)	
	2nd Trimester	3rd Trimester
Mild	≤7	≤9
Moderate	7–10	9–15
Severe	≥10	≥15

SFU grade 3 and 4 probably co-relates with the moderate and severe grades of Lee's classification. It is to be noted here that almost 80% of fetuses diagnosed in the second trimester show resolution or improvement of findings with low likelihood of postnatal sequelae.[5,10] Patients with persistence or worsening hydronephrosis in the third trimester show higher rates of postnatal pathology and require close follow-up.

In addition to renal pelvic APD measurements, renal calyceal dilation and renal parenchymal thinning, other findings that should be looked for at antenatal ultrasound are given in box 1.

Timing of ultrasound for diagnosing urologic abnormalities

Box 1. Additional findings in antenatal ultrasound

1. Unilateral bilateral
2. Presence of ureteral dilatation.
3. Renal echogenicity
4. Volume and thickness of bladder
5. Posterior urethral dilation(Key hole sign)
6. Amniotic fluid level and the urinary flow
7. Presence of other associated congenital abnormalities[11]
8. Presence of other soft signs like increased nuchal translucency, echogenic focus in the heart, absent nasal bone, etc.

Even though antenatal hydronephrosis can be diagnosed as early as 10-12 weeks of gestation, a repeat scan before 20 weeks of gestation is required for better characterization of the problem. In India, since the legal cut-off age limit for doing an MTP is 20 weeks, it implies that the scan be done one or two weeks prior to this so that we can give some time for the parents to think, discuss regarding the subsequent steps to be taken.

Management

General principles

1. Uretero-pelvic junction obstruction.

a) Unilateral hydronephrosis

• Presence of any degree of unilateral hydronephrosis with a normal contra lateral kidney and a normal liquor volume does not warrant any sort of antenatal intervention. Child to be delivered at term as per obstetric indications.

• Post natal ultrasound to be done in a timely but non-urgent fashion (3 to 8 weeks of life).[12]

• Children with moderate to severe persistent HDN will require a nuclear scan (EC/DTPA/MAG-3) after 4-6 weeks of birth to confirm the level of obstruction, relative functioning of the kidney.

• Children with relatively preserved function with maintained or improving parenchymal thickness shall be closely observed as per protocol.

• Children with confirmed PUJ-obstruction with a relative function of <40% with a t-1/2 of more than 20 minutes and having progressive thinning of the renal parenchyma on USG shall warrant a pyeloplasty.

b) Bilateral hydronephrosis

Children presenting with antenatal bilateral HDN are a cause for concern. These fetuses require close monitoring with serial ultrasound scans repeated every 4-6 weeks or more frequently depending on the degree of HDN, oligohydramnios, period of gestation. Again, the cause may be bilateral PUJ-obstruction, bladder outlet obstruction or any of the factors mentioned previously.

• If antenatal ultrasonography shows evidence of lower urinary tract obstruction, parents should be referred to specialized centres for counselling regarding prenatal diagnostic and therapeutic interventions.

• In mothers having progressive oligohydramnios, fetal vesico-centesis is done on two or more occasions to assess for the biochemical prognostic indicators (Box 2).[13]

Box 2. Fetal vesico centesis – Biochemical prognostic indicators

1. Fetal urinary sodium of >100 mEq/L after 20 weeks' gestation
2. Elevated urinary calcium (>1.2 mmol/L)
3. Increased levels of urinary and serum ≥ 2-microglobulin.
4. Urinary osmolality >200mosm/dL
5. Urinary chloride>90mg/dL
6. Total protein >20mg/dL

• In fetuses with favourable indices, parents should be counselled regarding the role of vesico-amniotic shunting or in utero endoscopic ablation of valves.[14,15] It is to be noted here that these procedures in the mid trimester are being done in very few advanced tertiary centres abroad. There is no evidence that this intervention improves long term renal outcome or reduces mortality in fetuses with less severe disease.[16,17] Moreover, vesicocentesis and other interventions carry considerable risk of fetal loss, chorioamnionitis and preterm labour.

• In fetuses with unfavourable indicators, if diagnosed before 20 weeks of gestation, parents can be counselled regarding medical termination of pregnancy. However, if the condition is diagnosed later, the option is to deliver the child after inducing lung maturity with steroids and to consider post natal management. Fig.1 explains the antenatal management.[17]

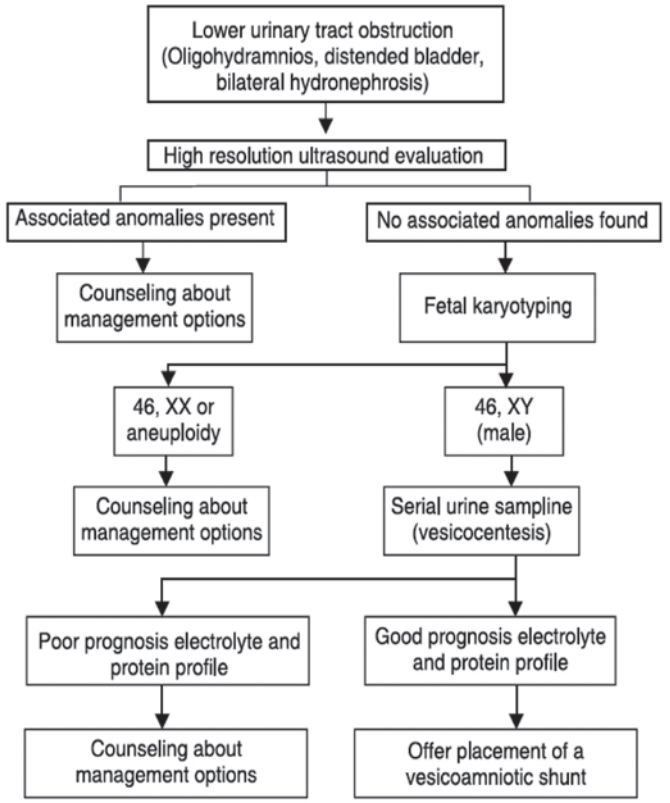

Fig.1. Algorithm for antenatal management of patients with lower urinary tract obstruction[17]

Post natal evaluation and management (Fig.2)

All children who had been detected to have some form of antenatal genito-urinary abnormality do warrant a post natal evaluation.[3] Since the urinary output of newborn infants is reduced in the first 24 hours of life, it is generally recommended that ultrasonography is deferred for 24-48 hours until a more physiologic urinary output has been re-established. The antenatal findings need to be re-assessed and a proper diagnosis needs to be made. Ultrasound screening needs to be completed within 1 week of life or prior to discharge from the hospital in patients with mild or unilateral hydronephrosis. However, in neonates with antenatal history of severe hydronephrosis and suspected bladder outlet obstruction, an early ultrasound, within 24-48 hours of birth would be required. If the findings suggest bladder outlet obstruction, then an early mictu rating cytourethorogram (MCUG) needs to be done after ruling out urosepsis for confirmation of valves. Early appropriate treatment needs to be instituted for the same.

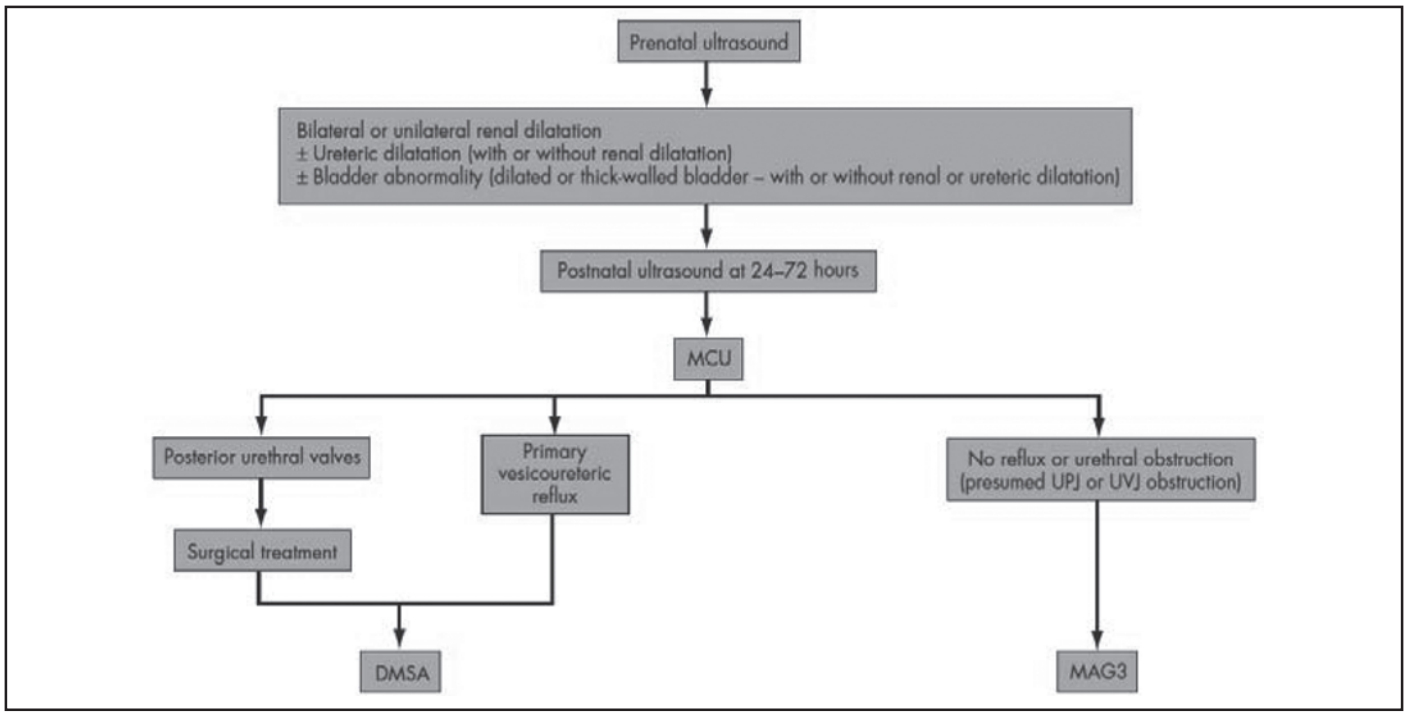

Fig.2. Approach to postnatal evaluation of neonates with history of ANH[18]

Neonates with suspected bilateral PUJ obstruction need to be delivered at term followed by an ultrasound and MCUG to rule out vesico ureteric reflux (VUR). An EC/DTPA/MAG-3 scan is done to confirm the obstruction, though relative functioning may not be reliable in such cases. Once confirmed, pyeloplasty is indicated, either simultaneously or in a staged approach with the better kidney operated first. All children with hydroureteronephrosis (HUN) need to be started on antibiotic prophylaxis till proper diagnosis and treatment is instituted.

Special situations

1. Ectopic kidneys: An ultrasound scan combined with a nuclear medicine scan/IVP would confirm the position and relative functioning of the kidney. Usually, they do not need any treatment, unless they present with complications like PUJ obstruction, infection, etc.

2. Ureterocoele: An ultrasound scan combined with a nuclear medicine scan/IVP would confirm the diagnosis and functioning of the involved kidney. The first step would be trans urethral incision of the ureterocoele followed by subsequent management if necessary.

3. Multicystic dysplastic kidney (MCDK): The management of MCDK has changed over the years from being aggressive on surgical removal to a more conservative approach with serial follow up. Contralateral kidney needs to be evaluated for associated anamolies. Surgical excision is warranted rarely if complications like hypertension, hematuria or infection develops.

Points to Remember

- ***Antenatally diagnosed unilateral HDN without any associated anomalies does not require any antenatal intervention and can be evaluated postnatally.***
- ***Antenatal intervention in the form of vesico-amniotic shunting is presently being performed on selected group of fetuses in very few centres abroad with no definite evidence of improved renal outcome.***
- ***Neonates with suspected bladder outlet obstruction warrant an early ultrasound scan followed by MCUG and appropriate treatment.***

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NEWS AND NOTES

3rd National Conference of Pediatric Education (NCPE), Chennai

Date: 12-09-2015 and 13-09-2015

Enquires

Dr. P. Ramachandran, Organizing Chairperson, 3rd NCPE
Email: ramachandran_dr@rediffmail.com

IAP Golden Hour in Emergency Medicine (GEM) Course, Aurangabad, MS

Date: 8th & 9th August, 2015

Contact
Dr.Mandar Deshpande
Mobile: 09850026542
Email: deshpanm@gmail.com

Workshop on "Clinical Evaluation in Pediatric Neurology"

Date: 8th & 9th August, 2015

Contact
Dr.Suvasini Sharma, Organizing Secretary,
Assistant Professor, Department of Pediatrics,
Lady Hardinge Medical College, New Delhi-110001.
E-mail id: sharma.suvasini@gmail.com, Mobile: 9910234344

RADIOLOGIST TALKS TO YOU

DISORDERS WITH DEFECTIVE MINERALISATION

***Vijayalakshmi G**
****Natarajan B**
****Jeya Rajiah**

It is very easy to appreciate increased density of bones and a diagnosis of sclerosing bone disorders can be quite straightforward. However, diagnosis of disorders with decreased density can be quite confusing. Bones are porotic with thin cortices or thinned shafts that in severe cases are hardly seen with clarity. Bent bones and fractures altering the configuration of the bones can also be misleading.

The most well known dysplasia with decreased bone density is osteogenesis imperfecta. It is a group of disorders consisting of mild and severe forms. The most severe form is osteogenesis imperfecta type 2 which is a lethal dysplasia with a dominant inheritance. It has specific radiographic features (Fig.1). There is generalised osteoporosis. The long bones show multiple fractures which have healed with exuberant callus giving the bones, a thick appearance. Though there is healing, bones cannot withstand stress and hence are bent and shortened.

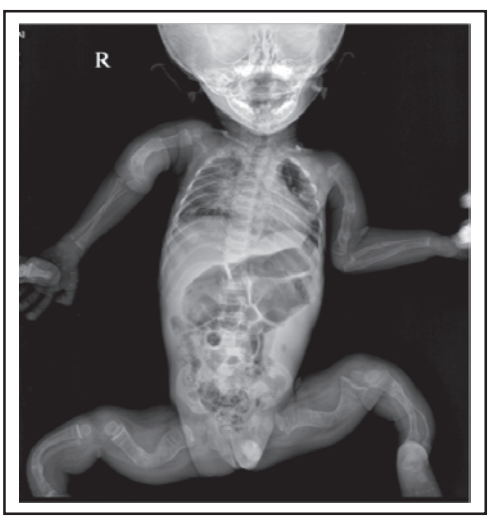

Fig.1. Osteogenesis imperfecta

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The vertebral bodies are flattened. The ribs are very thin. The child in fig.2 came with a complaint of crying on handling just as in osteogenesis imperfecta. An x-ray of the pelvis and lower limbs shows bent bones with numerous fractures. However, the bones are inherently normal as you can see in the areas where there are no fractures. These are normal bones with unnaturally innumerable fractures which should be diagnosed as Battered baby syndrome. Fractures may also be of varying ages. This distinction between osteogenesis imperfecta and child abuse is absolutely essential as both should not be mistaken for the other.

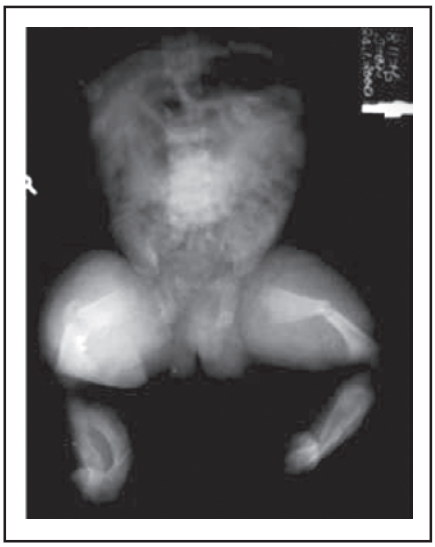

Fig.2. Battered baby syndrome

The other non-lethal types of osteogenesis imperfecta present at various stages of growth. The vertebrae may be flattened and ribs are thin and gracile (Fig.3). There may be variable number of fractures of the long bones. Fig.4 shows thin bent bones. There is a healed metaphyseal fracture at the lower end of the left femur.

Another dysplasia with defective mineralisation is "hypophosphatasia" where again there is a lethal type with recessive inheritance and autosomal dominant type with a later onset. Radiological features are accordingly very severe in the perinatal type. Bones are poorly ossified with some parts being invisible. Vertebrae are flat and porotic. There are multiple fractures. The late onset type again shows porotic bones as in Fig.5. Bones are extremely soft.

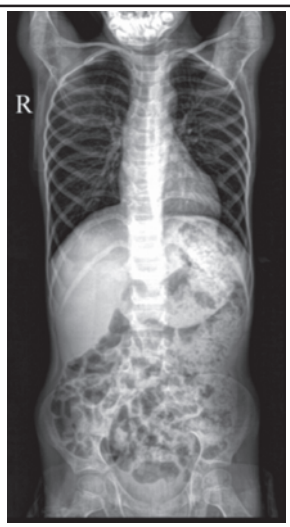

Fig.3. Osteogenesis imperfecta: Non-lethal type. Note thin ribs.

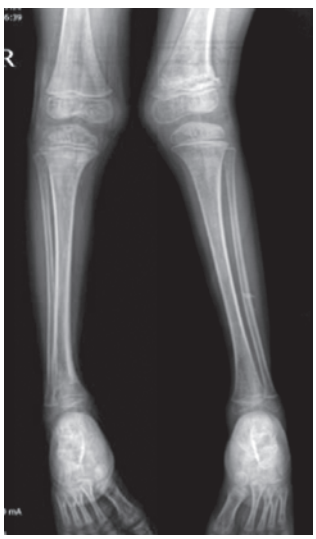

Fig.4. Osteogenesis imperfecta: Non-lethal type. Thin bent bones.

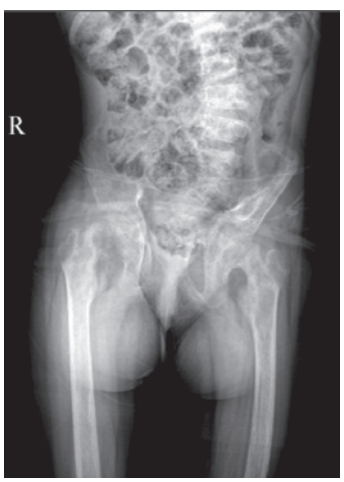

Fig.5. Hypophosphatasia: Late onset type.

There is a fracture of the neck of right femur and a deformed pelvis with protrusio acetabuli. Part of the metaphysis on the medial side of the upper right femur is not seen. Punched out metaphyseal defects like this and the ones in the distal radial metaphysis in Fig.6 are features of hypophosphatasia. Alkaline phosphatase levels are low. This enzyme is present in bone as tissue non specific alkaline phosphatase that provides phosphorus for the formation of hydroxyapatite crystals which leads to mineralisation of bone. Deficiency of this dephosphorylation enzyme causes hypophosphatasia.

Another rare congenital disorder with fragile bones is hereditary hyperphosphatasia or juvenile Paget's disease.

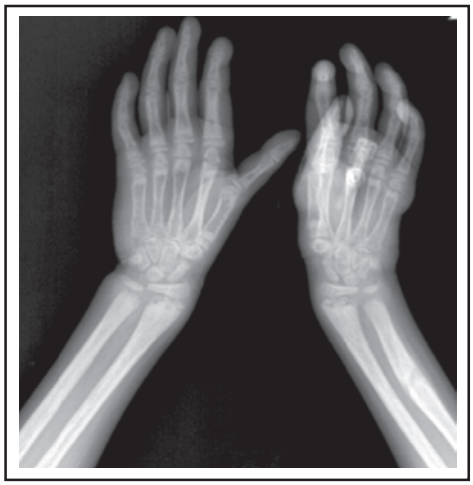

Fig.6. Hypophosphatasia (same child as Fig.5). Punched out metaphyseal defects at lower end of radius

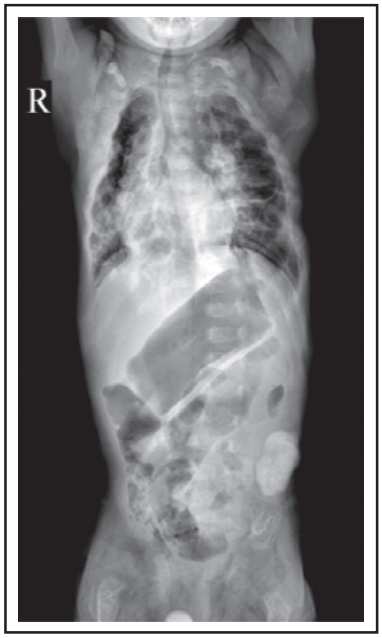

Fig.7. Florid rickets.

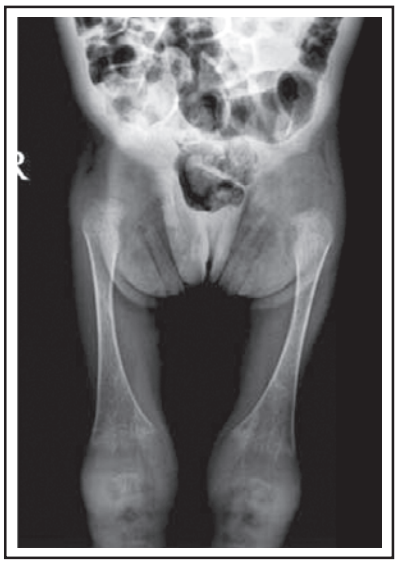

Fig.8. Florid rickets (same child as Fig.7). Widening of the growth plate.

In this, there is an increase in osteoblastic and osteoclastic activity which results in increased alkaline phosphatase levels. However, there is a failure to replace immature bone with compact Haversian bone. There is a continual subperiosteal deposition of disorganized bone that makes the diaphyses appear thickened but bones are weak and likely to fracture easily.

Fig.7 is another child with extremely porotic bones. The chest wall is collapsing because of the soft ribs, the cortices are thin and the outline of the long bones are not clearly visible. The vertebrae are flattened. On careful scrutiny you will be able to see that the physis in the upper femur is widened. An x-ray AP of both knees will remove all doubt that it is only the easily treatable rickets. The cartilaginous growth plate in Fig 8 is widened due to failure of mineralisation for want of vitamin D. This is the hallmark of rickets.

CLIPPINGS

Plant-Based, No-Added-Fat or American Heart Association Diets: Impact on Cardiovascular Risk in Obese Children with Hypercholesterolemia and Their Parents.

A randomized trial was performed to determine whether there is cardiovascular disease risk reduction from a plant-based, no–added-fat diet and the American Heart Association diet in children in a large, Midwestern hospital system's predominantly middle class outpatient pediatric practices. Thirty children (9-18 years of age) parent pairs with a last recorded child body mass index >95th percentile and child cholesterol >169 mg/dL were randomized to PB or AHA with weekly 2-hour classes of nutrition education. Children on PB had 9 and children on AHA had 4 statistically significant ($P < .05$) beneficial changes from baseline (mean decreases): body mass index z-scorePB ("0.14), systolic blood pressurePB ("6.43 mm Hg), total cholesterolPB ("22.5 mg/dL), low-density lipoproteinPB ("13.14 mg/dL), high-sensitivity C-reactive proteinPB ("2.09 mg/L), insulinPB("5.42 uU/mL), myeloperoxidasePB/AHA ("75.34/69.23 pmol/L), mid-arm circumferencePB/AHA ("2.02/"1.55 cm), weightPB/AHA ("3.05/"1.14 kg), and waist circumferenceAHA ("2.96 cm). Adults on PB and AHA had 7 and 2, respectively, statistically significant ($P < .05$) beneficial changes. The significant change favoring AHA was a 1% difference in children's waist circumference. Difficulty shopping for food for the PB was the only statistically significant acceptability barrier.

PB and the AHA in both children and adults demonstrated potentially beneficial changes from baseline in risk factors for CVD. Future larger, long-term randomized trials with easily accessible PB foods will further define the role of the PB in preventing CVD.

Macknin M, Kong T, Weier A, Worley S, Tang AS, Alkhouri N, Golubic M. Plant-Based, No-Added-Fat or American Heart Association Diets: Impact on Cardiovascular Risk in Obese Children with Hypercholesterolemia and Their Parents. 2015 Volume 166, Issue 4, Pages 953–959.e3 , DOI: http://dx.doi.org/10.1016/j.jpeds.2014.12.058.

NEWS AND NOTES

18th National Conference of IAP Pediatirc Infectious Diseases Chapter (NCPID 2015), Mahabaleshwar
Date: October 31st - November 1st , 2015

Enquiries to
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CASE STUDY

HISTIOCYTOSIS LYMPHADENOPATHY PLUS SYNDROME

***Hema Chitra J**
***Srinivasan G**
***Karthikeyan M**
***Dhakshayani R V**
****Rema Chandramohan**

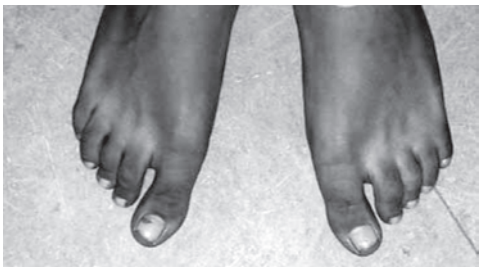
Abstract:*Histiocytosis-lymphadenopathy plus syndrome comprises of histiocytosis and lymphadenopathy occurring along with cutaneous, cardiac, endocrine abnormalities, joint contractures and deafness. It is caused by homozygous or compound heterozygous mutation in the SLC29A3 gene on chromosome 10q22. We present a case report of this rare genetic disorder.*

Keywords: *Histiocytosis, Lymphadenopathy, H syndrome.*

Histiocytosis-lymphadenopathy plus syndrome is a rare disorder, affecting approximately one hundred individuals worldwide.

Case report: A 12 year old female child was referred to our hospital for evaluation of mediastinal lymphadenopathy which was diagnosed incidentally when a chest X-ray was taken for nonspecific chest pain. She was the fourth born of nonconsanguineous parents with no live siblings. There was history a of two neonatal deaths and one sibling death at two years of age due to meningitis. This child was diagnosed with type I diabetes mellitus at two years of age and was on mixed insulin regimen with good glycemic control. She was diagnosed to have hypothyroidism at seven years of age and was on regular thyroxine supplements for the same. She also had sensorineural deafness and was using a hearing aid.

General examination revealed significant cervical, axillary and inguinal lymphadenopathy, camptodactyly (fixed flexion contractures of the proximal inter phalangeal joints of the fingers and toes), and hallux valgus (Fig.1). Her height was less than the 3rd percentile and her weight was appropriate for height. There was a hyperkeratotic patch in the thigh. Her SMR staging was 1.There was no pallor, edema, clubbing or jaundice. Cardiovascular, respiratory, abdomen and central nervous system examination was normal.



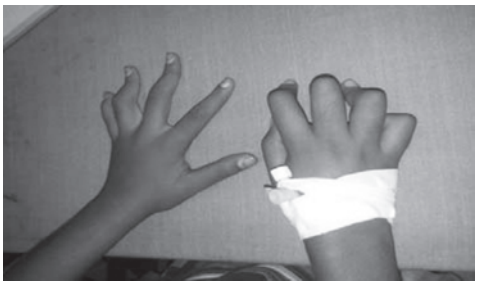


Fig.1. Observation showing hallux valgus and camptodactyly

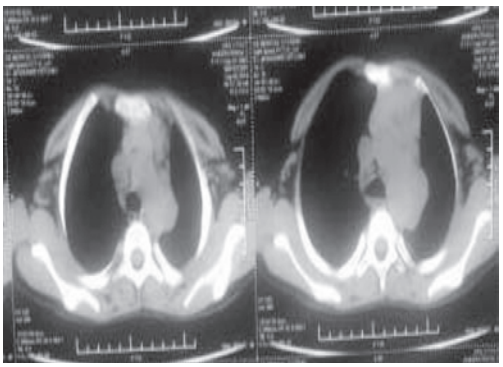


Fig.2. CT Chest-showing mediastinal lymphadenopathy

Although the child was asymptomatic, we investigated for possible causes for her generalised lymphadenopathy. Complete hemogram, peripheral smear and metabolic parameters were normal. Screening for tuberculosis and HIV was negative. CT chest showed mediastinal lymphadenopathy (Fig.2). Bone marrow aspiration was normal. Cervical lymph node biopsy showed the presence of histiocytes, plasma cells, lymphocytes and eosinophils with congested and dilated sinusoids. Sections from the skin showed orthokeratotic epidermis with dermis showing a collection of histiocytes. Immunohistochemistry of the

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lymphnode specimen showed mixed pattern with histiocytes which were positive for CD68+,CD20+, S100+, CD1A-.

With the above clinical picture of generalised histiocytic lymphadenopathy along with cutaneous and endocrine abnormalities (diabetes mellitus and hypothyroidism), skeletal abnormalities and deafness a literature search was done which narrowed the diagnosis to histiocytosis-lymphadenopathy plus syndrome.

Discussion

Histiocytosis-lymphadenopathy plus syndrome comprises features of 4 histiocytic disorders.

1. Faisalabad histiocytosis (FHC)
2. Sinus histiocytosis with massive lymphadenopathy (SHML)
3. 'H' syndrome
4. Pigmented hypertrichosis with insulin-dependent diabetes mellitus (PHID)

A feature common to the disorders in this spectrum is histiocytosis. FHC is described as an autosomal recessive disorder involving joint deformities, sensorineural hearing loss and generalized lymphadenopathy.[1] SHML or familial Rosai-Dorfman disease, is described as a rare cause of lymph node enlargement in children, consisting of chronic massive enlargement of cervical lymph nodes frequently accompanied by fever, leukocytosis, elevated erythrocyte sedimentation rate, and polyclonal hypergammaglobulinemia with extranodal involvement in approximately 25% of patients.[2] 'H' syndrome is characterized by cutaneous hyperpigmentation and hypertrichosis, hepatosplenomegaly, heart anomalies, and hypogonadism, hearing loss (50%) and short stature.[3] Hypogonadism could not be documented in our child, although she was lagging behind in SMR staging, her uterus and ovaries were appropriate for her age and a hormonal assay was not done as she was preadolescent. PHID is characterized by predominantly antibody-negative insulin-dependent diabetes mellitus associated with pigmented hypertrichosis and variable occurrence of other features of 'H' syndrome, with hepatosplenomegaly occurring in about half of the patients.[4]

Bolze et al. noted that mutations in the SLC29A3 gene had been implicated in 'H' syndrome, PHID, FHC and SHML, and that some patients presented a combination of features from two or more of these syndromes, leading to the suggestion that these phenotypes should be grouped together as 'SLC29A3 disorder'.[3] The disease mainly affects the lymph nodes, but there may be a simultaneous involvement of extranodal locations (eyelids, skin and subcutaneous tissue, gastrointestinal tract, upper airways and central nervous system) in 40% of the cases. Mediastinal involvement is extremely rare but was observed in our child.

In our patient, the presence of joint deformities-camptodactyly and hallux valgus(feature of FHC), sensorineural hearing loss (feature of FHC & 'H' syndrome), generalized lymphadenopathy with mediastinal involvement (feature of FHC & SHML), cutaneous hyperpigmentation (feature of 'H' syndrome), short stature and insulin dependant diabetes mellitus (feature of PHID) shows the overlap of clinical features of all the four histiocytic disorders. The characteristic histopathological features in lymph node biopsy and the presence of S100 protein and macrophage antigen CD68 positivity in immunohistochemistry favoured the diagnosis.

The syndrome is caused by mutations in the SLC29A3 gene which leads to decreased activity of the ENT3 protein. Researchers speculate that the resulting impairment of nucleoside transport leads to a build up of nucleosides in lysosomes, which may be damaging to cell function. It is unclear how the mutations lead to histiocytosis and other features of the condition or why affected individuals can have different patterns of signs and symptoms. This condition is inherited in an autosomal recessive pattern.

It is frequently a benign self limiting disease. In the presence of vital organ compression and or extranodal localization with important clinical signs, surgical debulking may be needed. Radiotherapy has shown limited efficacy, while chemotherapy in general is ineffective. More experience is needed to evaluate the role of interferon.

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3rd NATIONAL CONFERENCE OF PAEDIATRIC EDUCATION 2015

(NCPE 2015)

Dates: 12 & 13 September 2015

Venue: Sri Ramachandra Medical College and Research Institute,

Porur, Chennai, 600116

Organised by:

Department of Paediatrics, Sri Ramachandra Medical College and Research Institute,

IAP HOD/Professor cell & IAP-Chennai City Branch

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For any clarifications contact: Dr J. Dinesh Kumar (Jt. Org. Sec) Mobile: 9841111009 e-mail:ncpesrmc2015@gmail.com

15th NATIONAL CONFERENCE NEUROLOGY CHAPTER OF IAP

NEUROPEDICON 2015

Organizer: INDIAN ACADEMY of PEDIATRICS, NAGPUR

Venue: Hotel Centre Point, Ramdaspeth, Nagpur

Date: 16 to 18 October 2015

*** An interactive event with Workshop, discussion & debates ***

* Covering A to Z of Pediatrics Neurology *

Our Esteem Faculty:

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Dr KMP Suresh, Dr PAM Kunju, Dr A Agrawal, Dr Shefali Gulati, Dr Brajesh Udani,
Dr Anup Varma, Dr Lokesh Lingappaa, Dr S Kamath, Dr Pramod Jog, Dr V Yeole

Registration details:

Registration	Upto 30th June 15	Upto 30th Aug 15	Upto 30th Sep 15	Spot
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Welcome to South Pedicon 2015 !

The members of IAP North Arcot and Kanchipuram take great pride in inviting you all for the 40th Tamilnadu State Conference and 29th South Pedicon. The event is to be held in Vellore in the prestigious **Vellore Institute of Technology from 6th August 2015 to 9th August 2015**. We cordially invite you to join us in this academic extravaganza as we hold our academic deliberations in a serene background without the hustle and bustle of urban life.

The conference will cover a range of topics meticulously planned by our Scientific committee involving academic heads from Christian Medical college, Government Medical college and academically oriented private practitioners. This unique blend brings you a wide spread of topics useful for everyone from the aspiring postgraduates, the specialists involved in advanced care of the very sick children and the office practitioners.

We invite abstracts for the papers and posters that will steer us into evidence based practice and contribute to enriching our knowledge.

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Relax in our serenity and blend into our sophistication.

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IAP TNSC

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Dr. G.Kirubakaran
Organising Chairperson

Dr. S.Narmada
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TILL 15.07.2015	5500	6000	5000	3500	FREE
SPOT (FROM 15.07.2015)	6000	6500	5500	4000	3500

Note :
1. *70 years and above as on 6.8.2015 considered as Senior citizens(please attach age proof)*
2. *Separate registration mandatory for workshops*
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