CAPE NEWS

Newsletter of the Indian Society for Pediatric and Adolescent Endocrinology (ISPAE)

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Contents

From the Editor's Desk

Dear members,

This new issue of CAPE NEWS marks the beginning of a 2y term for the new Team ISPAE. We hope our Society will become stronger and serve the children of this country better in the capable hands of Drs Anju Seth, Anurag Bajpai, Rajni Sharma and the EC.

This issue brings you two interesting mini-reviews: one on the role of next-generation sequencing in the molecular diagnosis of monogenic forms of diabetes, and the second one on 'Bone Xpert', a new tool for assessment of bone age. It also includes the summary of recommendations from the recent Endocrine Society Guidelines on 'Treatment and prevention of pediatric obesity'. Also featured is an interesting case report of 'Infantile hypophosphatasia'. I am sure all of you will find this issue useful.

I thank Drs Rajni Sharma, Ravindra Kumar and Reetha Gopinath, members of previous Cape News editorial team who supported me in all possible ways. I am happy to receive the continued support of Drs Anju Virmani and Sachin Mittal during the period of 2017-18, and welcome the new members of the Cape News editorial team, Dr Sweta Budyal (Mumbai), Dr Tushar Godbole (Nashik) and Dr Vani H N (Bangalore). I thank all of them for their active participation in designing this issue and for their valuable contributions.

Dr Vijaya Sarathi, Editor, CAPE NEWS

ISPAE Biennial Meeting 2017: Coimbatore, Tamil Nadu, Nov- Dec 2017

Organizing Chairperson: Dr P Raghupathy, drp.raghupathy@gmail.com
Organizing Secretary: Dr Ahila Ayyavoo, ahila.ayyavoo@gmail.com

We are proud to announce the 44th annual ISPAD meeting, **ISPAD 2018,** to be held in Hyderabad, India 11-14 Oct 2018, in association with ISPAE.
Organizing Chairs: Dr Anju Virmani and Dr Banshi Saboo
This is the time to become an **ISPAD member** - the discounted 3y membership for us in India (LMIC) is just $ 80/ year. Membership allows discounted rates for attending the **43rd Annual ISPAD Conference, ISPAD 2017** in Innsbruck, Austria (18-21 October 2017), and of course **ISPAD 2018.** Click ispad.org and become part of this vibrant community.

Hearty Welcome to New ISPAE Members

1. Dr Nisha Bhavani, Kochi	2. Dr Pramila Dharmshaktu, New Delhi
3. Dr Mounika Anitha, Visakhapatnam	4. Dr Sree Divya Bekkam, Tirupati
5. Dr Aashish Sethi, New Delhi	

Message from the President and the Secretary, ISPAE

Dear Friends,
Greetings from the ISPAE Executive Council 2017-18!
The new ISPAE Executive Council that assumed office from 1st January 2017 is an eclectic mix of youth & experience, and hence, brimming with enthusiasm. In the very first meeting, the Executive has decided to initiate work on two important projects:
a. Development of T1DM Road Map for the years 2017-18, where different groups of experts would work on various aspects of T1DM, including empowerment of health care providers, support & advocacy, development of E-resources, and defining research & teaching priorities. The project would be overall supervised by Dr Anurag Bajpai.
b. Development of ISPAE guidelines on GH therapy. The convener for these guidelines would be Professor Rajesh Khadgawat.

In addition, Professor Vandana Jain has been nominated by our Society to participate in the Pediatric Endocrine Society (PES) Neonatal Diabetes Mellitus consensus group meeting upon invitation from PES. The next two years appear exciting, with many planned meetings, including the first APPES-ISPAE Fellows' School preceding the Biennial ISPAE conference at Coimbatore in November 2017. The ISPAD Annual Meeting in association with ISPAE is scheduled for October 2018 in Hyderabad. New members have been inducted into the Cape News & Web Committees, so that over the coming months we can look forward to some fresh changes there as well.

The current Executive looks forward to hearing from you regarding any new initiatives by you as well as suggestions for us to work on.

With warm regards,
Dr Anju Seth and Dr Anurag Bajpai

Applications invited for ISPAD fellowships 2017

1. JDRF-ISPAD Research Fellowship for ISPAD members below age 40, for a 6 month research visit to an ISPAD Center of Excellence.
2. ISPAD Alan Drash Clinical Fellowship for ISPAD members below age 45, for a 6 week clinical visit to an ISPAD Center of Excellence.
The last date for application is 1st May 2017. For full details, please visit the ISPAD website: www.inpad.org.

2nd Annual 'Masterclass for Fellows in Pediatric Endocrinology'

Dr Shaila Bhattacharyya, Manipal Hospital, Old Airport Road, Bengaluru

The 2nd Annual 'Masterclass for Fellows in Pediatric Endocrinology' is being organised by Dr Shaila Bhattacharyya on 30th April and May 1st at Manipal Hospital, Bengaluru. The program is particularly suited for fellows in pediatric endocrinology and those pursuing DM in endocrinology. The 2 day course will use 6 case-based modules- covering growth, puberty, thyroid, diabetes, calcium and bone, and electrolyte disorders. The number of delegates would be limited to ensure active participation.

Introduction to 'New ISPAE Guidelines on Type 1 Diabetes Mellitus in Children'

Dr. Aspi J Irani, Pediatric Diabetologist, Mumbai

Several excellent guidelines are available for the management of type 1 diabetes mellitus (T1DM) in children and adolescents. These include the International Society of Pediatric and Adolescent Diabetes (ISPAD) guidelines (2014), Australian guidelines (2011), Canadian Diabetes Association guidelines (2013) and the American Diabetes Association (ADA) guidelines (revised in 2016). One may therefore ask – what was the need for publishing the present set of guidelines?

In the year 2011, the Indian Society for Pediatric and Adolescent Endocrinology (ISPAE) brought out its own "Clinical Practice Guidelines for Type 1 Diabetes Mellitus in Children and Adolescents in India". The ISPAE guidelines have been written keeping in mind the situation prevailing in our country, facilities available in our country, and the constraints under which we work. These guidelines summarize the latest scientific data on the subject and offer suggestions on how best to apply the same for optimum results in the Indian scenario. Since the publication of the first edition, advances in management of T1DM have been taking place at a rapid pace, and hence the editors felt the need to revise and update the guidelines.

T1DM is the commonest metabolic-endocrine disease in children and adolescents. There has been a significant increase in the number of new cases in the past few years, especially in the age group of 1-5 years. There are very few specialized pediatric endocrinologists and pediatric diabetologists in our country. Some patients are managed by the "adult" diabetologist or endocrinologist. Most pediatric patients with diabetes are treated by the general pediatrician. Since a practicing pediatrician in India is not likely to encounter more than a couple of new cases each year, it is not possible for him / her to learn and apply the finer points of diabetes management. These guidelines should serve as a quick and ready reference manual for those caregivers who are not specialized in pediatric diabetes care.

In India, few centers are able to provide a team based approach for management of diabetes in the pediatric age group. A 24-hour helpline for these patients is virtually non-existent. Little attention is paid to the psychosocial needs of the patients. There are very few diabetes support groups. Most schools in our country are neither geared for, nor willing to take up, any responsibility for caring for the child with diabetes. The different types of foods we consume in various parts of our vast country and lack of freely available data on carbohydrate content of our foods poses a major challenge. The joint family system creates a problem especially with meal planning. The same system, if properly harnessed, can afford parents the benefit of additional support and assistance in managing the child with diabetes. Poverty, absence of government funding and illiteracy are some of the other important hurdles in the management of T1DM. Misconceptions about the condition, and those affected by it, are rampant. Blind faith in alternative systems of medicine often leads patients to omit insulin therapy, with disastrous results. Availability of the latest medications and devices for management of diabetes is no longer a problem. The challenge lies in making these available to all classes of patients and ensuring that they are utilized appropriately so as to derive maximum benefit.

For all the above mentioned reasons, the ideal therapeutic approach may not always be the most practical one to follow. This book on guidelines for diabetes management by the ISPAE has been prepared, keeping these factors in mind.

Four new chapters have been added in the present edition. *The first month after diagnosis of type 1 diabetes mellitus* is a crucial period. Patients and their families have to be helped to overcome the initial shock and denial, and to accept the diagnosis with a positive outlook. At the same time, insulin therapy must be initiated, patients must be trained in basics of diabetes self-management, and preliminary work-up to define the type of diabetes and to look for co-morbidities must be undertaken. Families need constant guidance as the phase of metabolic recovery (with high insulin requirements) gradually gives way to the honeymoon phase (with rapidly dropping insulin needs). The quality of care received during this period will have a bearing on the long term outcome.

T1DM is a lifelong disease and the treatment can be very expensive. Knowledge of *the economics of diabetes care in the pediatric age group* can help the treating doctor to choose the best treatment for a given patient, keeping in mind his / her financial status. This chapter will focus on how T1DM can be controlled reasonably well even without the newer costly medications or gadgets.

Neonatal diabetes mellitus (diabetes with onset in the first six months of life) is unlikely to be T1DM. It needs a special work-up, to distinguish between transient and permanent varieties and to detect as early as possible, with the help of genetic molecular studies, whether the patient would be sulfonylurea-responsive or insulin-dependent. Further, certain syndromes can present with neonatal diabetes. Making a precise diagnosis can improve the outcome by alerting the treating doctor about the appropriate treatment and possible known associations.

Type 2 diabetes mellitus (T2DM) is assuming epidemic proportions *in the adolescent age group* in some parts of the world and is also being encountered in urban India. Every pediatrician needs to be conversant with the primary prevention, early detection (by screening appropriate populations) and management of this disease. Guidelines on pediatric T1DM would be incomplete without a discussion on this variety of diabetes.

Next-generation sequencing based genetic testing for monogenic forms of diabetes

Dr Aaron Chapla, **Dr Riddhi Das Gupta**, **Prof Nihal Thomas**, Department of Endocrinology, Diabetes & Metabolism, Christian Medical College, Vellore- 632004, Tamilnadu.

Diabetes mellitus (DM) is now recognized as a global pandemic, affecting around 415 million people worldwide (1). The vast majority of these patients (85%) are classified as polygenic type 1 diabetes (T1D) or type 2 diabetes (T2D) (2). However, growing evidence from genomic research shows that monogenic forms could account for approximately 1–2% of all diabetes (2). Maturity onset Diabetes of the Young (MODY), neonatal diabetes (NDM), and a few of the syndromic forms of diabetes fall under the category of monogenic diabetes.

Neonatal Diabetes:

NDM is a non-autoimmune disorder characterized by the onset of hyperglycemia within the first 6 months or 26 weeks after birth (3). Diabetes in these patients can persist throughout life, i.e. permanent neonatal diabetes mellitus (PNDM), or can be transient i.e. hyperglycemia resolves over time (TNDM). Genes implicated in NDM include *KCNJ11, ABCC8, INS, GCK*, and *PDX1*. An accurate genetic diagnosis could be beneficial as patients carrying a mutation *in KCNJ11* or *ABCC8* can be well managed with higher doses of oral sulfonylurea drugs instead of insulin therapy. Till recently, the genetic diagnosis was based on Sanger sequencing of more than 40 PCR products from the *KCNJ11, ABCC8*, and *INS* genes (3).

MODY:

MODY is an autosomal dominant disease caused by mutations in one of the 13 different genes which are primarily involved in β cell development and functioning. Clinical suspicion is based on a typical presentation of diabetes before the age of 25 years, negative testing for T1D-related auto-antibodies, no history of DKA, and absence of signs of insulin resistance (4). However, due to overlapping clinical features with the more common polygenic diabetes, identification of patients with MODY is a diagnostic challenge. Till recently the molecular diagnosis of MODY included sequential screening of a few MODY-related genes based on the phenotype. In addition, due to the prohibitive cost and limitations associated with the scalability of Sanger sequencing, the majority of diagnostic laboratories limit the screen for hepatocyte nuclear factor 1alpha (*HNF1A*), glucokinase (*GCK*), and hepatocyte nuclear factor 4 alpha (*HNF4A*) mutations (5).

Due to lack of clinician awareness, limited genetic diagnostic facilities, and affordability, this subset of patients with NDM or MODY are often misdiagnosed as the more common T1D and T2D (6). In a heterogeneous disorder like MODY, comprehensive testing of all the known MODY genes would be beneficial (7), but so far has not been practical. Due to this, many of these patients may be treated inadvertently with insulin or an inappropriate oral antidiabetic drug (OAD), not only in developing countries but also in the developed countries (8).

Around 30 genes have been implicated in the pathogenesis of monogenic forms of diabetes. Depending on the gene implicated, patients may present with a severe phenotype, such as pancreatic agenesis (exocrine insufficiency) with neonatal diabetes, or a milder phenotype, with diabetes onset during adolescence or adulthood. Recently we have established and utilized novel Next-Generation Sequencing (NGS) based "2nd Generation Genetic Diagnosis of MODY" (2GDMODY) for screening of all the known 13 MODY genes (4) and also an additional 17 genes implicated in various monogenic forms of diabetes. The (13+17) NGS panel includes *ABCC8, AKT2, BLK, CEL, CISD2, CP, EIF2AK3 GATA6, GLUD1, HADH, HNF1A, HNF1B, HNF4A, IER3IP1, INS, INSR, KCNJ11, KLF11, NEUROG3, PAX4, PDX1, PTF1A, RFX6, SLC2A2, WFS1, ZFP57, GCK, GLIS3, NEUROD1, FOXP3*.

Utilizing this methodology, we have been able to confirm the diagnosis and provide appropriate therapy in many cases. Further, a confirmed diagnosis also helps in presymptomatic testing in other family members and for genetic counselling. We discuss a few cases which help to understand the importance of making a confirmed diagnosis in monogenic forms of diabetes.

Case 1: An 8 year old girl with recurrent urinary tract infections was incidentally diagnosed with mild fasting hyperglycemia. This patient, with a three generation family history of diabetes, was found to be negative for GAD & IA2 autoantibodies, but on testing was positive for a reported *GCK* (MODY 2) gene mutation (4). With a confirmed genetic diagnosis, this patient was managed with diet and exercise which is the standard of care in this form of MODY. However, in case of unawareness about MODY, this patient could have been misdiagnosed as T1D and may have been treated with insulin therapy which could prove fatal for her. Later in life, this patient can be managed appropriately during her pregnancy. Based on published data, if the developing fetus is negative for this mutation, it may result in increased fetal growth and hence insulin treatment is recommended during her gestation.

Case 2: A 21 year old lady, diagnosed at the age of 11 years with diabetes, and with autosomal dominant family history, was found to positive for *HNF1A* mutation (4). With a confirmed diagnosis of MODY 3, she was shifted from insulin to low dose sulfonylurea therapy, since the majority of

patients with MODY 1 (*HNF4A*) and MODY 3 (*HNF1A*) can be managed well with low dose sulfonylureas.

Case 3: A 25 year old lady with pre-gestational diabetes and history of 1st trimester miscarriage was being treated with OADs with suboptimal glycemic control. She was found to be positive for *HNF1B* (MODY 5) mutation (4), and started on insulin, which is the recommended standard of care in these patients, due to defective beta cell secretory function. This form of MODY is associated with renal cysts and diabetes syndrome (RCAD) and urogenital tract anomalies.

Case 4: A 1 year 11 month old boy, diagnosed as T1D elsewhere, was admitted for glycemic control. He presented with diabetic ketoacidosis and had been on regular insulin, with his last HbA1C being 10.2%. There was history of recurrent diarrhea, hypopigmented patch, oily stools and recurrent chest infections. He was a single child born to non-consanguineous parents, and had attained normal developmental milestones. Even though he was diagnosed after 6 months of birth, the treating physician wanted to rule out NDM since he was negative for auto-antibodies. Interestingly, he was found to be positive for *INS* gene mutation (c.265G>A, R89C), which has been reported to cause PNDM (9), and has been reported to be detected beyond 6 months of life (10). This finding corroborated earlier reports, and suggested the need for studies to estimate the prevalence of INS gene mutation in infants negative for auto-antibodies, and diagnosed after age 6 months.

These cases highlight the need for genetic testing, which would aid in making a firm diagnosis, and in a few cases, change the appropriate therapy, which can range from diet, exercise and OAD, to absolute need for insulin therapy. Therefore, NGS based testing in clinically suspected MODY/neonatal diabetes subjects could represent a cost-effective patient centric model of diabetes care in a clinical setting.

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Bone Xpert

Dr Anurag Bajpai, Pediatric & Adolescent Endocrinologist, Regency Center for Diabetes, Endocrinology & Research, Kanpur; Fortis Memorial Research Institute, Gurgaon

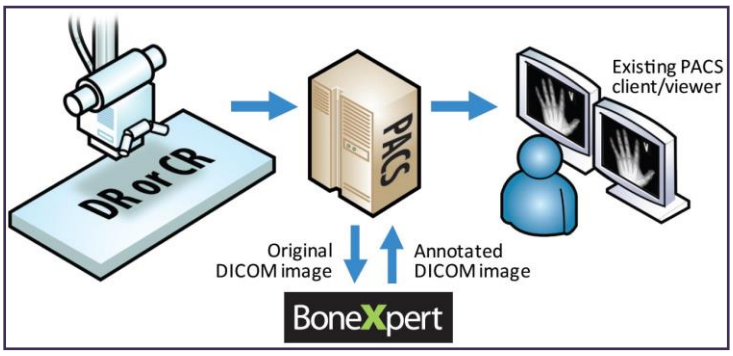
Bone age estimation is an essential part of pediatric endocrine practice. The Tanner Whitehouse and Greulich Pyle atlases are standard methods for bone age estimation, but are time consuming, and complicated by technical difficulties and significant inter- and intra-observer variation. This limits their universal use in clinical practice. Automated bone age estimation with rapid turnaround time is therefore a desirable goal. Bone Xpert provides an automated alternative to manual assessment of bone age.

Principle

Bone Xpert analyses a radiograph from the non-dominant hand and compares it to databases to provide an estimation of bone age. The database was developed using 3000 Danish X-Rays and validated on 1600 Dutch radiographs. The software provides bone age for each individual region of interest and provides an average bone age based on all the readings. The software provides information as per TW3 and GP methods, with a range of 2-19 years.

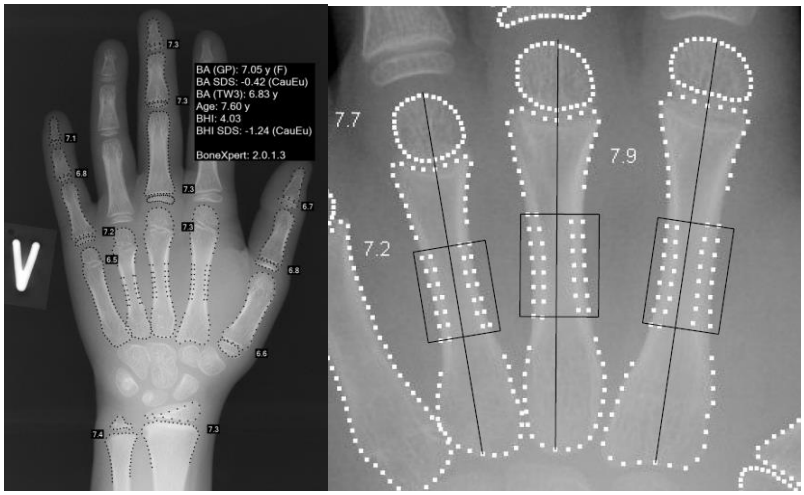
Evidence

Bone Xpert has been validated in different populations, and its accuracy found to be similar to manual GP readings, with precision three times the manual assessment.



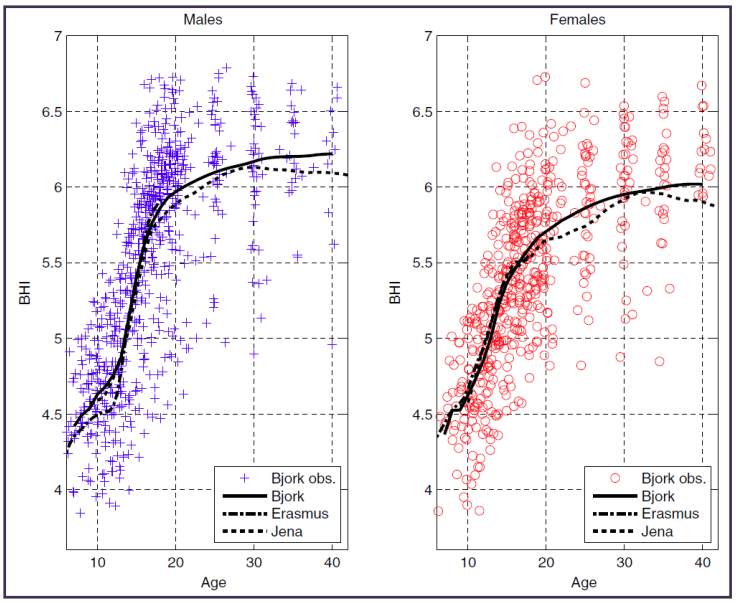
Workflow

1. BoneXpert is installed on a PC.
2. Radiographer sends X-ray from PACS to BoneXpert computer.
3. BoneXpert computer analyses in 3-5 seconds.
4. Annotated image with results is sent to PACS.
5. The doctor can view the result in office within a few minutes of acquisition of X Ray.



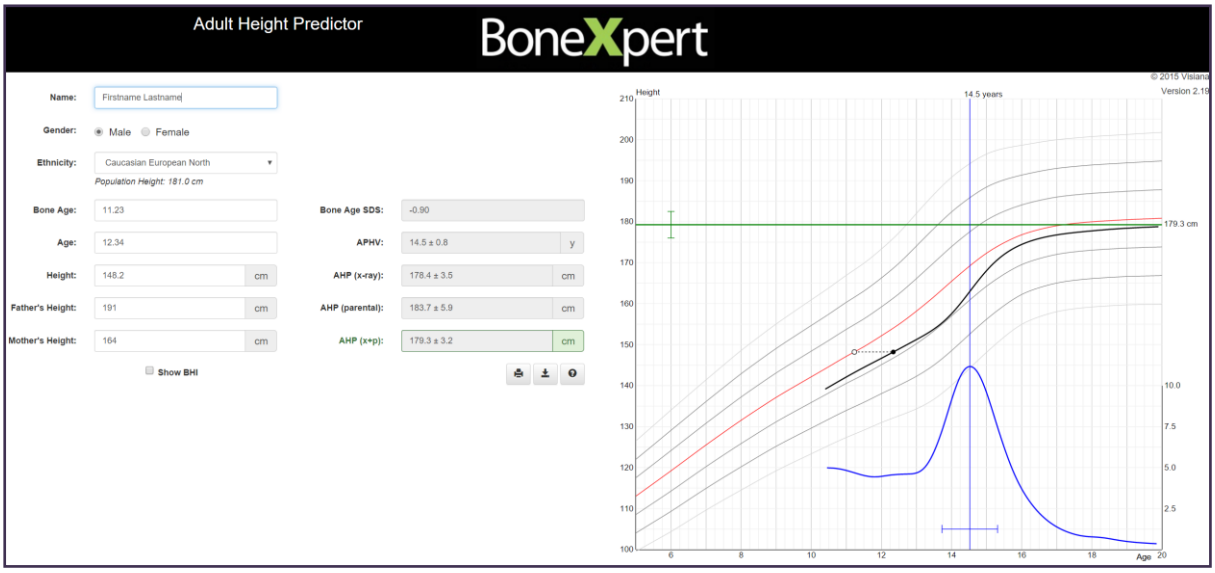
Results

The Bone Xpert annotated image provides information about precise GP and TW3 bone age, along with standard deviation score. Another information provided is the bone health index (BHI) Z Score, which is a marker of bone strength, validated in children and adults.



Adult height prediction

Bone Xpert Adult Height Predictor can be used to predict adult height using the current height, mid-parental height, bone age, and age at menarche (for girls) with prediction errors of 2.7-3.5 cm for 8-15 years of age. These results are, however, population dependent, and algorithms are being developed for Indian children.



Limitations

As Bone Xpert is an automated assessment, precise site positioning of the site is mandatory. Post image processing to enhance images can influence Bone Xpert estimations, and should be avoided. Indian population based adult height prediction is not currently available, limiting the generalisability of adult height prediction. Its use is quite expensive (5 Euros per analysis).

Further reading

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Prevention and Treatment of Pediatric Obesity: An Endocrine Society Clinical Practice Guideline Based on Expert Opinion
SUMMARY OF RECOMMENDATIONS

1.0 Diagnosing overweight and obesity

1.1 We recommend using body mass index (BMI) and the Centers for Disease Control and Prevention (CDC) normative BMI percentiles to diagnose overweight or obesity in children and adolescents ≥2 years of age.
1.2 We recommend diagnosing a child or adolescent >2 years of age as overweight if the BMI is ≥85th percentile but < 95th percentile for age and sex, as obese if the BMI is ≥95th percentile, and as extremely obese if the BMI is ≥120% of the 95th percentile or ≥35 kg/m2 .
1.3 We suggest that clinicians take into account that variations in BMI correlate differently to comorbidities according to race/ethnicity and that increased muscle mass increases BMI.
1.4 We suggest calculating, plotting, and reviewing a child's or adolescent's BMI percentile at least annually during well-child and/or sick-child visits. (Ungraded Good Practice Statement)
1.5 We suggest that a child < 2 years of age be diagnosed as obese if the sex-specific weight for recumbent length is ≥97.7th percentile on the World Health Organization (WHO) charts, as US and international pediatric groups accept this method as valid.
1.6 We recommend against routine laboratory evaluations for endocrine etiologies of pediatric obesity unless the patient's stature and/or height velocity are attenuated (assessed in relationship to genetic/familial potential and pubertal stage).
1.7 We recommend that children or adolescents with a BMI of >85th percentile be evaluated for potential comorbidities (see Table 2 and Fig. 1).
1.8 We recommend against measuring insulin concentrations when evaluating children or adolescents for obesity.

2.0 Genetic obesity syndromes

2.1 We suggest genetic testing in patients with extreme early onset obesity (before 5 years of age) and that have clinical features of genetic obesity syndromes (in particular extreme hyperphagia) and/or a family history of extreme obesity.

3.0 Prevention of obesity

3.1 We suggest that clinicians promote and participate in the ongoing healthy dietary and activity education of children and adolescents, parents, and communities, and encourage schools to provide adequate education about healthy eating.
3.2 We recommend that clinicians prescribe and support healthy eating habits such as:
- avoiding the consumption of calorie-dense, nutrient-poor foods (e.g., sugar-sweetened beverages, sports drinks, fruit drinks, most "fast foods" or those with added table sugar, high-fructose corn syrup, high-fat or high sodium processed foods, and calorie-dense snacks)
- encouraging the consumption of whole fruits rather than fruit juices.

3.3 We recommend that children and adolescents engage in at least 20 minutes, optimally 60 minutes, of vigorous physical activity at least 5 days per week to improve metabolic health and reduce the likelihood of developing obesity.
3.4 We suggest fostering healthy sleep patterns in children and adolescents to decrease the likelihood of developing obesity due to changes in caloric intake and metabolism related to disordered sleep.
3.5 We recommend balancing unavoidable technology related screen time in children and adolescents with increased opportunities for physical activity.
3.6 We suggest that a clinician's obesity prevention efforts enlist the entire family rather than only the individual patient.
3.7 We suggest that clinicians assess family function and make appropriate referrals to address family stressors to decrease the development of obesity.
3.8 We suggest using school-based programs and community engagement in pediatric obesity prevention.
3.9 We recommend using comprehensive behaviour changing interventions to prevent obesity. Such programs would be integrated with school- or community-based programs to reach the widest audience.
3.10 We recommend breast-feeding in infants based on numerous health benefits. However, we can only suggest breast-feeding for the prevention of obesity, as evidence supporting the association between breast-feeding and subsequent obesity is inconsistent.

4.0 Treating obesity

Lifestyle: general considerations
4.1 We recommend that clinicians prescribe and support intensive, age-appropriate, culturally sensitive, family-centered lifestyle modifications (dietary, physical activity, behavioral) to promote a decrease in BMI.
4.2 We recommend that clinicians prescribe and support healthy eating habits in accordance with the following guidelines of the American Academy of Pediatrics and the US Department of Agriculture:
- decreased consumption of fast foods
- decreased consumption of added table sugar and elimination of sugar-sweetened beverages
- decreased consumption of high-fructose corn syrup and improved labelling of foods containing high-fructose corn syrup
- decreased consumption of high-fat, high sodium, or processed foods
- consumption of whole fruit rather than fruit juices
- portion control education
- reduced saturated dietary fat intake for children and adolescents >2 years of age
- US Department of Agriculture recommended intake of dietary fiber, fruits, and vegetables
- timely, regular meals, and avoiding constant "grazing" during the day, especially after school and after supper
- recognizing eating cues in the child's or adolescent's environment, such as boredom, stress, loneliness, or screen time
- encouraging single portion packaging and improved food labelling for easier use by consumers. (Ungraded Good Practice Statement)

4.3 We recommend that clinicians prescribe and support the reduction of inactivity and also a minimum of 20 minutes of moderate to vigorous physical activity daily, with a goal of 60 minutes daily, all in the context of a calorie-controlled diet.
4.4 We suggest that clinicians encourage and support patients to limit non-academic screen time to 1 to 2 hours per day and decrease other sedentary behaviors, such as digital activities.
4.5 We suggest that the health care team identify maladaptive rearing patterns related to diet and activity and educate families about healthy food and exercise habits.
4.6 We suggest that the health care team probe for and diagnose unhealthy intra-family communication patterns and support rearing patterns that seek to enhance the child's or adolescent's self-esteem.
4.7 We suggest that the health care team evaluate for psychosocial comorbidities and prescribe assessment and counseling when psychosocial problems are suspected.
4.8 We suggest pharmacotherapy for children or adolescents with obesity only after a formal program of intensive lifestyle modification has failed to limit weight gain or to ameliorate comorbidities. We recommend against using obesity medications in children and adolescents <16 years of age who are overweight but not obese, except in the context of clinical trials.
4.9 We suggest that Food and Drug Administration (FDA)–approved pharmacotherapy for obesity be administered only with a concomitant lifestyle modification program of the highest intensity available and only by clinicians who are experienced in the use of anti-obesity agents and are aware of the potential for adverse reactions.
4.10 We suggest that clinicians should discontinue medication and re-evaluate the patient if the patient does not have a .4% BMI/BMI z score reduction after taking anti-obesity medication for 12 weeks at the medication's full dosage.
4.11 We suggest bariatric surgery only under the following conditions:
- the patient has attained Tanner 4 or 5 pubertal development and final or near-final adult height, the patient has a BMI of >40 kg/m2 or has a BMI of >35 kg/m2 and significant, extreme comorbidities
- extreme obesity and comorbidities persist despite compliance with a formal program of lifestyle modification, with or without pharmacotherapy
- psychological evaluation confirms the stability and competence of the family unit [psychological distress due to impaired quality of live (QOL) from obesity may be present, but the patient does not have an underlying untreated psychiatric illness]
- the patient demonstrates the ability to adhere to the principles of healthy dietary and activity habits
- there is access to an experienced surgeon in a pediatric bariatric surgery center of excellence that provides the necessary infrastructure for patient care, including a team capable of long term follow-up of the metabolic and psychosocial needs of the patient and family.
4.12 We suggest against bariatric surgery in preadolescent children, pregnant or breast-feeding adolescents (and those planning to become pregnant within 2 years of surgery), and in any patient who has not mastered the principles of healthy dietary and activity habits and/or has an unresolved substance abuse, eating disorder, or untreated psychiatric disorder.

TABLE 1. Screening tests for the more common obesity comorbidities	
Comorbidity	Tests and interpretation
Prediabetes Impaired fasting plasma glucose (verify fasting status) Impaired glucose tolerance (if OGTT is used)	HbA1c 5.7% to 6.5% Fasting plasma glucose ≥100 but ≤126 mg/dl Two-hour plasma glucose > 140 but <200 mg/dl
Diabetes mellitus	Fasting plasma glucose ≥126 mg/dl, or Two-hour plasma glucose ≥200 mg/dl, or In a patient with classic symptoms of hyperglycemia, a random plasma glucose of ≥200 mg/dL
Dyslipidemia	Fasting (12–14 h) lipids Triglycerides (mg/dL): 0–9 y: <75 (acceptable), 75–99 (borderline high), ≥100 (high); 10–19 y: 90 (acceptable), 90–129 (borderline high), ≥130 (high) LDL cholesterol (mg/dL): <110 (acceptable), 110–129 (borderline high), ≥130 (high) Total cholesterol (mg/dL): <170 (acceptable), 170–199 (borderline high), ≥200 (high) HDL cholesterol (mg/dL): <40 (low), 40–45 (borderline low), >45 (acceptable) Non–HDL cholesterol (mg/dL) (can be non-fasting): <120 (acceptable), 120–144 (borderline high), ≥145 (high)
Hypertension	3–11 y: BP >90th percentile to <95th percentile = prehypertension BP ≥95th percentile to <99th percentile + 5 mm Hg = stage 1 HTN BP ≥99th percentile + 5 mm Hg = stage 2 HTN 12–17 y: BP of >90th percentile to <95th percentile or >120/80 = prehypertension BP ≥ 95th percentile to <99th percentile + 5 mm Hg = stage 1 HTN BP ≥ 99th percentile + 5 mm Hg = stage 2 HTN 18 to 21 y: BP ≥120/80 to 139/89 mm Hg = prehypertension BP ≥140/90 to 159/99 mm Hg = stage 1 HTN BP ≥160/100 to 179/109 mm Hg = stage 2 HTN BP >180/110 mm Hg = stage 3 HTN
NAFLD	ALT >25 U/L (boys) and >22 U/L (girls)
PCOS	Free and total testosterone and SHBG, per Endocrine Society PCOS guidelines
Obstructive sleep apnea	If positive history, refer to pulmonary for nocturnal Polysomnography, and if not available, overnight oximetry
Psychiatric	If positive history, refer to mental health specialist

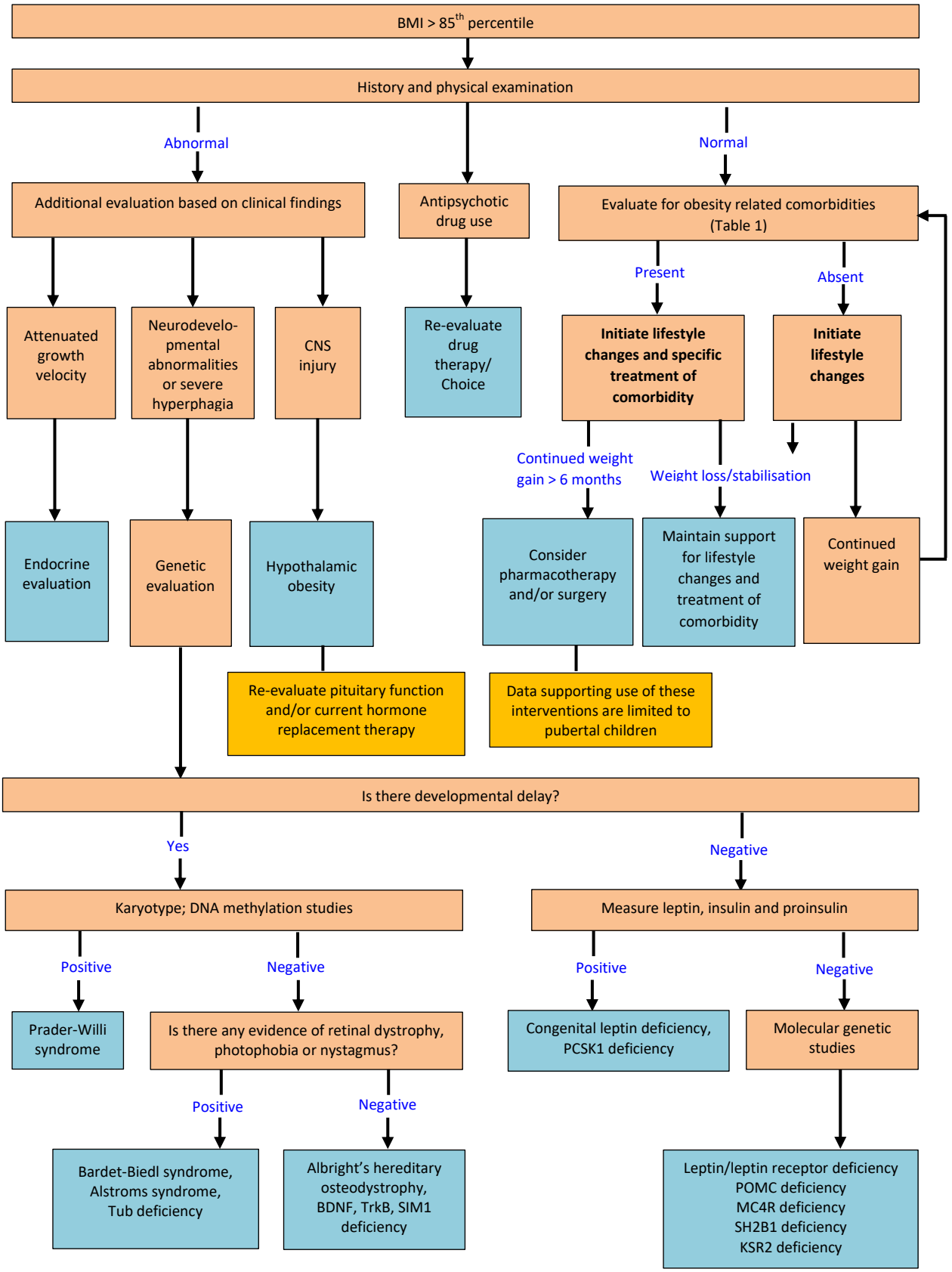

Fig 1: Algorithm for diagnosis and management of pediatric obesity

Infantile hypophosphatasia: a case report

Dr Shaila M Sankeshwar, Dr Vani H N, Dr Raghupathy P. Indira Gandhi Institute of Child Health, Bangalore

A 7 month old male infant, born to non-consanguineous parents, presented with the history of failure to gain weight since birth, while being exclusively breastfed. There was no significant antenatal history (normal USG scans reported) and the baby was born at term, by normal vaginal delivery with birth weight of 2.3 kg. Meconium stained liquor was recorded at birth, but no NICU admission was needed for respiratory distress. There was no history of other predisposing illnesses for failure to thrive, such as recurrent chest infections/ loose stools/ persistent vomiting/ constipation/ polyuria. Urine output was documented to be 2.5 ml/kg/hour. The infant had been admitted elsewhere once at 2 months of age. He weighed 2.45 kg then and was found to have dehydration, acute renal failure and anemia, which resolved with treatment. Weight at subsequent visits was always found to be constant, viz., 3 kg at 3.5 months and also at present. Oral vitamin D drops was being given as 400 IU/day since 2 months of age. The infant had severe delayed motor milestones with generalized floppiness and a floppy neck, but social and language milestones were appropriate for age. The mother had medical termination of her first pregnancy as her antenatal scan done at 3 months of gestation revealed a short limbed fetus. Her second pregnancy ended in spontaneous abortion at 2 months of gestation.

Examination revealed a severely malnourished child, active in his mother's lap, with vitals being stable, but signs of dehydration present. Weight: 3 kg, length: 49.5 cm, HC: 36 cm. Bulging anterior fontanelle and wide open sagittal and metopic sutures were noted. Clinical signs of rickets in the form of widened wrists and rickety rosary were present. Mild hepatosplenomegaly was also noted. Genitalia: normal.

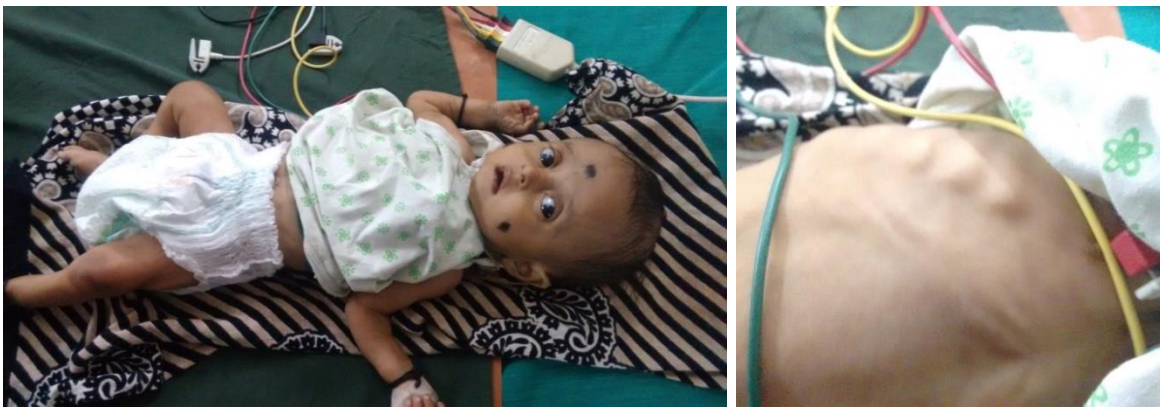

Figure 1: Gross hypotonia in a 7 month old infant with wrist widening and rickety rosary

Investigations:

Peripheral blood smear: Normocytic normochromic anemia with PCCS +, occasional reactive/ atypical lymphocytes seen. Ammonia/ lactate: 72µmol/l / 15.1 mg/dl. Serum sodium/ potassium/ chloride: 131 / 4.4 / 103 mEq/L; Urea/ creatinine: 46/ 0.91 mg/dl; blood gases and thyroid function tests were normal. Serum protein / albumin: 7.5/ 4.3 g/dl; SGOT / SGPT: 46/ 10 IU/L; CRP: < 6 mg/l. 24 hour urinary calcium: 7 mg/ kg/ day. USG abdomen: s/o medullary nephrocalcinosis. Radiographic findings are shown in Figure 2.

Table 1: Biochemical parameters

Date	S. Calcium (mg/dl)	S. Phosphorus (mg/dl)	ALP	S. Mg (mg/dl)	25OHvitD (ng/ml)	PTH (pg/ml)	Hb (g/dl)
12/9/16	11.6	-	220	1.75	-	-	6.3
16/9/16	13	-	-	-	-	-	-
Present admission							
16/2/17	17.1	3.9	11	-	33.82	3	6.7
19/2/17	17.5	3.1	16	-	-	-	-
20/2/17	17.4	2.9	11	-	-	-	10.6
21/2/17	15.6	3.3	-	-	-	-	-
22/2/17	16.3	2.8	17	-	-	-	-
23/2/17	16.7	3.2	16	-	-	-	-
24/2/17	16.6	2.5	17	-	-	-	-
25/2/17	15.0	2.9	7	-	-	-	-
26/2/17	14.0	2.7	12	-	-	-	-
2/3/17	13.5	2.5	15	-	-	-	-

The infant is being treated with IV fluids and Inj. furosemide 1 mg/kg/dose thrice daily. There has been improvement in the form of normalization of serum calcium only, but no clinical improvement in weight and activity. He was started with compliment feedings during the hospital stay.

Discussion: Hypophosphatasia (HPP) is a rare inborn error of metabolism caused by mutations in the gene encoding tissue non-specific alkaline phosphatase (TNSALP). It affects the skeleton and teeth mainly (1). ALP is required for breakdown of pyrophosphate (a mineralization inhibitor). It also provides inorganic phosphate required for bone formation.

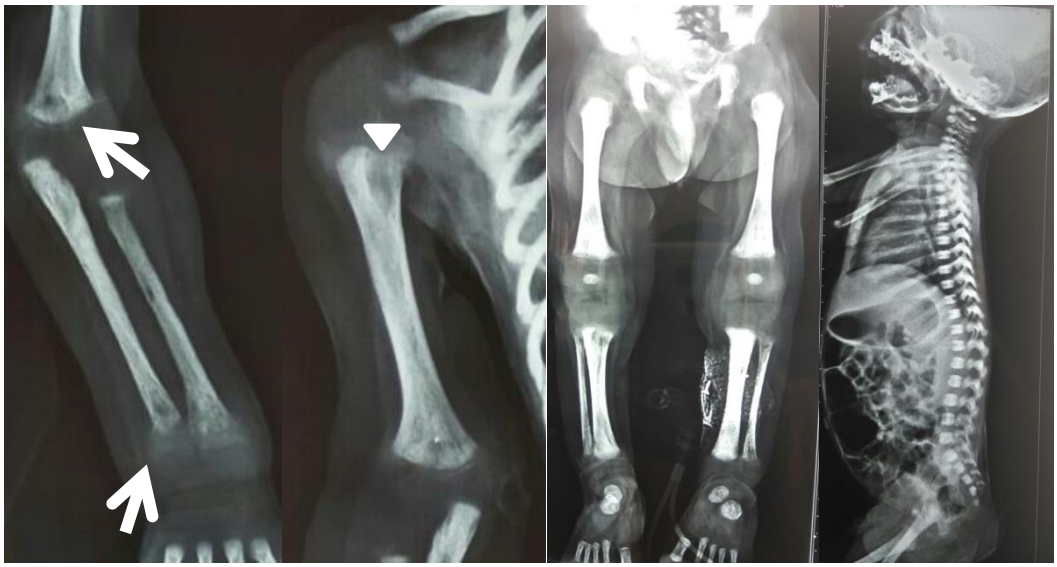

Figure 2: Radiographs showing poorly mineralized bones, evidence of rickets (arrows), flared metaphyses, poorly ossified epiphyses, focal bony defects of the metaphyses resembling radiolucent “tongues” (arrow heads), short vertebral bodies with lack of neural arches.

The incidence of HPP varies, with severe forms being approximately 3.3/million live births; whereas the incidence of milder forms is 1 in 6-7,000 (1). HPP is inherited as autosomal dominant or recessive manner.

HPP has a wide clinical spectrum, with severe forms presenting in utero as stillbirth without mineralized bone, to milder forms with either fracture of long bones or loss of teeth. Six types of HPP are usually described, based on age at diagnosis and severity of features: perinatal (severe), perinatal (benign), infantile, childhood (juvenile), adult, and odonto HPP (2). Hypercalcemia is common in the neonatal and infantile forms. HPP should be suspected in infantile rickets without elevated ALP activity, with other features of failure to thrive, wide open fontanelles and sutures, blue sclerae, hypotonia, and bowing of long bones. Vitamin B6-dependent seizures can also be observed as pyridoxal phosphate (PLP) is a substrate for ALP. Biochemical investigations will reveal low ALP, high/normal calcium and vitamin D levels. Urinary levels of phosphoethanolamine and pyridoxal 5' phosphate will be elevated.

Radiological signs (Fig. 2) vary with age and type, and include osteopenia, rickets, brachycephaly, flail chest, flared metaphyses, focal bony defects of the metaphyses resembling radiolucent "tongues", metatarsal stress fractures, alveolar bone loss, and osteomalacia, with lateral pseudofractures (in adults) (3).

A multidisciplinary approach is required. Treatment consists of respiratory support (even requiring ventilator support) and managing hypercalcemia/ hypercalciuria. Bisphosphonates should not be used as they accumulate in the absence of ALP, and further worsen the condition. Neurosurgical intervention is required in case of craniosynostosis. Other modalities include treatment of seizures with vitamin B6 when indicated, and specific enzyme replacement therapy. Asfotase alfa is a recombinant ALP administered as subcutaneous injections (either as 1 mg/kg six times per week or 2 mg/kg thrice weekly) (4). It was mainly tried in the perinatal and infantile forms. Enzyme replacement therapy improves skeletal healing and mineral content, pulmonary status, and overall physical activity. Whyte et al reported 77% survival in infants who suffered convulsions, also reduction of mortality from 97% to 60% in cases presenting later in infancy (4).

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Pedendoscan

Dr Sachin Mittal, Consultant Endocrinologist, Fortis Hospital, Chandigarh

Growth

The influence of GH treatment on glucose homeostasis in girls with Turner Syndrome: a 7 year study. Baronio F et al. J Clin Endocrinol Metab. 2017;102(3):878-83.

This was a longitudinal study of the insulin sensitivity (HOMA-S), insulin secretion (insulinogenic index - IGI) and capacity of β-cells to adapt to changes in insulin sensitivity (oral disposition index- ODI) in 104 girls with Turner syndrome (TS) undergoing GH treatment (9.1 ± 3.4 years) for a median period of 7.2 years. Every year the children underwent an oral glucose tolerance test (OGTT), which was used to calculate HOMA-S, IGI and ODI. No significant changes were observed in term of HOMA-S, IGI or ODI. The study showed no negative influence of GH treatment on insulin sensitivity and on beta cell secretory capacity in girls with TS.

The Influence of Growth Hormone Treatment on Glucose Homeostasis in Growth Hormone-Deficient Children: A Six-Year Follow-Up Study. Baronio F et al. Horm Res Paediatr. 2016;86(3):196-200.

In this longitudinal study the same group looked at HOMA-S, IGI and ODI in 99 GHD (62 male, 37 female; age 8.9 ± 3.5 years) children on GH treatment for a median period of 6 years (range 1.5-16.2). Every year, patients underwent an oGTT to calculate the HOMA-S, IGI and ODI. Although HOMA-S remained unchanged, an increase in IGI and ODI was observed, becoming significant after 6 years of treatment. These results suggested a positive influence of GH treatment on the β-cell secretory capacity in children with GH deficiency.

Thyroid

Characterization of Thyroid Abnormalities in a Large Cohort of Children with Down Syndrome (DS). Pierce MJ, LaFranchi SH, Pinter JD. Horm Res Paediatr. 2017;87 (3):170-78.

Thyroid abnormalities were assessed in 508 children with DS. 120 (24%) had a thyroid-related diagnosis, the majority having elevated thyrotropin treated with levothyroxine. A Kaplan-Meier estimate projects that 50% have a thyroid disorder by adulthood, with 20% of hypothyroidism diagnosed before the age of 6 months. The authors concluded that thyroid disease in DS is more common and occurs earlier than in the general population, and is often transient. Thyroid disease is unrelated to gender, obesity, or other comorbidities. An additional screen for thyroid disease between the newborn screen and the 6-month well-child visit will detect early cases of hypothyroidism in babies with normal newborn screen.

Early Maternal Thyroid Function During Gestation Is Associated With Fetal Growth, Particularly in Male Newborns. Vrijkotte TG, et al. J Clin Endocrinol Metab 2017;102 (3): 1059-1066.

Data was taken from a community-based cohort study of pregnant women living in Amsterdam (Amsterdam Born Children and Their Development study). TSH and fT4 levels were determined during the first prenatal screening at median 13 weeks. After adjustments, 1 pmol/L increase in maternal fT4 levels was associated with a reduction in birth weight of 33.7 g ($P < 0.001$) in male newborns and 16.1 g ($P < 0.05$) in female newborns. Maternal subclinical hypothyroidism in early pregnancy (TSH > 2.5 mU/L, 7.3%) was associated with increased odds for LGA in male newborns (OR, 1.95; 95% CI, 1.22 to 3.11). The authors concluded that maternal fT4 in early pregnancy was observed to be inversely associated with birth weight, with a stronger relationship in males. Male infants also had increased odds for LGA in mothers with subclinical hypothyroidism.

Diabetes

Initiation of insulin pump therapy (IPT) in children at diagnosis of type 1 diabetes resulted in improved long-term glycemic control. Lang EG et al. Pediatr Diabetes. 2017;18(1):26-32.
The study sought to determine if early initiation of IPT in children with type 1 diabetes (T1D) leads to improved glycemic control and quality of life (QOL) compared with later introduction of IPT. Data on HbA1c, rate of severe hypoglycemia, and diabetic ketoacidosis (DKA) was collected retrospectively over a 48-month period. HbA1c measurements were lower in the early pump group (EPG) (mean age 12.6, n = 38, 23 male) compared to the later pump group (LPG) (mean age 13.1yr, n =37, 19 male). There was no significant difference in the severe hypoglycemia, episodes of DKA and quality of life (QOL) between the groups. The authors concluded that initiation of IPT at diagnosis of T1D resulted in consistently lower HbA1c with no apparent change in hypoglycemia, DKA, or QOL.

Glycated hemoglobin A1c as a risk factor for severe hypoglycemia in pediatric type 1 diabetes. Karges B et al. Pediatr Diabetes. 2017;18(1):51-58.
The association between HbA1c levels and severe hypoglycemia (defined as requiring assistance from another person) or hypoglycemic coma (loss of consciousness or seizures) was analyzed in children and adolescents with type 1 diabetes from the DPV Diabetes Prospective Follow-up in Germany and Austria in 1995-2003 (n = 15 221) and 2004-2012 (n = 22 318). Mean adjusted rates of severe hypoglycemia and hypoglycemic coma decreased from 19.18 and 4.36 per 100 patient-years to 15.01 and 2.15, respectively ($p < 0.001$). The relative risk (RR) for severe hypoglycemia and hypoglycemic coma per 1% lower HbA1c decreased from 1.22 to 1.06 and from 1.27 to 1.04, respectively. The authors concluded that in later years, low HbA1c has become a minor risk factor for severe hypoglycemia and coma in pediatric T1D.

Genetic and environmental factors affect the onset of type 1 diabetes mellitus. Altobelli E et al. Pediatr Diabetes. 2016;17(8):559-66.
To investigate T1DM time trends from 1989 to 2008 and try to establish whether breast/bottle feeding, a family history of diabetes, and childhood infectious diseases influence age at onset, the data from population-based registry of childhood diabetes of Abruzzo (central Italy) was analysed. Overall standardized incidence rates (SIR) increased by 73.38% from 8.94 (1989-1993) to 15.50 (2004-2008). A rising trend was found in all age groups. Early T1DM onset was related to mixed feeding and a family history of T1DM, whereas multiple infections delayed age at onset.

Obesity

The association of weight loss and cardiometabolic outcomes in obese children: Systematic review and meta-regression. Rajjo T et al. J Clin Endocrinol Metab. 2016;101(12):4764-68.
To identify the degree of reduction in excess body weight associated with cardiometabolic changes (lipid panel, liver function tests, systolic blood pressure (SBP), diastolic blood pressure, glycosylated hemoglobin, and fasting blood glucose) in overweight and obese children, 42 studies (37 randomized controlled trials and five cohorts) including 3807 children (mean age, 12.2 years; weight, 74.7 kg; and BMI, 31.7 kg/m^2 were reviewed. A 1 mm Hg decrease in SBP was significantly associated with a decrease of 0.16 kg/m^2 ($P = .04$) in BMI. A 1 mg/dL increase in HDL was significantly associated with a 0.74 kg decrease in weight ($P = .02$). A 1 mg/dL decrease in triglycerides was significantly associated with a 0.1 kg decrease in weight ($P = .03$). The authors concluded that weight reduction in children is associated with significant changes in several cardiometabolic outcomes, particularly HDL, SBP, and triglycerides.

Obesity

Birth weight in different etiologies of disorders of sex development. Poyrazoglu S et al. J Clin Endocrinol Metab. 2017;102(3):1044-50.

It is well established that boys are heavier than girls at birth. Although the cause of birth weight (BW) difference is unknown, it has been proposed that it could be generated from prenatal androgen action. The aim of the current study was to determine the BW of children with disorders of sex development (DSD) of different etiologies and to evaluate the effects of androgen action on BW. Data regarding diagnosis, BW, gestational age, karyotype, and concomitant conditions collected from the International Disorders of Sex Development (I-DSD) Registry for a total of 533 (75% 46,XY, and 25% 46,XX) cases was analysed. Eighty cases (15%) were born small for gestational age (SGA). Frequency of SGA was higher in the 46,XY group (17.8%) than in the 46,XX (6.7%) group (P = 0.001). Mean BW standard deviation scores of cases with androgen excess and androgen deficiency were similar to normal children with the same karyotype.

The authors concluded that BW dimorphism is unlikely to be explained by fetal androgen action per se. 46,XY DSDs due to nonspecific disorders of under-masculinization are more frequently associated with fetal growth restriction, SGA, and concomitant conditions.

Photo Quiz

Dr Vani H N, Dr Raghupathy P. Indira Gandhi Institute of Child Health, Bangalore

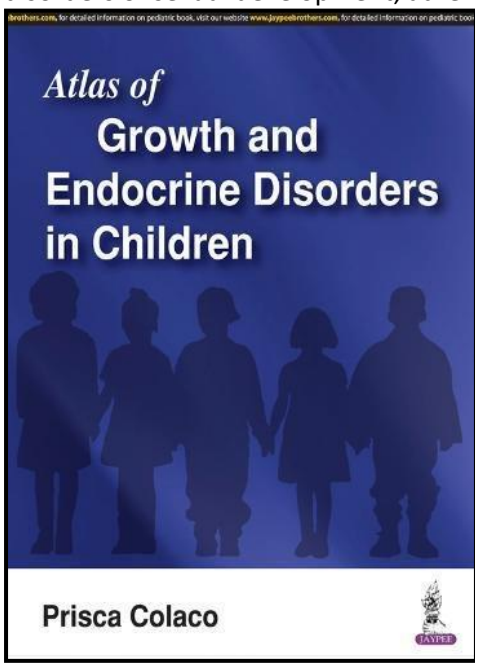
A 14 year old boy born to a second degree consanguineous couple presented with short stature. His 10 year old sister had similar complaints. On examination, the boy had disproportionate short stature with rhizomelic limb shortening. His skeletal survey had typical findings. What are the findings, and what is the diagnosis?

Identify the radiological abnormalities and mention the most likely diagnosis.

Book Review: "Atlas of Growth and Endocrine Disorders in Children"

Dr Tushar Godbole, Pediatric Endocrinologist, Nashik, Maharashtra

The 'Atlas of Growth and Endocrine Disorders', authored by our senior ISPAE member **Professor Prisca Colaco** (MGM Medical College, Navi Mumbai), was recently published under the banner of Jaypee. It is a single convenient volume, printed fully in color, with clinical images of all the rare disorders, radiographs and nuclear scans. The illustrative tables and charts also help clinicians to make correct diagnoses easily. The book has 24 chapters, broadly divided into stature, puberty, disorders of sexual development, adrenals, thyroid, sugar and calcium homeostasis and rickets.



The chapter on type 1 diabetes discusses in a very precise manner the insulin regims, home based glucose monitoring and complications of diabetes. The dedicated chapters on gynecomastia, micropenis and thyroid nodules deserve a special mention. The book also mentions in brief the management of various endocrine conditions, including the drug doses and available drug preparations in Indian settings.

Fluent language, easily readable formats are the merits of this book. This book is targeted towards practising paediatricians and postgraduates. It is surely recommended as a must-read, in addition to the usual textbooks of pediatric endocrinology, and also as a ready reckoner to a busy pediatrician.

The book is available at all major medical book stores across the country, and also online.

Pediatric Endocrine Workshop, PEDICON 2017

Dr Raghupathy P, Dr Vani H N, IGICH, Bangalore

A pediatric endocrine workshop was organised on January 18th as a part of the 54th Annual IAP conference, PEDICON 2017, in Bangalore. Dr Raghupathy was the National Coordinator and Dr Vani the local coordinator for the workshop. There was an overwhelming response, with 70 participants (mostly practicing pediatricians and postgraduates with interest in endocrinology) from all over India.

Eminent experts from all over India were the speakers. Pre-test questions were given to all the participants. Common endocrine topics like growth disorders, pubertal disorders, hypothyroidism, type 1 diabetes (T1D), DKA management and obesity were covered.

Dr Vaman Khadilkar and Dr Rahul Jahagirdar conducted hands on training on growth chart plotting and identifying different varieties of growth disorders. Dr Vijaya Sarathi spoke on pubertal disorders while Dr Santhosh Olety spoke on practical issues of T1D management, and Dr Ahila Ayyavoo on management of DKA. Dr Shaila Bhattacharyya discussed the approach to a child with disorder of sex development, while Dr Anjana Hulse discussed recent concepts in assessment, prevention and management of obesity. Dr Raghupathy P explained the importance of neonatal screening for congenital hypothyroidism, and Dr Riyaz spoke on diagnosis and management of juvenile hypothyroidism.

The post-test conducted at the end of the program showed a fair gain of knowledge on the discussed topics.

Endocrinology activities in Pedicon 2017

Dr Raghupathy P, Dr Vani H N, IGICH, Bangalore

A number of endocrine topics were discussed during PEDICON 2017 at Bangalore. In the CME on 'Adolescent Health' organized on 19.01.2017, Dr Raghupathy spoke on juvenile hypothyroidism and Dr Kumar Angadi (Gulbarga, Karnataka) spoke on type 2 Diabetes Mellitus.

The IAP-Endocrinology Chapter Symposium organized on 21.01.2017 focused on 'Examination and practical management of gonadal disorders'. Dr Preeti Singh (New Delhi) spoke on 'Examination of genitalia for puberty staging', while Dr Smita Koppikar from Mumbai spoke on 'Management of undescended testes'. The symposium on 'Treating disorders of and delay in puberty' was moderated by Dr Kavitha Bhat (Bangalore). Dr Rakesh Kumar (Chandigarh) spoke on 'Management of gynecomastia', Dr Reetha Gopinath (Pariyaram, Kerala) spoke on induction of puberty in boys, while Dr Abraham Paulose (Ernakulam, Kerala) spoke on induction of puberty in girls.

On 20.01.2017, a panel discussion on 'Approach to a patient of short stature' was conducted. The session was moderated by Dr Abhishek Kulkarni (Mumbai) and Dr Archana Arya (New Delhi) and Dr Shaila Bhattacharya (Bangalore) were the expert panelists.

On 21.01.2017, Dr Vaman Khadilkar (Pune) moderated a session on 'Obesity and Metabolic syndrome' in which Dr Ganesh Jevalikar (Gurugram) and Dr Archana Arya were the expert panelists. A panel discussion was also conducted on 'Precocious and delayed puberty' on 21.01.2017, moderated by Dr Garima Chawla (New Delhi). Dr Ahila Ayyahoo (Coimabatore), Dr Subrata Dey (Kolkatta) and Dr Raghupathy were the panelists.

On 21.02.2017, Dr Vijaya Sarathi spoke on 'Diagnosis and management of vitamin D resistant rickets' in the Mixed Bag section, and Dr Jayanthi, a gynecologist from Bangalore, on 'Adolescent PCOS'. A session on 'Nuclear Medicine in Endocrinology' was conducted by Dr Anurag Bajpai (Kanpur), Dr Riyaz (Thiruvanathapuram) and Dr Prashanth.

As part of the scientific program, many oral papers and e-papers on endocrinology were presented. In the oral paper section, the first place was awarded to Dr Garima Chawla for 'Growth hormone therapy in Turner syndrome-An Indian experience' and second place to Dr Ahila Ayyavoo for 'Management of a child with recurrent pancreatitis in early infancy due to severe hypertriglyceridemia', whereas in the e-papers section, the first place was awarded to Dr Satheesh and the second place to Dr Ahila Ayyavoo.

Answer to Photoquiz: Radiological findings: Cupping of metaphysis of proximal phalanges and cone shaped appearance of distal phalanx, lace like appearance in the iliac bone, central notching and dumbbell appearance of the spine.

Diagnosis: Dyggve Melanchior Clausen syndrome

Activities by ISPAE Members

PEDIATRIC ENDOCRINOLOGY SYMPOSIUM/WORKSHOP FOR POSTGRADUATES, PEP 2017
Dr Raghupathy P, Bangalore and Dr Amarnath Kulkarni, Hyderabad

A 2-day Symposium/ Workshop was held for postgraduates pursuing MD or DNB courses in Pediatrics, in Hyderabad on 18 and 19 March 2017. It was organized by Dr P Raghupathy and Dr Amarnath Kulkarni, under the auspices of ISPAE and Growth Hormone Research Society (GHRS). The objective was to provide teaching sessions in pediatric endocrinology, in preparation for postgraduate clinical and theory examinations. It was attended by 37 postgraduates from Telengana, Karnataka and Tamil Nadu. Interactive sessions were held, with clinically oriented case presentations by the attendees, discussed by the faculty members. Common topics in pediatric endocrinology were included, with practical sessions, MCQs, and OSCE. Drs Anurag Bajpai (Kanpur), Raghupathy, Sudha Rao (Mumbai), Shaila Bhattacharyya (Bangalore), Sarah Mathai (Vellore), Leenatha Reddy (Hyderabad), Hemchand Prasad (Chennai), Leena Priyambada (Hyderabad), M Srinivas (Hyderabad) and Suhasini (Hyderabad) were the faculty. The Telengana State Medical Council awarded 4 CME accreditation points to the participants.

Dr Santosh Olety, Pediatric Endocrinologist, Karnataka Institute of Endocrinology and Research, Bangalore

In celebration of World Health day 2016, theme "*Beat Diabetes*", the Karnataka Institute of Endocrinology and Research, Bangalore initiated a Diabetes Awareness Program in April 2016. The program includes visiting various schools to educate teachers and students through interactive sessions regarding healthy life styles and prevention of diabetes, also creating awareness about childhood diabetes and the need for schools to provide support to children with diabetes, to optimise their quality of life. A dozen schools in south Bangalore have been visited from April 2016 till date, with many more to go... The last one was held in January 2017.

On November 13th, a World Diabetes Day program for children with diabetes, organised in our Institute premises, was well attended by 50 children along with their parents. It was a fun filled day with educative activities such as building a food pyramid, blindfold tasks to make them realise the importance of protecting their sight with good glucose control, a balloon race for understanding team work and good communication skills, a drawing competition to express their life with diabetes, and parental networking to empower and build their confidence.




Hemchand K Prasad, Pediatric Endocrinologist, Mehta Children's Hospital, Chennai

A pediatric endocrine conference was conducted in Hotel Raintree under the auspices of IAP Chennai. The Organising Chairpersons were Prof P G Sundararaman, Prof P Venkataraman and Dr C V Ravisekar; Dr Hemchand KP was the Organising Secretary. The meeting was attended by 250 pediatricians from all over south India; all common problems in pediatric endocrinology were discussed. The first Pediatric Endocrinology Oration was held, and PGs made oral and poster paper presentations. Eminent national faculty were Prof P Raghupathy, Dr Vaman Khadilkar, Dr Anna Simon, Dr Anurag Bajpai, Dr GR Sridhar and Dr Archana Arya.


World Thyroid Day was celebrated on 11.2.2017. Twenty families with patients of congenital hypothyroidism (CH) participated; booklets on CH were released.

Dr Anurag Bajpai, Pediatric Endocrinologist, Kanpur

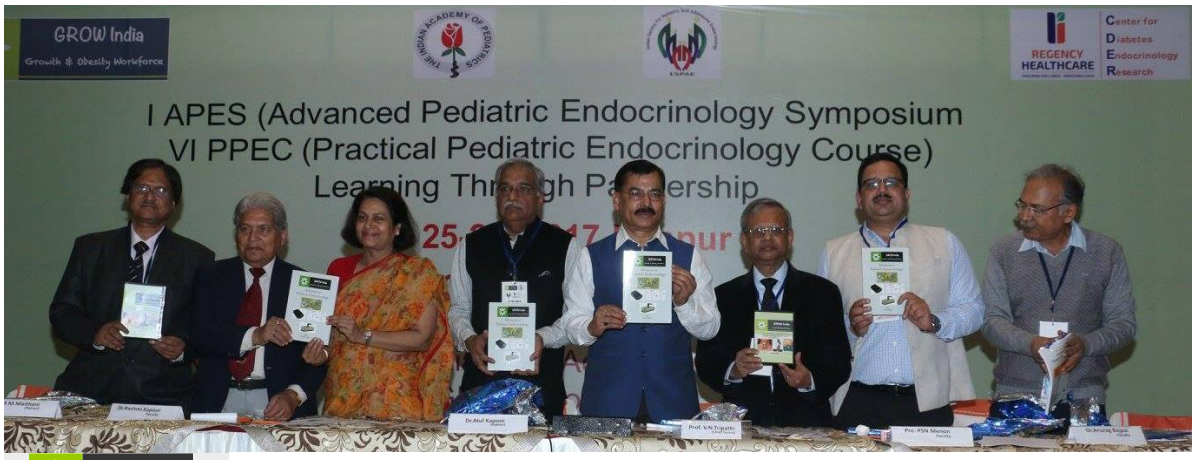
I Advanced Pediatric Endocrinology Symposium, Feb 25 2017, Kanpur

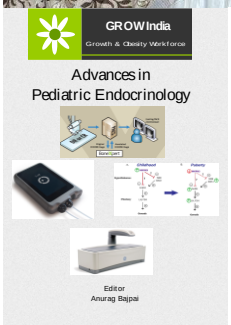
I Advanced Pediatric Endocrinology Symposium was organized under the auspices of GROW Society, Kanpur Academy of Pediatrics, ISPAE, and Regency Center for Diabetes, Endocrinology & Research on Feb 25 2017. The program was attended by 100 Pediatric Endocrinologists, Pediatric Endocrine Trainees, Endocrinologists, and Pediatricians with special interest in Endocrinology. Stalwarts in Pediatric Endocrinology from across the country including Prof. PSN Menon, Prof. P Raghupathy, Dr Subrata Dey, Dr Anuradha Khadilkar, Dr Raman Marwaha, Dr Smita Koppikar, Dr Meena Mohan, Dr Anjana Hulse, Dr Vijaya Sarathi, Dr Rakesh Kumar, Dr Hemchand Prasad, Dr Rishi Shukla and Dr Saurabh Uppal, dwelt on all aspects of pediatric endocrinology, including bone mineral disorders, Year in Review, insulin pumps, CGMS, bone age assessment, disorders of sexual development and type 2 diabetes.

VI Practical Pediatric Endocrinology Symposium (PPEC), Feb 26 2017, Kanpur

VI Practical Pediatric Endocrinology Course was organised by GROW Society in association with ISPAE, Kanpur Academy of Pediatrics, and Regency Center for Diabetes, Endocrinology & Research. The course, attended by 200 participants from across the country, focused on key aspects of pediatric endocrinology including growth, thyroid, puberty, calcium disorders and glucose disorders. Our eminent national faculty included Prof. PSN Menon, Prof. P Raghupathy, Dr Subrata Dey, Dr Raman Marwaha, Dr Sangeeta Yadav, Dr Smita Koppikar, Dr Yuthika Bajpai, Dr Vijaya Sarathi, Dr Anjana Hulse, Dr Abhishek Kulkarni, Dr Meena Mohan, Dr Rakesh Kumar, Dr Saurabh Uppal and Dr Rashmi Kapoor.

Book release "Advances in Pediatric Endocrinology"





Advances in Pediatric Endocrinology" was released as part of the I APES-VI PPEC at Kanpur. The book published by GROW Society Publications takes a bird's eye view of the state of art in pediatric endocrinology, including GH therapy, Turner syndrome, calcium and vitamin D, puberty, adrenal disorders, PCOS, disorders of sexual development, assessment of bone mineralization and of bone age, and diabetes in children.

Dr J Dhivyalakshmi, Consultant Paediatric Endocrinologist, Sri Ramachandra Medical College & RI, Chennai

Between 14.11.2016 and 26.11.2016, a childhood obesity awareness camp was conducted at Sri Ramachandra Medical College (SRMC), Chennai. Free pediatric endocrinologist consultation and nutritional counselling were provided. Screening for metabolic syndrome was done at concessional prices. The camp ended with a fitness therapy session by a fitness specialist. Around 70 children participated in the camp.

On 26th November 2016, "Growth Module for Postgraduates" was organised at SRMC, Chennai. Around 40 postgraduates from various institutes participated in the program. Dr Shriram Mahadevan (Endocrinologist, SRMC), Dr Adylene Reena (Endocrinologist, SRMC), and Dr Shruti Chandrasekar (Endocrinologist, Global Hospital, Chennai) were the speakers. The program included lectures on growth charts, approach to short stature, and idiopathic short stature. Lecture sessions were followed by small group discussions on case scenarios, and diagnosing various growth disorders with growth charts. All sessions were highly appreciated by the postgraduates.

On 4th March 2017, "Type 1 Diabetic Children meet" was organised at SRMC. Twenty children with type 1 diabetes attended it. Program highlights were diabetic education, demonstration of recipes with carbohydrate counting, diabetic fun activity booklet (colouring book and activity book) and Diaversary medals. Insulin pens, insulin syringes and 10 glucometer strips were provided free of cost. Snacks and lunch for the children and parents were also provided.

Publications by ISPAE members

Dr Devi Dayal, Pediatric Endocrinology & Diabetes Unit, Advanced Pediatric Center, PGIMER, Chandigarh

1. Dayal D, Prasad R, Kumar R, Sodhi KS, Bhattacharya A, Didi M. Clinical and morphological characteristics of ectopic thyroid gland in children: A series of 24 patients from Northwest India. *Pediatr Pol*. 2016;92(1):17-21.
2. Kamble R, Sodhi KS, Thapa BR, Saxena AK, Bhatia A, Dayal D, Khandelwal N. Liver acoustic radiation force impulse (ARFI) in childhood obesity: comparison and correlation with biochemical markers. *J Ultrasound.* 2017;20(1):33-42.
3. Dayal D, Prasad R, Bhunwal S, Kumar R, Kumar RM, Sodhi KS. Spectrum of extrathyroidal congenital malformations in a cohort of North Indian children with permanent primary congenital hypothyroidism. *Thyroid Res Pract.* 2017;14:8-11.
4. SK Angurana, J Muralidharan, D Dayal, J Ismail. Status Dystonicus in a Child with Familial Idiopathic Hypoparathyroidism. *Indian J Pediatr*. 2017. doi:10.1007/s12098-017-2295-31-3 (epub).

Dr Satendra K Multani, Consultant Endocrinologist, Prime Medical Center , Salh Eldin Oud Al Muteena, Dubai

Sridhar SB, Rao PG, Multani SK, Jain M. Assessment of prevalence of hypovitaminosis D in multiethnic population of the United Arab Emirates. J Adv Pharm Technol Res. 2016 Apr-Jun; 7(2): 48–53.

Dr Deep Dutta, Consultant & Head, Department of Endocrinology, Diabetology & Metabolic Disorders, Venkateshwar Hospital, Dwarka, New Delhi

Sharma LK, Sharma N, Gadpayle AK, Dutta D. Prevalence and predictors of hyperprolactinemia in subclinical hypothyroidism. Eur J Intern Med. 2016 Nov; 35:106-110.

Dr Santosh Olety, Pediatric Endocrinologist, Karnataka Institute of Endocrinology and Research, Bangalore

Olety SS, Vellakampadi D. TRMA syndrome (thiamine-responsive megaloblastic anaemia): An example of rare monogenic diabetes: is thiamine a magic pill for anaemia and diabetes? Int J Diabetes Dev Ctries. 2016;36:389.

Upcoming Activities

Dr Raghupathy P, IGICH, Bangalore

Growth Hormone Research Society, India, has organised "Growth Symposium 2017 - Recent trends in management of growth disorders" to be held in Bengaluru on **22nd and 23rd July 2017.** The symposium will focus on the recent Paediatric Endocrine Society Guidelines, the new country specific growth standards that were recently published, along with interesting panel discussions and case discussions with leading national and regional faculty from India, Egypt and UAE.

5th Biennial Meeting

Indian Society for Pediatric and Adolescent Endocrinology (ISPAE)

November 24th to 26th, 2017

Coimbatore, India

Organising Chairperson: Prof P. Raghupathy; email – drp.raghupathy@gmail.com

Organising Secretary: Dr Ahila Ayyavoo; email – ispae2017coimbatore@gmail.com

ahila.ayyavoo@gmail.com

Registration Form

Title: Prof/ Dr/ Ms/ Mr ISPAE Number: _____

First Name: _____

Last Name: _____

Institution: _____

Affiliation: _____

Correspondence Address:

_____

_____

_____

Mobile numbers: _____

Email id: _____

Accompanying persons if any:

_____

Address for Communications:
Dr Ahila Ayyavoo
Department of Pediatrics & Pediatric Endocrinology,
G. Kuppuswamy Naidu Memorial Hospital,
Pappanaickenpalayam,
Coimbatore 641037, India
Phone +91 422 4305372/ +91 422 4305261

Registration fees for the Biennial Meeting of ISPAE 2017
November 24 – 26, 2017

Dates	ISPAE members	Non-members	Students & Accompanying persons	Registration for 26/11/2017 (single day) - Paediatricians and General Practitioners
Upto 30/04/2017	Rs 4500	Rs 5500	Rs 3000	Rs 1500
01/05/2017 – 30/06/2017	Rs 5500	Rs 6500	Rs 3500	Rs 1500
01/07/2017 – 31/10/2017	Rs 6000	Rs 7000	Rs 4000	Rs 1750
Nov 1st onwards	Rs 7000	Rs 8000	Rs 4500	Rs 2000
International delegates: Rs 7000 (up to 31/10/2017)				
Spot Registration: Rs 8000				

From 1st of April, please add 14.5% service tax to your registration fees.

Bank details:
Corporation Bank,
Branch: GKNM Hospital Branch, Coimbatore 641037
Account number: 510101000638543
Account holder: ISPAE 2017
IFSC Code: CORP0000650

Cheques and DDs can be drawn in favour of "ISPAE 2017". DDs will be payable at Coimbatore.

Amount of money paid: INR_____

Details of mode of money transfer:
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