NEWSLETTER
MARCH 2024
Volume 28 - Issue 1

CAPE News

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

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Next Issue: Bone and Calcium disorders

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EDITOR'S MESSAGE

Dear Readers,

We are pleased to release the first issue of 2024, focused on obesity and metabolic disorders, that has emerged as a global problem affecting children and young adults. India faces the dual burden of malnutrition in the form of under-nutrition and over-nutrition/ obesity. As per the Comprehensive National Nutrition Survey, the countrywide prevalence of overweight and obesity is 1.6% (0-4 years) to 4.8% (10-19 years).

With this background, we present a compendium of reviews and learning points -a summary of the IAP guidelines on obesity, interesting brief reviews on diagnosis and management of obesity, and learning pearls summarized from recently conducted scientific deliberations. We hope this information will be found useful.

There is 'tech news' for our readers. The Trainees' Section is now in an online version. You are requested to scan the QR code and complete the quiz before the deadline. Most of the answers are hidden in the issue itself. We hope this will enthuse reading and participation!

The next issue will be on **Bone and calcium disorders**. We look forward to your generous ideas and contributions.

Best wishes,

Aashima Dabas

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MESSAGE FROM THE ISPAE PRESIDENT

Dear friends,

Greetings from ISPAE!

World obesity day was celebrated on the 4th of March. The campaign theme for this year has been "Let's talk about obesity and ...."The Global Health Coalition consisting of WHO, UNICEF, and the World Obesity Federation has encouraged the engagement of youth and programs led by them. These steps could help in the analysis of how obesity is affecting their lives and guide towards the steps that can be taken towards its prevention.

To address this burning issue among the children and youth of India, members of ISPAE have initiated several programs around the country. The publication of the IAP revised guidelines on Evaluation, Prevention and Management of Childhood Obesity by Khadilkar et al is an important step forward towards this goal.

We hope this issue of CAPE NEWS focusing on Obesity and Metabolic disorders stirs further activities to improve the health of our children with obesity.

We would like take this opportunity to remind you about the APPES-ISPAE Joint Meeting happening in New Delhi from October 2-4 and invite your active participation.

Best wishes,

Ahila Ayyavoo, President
Rakesh Kumar, Secretary cum Treasurer,
Sirisha Kusuma Boddu, Joint Secretary & EC 2023-2024.

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WELCOME NEW ISPAE MEMBERS

Life members	
• Dr Roli Bansal, Uttar Pradesh • Dr Sunitha GS, Kerala • Dr Rohit Khandelwal, Karnataka • Dr Dharm Raj Maurya, Uttar Pradesh • Dr Bhavna Anand, Delhi	• Dr Sri Lakshmi Chordia, Tamil Nadu • Dr Muhammed Saleeq PK, Kerala • Dr Anju Bala, Himachal Pradesh • Dr Archana K, Uttar Pradesh • Dr Rakhi Jain, Uttar Pradesh • Dr Vaishnavi Agrawal, Madhya Pradesh
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WINNER- December 2023 Quiz	
FIRST THREE WINNERS- 1) DR. CHIRANTAP OZA- Pediatric Endocrinologist, Ahmedabad 2) DR. TRISHYA REDDY- Fellow, Pediatric Endocrinology, Manipal Hospital, Bangalore *3)* DR. MOUMITA SAHA- Pediatric Endocrinologist, CMC Vellore	
THE QUIZMAS ANAGRAM	
1) HYPOGLYCEMIA 2) ICA 3) LACTIC ACIDOSIS 4) IPEX 5) AUTOSOMAL DOMINANT 6) DEND 7) INCRETIN	8) DEGLUDEC 9) ACETOACETIC ACID 10) LEPRECHAUNISM 11) GLUCOKINASE 12) GLUCOSE OXIDASE 13) THIAMINE 14) EMPAGLIFLOZIN
COMPLETE THE PHRASE- THE PANCREAS STRIKE	

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GUIDELINES: INDIAN ACADEMY OF PEDIATRICS REVISED GUIDELINES ON EVALUATION, PREVENTION AND MANAGEMENT OF CHILDHOOD OBESITY

Vaman Khadilkar, *Senior Consultant Pediatric Endocrinologist, Hirabai Cowasji Jehangir Medical Research Institute, Jehangir Hospital, Pune,*
Nikhil Shah, *Consultant Pediatric Endocrinologist, Surya Children's Hospital, Mumbai and MRR Children's Hospital, Thane, Visiting Consultant, LTMMC and Sion Hospital, Mumbai*

India has the second highest rate of childhood obesity (14.4 million obese children in 2017) in the world, only behind China [1]. It is a well-known fact that childhood obesity tracks into adulthood, adversely affecting physical and psychological health. As the previous Indian guidelines on childhood obesity were released over two decades ago, a need was felt to revise and update the guidelines.

The Executive Board of the Indian Academy of Pediatrics (IAP),in February 2023, established a writing committee chaired by the IAP President to formulate these recommendations. Five IAP chapters (Pediatric and Adolescent Endocrinology, Infant and Young Child Feeding, Nutrition, Non-Communicable Disease Prevention Academy, and Adolescent Health Academy) were invited to contribute to the guidelines. Evidence was assessed and graded according to the method used by the American Academy of Pediatrics (AAP) guidelines [2]. Here is a summary of the recommendations (evidence level and recommendation level), suggested by the writing committee, which were published recently [3].

Recommendations:

1. Health care providers should identify and treat childhood obesity as a chronic disease characterized by excessive or dysfunctional body fat (adiposity), which leads to impairment of health, resulting in long-term morbidity and early mortality. (evidence level B, recommendation level moderate)
2. The increasing incidence and prevalence of obesity in infancy, childhood and adolescence in India (including the rural children) needs to be addressed critically by all the stakeholders (pediatricians, school officials, state and national medical bodies, and policy makers) involved in the management and prevention of childhood obesity. (A, strong)
3. Exogenous or primary obesity is responsible for the majority of cases of pediatric obesity. It is important to distinguish it from endogenous or secondary obesity, as evaluation and treatment depends on the cause. (A, strong)
4. ***Recommendations for evaluation of childhood obesity***

4.1 All overweight and obese children should have a comprehensive history and clinical examination, including measurements such as body mass index (BMI), waist circumference (WC) and blood pressure (BP), to recognize the etiology and associated comorbidities. (B, strong)

4.2 In 5-18 years old Indian children, BMI plotted using IAP 2015 BMI charts should be used to diagnose overweight and obesity. (B, moderate)

4.3 BMI cutoffs (on IAP 2015 BMI charts) of 23rd adult equivalent and 27th adult equivalent should be used to define overweight and obesity in Indian children and adolescents aged 5-18 years, respectively. (B, moderate)

4.4 In Indian children below 5 years of age, weight for length or height (WFL/ WFH) using WHO charts should be used to diagnose overweight and obesity. A child whose WFL or WFHis ≥+2 SD (≥97th percentile) but less than + 3 SD (<99.9th percentile), and value ≥3 SD (≥99.9th percentile) is diagnosed as overweight and obese, respectively. (B, moderate)

4.5 In children below 5 years,WFL/ WFH should be plotted at all the vaccine visits and at least 6 monthly (after the 1st birthday). In children above 5 years, BMI should be plotted at least once a year. (A, strong)

4.6 WC, which is a key measure of cardio-metabolic risk, should be measured in all overweight/ obese children and plotted on India specific charts (B, strong)

4.7 WC > 70th percentile can be used as a cutoff for recognising children with central adiposity and at risk of developing metabolic syndrome. (C, weak)

4.8 Routine investigations for endocrine causes need not be done except in short and obese children with additional diagnostic clues. (C, moderate)

4.9 Genetic testing should be reserved only for children with early onset obesity (<5 years of age), with hyperphagia, clinical clues and/or family history of suspected syndromic or monogenic obesity. (B, moderate)

4.10 All obese children older than or equal to 10 years (with or without risk factors) and obese children aged 2-10 years (with risk factors like family history of obesity/ dyslipidemia/ premature coronary artery disease/ diabetes/ hypertension)or WC> 70th percentile, should be screened for hypertension, dyslipidemia, hyperglycemia, metabolic dysfunction-associated steatotic liver disease (MASLD) and other comorbidities. (B, moderate)

4.11 All overweight children older than or equal to 10 years of age should be screened for comorbidities like hypertension, dyslipidemia, hyperglycemia, MASLD, etc. in the presence of risk factors or if WCis > 70th percentile. (B, moderate)

5. Recommendations for prevention on childhood obesity

5.1 Prevention of pediatric obesity should be by encouraging healthy maternal weight and cessation of smoking before pregnancy, appropriate diet and weight gain during pregnancy, exclusive breast feeding for the first 6 months, ensuring appropriate weight gain in infancy, transition to balanced home-made complementary foods, avoid salt (first year of life), avoid extra sugar (first two years of life), avoid packaged foods and forced feeding. (B, strong)

5.2 Childhood nutrition should ensure a balanced diet with healthy eating patterns. The diet should have diverse food groups to provide sufficient energy, protein, fats, micronutrients and vitamins in optimal proportions. (A, strong)

5.3 Children should be offered appropriate portion sizes, depending upon their age and the energy density of the food. (C, moderate).

5.4 Consumption of JUNCS food (Junk foods, Ultra-processed foods, Nutritionally inappropriate foods, Caffeinated/colored/carbonated foods/beverages and Sugar-sweetened beverages)should be avoided. (B, strong)

5.5 Age-appropriate, moderate to vigorous physical activity for at least one hour per day, is recommended for children and adolescents (age 5-18 years). (B, moderate)

5.6 Infants, toddlers and preschoolers should be encouraged to remain active throughout the day through age-appropriate activities and play. (B, moderate)

5.7 Sleep hygiene should be ensured for getting recommended age-appropriate good quality sleep. (B, moderate)

6. Recommendations for management of childhood obesity

6.1 The initial management is lifestyle modification with the help of all care givers, including parents, family and school. If this fails to give results after six months, the management is transferred to a multidisciplinary team for multimodal approach. This team involves pediatricians, pediatric specialists for comorbidities, psychologists or counsellors and dietitians in the management and follow-up care of overweight/obese children. (A, strong)

6.2 Interventions for obese children and adolescents should initially focus on weight maintenance, using healthy dietary practices that are culturally acceptable, affordable, and ensure long-term compliance. (B, strong)

6.3 Children with severe obesity and/or comorbidities can be considered for energy-restricted supervised intensive dietary interventions (e.g., very low-carbohydrate diets, very low-energy diets and lower glycemic index diets) to achieve gradual weight loss. (C, weak)

6.4 Exercise interventions in obese children should be tailored as per the age, sex, preference, socioeconomic status and fitness/ disability level of the child, and should be a combination of aerobic and strength training exercises. (B, moderate)

6.5 Use of pharmacotherapy as an adjunct to comprehensive lifestyle modifications may be recommended in adolescents older than 12 years,with class 2 obesity and immediate or life-threatening comorbidities, or class 3 obesity with/without comorbidities. (B, moderate)

6.6 Surgical options may be offered in children older than 12 years, with class 2 obesity and associated comorbidities, or class 3 obesity with/without comorbidities, only after failure of a proper trial of intense lifestyle modifications and pharmacotherapy for at least 6 months. (C, moderate)

Finally, in addition to the above recommendations, the revised guidelines have defined classes of severe obesity in Indian children. **Class 2 obesity** has been defined as 120% of the IAP obesity cutoff values of 27 adult equivalent, and **Class 3 obesity** has been defined as 140% of the IAP obesity cutoff values of 27 adult equivalent.

The link to IAP charts is https://iapindian.org/iap-growth-charts

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DYSLIPIDEMIA IN CHILDHOOD

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Dyslipidemia is detected in a significant proportion of children and adolescents as per the global studies. It often begins in childhood and adolescence and tracks into adulthood and is the most significant risk factor for early atherosclerosis and premature cardiovascular diseases.

Dyslipidemias are congenital or acquired (secondary) disorders manifesting as qualitative or quantitative changes in lipids and lipoproteins. Elevated lipids do not manifest with any symptoms in early stages and can only be diagnosed by blood tests. The levels of lipids and lipoproteins observed in childhood are stable throughout life and have an independent prognostic value. Thus, there is a need for timely diagnostic, therapeutic and preventive measures.

Dyslipidemia is defined as the presence of any one or more of the following abnormalities on a fasting venous sample:

- Total cholesterol (TC) ≥200 mg/dL (5.2 mmol/L)
- Low-density lipoprotein cholesterol (LDL-C) ≥130 mg/dL (3.4 mmol/L)
- Triglycerides ≥100 mg/dL (1.1 mmol/L) in children <10 years and ≥130 mg/dL (1.5 mmol/L) in children ≥10 years
- High-density lipoprotein cholesterol (HDL-C) ≤40 mg/dL (1.0 mmol/L)
- Non-HDL-C ≥145 mg/dL (3.8 mmol/L)

An abnormal values needs to be confirmed on a second sample performed after a few weeks.

Genetic factors, eating behaviors, environmental factors and metabolic disturbances influence the level of lipids. Obesity, insulin resistance, metabolic syndrome, prediabetes and type 2 diabetes (T2D) are the secondary causes of dyslipidemia that may manifest in childhood and increase the severity of inflammation and endothelial dysfunction that accelerates plaque formation leading to atherosclerosis.

The causes of pediatric dyslipidemia can be genetic (including monogenetic and polygenic defects) like Familial hypercholesterolemia, or secondary to obesity (excessive dietary intake of saturated and transfats), T2D, and nephrotic syndrome (see Box below)

Risk factors for Dyslipidemia

- Dietary causes
  - *Excessive dietary intake of saturated and trans fats* increase low-density lipoprotein
  - *Excessive refined carbohydrates and simple sugars* raise triglyceride levels.
- Secondary causes Obesity, type 2 diabetes mellitus, nephrotic syndrome.
- Genetic causes Monogenic and polygenic defects
  - Monogenic-Familial hypercholesterolemia (FH), Familial defective apolipoprotein B or *PCSK9*, and Familial hypertriglyceridemia
  - Polygenic- polygenic hypercholesterolemia, mixed dyslipidemia (May have clinical phenotype of FH but without a single mutation)

The rationale of screening for lipid disorders in childhood is early identification of high risk individuals and timely management to reduce the risk and severity of atherosclerosis. The recommendations for screening for lipid disorders in childhood are:

- ***Universal screening***- for children and adolescents at ages 9–11, and again between the ages 17–21 years.
- ***Selective screening***- for children between the ages 2–8 and 12–16 years, who have risk factors/risk conditions. The atherosclerosis risk factors are positive family history of hypercholesterolemia or premature cardiovascular disease, high-risk conditions including Kawasaki disease, type 1 or type 2 diabetes, HIV, a solid organ transplant, or nephrotic syndrome.

Obesity associated dyslipidemia includes hypertriglyceridemia, elevated small dense LDL particles, depressed HDL-C levels, and increased apolipoprotein B levels. Obesity-associated dyslipidemia overlaps phenotypically with congenital hypertriglyceridemia, which is associated with increased risk of atherosclerosis and early cardiovascular disease. In the absence of modifiable secondary causes of hypertriglyceridemia, lifestyle, or non-pharmacologic management is the cornerstone.

All patients identified with dyslipidemia should be counseled for healthy lifestyle. It is also recommended to reassess the risk factors at every visit and to assess for secondary causes including hypothyroidism, diabetes, chronic liver, and/or kidney disease, use of medications such as steroids that may alter the lipid profile.

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PATIENT CORNER

STORY FROM A PARENT OF CHILDREN WITH FAMILIAL CHYLOMICRONEMIA SYNDROME

***Ruchi Shah**, Pediatric Endocrinologist, EndoKids, Ahmedabad*

"Hello! I am a mother of two, one boy and one girl, aged 10 and 3 years respectively. Both my kids are living with familial chylomicronemia syndrome (FCS). My son was diagnosed with FCS at the age of 2. Initially we didn't get much help from anywhere; as such the case was new for the pediatricians around us. He used to complain about stomach pain very often and also used to vomit a lot. Many a times the pain was so severe that we had to hospitalize him and doctors would starve him for 2-3 days to get the levels of triglycerides down. In 2016 (a terrifying year for us), he was hospitalized each and every month. In spite of such strict diet and medications, my baby was suffering a lot and we were helpless. His diet was 1 bowl poha/upma for breakfast, 1 bowl dal rice for lunch, 1 banana/apple for snacks and 1 bowl khichdi, every single day. This was on this strict diet till the age of 7. He was afraid to attend school and make friends. He developed frequent pancreatitis.

We then consulted a pediatric endocrinologist who advised to switch to coconut oil as cooking medium and addSimyl MCT oil on salads as dressing. We discontinued all plain sugar sources, and started using Sagar skimmed milk powder as milk substitute. My son improved and did not develop pancreatitis for last 3 years. We have gradually started to enjoy life with him. His growth remains poor and he weighs only 17-18 kg. Otherwise, he is very brilliant and always scores gold medals in academics.

Unfortunately, my daughter was born with the same condition and was detected when she developed the first episode of pancreatitis at the age of 4 months. Luckily, insights into management of my son made us wiser to institute timely dietary modifications for her. Her growth is better than my son's. Our doctor suggested to try insulin to cover certain meals, but we're hesitant to do that yet.

Thank you very much, doctors. I hope that a cure for this will soon be found and made available."

DRUG CORNER

TREATMENT OF FAMILIAL CHYLOMICRONEMIA SYNDROME

***Ruchi Shah**, Consultant Pediatric Endocrinologist, EndoKids, Ahmedabad*

Familial Chylomicronemia Syndrome (FCS), also referred to as lipoprotein lipase deficiency (LPLD), is a rare genetic lipid disorder characterized by severe elevations in circulating triglycerides (Normal <150, elevation mild 150-199, moderate 200-999, severe 1000-1999, very severe >2000 mg/dL) [1].

LPL breaks down chylomicrons through hydrolysis of serum triglycerides into free fatty acids. Patients with FCS have inherited defects that limit the production or impair functionality of LPL. These loss-of-function mutations occur in either the *LPL* gene or in genes related to the function of LPL, mostly including *APOC2*, *APOA5*, *GPIHBP1*, and *LMF*. Clinical features of FCS include xanthomas, lipemia retinalis, fatigue, and hepatosplenomegaly, and often accompanied by episodes of abdominal pain and pancreatitis.

Pancreatitis is a common complication that has multifactorial etiology in FCS [2]. Accumulation of chylomirons in the pancreatic capillaries, resulting in lipolysis by the pancreatic lipase, and accumulation of FFA in the acinar cells, causing direct cytotoxicity, formation of microthrombi, ischemia and tissue necrosis etc. The hypoxic process explains the high frequency of severe necrotic pancreatitis.

Diagnosis is based on severe refractory hypertriglyceridemia, observed on 3 different occasions, where secondary causes can be excluded [3]. The presence of either one of three- acute pancreatitis, recurrent abdominal pain without any explainable cause or positive Genetic mutation confirms the diagnosis.

Management: Management of acute pancreatitis resolves around fluid replacement, pain control, plasmapharesis (reserved for patients developing multiorgan failure) and insulin – 0.005 – 0.01 u/kg/hr with dextrose infusion and close monitoring of sugars. Long term management involves dietary modifications with limited role of pharmacotherapy at present [4]. A multidisciplinary team is advocated for improving disease outcomes.

Dietary modifications:

Lipids:	**Other nutrients:**
1. Very-low-fat diet by either consuming less than 10-15 g of fat per day or restricting total dietary fat to less than 10-15% of total daily caloric intake (essentially exclude all types of oils, ghee, butter, cheese etc., i.e. sources of fat in diet) 2. Meet Essential Fatty Acid requirements of 2-4% of daily calories in the very-low-fat diet (include walnut, flaxseeds, chia seeds etc.) 3. Medium-chain triglyceride (MCT) oil may be used for salad dressing, coconut oil may be used for cooking 4. Monitor levels of fat-soluble vitamins and replace as necessary	1. Protein rich foods 2. Ensure complex carbohydrate foods, avoid simple and refined carbohydrates as much as possible 3. Distribute calorie and fat intake throughout the day, while avoiding excess caloric intake. 4. Avoid alcohol 5. At least 150 minutes of physical activity/wk 6. Adequate Hydration 7. Breast milk has to be replaced with low fat formula (Nutricia Monogen, fat content 11.8 gm/100 gm, INR 6500/- per 400 gm). In India, a very useful alternative is skimmed milk spray dried powder (Sagar, fat content 1 gm/100 gm, INR 190/- per 500 gm)

Pharmacotherapy:

- Currently no FDA approved medicines for FCS
- Fenofibrate can be used with variable response
- Insulin: Activates lipoprotein lipase. There is some role of Regular insulin used in very low doses, with frequent sugar monitoring [5].
- Future therapies- LPL gene therapy and Apo C-III Anti-Sense Oligonucleotides (ASO)

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FDA APPROVED ANTI-OBESITY MEDICATIONS IN CHILDREN & ADULTS

Kochurani Abraham, *Consultant Pediatric Endocrinologist, Ankura Hospitals, Pune*

Drug	Mechanism of action	Dose	Adverse Effects	Contra Indications	Mean % Weight Loss
ORLISTAT	Reversible inhibitor of gastric and pancreatic lipase, inhibits fat absorption by 30%. Available as OTC	Minimum age: 12 years OTC: 60 mg TID Prescription: 120 mg TID (skip dose for skipped meals)	Oily rectal leakage, abdominal pain, flatulence with discharge, bowel urgency, steatorrhea	Pregnancy, breastfeeding, chronic malabsorption syndrome or cholestasis	2.9–3.4% weight loss
LIRAGLUTIDE	Mimics GLP-1 incretin, stimulates postprandial insulin secretion, reduces glucagon, reduces hunger and food intake. **Phenotype Hungry Gut**	Minimum age: 12 years Week 1: 0.6 mg SC/ d Week 2: 1.2 mg SC/d Week 3: 1.8 mg SC/d Week 4: 2.4 mg SC/d Week 5: 3.0 mg SC/d	GI symptoms: abdominal pain, nausea, vomiting, diarrhoea, and hypoglycaemia	Personal or family history of medullary thyroid carcinoma, MEN2	7% weight loss over placebo
SEMAGLUTIDE		Minimum age: 12 years Week 1–4: 0.25 mg SC/wk Week 5–8: 0.5 mg SC/wk Week 9–12: 1 mg SC/wk Week 13–16: 1.7 mg SC/wk Week 17-on: 2.4 mg SC/wk			16.1% BMI reduction
SETMELANOTIDE	MC4R agonist,Appetite suppression via stimulation of hypothalamus to release norepinephrine . Used in SYNDROMIC OBESITY	**6-12 years**-starting dose 1 mg SC once daily for 2 weeks. **>12 years**-starting dose 2 mg SC once daily for 2 weeks. Monitor patients for gastrointestinal (GI) adverse reactions.	Hyperpigmentation, GI symptoms, depression, dizziness, fatigue, headache, insomnia, prolonged penile erections	Pregnancy	9.7% weight loss (LEPR), 23.1% weight loss (POMC/PCSK1), 7.9% BMI reduction
PHENTERMINE	Appetite suppression via stimulation of hypothalamus to release norepinephrine	Minimum age: 16 years Starting dose (8mg/15 mg daily) with increase to bid if needed	Increase in BP, heart rate, anxiety	Pregnancy Use caution in congenital heart disease, hypertension, renal impairment	5–7.8% weight loss

PHENTERMINE/ TOPIRAMATE	Appetite suppression via release of norepinephrine , enhances GABA release while inhibiting AMPA and NMDA **Phenotype Hungry Brain**	Minimum age: 12 years Starting dosage is 3.75 mg/23 mg daily for 14 days up to15 mg/92 mg Dose escalated based on BMI reduction in pediatric patients.	Increase in BP, HR, anxiety, paraesthesia, cognitive dulling, dizziness, dysgeusia, dry mouth, constipation , suicidal ideation, seizures if stopped abruptly.	Pregnancy, use of MAOIs, glaucoma, black-box warning for worsening depression in 18–24 year olds	Moderate dose: -8.11% BMI Top dose: -10.44% BMI

FDA APPROVED MEDICINES FOR OTHER INDICATIONS WITH OBESITY

Drug	Mechanism of action	Dose	Adverse effects	Contra Indications	Mean % Wt Loss
METFORMIN	Increases insulin and leptin sensitivity, may increase secretion of GLP-1	Minimum age 10 years with T2DM 500 mg with dinner, titrate to 500 mg bid, if tolerated & needed, can titrate to 1000 mg bid. If using ER, can take full dose once daily	GI upset, vitamin B12 deficiency, lactic acidosis	Severe renal dysfunction and acute/ chronic metabolic acidosis	2.5% weight loss over placebo
NALTREXONE/ BUPROPION	Reuptake inhibitor of norepinephrine and dopamine, and opioid antagonist. Useful in compulsive eating disorders	Approved for adult use only Week1:1 tablet in am, Week2:1 tablet bid, Week 3:2 tablets in am, 1 tabletin pm, Week 4 onward: 2 tablets bid ER Tab-8mg Naltrexone/90mg Bupropion	Nausea, vomiting, headache, dizziness	***Black-box warning*** worsening depression or suicidal ideation in youth, uncontrolled hypertension, seizure disorder, anorexia, bulimia, drug or alcohol withdrawal MAOI use	4.8–6.0% weight loss over placebo
TOPIRAMATE	Appetite suppression via enhanced GABA release while inhibiting AMPA and NMDA. Useful in compulsive eating disorders	Approved for adult use only 25 mg daily x 1week Titrate to 50 mg daily for 1 week Titrate to 75–100 mg if needed.	Paraesthesia, cognitive dulling, fatigue, worsening depression	Pregnancy	2–4% BMI reduction

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DIAGNOSIS CORNER

GENETIC TESTING IN PATHOLOGICAL OBESITY

***Richa Arora**, Consultant Pediatric Endocrinologist, Child Clinic and Endocrine Center, New Delhi*

Obesity results from complex interaction of genetic, hormonal, nutritional, and environmental factors [1]. Pathological obesity is classified into syndromic and non-syndromic obesity with or without congenital defects and developmental delay.

- ***Syndromic causes*** include Prader–Willi, Fragile X, Bardet–Biedl syndrome, Cohen syndrome, and Albright Hereditary Osteodystrophy (AHO) syndromes, which are associated with early onset obesity and developmental delay.
- ***Non-syndromic obesity*** can be monogenic, polygenic, or chromosomal in origin. Monogenic obesity is caused by variants of single genes, while polygenic obesity includes several genes with the involvement of members of gene families, with or without syndromic findings, but accompanied with obesity and recognized phenotypes.

About 127 informative sites in the human genome have been reported to show linkage with obesity by genome-wide association studies (GWAS) [2], and over 500 obesity-related genes are recognized in humans. There are approximately 30 neuro-endocrine peptides in humans known to inhibit eating behavior. Ghrelin increases eating with an important role in appetite regulation and energy balance. Many monogenic neuroendocrine disorders involving the leptin pathway are recognized and associated with early onset obesity in childhood. Mutations of the *MC4R* gene are most frequently found among children with non-syndromic severe obesity, in whom the incidence ranges from less than 1% to 6% depending on nationality and variant [3]. These patients present with hyperphagia, severe hyperinsulinemia, tall stature, and high fat and lean mass. Several of the genes associated with early-onset severe obesity belong to the leptin-melanocortin pathway, including the genes encoding leptin (*LEP*), leptin receptor (*LEPR*), melanocortin-4 receptor (*MC4R*), prohormone convertase 1 (*PCSK1*), proopiomelanocortin (*POMC*), single-minded homolog 1 (Drosophila) (*SIM1*), and brain-derived neurotrophic factor (*BDNF*). In addition to developing severe obesity at an early age, carriers of mutations in some of these genes also have intellectual disabilities and exhibit developmental delays, which suggests that there is an interplay between neuro-development and the hypothalamic functions of energy homeostasis and body-weight regulation.

Genetic testing may be considered in the following conditions [4,5]:

- Children with syndromic early onset obesity (before 5 years old), such as Prader-Willi or Bardet-Biedl syndromes (BBS)

- Children with early onset obesity (before 5 years old) and hyperphagia, and/or a family history of severe obesity
- Developmental delay, dysmorphic features, hormonal deficiencies (e.g., hypogonadism, adrenal insufficiency), congenital anomalies, or vision loss- Threshold for genetic testing low.

There is increasing interest in genetic findings to be applied in the clinical setting to improve risk prediction and facilitate personalized therapy for obesity. The translation of genetic discovery into risk prediction is challenging at the population level. High penetrant variants associated with severe early-onset or syndromic forms of obesity may serve as a diagnostic tool and could assist in designing personalized therapy for individuals. Screening of identified variants in family members also assists early diagnosis. Long-term follow-up studies will be necessary to evaluate the genetic interaction with therapeutic outcomes.

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OBESITY-METABOLICALLY HEALTHY OR UNHEALTHY?

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Metabolic healthy obesity (MHO) is a useful term that denotes obesity without an unfavorable cardiometabolic profile. Individuals with metabolic unhealthy obesity (MUO) phenotype are at more risk of cardiovascular disease as compared to those with MHO. This categorization helps to prioritize the category of patients who need intensive lifestyle modification, close monitoring and possible upgrading of interventions. Those with MHO need to be considered as being at a transitional stage, with risk of going into MUO, besides having more risk than their normal weight counterparts.

MHO- A uniformly acceptable set of criteria has been long awaited. Studies have estimated different prevalence rates as per National Cholesterol Education Program (NCEP) Criteria and as per International Diabetes Federation (IDF) Criteria. MHO is defined as obesity *with absence of any risk factor* as defined below:

- ***Modified NCEP criteria*** - elevated BP (systolic/ diastolic BP ≥90th percentile for sex, age, and height, elevated TG (≥110 mg/dL), low HDL-C (<40 mg/dL), and elevated fasting blood glucose, FBG (≥110 mg/dL).
- ***Modified IDF criteria***- systolic/diastolic BP≥ 120/80 mmHg for age <10 years and ≥130/85 mmHg for ≥10 years, TG ≥150 mg/dL, HDL-C <40 mg/dL for all males and<50 mg/dL in females≥16 years, and FBG ≥100 mg/dL.

An evidence- based consensus definition of MHO by an international panel of 46 experts has recently been arrived:HDL-c> 40 mg/dl (or > 1.03 mmol/l), triglycerides ≤ 150 mg/dl (or ≤ 1.7 mmol/l), systolic and diastolic BP ≤ 90th percentile, and a measure of glycemia [1]. Most experts agreed on FBG level of ≥100 mg/dL as being a risk factor. MHO has also been described as absence of insulin resistance as characterized by HOMA IR<3.16 or less than 95th percentile for age and sex as per population based data [2].

MUO- Those with MUO have higher weight, height and BMI;higher levels of insulin, leptin, insulin-like-growth factor and adrenal androgens may be the contributing factors. Notable predictors of increased cardiovascular remodeling include severity of obesity, insulin resistance, waist circumference (WC) and waist-height ratio [3]. WC also predicts increased risk of liver fibrosis in those who have MASLD. Abdominal fat accumulation and hepatic fat content also correlate with unfavorable metabolic health. Serum uric acid has been shown to be an independent predictor of poor metabolic health in obesity [4].

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ELASTOGRAPHY IN PEDIATRIC OBESITY

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Obesity is one of the independent risk factors for non-alcoholic fatty liver disease (NAFLD). NAFLD is defined as accumulation of fat in the liver which is generally a silent, potentially reversible condition, but might progress into a more severe form, i.e., non-alcoholic steatohepatitis (NASH), with an increased risk for cirrhosis and end-stage liver disease. NASH comprises a triad of fatty infiltration, inflammation, and fibrosis. The prevalence of pediatric NAFLD worldwide ranges from 9–37%. Children with NAFLD have a high prevalence of insulin resistance, Type 2 diabetes mellitus, hypertension and metabolic syndrome.

Liver biopsy is the gold standard to diagnose NAFLD but is restricted in children as it is invasive. Among non-invasive methods, the screening method for pediatric NAFLD is B-ultrasonography (B-scan), combined with measurement of serum alanine transferase (ALT). However, the poor sensitivity and specificity of the B-scan and the variability of liver enzymes with age and body mass index, precludes its use in the assessment of NAFLD.

Elastography has emerged as a non-invasive tool for diagnosis and staging of NAFLD. It can be assessed by Magnetic resonance elastography (MRE) or ultrasound.

The different modalities using ultrasonography are:

- Transient elastography (TE)
- Acoustic radiation force impulse (ARFI) that can be Point shear wave elastography (p-SWE) and 2-dimensional shear wave elastography (2D SWE)
- Strain elastography.

MECHANISM- The non-invasive evaluation of tissue elasticity and its mechanical characteristics is brought about by applying external forces that characterize elastography techniques. TE assesses liver stiffness using a vibrator or specialized probe placed on the thoracic wall between the ribs; the TE device creates an elastic wave projected into the right hepatic lobe. Once the probe is positioned correctly, it will send a low-amplitude signal to the hepatic tissue, which causes an elastic shear wave to start and spread across the tissue. TE detects wave velocity, measured in kPa, and correlates with liver elasticity, ranging from 2.5 to 74 kPa.

PRINCIPLE - A modified phase contrast MRI sequence is used in the MRE of the liver to acquire images of mechanically generated shear waves propagated into the organ. Transient elastography, also known as fibroscan of the liver, is a dynamic shear wave elastic imaging technology. The principle is to detect the propagation velocity of shear waves in liver tissue to obtain the elastic modulus of liver tissue, namely liver stiffness measurements (LSM). The ARFI method, which involves shearing the tissue under examination, generating a circular wave in the tissue that resembles a stone thrown into water, and using a controlled attenuation parameter (CAP) tool to measure the decrease in amplitude of ultrasound signal in the liver, can be used to achieve real-time elastography. A novel ultrasound method called 2D-SWE allows real-time tissue visualization while accounting for the viscosity and flexibility of the tissue being studied. The device measures velocity and stiffness in real-time, independent of respiratory phases, so it does not require children to hold their breath while being examined. Compared to TE, this method is increasingly used to assess hepatic elasticity.

The cut-off values on 2D SWE are 4.13 kPa in children aged between 3–5 years and 4.88 in those between 12-15 years; while on TE these values varies from 4.40 kPa between 3–5 years of age and 5.1 kPa in adolescents aged 15–18 years.

LIMITATIONS

- Increased blood flow as a result of food intake leads to increased liver stiffness. A period of at least 4 hours of fasting is recommended before the examination in order to avoid this confounding factor.
- It can lack proper differentiation between mild fibrosis and normal liver tissue. For BMI ≥ 30 kg/m2, when it is most needed, elastography has a relatively low accuracy in differentiating between the first two fibrosis stages.
- Excessive movement during the examination and small intercostal spaces may result in invalid or falsely increased liver stiffness values.

The accuracy of elastography methods might be improved if combined with other clinical or laboratory parameters. Studies have revealed positive associations between ultrasound-based elastography methods and several laboratory parameters such as ALT, APRI (aspartate

aminotransferase to platelet ratio index), or AST/ALT ratio (AAR), which are known to be increased in children with obesity.

To conclude, NAFLD is a challenge to diagnose. Quantitative US-SWE is a superior imaging modality for evaluating liver stiffness in children and complementary method to conventional ultrasonography, but limitations remain.

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PEDSENDOSCAN [ISPAE-JPED and Others]

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ISPAE-JPED

Mynepally M, Yadav BR, Mythili A, Vivekananda B, Subrahmanyam KA. Study of growth parameters in children and adolescents with Type I diabetes mellitus. JPED. 2023;3:58-62.

The observational cross-sectional study included 57 subjects (30F) with T1D between ages 1-18 years, with diabetes duration of at least one year. Height for age Z-scores (HAZ), weight for age Z-scores (WAZ), and body mass index for age Z-scores (BAZ) were calculated using standard IAP 2015 growth charts. The median (IQR) age was 13.8 (9–17.1) years, age at diagnosis was 8 (4–11.5) years, and mean duration of T1D was 5 (3–7) years. The mean HbA1c was 10.33 ± 1.88%. The growth parameters as assessed by HAZ −0.83 (−1.98–−0.16), WAZ −0.98 (−1.72–−0.11), and BAZ −0.55 (−1.41–0.1) were low in comparison to the population medians. The age at diagnosis, duration of diabetes, and the type of insulin regimen did not significantly impact HAZ and WAZ. Children with HbA1c <8.5% had better HAZ −0.21 (−0.94–0.35) versus −1.07 (−2.07–−0.25), (P = 0.069), and WAZ −0.33 (−0.73–0.23) versus –1.07 (−1.77–−0.29), (P = 0.041) compared to those with HbA1c >8.5%.**Children with T1D were shorter and leaner than age- and sex-matched controls. Children with lower HbA1c had better HAZ and WAZ.**

Lim RJ, Abraham MB, Nicholls R, Fournier PA, Harray AJ. Efficacy of the MiniMed™ 670G hybrid closed loop system in managing postprandial glucose excursion with high protein high fat foods in children and adolescents under free-living conditions. JPED. 2023;3:63-70.

Children and adolescents (8–18 years) with type 1 diabetes, using the MiniMed™ 670G were recruited to a free-living randomized cross-over study. Participants consumed a

standardized lasagne or pizza meal two nights a week for 4 weeks while in auto mode and manual mode. Postprandial continuous glucose monitoring data were collected for 7 h post-meal. The primary outcomes were mean postprandial net incremental area under the glucose × time curve. User experiences were collected during end-of-study interviews administered to parents. Postprandial excursions from 38 meals in seven participants were analyzed. There were no significant differences between auto mode and manual mode for the mean net incremental area under the glucose × time curve, irrespective of meal type. Semi-structured end-of-study interviews revealed that five of seven families felt more confident eating high protein high fat (HPHF) meals in auto mode. **Although most families felt confident with auto mode for postprandial HPHF excursions, this was not reflected in the postprandial glucose levels.**

Other Journals

Southcombe F, Lin F, Krstic S, Sim KA, Dennis S, Lingam R, Denney-Wilson E. Targeted dietary approaches for the management of obesity and severe obesity in children and adolescents: A systematic review and meta-analysis. Clin Obes. 2023;13(2):e12564

This systematic review examined the effectiveness of diets of varying energy content as a component of weight treatment in children and adolescents with obesity, severe obesity and obesity-related comorbidity. Dietary interventions were grouped according to diet type and energy target. Meta-analysis examined change in body mass index (BMI) at intervention end. When dietary types were considered by energy target, a gradient effect was observed. Very-low energy diets were most effective, with BMI change of - 4.40 kg/m^2,while dietary interventions with no specified energy target were ineffective, resulting in a BMI gain of +0.17 kg/m^2. **Practical definitions of dietary energy target in the management of obesity and severe obesity are urgently required to ensure treatment seeking children have timely access to efficacious interventions.**

Vourdoumpa A, Paltoglou G, Charmandari E. The Genetic Basis of Childhood Obesity: A Systematic Review. Nutrients. 2023;15(6):1416.

The study aimed to systematically review the scientific evidence and to explore the relation of single-nucleotide polymorphisms (SNPs) and copy number variants (CNVs) with changes in body mass index (BMI) and other measures of body composition in children and adolescents with obesity, as well as their response to lifestyle interventions. The effect of polymorphisms in 92 different genes was assessed and revealed SNPs in 24 genetic loci significantly associated with BMI and/or body composition change, which contribute to the complex metabolic imbalance of obesity, including the regulation of appetite and energy balance, the homeostasis of glucose, lipid, and adipose tissue, as well as their interactions. **The decoding of the genetic and molecular/cellular pathophysiology of obesity and the gene-environment interactions, alongside with the individual genotype, will enable designing of targeted and personalized preventive and management interventions for obesity early in life.**

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CASE REPORTS

A RARE CASE OF SYNDROMIC OBESITY

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Syndromic causes of obesity in children are usually rare and difficult to diagnose, because of the varied manifestations at presentation.

We describe a case of a 2.5year old boy, first child born at term to non-consanguineous parents, with a birth weight of 2.9 kg, who presented to us with complaints of increased hunger since the age of 1.5 years, with violent tantrums when food was not provided, resulting in steadily increasing weight gain. He had been exclusively breastfed till 6 months of age. His parents had noted that he had progressive loss of vision and photophobia for nearly 7mo;he had developed seizures a month prior to presentation. There was mild delay in motor and language milestones:he attained neck control at 6 months of age, started walking at 18 months, and at 2.5 years was speaking 2-3 word sentences.

On examination, he was 19kg (weight Z score +3,weight for height Z score +4.46),height was 91cm(height Z score of -0.27). He was obese with acanthosis nigricans Grade 3 and lipomastia. He had pendular nystagmus,hypodontia, brachydactyly, and retinitis pigmentosa on ophthalmological exam. He was prepubertal, with bilaterally descended testis (2cc). Systemic examination was normal. He had dyslipidemia (total cholesterol 156 mg/dl, HDL 29mg/dl,triglycerides 232 mg/dl), with elevated transaminases (SGOT 50U/L, SGPT 43U/L, ALP 224U/L). Pre and postprandial blood sugar levels and renal parameters,2-D echocardiography and abdominal ultrasonography were normal.

With history of mild developmental delay, visual impairment and syndromic obesity, genetic evaluation was done, which showed findings suggestive of Alstrom syndrome - pathogenic heterozygous nonsense variation in exon 10 of the *ALMS1* gene (chr2:73717247C>T; Depth:143x) that results in a stop codon and premature truncation of the protein at codon 2720 (p.Arg2720Ter; ENST00000264448.6) and a pathogenic heterozygous single base pair duplication in exon 10 of the *ALMS1* gene (chr2:73717794dupA;Depth: 130x) that results in a frameshift and premature truncation of the protein 23 amino acids downstream to codon 2902 (p.His2902GInfsTer23; ENST00000264448.6). The parents were advised lifestyle modification, including a meal plan; and genetic counselling. Presently the child is 7 years old (***Figure***) and has behavioral issues, aggressiveness and impaired hearing. BERA is yet to be done.

Alstrom syndrome (ALMS) is a rare autosomal recessive, multisystem, monogenic disorder caused by pathogenic variants of *ALMS1*gene located on chromosome 2p13. It is characterized by progressive retinal degeneration, obesity, neuronal hearing loss and insulin resistance [1]. Clinical symptoms first appear in infancy, with wide variability in age of onset and severity. ALMS has an estimated incidence of 1 case per 1,000,000 live births; ethnically or geographically isolated populations have a higher-than-average frequency [2].

The diagnosis is based on the presence of major and minor criteria. ***Major criteria*** include: a pathogenic mutation in one *ALMS1* allele or family history of Alström syndrome, nystagmus, legal

blindness, cone-rod dystrophy. ***Minor criteria*** include obesity and/or insulin resistance and/or type 2 diabetes mellitus, dilated cardiomyopathy with congestive heart failure, hearing loss, hepatic dysfunction, renal failure, short stature, hypogonadism (males), hyperandrogenism (females), and other variable supportive evidence [3].

Currently, there is no known cure for ALMS other than managing underlying systemic diseases. Supportive treatment includes a diet high in anti-oxidants, correction of refractive errors, low-vision aids and wearing sunglasses outside.

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BARDET-BIEDL SYNDROME: ROLE OF MDT

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Bardet–Biedl syndrome (BBS) is a rare, genetic multisystem disorder due to dysfunction of the primary cilia. This ciliopathy is characterized by polydactyly, rod–cone dystrophy, learning disabilities, renal malformations, obesity, and hypogonadism. According to the diagnostic criteria published by Beales et al., the diagnosis of BBS is based on the presence of at least four primary features, or three primary features and at least two secondary features as shown in the table below.

Primary Features	Secondary Features
• Rod-Cone Dystrophy (100%) • Obesity- seen (72-96%) • Polydactyly- post-axial • Learning Disabilities • Hypogonadism In Males • Renal Anomalies	• Speech Disorder/Delay • Strabismus/Cataract/Astigmatism • Brachydactyly/Syndactyly • Developmental Delay • Polyuria/Polydisia • Ataxia/Imbalance • Mild Spasticity • Diabetes Mellitus • Dental Crowding/ Hypodontia/High Arched Palate • Left Ventricular Hypertrophy/ Congenital Heart Disease • Hepatic Fibrosis

Twenty-one BBS genes (*BBS1–BBS21*) have been identified; he two main genes are *BBS1* and *BBS10*, which are present in more than 20% of the cases. Patients with mutations in *BBS1*

generally present later than patients with mutations in *BBS10*, owing to a milder phenotype and a later onset of retinal degeneration. BBSomes are proteins that are encoded by BBS genes, which function to promote the biogenesis and functions of the primary cilia.

A 14 year old boy, first born to a third degree consanguineously married couple with a birth weight of 3.3 kg, presented with lower respiratory tract infection. He had a learning disability, and history of poor academic performance, developmental delay and visual impairment. History of snoring and mouth breathing was present, but notof polyuria or polydipsia. On examination, he was103 kg,163 cm, BMI 38.9 kg/m^2; vital signs were normal. He had acanthosis nigricans over nape of neck, axilla and groin, abdominal striae; post-axial polydactyly of both upper limbs, and retinal dystrophy on ophthalmic exam; the rest of his physical examination was unremarkable. He was Tanner stage 1: prepubertal, with testicular volume 2cc and stretched penile length (SPL) 4cm.

Laboratory investigations revealed HbA1C of 6% (prediabetes), HOMA IR- 3.6, low LH (< 0.22 mIU/ml), FSH (1.05 mIU/ml) and total testosterone (0.593 ng/mL), suggestive of hypogonadotropic hypogonadism. 2D ECHO showed bicuspid aortic valve with mild aortic stenosis and concentric left ventricular hypertrophy. Pulmonary function tests and sleep study were done; he had obstructive sleep apnea.

He was diagnosed with BBS because he had four major features (retinal dystrophy, post-axial polydactyly, obesity, and learning impairments), as well as three secondary features (developmental delay, congenital heart disease and speech disorder). Clinical exome sequencing to confirm the diagnosis showed a homozygous deletion in*BBS2* gene exon 14 to 15 (likely pathogenic).

A Multidisciplinary team (MDT) was planned for further management of the child.

- Endocrinology- He was started on Tab Metformin, along with diet and lifestyle measures, and HCG injections 2500 IU twice weekly and HMG injections 75 IU twice weekly for hypogonadism.
- Cardiology- 2D ECHO was reviewed by pediatric cardiologist and advised conservative management.
- Ophthalmology- for retinal dystrophy: counselling was done.
- Nutrition- Detailed nutritional counselling was done by a registered dietitian.
- Pulmonology- He was started on inhalers (Asthalin and Foracort) along with auto- CPAP support at night.
- Development and Speech- Development and IQ assessment done and speech therapy was given.

Presently he is 86 kg, having lost 17 kg over 7 months; SPL is 4.9cm, and testicular volume 6cc. Latest investigations revealed HbA1c- 5.3%, LH- 0.92 MIU/ML, FSH- 2.26 MIU/ML. Metformin has been tapered and stopped. He continues to be on HCG and HMG injections, along with diet and lifestyle measures, and CPAP support at night, with improvement in sleep.

The management of BBS is supportive through a MDT. Genetic analysis for confirmation of BBS is not available in many resource-limited settings. The rarity of the syndrome, and its slowly

progressive course, pose a significant challenge for early diagnosis, which is critical for halting the progression of renal impairment since it is the leading cause of morbidity and mortality in patients with BBS.

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SCHAAF-YANG SYNDROME

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Schaaf-Yang syndrome (SYS) is a rare neuro developmental disorder first described in 2013. It is caused by a truncating mutation in the maternally imprinted, paternally expressed, single exon *MAGEL2* gene that regulates endosomal protein trafficking and recycling [1]. Only mutations in the paternal allele lead to SYS, as the maternal allele is silenced through methylation. This mutation is mapped to the Prader-Willi region (15q11-q13), with an observed phenotype partially overlapping that of Prader-Willi syndrome. [2] The syndrome is characterized by neonatal hypotonia, developmental delay, respiratory abnormalities, interphalangeal joint contractures, autism spectrum disorder, and endocrine abnormalities. A subgroup of patients also developed obesity and hyperphagia.

An eleven-year-old boy presented with weight gain since 5 yrs of age, dyspnea on exertion, snoring, increased perspiration, hyperphagia and poor scholastic performance. He was the second child born to nonconsanguineous obese parents with family history of type 1 diabetes and hypertension. Hewas 145 cm, 62.3 kg, and body mass index 29.6kg/m^2 (obese); prepubertal with lipomastia, acanthosis nigricans, and normal blood pressure. His baseline workup was normal. The family was counselled for lifestyle modifications and regular follow up.

He then presented at the age of 15.8 years with rapid weight gain, hyperphagia, obstructive sleep apnea and gyneco-lipomastia. He now measured 161 cm (10-25th centile, for a mid-parental height 172 cm (25-50th centile), weight 99.9kg, BMI 36 kg/m^2 (morbidly obese) and testicular volume 3-4mL. He was euthyroid with peripubertal gonadotropin levels. He was advised stricter lifestyle measures, and in view of rapidly increasing weight, whole genome exome sequencing. This revealed a heterozygous variant c.142C>A in exon 1of the *MAGEL2* gene (chr15:23647601G>T;Depth:93x) suggestive of Schaaf-Yang syndrome. Reassessment after 6 months revealed height gain of 3 cm and reduction in BMI by 0.6 kg/m2, with normal pubertal progression.

To conclude, early onset obesity with developmental issues should be evaluated for syndromic or genetic obesity.

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LEARNING PEARLS

PRADER WILLI SYNDROME SCIENTIFIC AND FAMILY CONFERENCE

*Compiled by **Jahnavi M**, Fellow Pediatric Endocrinology,*
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Physical therapy in PWS by Miss Janice Agarwal

- Physical therapy, including sensory integration, is a vital part of the management of PWS.
- Poor vestibular system results in hypotonia/ clumsiness/ unpredictable behavior/tantrums/ overly impulsiveness/ poor attention/ may not develop handedness.
- Techniques to improve the vestibular system: swinging/ rocking/ spinning/ scooter board/tyre swing/ bouncing on large ball/ hopscotch/ riding on bikes.
- Poor proprioception is indicated by: Biting/ chewing of hand nails/ grinding of teeth/ mushy speech/ walking next to a wall or touching the wall while walking.
- Techniques to improve proprioceptive system: Deep and slow massaging/ use of heavy blankets/ carrying a backpack/ heavy items.
- Poor tactile sensation is indicated by insensitivity to cuts or bruises or temperature/ not liking to have hair or teeth brushed/ constantly picking at skin.
- Techniques to improve the tactile system: Brushing/ rolling/ deep massage of skin/ joint compression/ dressing up with gloves/ hats/ painting/ use of sensory toys/ playing with rice trunk.
- Oral motor activities include eating, sucking, blowing, biting. These help the child to calm or alert himself, promote trunk flexion, trunk extension and jaw, neck, shoulder stability respectively.
- Techniques to improve oromotor system include blowing bubbles or whistles, use of cold popsicles or bananas, chewing dry fruits, crunchy vegetables/ tugging/ biting/ pulling with teeth/using warm/ sour food items.

Nutrition in PWS by Mr Chris Smith

Four key messages:

- Calorie restriction is not recommended in the phase of undernutrition, it should be only after phase 2a.

- Important to track and monitor growth
- Quantity and quality of diet
- Think calories beyond food
- Be flexible through the ages

- Calories for weight maintenance: 10- 12 kcal/ cm height, weight loss: 6-9 kcal/ cm
- Food security is the foundation for a good diet plan

Multidisciplinary care for patients with PWS by Dr Shankar Kanumakala

- Multi-disciplinary team involving an endocrinologist, nutritionist, physiotherapist, pulmonologist, psychiatrist, orthopedician, ENT physician, developmental pediatrician helps to improve treatment and patient care.
- Early initiation of growth hormone (GH) is useful for normalization of height, weight, body proportions, muscle tone and muscle mass.
- GH is not indicated in the following conditions: Worsening scoliosis/ sleep disordered breathing/ severe uncontrolled diabetes/ acute psychosis.

Behavioral manifestations in children with PWS by Dr Deepan Singh

- Behavioral manifestations seen include hyperphagia, skin picking, impulsivity, inflexible behavior, repetitive behaviors, impatience, confabulation, inattention, and response perseveration.
- Response perseveration is the inappropriate repetition of response, despite the absence or cessation of reward.
- Response monitoring is the capacity to flexibly adapt to dynamic environments. Children with PWS syndrome have difficulty in response monitoring.
- Psychosis in PWS is subtle, may present with sleep disturbances or catatonic symptoms, decrease in food seeking behaviors.
- Behavioral modification and applied behavior analysis are helpful for children with PWS.
- Pharmacological agents include antipsychotics, mood stabilizers, antidepressants, N acetyl cysteine.

Orthopedic management in children with PWS by Dr Harold Van Bosse

- Development delay, flat foot deformity, osteopenia, hip dysplasia, spine deformities are among the main orthopedic issues.
- Flat feet are secondary to ligament laxity and hypotonia, and lead to poor foot positioning for walking/ running. Treatment is bracing- Supra malleolar orthotics (SMO) and University of California Biomechanics Laboratory (UCBL).
- Spine deformity including scoliosis and kyphosis are common, with 2 peaks: first before 4 years and second by 10 years of age.

Management of scoliosis:

- Prevention- Delay upright sitting till the child can come to sitting position independently, emphasize tummy time activities, physical therapy.
- **Monitoring:** Spine X-ray once child starts sitting independently.
- Treatment includes:
  - Casting between sitting age and 5 years old.
  - Bracing for curves less than 50 degrees.

- Surgery for curves more than 50 degrees.
- Non-fusion spinal intervention (NFSI) surgery between 5- 10 years.
- Spine fusion surgery: 12 years and older.

Pulmonary/ sleep problems by Dr Amee Revana

- Children with PWS have weak or uncoordinated swallowing and at risk of aspiration.
- Expiratory muscle weakness leads to poor ineffective cough, leading to respiratory failure and sudden death.
- Children with PWS do not demonstrate symptoms of acute illnesses- these are very subtle due to dysautonomia. This requires a high index of suspicion for any illness.
- Sleep-disordered breathing includes obstructive sleep apnea (OSA), central sleep apnea syndromes, sleep-related hypoventilation, or sleep-related hypoxemia.
- OSA presents as snoring, pauses in breathing, apneic or near apneic episodes, choking or gasping for air during sleep, restless sleep, excessive daytime sleepiness or fragmented sleep.
- Factors contributing to OSA include abnormal ventilatory control, anatomical abnormalities like micrognathia or small naso/ oropharynx or scoliosis, airway collapse due to hypotonia of pharyngeal wall, adenoid or tonsillar enlargement or airway narrowing due to obesity.
- Gold standard for diagnosis of Sleep-disordered breathing: Polysomnography.
- Treatment: Supplemental oxygen/ Positive airway pressure therapy/ medical management or surgical intervention.
- GH can cause growth of lymphoid tissue due to increase in IGF-1, leading to adenotonsillar hypertrophy.
- Sleep study is needed before and after initiation of GH.
- Trials of Pitolisant to improve daytime sleepiness in children with PWS are ongoing.

26TH ISPAE ACES MEETING: TECHNOLOGY IN TYPE 1 DIABETES MELLITUS (24 Dec 2023)

Experts- Professor Ben Wheeler, *Pediatric Endocrinologist, University of Otago and the Southern District Health Board,* ***New Zealand*** *and* ***M. Harsh Kohli, Idealite, Living with Type 1 Diabetes***

(Compiled by ***Zalak Upadhyay****, Pediatric and Adolescent Endocrinologist, Endocare for Kids, Rajkot)*

Measurement of Glucose: Glycated hemoglobin (HbA1c) provides only an average of blood glucose (BG) levels. Continuous Glucose monitoring (CGM) is the gold standard for BG monitoring, and should be started in every child with T1D at the time of diagnosis. Early initiation of CGM is associated with significant HbA1C reduction and better long term outcomes.

Real-time CGM is better than intermittently scanned CGM; gradually real-time CGM will take over. If one has to choose between CGM and insulin pump, CGM is far more important than insulin pump. The combination of CGM plus MDI also works very well.

On CGM, the 'time in range' (BG 70-180 mg/dL) should be >70%, 'time below range' (< 70 mg/dL) should be < 4%, and 'time above range' (> 180 mg/dl) < 25%. The emerging concept of 'time in tight range' expects the BG to be between 70-140 mg/dL for > 50% of time.

The CGM devices available in India are Medtronic Guardian and Abbott Libre Pro, Libre. As Libre Pro costs less, and lasts for 14 days, it is more widely used.

After insertion of a Libre/Libre Pro sensor, if it is activated after 8-12 hours (soak in time), the variability of readings during the first 24 hours is reduced. So activating the sensor 12h after insertion can be advised.

LibrePro and Libre are factory calibrated. If activation is done when BG readings are stable and between 70-180 mg/dL, the quality ofself-calibration is improved.

LibrePro has been developed for use by doctors (readers are supposed to be available only with doctors). The Glimp app can be used in place of a reader; Glimp works only on Android phones that are NFC enabled. OnePlus mobile phones may break the sensors and hence should be used with caution.

The Glimp app allows BG reading within 15 minutes of activation.

The Ultrahuman app has also been used to activate LibrePro sensors.

For Libre CGM, the Libre Link app can now be used on mobile phones that are NFC enabled.

LibrePro is intermittently scanned CGM. Devices like Miao Miao (MM) convert it to real-time CGM; MM can be used with all phones - NFC is not required. Other such devices are Bubble and Bluecon, but they have difficulties. Apps like Night Scout allow remote monitoring of the child's BG, by care givers, and provide for alarms. A mobile phone and an active internet connection are essential.

Downsides of technology- Skin hypersensitivity reactions to the adhesives can occur. Skin solutions are available on the website www.pantherprogram.org. There may be stress due to information overload, or the feeling of a device constantly attached to the body.

Insulin delivery: Automated insulin delivery (AID) is the current gold standard for management of T1D, but worldwide, as in India, equity of access is a big issue. AID provides better control, a more positive relationship with diabetes, and improved engagement of the diabetes team with parents. For the 780G insulin pump, we should remember the ABC approach.

- 'A' stands for active insulin time, which should be anywhere between 2-4 hours. If we want aggressive control, active insulin time can be reduced to 2 hours.
- 'B' is for basal target, which can be 120, 110 or 100 mg/dL. Basal insulin is secreted every 5 minutes, depending on the BG values.
- 'C' is for insulin:carb ratio. It's better to choose the formula of 400/total daily dose, as this helps control BG better. Adherence should be considered: sensor wear should be > 90%, and the infusion set should be changed every three days.

The next meeting will be held on **6th April 2024, 7pm onwards, on Turner syndrome.**

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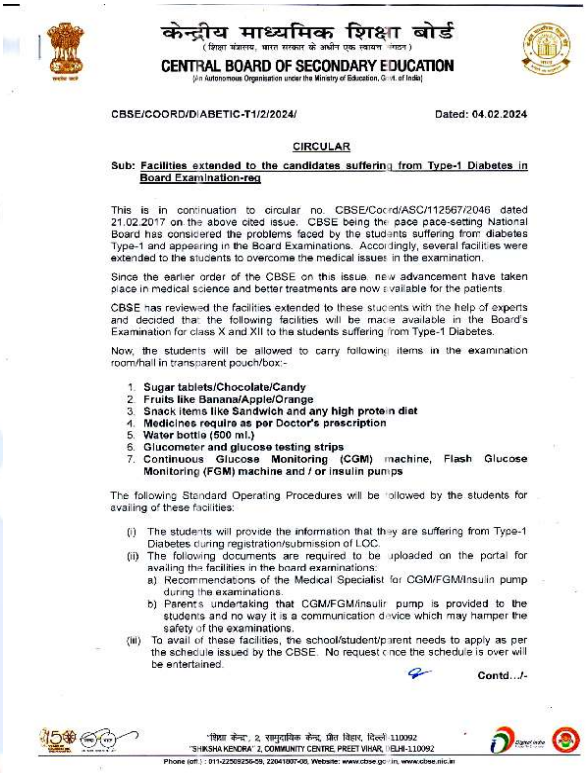
IDEAL/ IDEAS Report

PROVIDING HOLISTIC CARE - ISPAD - BEST - IDEAS - THE JOURNEY CONTINUES

Anju Virmani, Sirisha Kusuma Boddu, Preeti Singh, *Core Committee- IDEAL*

After the excitement of actually receiving the ISPAD award for "Innovation in Pediatric Diabetes Care 2023" in Rotterdam in October, meeting the entire IDEAL family in Bangalore in November, and all 6th batch IDEAL trainees (physicians) completing Exit exams with flying colors and becoming IDEALites, we have entered another promising year, another phase, another batch.

Batch 7 commenced in mid-January, and as per our past pattern, consists of non-physicians: nurses, dieticians, and educators already working in the T1D space. The program continues to generate excitement, and outreach results from IDEALites. The IDEALite WhatsApp group provides information, thoughtful discussions, and experience sharing, on an ongoing basis.



केन्द्रीय माध्यमिक शिक्षा बोर्ड
CENTRAL BOARD OF SECONDARY EDUCATION

CBSE/COORD/DIABETIC-T1/2/2024/ Dated: 04.02.2024

CIRCULAR

Sub: Facilities extended to the candidates suffering from Type-1 Diabetes in Board Examination-reg

This is in continuation to circular no. CBSE/Coord/ASC/112567/2046 dated 21.02.2017 on the above cited issue. CBSE being the pace pace-setting National Board has considered the problems faced by the students suffering from diabetes Type-1 and appearing in the Board Examinations. Accordingly, several facilities were extended to the students to overcome the medical issues in the examination.

Since the earlier order of the CBSE on this issue, new advancement have taken place in medical science and better treatments are now available for the patients.

CBSE has reviewed the facilities extended to these students with the help of experts and decided that the following facilities will be made available in the Board's Examination for class X and XII to the students suffering from Type-1 Diabetes.

Now, the students will be allowed to carry following items in the examination room/hall in transparent pouch/box:-

1. Sugar tablets/Chocolate/Candy
2. Fruits like Banana/Apple/Orange
3. Snack items like Sandwich and any high protein diet
4. Medicines require as per Doctor's prescription
5. Water bottle (500 ml.)
6. Glucometer and glucose testing strips
7. Continuous Glucose Monitoring (CGM) machine, Flash Glucose Monitoring (FGM) machine and / or insulin pumps

The following Standard Operating Procedures will be followed by the students for availing of these facilities:

(i) The students will provide the information that they are suffering from Type-1 Diabetes during registration/submission of LOC.
(ii) The following documents are required to be uploaded on the portal for availing the facilities in the board examinations:
a) Recommendations of the Medical Specialist for CGM/FGM/Insulin pump during the examinations.
b) Parents undertaking that CGM/FGM/insulin pump is provided to the students and no way it is a communication device which may hamper the safety of the examinations.
(iii) To avail of these facilities, the school/student/parent needs to apply as per the schedule issued by the CBSE. No request once the schedule is over will be entertained.

Contd.../-

"शिक्षा केन्द्र", 2, सामुदायिक केन्द्र, प्रीत विहार, दिल्ली 110092
"SHIKSHA KENDRA" 2, COMMUNITY CENTRE, PREET VIHAR, DELHI-110092
Phone (off.): 011-22509256-59, 22041807-08, Website: www.cbse.gov.in, www.cbse.nic.in

We had a flurry of activity in February when a child in Bangalore was refused permission for his insulin pump during the Board exams by CBSE, Karnataka. A protest lodged with Mr Priyank Kanungo, Chairperson, NCPCR, and with Ms Anu Choudhary, who recently completed her term as Registrar at NCPCR, yielded swift results. CBSE came out with their circular clarifying that children with T1D will be permitted sugar, snacks, glucometer, CGMS sensor, and insulin pump, as applicable, during Board exams. Most other Boards had already issued such letters last year, following the NCPCR directive in March 2023.

The order can be downloaded from here: https://ispae.org.in/diabetes-care/

The "Basic Education Series in Type 1 Diabetes" (BEST) course continues with its journey head-on, with 6 batches trained over the last 20 months (April 2022- Dec 2023). A total of 165 participants including parents/caregivers of T1D (38%), young adults with T1D (27%), nursing staff (19%) and healthcare assistive personnel (16%) have been trained from across the country. Notification is out for the next BEST program for parents (batch 7) due to start in April.

The monthly IDEAS sessions (1st Sunday of each month) of Diabetes Education for school teachers are slowly gaining traction. Dr Leenatha Reddy, Dr Shreeja M, and Ms Aishwarya were the faculty in January 2024. Then we went regional, with Dr Santhosh Olety, Dr Shaila Bhattacharyya, and Ms Sharanya Shetty doing a Kannada IDEAS in February, which was very well attended. In March, we had a session in Hindi, with the faculty being Dr Preeti Singh, Dr Deepika Harit, and Mr Sam Gulati. In April, we will return to English. We also have the Gujarati team of Dr Shalmi Mehta and Dr Ruchi Shah all set to go! In each one of these virtual programs, the backbone is Mr. Harsh Kohli, who was also faculty in the December ACES session.

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ACTIVITIES BY ISPAE MEMBERS

PEDICON 2024, Kochi, 24-28 January 2024

Preconference workshop- Dr Parvathy, Aster Medcity, Kochi

*Compiled by **Dr Dhanya Soodhana**, Pediatric Endocrinologist, Aster MIMS, Calicut*

The PEDICON 2024 pediatric endocrinology pre-conference workshop was meticulously organized by Dr Parvathy at Aster Medcity, Kochi, located against a picturesque backdrop of the Kochi backwaters, on 24 January, 2024. It was attended by 30 delegates from all over the country. The scientific sessions were led by Dr Vijayakumar M, Dr Riaz I and Dr Ahila Ayyavoo. The other speakers included Dr Zalak Upadhyay, Dr Deepa Anirudhan, Dr Ambika P Ashraf, Dr Veena V Nair, Dr Sheeja Madhavan and Dr Dhanya Soodhana. Growth charts and approach to short stature were discussed in detail. Dr Ambika P Ashraf (Birmingham, Alabama) spoke about obesity, type 2 diabetes mellitus and lipid disorders. The need for motivational speaking that is **O**bjective, **A**ffirmative, includes **R**eflective listening and **S**ummarizing (**OARS**), was emphasized. Thyroid disorders, atypical genitalia and pubertal disorders were also covered. The last session included practical aspects of insulin administration, meal planning, exercise, glucose monitoring and case-based discussion of complications of type 1 diabetes mellitus.

Main Conference

*Compiled by **Dr. Zalak Upadhyay**, Pediatric and Adolescent Endocrinologist, Endocare for Kids, Rajkot*

This year's National Conference of the Indian Academy of Pediatrics was held at Kochi from 24-28 January 2024. The theme was 'Global Warming and Child Health', emphasizing the detrimental effects of global environmental changes on children. Around 7,000 delegates from the country and abroad attended the four-day event.

The pediatric endocrinology symposium was held on 27 January. The unique concept of lectures by international faculty, under the banner of 'IAP Beyond the Borders CME', included Dr PSN Menon from Kuwait, Dr Ambika Ashraf from USA and Dr Pratik Shah from London. The topcs covered were Metabolic syndrome, advances in glucose monitoring, HRT in pubertal disorders in adolescents, short stature, newborn screening for congenital hypothyroidism, MODY, case based approach to pubertal disorders & thyroid disorders and discussion on DKA, hypoglycemia, precocious puberty and rickets. The event was graced by 30 National Faculty from different zones of India..

The more than 200 attendees appreciated the coverage of the topics by the esteemed Faculty.

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INTERNATIONAL PWS SCIENTIFIC AND FAMILY ORIENTED CONFERENCE, 2023

***Dr Jahnavi M**, Fellow, Dr **Kavitha Bhat,** Lead Consultant, Dr **Namratha Upadhya**, Consultant, Pediatric Endocrinology, Aster Hospitals, Bengaluru*

The Pediatric Endocrinology team from Aster Hospitals, Bangalore, backed by a conference grant from the International Prader-Willi Syndrome Organization (IPWSO) and Friends of IPWSO (USA),with support from the Indian Prader-Willi Syndrome Association (IPWSA), organized a groundbreaking scientific and family-oriented conference in Bangalore on December 16, 2023.

The hybrid event witnessed the active participation of 30 patients with their families and an audience of about 100 healthcare professionals physically and virtually. Experts specializing in PWS from renowned medical institutions all over the world were invited to give talks on their field of expertise. The conference also featured a unique pre-conference clinic, allowing patients from all over India the opportunity to consult various specialists under one roof.

WORLD OBESITY DAY 2024- LET'S TALK ABOUT OBESITY

***Dr. Priti Phatale and Dr. Hemant Phatale**, Samrat Endocrine Institute of Obesity, Diabetes & Thyroid, Aurangabad*

A series of events were organized with World Obesity Federation on the occasion of World Obesity Day. Dr Priti was faculty in an online webinar organized by the IAP subspeciality chapter on Nutrition on 4 March 2024. Awareness campaigns were organised at three different schools - 'Anant Bhalerao Vidya Mandir' attended by 85 parents and 25 school staff; Bhondawe Patil Public School' attended by 450 students, 40 teachers and 10 nonteaching staff; and 'Shishu Vikas Mandir', where more than 300 students and school staff were present.

An article on causes and consequences of obesity was published in Marathi in a daily newspaper of Aurangabad. All awareness campaigns were well appreciated, and impacted the knowledge and attitudes towards adopting a healthier lifestyle of different stakeholders.

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PEDIATRIC ENDOCRINE UPDATE, AHMEDABAD

Dr Shalmi Mehta and Dr Ruchi Shah at Endokids Clinic, Ahmedabad

A half day CME was organized on 25 February on Pediatric Endocrinology, in association with Academy of Pediatrics, Ahmedabad. Important areas like newer advances in diagnosis and management of short stature, new obesity guidelines, early maturity in girls, neonatal endocrinology and management of type 1 diabetes in resource limited and resourceful settings were discussed. Dr Vaman Khadilkar, Dr Shalmi Mehta, Dr Tushar Godbole, Dr Ruchi Shah and Dr Chirantap Oza graced the occasion as faculty. It was well attended by 60 practicing pediatricians and much appreciated by all.

CHILDHOOD AND ADOLESCENT DIABETES SUPPORT GROUP CAMP

Dr Sapna Nayak, Dr Sarah Mathai, Pediatric Endocrinology division, Department of Pediatrics, Christian Medical College

The 33rd Childhood and Adolescent Diabetes Support Group camp was held on 3rd February 2024 at Sneha Deepam, Vellore, to empower children living with Type1 Diabetes Mellitus (T1DM) and their families. The event was attended by 114 children and their parents. Families were educated on various aspects of T1DM management, including their mental health, through workstations, talks and role-plays. The Pediatric Endocrine, Pediatrics, Dietary, and Child and Adolescent Psychiatry teams of CMC, Vellore, were involved. Our previous beneficiaries with T1DM(now adults) shared their struggles and success stories that encouraged the families. All children received gifts.

The camp was held at subsided expenses. The subsidy was possible due to the fundraising “Give for Diabetes” sale and “5K Run for Diabetes” fun-run events conducted by the team on 25th November and 2nd December 2023, respectively.

EMPOWERING INDIVIDUALS WITH TYPE 1 DIABETES

Dr Ajinkya Patil, Pediatric Endocrinologist, HormoCare Clinic, Jalgaon, Maharashtra

A single day meeting was organized for the Type 1 Diabetes Support Group by HormoCare Clinic and ClubOne Adda, Jalgaon, in association with Club One KEM Pune and Mukul Madhav Foundation. It got an overwhelming response with 82 T1Ds and over 160 care givers. The event kick-started with an energetic Zumba session and outdoor games for care givers and kids. In order to empower families, measuring cups as well as educational material were distributed, along with Support Group T shirts to boost camaraderie.

The main event started with the annual report and general insights on activities of ClubOne Adda by Dr. Ajinkya Patil, followed by an interactive session on carbohydrate counting by IDEALite Mrs. Nilam Jani. The session aimed at busting myths and educating regarding carb counting in the context of local food patterns. Senior T1Ds shared their experiences to boost the morale of newly diagnosed T1D families. Children with outstanding management practices were awarded and felicitated. The event planning and coordination was handled by Mrs. Pritee Mandore, Mrs. Bhagyashri Dandi and IDEALite Mrs. Utpala Daryapurkar.

CELEBRATING DIABETES

***Dr Shalmi Mehta** and **Dr Ruchi Shah** at Endokids Clinic, Ahmedabad*

This event organized on 21st January, 2024, was attended by more than 70 children and their families. It began with Zumba dance, followed by a Fashion Show by young children. The older children performed skits based on school bullying and hypoglycemia that they had practiced through zoom meetings! Parents wholeheartedly participated in the friendly Educational Quiz. All children received a kit containing T1D ID card, Guide to school brochure, Diabetes management plan and a mug. Five teenagers living with T1D helped organize the event and explained the basic care of diabetes. The event ended with cake cutting (including a gluten free cake), followed by dinner. The event helped children and parents make new friends; new families got reassurance and confidence; we all got lifetime memories to cherish. The excerpts of the event can be viewed at this link: https://www.youtube.com/watch?v=Y1DUJxgYrw8.

HAPPIEST MIND RUN

***Dr Vani H N**, Pediatric Endocrinologist, Indira Gandhi Institute of Child Health (IGICH), Bengaluru, and **Dr Santhosh Olety**, Karnataka Institute of Endocrinology and Research (KIER), Bengaluru*

Happiest Minds Technologies organized the fourth edition of Happiest Minds Run, at HSR BBMP Ground, Bengaluru, on Sunday 17th December 2023, raising INR 10 lacs for Idhayangal Charitable Trust to aid children with Type 1 diabetes. Around 1300 participants came together to make it a success. Around 30 children with T1D participated in the event and completed 3-6 miles run. Executives Mr Venkatataraman Naarayan MD, Mr Joseph Anantharaju, Executive Vice Chairman of Happiest Minds Technology presided over the event. They emphasized the event's harmony between corporate excellence and health, promoted an active lifestyle and highlighted concerns of diabetes in children and adolescents. Proceeds will support around 40 underprivileged children with T1D for a duration of 12 months, benefiting from standard treatment at KIER and IGICH. The

event, sponsored by RBL Bank, ICICI Bank, and others, showcased the organization's wellness culture, celebrating fitness and contributing to charitable causes. Dr SanthoshOlety and Dr Vani received the donation on behalf of patients.

NZ-PEDICON 2023

Dr Sangita Yadav delivered a talk on Precocious Puberty highlighting the Recent trends of Puberty and Early Puberty at the NZ –PEDICON and PUNPEDICON 2023 organised by Sutlej Academy of Pediatrics and Jalandhar Academy of Pediatrics on 16 -17 December 2023 at Jalandhar. The conference was attended by more than 500 delegates. The update was found interesting, practical and useful and highly appreciated by the Practicing Pediatricians.

TRAINEES SECTION

BMI- BEST MATCH INDEX

Tejasvi Sheshadri, *Consultant Pediatric and Adolescent Endocrinologist, Sparsh Super Speciality Hospital, Shishuka Children's Hospital, Bengaluru*

Choose the best single match for the responses in column A with those in column B in both Sets of eight questions each.

https://forms.gle/nduraDPtP36XuXea9

Correct answers and individual quiz scores will be mailed at respective email ID after the quiz closes!

"LAST DATE- 10 April 2024"

APPES-ISPAE Joint Meeting 2024

The 13th Biennial Scientific Meeting of APPES, co-organised by the Asia Pacific Pediatric Endocrine Society (APPES) & Indian Society for Pediatric and Adolescent Endocrinology (ISPAE)

October 2 - 5, 2024
Yashobhoomi (India International Convention & Expo Centre), Dwarka, New Delhi

Each award with **1000 USD** Cash Prize

First registration slab closes 15 April 2024

APPES 2024 Best Presentation Award

The top 6 abstracts will be selected for oral presentation at the Kaichi Kida session. The top-scoring Oral presentation in the session will be awarded as APPES 2024 Best Presentation Award.

Human Growth Foundation Award

To be eligible for the Human Growth Foundation Award, your abstract must be related to Growth-related disorders.

Last date for abstract submission: 15th May 2024

Click here to submit your abstract now!

E: appes2024.delhi@gmail.com | W: www.appes2024.org