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# **Editor's Note**

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### Dear friends,

Greetings from Child India - post the mega success (from available feedbacks) of PEDICON 2024, Kochi.

On February 12th we celebrated the International Epilepsy Day 2024 focusing on the theme "Milestones on My Epilepsy Journey". The theme emphasises to highlight personal achievements despite the challenges brought about by the condition. It aims to encourage individuals to break the silence and share their successes.



**February** 

February 14 was Congenital Heart Defect (CHD) Awareness Day. This special day comes at the end of CHD Awareness Week (February 7-14) — a time to raise awareness about CHD, the most common type of birth defect. In India, approximately 2 lakh children are born with congenital heart disease every year. Around one-fifth of these suffer from critical heart disease.

International Childhood Cancer Day (ICCD) is observed on February 15 every year. The theme for this tear is 'Reducing the survival gap' to highlight the vital role of parents, as well as family doctors and pediatricians, in the early detection of childhood cancers. The target goal of the WHO Global Childhood Cancer Initiative is to achieve at least 60 percent survival for all children diagnosed with cancer around the world by 2030.

Rare Disease Day is a globally coordinated movement that occurs annually on the last day of February to raise awareness about rare diseases and their impact on patients and their families. This year it falls on 29th February with the theme "Year of the Zebra". The theme highlights the unique needs of the rare disease community. The zebra is the official symbol of rare diseases. The "Year of the Zebra" initia-tive highlights one rare disease each week on the healthcare video education channel, Osmosis from Elsevier. The goal is to educate healthcare professionals and students and to speed up rare disease diag-nosis and research.

Child India's academics will focus on pediatric emergencies as our dear President, Dr Basavaraja GV is an intensivist. This year's February issue, deals with Pediatric Endocrine Emergencies and we profusely thank the contributors, especially Dr Parvathy L for coordinating the inputs for this issue.

### Happy reading,

Regards and wishes for a great year of IAP activities.

Jai IAP!

Dr Jeeson C Unni Editor-in-Chief

# **President's Address**

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Greetings fellow pediatricians!,

As we step into the month of March, let's dive deep into the topics covered under the IAP Presidential Action Plan's Training of Trainers (TOT) programs. These initiatives are designed to equip us with the latest knowledge and skills to enhance pediatric healthcare delivery.

The IAP Presidential Action Plan has initiated a series of Training of Trainers (TOT) programs across various zones, each addressing crucial aspects of pediatric healthcare. These programs serve as

pivotal platforms for advancing knowledge and skills among healthcare professionals, ensuring optimal care for children and families.

In the South Zone TOT program, the focus is on bridging theory with practice in vaccinology. This initiative recognizes the paramount importance of vaccines in preventing infectious diseases and safeguarding child health. By providing practical training on vaccine administration techniques and immunization schedules, healthcare providers are equipped to effectively implement vaccination programs, thus bolstering community immunity and reducing the burden of vaccine-preventable illnesses.

Moving to the Central Zone, the IAP-FM module at Vizag delves into the management of fever, a common yet significant concern in pediatric practice. Fever serves as a cardinal symptom of various underlying infections and systemic illnesses in children. Through comprehensive training on fever assessment, differential diagnosis, and appropriate management strategies, healthcare professionals can accurately identify and treat febrile conditions, thereby promoting timely recovery and minimizing complications.

In the North Zone TOT in Vaccinology, the emphasis lies on translating theoretical knowledge into practical applications in vaccine delivery. This initiative aims to enhance healthcare providers' proficiency in vaccine storage, handling, and administration, ensuring vaccine potency and efficacy. By strengthening the immunization infrastructure and promoting vaccine adherence, this program contributes to achieving optimal vaccine coverage and disease prevention.

The launch of the Diagnostic Stewardship Module in the South Zone, specifically in Chennai, underscores the importance of judicious utilization of diagnostic tests in pediatric practice. Healthcare providers are trained to employ evidence-based diagnostic algorithms and guidelines, thereby optimizing diagnostic accuracy and minimizing unnecessary testing. This prudent approach to diagnostics not only enhances patient









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care but also conserves healthcare resources and mitigates the risk of diagnostic errors.

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Furthermore, the West Zone and East Zone IAP FM TOT programs, both focusing on fever management, equip healthcare professionals with the knowledge and skills to effectively evaluate and manage febrile illnesses in children. By emphasizing evidence-based approaches to fever assessment, diagnosis, and treatment, these programs empower clinicians to deliver timely and appropriate care, thereby improving clinical outcomes and patient satisfaction.

Overall, the launch of these TOT programs under the IAP Presidential Action Plan signifies a concerted effort to enhance pediatric healthcare delivery through targeted training and capacity-building initiatives.

By addressing key areas such as vaccinology, fever management, and diagnostic stewardship, these programs aim to elevate the standard of care for children across different regions, ultimately contributing to improved health outcomes and well-being.

Let's make the most of this month by delving into these critical topics and enhancing our expertise in pediatric care. Together, we can strive towards providing optimal care for our young patients and their families. Stay tuned for valuable insights and discussions on these essential subjects throughout March!

Warm regards,

**Dr. G V Basavaraja** National IAP President, 2024

# Secretary's Message

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Respected Seniors and Dear Friends,

### "Teamwork is the secret that makes common people achieve uncommon results."

February is the month where activities and IAP Action Plan modules under the Presidential ship of Dr G V Basavaraja, National President IAP 2024 were implemented as follows. Indian Academy of Pediatrics (IAP) has virtually launched a pioneering flagship program called "IAP Ki Baat, Community Ke



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Saath" to actively engage with communities and provide essential information on child health at National Indian Pediatrics Office at Navi Mumbai on 13th February, 2024. The first topic to be covered is "Anemia Ki Baat, Community Ke Saath,". Distinguished guests, including Dr Vinod K Paul- Member, NITI Aayog, Govt of India and Dr Pukhraj Bafna – Padmashree Awardee and Sr Consultant Pediatrician, virtually unveiled the awareness poster and video on Anaemia.

This day also witnessed virtual launch of IAP PG Clinic Program also, by National President Dr G V Basavaraja 2024 along with Secretary General 2024-2025 Dr Yogesh N Parikh. This event took place from National IAP Office, Navi Mumbai.

The following meetings were conducted via video conferencing in the month of February 2024 such as 3rd Office Bearer Meeting, Charity Advocate Meeting with Advocate Ghanshyam Hule, Finance Committee Meeting with the committee members, NC ECD Committee Meeting, IAP NRP FGM meeting with Kenvue, Meeting with ICP new office bearers, SOP Book Committee meeting also took place followed by ACVIP Committee meeting and also "HOW TO INTERACT WITH THE MEDIA EFFECTIVELY" : Using Print, TV and Social Media Platforms workshop took place under "IAP ki Baat, Community Ke Sath"

Along with this, Indian Academy of Pediatrics conducted 7 Zonal ToTs on the following modules under the Presidential Action Plan 2024 - East Zone : IAP FM ToT at Guwahati, South Zone : IAP FM ToT at Bangalore and IAP Vaccinology ToT at Coimbatore, Central Zone : IAP FM ToT at Vizag, West Zone : IAP FM ToT at Navi Mumbai, North Zone : IAP Vaccinology ToT and Diagnostic Stewardship ToT at Jaipur.

Also, this month observed successful conduction of total 30 Basic NRP and 9 Advanced NRP provider workshops

On behalf of IAP, I urge you to organize various activities in the best interest of the health and welfare of the country's children.

### "Let's Lead IAP Into A New Era of the Growth"

In service of Academy,

**Dr Yogesh N Parikh** Secretary General, IAP 2024 & 2025





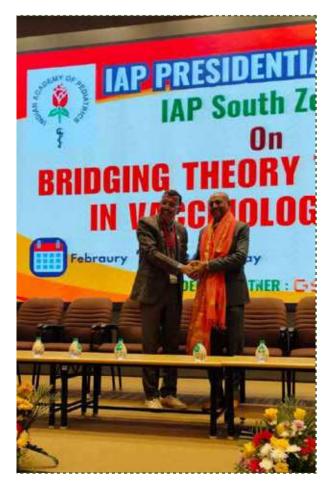
All about fever - Zonal ToT at Bangalore on 4th February



IAP Presidential Action Plan - IAP South Zone TOT in Coimbatore















Launch of "IAP ki Baat, Community Ke Saath" - first topic "Anemia Mukt Bharat"





# IAP Ki Baat, Community Ke Saath is now a Sensation!

## We are on every news paper across country! Lets thrive to ensure Authentic, Relavant and reliable Childcare Information reaches to all





IAP-FM module at Vizag, **Central Zone TOT Guidance of National President Dr G V Basavaraja Central Zone Vice President Dr Ajay Srivastava , National Treasurer Dr Athanu Bhadra** 













**Daignostic Stewardship Module** Launched under the guidance of South Zone Vice President Dr Singaravelu on 18-02-204 at Chennai





Infectious Diseases CME at Bangalore under Guidance of Dr Raghunath C N,Dr A R Somashekar inauguration by National President Dr G V Basavaraja on 18-02-2024 Sunday Epidemics of Academics accross the Nation





Vaccinlogy North Zone TOT Bridging Theory to Practice under the guidance of President Elect Dr Vasanth Khalatkar, HSG- Dr Yogesh Parikh, Senior Vice President Dr Satish Sharma at Jaipur on 18-02-2024









### West Zone IAP-FM TOT at Mumbai on25-02-2024

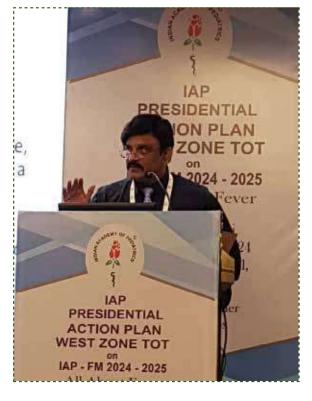














East Zone IAP-FM TOT at Gawathi on25-02-2024

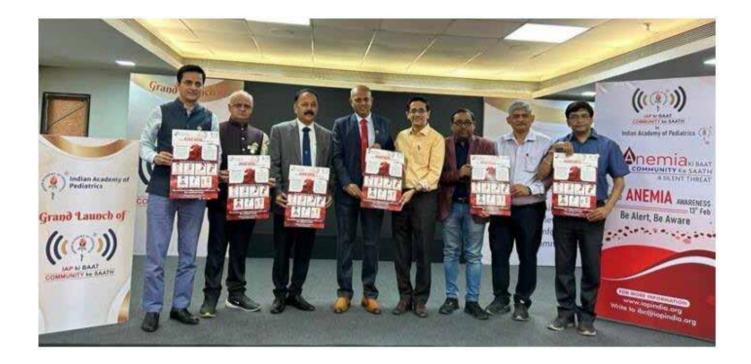
























**Greetings from Indian Academy of Pediatrics!** 

Kindly note the following official email Id's of Indian Academy of Pediatrics for communication and reference.

For all correspondence

Central office - centraloffice@iapindia.org

■ For submitting activities report, UG & PG quiz communication, changes in office bearers of State/City/District branches

IAP Activities - iapactivities@iapindia.org

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Communication related to NC ECD, NTEP

IAP Project - iapprojects@iapindia.org

■ Communication related to membership, branches (formation, submission of branch documents)

Membership IAP - iapmembership@iapindia.org

Communication realted to Presidential Action Plan 2024 – IAP ki Baat Community ke Sath

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# **Diabetic Ketoacidosis**

### DR. AKANKSHA PARIKH Consultant Paediatric and Adolescent Endocrinologist Kokilaben Dhirubhai Ambani Hospital Mumbai



Diabetic ketoacidosis (DKA) is a lifethreatening complication of uncontrolled diabetes. It is the presenting manifestation in up to 70% of children with type 1 diabetes and up to 25% of children with type 2 diabetes [1]. In children with known diabetes, DKA occurs due to missed insulin doses or is precipitated by an infection.

Presenting complaints are non-specific and include abdominal pain, nausea, vomiting, rapid breathing and altered sensorium. Children are often misdiagnosed with a gastrointestinal illness, acute abdomen, or pneumonia. Thus, all children with such complaints, especially when unexplained, should have a blood glucose level checked.

### Diagnostic criteria for DKA (all three criteria required)

- Hyperglycemia blood glucose (BG) >200 mg/ dL
- Ketosis Urinary ketones < 2+ (moderate to large) or blood ketone (beta-hydroxybutyrate)
   > 3 mmol/L
- Acidemia pH > 7.3 or serum bicarbonate < 18 meq/L

### **Severity of DKA**

- Mild: venous pH < 7.3 or serum bicarbonate</li>
   < 18 meq/L</li>
- Moderate: venous pH < 7.2 or serum bicarbonate < 10 meq/L</li>

Severe: venous pH < 7.1 or serum bicarbonate</li>
 5 meq/L

### Initial management of DKA

- Follow PALS technique
- Assess vitals including BP and measure weight and height/length
- Assess level of consciousness by Glasgow coma scale (GCS)
- In an unconscious child secure airway, provide oxygen and insert nasogastric tube
- Assess severity of dehydration (clinical markers may be unreliable); > 10% dehydration is indicated by weak peripheral pulses, hypotension or oliguria
- Initiate O2 therapy in circulatory shock
- Insert two separate peripheral intravenous (IV) catheters for insulin and fluid therapy (avoid central line)
- Send blood investigations as shown in Table 1
- Avoid bladder catheterization unless severely ill

### **Goals of therapy**

- Correct dehydration and electrolyte imbalance
- Correct acidosis and reverse ketosis with insulin
- Restore blood glucose gradually to normal

• Monitor for complications of DKA and its treatment

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Identify and treat any precipitating event

### **Fluid therapy**

### **Initial resuscitation**

- 10-20 ml/kg over 20 to 30 mins with 0.9% normal saline (NS)
- In shock administer 20 ml/kg rapidly and repeat as necessary; consider use of inotropes
- Insulin is initiated only after the fluid bolus

# Fluid deficit and maintenance over 24 to 48 hours:

- Assume deficit of 5%, 7% and 10% in mild, moderate, and severe DKA respectively
- Administer fluids (ml/hr) as (deficit + maintenance bolus)/24 or 36 or 48 hrs; consider 1 x maintenance fluid or 1.5 x or 2 x accordingly
- Choice of fluids: 0.45 0.9% NS or Ringer's lactate or Plasmalyte
- Switch to 5% DNS if BG < 250 300 mg/dL and 10% dextrose with NS if BG < 150 – 200 mg/dL

### Serum electrolytes

### Potassium

- If < 3.5 meq/L Administer potassium bolus concomitantly with initial fluid bolus through a separate IV line at the rate of 0.5 meq/kg/ hour under cardiac monitoring; defer insulin till potassium is corrected
- If 3.5 5.5 meq/L initiate potassium in fluid therapy after initial bolus @ 40 meq/L
- If > 5.5 meq/L add potassium after the child passes urine; defer potassium only in cases of renal failure

Potassium can be administered as a mixture of 20 meq/L potassium chloride and 20 meq/L potassium phosphate to prevent hyperchloremia and hypocalcemia as adverse effects respectively which occur when a single formulation is used

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- **Phosphate:** Requires treatment if < 1mg/dl or symptoms such as rhabdomyolysis, ileus, encephalopathy or respiratory failure develop
- **Bicarbonate:** Bolus is contraindicated; except in life-threatening hyperkalemia and impaired cardiac contractility

### **Insulin therapy**

- Dilute 50 units of regular insulin in 50 ml of 0.9% NS such that 1 ml = 1 unit
- Flush the insulin tubing before initiating insulin infusion
- Start insulin infusion only after initial fluid bolus @ 0.05 0.1 units/kg/hour; lower dose is preferred in younger children and in whom risk of cerebral edema is greater
- Insulin bolus is contraindicated and increases risk of circulatory shock due to sudden drop in osmolality and life-threatening hypokalemia
- Target reduction in blood glucose by 40 – 90 mg/dL/hour, increase in serum sodium by 0.5 meq/L/hour and reduction in betahydroxybutyrate by 0.5 mmol/L/ hour
- Insulin infusion is to be continued till resolution of DKA (pH > 7.3, serum bicarbonate > 18 meq/L and blood ketones < 1 mmol/L)</li>
- In case IV insulin infusion is not feasible, rapid acting or short-acting (regular) insulin can be administered subcutaneously at the dose of 0.15 units/kg/hour 2nd hourly and 4th hourly respectively (after the initial fluid bolus)

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# Transitioning from IV to subcutaneous (SC) insulin in DKA

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- Transition to SC insulin only when acidosis resolves, ketosis improves, and child is ready to eat; absence of ketosis should not be a criterion, especially when monitoring urine ketones, which take longer to resolve than blood ketones
- Administer rapid acting or short acting insulin SC in the dose of 0.25 units/kg, 15 to 30 mins before a meal respectively; IV insulin infusion is continued till 15-30 mins after rapid-acting and 1-2 hours after regular insulin bolus
- Initiate SC long-acting basal along with the bolus dose according to the basal bolus regimen; basal insulin can also be administered the previous evening with tapering and stopping of IV insulin infusion next morning to ensure a smoother transition and relative euglycemia
- Monitor BG levels frequently (3-4 hourly) to address any hypo/hyperglycemic episodes

### **Clinical and biochemical monitoring**

- Hourly heart rate, respiratory rate, blood pressure, GCS score, input and output charting, BG, serum osmolality and IV insulin rate
- 2-4 hourly pH, serum bicarbonate, pCO2, serum electrolytes, BUN, calcium and phosphate
- Persistence of acidosis with improvement in clinical signs usually occurs due to benign hyperchloremia (Cl: Na > 0.79) secondary

to high chloride content of fluid therapy; in contrast, a lack of clinical improvement indicates inadequate fluid resuscitation or insulin delivery, and untreated underlying infection

### **Complications of DKA**

### Cerebral edema

- Usually occurs in the first 12 to 18 hours of starting treatment; rarely at presentation or after 24 hours of therapy
- High index of suspicion is required and management should start early; risk factors include young age, longer duration of symptoms, severe dehydration and acidosis at presentation, administration of bicarbonate/ insulin bolus in first hour of treatment
- Can present as new onset or worsening of headache, altered sensorium, focal neurological deficit, elevated blood pressure, bradycardia and abnormal respiration
- Secure airway if required, elevate head end by 30°, adjust fluids to 2/3rd of calculated rate, administer hyperosmolar agents such mannitol or 3% NS @ 0.5-1 g/kg and 2.5 – 5 mmol/L over 10-15 minutes respectively; dose can be repeated after 30 minutes
- Cranial imaging should be performed only after initial management of cerebral edema
- Other complications: dyselectrolemia, hypoglycemia, deep vein thrombosis, stroke, mucormycosis and pulmonary thromboembolism



Table 1. Investigations and calculations at diagnosis

Parameter	Comment
VBG – pH, bicarbonate, pCO <sub>2</sub>	To look for acidemia and classify
	severity of DKA; pCO <sub>2</sub> < 20 mmHg
	indicates severe DKA
Anion gap (mmol/L)	$AG = Na - Cl + HCO_3$ ; normal: $12 \pm 2$ ;
	in DKA: 20-30; suspect concomitant
	lactic acidosis if $> 35$
RBS (mg/dL)	To confirm hyperglycemia observed on
	point of care device
Blood beta-hydroxybutyrate (mmol/L)	To confirm diagnosis of DKA; urine
	ketones to be checked if bedside serum
	ketones not available
CBC	A high WBC count without fever is
	commonly seen as stress response and
	does not always indicate an underlying
	infection
Serum sodium (meq/L)	Corrected sodium: measured Na +
	1.6([plasma glucose– 100]/100) mg/dL
Serum potassium (meq/L)	Maybe low, normal or high despite a
	low total body potassium level
Serum chloride (meq/L)	Usually normal at diagnosis; can
	increase with subsequent fluid therapy
Effective osmolality (mOsm/kg)	(2 x Na) + BG/18 (normal 275 – 295)
BUN, creatinine	BUN > 20 mg/dl indicates severe
	dehydration
Serum calcium/phosphorus/magnesium	Phosphorus, magnesium may be low at
	presentation
HbA1c	To confirm diabetes and assess duration
	of hyperglycemia
Blood cultures	If positive history of fever
ECG	If potassium levels cannot be assessed in
	time
Urine routine	To look for urinary tract infection

Reference:

1. Glaser N et al. ISPAD clinical practice consensus guidelines 2022: Diabetic ketoacidosis and hyperglycemic hyperosmolar state. Pediatric diabetes. 2022 Nov;23(7):835-56.

# **Adrenal Insufficiency In Children**

**DR. V. SHOBI ANANDI** Consultant Pediatric Endocrinologist Sri Ramakrishna Multispecialty Hospital Coimbatore



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### Definition

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insufficiency(AI) Adrenal is а lifethreatening condition that occurs secondary to impaired secretion of adrenal glucocorticoid and mineralocorticoid hormones. Primary AI occurs due to adrenal gland pathologies (such as autoimmune Addison's disease or congenital adrenal hyperplasia) and is usually associated with both glucocorticoid and mineralocorticoid deficiency. Secondary AI is due to pituitary gland pathologies (e.g. hypopituitarism) resulting in deficiency of ACTH causing cortisol deficiency. Tertiary AI may result due to exogenous high dose glucocorticoid use causing suppression of ACTH.

An acute adrenal crisis (AC) is a lifethreatening deterioration due to glucocorticoid insufficiency which may result in cardiovascular instability, abnormalities of plasma electrolytes, seizures and death. It may be the first presentation of an underlying AI or may be precipitated by physiological stress like intercurrent illness, surgery or trauma in a susceptible patient.

### **Clinical features**

The clinician must have a high index of suspicion because the symptoms of AI may be vague and undefined. The major presenting symptoms and signs include those related to cortisol deficiency causing weakness, fatigue, weight loss, anorexia, nausea, vomiting, hypotension and hypoglycemia. In primary adrenal hypofunction, cortisol deficiency is associated with mineralocorticoid deficiency resulting in hyperkalemia, hyponatremia, acidosis, hypochloremia, dehydration, cardiovascular collapse, shock and death.

Associated findings may include the appearance of small heart on chest Xray, anemia, azotemia, eosinophilia and lymphocytosis.

In primary adrenal failure, low concentrations of plasma cortisol stimulate hypersecretion of ACTH and other Pro-opiomelanocortin (POMC) peptides like Melanocyte Stimulating Hormone (MSH) causing hyperpigmentation of skin and mucus membrane.

Additional signs and symptoms may arise from underlying cause of adrenal insufficiency; eg. associated autoimmune disorders, neurological features of adrenoleukodystrophy or disorders leading to adrenal infiltration.

Adrenal crisis is a medical emergency with hypotension, marked acute abdominal symptoms and specific laboratory abnormalities requiring immediate treatment.

### Investigations

Investigations to be done in all cases of possible adrenal insufficiency / crisis include:

- Immediate blood glucose using a bedside glucometer.
- Serum glucose, urea, sodium and potassium.



• Arterial or capillary blood gas.

In many cases the underlying diagnosis will be known. However, if this is the initial presentation, the following investigations will help with diagnosis:

- Serum Cortisol, ACTH
- Plasma Renin Activity and Plasma Aldosterone levels
- 17 hydroxyprogesterone (in suspected CAH)

Acute adrenal insufficiency is a life threatening emergency, and treatment should not be delayed while waiting for definitive proof of diagnosis. However, when possible, these investigations should be performed prior to administration of steroids, as this helps greatly with the interpretation.

The diagnosis of primary adrenal insufficiency is verified by low morning cortisol levels (<5 mcg/dL) with high ACTH (>2-fold the upper limit of the reference range) and confirmed by a minimal response of cortisol to a 60 min intravenous ACTH test. An elevated plasma renin activity in combination with an (inappropriately) normal or low serum aldosterone levels is suggestive of associated mineralocorticoid deficiency.

### Treatment

Rapid recognition and prompt therapy of a salt losing crisis are critical to survival. Any patient with vascular tone insufficiency (hypotension), shock that is unresponsive to initial resuscitation, and/or an abnormal electrolyte profile (low sodium or high potassium level, low glucose level, or metabolic acidosis) should be considered to have adrenal insufficiency until proven otherwise.

- 1. Fluid replacement: Fluid and electrolyte therapy must be instituted as soon as possible.
- Rapid infusion of 10-20 ml/kg boluses of isotonic saline within the first hour of treatment; boluses may be repeated (up to 60

ml/kg) until circulation is restored.

- Subsequent fluid administration includes deficit, on-going losses and daily maintenance fluids requirements. The calculated fluid is administered as isotonic crystalloids containing dextrose, usually 5% dextrose with normal saline.
- 2. Steroid replacement:
- Patients with suspected adrenal crisis should be treated with an immediate parenteral injection of Hydrocortisone at a dose of 50 -100 mg/m<sup>2</sup>. If intravenous (iv) access is not immediately available, the dose is given as an intramuscular injection while establishing intravenous access. This is followed by iv Hydrocortisone at 50 - 100 mg/m<sup>2</sup>/day in 4 divided doses. Instead, a continuous infusion of hydrocortisone at 100mg/m<sup>2</sup>/24 hours after the initial bolus dose can be given.
- If hydrocortisone is unavailable, prednisolone can be used as an alternative.
- Rapid tapering and switch to oral regimen depending on the clinical state of the child is preferred. Once child is stable and tolerating oral intake, oral hydrocortisone is started at 30-50 mg/m<sup>2</sup>/day and reduced to maintenance levels depending on the etiology (10-15 mg/ m<sup>2</sup>/day in primary adrenal insufficiency or 6-8 mg/m<sup>2</sup>/day in secondary adrenal insufficiency) over about 5 days.
- Mineralocorticoid replacement: Initial correction is achieved with saline, fluids and the mineralocorticoid activity of stress dose hydrocortisone. Oral fludrocortisone should be re-commenced when tolerated
- 3. Management of hypoglycemia:
- Hypoglycemia is common in infants and small children with adrenal insufficiency.
- It can be corrected with intravenous bolus of 2-5 ml/kg 10% dextrose followed by maintenance fluids in Dextrose Normal Saline.



10% dextrose normal saline may be needed to maintain normoglycemia.

- Blood Glucose levels should be monitored hourly initially, then 2-4 hourly and less frequently once stable.
- 4. Management of hyperkalaemia:
- Hyperkalemia usually normalizes with fluid, electrolyte and steroid replacement.
- Patients with a K+ above 6.0 mEq/l should have ECG and be on cardiac monitor.
- Hyperkalaemia is potentially life-threatening and can lead to cardiac arrhythmias so additional measures such as the use of IV calcium gluconate, nebulised salbutamol, IV insulin and dextrose or IV bicarbonate and cation exchange resins should also be considered.
- 5. Management of Precipitating Causes:
- Identification and treatment of the stressor precipitating the adrenal crisis is of paramount importance.

### Prevention of adrenal crisis in a susceptible child

Adrenal crisis in patients with known primary adrenal insufficiency is best prevented by patient education and increasing the glucocorticoid dosage in situations of stressors known to increase cortisol requirements.

 During illness with fever: Hydrocortisone replacement doses are doubled (>38°C) or tripled (>39°C) until recovery (usually 2-3 days); increased consumption of electrolyte containing fluids as tolerated is recommended.

- If the child is unable to tolerate oral medication due to gastroenteritis or trauma: Hydrocortisone 50mg/m<sup>2</sup> is given as an intramuscular injection. A rough estimate of the required stat dose being; Infants- 25 mg, School age children- 50mg, adolescents-100 mg.
- Surgery or anesthesia: Hydrocortisone 50 mg/m<sup>2</sup> iv followed by 50-100 mg/m<sup>2</sup>/day in 4 divided doses.
- All susceptible patients should be equipped with a steroid emergency card and medical alert identification to inform health personnel of the need for increased glucocorticoid doses to avert or treat adrenal crisis and the need of immediate parenteral steroid treatment in the event of an emergency.

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- 4. Emergency and peri-operative management of adrenal insufficiency in children and young people: BSPED consensus guidance. BSPED: Paediatric Adrenal Insufficiency. Full Guidance. July 2023
- 5. Miller WL, Fluck CE. Adrenal cortex and its disorders. In: Sperling MA, editor. Pediatric Endocrinology 5th Edition. Elsevier; 2021. p. 460-467.



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Hypocalcemia is one of the commonest disorders of calcium and phosphate metabolism seen in children. It can be asymptomatic or have clinical symptoms which varies with age of presentation.Hypocalcemia is more common in the newborn period.It can occur as a result of inadequate calcium supply following an acute increase in plasma phosphate concentration, impaired PTH (Parathyroid hormone) secretion or PTH resistance (Pseudohypoparathyroidism). Hypomagnesemia can impair PTH secretion and also cause resistance to PTH action and hence manifest with symptoms of hypocalcemia. Impaired absorption of calcium from the gut due to Vitamin D deficiency/errors in Vitamin D metabolism can also cause hypocalcemia. The definition of hypocalcemia in term neonate is total serum calcium concentration less than 8 mg/dl and less than 7mg/dl in preterm neonate and in children is below 8.5 mg/dl.

hild India

### Normal calcium homeostasis

Two major interrelated calciotropic hormonal systems, Parathyroid hormone(PTH) and vitamin D are central to calcium homeostasis and function as described below. **Parathyroid hormone** – PTH serves as the primary protection against hypocalcemia and is released from the parathyroid chief cells.The calcium sensing receptor (CaSR) resides on the plasma membrane of the chief cells and serves as a "calcistat" which continuously senses ionised calcium concentration leading to rapid increase or decrease in PTH concentration.

PTH acts to increase serum calcium by mobilizing calcium from the bone by stimulating osteoclastic bone resorption and stimulating 1-alpha-hydroxylase in the kidney thereby converting 25-hydroxyvitamin D to 1,25-dihydroxyvitamin D which directly increases intestinal calcium absorption.PTH also increases calcium reabsorption in the distal renal tubule.

**Vitamin D** – 1,25-dihydroxyvitamin D,the active metabolite of Vitamin D,promotes calcium and phosphorus absorption from the gut.Lesser effects on calcium reabsorption from the renal tubules may also occur.

February



### Aetiology of hypocalcemia in newborn

Thild India

Early neonatalLate neonatalhypocalcemia(<72 hrs)</td>hypocalcemia (>72 hrs)

### 1. Neonatal illness

Prematurity	High dietary phosphate load (cow milk )
Birth asphyxia	Maternal Vitamin D deficit
IUGR	Hypomagnesemia
Sepsis	Hypoparathyroidism
Respiratory distress	Chronic renal insufficiency
Citrated blood/alkali	Pseudohypoparathyroidism
Phototherapy	Mitochondrial fatty acid d/s
	Infantile osteopetrosis

### 2. Maternal illness

Diabetes mellitus

Toxemia of pregnancy

Hyperparathyroidism

### Aetiology of hypocalcemia in children

# 1. Inadequate Calcium supply/increased requirement by skeleton

Maternal nutritional Calcium and Vitamin D deficiency

Malabsorption

'Hungry bone syndrome'-post parathyroidectomy/rapid healing of severe Vitamin D deficiency,rickets.

# 2. Decrease in ionised calcium concentration

Chelation eg-blood transfusion

Respiratory or metabolic alkalosis

### 3. Phosphate overload

Acute or chronic renal failure

Crush injuries and rhabdomyolysis

# 4. Hypoparathyroidism and parathyroid hormone resistance

Transient (secondary to maternal hypercalcemia)

Autosomal dominant hypocalcemia

Congenital agenesis and hypoplasia of parathyroid glands(DiGeorge syndrome,HDR syndrome,Sanjad-Sakati syndrome,Kearns-Sayre syndrome,Pearson syndrome)

Idiopathic hypoparathyroidism

Autoimmune polyendocrinopathycandidiasis-ectodermal dystrophy(APS Type 1)

Post thyroidectomy/parathyroidectomy

Radiation to neck

Iron deposition in parathyroid glands in thalassemia major

Pseudohypoparathyroidism

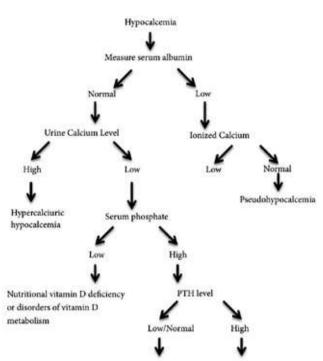
Hypomagnesemia

### History and physical examination

Children who may be prone to hypocalcemia include those whose dietary problems predispose to vitamin D deficiency, premature infants, congenital heart defects, hypoparathyroidism, pseudohypoparathyroidism or chronic renal disease. Signs of hypocalcemia include lethargy, poor feeding, apnea and vomiting in infants, jitteriness, irritability, muscle spasm, laryngeal stridor, tetany, tachycardia and congestive heart failure. Generalised seizures are common, so hypocalcemia should be ruled out in any child with an unexplained seizure. Carpopedal spasm is typical sign of hypocalcemia and facial muscle hyperirritability (Chvostek sign) may be elicited by tapping on the maxilla.Trousseau sign may be elicited in hypocalcemic patients by inflating a sphygmomanometer to 20 mm Hg above the systolic pressure.

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Hypoparathyroidism Pseudohypoparathyroidism

### Approach to diagnosing Pediatric hypocalcemia

- Obtain a complete history and perform a physical examination to detect clinical features of hypocalcemia/those that may point to a specific underlying disorder
- Confirm the diagnosis of hypocalcemia by measuring total serum calcium and albumin simultaneously or by measuring ionized serum calcium
- If the diagnosis is confirmed, order a hypocalcemic panel - Calcium, Phosphorus, Alkaline phosphatase, Magnesium, Creatinine, 25 hydroxy vitamin D, intact Parathormone (PTH) levels and test the ratio of calcium to creatinine in patient's urine.
- Order a test for 1, 25-dihydroxyvitamin D – However this test is of limited clinical use outside subspecialty clinics due to slow laboratory turnaround and variable assay quality.

### **Treatment of hypocalcemia**

IV 10% Calcium gluconate solution (0.5 ml/kg) to a maximum of 20 ml diluted 1:1 with 5% dextrose should be administered to infants and children with symptomatic hypocalcemia like seizures.This is infused over 10 minutes under cardiac monitoring.Caution to place IV line in a large vein to avoid extravasation into subcutaneoustissues,scarringandtissuenecrosis. Once patient is asymptomatic,a maintenance infusion of 10% Calcium gluconate(1-5 ml/kg/ day) or oral calcium supplements(50-75 mg/kg/ day of elemental calcium in four divided doses) can be supplemented.For older children and adolescents,the maximum dose of oral calcium supplements is 2gm/day.

In the management of hypoparathyroidism or pseudohypoparathyroidism,the use of a vitamin D analogue such as alphacalcidol or calcitriol in a dose of 15-25 ng/kg/day with calcium supplementation is the line of management to maintain serum calcium in the low normal range.

In hypocalcemia secondary to magnesium deficiency,intramuscular (IM) 50% Magnesium sulphate solution (100 mg/kg or 0.2 ml/kg/dose) is used. Two doses 12 hours apart are sufficient for most cases of neonatal hypocalcemia. Oral magnesium salts at a dose of 1-2 meq/kg are continued in patients with primary defects of magnesium metabolism.

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# Hypoglycemia in neonates and children

Thild India

**DR ZALAK UPADHYAY** Pediatric And Adolescent Endocrinologist Endocare For Kids, Rajkot, Gujarat



February

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Hypoglycemia constitutes one of the major metabolic emergencies at any age with potentially devastating consequences for the brain and neurological sequelae. It could be an important presenting feature of underlying endocrine/ metabolic problems requiring prompt treatment. Hypoglycemia should be excluded as the cause of an initial episode of convulsions, coma or neurobehavioural alteration in children.

Blood glucose homeostasis in the human body requires synchronous coordination of substrate availability, hormonal activities and metabolic events. The glucose requirement of infants is quite large (8mg/kg/min) as compared to that of adults (2-3 mg/kg/min) and is related to larger percentage of total body mass that the newborn brain occupies.

Hypoglycemia is the most frequent metabolic abnormality in the newborn and is a source of clinical concern and controversy, as no consensus exists on what level of blood glucose is actually hypoglycaemia and when to start treating. Hypoglycemia occurs in 1.3–4.4 per 1000 full-term newborns and 15–55 per 1000 preterm newborns. According to current evidence, the prevalence of hypoglycemia is approximately 10% in full-term neonates; 6.5% in appropriate for gestational age (AGA), 8% in large for gestational age (LGA), and 15% in small for gestational age (SGA) newborns; and 15.5% in late-preterm infants.

Hypoglycemia is less frequent in an older child. Mild congenital or often acquired defects can produce hypoglycemia in older child. The physiologic threshold for diagnostic evaluation and therapeutic intervention is a plasma glucose of 50 mg/ dL. Target above 70 mg/ dL should be maintained during ongoing management of hypoglycaemia.

Early diagnosis, urgent treatment, and prevention of future episodes of hypoglycemia are the cornerstones of management.



INTRODUCTION

Current evidence is still unable to define a specific glucose concentration that is safe to prevent acute neurological damage or chronic, irreversible neurological injury in the neonate. Weight and gestational age, as well as the age at onset, severity, duration, and number of episodes of hypoglycemia, are all determinants of the blood glucose level.Neonatal hypoglycemia can be transient or persistent. Hyperinsulinemichypoglycemia is the most common cause of recurrent and severe form of hypoglycaemia in the newborn.

A plasma glucose level below 30 mg/dL (1.65 mmol/L) in the first 2 h of life or below 45 mg/dL (2.5 mmol/L) after these first 2 hour has been considered diagnostic of hypoglycemia.

The Endocrine Society has emphasized on Whipple's triad for confirming hypoglycemia, which includes:(1)- symptoms or signs of hypoglycemia, (2)- low plasma blood glucose, (3)- resolution of clinical symptoms and signs when blood glucose is raised. This applies to children and adults. In practice, blood glucose levels below 50 mg/dL as measured by a glucometer should warrant careful monitoring, and should prompt initiation of diagnostic measures and immediate treatment.

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### MANIFESTATIONS

In most cases, neonates—even those at risk—are asymptomatic. Nevertheless, a neonate who is refusing feeds and has a feeble cry, or has irritability, tremor, and hyperexcitability or may have hypotonia, lethargy, and weak suckling should heighten suspicion of hypoglycemia. In high-risk neonates, major findings include fine tremors, acrocyanosis, seizures, and apnea; if left untreated, coma and death may follow.

In older children, epinephrine mediated symptoms and signs such as tremors, shakiness, sweating, pallor, anxiety, hunger, tingling and tachycardia often precede other signs. Symptoms of CNS depression or neuroglycopenia include mental confusion, headache, irritability, lethargy, psychotic behaviour, personality changes, and loss of intellectual ability, speech difficulties, visual disturbances or acute transient cortical blindness and loss of consciousness, coma.

### ETIOLOGY OF HYPOGLYCEMIA

# A) Transient Neonatal Hypoglycemia 1.Inadequate substate, enzyme function, homeostatic adaptation Transitional hypoglycemia (first 12 hours) Prematurity, SGA Severe respiratory distress Infant of toxemic mother 2. Associated with hyperinsulinemia Infant of diabetic mothers Infant with erythroblastosis fetalis Transient perinatal stress hyperinsulinism

3. Maternal medications

- Oral hypoglycemia agent
- Beta adrenergicstimulating and blocking agent

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Large maternal dextrose infusions



### B) Persistent (Recurrent) Hypoglycemia- Neonatal, infantile, childhood

- 1. Hyperinsulinemic states
  - Genetic forms of hyperinsulinism (focal/ diffuse- SUR1 and Kir 6.2 defect, glucokinase deficiency)
  - Beckwith- Wiedemann syndrome
  - Islet cell tumour
  - Factitious hyperinsulinism

### 2. Hormone deficiency

- Panhypopituitarism
- Isolated growth hormone deficiency
- CRH/ACTH deficiency or resistance
- Primary adrenal insufficiency
- Glucagon deficiency
- 3. Glycogen storage disease type I,III,VI, IX
- 4. Substrate limited-
  - Ketotic hypoglycemia
  - Maple syrup urine disorder
- 5. Disorders of gluconeogenesis
- 6. Enzyme defects galactosemia, fructose intolerance
- 7. Disorders of fatty acid oxidation
- 8. latrogenic
  - Drug induced salicylates, sulfonylureas
  - Abrupt discontinuation of IV high glucose drip

### **DIAGNOSTIC APPROACH**

### **HISTORY AND PHYSICAL EXAMINATION:**

A detailed history for factors that predispose to hypoglycemia including onset of symptoms, relation to timing of feed (special foods or period of starvation- short duration < 4 hours is s/o hyperinsulinism or glycogen storage disease and long duration of fasting of10-12 hours- is s/o fatty acid oxidation defect or defect in pathway of gluconeogenesis, hormone deficiency), family history ( consanguinity, infantile deaths, and diabetes mellitus), pregnancy issues (gestational diabetes), abnormal birth weight, and problems at delivery is important. Any history of potential drug exposure in children (Insulin,sulfonylurea).

A thorough physical examination is essential. Any presence of dysmorphic features, organomegaly, midline defects, micropenis, short stature, hyper pigmented skin, ambiguous genitals, any gallop or murmur .

**INVESTIGATIONS :** Glucose level monitoring is necessary in unwell and at risk babies. It is common to encounter transient hypoglycemia in the first few hours of life, but a persistent hypoglycemia that is unresponsive to feeding should trigger further investigations. Hypoglycemia should be excluded as the cause of an initial episode of convulsions, coma or neurobehavioural alteration in children.

Glucometry is the method of choice for initial screening of glucose level. However, levels should be confirmed through laboratory measurement

A critical sample taken during hypoglycemic episode is very important to diagnose the condition, which is included in the following:-

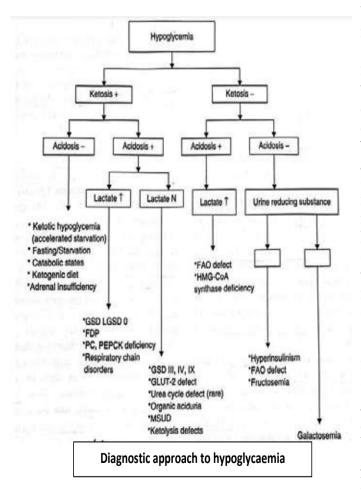
February 2024



SAMPLE	TEST
BLOOD	Glucose
	Ketones ( 3- beta hydroxybutyrate- BOHB)
	ABGA
	Lactate and ammonia
	Free fatty acids
	Insulin
	Cortisol and growth hormone
URINE	Reducing substance
	Ketones

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(To note – any detectable insulin during hypoglyemic episode is suggestive of hyperinsulinism. Serum cortisol and growth homrone levels may have delayed response to hypoglycemic episode, so may need further work up to confirm their deficiency if found low during the critical sampling)



**MANAGEMENT :-** As mentioned previously, there are still no clearly set values to define hypoglycemia.

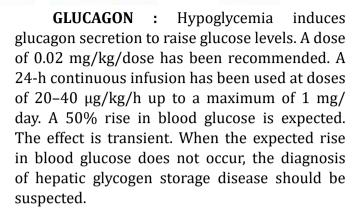
Although one may consider a diagnosis of hypoglycemia in a neonate when plasma glucose levels are below 45 mg/dL, this is not an absolute cutoff.SGA and late-preterm infants should be fed every 2–3 h and screened before each feeding in the first 24 h. After 24 h, screening needs only be continued in those whose glucose levels remain below 50 mg/dl.

The physiologic threshold for diagnostic evaluation and therapeutic intervention is a plasma glucose of 50 mg/ dL in children. Target above 70 mg/ dL should be maintained during ongoing management of hypoglycaemia.

**GLUCOSE** : Symptomatic neonates should be treated with glucose intravenously. A 2ml/ kg bolus of 10% dextrose glucose should be administered over 1 min. This should be followed by IV infusion at 6-8 mg/kg/min ( Glucose infusion rate). Glucose levels should be monitored after 30-60 min, with a therapeutic target of > 70 mg/dL. Control measurements should be obtained every 1-2 h. Once levels are stable, they can be reassessed every 4-6 h. If values do not reach a normal range, the rate of glucose infusion is increased by 1-2 mg/kg/min every 3-4 h. Suspect hyperinsulinism if dextrose requirement exceeds 8 mg/ kg/ min. Oral feedings should only resume once blood glucose levels have been stable for 6 h. High glucose concentrations (20–25%) may be necessary to maintain a rate of infusion of 15-30 mg/kg/ min; concentrations above 12.5% will require a central venous catheter.

**GLUCOCORTICOIDS** promote increased resistance to insulin action, reduce the secretion of insulin, and activate enzymes involved in gluconeogenesis. Thus, although such effects should theoretically induce an increase in blood glucose, there is no evidence to support glucocorticoid therapy in the treatment of hypoglycemia other than that caused by primary or secondary adrenal insufficiency.

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**DIAZOXIDE:** This agent is indicated in cases of hypoglycemia associated with hyperinsulinism. Diazoxide is a benzothiazine derivative that acts by opening ATP-sensitive potassium channels, causing inhibition of insulin secretion by pancreatic beta cells. It promotes an increase in hepatic glucose production and decreases peripheral glucose utilization. The recommended dose ranges from 10 to 15 mg/kg/ day, divided in two or three oral doses, up to a maximum dose of 30 mg/kg/day.

Sodium and water retention, plasma volume expansion, edema, thrombocytopenia, anorexia, vomiting, ketoacidosis, and hyperuricemia are possible complications of the use of this drug. Diazoxide treatment is hence combined with thiazide diuretic to counteract the side effects.

When the drug is effective, blood glucose levels will return to normal range within 2–4 days. Any trial of diazoxide therapy should last at least 1 week before the possibility of treatment failure is considered. Failure of diazoxide therapy suggests an abnormality in ATP-sensitive potassium channels. In these cases, a course of octreotide therapy should be considered.

Octreotide was the first somatostatin analogue approved for clinical use, due to its more prolonged effect. This substance inhibits the secretion of glucagon, insulin, growth hormone, and thyrotropin, as well as the exocrine secretions of the bowel. Due to its ability to inhibit hormones, this drug can be used in infants with congenital hyperinsulinemichypoglycemia. A dose of 5–35 mcg/kg/day via subcutaneous injection has been recommended. It may cause tachyphylaxis, necrotizing entero colitis, cholelithiasis and growth suppression. A long acting octreotide preparation , Lanreotide can be administered as a monthly injection ( 30-60 mg).

Sirolimus (rapamycin) The management of diffuse hyperinsulinemichypoglycemia, which does not respond to diazoxide, is a major therapeutic challenge. The successful use of sirolimus, both alone and as adjunctive therapy with octreotide, appears to be a potential alternative to subtotal pancreatectomy.

Exendin, a GLP-1 receptor antagonist that raises blood glucose levels in adults, has been introduced as a possible novel therapy for management of hypoglycemia in neonates with hyperinsulinism.

Growth hormone is used in cases of hypoglycemia associated with deficiency of this hormone or with hypopituitarism.

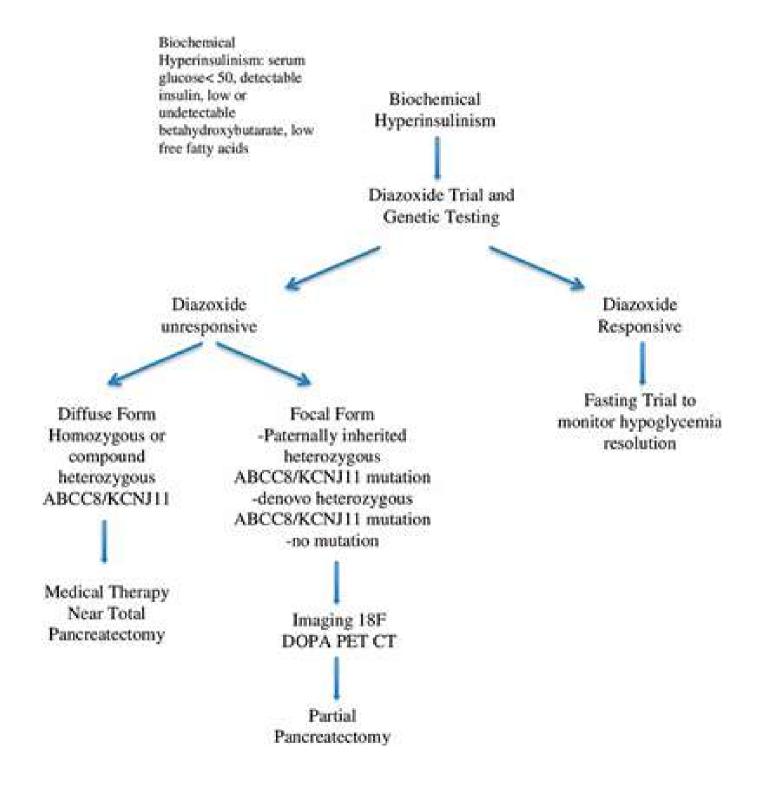
Calcium channel blockers, like Nifedipine, have been reported to be effective in treatment of hyperinsulinism, in combination with octreotide. It is started at doses of 0.3 mg/kg/ day,uptomaximum of 2.5mg/ kg/ day in 3 divided doses Therapeutic window is variable and need individual customization.

In cases of hypoglycemia due to persistent hyperinsulinemichypoglycemia that does not respond to treatment with diazoxide, octreotide and sirolimus, partial pancreatectomy may be indicated.

Also, avoiding prolonged periods of starvation, frequent feeding,bedtime snacks, intake of liquids during intercurrent illness and checking blood glucose during illness,use of raw corn starch may help when dealing with ketotichypoglycemia.



### MANAGEMENT OF A CHILD WITH HYPERINSULINISM



**CONSEQUENCES :** Recurrent or sustained hypoglycemia can cause neurological damage, mental retardation, epilepsy, and personality disorders. Transient episodes of hypoglycemia are also associated with deficits in math learning around age 10 years. Severe hypoglycemia can lead to impairment of cardiovascular function and is associated with high rates of neonatal mortality in very low-birth-weight infants. Permanent brain damage is found in 25-50% of patients with recurrent severe symptomatic hypoglycemia under age 6 months. The pathological changes described include gyral atrophy, reduced white-matter myelination, and cerebral cortical atrophy.

Child India

CONCLUSION : Generally, neonatal hypoglycemia is typically seen in at risk neonates and tends to be transient. Any persistent, severe or recurrenthypoglycemia, appropriate investigations including a detailed hypoglycemia screen (critical sample) is essential to understand the etiology in children. Hyperinsulinemichypoglycemia is a complex heterogenous condition associated with prolonged and recurrent hypoglycemia. Although a significant progress has been made in genetic analysis, novel imaging techniques, newer medications, is it still a challenging condition. Early detection and appropriate management iscrucial to avoid permanent brain injury in children.

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**February** 

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# **IAP Maharashtra**

### **MAHARASHTRA IAP REPORT :**

(Activities conducted From 26 th Jan 2024 to 29th Feb 2024 )

**<u>1.</u>** Wednesday Wisdom : 31/1/2024

Theme : Congenital Infections

Chief Guest : Dr M. Sinagaravelu (National Vice President IAP, South Zone , Tamilnadu ) Guest of Honour : Dr Sanjay Aher President, Maharashtra NNF (Nashik)

### **Experts** :

### 1. Dr Ashish Mehta

Senior Neonatologist (Ahemdabad)

### 2. Dr Omprakash Jamadar

Senior Neonatologist (Navi Mumbai)

Topics & Speakers were :

- 1. Congenital Toxoplasmosis : Dr Tanushree Mukharjee (Mumbai IAP)
- 2. Congenital Syphilis : Dr Deepa Joshi (Nashik IAP)
- 3. Congenital Varicella Zoster : Dr Ajay Jadhav (Beed IAP)
- 4. **Congenital Rubella** : Dr Bhushan Thorat (Amravati IAP)
- 5. Congenital CMV : Dr Shila Kalane

The session was moderated by :

### 1. Dr Manjusha Sherkar,

chairperson WC and

2. Dr Grivita Raikar ,co chairperson WC

The session was a power packed session with over 300 attendees in digital channel and zoom platform

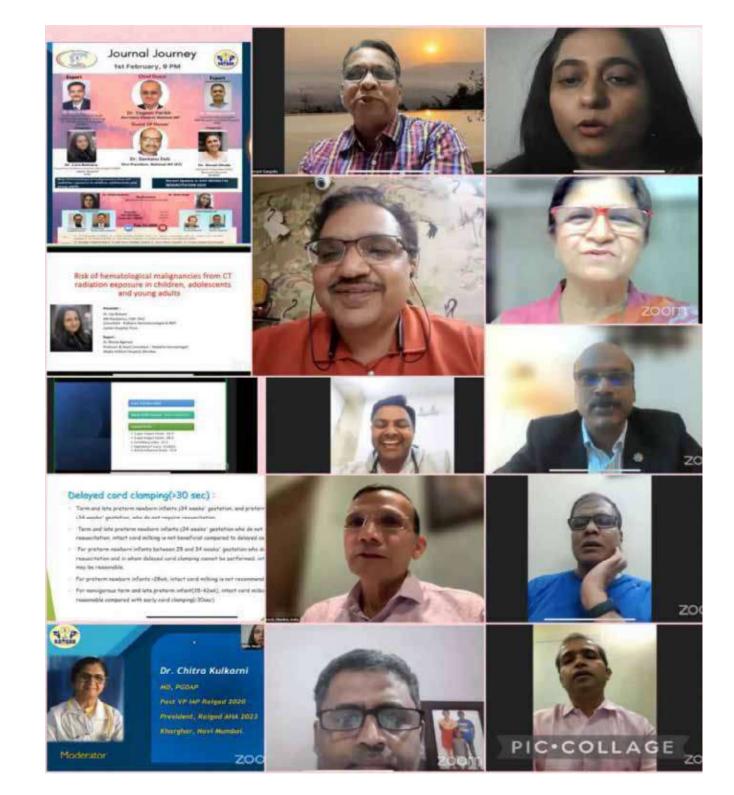


YouTube Link : https://www.youtube.com/live/YRPSNyPbWTw?si=0tOQjH-rdOxE9Iep



### WEDNESDAY WISDOM

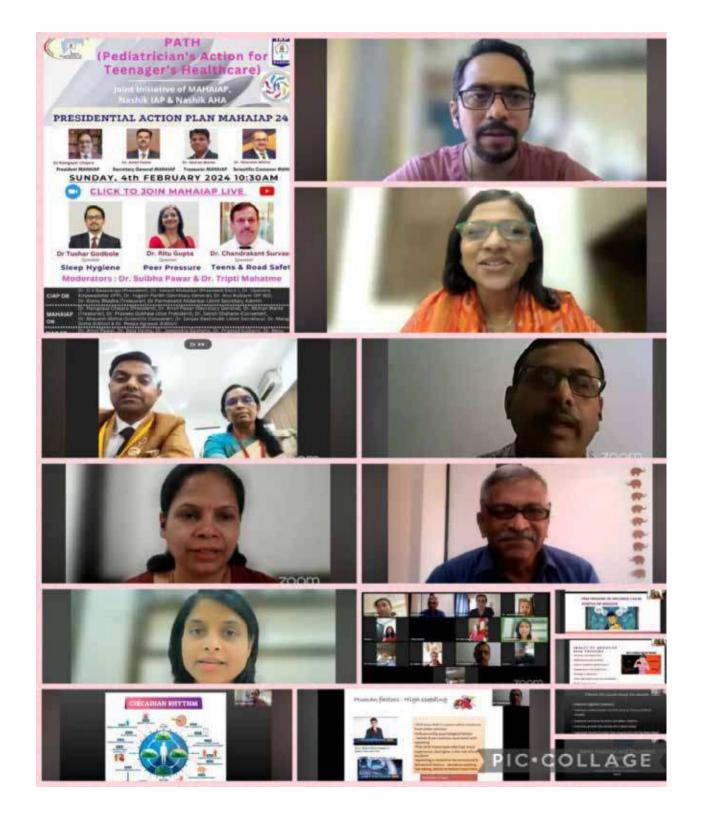




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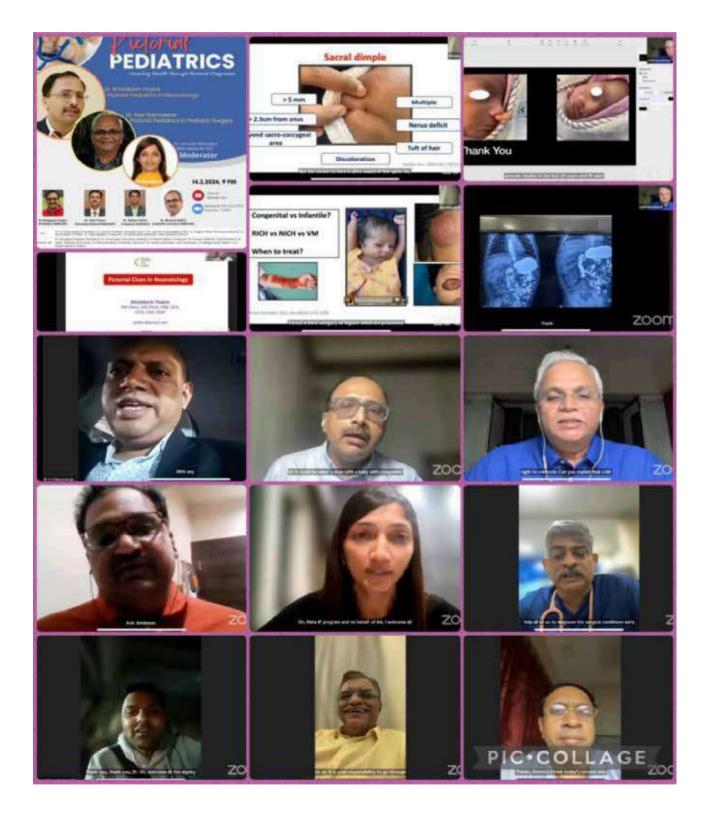
## **JOURNAL JOURNEY**





### P.A.T.H ( Pediatrics Action for Teenagers Health care )





## **PICTORIAL PEDIATRICS**





### Webinar on Medicolegal Protection Day For Doctors





### MahaVacciCon 2024





### MahaVacciCon 2024





### WEDNESDAY WISDOM



### 8. LEARNING WITH LEGENDS : 22/02/2024

### Topic -:

### **Pediatric Epilepsy for Pediatricians :**

"Febrile seizures, All that shakes is not seizures, and Eloquent resection epilepsy surgery"

### LEGEND : Dr Nandan Yardi

Senior Pediatric Neurologist, PUNE

Guest Of Honor: **Dr Santosh Kadam** President Elect, Maharashtra State IMA, THANE

Moderator: **Dr Jayant Shah** EBM MAHAIAP, Nandurbar





### 9. MAHAIAP KATTA : WANDERING IN THE HIMALAYAS : 24/02/2024

#### Narrator : Dr Mandar Bapaye

### Moderator : Dr Praveen Gokhale

In our busy hectic but monotonous schedules of a medical professional lies a hidden passion.

MAHAIAP katta is making an effort to bring to fore such passions and the pediatricians inculcating them.

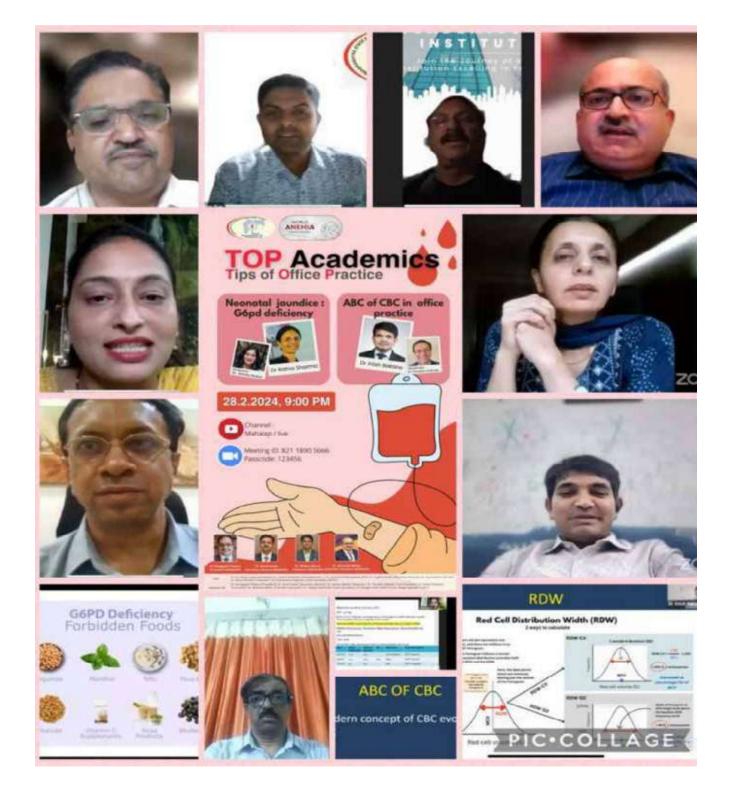
Trekking is one such passion followed by Dr Mandar Bapaye from Mumbai,who will be highlighted his journey through a very picturesque presentation.

Dr Mandar Bapaye brought forth in audio visual format the Himalayas to our screen and our imagination.

He was interviewed by Dr Praveen Gokhale another passionate traveller trekker mountaineer from thane.







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## **TOP Academics**



# **IAP Kolhapur**

# Installation of 2024 body



President- Dr Sainath powar, Secretary- Dr Rahul Patil, Traesurer- Dr Prama Bafna, Executive Board Members – dr Ruchika Yadav, dr rupail patil, dr Kashmiri badbade, dr Milind sadavarte, dr shradha kulkarni, dr Maheshwari jadhav



# **IAP Kolhapur**

# Journal club 13 jan 2024



Journal club Speakers – dr amit Chavan, topic- Acute liver failure in children Dr Aniket kumbhojkar- IAP revised guidelines on evaluation, prevention and management of childhood obesity.

# PG Clinic on 16th jan 2024



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# **IAP Kolhapur**

## Casper CME on 21 jan 2024



# Monthly Clinical meet on 31 jan 2024



Clinical meeting Lecture-Dr Salnath Powar , Topic- Intestinal Dysblosis Cases discussion by Dr Narendra Nanivadekar, Dr Rupali Patil



## **IAP Amravati**

# Indian Academy of Pediatrics (IAP) Amravati Branch (2024) President: Dr. Nitin Bardiya

Secretory: Dr. Hrishikesh Ghatol Treasurer: Dr. Nilesh Pachakawade World Anemia Awareness Day (13 Feb 2024)



World Anemia Awareness Day celebration at Government Superspeciality hospital, Amaravati (80 Beneficiaries)



World Anemia Awareness Day celebration at Prayas Child Development Center, Amaravati (20 Beneficiaries)



World Anemia Awareness Day celebration at District Women's hospital, Amaravati (110 Beneficiaries)



# **IAP Hingoli**

# IAP HINGOLI MONTHLY ACTIVITY FEBRUARY 2024

National Deworming Day: We IAP Hingoli celebrated National Deworming day on 10<sup>th</sup> February 24 at Choudhary Hospital Hingoli by distributing Tablet & Syrup Albendazole to OPD children above 2 yr & told them importance of hand washing & taking care about hygiene.



## National Deworming day



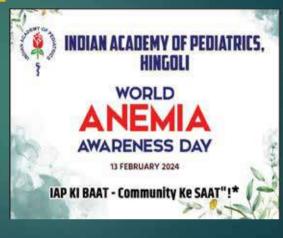




# **IAP Hingoli**

# WORLD ANEMIA AWARENESS DAY

We IAP Hingoli celebrated World Anemia Awareness Day at Choudhary Hospital Hingoli under the theme of 'The Best Blood is Your Blood'. We have organised short talk on various causes, prevention & treatment of Anemia in children under the Presidential Action plan 2024 with the slogan 'IAP KI BAAT-COMMUNITY KE SATH''. Also we hava distributed Jaggery Chicky' to our patients to create awareness about Anemia.



# World Anemia Awareness Day





# **IAP Hingoli**

# WORLD ANEMIA AWARENESS DAY





DR TU SUKUMARAN ORATION BY DR GOWRISANKER AT STATE RESPICON CALICUT 54





IAP Thiruvananthapuram - Adolescent Health education for Poovathoor Higher Secondary School on 26/02/2024 by Dr Bennet Xylem P.



























ELS/BLS Training - IAP Kasargode



**IAP Kerala** 



Anemia Detection camp - IAP Vadakara





IAP Thalassery - Anemia awareness campaign





Monthly GB and CME of IAP Malanad



**IAP Kerala** 



**BLS/ALS Training IAP Kannur** 





IAP Pathanamthitta Installation



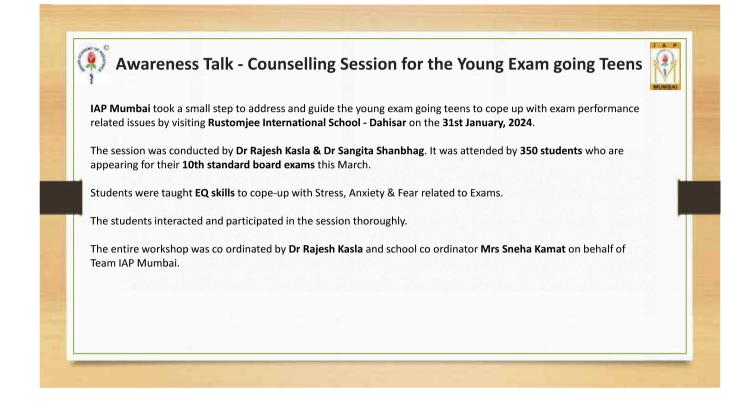
IAP ALAPPUZHA BLS TRAINING





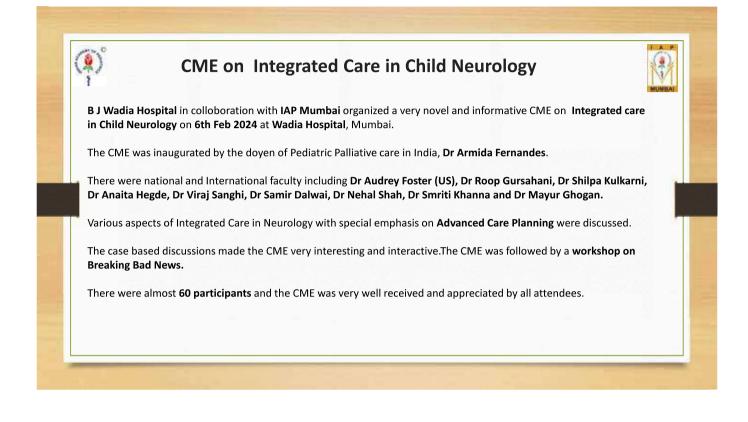
Iap Kozhikode - monthly clinical club meeting









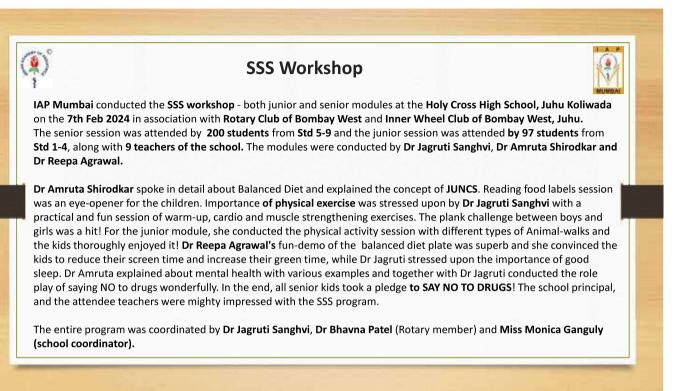




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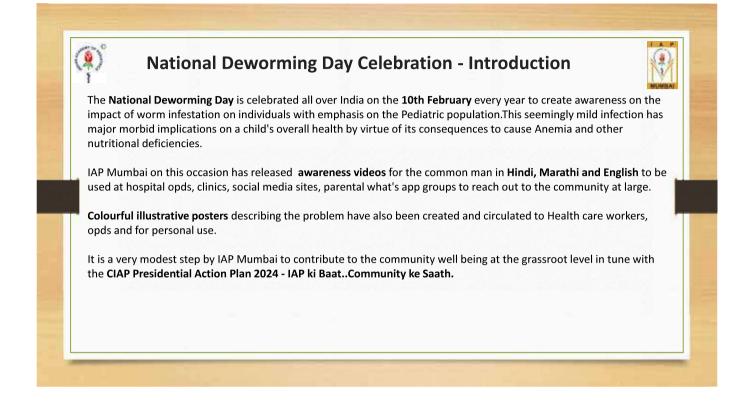
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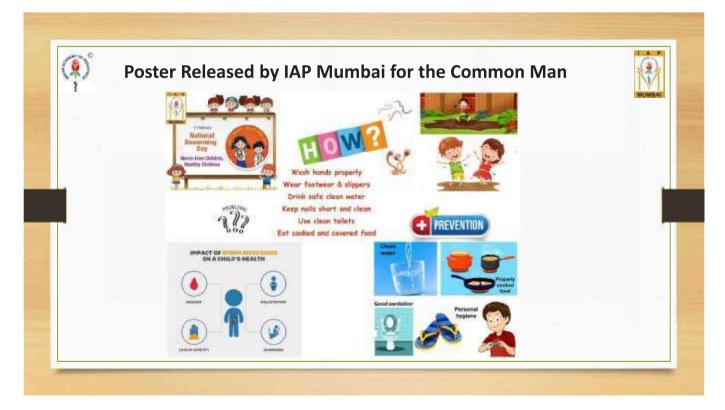
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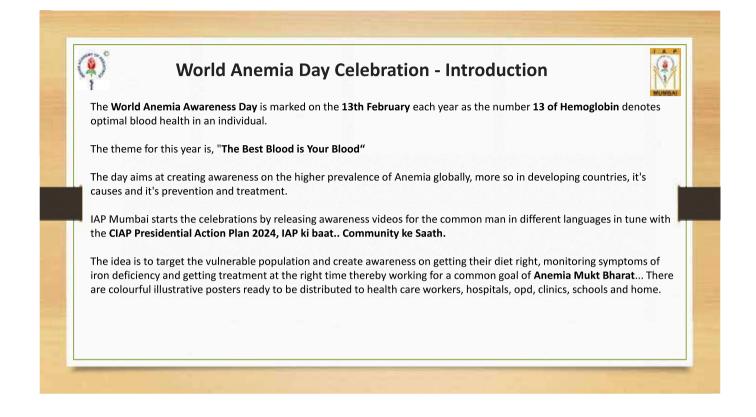






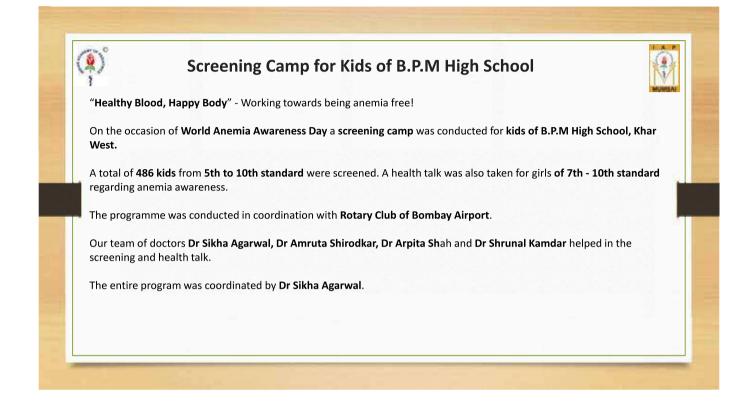






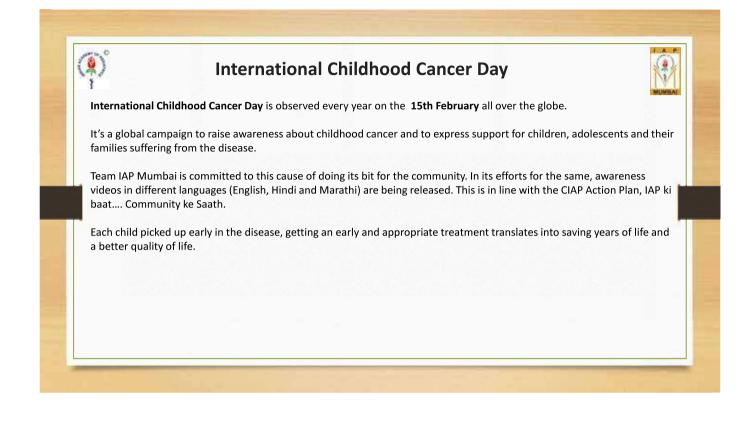








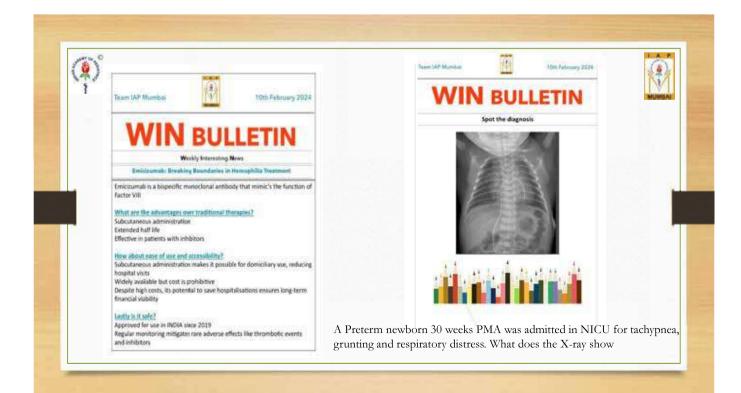














#### NAVI MUMBAI IAP BRANCH REPORT FEBRUARY 2024

#### <u>ACADEMIC –</u>

- 9<sup>th</sup> Feb 2024 Navi Mumbai IAP PG teaching clinic
   Experts Dr S Balasubramanian & Dr S Srinivasan
   Convenors Dr V N Yewale, Dr Jeetendra G, Dr Satish S, Dr Snehal M.
   <a href="https://youtube.com/c/IAPNaviMumbai">https://youtube.com/c/IAPNaviMumbai</a>
- 17<sup>th</sup> Feb 2024 E Gurukul Pathshala PG CLINIC
   Case discussion on Fever with rash
   Expert Dr. Vijay Viswanathan Consultant Pediatric Rheumatologist, Thane.
   <a href="https://youtube.com/@egurukulpathshala7559">https://youtube.com/@egurukulpathshala7559</a>

#### 3. 18th Feb 2024 – MAHA VACCICON – 1st STATE LEVEL CONFERENCE ON VACCINOLOGY

Navi Mumbai Association Of Pediatrics in collaboration with MahalAP hosted

"The MahaVacciCon" which was the first state level vaccine conference held ever.

The conference acted as a strong booster dose to our primary vaccine knowledge increasing our efficacy of implementing this knowledge in our day to day practice with our knowledge memory cells lasting for a long time.

The session started with **Dr. Srinivas Kasi** building a strong foundation of Vaccinology basics making the complex immune mechanisms so simple to understand.

This was followed by our master blaster, **Dr. Nitin Shah's** informative talk on dropping hesitancy to HPV Vaccines.

This was followed by another astute academician, **Dr. Suhas Prabhu** giving us valuable insights on choosing the right PCV.

The Panel discussion moderated by **Dr. Satish Shahane and Dr. Amit Saxena** with expert **panelists Dr Kasi, Dr Sanjay Marathe, Dr Bakul Parekh** cleared the Purple Book Blues and created a fresh colourful hue of correct information

Our own Dr. Vijay Yewale spoke on TCV - Mission make in India...a subject close to his heart.

The **KeyNote Address** was delivered by **Dr. Srinivas Kasi** navigated us through newer and future vaccines which was one of the best talks of the conference. This talk opened our minds to a whole new universe of the vaccine therapeutics that awaits us in the future.



Dr. Sanjay Marathe then spoke on influenza vaccine in pediatric practice.

The Jabbs in special situations panel 'Jabb we met' was moderated by our Jabardast duowitty Dr. Shilpa Aroskar and wise Dr Shubhash Rao with experts Dr Pravin Mehta & Dr Sanjay Marathe, this panel tickled both our grey cells and our humour cells.

The Chai Pe Charcha had doyens **Dr. Nitin Shah and Dr. Vijay Yewale** and the discussion was definitely more stimulating than our chai.

The Ru Ba Ru session was the cherry on the cake: a session moderated by **Dr Vikram Patra** addressing our audience queries and getting doubts cleared from the experts in the field **Dr Srinivas Kasi, Dr Vijay Yewale, Dr Nitin Shah and Dr Sanjay Marathe.**The session was conducted graciously by our **MOC** for the event **Dr Sushmita Gupta and Dr Gargi Bangar.** 

The delegates received an exciting goody bag as **Delegate kit** in the form of **Emergency Kit** containing inj adrenaline for rapid response to anaphylaxis, a Vaccine Temperature Monitor and a Fridge Magnet. At the end of the conference, copies of ' **IAP Purple Book** ' were distributed to those who haven't received yet.

4. 22<sup>nd</sup> Feb 2024- On occasion of World Anemia Day, NMAP in association with Dr.D.Y. Patil Hospital & Medical College have organised a CME with esteemed faculties renowned in the world of Hematology, Dr Ratna Sharma and Dr Govind Kendre, where they will be discussing a difficult case of anemia followed by a panel discussion on common queries in day-to-day practice. Our Dynamic president Dr Satish Shahane and Secretary Dr Mangayarkarshi Sinha attended the program. HOD Dr Kotrashetty has taken active part in discussion and made session more interesting

Venue: Pushpanjali Auditorium, Dr. D. Y. Patil Hospital

5. 23rd Feb 2024 – Navi Mumbai IAP PG teaching clinic

Experts - Dr S Balasubramanian & Dr S Srinivasan

Convenors - Dr V N Yewale, Dr Jeetendra G, Dr Satish S, Dr Snehal M

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6. 25th Feb 2024 – Webinar Series educating our future today

Topic – Creativity an essential catalyst for education

Expert – Dr Leena Deshpande

7. 25th Feb 2024 – IAP Presidential Action Plan West Zone TOT

Topic – All About Fever



#### Venue – Vashi, Navi Mumbai









#### AWARDS, PUBLICATIONS & ACHIEVEMENTS -

- 1. Dr Satish Shahane installed as Scientific Convenor Maharashtra IAP at Nagpur
- 2. Dr Mangai S installed as Editor Maharashtra IAP at Nagpur
- 3. Dr Prashant Patil et all published original article describing a " simplified method to calculate Bone Age utilizing three bones of the hand and wrist" in a reputed International Journal ' Endocrine'.
- 4. Dr.Kiran Vaswani was awarded International Board Certified Diplomate and Lifestyle medicine (LSM) Physician





#### <u>SOCIAL-</u>

- 1. On the occasion of World Anaemia Day 2024 and Deworming Day with context of Mission Anaemia Mukt Bharat Navi Mumbai IAP in collaboration with Maha IAP has released a parental awareness poster to be displayed in our OPD (Outpatient Department) area to sensitize parents about the need for deworming in their children. This poster was made available in two languages (English and Hindi) for better understanding.
- 2. Cervical cancer is one of the leading causes of morbidity and mortality in women. The awareness regarding this dreaded disease is increasing day by day and due to the availability of affordable vaccination and pro activeness of Paediatricians and gynaecologists alike, acceptance has grown regarding the use of Cervical cancer vaccination. An awareness session and administration of HPV vaccination was conducted to 50 girl students at Murbi Zilla Parishad school, Kharghar in collaboration with Rotary club Kharghar mid-town by Navi Mumbai AHA Treasurer and EB member Dr Amog Shahane. The program was successful and the beneficiaries were filled with gratitude with this noble work. It was supported extensively by IAP Raigad and Dr Chitra Kulkarni and it was a joint venture appreciated by all.
- 3. iCAN child development centre started the parental training series on ADL (Activities Of Living) today. The first session was an interactive, hands on practical session on toilet training.
- 4. Dr Prashant Patil et all published original article describing a "simplified method to calculate Bone Age utilizing three bones of the hand and wrist" in a reputed International Journal 'Endocrine '<u>https://link.springer.com/article/10.1007/s12020-024-03684-9</u>
- 5. Childhood anaemia is a subtle and often missed cause of inattentiveness, irritability and poor scholastic performance in children and especially adolescents. Many a times, kids present to the clinic with recurrent infections, mood swings and as a 'difficult temperament child', but the underlying cause of anaemia is detected on detailed history and examination. Curbing this issue in the budding stage goes a long way in improving the life of these kids by early screening, detection and treatment. Navi Mumbai IAP on World Anaemia Day 2024 conducted an anaemia screening camp for 450 kids of Greenfingers Global school, Kharghar in association with Kharghar Doctor's association from Senior kg to 9th STD and the importance of iron rich food and balanced nutrition was emphasized in a session by NMAP President Dr Satish Shahane and EB member Dr Amog Shahane
- 6. IAP Ki Baat Community Ke Saath <u>https://www.youtube.com/@indianacademyofpediatrics/</u><u>featured</u>

https://www.facebook.com/events/921517812682960/







**IAP Jalandhar** 















World Anemia Awareness Day Celebrations on 23rd February 2024