Child India

December 2022

Monthly e-Newsletter of Indian Academy of Pediatrics

IAP EXECUTIVE COMMITTEE - 2022

PRESIDENT	DR REMESH KUMAR R
PRESIDENT-ELECT	DR UPENDRA S KINJAWADEKAR
IMM. PAST PRESIDENT	DR PIYUSH GUPTA
VICE-PRESIDENT (CENTRAL ZONE)	DR K RADHAKRISHNA
VICE-PRESIDENT (EAST ZONE)	DR SUDHIR MISHRA
VICE-PRESIDENT (NORTH ZONE)	DR HARINDER SINGH
VICE-PRESIDENT (SOUTH ZONE)	DR SUBRAMANYA NK
VICE-PRESIDENT (WEST ZONE)	DR CHETAN B SHAH
SECRETARY GENERAL	DR VINEET K SAXENA
TREASURER	DR SAMIR HASAN DALWAI
EDITOR-IN-CHIEF, IP	DR DEVENDRA MISHRA
EDITOR-IN-CHIEF, IJPP	DR S THANGAVELU
JT. SECRETARY - LIAISON	DR ALOK BHANDARI
JT. SECRETARY - ADMIN	DR PURNA A KURKURE

Executive Members

DR ANTHATI S KIREETI
DR P ANIL KUMAR
DR R RAMAKRISHNA PARAMAHAMSA
DR MRITUNJAY PAO
DR CHANDRA MOHAN KUMAR
DR SHEO BACHAN SINGH
DR ARUN PRASAD
DR SUSHIL KUMAR
DR AJAY KUMAR GUPTA
DR LALAN K BHARTI
DR MANISH GUPTA
DR VIRENDRA SADANAND GAOKAR
DR KANAKSINH U SURMA
DR RAMESH M BAJANIA
DR SAMIR R SHAH
DR AVS RAVI
DR MAHAVEER PRASAD JAIN
DR RAVINDER K GUPTA
DR JOY BHADURI
DR GEETA PATIL
DR MADHU S PUJAR
DR SN PRASHANTH
DR SHIVA PRAKASH SOSALE C
DR JOSE OUSEPH
DR M NARAYANAN
DR MK NANDAKUMAR
DR RIAZ I
DR KEWAL KISHORE ARORA
DR MAHESH MAHESHWARI
DR NARENDRA R NANIVADEKAR
DR PRAMOD M KULKARNI
DR RAMAKANT D PATIL
DR SANJAY B DESHMUKH
DR BELA VERMA
DR SHYAMKUMAR LAISHRAM
DR SANTANU DEB
DR PRASANT KUMAR SABOTH
DR SUSRUTA DAS
DR GURDEEP SINGH
DR HARPREET SINGH
DR ANURAG TOMAR
DR RAMESHWAR L SUMAN
DR A CHENTHIL
DR A SOMASUNDARAM
DR J BALAJI
DR R SOMASEKAR
DR G VIJAYA KUMAR
DR KOTTOORU SRISAILAM
DR S SRIKRISHNA
DR DHIRAJ M GUPTA
DR OM SHANKAR CHAURASIYA
DR RAJEEV KRASHAK
DR UTKARSH SHARMA
DR ASOK KUMAR DATTA
DR KAUSTAV NAYEK
DR SUTAPA GANGULY
DR SHEILA S MATHAI (SERVICES)

CONTENT

Editor's Note

Dear friends,

With this December issue of Child India, we convey Season's Greetings and wishes to all for a Happy and Prosperous New Year.

After an eventful and academically rich 2022 we are to enter into another year that would take our knowledge in child health to even greater heights.

December celebrates World Aids Day on 1st (theme 'Equalize' to address the inequalities which are holding back progress in ending AIDS; and equalize access to essential HIV services particularly for children and key populations and their partners) and International Day of Disabled Person on the 3rd (Theme "Transformative solutions for inclusive development: the role of innovation in fueling an accessible and equitable world.").

Most States have had their respective IAP State Annual Conferences and the resultant scientific sessions and a slew of activities on the IAP Action plan front has turned this year end into a teaching and learning experience for beyond one's fondest imagination.

We are eagerly awaiting the results of IAP Awards for 2022 and here is wishing all performers are appreciated.

We profusely thankful to Dr PSN Menon for his time and expert guidance in having most of the pediatric endocrinologists in the country contribute to the November and December issues of Child India.

We place on record our sincere thanks to our ever-smiling, most approachable and active IAP President 2022 Dr Remesh Kumar, our most efficient HSG Dr Vineet Saxena, IAP OB and EB 2022 for their support and encouragement in publishing this monthly e-newsletter of IAP – Child India.

Have a great ending to year 2022 and pray for cheer and joy in 2023

Jai IAP!

Dr Jeeson C Unni
Editor-in-Chief

President's Address

Bidding adieu IAP Year 2022 - My lovely Dears,
I just walked behind you only;
and You made it happen awesome.

Dear Friends,

The year 2022 was quite exciting and invigorating, with the Covid blues quickly moving out and people racing out of the covid shell . And at IAP too, our Friends started rejoicing "Academics with Camaraderie " and the infectious vibe was too contagious that we had an unbelievable number of physical activities across the country this year. Our membership too have been growing at a quick pace of around 1500 new members per year , taking us past the magic figure of 40K to 40034 as on Dec 31st 2022. We had 9 new district branches coming up this year providing greater opportunities for our members to contribute to the cause of IAP.

PEDICON 2022, the National Conference of the Academy at Noida gave Central IAP the much needed confidence and belief that Covid has not shaken the spirit of the IAPians. Along with the flagship activities of the yester years like NRP, NC ECD & NTEP, the 13 new academic modules in various sub specialties floated in the year kept the branches on the toes.

CIAP could successfully enlist around 240 out of its 343 branches in this year's activities and much more heartening was the fact that as many as 40 of them were undertaking CIAP activities for the 1st time. Initiating academic and community activities under the banner of Kashmir branch of IAP has been a long awaited dream which got materialized in Nov 2022.We could successfully launch the UK & Bahrain branches of the Academy this year.

The Standard Treatment Guidelines in Pediatric Office Practice have been meticulously reaching our members every Monday, Wednesday & Friday from the 1st of Jan 2022 itself . It is quite heartening to note that more than 75 % of the authors for STG are young debutants and we wish to see them contribute to the academic corpus of IAP in a big way in the years to come.

We had the 3rd updated edition of IAP Immunisation Book (PURPLE BOOK) & the Standard Treatment Guidelines - STG Book released at our Navi Mumbai IAP Office at the celebrated hands of the living legend of Pediatrics Dr Y K Amdekar . The Immunisation Book will be delivered complimentary at the doorstep of every IAP member by Jan 31st 2023.

The year also saw our mother body taking up community and charity activities in a big scale. The U5MR 25 by 2025 project which focused on 57 high burden aspirational districts with a Under Five Mortality Rate more than 50 has taken off with much zeal and enthusiasm with our District Champions passionately behind it and UNICEF and District Health administrations proactively facilitating the initiative.

President's Address

The distribution of Superhero Kits with items which focused on strong messages on Undernutrition & Anemia was launched on Children's Day at Barabanki in Uthar Pradesh . These kits, each worth around Rs 1000/- have already been distributed to 2800 under privileged children and another 3000 are ready to be distributed (100 families each in the selected 50 districts) and this project is to be continued as a Charity Project from Central IAP , with deserving children as direct beneficiaries.

Central IAP have been able to provide a single point access to all IAP portals through the revamped IAP website and the introduction of the PEDCARD. Self updation of member data and profile is an important feature in the new web site. The PEDCARD mobile app once installed on your home screen can take you to any IAP site or activity with a single click . Apart from this, the Pedcard will be offering loyalty services on purchase of academic subscriptions like Uptodate/ Clinical Key/ books from Jaypee Brothers and products of many hospitality and retailer industries in the near future.

The regular flagship academic journals of Central IAP ,Indian Pediatrics & Indian Journal of Practical Pediatrics and the Drug Formulary were in full swing last year.

I would like to congratulate all the 40000 plus members in IAP Family for their whole hearted involvement to keep the IAP flag flying high all through . My sincere gratitude to all the 2022 OB & EB members for their unstinted support and for keeping the team inspired throughout the year .I am short of words to thank my HSG Dr Vineet Saxena who was a man on mission , who never compromised on work ethics and had the systems in full operational mode from day 1 itself.

Friends, IAP has lived up to the legacy built up over the years by our illustrious predecessors and has always been standing tall with its commitment of its members to the declared mission and goals of bettering child health in the country. Let's move together and make ourselves proud as noble custodians of child welfare.

I sincerely wish the new Team led by Dr Upendra Kinjawadekar, Dr Basavaraj & Dr Vineet Saxena a most fruitful year at the helm of Indian Academy of Pediatrics.

Wishing all my DEARS in IAP Family a very Happy and Prosperous New Year- Here is your Remesh Kumar signing off from the Presidency of Indian Academy of Pediatrics.

Loving Regards,

Jai IAP, Jai Hind

Dr Remesh Kumar
National President, IAP 2022

Secretary's Message

Dear Friends,

"Wish you a very happy, healthy and prosperous new year 2023."

December is the month of reviewing all past and ongoing activities as well as all activities to be implemented in the upcoming year. I am extremely happy with the success of all the previous activities and excited about the activities to be implemented in the upcoming year.

I heartily welcome my dear friend and IAP President-elect 2023, Dr GV Basavaraja, and all the newly elected OB and EB members on the board of 2023, and assure every IAPan that we shall continue to good work and commit to the health and well-being of all children across the country.

The month of December has significantly added to the success of year 2022. On December 18th, the release ceremony of two much-awaited and very important books 1) the PURPLE BOOK of IAP Immunisation Guidelines 2022 2) STG - Standard Treatment Guidelines in Pediatric Office Practice, was held at the central office of Indian Academy of Pediatrics. Hon'ble Dr YK Amdekar was the chief guest for the function. Also, Dr Rohit Agarwal, Dr Deepak Ugra, Dr Ramesh Kumar, and Dr Upendra Kinjwadekar were all present for this ceremony. Their presence doubled the enthusiasm of the ceremony.

Also, on 29 December 2022, we held a joint meeting of the Executive Board for the year 2022 and the New Year 2023. In this, farewell was given to the outgoing Executive Board and the new Executive Board was welcomed. Also, brainstorming and various activities for the health and welfare of the child were discussed in this meeting. This new initiative will benefit a lot. I thank to all Office bearers and Executive Board members for their active participation and fruitful discussion in the best interest of child health.

We are happy to share that in the month of December the following conferences have been successfully organised i.e.RESPICON 2022 at Chennai, PEDICRITICON 2022 at Bhubaneswar, HARYANA PEDICON at Karnal and GUJPEDICON 2022 at Ahmedabad. I Congratulate all committee members of these Mega Events and Conferences for their extraordinary efforts and teamwork to push IAP to greater heights in academics and child welfare.

Along with this, Indian Academy of Paediatrics conducted workshops on the following modules under the Presidential Action Plan 2022. 4 Workshop of Pediatric Emergency Care & Resuscitation Training Module (PECART), 6 of Pyrexia of Infection & Non-Infection (POINT), 1 of Ped Gastro, 3 of Growth & Puberty- A Challenging Journey-Pediatric Endocrinology Module, 2 of Perinatology - Caring both ends of the Cord.

Regarding the ECD, A total of 113 workshops of ECD have been done to date and 7 workshops of ECD in November 2022. This month total of 118 Basic NRP and 14 Advanced NRP provider courses have been successfully conducted.

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.

Long Live IAP, Jai IAP

In service of Academy,

Dr Vineet Saxena
Hon. Secretary General 2022 & 23

President's Engagements

A totally plastic free environment friendly conference was awaiting to happen - NCPID , the showpiece event of the Infectious Diseases Chapter of Indian Academy of Pediatrics in its 2022 edition at VIT Campus, Vellore took the bold challenge of going " Green" all the way . The all round skills of Dr Narmada Ashok coupled with her passion and enthusiasm for IAP made the conference unique on its own . Dr S Balasubramanian, the ID Chapter Chairperson and the stalwart ID specialist from Chennai and his Secretary Dr Bhaskar Shenoy left no stone unturned to provide mesmerising scientific sessions from D1. . The academic giants right from world renowned immunisation expert Dr Jacob John were there to enrich the scientific deliberations. The new Team of 2023 led by Dr Vasanth Khalatkar was installed at the inaugural function. Congratulations to the Organising Team of Dr Janani Sankar & Dr Arulalan, Organising Chairpersons, Dr Narmada Ashok, Org Secretary ,Dr Suresh Babu, Treasurer & Dr Sathish Kumar , for carving

President's Engagements

CALPEDICON , the 2022 Annual Conference of IAP at K Hills, Kozhikode from 18 th to 20 th Nov made its mark as a benchmark conference in the annals of IAP Kerala. Excellent scientific sessions spiced with 6 pre conference workshops made it a worthy time for our delegate friends . The mouth watering Malabar taste never went begging at the food counters . And personally for me , being at home turf gave a most cozy weekend with my dear teachers , seniors as well as buddies . Congratulations dear Dr Balraj, Dr Shaji Thomas John , Dr Ashraf, Dr.Nihaz, Dr Ajith, Dr. Suresh , Dr.Rahul, Dr.Mohandas , Dr.Ajay , Dr Sayyid , Dr.Ranjith , Dr. Krishnamohan, Dr. Vishnumohan & all at the Organising Team who made us proud with a big bang conference . Best wishes to the Team 2023 of Dr Jose. O, Dr Shimmy Paulose, Dr Krishnamohan, Dr Gopimohan & Dr Ranjith who were installed at CALPEDICON .

President's Engagements

Silchar on 26th & 27th Nov hosted the EZ Conference of IAP .Privileged to deliver Dr Tapan Ghosh Oration on a novel topic " Human Intelligence to Artificial Intelligence- Future of Pediatric Practice " . Was excited to have Dr Rajdeep Roy, MP of Silchar , more so a practising Orthopedician as our Guest of Honour . Congratulations to our vibrant friends at Barak Valley led by Dr Sumit Das , Dr Anjan Paul, Dr S Dam, Dr Reeta Bora, Dr Janet ,Dr SK Roy, Dr DJ Nath , Dr Priyanka Deb , Dr Hameed and other course , the back office and front office vibrant dynamo Dr Pinaki Chakraborty for organising a conference of this magnitude . The patronage of seasoned veterans of Assam like Dr AK Dutta, Dr Shahina Ahmed, Dr Rashna Das, Dr Mrityunjay Pao , Dr Sailendra Kumar Das, Dr Rupam Das , and Dr Devajit made the conference a flawless one . It was quite refreshing to have all the pVice Presidents of EZ past, current and future - Dr Arup Roy, Dr Jaydeep Chowdhary, Dr Atanu Bhadra, Dr Nigam Narain , Dr Sudhir Mishra and Dr Biswajit - in one platform. Hats off to Dr Pinaki and all at Team Silchar

President's Engagements

Hyderabad on 10th & 11 th Dec had the biggest event of the year for both PATS & TCB . The 2022 annual conference of both Telengana State and Twin Cities Branch was conducted with much splendour and plomb . Had the honour of delivering the prestigious Dr YC Mathur Oration of TCB . We had a special Issue on Breastfeeding released on the same day with UNICEF support . Congratulations to the Organising Team led by Chairmen Dr Surendranath & Dr E.Arjun & the Presidents Dr Arkala Bhaskar & Dr Sunkoj Bhaskar and Secretaries Dr Pavan Kumar & Dr Sreekrishna RSV for hosting a wonderful conference. The Telengana stalwarts especially Dr Sanjay Srirammpur, Dr Indrasekhar Rao, Dr Ajoy Kumar, Dr Ravi Kumar, Dr Himabindu, Dr Yashwanth Rao , Dr Sreekrishna, Dr Srisailam, Dr Vijay Kumar Dr Venkiteswara Rao, Dr. Lakshman Garlapati, Dr Laxman Kumar, Dr Usha Rani, Dr Nirmala, Dr Dr Mallesh, Dr Daruru Ranganath, Dr. Krishnamurthy , Dr Sridhar , Dr Srinivasa Rao and Dr Vamshi Kondle contributed their might to have PATSCON & TCBC

President's Engagements

Release of IAP Standard Treatment Guidelines 2022 at IAP Office, Mumbai. A compilation of standard treatment guidelines (STG) for 150 common office practice issues in pediatrics mainly focused to help common paediatricians in day today practice. The wonderful culmination of a project conceived 18 months back as one of the major presidential action plans of IAP year 2022 by our own President dear Remesh Kumar sir. Our team of 9 members constituted from all 5 zones of IAP under the leadership of our dear leader Sachi sir was a real learning experience. Communicating and interacting with 450 pioneer academician's of our Pediatric fraternity in country was a real privilege and great experience. A big thanks to our Chief editors Remesh sir, Vinod Ratageri sir and Piyush Gupta sir for guiding us throughout the journey. Our team members Dr Santanu Deb, Dr Surender Bisht, Dr Narmada Ashok, Dr Pawan Kalyan and Dr Prashant Kariya were like a family and a joy to work with. Thank you all dear seniors and friends at IAP Kerala for the wonderful support through out the journey. Once again a big thank to our visionary leader and inspiration of all young IAPian's of our country, dear president Dr Remesh Kumar R for the great vision and opportunity

President's Engagements

PURPLE BOOK on Immunization : Release of IAP Immunisation Book 2022 at CIAP Office, Navi Mumbai on 18 th Dec 2022 Sunday by the doyen of Pediatrics Dr Y K Amdekar. Congratulations to the ACVIP Team led by Chairman Dr Indrasekhar Rao & Advisor Dr S G Kasi. Proud to have the presence of seasoned stalwarts like Dr Sanjay Lalwani, Dr Rajendra Khadke , Dr Arun Wadhwa , Dr Shashikant Dhir, Dr Chandramohan Kumar, Dr. Kripasindhu Chatterjee, Dr Srinivas Kalyani, Dr Rajasekhar, Dr. Ananthakesavan & Dr Bhaskar Shenoy as members of ACVIP .The purple book with updated guidelines will be delivered to all the members at their doorstep by Jan 2023 end

President's Engagements

Gujarat State Conference at Ahmedabad on 17th Dec was an experience on its own . Being the curtain raiser for the upcoming International Congress at Gandhinagar, the GUJPEDICON had a lot to showcase to the IAP fraternity. The prowess and splendour of the academics and the venue was befitting the grit of the organising team led by Dr Chetan Trivedi , Dr Rakesh Sharma , Dr Rakesh Desai,Dr Manish Mehtha,Dr Nischal Bhat , Dr Thushara Shah , Dr Unmesh Dr Sunil Patel & Dr Monish Shah . The inaugural function was blessed by the presence of Dr Upendra Kinjawadekar, President Elect CIAP & Dr Chetan Shah, Vice President CIAP . Guidance from Senior Guj Stalwarts like Dr Shashi Vani, Dr Naveen Thacker , Dr Raju Shah, Dr Baldev Prajapati, Dr Digant Shastri , Dr Abhay Shah, Dr Satish Pandya , Dr Prothima Shah , Dr Yagnesh Popat, Dr Swathi Popat , Dr Bela Shah , Dr Samir Shah, Dr Ramesh Bajania, Dr Rakesh Sarma, Dr Takwani , Dr Prasanth Katia& Dr Kirit Sisodia went a long way in the successful conduct of the conference. Congratulations dear Dr Chetan Trivedi and Team

President's Engagements

Sri Ramachandra Medical College, Chennai- RESPICON 2022 , the 34th National Conference of IAP National Respiratory Chapter - Dec 17th & 18th- hosted by IAP TN State Respiratory Chapter, IAP TN State Chapter & IAP Chennai City Branch. Was privileged to be the Chief Guest for the inaugural function. Dr D Vijayasekharan, Dr NC Gowri Shankar , Dr Shivabalan & Team could provide the best of science from faculty of international repute . The Respiratory Chapter OB led by the vibrant Dr NK Subramanya , Dr B S Sharma and Dr SS Rawat and the TN stalwarts Dr Ramesh Babu, Dr Annamalai Vijayaraghavan, Dr Ezhilarasi, Dr Kalpana & Dr Dakshayani were on the floor to make it a conference with impeccable reach . The gracious involvement of senior mentors like Dr L Subramanyam, Dr TU Sukumaran , Dr P Ramachandran, Dr A Balachandran, Dr Mahesh Babu , Dr Thangavelu, Dr K Nedunchelian & Dr Doraiarasan was quite heartwarming. Was so proud to have my Teacher & Guide Dr T U Sukumaran delivering the 26th Prof N Somu oration earlier in the day . Eminent academician in the Pediatric Respiratory domain Dr SK Kabra was honoured with Life time Achievement Award while Dr Varinder Singh, Dr Meenu Singh, Dr Krishan Chugh & Dr D Vijayasekaran, were felicitated with honorary fellowship awards at the inaugural function . Congratulations again to Team Chennai especially the trio of Vijayasekharan Sir , Gowrishankar & Sivabalan for having a successful RESPICON 2022

Diabetes Education for Patients and Families

SAURABH UPPAL
Consultant Pediatric Endocrinologist, ENDOKIDZ
Centre for Growth, Diabetes and Hormones for Children
Jalandhar, Punjab

INTRODUCTION

A structured dialogue at diagnosis and customized educational support to families all along the treatment journey is one of the most cost-effective measures in diabetes management, as it has shown to improve outcomes, reduce hospital admissions and reduce long-term complications due to uncontrolled diabetes [1]. Misinformation by society and lack of empathic diabetes education is an important factor impacting adoption and adherence to treatment.

As is the case with any disease requiring chronic care, education and empathy enable the formation of a mutually beneficial relationship between the caregiver and the patient-family. Effective education at the starting stages of therapy not only improves disease outcomes, but also enables better utilization of consultation time during future visits by allowing incremental learning for families. As with other aspects of management, the importance of a multidisciplinary team approach in diabetes education cannot be overstated. However, the lack of resources and educators in several clinical settings should not deprive the families of the necessary education and healthcare professionals should devise innovative strategies to provide a structured educational model of care as per the regional, cultural necessities as well as the education and comprehension level of the families.

An important difference between diabetes care and care in many other chronic diseases is the multitude of decisions that need to be taken regularly by persons with diabetes or their families. As the decision making cannot always be undertaken by the pediatrician/pediatric endocrinologist/diabetologist caring for the child with diabetes, it becomes imperative that our role as caregivers extends further beyond clinic prescription to promote an empowering patient-centric program that encourages families to manage insulin dosing and lifestyle decisions on a day-to-day basis and make necessary alterations in special situations such as traveling, exercise, school and during intercurrent illness.

THE FIRST VISIT AFTER DIAGNOSIS

The first visit at diagnosis should be utilized in the best possible manner to establish a rapport with the family and impart the basic information necessary to keep the child safe in the initial phase of therapy and education. Care should be taken to customize the information to the parents' educational as well as emotional status. Words may be chosen carefully and the family should not be overwhelmed with too much information. The following information may be imparted at once or in different sessions as part of the initial education:

1. A simplified explanation of the pathophysiology of childhood diabetes and the reason for symptoms.
2. Reassurance about no imminent danger to the child's life because of the availability of recombinant insulin and the rationale behind the lifelong need for insulin replacement.

3. Discussion about the probable causes of diabetes, exploration and redressal of the parental feelings of guilt, blame and grief.
4. Reassurance about the availability of diabetes management techniques enabling patients to achieve a good quality of life.
5. A very brief introduction on different types of insulin (short and long-acting) to be used, their action profiles and initial doses.
6. The rationale behind blood glucose measurement at different times of the day, maintaining a log and its importance in effective diabetes management.
7. Basic dietetic counseling with an emphasis on healthy food choices. Diet advice at initial stages should be kept as simple as possible to encourage routine balanced diet and to avoid overwhelming the family . The practice of providing template based 'diabetic diet" charts should be discouraged. Depending on the opportunity in subsequent sessions and the ability of parents to learn, discussion should progress to involve mindful eating and addressal of myths related to food. More specific nutritional knowledge should be discussed with a gradual transition to carbohydrate counting in the subsequent visits [2].
8. Explanation of symptoms of hypoglycemia and emergency hypoglycemia treatment plan must be shared. Patients should be advised to prepare hypoglycemia kits for school and/or work and for traveling for managing blood glucose fluctuations. Parents/caregivers must be taught about glucagon—indications and technique for its use in times of severe hypoglycemia (blood glucose <45 mg/dL).
9. Clear and consistent instructions about treatment goals, glycemic targets and long-term follow-up plan.
10. Advocating for good injection technique and self-care.

IMPORTANT SKILLS TO BE IMPARTED AT THE FIRST VISIT

The discussion at first visit should be followed by a structured hands-on training session which should cover:

- Insulin delivery devices – Injections/syringes/insulin pump
- Safe injection practices
- Insulin storage and usage
- Blood glucose and blood/urine ketone tests
- Record keeping (diabetes log book/chart)
- Guidelines for disposal of sharps and diabetes technology waste at home.

It is advisable to involve all the caregivers in discussion and the opportunity should be utilized for the sharing of responsibilities. In the authors' opinion, encouraging families to join support groups in the geographical area early in the treatment journey serves the benefit of shared learning, provides the much-needed emotional support for families and improves patient outcomes.

CONTINUING DIABETES EDUCATION

Every clinic visit should be utilized as an opportunity to impart diabetes education. Regular consultations should be structured in a manner that encourages families to discuss their doubts and questions. The clinician should not limit oneself to merely making the required changes in the treatment regimen; explanation of the reasoning behind problem-solving and the suggested adjustments in treatment should be utilized as an important teaching opportunity to improve the understanding among the families. It can improve their understanding of insulin action, diet management, exercise, blood glucose patterns and diabetes technology. The psychological well-being of the child as well as the family should be regularly assessed and issues if any must be addressed during the session or by involving relevant experts [3].

Discussion forums and distant support groups on social media provide new avenues of information, communication, experience sharing as well as continuous education for the families of children with diabetes. However, the relative lack of scientific knowledge in participants, unverified/unvalidated information, personal bias and the absence of accountability in such forums presents a new challenge to the treating physician who now has to ensure that families follow the scientifically validated and evidence-based treatment advice. To keep pace with the rapidly changing diabetes landscape, the need for self-education and updation on part of the clinician cannot be overstated. Only a well informed and empathic clinician can educate the families against sup-optimal and sometimes harmful information. Hence, the establishment and maintenance of a healthy relationship and mutual trust between the family and the treating physician hold much greater importance in today's time.

SUMMARY

Diabetes education is a continuous and progressive process. As management of childhood diabetes improves with addition of new knowledge and technology, effective education is an important contributing factor in ensuring optimal clinical outcomes.

REFERENCES

1. Olinder AL, DeAbreu M, Greene S, Haugstvedt A, Lange K, Majaliwa ES, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Diabetes education in children and adolescents. Pediatr Diabetes. 2022; Sep 19.doi: 10.1111/pedi.13418.
2. Uppal, S. Dietary Management Of Childhood Diabetes. In Khadilkar V, et al (Ed.), IAP Textbook on Pediatric Endocrinology (1st ed.). New Delhi; Japyee Brothers Medical Publishers Pvt Ltd 2019; pp 424-428.
3. Limbert C, Tinti D, Malik F, Kosteria J, Messer L, Jalaludin YM, et al. ISPAD Clinical Practice Consensus Guidelines 2022: The delivery of ambulatory diabetes care to children and adolescents with diabetes. Pediatric Diabetes. 2022; In press.

Psychological Care of Children with Type 1 Diabetes Mellitus

DR. SHALMI MEHTA[1]
DR. RUCHI SHAH[1]
DR MEGHA DESAI[2]
Consultant Pediatric Endocrinologist[1], Endokids Clinic, Ahmedabad
Consultant Child and Adolescent Psychiatrist[2], Ahmedabad, Gujarat

INTRODUCTION

The incidence of type 1 diabetes mellitus (T1D) in children in our country is significantly increasing (4.9 cases/100,000) [1]. The typical day of a child with T1D involves checking blood glucose (BG) frequently, getting multiple injections of insulin, calculating carbohydrates in the diet, and controlling cravings for food etc. Striking a balance among hypoglycemia, hyperglycemia, growth and development and other life events is not easy for healthcare providers, patients or families [2]. The child and the family hence experience a lot of physical, social, emotional and financial burdens.

Almost every fifth child with T1D (20.24%) in India exhibits psychosocial issues of the severe kind [3]. Another study also reported a high incidence of psychosocial morbidity in children with T1D [4]. The main risk factors for depression include being a girl, dysfunctional family, low socioeconomic status, and stressful experiences including diagnosis of T1D [5]. While most efforts are directed towards managing glycemic control, psychosocial care is the most neglected aspect of T1D care.

Let us look at some examples to understand the need of continued psychosocial assessment in children with T1D.

- Masters (14 y) had unusually low requirement of insulin only around 3–4 units/day. In spite of this, child had frequent episodes of hypoglycemia. All medical causes were ruled out carefully. It came as a shock to parents to realize child had his entire set of pens and penfills with him with which he will often overdose to enjoy foods of his choices.
- Miss A (17 y) had lost 7 kg weight in last 2 months. On detailed history, it was found that she had been skipping doses to look skinny.
- Master P (4 y) had come for a regular visit with his parents. HbA1c is 11%, however his logbook shows perfect sugars. On enquiring, it was brought to the attention, that to avoid arguments between parents regarding high BG levels, mother used to alter the readings in the logbook.

These cases highlight the importance of not just glycemic control but also taking care of psychosocial health in these families.

The causes of distress vary according to the various age groups and are listed in Table 1[6]. Thus it is recommended that screening for psychological morbidity and regular psychological assessment should begin from diagnosis. Poor psychosocial health has also shown to affect the glycemic control adversely [7].

Table 1. Causes of distress in children and adolescents with diabetes mellitus

Infants and toddlers	1. Hospitalization itself 2. Frequent insulin injections and blood glucose (BG) checks 3. Dependent on parents for health care 4. Increase in temper tantrums due to inability to express verbally 5. Exacerbation of underlying behavioral disorders 6. Continuous worry and anxiety in the parents regarding management
School going children	1. Reluctance in accepting the diagnosis 2. Sudden change in the lifestyle, change in the way parents and relatives treat them 3. Fear of pricks 4. Fear of disclosure at school 5. Refusal to check BG and take insulin at school 5. Wanting to be back to normal 6. Missing school frequently due to hospitalization 7. Fear of hypoglycemia
Adolescents	1. Realization of the permanent nature of the condition 2. Fear of complications 3. Tired of reminders and getting reminded 4. Peer pressure 5. Desire for more independence leading to distress among adolescents and parents 6. Injecting more/less insulin causing hospitalization 7. Prevalence of eating disorders, alcohol, cigarette smoking, drug abuse
Parents	*T1D is a life altering diagnosis not only for child but also for parents and care-givers* 1. Denial to accept the diagnosis, use of alternative therapies, and ways to reduce pricks to the child 2. Parental disputes over child's management, separation, job changes, many mothers prefer to become stay at home mothers to take care of the child 3. Added financial burden increases mental stress 4. Fear of hypoglycemia, especially when the child is not with parents 5. Sleep disturbances – due to BG checks at night 6. Worry about future 7. Treating all children in the family with or without diabetes equally is a task
Siblings	1. Often feels less care and time are given compared to diabetic child 2. Restrictions in lifestyle due to diabetic sibling

HOW TO IDENTIFY SIGNS OF PSYCHOLOGICAL DISTRESS

Along with the routine causes of depression and anxiety, children and families undergo through diabetes distress (Table 2). It is the negative emotion or effect experienced by approximately 30% of adolescents with T1D [8]. Along with these, eating disorders, substance abuse and issues with body image are seen more in these children, affecting the glycemic control further (Table 2)[9].

MANAGEMENT OF PSYCHOLOGICAL DISORDERS IN CHILDREN WITH T1D

The salient features in the management of psychosocial disorders in children and adolescents are listed below briefly.

- It is very important for the treating physician to be able to suspect symptoms of maladjustment or psychological issues. Hence an open communication link is to be established between the physician, family and the child.
- Also healthcare providers should be given basic training in the identification and primary intervention for psychological disorders. They should be able to timely refer these patients to appropriate personnel.
- Interdisciplinary diabetes healthcare teams should include mental health specialists namely psychologists, social workers, and psychiatrists.
- Regular screening for depression and co-occurring anxiety and diabetes distress will help identify the patients in need of an intervention[10].
- Age-appropriate and validated assessment tools should be routinely implemented in clinical practice to monitor and discuss overall psychosocial well-being and quality of life of all youth with diabetes [11], especially for children > 12 years, once a year.

PATIENT ORIENTED INTERVENTION

These include:

- Shared decision making should be practiced right from the diagnosis or at an appropriate age so that the child feels more involved and responsible for her/his health.
- Patient awareness programs should be regularly conducted which gives an opportunity for the child to learn more about her/his condition.
- Technology especially continuous glucose monitoring (CGM) system has proven quite

Table 2. Clinical features of depression, anxiety and diabetes distress

Depression	Anxiety	Diabetes distress
Low mood	Excessive worry	Feeling of life controlled by diabetes
Low energy levels	Restlessness	Fear, anger or despair about living with diabetes
Lack of concentration	Fatigue	Social isolation or feeling lack of support
Altered appetite	Impaired sleep	Fear of not being able to keep up with diabetic care
Anger outbursts	Poor concentration	Lying about the blood glucose (BG) levels, malingering in the logbook
Irritability Suicidal thoughts/attempts	Physical symptoms of palpitations, breathlessness, sweating	Injecting too much insulin for foods or too less insulin to lose weight

effective in improving glycemic control. It also helps in decreasing the parental anxiety regarding hypoglycemia, school hours and sleep. Insulin pumps are also beneficial since they provide 24 hr delivery of insulin, thus helping improve the overall quality of life of the child and parents.

- Support groups play a very important role in preventing feeling of left alone. Hence children should regularly attend these meetings.
- Timely referral to the mental health specialists should be done if any symptoms or signs of anxiety, depression or diabetes stress or substance abuse are seen.

FAMILY ORIENTED INTERVENTION

The important aspects include the following:

- Both the parents should share responsibility equally to prevent diabetes burnout. Other family members should also be involved in diabetes care. All the members should be on the same page while communicating with the child.
- Parents should be made well equipped in handling emergencies like hypoglycemia and sick days.
- Lot of anxiety exists among the parents regarding the diagnosis, treatment and long-term complications. Hence parents often resort to alternative therapies. Diabetes education at the time of diagnosis and with ongoing care plays a very important role in allaying the anxiety in the parents and clearing their doubts and myths.
- Family talks should not be always centered on diabetes.
- Parents should also be encouraged to be a part of the diabetes support groups and thus build a community among themselves.
- If needed, parents should be provided counseling and therapy to avoid worsening of mental health.

COMMUNITY INTERVENTION

The major interventions to be done at the community level include the following:

- Schools play a very important role since children spend a lot of a day at school. The nurse or a teacher at the school should come forward to help in managing diabetes at school.
- Any abnormal behaviors can be brought to the attention of the parents and the healthcare professionals since these could be signs of psychological disorders.
- It is important to create awareness about T1D in the community via newspapers, television, social media so that this condition is not considered as a taboo.
- Mental health specialists should be made a part of the health care team at all levels of health care including the primary health care.

SUMMARY

Type 1 diabetes mellitus is a permanent condition, thus requiring lifelong care and management. The injectable treatment, regular monitoring, fear of hypoglycemia, diabetic ketoacidosis and long-term complications, and the various developmental stages of a child can lead to signs and symptoms of anxiety, depression and diabetes stress and burnout in the patient and the family, leading to poor glycemic control. Thus timely intervention in terms of identifying patients at risk of developing psychosocial problems and offering help becomes utmost important.

Since pediatricians are most often the first line of contact for the family and most trusted, every one of us should be able to identify such children and adolescents and refer them timely to mental health specialists.

REFERENCES

1. Jensen TE, Dabelea DA, Praveen PA, Amutha A, Hockett CW, Isom SP, et al. "Comparison of the incidence of diabetes in United States and Indian youth: An international harmonization of youth diabetes registries." Pediatr Diabetes. 2021;22(1):8-14.
2. Guthrie DW, Bartsocas C, Jarosz-Chabot P, Konstantinova M. (2003). Psychosocial Issues for Children and Adolescents With Diabetes: Overview and Recommendations. Diabetes Spectr. 2002;16(1):7-12.
3. Khandelwal S, Sengar GS, Sharma M, Choudhary S, Nagaraj N. Psychosocial Illness in Children with Type 1 Diabetes Mellitus: Prevalence, Pattern and Risk Factors. J Clin Diagn Res. 2016;10(9):SC05-SC08.
4. Chatterjee S, Bakhla AK, Biswas P, Singha S, Dubey S, Sharma CB, et al. Psychosocial morbidity among children with type-1 diabetes mellitus. J Family Med Prim Care 2020;9(2):652-656
5. Grey M, Whittemore R, Tamborlane W. Depression in type 1 diabetes in children: natural history and correlates. J Psychosom Res 2002;53(4):907–911.
6. ChaoM, Minges KE, Park C, Dumser S, Murphy KM, Grey M. et al. General Life and Diabetes-Related Stressors in Early Adolescents With Type 1 Diabetes. JPediatr Health Care 2016;30(2):133-42.
7. Buchberger B, Huppertz H, Krabbe L, Lux B, Mattivi JT, Siafarikas A. Symptoms of depression and anxiety in youth with type 1 diabetes: A systematic review and metaanalysis. Psychoneuroendocrinology 2016;70:70-84.
8. Hagger V, Hendrieckx C, Sturt J, Skinner TC, Speight J. Diabetes Distress Among Adolescents with Type 1 Diabetes: a Systematic Review. Curr Diab Rep 2016;16(1):9.
9. Wagner G, Karwautz A. Eating disorders in adolescents with type 1 diabetes mellitus." Curr Opin Psychiatry 2020;33(6):602-610.
10. Hilliard ME, De Wit M, Wasserman RM, Butler AM, Evans M, Weissberg-Benchell J, et al. Screening and support for emotional burdens of youth with type 1 diabetes: Strategies for diabetes care providers. Pediatr Diabetes 2018;19(3):534-543.
11. de Wit M, Gajewska KA, McDarby V, Zhao X, Hapunda G, Goethals ER, et al. SPAD Clinical Practice Consensus Guidelines 2022: Psychological care of children and adolescents with type 1 diabetes. Pediatr Diabetes 2022; In press

Care of Children with Diabetes in Low Resource Settings

ANJU VIRMANI
Director, Pediatric Endocrinology
Max Smart Super Specialty Hospital, Saket, New Delhi &
Madhukar Rainbow Children's Hospital, New Delhi
Founder President, ISPAE; Former AC member ISPAD, Director, IDEAL

INTRODUCTION

Umang is a 11-year-old boy in a small village in Gujarat, diagnosed with type 1 diabetes (T1D) two years ago. He goes to the village school, but missed it when he must work in the fields to supplement the meagre family income. He and his family eat two meals a day—mid-morning and at night—but even those meals are without any certainty. School provides a mid-day meal on many days (but not always). The nearest Health Center is 50 km and about 3–5 hours away—if the bus comes. The doctor there prescribed premixed (30/70) insulin to be injected twice a day. He got it free from the health center—if supplies had been received. He often felt hungry, tired and shaky, his hands trembled, and he would not be able to work. Last year, he got hypoglycemic seizures at night because there was not enough food for dinner. His mother managed to put some shakkar (raw sugar) into his mouth and save his life. Since then, if he had to work in the fields, he would skip the morning insulin for fear of hypoglycemia. He had become poor in studies. He would get up 2–3 times every night to pass urine, and had not grown much in height since diagnosis.

Six months ago, a new doctor was posted at the Health Center. Umang and his parents were provided two different types of insulin. They were taught to take NPH at night, and Regular insulin (RI) half an hour before each meal, as many times as he ate food. The insulins had to be kept cool in a double matka (Fig. 1) The doctor provided them a glucometer and bottle of strips, taught them how to test blood glucose (BG) and increase or decrease the RI dose according to the BG level and amount of food. He has to write his BG and dose numbers in a copy. Umang finds his hunger and urine frequency have reduced, energy levels have increased; he can sleep, work and study better. Now he can quickly complete farm work early in the morning and after school. Better school attendance means he gets more mid-day meals (if food is given, he runs home to takes his insulin). He has gained weight and height; understanding words and numbers in school is better. So he has learnt to maintain diabetes records and adjust doses well. Suddenly, Umang is not the butt of everyone's jokes and pity, just another young boy, who has the energy to sometimes play pranks also.

Figure 1: Matka – Make your own insulin cooler
(Courtesy: Dr Archana Sarda)

In the last 100 years, it has become possible to live after being diagnosed with T1D. However, for decades, most doctors and policy makers in developing countries, in limited resource settings (LRS) were convinced that T1D is too infrequent a problem to waste time and resources on, so children and adolescents continued to die undiagnosed or within a few years of diagnosis, of acute complications [1]. The "lucky" few who survived despite pity and neglect, would often develop chronic complications like blindness or renal failure. The recently released T1D Index has used mathematical modelling to get estimates of the ground reality, in the absence of reliable data in LRS [1]. However, this unremittingly bleak picture need no longer be true [2]. Many changes in the last few decades have transformed lives, and we start the next 100 years with hope. Different aspects of management are well covered elsewhere in this and an earlier issue, so this is an overview of the scenario which can be achieved in LRS.

EXPECT NORMALCY, NOT JUST SURVIVAL

Several changes over the past few years have made it possible for the child, adolescent or young adult with diabetes to live, study, work and marry near-normally, without constant fear of hypoglycemia, ketoacidosis or other complications. When the young person is diagnosed with diabetes, the shocked parents and other family members turn to the pediatrician they are familiar with, for validation. It is important for pediatricians to have basic awareness of good diabetes management, in order to help the family accept the situation, and implement and coordinate care with the diabetes care team (DCT), so that the attitude of the person with diabetes (PwD) is not "at least I am alive", but "I want good quality of life (QoL)".

REDUCING GLYCEMIC VARIABILITY

It was astonishing to find that chronic complication rates were lower in the early decades when RI was given 5–8 times every day (including the middle of the night), than when discovery of longer acting insulins like NPH, Lente, and Ultralente reduced the number of injections taken. We now understand the reason was that the fluctuating BG levels (glycemic variability) caused organ damage. Extensive data shows that good glycemic control i.e. reduced glycemic variability, greatly improves QoL, while reducing the burden of acute complications (hypoglycemia, diabetic ketoacidosis [DKA], and decreased immunity) and chronic complications (renal failure, blindness, and heart disease).

REPLACING INSULIN PHYSIOLOGICALLY

The body secretes insulin in a basal + bolus manner. The importance of replacing insulin in the same way, i.e. the most physiologic manner possible, has been realized. The almost painless injections with current-day ultrafine 31G needles cause less distress than fluctuating BG. This has resulted in strong advocacy for basal-bolus (multiple daily injections: MDI) regimens for each and every person with T1D [3]. Premixed insulins drastically decrease flexibility and ability to adjust doses. Even worse are 2-dose regimens—insulin given twice a day inevitably causes hyperglycemia after mid-day,

and frequent nocturnal hypoglycemia (which is the most dangerous).

AFFORDABLE INSULINS

We should be thankful that in India, access to insulin is the best it has ever been. A range of insulins are available—from the older "conventional" insulins (Regular and NPH) to the newer, expensive analog insulins. Costlier insulins are not necessarily better. Used intelligently, the inexpensive conventional insulins can control glycemia well. Cheaper insulins are more sustainable, and reduce the need to ration or miss doses. An additional benefit is that the money saved can be used to buy glucostrips and ensure regular self-monitoring of BG (SMBG), discussed below.

India is the largest producer of generic drugs. Multiple Indian companies manufacture biosimilar conventional insulins and glargine, so we are not at the mercy of the 'Big 3'. The actual prices of the conventional insulins and glargine have remained almost unchanged for the last two decades while purchasing power has increased, i.e. they have become much more affordable. Increased manufacturing capacity has increased availability [4]. Glargine was included in the Essential Medicines List (EML) by WHO in 2021 and by India in 2022, leading to price control. Since 2008, increasing numbers of Jan Aushadhi Kendras set up by the Government of India, provide low-cost generic drugs, including insulins. With increased awareness, more donors are helping T1D children. Government and charity donors should be persuaded to provide the cheaper insulins, and provide for self-monitoring of BG (SMBG). Families buying insulin out of pocket can also buy from Jan Aushadhi Kendras or form groups to buy in bulk from distributors to reduce costs.

USING INEXPENSIVE INSULINS INTELLIGENTLY

In basal-bolus regimens using Regular and NPH insulins, the 'BASAL' needs are met with one or two doses of glargine (or NPH if socioeconomic status is very low). The BOLUS needs are met by RI, given before each meal and large snack, i.e. in the morning before breakfast, in school before "tiffin", before lunch, and before dinner. It enables tailored insulin dosing—if the BG is low/ food amount is less/ physical activity has been or will be high, the insulin dose is reduced. If BG is high/ extra food is anticipated (festivals, celebrations, just impulsively)/ activity is less, the dose is increased. If food is delayed (or even unavailable), there is no need to panic or to suffer hypoglycemia—the bolus dose is delayed (or missed). Glycemic variability, crippling hypoglycemia, and parental fear are reduced. Earlier, we would hear of families who stopped attending weddings or parties because their child was forbidden festive foods, especially sweets. This caused rebellion in the child, and resentment among family members. Now, there is no "poor, sick diabetic boy/ girl" who feels like a burden, just a child who tests and self-injects before eating.

Thus, various options can be:

a) once-daily glargine in the morning (or at night) with R+R+R insulin (and R+R+R+R on school days), or

b) NPH at night with R+R+R insulin (and R+R+R+R on school days), or

c) R+NPH insulin (mixed in the same syringe) before breakfast, R insulin before day meal/ snack/ both, and R+NPH insulin (mixed in the same syringe) before dinner.

This would be discussed in detail in the ambulatory care section.

Are MDI regimens practical in India?

The group from SGPGI, Lucknow, a government teaching organization catering mostly to low SES families mainly from UP, reported recently that 97% of their T1Ds are on MDI regimens, regardless of age, education, SES, or residence (urban or rural) [5]. Incidentally,

their mortality had dropped to 1.1%, compared to 7% they had earlier reported in 2004 [6].

Must insulin be wasted?

Manufacturers variably recommend discarding insulin after 4–6 weeks of opening, though there is insufficient data to support this. These recommendations have even varied for the same insulin in different countries. In LRS, we suggest not wasting any insulin, but using it fully, the doses being adjusted as needed based on SMBG. The families are taught to not miss any BG testing and to exercise caution whenever a new vial is started, in case the dose needs to be decreased.

THE IMPORTANCE OF SELF-MONITORING OF BLOOD GLUCOSE

It has been realized that SMBG is crucial to improve glycemia and QoL, i.e. it is an essential pillar of T1D management. We have progressed from boiling urine in Benedict's solution to glucostrips with glucometers, and now continuous glucose monitoring (CGM). In general, the more frequent the monitoring, the easier it is to improve glycemia. At the very least, 4 tests per day are needed: before each meal to decide insulin dose, and at bedtime to decide amount of bedtime snack or need for correction. As with insulins, increased Indian manufacturing and decreased prices have meant glucostrips which cost Rs 50 each in 1988, today cost even Rs 7–8 each. In LRS, BG testing meters and strips must be arranged through governments or charity, to enable maximal SMBG for all persons with T1D. Some state governments now provide monthly supplies of glucostrips. Apart from routine SMBG, testing is crucially needed if hypoglycemia is suspected/ has occurred; and during sick days, when hyperglycemia may lead to ketoacidosis if uncorrected. Such access to BG testing is particularly important in LRS, especially in remote areas, because timely medical care for an emergency may be unavailable, or very expensive.

Is SMBG impractical in LRS?

A common refrain is that SMBG is expensive. However, not testing is often even more expensive. One episode of severe hypoglycemia or DKA may cost the family (travel, hospital costs, lost wages, other losses) and the Health Care System, as much as several weeks or even months of SMBG, and may even prove fatal. In the same way, use of CGM can further reduce the burden of acute and chronic complications and improve QoL. If regular use of CGM cannot be afforded, anecdotally intermittent use (e.g. using once every few weeks or months) has been shown to help improve understanding of BG patterns and glycemic control. This can be particularly useful when routines are difficult to follow, e.g. sick days or travel or examinations.

THE MYTH OF THE "DIABETIC DIET"

PwD may be needlessly advised restrictive diets (so called "diabetic diet") and discouraged from physical activity, increasing their sense of being different and a burden. The current-day concept is to encourage them and all their family members to have healthy, balanced food, and avoid processed foods containing high fat, sugar, and salt (HFSS). Ensuring adequate protein, fat, fiber and complex carbohydrates in the right proportions need not be costly, but does need awareness and education. They should all also be encouraged adequate physical activity. The PwD and family are taught how to manage exercise by testing BG and adjusting insulin and food to avoid hypoglycemia. T1Ds have even participated in extreme sports.

MONITORING IS HIGHLY COST-EFFECTIVE

Pediatrics is the science of the growing child. Serial monitoring and recording of growth, BP and puberty costs nothing, but yields valuable information. A PwD with optimal glycemia should have normal growth and puberty. Care is needed to avoid obesity, even in low SES families! In addition, it is important to monitor

injection sites, insulin handling and techniques of injection, SMBG, and other aspects of care. HbA1c should be checked every 3 months; this may be practical if periodic camps are held with point-of-care testing. TSH can be tested annually, or earlier if growth velocity is abnormal; other thyroid tests are hardly ever needed. Other screening modalities are more nuanced, and discussed elsewhere.

THE IMPORTANCE OF DIABETES SELF-MANAGEMENT EDUCATION (DSME)

It is obvious from the above discussion that proper management of the PwD is possible only if the DCT is knowledgeable, and DSME is given systematically and reinforced regularly. However, expert DCT are not available everywhere. Here telemedicine, used extensively during the Covid lockdowns, can be handy. The pediatrician and family should be in touch via telemedicine with any DCT (pediatric endocrinologist + pediatric diabetes educator [PDE] + dietician familiar with T1D + mental health specialist) they are comfortable with. More PDEs are urgently needed in LRS like India. ISPAE (Indian Society for Pediatric & Adolescent Endocrinology) recently initiated IDEAL (ISPAE Diabetes Education And Learning), which is discussed elsewhere. This is a comprehensive online course to impart PDE training to nurses, dieticians, and educators treating persons with T1D, to help fill this gap [7]

TAKING CARE OF MENTAL HEALTH

Managing T1D for a lifetime is mentally, physically and financially exhausting, and can cause diabetes distress, or even depression. Mental support by the DCT, or by self-help groups, NGOs, and "senior" PwDs or families, can significantly help in coping, and maintaining good control and QoL. Widespread access to mobile phones has enabled even low SES families to stay in touch with each other and offer practical medical and emotional support. In addition, avoiding words like "diabetic", "sick", "suffering from" which have a negative impact, preferring more positive options e.g. "person with diabetes", "has diabetes" costs nothing, but the changed mindset yields good results.

SUPPORT SYSTEMS

With increasing awareness of T1D, many organizations now offer financial, emotional and other help. The Indian Central Government, through Ayushman Bharat and other programs, and State Governments are gradually increasing support. Since 2013, the state of Kerala is providing insulin, glucostrips, DSME, and lab support under the comprehensive Mittayi Program (described elsewhere). International bodies including LFAC and CDiC, and many local NGOs offer supplies and logistic support. A regularly updated list of Indian organizations (including NGOs) is available on the ISPAE website (ispae.org.in). National bodies like ISPAE and international organizations like International Society for Pediatric & Adolescent Diabetes (ISPAD) and Juvenile Diabetes Research Foundation (JDRF) offer education and research.

In short, greater experience, improved technology, and several resources are currently available for managing the child, adolescent and young adult with diabetes, yet, many PwD continue getting sub-optimal care. It is our responsibility as health care personnel to improve this situation and ensure care is best possible under the available circumstances, while striving for better care when feasible.

SUMMARY

In summary, T1D care can be substantially improved by an aware pediatrician in coordination with a competent DCT. The aim is to offer these children the ability to grow up physically and mentally healthy, with good glycemic control and QoL, and minimal acute and chronic complications, into well adjusted, productive adults.

Declaration

I declare no competing interests and no funding support.

REFERENCES

1. Gregory GA, Robinson TIG, Linklater SE, et al. Global incidence, prevalence, and mortality of type 1 diabetes in 2021 with projection to 2040: a modelling study. The Lancet Diabetes & Endocrinology. 2022;10(10):741-760. doi:10.1016/S2213-8587(22)00218-2.

2. Virmani A. Type 1 Diabetes in India: The Numbers Show the Way Ahead. Indian Pediatr 2019; 56(3):189-190. PMID: 30954987.

3. Virmani A, Brink SJ, Middlehurst A et al, Management of the child, adolescent and young adult with diabetes in limited resource settings. ISPAD Clinical Practice Consensus Guidelines 2022 (in press)

4. Gotham D, Barber MJ, Hill A. Production costs and potential prices for biosimilats of human insulin and insulin analogues. BMJ Glob Health. 2018; 3e000850

5. Sudhanshu S, Nair VV, Godbole T, Reddy SJB, Bhatia E, Dabadghao P, et al. Glycemic Control and Long-term Complications in Pediatric Onset Type 1 Diabetes Mellitus: A Single-center Experience from Northern India. Indian Pediatr 2019; 56: 191-195.

6. Bhatia V, Arya V, Dabadghao P, Balasubramanian K, Sharma K, Verghese N, et al. Etiology and outcome of childhood and adolescent diabetes mellitus in North India. J Pediatr Endocrinol Metab. 2004;17:993-9.

7. Boddu SK, Virmani A, Salis S, Singh P, Olety S, Irani A, et al. A comprehensive and completely virtual pediatric diabetes educator training model which is affordable, accessible, and hence "IDEAL" (ISPAE Diabetes Education And Learning) for developing countries and other limited-resource settings. Presented at ISPAD Annual Scientific Meeting, Abu Dhabi, October 2022.

ISPAE at the Forefront for Improving Care of Children with Diabetes

DR SHAILA S BHATTACHARYYA
President ISPAE 2021-22
Professor of Pediatrics and Pediatric Endocrinology
Shivajoyti, Indiranagar, Manipal Hospital, Bengaluru, Karnataka

INTRODUCTION

Since its inception 25 years ago, the Indian Society for Pediatric and Adolescent Endocrinology (ISPAE) has been working towards improving the care of children with various endocrine disorders. When I took over as the president of ISPAE in January 2021, we were in the peak of the COVID-19 pandemic. We had a young dynamic and passionate team of pediatric endocrinologists with brilliant ideas in the ISPAE executive committee — Ganesh Jevalikar (Secretary-Treasurer), Rakesh Kumar (Joint Secretary), Mahesh Maheshwari, Sirisha Kusuma Boddu, J Dhivyalakshmi, Preeti Singh, Amarnath Kulkarni, Hari Mangtani and Deepa Anirudhan (Executive Council Members).

With a large number of children with diabetes in our country, we realized that there is a lacunae of trained healthcare professionals and there is an urgent need to develop a cadre of certified pediatric diabetes educators to help ease the burden of care and improve the quality of lives of our children living with diabetes. We thought that this is the best time to execute a structured, comprehensive and economical type 1 diabetes (T1D) course using a virtual platform so that we can have a wider reach and train physicians, diabetes educators, nurses, dietitians and T1D advocates. IDEAL (ISPAE Diabetes Education and Learning) program, the first-of-its-kind dedicated certified course on diabetes education for childhood diabetes thus came into being.

IDEAL (ISPAE DIABETES EDUCATION AND LEARNING) PROGRAM

Team IDEAL

As the saying goes "alone we can do so little; together we can do so much". I was fortunate to have a cohesive and passionate team who helped realize my vision and mission.

IDEAL has a nine-member core committee and a team of experienced and learned faculty from all over India with expertise in the management of childhood diabetes. After long hours of brainstorming and intensive review sessions, the core committee along with the faculty team conceptualized and developed a comprehensive curriculum that covers both basic and advanced aspects of managing pediatric diabetes. The core committee comprised of Dr. Shaila Bhattacharyya, Dr. Anju Virmani, Dr. Aspi Irani, Dr. Santhosh Olety, Dr. Preeti Singh, Dr. Sirisha Kusuma Boddu and Ms. Sheryl Salis (registered dietitian and certified diabetes educator) with Drs. Ganesh Jevalikar and Dr Rakesh Kumar as ISPAE office-bearers. Our team of 54 faculty members who were well versed in managing children with diabetes hailed from all over India.

Curriculum

IDEAL is a structured online training course consisting of 17 teaching sessions, 6 review sessions, and an exit examination. The

two-hour-long sessions are conducted twice a week. Each session has a couple of didactic lectures (30 minutes each) followed by an hour-long interactive and case-based discussion on the module. Both pre- and post-tests are conducted using Google forms before and after each session respectively. Practical assignments are given to the participants based on each module, to evaluate their understanding, application of knowledge, and communication skills. The participants are expected to complete the entire course module, with >80% attendance, including the pre- and post-tests along with the timely submission of practical assignments. The final certificate of successful completion of the course is awarded only if the participant fulfils the above requirements and exhibits reasonable competence as a pediatric diabetes educator in the exit examination (written and viva).

Application and enrollment

Applications are invited a month prior to the start of each batch. The pre-requisite is that the applicants must be counseling children with T1D. The online application form also requires submission of a recommendation letter from the head of department — preferably a pediatrician/pediatric endocrinologist/adult endocrinologist/dietitian. Application procedure and links for registration and entry assessment can be found on the ISPAE website www.ispae.org.in/ideal. Contact: ispae.ideal@gmail.com

Journey so far

It has been a very enriching and fulfilling journey so far. The IDEAL core committee is proud to announce that since the course commencement in October 2021, we have successfully trained three batches of students, the last one being physicians from urban and rural areas. The third batch (June-Aug 2022) was specially tailored for physicians with an enhanced and much more detailed curriculum that elaborates more on the etiopathogenesis of T1D, management of diabetic emergencies, insulin dose adjustments, diabetes care at school, etc., in addition to the existing modules. The fourth batch of IDEAL is currently ongoing.

The three-month course continues to be as rigorous as ever, with pre and post-tests before and after each teaching session, ample time for small group discussions during teaching sessions, and assignments for the trainees to submit within a week of the respective teaching session. The assignments are designed in such a way that they improve the retainment of the taught principles. They also help the trainee practice what he/she has learnt in the preceding session by implementing the principles of patient care and counseling in the clinic setting.

The success of IDEAL would not have been possible without the dedication and commitment of our team of faculty hailing from all over India, volunteering their time, efforts, and expertise. As the veteran faculty continue to lend their support, new faculty members are also joining in to contribute to the noble cause.

BASIC EDUCATION SERIES IN TYPE 1 DIABETES (BEST)

Somewhere along the way, we realized that just training educators would not suffice, we need to reach out and educate our parents and caregivers of children with diabetes, especially if educators are not available in their cities or towns. Keeping this in mind, we designed a structured diabetes education module called “Basic Education Series in Type 1 Diabetes” (BEST) for children with T1D and their caregivers. This course is open to parents/caregivers of children with T1D, adults with T1D, nursing assistants, physician assistants, and school personnel to learn the basics of T1D and its ambulatory management.

Program implementation

BEST has an eight-member core committee and an excellent team of 27 faculty across the country with rich experience and expertise in managing childhood diabetes. The team has

developed a series of eight structured online educational modules to train the participants with the basics and essential skills in managing T1D.

BEST teaching sessions are conducted over four weeks: one session of 2 hours duration per week (Tuesday 7–9 PM). A maximum of 30 participants are selected per course to ensure engagement and one-to-one interaction between the faculty and the participants. The first and second batches of BEST participants were successfully trained in April and July 2022, respectively. The enrolment for the third batch is underway and will begin on the 6 December, 2022. Contact ispae.best@gmail.com

WORLD DIABETES DAY – ISPAE (14 NOVEMBER 2022)

November 14 is marked as 'World Diabetes Day' worldwide to create awareness about diabetes. To commemorate this day, under the able leadership and guidance of Dr. Shaila Bhattacharyya, ISPAE organized an awareness and education program addressing, in particular, persons with T1D (PwD) and their parents/ caregivers.

Living with T1D is associated with many challenges as the child's needs change with his/ her age and thus the requirements of a toddler with diabetes are completely different and their parents, ISPAE organized a public forum by conducting a panel discussion with the entire team of members, Dr. Shaila Bhattacharyya, as the moderator and Drs Ganesh Jevalikar, Rakesh Kumar, Mahesh, Sirisha Kusuma Boddu, J Dhivyalakshmi, Preeti Singh, Amarnath Kulkarni, Hari Mangtani and Deepa Anirudhan as panelists.

It was well attended and is now available on YouTube for people to view and benefit from the session. The panel discussed various aspects of TID, including its definition, cause, and the basic pathophysiology of Insulin deficiency. The diagnosis and treatment were discussed in detail. The need for insulin and the management of hypoglycemia and hyperglycemia was also discussed. Useful tips were given by the experts about the management of T1D at school and during sick days. The difficulties in managing adolescent diabetes were also addressed and many myths and misconceptions associated with T1D were busted by the panelists.

There were several questions from participants which were patiently answered by the panelists in detail. The well-moderated session was greatly appreciated by the participants.

ASSOCIATION OF ISPAE WITH ISPAD (INTERNATIONAL SOCIETY OF PEDIATRIC AND ADOLESCENT ENDOCRINOLOGY)

The members of ISPAE have contributed to various chapters in the ISPAD Clinical Practice Consensus Guidelines, 2022. These include Dr Preeti Dabadghao on type 2 diabetes, Dr Ahila Ayyavoo on insulin treatment, Ms. Sheryl Salis for medical nutrition therapy, Dr Ganesh Jevalikar for hypoglycemia, Dr Leena Priyambada on diabetic ketoacidosis, Dr Banshi Saboo on glucose monitoring, Dr Leenatha Reddy on new technologies on insulin delivery, Dr Kriti Joshi on diabetes in adolescence, Dr Anju Virmani and Archana Sarda for care of children in limited resource settings. Dr Leena Priyambada was the lead project coordinator.

Dr Leena Priyambada and Dt Sheryl Salis were elected as ISPAD council members from 2022 to 2026.

SCHOOL MODULE TO EDUCATE TEACHERS

ISPAE will be releasing a school module soon to educate teachers in school to take care of children with diabetes especially when they are in the school. Teachers will be educated about day-to-day care, hypoglycemia recognition and treatment, exercise and nutrition for these children.

SUMMARY

IDEAL is a structured three-month online training program for physicians and educators which has a comprehensive curriculum that covers both basic and advanced aspects of managing pediatric diabaetes. BEST is a structured diabetes education module for children with T1D and their caregivers. This course is open to parents/ caregivers of children with T1D, adults with T1D, nursing assistants, physician assistants, and school personnel to learn the basics of T1D and its ambulatory management. In the coming years we plan to come up with more awareness and educative modules for parents and public about not only Diabetes, but also other common endocrine problems.

Public and Private Partnership to Improve the Care of Children with Diabetes – The Kerala Model

DR M VIJAYAKUMAR
Professor, Department of Pediatrics
Government Medical College, Kozhikode, Kerala

INTRODUCTION

Childhood diabetes is a chronic illness and understanding the disease and managing it effectively is often complicated and challenging, both to the caretakers and caregivers. The patient requires parenteral medications (as of now) and (almost) continuous blood glucose (BG) monitoring day in and day out. The child and caretakers should be vigilant about the frequent acute complications including hypoglycemia and hyperglycemia. The development of long-term complications, which can affect all the major organs, is a potential threat which have to be anticipated and prevented. The child and family may pass through all the steps of any chronic illness and need emotional support and care. Financial burden is tremendous which adds to the misery. Hence without public support, attainment of optimal treatment goal is a myth or illusion. The comprehensive social support should be given to all patients in the form of financial, emotional, informational and physical measures [1].

Childhood diabetes is not a rare disease. The incidence in USA is 22.3 per 100,000 population [2]. We do not have a robust data in India and the available studies show a prevalence between 10.2 to 17.93 per 100,000 children below the age of 15 years [3].

THE CONCEPT OF COMPREHENSIVE CARE AND THE INTRODUCTION OF MITTAYI PROGRAM

With the intention of comprehensive care for children with diabetes, the Department of Social Justice, the Government of Kerala decided to initiate a comprehensive program for children with diabetes and their family members. The Mittayi program was officially launched in May 2018 by the Government of Kerala. Kerala Social Security Mission (KSSM) is the implementing agency for this initiative [4]. The program was initially started with enrolment of 300 children. Currently, about 1200 children with diabetes mellitus have enrolled and it is planned to register 300 more children soon. Mittayi clinics have been established in all the major Government Medical Colleges in Kerala (Thiruvananthapuram, Kozhikode, Kottayam, Thrissur and Alappuzha). Nine satellite canters have also been started in the year 2020 in major government hospitals, one hospital in each district (District Hospital Kasaragod, Government Medical College Kannur, Government Hospital Kalpetta, District Hospital Perinthalmanna, Women and Children Hospital Palakkad, Government Medical College, Ernakulam, District Hospital Idukki, District Hospital Pathanamthitta and District Hospital, Kollam).

Aims and Objectives of the Program

1. Early identification of type 1 diabetes mellitus (T1D). Awareness creation to health care personnel, social workers, teachers and public
2. Strengthening institutional mechanisms for effective clinical and laboratory diagnosis
3. Providing free and comprehensive treatment and monitoring with most appropriate technologies
4. Systematic and scientific nutritional management and monitoring
5. Ensure special care and attention at school
6. Physical activity management including prevention and management of complications during exercises
7. Awareness creation and training of parents, self and teachers about sick day rules, management of complications and hypoglycemia
8. Address the psychological aspects of the patients and family members
9. Training to deal with special situations like travel and holidays
10. Conducting diabetic camps and other outdoor activities.

Eligibility Criteria

- The beneficiary (child/adolescent) should be below the age of 18 years (However the state level technical committee would be empowered to make exceptions on a case-by-case basis if the committee feels that there are compelling technical reasons warranting an exemption)
- The annual family income of the applicant should be below Rs. 200,000/-
- The applicant/parent should be a permanent resident of Kerala
- The child/adolescent should be certified by an empanelled doctor (under the scheme) regarding the diagnosis of childhood diabetes and recommended therapy schedule under the project
- Parents of the child/adolescent should be ready to sign an informed consent before beginning the therapy
- Parents of the child/adolescent should be ready to undergo mandatory training including residential diabetes camps.

State Level Technical Committee

The State Level Technical Committee is constituted to develop criteria for empanelment for doctors and institutions and technical advice to KSSM and the Government in the implementation of the scheme from time to time. The committee will recommend standards and specifications for insulin preparations, glucometers, insulin pump, continuous glucose monitoring (CGM) systems or any other drug, device or equipment required as a part of the project. It is responsible for the selection of beneficiaries of the project after scrutinising the applications and relevant reports based on the inclusion criteria.

Members of the Committee

Executive Director, KSSM is the convener of the committee, other members include:

- Director, Indian Institute of Diabetes, Kerala
- State Nodal Officer, NCD program, Directorate of Health Services, Kerala
- Two pediatricians, not below the rank of Assistant Professor, from Government Medical Colleges in Kerala, and having experience in treating children with diabetes
- One physician/pediatrician from private, specialized in managing diabetes mellitus

The committee is empowered to invite other experts for technical advice if required. The

state level technical committee may also call up on the children/adolescents with diabetes mellitus with their parent/guardian if required, for making appropriate decision.

Patient Enrolment

Mode of selection is based on a 3-step process. In step 1, patient will upload the details in the website in the prescribed format along with other enclosures (e.g. medical certificate from the treating physician, income certificate from appropriate authorities, Aadhaar card etc.). in step 2, the state level technical committee scrutinizes each application and will select the case for inclusion under the scheme (if the patient's application satisfies the approved criteria). In step 3, the name and details of approved cases are forwarded to the Mittayi clinic for initiation of treatment and follow-up.

The upper age limit is 18 years and once enrolled, the child will get the benefit for 20 years. Even though the scheme is primarily intended for type 1 diabetes, children with other forms of diabetes also will get the benefit (monogenic diabetes including neonatal forms, type 2 diabetes, etc.) once technical committee approves the application.

Functioning of the Clinic

Each Mittayi clinic is managed by a pediatrician who has special training in treating childhood diabetes. A fulltime nurse educator is posted to look after these children, provide them the medication and other accessories needed for management. A part-time dietician is also posted in all major centers. Insulin pens, both bolus (regular/aspart/lispro) and basal (glargine), needles, glucometer, strips, lancets and urine ketone strips are procured through Kerala Medical Service Corporation Limited (KMSCL) and distributed to the beneficiaries via Mittayi clinics. In children with frequent fluctuations in blood glucose levels, continuous glucose monitoring devices are being applied. Insulin pumps were given to 9 children so far. Parental empowerment programs, family support programs and outreach activities like diabetic camps are also being conducted regularly. During COVID-19 outbreak, medicines and accessories were supplied at the residences of the beneficiaries.

The functioning of Mittayi clinic is from 9.00 AM to 4.00 PM. The medical officer will examine the enrolled child, decide the treatment and the drug dose. Diabetic outpatient clinic is conducted once in a week. The nurse educator will see the patients on every day and advise regarding drug administration, dosage changes, go through blood glucose readings and logs, dispense the drugs and inform the medical officer as and when required. The part-time dietician will give the advice regarding diet modifications based on the child's diet chart, once in a week. Insulin supplies, glucometer strips and other accessories will be provided on monthly basis.

Criteria for Selection of Patients for Continuous Subcutaneous Insulin Infusion

1. Child should have registered in Mittayi scheme
2. Patient is having symptoms like severe hypoglycemia including nocturnal hypoglycemia, activity induced hypoglycemia or hypoglycemia unawareness
3. Patient is having recurrent diabetic ketoacidosis while on proper basal bolus treatment
4. Patient is having severe glycemic variability which cannot be corrected with multiple injections
5. Child/caregiver should have good knowledge regarding insulin dose adjustments based on glucose readings and food intake
6. Should know the dose and diet adjustments based on CGM reading
7. There should be a second CGM applied after carrying out the corrections suggested by the first CGM

8. Child/caregiver should have undergone the educational training regarding the insulin pump technology, food pattern, insulin dose modification based on carbohydrate counting and insulin sensitivity factor

Capacity Building for Doctors, Nurses, Dieticians, Parents And School Teachers

All the medical officers, nurse educators and dieticians are trained by experienced pediatric endocrinologists at the state level workshops. During COVID-19 pandemic also, the workshops continued, in online platforms. Education programs are being conducted for parents and school teachers (where the child is studying), to empower them to help the child in taking medications and identifying and managing acute complications sufficiently early.

Budget Allocation

The budget allocation for the scheme since its initiation and for current year are provided in Table 1.

Table 1. Budget allocation under Mittayi program

Year	Budget
2017–18	2 crore
2018–19	3 crore
2019–20	3.8 crore
2020–21	3.8 crore
2021–22	3.8 crore + Extra 50 lakhs for CSII

The cost for one patient for a year-long insulin therapy (including insulin analog cartridges, glucometer strips and other accessories comes around Rs. 14000/- per year.

CONCLUSION

Childhood diabetes cannot be managed by just prescribing insulin. This is a chronic illness with frequent complications, both acute and chronic. The psychological effects are tremendous, both to the child and care givers. Financial burden is massive, beyond the reach of most families and caregivers. Hence a government-sponsored comprehensive program taking care of financial, social and psychological aspects of the disease management is a must for the optimal care of these children.

REFERENCES

1. Libman I, Haynes A, Lyons S, Pradeep P, Rwagasor E, Tung JY, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Definition, epidemiology, and classification of diabetes in children and adolescents. Pediatr Diabetes 2022: In press.
2. Lawrence JM, Divers J, Isom S, Saydah S, Imperatore G, Pihoker C, et al. Trends in prevalence of type 1 and Type 2 diabetes in children and adolescents in US, 2001-2017. JAMA 2021;326:717-27.
3. Das AK. Type 1 diabetes in India: Overall insights . Indian J Endocrinol Metab 2015;19(Suppl 1):S31-S33
4. Kerala Social Security Mission. Proposal for Working Group 2021-2022. A Comprehensive Social Support Project for Children and Adolescents with Type 1 Diabetes Mellitus.

Type 2 Diabetes and Monogenic Diabetes of the Young – Challenges and Approach to Precision Medicine in Childhood Diabetes

SANTHOSH S OLETY
Consultant Paediatric and Adolescent Endocrinologist
Karnataka Institute of Endocrinology and Research
Bengaluru, Karnataka

INTRODUCTION

Diabetes mellitus is the most common metabolic endocrine disorder in children. Ancient Indian medical scholars recognized it as 'madhumeha' (honey urine) as it attracted ants. Among children and adolescents, type 1 diabetes (T1D) is more common; the incidence of type 2 diabetes (T2D) is also on the rise usually above 10 years of age, at around puberty. In addition, less common types of diabetes such as monogenic diabetes syndromes of childhood which account for 2–5% of diabetes need to be accurately identified to initiate the most appropriate treatment. Depending on the type of diabetes, management needs a comprehensive approach with insulin and/or oral antidiabetic drugs, physical exercise, meal planning, and psychological support to the child and the family.

ETIOLOGICAL CLASSIFICATION OF DIABETES

Diabetes mellitus in children can be classified into various types as follows:

1. ***Type 1 diabetes*** – this is due to β-cell destruction, usually leading to absolute insulin deficiency. This may be immune-mediated or idiopathic.
2. ***Type 2 diabetes*** – this is due to insulin resistance with relative insulin deficiency. Destruction of β-cells leading to impaired insulin secretion occurs at variable rates and is progressive throughout.
3. ***Monogenic diabetes:*** The common forms are maturity onset diabetes of young (MODY) and neonatal diabetes mellitus (NDM).
4. ***Other specific types:*** These include the genetic defects in insulin action, diseases of the exocrine pancreas, immune-mediated diabetes, drug or chemical-induced diabetes, diabetes secondary to infections and endocrinopathies.

In recent years, there has been a significant rise in obesity and overweight among Indian children. An estimated combined prevalence of 19.3 percent of childhood overweight and obesity from the data after 2010 showed a significant increase from the earlier prevalence of 16.3 percent reported in 2001–2005 [1]. This has resulted in many T1D children presenting with pre-existing obesity. Indians are inheritably at high risk for insulin resistance which is further compounded by a high proportion of visceral fat, presenting as T2D with normal body mass index. There are no single clinical criteria or a single investigation to distinguish T1D from T2D or other types, and this needs a multi-facetted approach. Hence it is important to distinguish amongst T1D, T2D, and monogenic forms of the disorder and appreciate the diagnostic,

therapeutic, and prognostic significance of establishing the correct type of diabetes (Table 1). Here we focus on the approach and management of T2D and MODY, both of which are commonly prone to be misdiagnosed as T1D.

TYPE 2 DIABETES

T2D is a multifactorial disorder. It is characterized by insulin sensitivity defects and insulin secretion defects which tend to coexist and both are important phenomena in the physiopathology of the disease. They are genetically determined directly and modulated by acquired factors. More than 90% of children have a family history of diabetes in the first or second-degree relatives. The risk of T2D is 30–40% with one parent with diabetes, which increases by 70% when both parents are diabetic. T2D is usually diagnosed above 10 years of age, at around puberty. School screening of obese and overweight children between 5–18 years in Delhi showed a prevalence of 1.3% of T2D and impaired glucose tolerance (IGT) of 18–24.8% [2]. In the US and the European Union, adolescent T2D makes up around 10–40% of diabetes. Indian studies have shown that T2D accounted for 8% of cases of diabetes in children below 18 years of age [3].

In the last decade, it has become a global epidemic due to the increasing prevalence of overweight and obesity resulting from easy access to calorie-rich foods, sub-optimal physical activity, and more screen time. This causes an increase in visceral adiposity which plays a higher role in insulin resistance than subcutaneous fat. Insulin resistance leads to impaired glucose utilization, especially in the skeletal tissue and unopposed hepatic glucose production with compensatory hyperinsulinemia. Adipocytokines formed due to raised fatty acid levels and an increase in oxidative stress lead to inflammation and β-cell

Table 1. Clinical characteristics of T1D, T2D and MODY

Clinical features	T1D	T2D	MODY
Age of onset (yr)	Any age after 6 months; childhood and early adolescence	Usually pubertal or adulthood	Usually presents before 25–35 yr, except GCK-MODY and NDM
Family history of diabetes	Usually sporadic (>85 %)	Strongly positive (75 to 90%)	Multigenerational (>2 generations)
Weight	Usually thin, but overweight and obesity at diagnosis not uncommon	>90% at least overweight	Same as population frequency
Markers of insulin resistance	Unusual	Common	Unusual
C-peptide levels	Low or undetectable, particularly after 2 to 3 years of diagnosis	Normal or high	Usually lower than normal
Islet autoantibodies	Usually present	Usually absent	Usually absent
Insulin-dependence	Yes	No	No
Risk of DKA	High	Low	Low
Lipids	Normal	HDL low, TG high	Normal

Abbreviations: GCK – Glucokinase, MODY – Maturity onset diabetes of the young, NDM – neonatal diabetes mellitus, DKA – Diabetic ketoacidosis, HDL – high density lipoprotein-cholesterol, TG – triglycerides

loss. T2D should be suspected in children and adolescents who are obese, with evidence of insulin resistance, strong family history of T2D, and high C-peptide levels.

T2D is usually associated with features of metabolic syndrome such as hypertension, hyperlipidemia, acanthosis nigricans, fatty liver disease, and polycystic ovary syndrome (PCOS). Initially the majority of them respond to oral antidiabetic medications but later need insulin treatment because of the progressive reduction of insulin secretory nature of the condition. Furthermore, evidence suggests that early onset of T2D may be associated with more aggressive development of microvascular and macrovascular complications than T2D appearing at later ages.

MONOGENIC DIABETES

Monogenic diabetes is defined as diabetes that occurs due to a single gene mutation affecting β-cell function. These mutations involve genes coding for transcription factors involved in β-cell development, differentiation, insulin synthesis, processing, secretion, and survival; as well as enzymes like glucokinase (Fig 1). Two clinically recognizable types are neonatal diabetes (NDM) before infancy and maturity-onset diabetes of the young (MODY) commonly seen in adolescents and early adulthood.

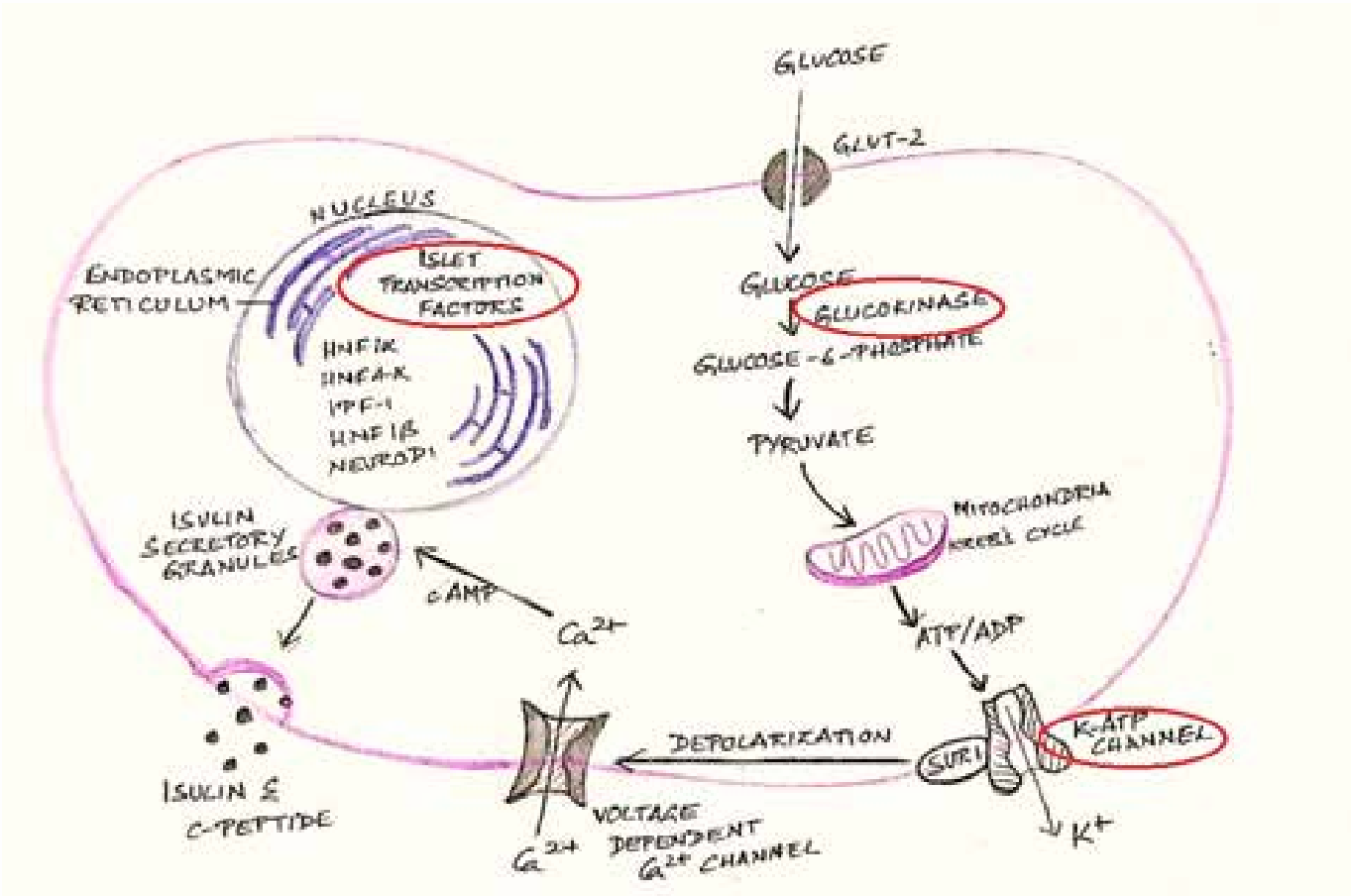

Figure 1: Physiology of Insulin formation, storage and secretion#

*Note: *highlighted genes are involved in MODY*

#Source: Reproduced with permission from: Santhosh Olety and Shruthi Appaji. Diabetes Mellitus, Figure 1. Practical Pediatric Endocrinology, 2021, page 380, copyright with Indian Journal of Pediatrics

MATURITY ONSET DIABETES OF THE YOUNG

MODY is a clinically heterogeneous disorder that occurs as a result of impaired insulin secretion, with minimal or no defects in insulin action and is characterized by hyperglycemia, nonketotic disease, the autosomal dominant mode of inheritance, age at onset less than 25 years, and lack of auto-antibodies [4]. It accounts for 2–5% of all cases of diabetes in people aged 20 years and younger. At least 14 different genes have been reported to cause diabetes with a MODY-like phenotype. The most common forms are due to mutations in hepatocyte nuclear factor (HNF)-1A (known as HNF1-MODY3), GCK gene (GCK-MODY2), and HNF4A (MODY1). An Indian study found MODY 3 to be the commonest (7.2%) followed by MODY 12 (ABCC8 3.3%) [5]. Although diagnosis may be made by careful clinical evaluation, the exact sub-typing is possible only by genetic analysis. The different genetic subtypes of MODY differ in their age at onset, the pattern of hyperglycemia, and response to treatment. MODY 1 and 3 initially respond well to oral sulfonylurea, while GCK-MODY does not require active treatment except in the setting of pregnancy to reduce the risk of fetal macrosomia [6,7]. The genetic findings have important clinical implications, allowing for proper genetic counseling, early diagnosis, and better care of patients in the form of precision medicine and foresee clinical courses such as renal developmental disease or renal cysts (HNF1B-MODY5).

CLINICAL PRESENTATION

The approach to hyperglycemia is detailed in Fig 2.

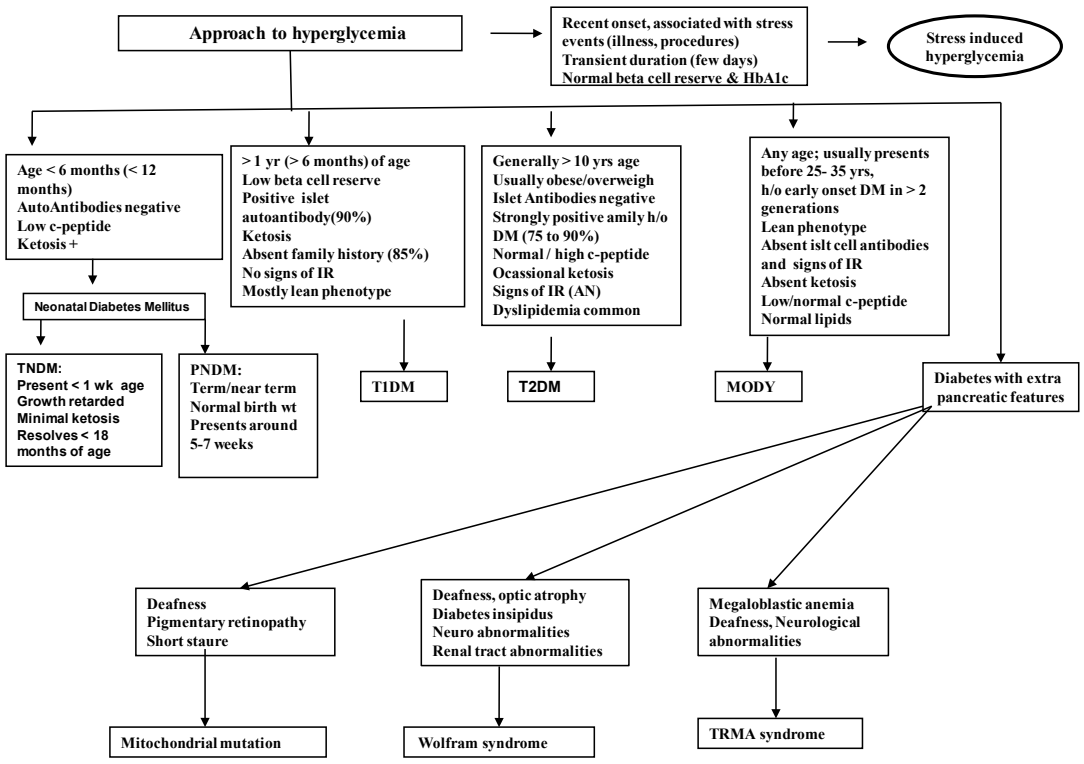

Figure 2: Approach to hyperglycemia in a child and adolescent*

Abbreviations: AN – Acanthosis nigricans, TNDM/PNDM – Transient/permanent neonatal diabetes mellitus, TRMA – Thiamine responsive megaloblastic anemia

**Source: Reproduced with permission from: Santhosh Olety and Shruthi Appaji. Diabetes Mellitus, Figure 5. Practical Pediatric Endocrinology, 2021, page 386, copyright Indian Journal of Pediatrics*

- Children present with characteristic hyperglycemia symptoms such as polyuria, polydipsia, nocturia and weight loss, which may be accompanied by polyphagia, diminished vision, and increased susceptibility to certain infections.
- Diabetic ketoacidosis (DKA) at the time of the first diagnosis is more commonly seen with T1D but occasionally can be present with T2D and not seen in monogenic diabetes.
- Rarely, the non-ketotic hyperosmolar syndrome may develop in T2D.
- T2D is diagnosed by screening for metabolic syndrome in at-risk children and adolescents with obesity, family history of T2D, early coronary-vascular disease (CVD), and markers of insulin resistance (acanthosis nigricans, hyperandrogenism and PCOS)
- Diabetes in the absence of ketosis with a family history of diabetes in two or more generations and a thin phenotype, the clinician should suspect monogenic diabetes in a child.

LABORATORY EVALUATION

- Initial simple tools used in the presence of symptoms are the measurement of blood glucose (BG) and ketones by a bedside glucometer, They guide us for emergency management.
- A formal laboratory plasma glucose measurement is always required to confirm the diagnosis.
- Oral glucose tolerance test (OGTT), is a recommended tool to diagnose prediabetes, T2D, and monogenic forms of diabetes. Children should be on a normal carbohydrate diet for 72 hours with overnight fasting for at least 8 hours. Anhydrous glucose, 1.75 g/kg (maximum of 75g) is given orally. Pre- and 2-hour post-glucose load BG values for the diagnosis are shown in Table 2. There is no role for OGTT and it should be strictly avoided in the diagnosis of T1D.
- Hemoglobin A1c (HbA1c) or glycosylated hemoglobin reflects the average blood glucose level in the past 3 months. A HbA1c level of >6.5% is suggestive of diabetes mellitus. HbA1c should always be used in adjunct to blood glucose values.
- Fasting serum C-peptide levels help to assess beta-cell reserve and levels less than 0.6 ng/mL indicate poor beta-cell reserve and point towards T1D. It is important to remember that sometimes it may be suppressed during glucose toxicity and may need to be repeated once glucotoxicity has been resolved.
- Islet cell autoantibodies aid in differentiating T1D from other types of diabetes, especially in the pubertal age group where the overlapping of diagnosis can occur.

Table 2: Diagnostic criteria for Impaired glucose tolerance and diabetes mellitus

Impaired glucose tolerance (IGT)	Diabetes mellitus
Fasting plasma glucose: 100–125 mg/dL **OR** 2-hour plasma glucose during OGTT: 140–199mg/dL **OR** HbA1c: 5.7–6.4%	Classic symptoms of diabetes with one of the following: Fasting plasma glucose: ≥126 mg/dL **OR** 2-hour plasma glucose during OGTT: ≥200 mg/dL **OR** Random plasma glucose: ≥200 mg/dL **OR** HbA1c ≥6·5%

MANAGEMENT

The aim of management is to achieve euglycemia targets (fasting plasma glucose [FPG] < 130 mg/dL, post-prandial plasma glucose [PPPG] < 180 mg/dL, and HbA1c <7 %)[8].

Lifestyle changes

For T2D, focusing on lifestyle parameters by adopting behavioral changes towards healthy

eating and recommended daily 45 to 60 minutes of moderate-intensity physical activities to achieve age and gender-appropriate BMI. These should involve a holistic approach with active participation from the entire family support.

Pharmacological intervention

For T2D, currently limited therapeutic options are available. Metformin is the first choice and most commonly used. Gastrointestinal side effects are common and self-resolve usually. Generally, start with a smaller dose (500 mg BD); once tolerated can be titrated fortnightly up to a maximum dose of 2 g per day if required. Those who initially present with HbA1c > 9.5 % or ketosis may need basal insulin-like glargine to be added (0.3 to 0.5 units/kg), dose titrated, and can be weaned gradually over a few weeks after achieving euglycemic targets. Sometimes when a diagnosis is unclear and T1D cannot be ruled out, it is acceptable to start on both metformin and insulin and refer to the specialist. Usually, over time the overlapping diabetes types would evolve and diagnosis can be narrowed down. Those who fail to respond to metformin would need the addition of basal insulin and later prandial insulin as required. The only other drug approved for T2D in children above 10 yr is an injectable GLP-1 agonist (glucagon-like peptide) but the cost is a limiting factor.

Prevention

Prevention is most important and different stake-holders need to work as a team in coordination to reduce the prevalence of obesity and overweight. Lifestyle modification programs starting in childhood are urgently needed and society needs to change its attitudes to childhood nutrition, play, and exercise. Screening and treating other co-morbid conditions as a part of metabolic syndrome is another strategy.

Managing MODY

MODY 3 and 1 respond well to sulphonylurea such as glimepiride and generally require a lesser dose (0.5 to 1 mg once a day) than adult T2D requirement. MODY 2 is usually asymptomatic and managed with medical nutritional changes and does not need any pharmacotherapy.

Monitoring

Unlike T1D, self-monitoring blood glucose (SMBG) in T2D and Monogenic diabetes is not frequently required.

SUMMARY

T2D is rising in alarming numbers. T2D has a more aggressive course in children and adolescents with early onset of long-term complications. There is no single diagnostic test and challenges are more in differentiating and diagnosing MODY. This involves careful selection of clinical features that are unusual for T1D and T2D and performing physiological tests like C-peptide and autoantibody measurement. Enormous improvement in our understanding and the knowledge of genetic etiology of diabetes has enabled appropriate treatment, better prediction of disease progression, screening of family members, and genetic counseling. It is always a challenge for the treating physician to optimize the glycemic levels in a child with diabetes.

REFERENCES

1. Ranjani H, Mehreen TS, Pradeepa R, Anjana RM, Garg R, Anand K, et al. Epidemiology of childhood overweight & obesity in India: A systematic review. Indian J Med Res 2016;143(2):160–174.
2. Kapil U , Singh P, Pathak P, Dwivedi SN, Bhasin S. Prevalence of obesity amongst affluent adolescent school children in Delhi. Indian Pediatr. 2002;39(5):449-52.
3. Bhatia V. IAP National Task Force for Childhood Prevention of Adult Diseases: Insulin Resistance and Type 2 Diabetes Mellitus in Childhood. Indian Pediatr. 2004;41:443-457.
4. Murphy R, Ellard S, Hattersley AT. Clinical implications of a molecular genetic classification of monogenic beta-cell diabetes. Nat Clin Pract Endocrinol Metab. 2008;4(4):200-13.
5. Mohan V, Radha, V, Nguyen TT, Stawiski EW, Pahuja KB, Goldstein LD, Tom J, et al. Comprehensive genomic analysis identifies pathogenic variants in maturity-onset diabetes of the young (MODY) patients in South India. BMC Med Genet. 2018;13;19(1):22
6. Chakera AJ, Steele AM, Gloyn AL, Shepherd MH, Shields B, Ellard S, et al. Recognition and Management of Individuals With Hyperglycemia Because of a Heterozygous Glucokinase Mutation. Diabetes Care. 2015;38(7):1383-92.
7. Byrne MM, Sturis J, Menzel S, Yamagata K, Fajans SS, Dronsfield MJ, et al. Altered insulin secretory responses to glucose in diabetic and nondiabetic subjects with mutations in the diabetes susceptibility gene MODY3 on Chromosome 12. Diabetes 1996:45:1503–10
8. Shah AS, Zeitler PS, Wong J, Pena AS, Wicklow B, Arslanian S, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Type 2 diabetes in children and adolescents. Pediatr Diabetes. 2022;23(7):872-902.

Metabolic Syndrome and Diabetes in Obese Children – The Time to Act

MANOJ AGARWAL, ANURAG BAJPAI
Department of Paediatric Endocrinology
Regency Center for Diabetes, Endocrinology & Research
Kanpur, Uttar Pradesh

INTRODUCTION

The dramatic increase in childhood and adolescent obesity has resulted in metabolic complications at a young age [1,2]. Central obesity and visceral fat deposition cause insulin resistance, the key determinant of metabolic syndrome (dysglycemia, hypertension, and dyslipidemia) [3].

WHAT CAUSES METABOLIC COMPLICATIONS IN OBESITY?

Visceral fat deposition is the forerunner of metabolic complications of obesity. The capacity to store fat is fixed and determined by size at birth. Excess fat above this is deposited in the visceral tissues (liver and pancreas), causing insulin resistance, leading to type 2 diabetes (T2D), dyslipidemia, polycystic ovarian syndrome (PCOS), and hypertension (Figure 1) [4]. Reduced fetal growth limits the adipocyte number and size at birth, compromising postnatal fat storage capacity. The severity of obesity (greater for higher body mass index [BMI]), fat distribution (greater for abdominal obesity), birth weight (higher for lower birth weight), and genetics (family history) determine the development of metabolic complications.

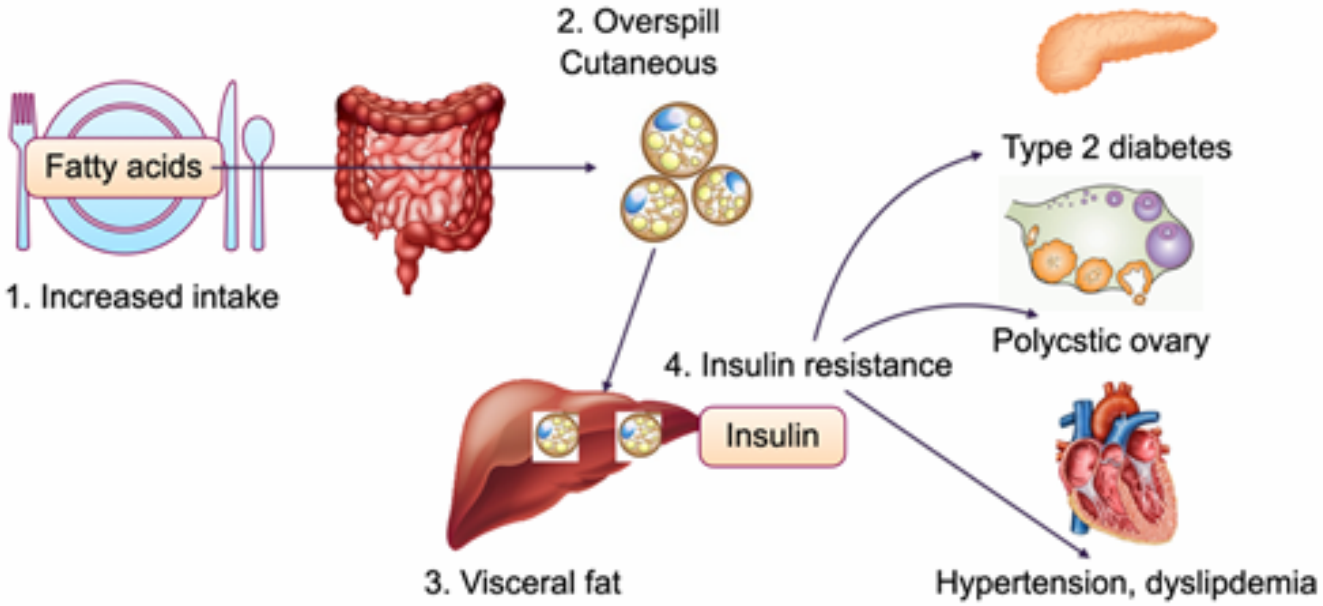

Figure 1: Pathogenesis of insulin resistance and metabolic complications of obesity*

**Adapted with permission from Advanced Pediatric Endocrinology Volume II. Eds: Bajpai A, Kanpur, 2021.*

WHAT IS THE COURSE OF METABOLIC SYNDROME IN CHILDREN AND ADOLESCENTS?

The impaired insulin effect is central to the pathogenesis of metabolic syndrome. Obesity lowers insulin sensitivity; this is, however, initially compensated by increased insulin secretion. Over time the beta-cells fail to cope with the increasing requirements leading to impaired glucose intolerance, elevated fasting blood glucose (BG), and finally, frank diabetes mellitus. The physiological insulin resistance of puberty exacerbates the development of the metabolic syndrome. The likelihood of remission of dysglycemia dramatically decreases after puberty making it the ideal window of opportunity for preventive and therapeutic interventions [5]. T2D in adolescents has a more aggressive course than in adults, with an annual loss of 10% of beta-cell reserve [6].

HOW TO ASSESS PEDIATRIC AND ADOLESCENT OBESITY?

The key components of assessing an obese child include its confirmation, exclusion of pathological causes, and identification of metabolic complications.

Is the child obese?

BMI is the most widely used marker of obesity, with tools for fat assessment reserved for research. BMI above 27 kg/m^2 adult equivalent for age and gender as per the IAP 2015 criteria suggests obesity [7].

Is there a pathological cause?

Most obese children do not have an underlying cause limiting the role of etiological work-up. Normal growth, development, and puberty suggest a physiological cause, while early onset, hyperphagia, as well as delayed growth, development, and puberty suggest a pathological cause [8].

WHAT ARE THE METABOLIC COMPLICATIONS?

The metabolic screening for obesity complications includes assessment for dysglycemia, fatty liver, hypertension, and dyslipidemia (Table 1). The tests, if normal, should be repeated after one year in obese and three years in overweight individuals.

Table 1: Complication assessment for obese children and adolescents [9]

Investigation	Level of concern	Pathological level
Blood glucose, fasting	100–125 mg/dL	> 126 mg/dL
Blood glucose, 2 hour after glucose	140–199 mg/dL	> 200 mg/dL
Hemoglobin A1c (HbA1c)	5.7–6.4%	> 6.5%
Total cholesterol	170–199 mg/dL	> 200 mg/dL
Low-density lipoprotein (LDL) cholesterol	90–129 mg/dL	> 130 mg/dL
Triglyceride	90–129 mg/dL	> 130 mg/dL
High-density lipoprotein (HDL) cholesterol	40–45 mg/dL	< 40 mg/dL
Alanine transaminase (ALT) or serum glutamic-pyruvate transaminase (SGPT)	> 25 IU/L (boys) > 22 IU/L (girls)	> 60 IU/L

Dysglycemia

Glucose abnormalities are common in obesity (pre-diabetes in 20% and T2D in 5%); most do not have clinical features. This emphasizes the need for screening.

How to screen for dysglycemia?

The options include fasting blood glucose (FBG), glucose tolerance test (OGTT), and HbA1c levels. Sample for BG is drawn in a fasting state and two hours after a glucose load of 1.75 g/kg (up to 75 g). HbA1c should be done using assays validated against the DCCT NGSP standard. Insulin levels have a limited role as they represent systemic levels, while the portal system is the leading site of insulin action.

Is it diabetes?

The American Diabetes Association (ADA) criteria for diagnosing diabetes in children and adolescents are the same as for adults [10]. Fasting BG levels have high specificity, while post-glucose levels are highly sensitive to diagnosing diabetes. HbA1c levels should be interpreted cautiously as their correlation with glycemic status is lower in children and adolescents.

Is it type 1 diabetes?

The proportion of obese children and adolescents with type 1 diabetes (T1D) with a family history of diabetes has increased dramatically, causing diagnostic confusion. Labeling these individuals as T2D may prompt insulin discontinuation predisposing to diabetic ketoacidosis (DKA). C-peptide levels are low in both T1D and T2D at diagnosis. The best approach to manage these individuals is to initiate insulin and obtain a glutamic acid decarboxylase (GAD) antibody test. T2D should be diagnosed only if the GAD antibody test is negative [11].

Dyslipidemia

Lipid assessment involves the estimation of total HDL, LDL, non-HDL cholesterol, and triglyceride levels [12].

Nonalcoholic fatty liver disease (NAFLD)

NAFLD should be screened with ALT levels. The optimal cut-offs predicting hepatic fibrosis are unclear; levels twice the upper limit of normal are a cause of concern [13]. This should be followed by hepatic ultrasound and work-up for other causes of chronic liver disease.

Hypertension

Blood pressure should be measured at each visit and compared to age and gender-specific norms (Table 2).

Table 2. Criteria for elevated blood pressure in children and adolescents

Category	Less than 13 years	Above 13 years
Normal	Less than 90th percentile	Less than 120/80 mm Hg
High	90-95th percetile	120-129/< 80 mm Hg
Stage I	> 95th- < 95th percetile + 12 mm Hg	130-139/80-89 mm Hg
Stage II	Above 95th percentile + 12 mm Hg	Above 140/90 mm Hg

MANAGEMENT OF ADOLESCENTS

The management goals include weight reduction and prevention and reversal of complications.

What should be the weight loss target?

The aim is to bring the weight into the normal range with a 7–10% reduction in BMI SDS (standard deviation score) over six months. Weight loss should be gradual (between 0.5–1 kg per month) in growing children and faster after completion of growth (up to 2 kg/month) [9].

What nutritional measures should be advised?

Critical interventions include regular meals, including breakfast, avoidance of screen exposure while eating, and reduced sugar-containing drinks. Rigorous dieting should be avoided. Family meals should be advised to avoid eating out and screen watching during eating.

What should be the level of physical activity?

Regular physical activity for at least 45 minutes daily is recommended. Combined screen time, including television, tablet, laptop, and mobile, should be limited to less than one hour daily.

What is the role of pharmacotherapy and surgery for obesity?

There is a limited role of medical and surgical management of obesity in children and adolescents. The gastric lipase inhibitor, orlistat is approved for use in children above 12 years but it causes only modest weight loss. Bariatric surgery should be reserved for morbid obesity (BMI above 35 kg/m2 with comorbidity) after completion of linear growth [9].

What is the specific treatment option?

The treatment options depend on the metabolic complications.

Dysglycemia

Currently approved drugs for management of dysglycemia in adolescents with T2D include insulin, metformin, and glucagon-like peptide-1 (GLP-1) receptor analog (liraglutide) (Clinical Practice Recommendations of International Society for Pediatric and Adolescent Diabetes [ISPAD]) [110. Studies on dipeptidyl peptidase IV inhibitors (DPP4 or gliptins) and sodium-glucose cotransporter-2 (SGLT2) inhibitors are ongoing.

Metformin. Metformin is the drug of choice and should be used unless there are contraindications. It is started at a dose of 500 mg daily with a build-up to a maximum dose of 2000 mg. Gastrointestinal symptoms (abdominal bloating, nausea, and vomiting) are common but improve over time.

Insulin. Insulin is reserved for children and adolescents with high BG levels (HbA1c above 8.5%) or osmotic symptoms. A basal-bolus regimen is recommended after stabilizing hyperglycemic emergency, while basal insulin (glargine or degludec at 0.3 unit/kg/day once at night) with metformin may be considered without ketoacidosis. Insulin may be required with the deterioration of beta cell functions on follow-up.

GLP-1 receptor analog. GLP-1 receptor analogs are helpful in obese individuals. They lower BG by delaying gastric emptying, inducing early satiety, increasing insulin and inhibiting glucagon release; and allowing discontinuation of insulin in most cases. The FDA has approved liraglutide in adolescents between 16–18 years with T2D. The side effects include abdominal bloating, nausea, and anorexia. Weekly dulaglutide, exenatide, and semaglutide are not approved in adolescents.

How to decide on treatment? A basal-bolus regimen should be initiated after hyperglycemic crises with the addition of metformin after the resolution of acidosis (Figure 2). Basal insulin with metformin is given with an HbA1c above 8.5% or osmotic symptoms. Prandial insulin should be added with persistent hyperglycemia despite a 1.5 units/kg/day basal insulin dose. Individuals with no osmotic symptoms and an HbA1c below 8.5% should be started on metformin. HbA1c above the target range despite the maximum metformin dose (2000 mg daily) should prompt the addition of basal insulin or GLP1 analog [11].

Dyslipidemia

Non-HDL cholesterol levels below 120 mg/dL are normal, while those between 120–144 mg/dL suggest the need for lifestyle measures (Figure 3). Fasting LDL cholesterol levels are estimated with non-HDL cholesterol levels above 145 mg/dL at two and 12 weeks. The mean of the two levels guides the subsequent management [12]. Levels below 110 mg/dL need follow-up, while a repeat sample after lifestyle modification is suggested with levels between 110 and 129 mg/dL. Lifestyle measures, evaluation for secondary causes, and treatment with atorvastatin (10 mg daily) are indicated with levels persistently above 130 mg/dL.

NAFLD

Weight loss is the mainstay of management with the experimental role of metformin, orlistat, and ursodeoxycholic acid.

Hypertension

Lifestyle measures (low salt and physical exercise) are advised for elevated blood pressure and stage 1 hypertension. ACE inhibitors (ramipril, 2.5–5 mg; perindopril, 2–4 mg) should be started with the failure of lifestyle measures or stage 2 hypertension. Angiotensin receptor blockers (losartan 50–100 mg; Olmesartan 20–40 mg), thiazide, and calcium channel blockers may be considered with no response or adverse effects on ACE inhibitors [14].

SUMMARY

Childhood obesity poses a substantial public health risk. Given the lack of specific therapeutic options and the rapid progression of metabolic complications, preventive strategies to reduce the development of childhood and adolescent obesity are urgently required.

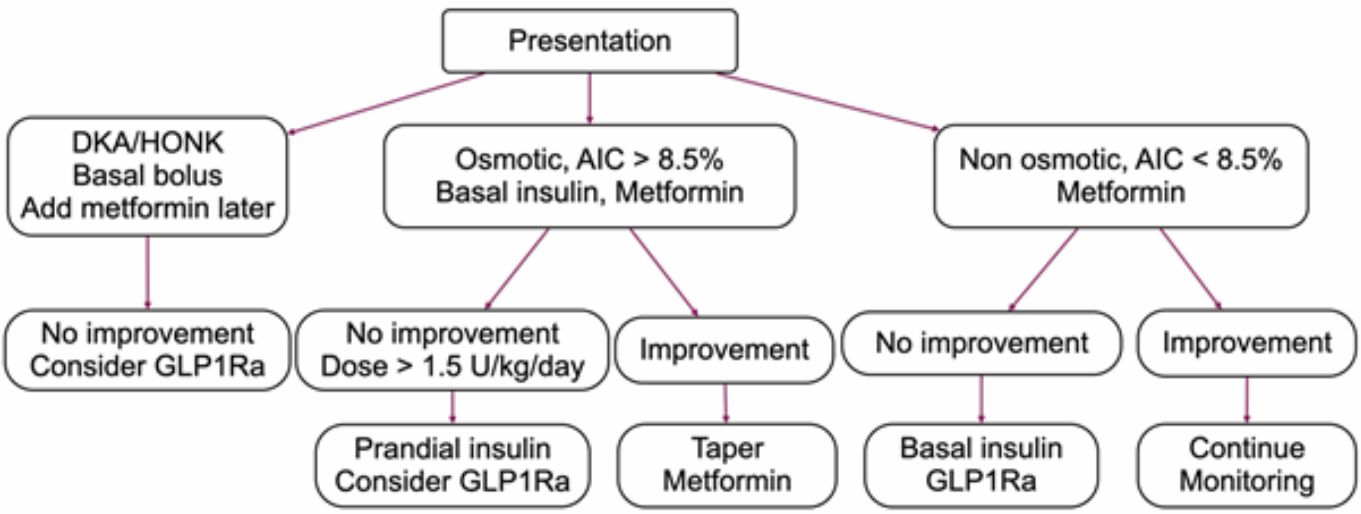

Figure 2: Approach to the management of Type 2 diabetes in children and adolescents

Abbreviations: DKA – Diabetic ketoacidosis, HONK – Hyperglycemia hyperosmolar nonketotic diabetes, A1C – Hemoglobin A1c, GLP1Ra – Glucagon like peptide-1 receptor analog,

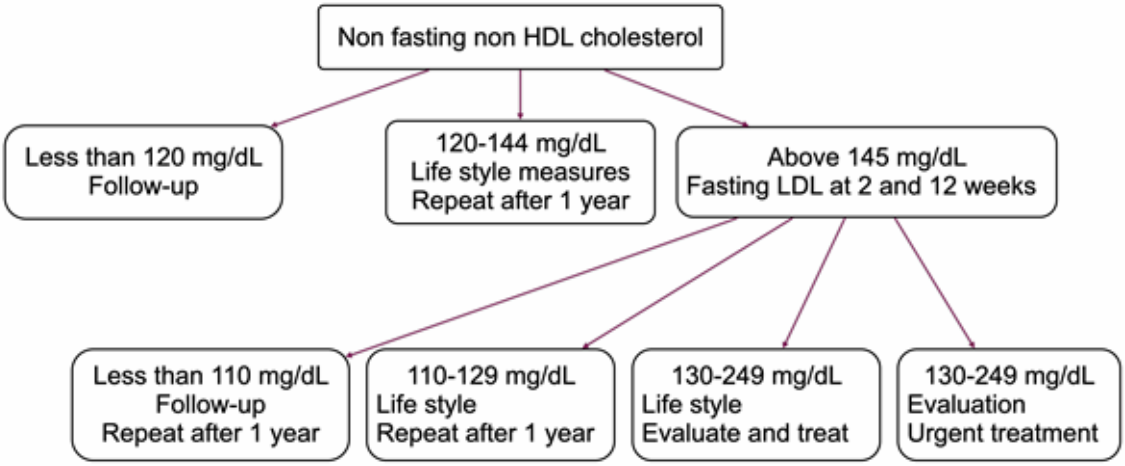

Figure 3: Approach to the management of dyslipidemia in children and adolescents

Abbreviations: HDL – High density lipoprotein cholesterol, LDL – Low density lipoprotein cholesterol

REFERENCES

1. Dave C, Agarwal N, Patel R, Shukla R, Bajpai A. Determinants and Predictors of Body Fat in Indian Children and Adolescents. Indian J Pediatr. 2022;89(5):473-476.
2. Dave C, Agarwal N, Patel R, Shukla R, Bajpai A. Predictors of Metabolic Complications in Obese Indian Children and Adolescents. Indian J Pediatr. 2021;88(3):252-256.
3. Bajpai A, Narayanan R. Type 2 Diabetes. Bajpai A, Ed. Advanced Pediatric Endocrinology Volume II, Kanpur, First Edition 2021, pp. 300-311.
4. Dave C, Bajpai A. Obesity. Bajpai A, Ed. Basic Pediatric Endocrinology II, Kanpur, First Edition 2018, pp. 65-73.
5. Ighbariya A, Weiss R. Insulin Resistance, Prediabetes, Metabolic Syndrome: What Should Every Pediatrician Know? J Clin Res Pediatr Endocrinol. 2017;9(Suppl 2):49-57.
6. TODAY Study Group, Zeitler P, Hirst K, Pyle L, Linder B, Copeland K, Arslanian S, et al. A clinical trial to maintain glycemic control in youth with type 2 diabetes. N Engl J Med. 2012;366(24):2247-56.
7. Dave C, Agarwal N, Patel R, Shukla R, Bajpai A. Comparison of the Predictive Value of IAP 2015 and WHO Body Mass Index Criteria for Hypertension in Indian Children and Adolescents. Indian J Pediatr. 2022;89(12):1175-1179.
8. Bajpai A, Shenoy M, Karia P. Childhood Obesity. IAP Standard Treatment Guidelines, https://iapindia.org/pdf/Ch-026-Childhood-Obesity.pdf, Accessed November 2022.
9. Styne DM, Arslanian SA, Connor EL, Farooqi IS, Murad MH, Siverstein JH, et al. Pediatric Obesity-Assessment, Treatment, and Prevention: An Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2017;102(3):709-757.
10. American Diabetes Association Professional Practice Committee. 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes-2022. Diabetes Care. 2022;45(Suppl 1):S17-S38.
11. Shah AS, Zeitler PS, Wong J, Pena AS, Wicklow B, Arslanian S, et al. ISPAD Clinical Practice Consensus Guidelines 2022: Type 2 diabetes in children and adolescents. Pediatr Diabetes. 2022;23(7):872-902.
12. Bamba V. Update on screening, etiology, and treatment of dyslipidemia in children. J Clin Endocrinol Metab. 2014;99(9):3093-102.
13. Vos MB, Abrams SH, Barlow SE, Caprio S, Daniels SR, Kophli R, et al. NASPGHAN Clinical Practice Guideline for the Diagnosis and Treatment of Nonalcoholic Fatty Liver Disease in Children: Recommendations from the Expert Committee on NAFLD (ECON) and the North American Society of Pediatric Gastroenterology, Hepatology and Nutrition (NASPGHAN). J Pediatr Gastroenterol Nutr. 2017;64(2):319-334.
14. Flynn JT, Kaelber DC, Baker-Smith CM, Blowey D, Carroll AE, Daniels SR, et al.; Subcommittee on Screening and Management of High Blood Pressure in Children. Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children and Adolescents. Pediatrics. 2017;140(3):e20171904.

Neonatal Diabetes Mellitus

PREETI SINGH[1], DOLLY MADAN[2]
[1]Associate Professor and [2]Senior Resident, Department of Pediatrics
Lady Hardinge Medical College and Kalawati Saran Children's Hospital
New Delhi

INTRODUCTION

Hyperglycemia in the newborn period is not uncommon, especially in premature and low birth weight/intrauterine growth restriction (IUGR) neonates. The risk factors for hyperglycemia include parenteral glucose administration, the stress response to critical illness, sepsis, and drugs such as phenytoin, glucocorticoids, and beta-adrenergic agents. It is identified in an admitted newborn on routine testing in the first 3–5 days of life that usually resolves within 2–3 days of onset but can persist up to 10 days.

Neonatal diabetes mellitus (NDM) is a rare metabolic disorder characterized by persistent hyperglycemia with onset usually before six months and rarely up to 1 year of age. It is caused by a mutation in a single gene affecting the development and function of pancreatic beta-cells resulting in impaired insulin secretion and/or function [1,2]. Based on the duration of insulin dependency, NDM can be sub-grouped into the transient (TNDM) and permanent (PNDM) forms of NDM. The syndromic forms of NDM have PNDM with extrapancreatic features at presentation or develop later with time.

EPIDEMIOLOGY

The reported incidence of NDM ranges from 1 in 90,000 to 160,000 live births [3,4]. Advances in molecular genetics over the past decade have led to the identification of over 30 known genetic mutations responsible for NDM. In a large international cohort of 1020 infants clinically diagnosed with NDM before 6 months of age, a genetic cause was recognized in 80% of them [1]. Mutations in the genes *(KCNJ11* and *ABCC8*) encoding ATP- sensitive potassium (KATP) channel were the most common cause (38.2%) of NDM, while mutations in the *INS* gene were reported in 10% of patients. Wolcott-Rallison syndrome due to homozygous mutation in the *EIF2AK3* gene was the commonest genetic cause in consanguineous families (24%). An early genetic diagnosis is essential as it shall predict the clinical course and prognosis and guide appropriate management.

PATHOGENESIS

The deficit in insulin production in NDM can arise either from delayed maturation of pancreatic islets and beta-cells or beta-cell dysfunction that impairs insulin secretion. To understand its pathogenesis, it is essential to have an insight into the normal pancreatic beta-cell physiology of insulin secretion. Glucose is transported into the beta-cell by insulin-independent glucose transporter (GLUT2); it undergoes phosphorylation by glucokinase and gets metabolized. The resultant rise in ATP/ADP ratio causes the closure of the KATP channel. This initiates a cascade of events characterized by decreased potassium efflux, membrane depolarization, calcium influx, and release of insulin from storage granules (Figure 1). Besides, leucine can also stimulate insulin secretion by allosterically activating glutamate

dehydrogenase (GDH) and increasing glutamate oxidation; this increases the ATP/ADP ratio and hence the closure of the KATP channel.

The KATP channel comprises four outer SUR1 subunits (encoded by the *ABCC8* gene) surrounding the inner four pore-forming Kir6.2 subunits (encoded by the *KCNJ11* gene). Activating mutations of *KCNJ11* or *ABCC8* reduces the sensitivity of the KATP channel to ATP. This results in persistent opening of the KATP channel and inhibition of the cascade of events involved in insulin secretion, leading to NDM. Almost 90% of children with *KCNJ11* mutations have PNDM and the remaining 10% develop TNDM. On the contrary, mutations in the *ABCC8* gene are more commonly (66%) associated with TNDM.

Mutations in genes *INS, GCK, SLC2A2, SLC19A2,* and *RFX6* are also related to abnormal beta-cell function. Other mechanisms associated with impaired insulin secretion are beta-cell destruction due to cellular stress or autoimmunity (*INS, EIF2AK3, IER3IPI, FOXP3, WFS1, LRBA, STAT1,* and *STAT3*); and abnormal pancreatic development (pancreatic aplasia or hypoplasia) (*PDX1, PTF1A, HNF1B, RFX6, GATA4, GATA6, CNOT1, GLIS3, NEUROG3, NEUROD1, PAX6, NKX2-2,* and *MNX1*) [5,6].

CLINICAL FEATURES

Infants with NDM usually present within the first 6 months of life, while a few may come to clinical attention between 6 and 12 months. They present with hyperglycemia, polyuria,

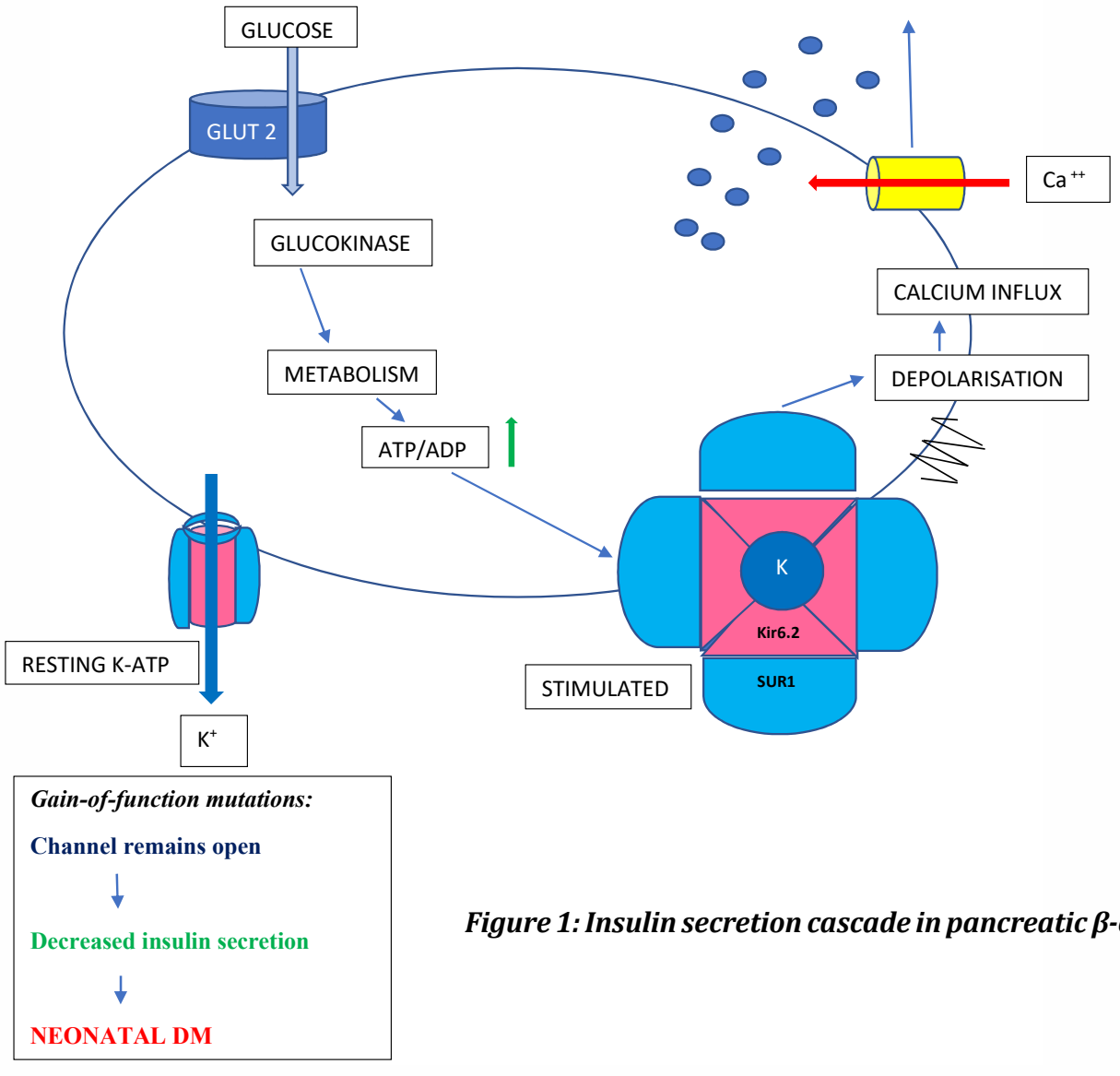

Figure 1: Insulin secretion cascade in pancreatic β-cells

polydipsia, poor postnatal growth, and sometimes diabetic ketoacidosis (DKA) precipitated by intercurrent infections. The frequency of DKA at presentation varies depending on the underlying specific genetic disorder (e.g. TNDM due to overexpression of 6q24 doesn't present with DKA) [7]. Around 50% of cases of NDM are transient forms (TNDM) that are clinically indistinguishable from the permanent variety (PNDM) at the time of diagnosis. There are no clinical cues to predict whether NDM will have a transient or a permanent course. The hyperglycemia of TNDM results from reduced or absent insulin production during the fetal period that extends for a variable time into postnatal life. Therefore, infants with TNDM are born SGA and show features of IUGR due to prenatal insulin deficiency. Two-thirds of transient forms of NDM are caused by abnormalities in an imprinted region on chromosome 6q24, while the rest have mutations in KATP channels. The dependency on insulin in the transient form of NDM usually resolves spontaneously by 12–14 weeks (up to 18 months) [8], but it may relapse during periods of metabolic stress like puberty and pregnancy.

In contrast, infants with PNDM require lifelong treatment, either insulin or sulphonylureas. Up to 50% of cases with PNDM are associated with a mutation in the gene encoding the KATP channel, while 15–20% have mutations in the *INS* gene [8]. KATP channels are also found in the brain and muscles. *KCNJ11* mutations can be associated with severe developmental delay, epilepsy, muscle weakness, and dysmorphic features (DEND syndrome). In some cases, chronic malabsorptive diarrhea due to exocrine pancreatic insufficiency (associated with mutations of *GATA6, EIF2AK3*, and *PTF1A*) is observed with PNDM.

The syndromic forms of NDM are associated with extrapancreatic features at initial presentation or evolve later in life. Wolcott-Rallison syndrome, an autosomal recessive disorder, is the most common syndrome presenting with PNDM. It is caused by mutations in *EIF2A*, which encodes the translation initiation factor 2-alpha kinase 3 that regulates the endoplasmic reticulum. Rogers syndrome is an autosomal recessive disorder with PNDM (mutation of the *SLC19A2* gene), characterized by thiamine-responsive megaloblastic anemia and sensorineural hearing loss. IPEX syndrome (immune dysregulation, polyendocrinopathy, enteropathy, and X-linked syndrome), a rare X-linked disorder, is caused by mutations in the gene that encodes the transcription factor FOXP3. Common genetic forms of transient and permanent NDM, along with their inheritance and characteristic phenotypic features, are given in Table 1 [6,8].

DIAGNOSIS

It is often challenging to diagnose NDM in infants <6 months of age and a high index of suspicion is needed. A clinical diagnosis of NDM is made for infants with persistent hyperglycemia (blood glucose levels >200 mg/dL) without an alternative cause for elevated blood glucose levels.

A thorough evaluation using a good history and examination supported by appropriate laboratory investigations is needed to differentiate NDM from neonatal hyperglycemia and autoimmune type 1 diabetes. Identify risk factors for hyperglycemia like parenteral glucose administration, especially in preterm and low birth weight or IUGR infants, sepsis, stress during critical illness, and drugs like corticosteroids and beta-adrenergic agents.

Laboratory assessment of blood glucose, urine/blood ketones, C-peptide, and/or insulin is performed for a suspected case of NDM. Ultrasound of the abdomen may help identify the structural defects of the pancreas, as pancreatic hypoplasia or agenesis is associated with specific genetic mutations. Test for autoantibodies (glutamic acid decarboxylase, islet cell, insulin, zinc transporter, and tyrosine phosphatases) is helpful in infants between 6 months and 12 months of age. Most children with type 1 diabetes

Table 1: Genetic and phenotypic profile of transient and permanent NDM

Transient Neonatal Diabetes mellitus (TNDM)

Genes	Locus	Inheritance	Clinical features
PLAGL1, HYMAI *ZFP57*	6q24 6p22.1	Variable (imprinting defect)	TNDM ± macroglossia ± umbilical hernia, cardiac and brain malformations Multiple hypomethylation syndrome- macroglossia, developmental delay, congenital heart defects
KCNJ11	11p15.1	Sporadic/ AD	Activating mutations of *KCNJ11/ABCC8* that encode subunits of the K_{ATP} channel, can lead to both TNDM or PNDM
ABCC8	11p15.1	Sporadic/AD/AR	
HNF1B	17q21.3	AD	*TNDM + pancreatic* hypoplasia, renal cyst

Permanent Neonatal Diabetes mellitus (PNDM)

Genes	Locus	Inheritance	Clinical features
KCNJ11	11p15.1	Spontaneous/ AD	PNDM ± DEND
ABCC8	11p15.1	Spontaneous/ AD/ AR	PNDM ± DEND (rare)
INS	11p15.5	AR	Isolated PNDM
GCK	7p15-p13	AR	Isolated PNDM, parents can have fasting hyperglycemia
EIF2AK3	2p11.2	AR	Wolcott-Rallison syndrome: PNDM + skeletal dysplasia + recurrent liver &/or renal dysfunction
SLC19A2	1q23.3	AR	Roger's syndrome: PNDM + thiamine responsive mega-loblastic anemia (TRMA), sensorineural hearing loss
FOXP3	Xp11.23-p13.3	X linked recessive	Immune dysregulation, polyendocrinopathy, exfoliative dermatitis, enteropathy, and X-linked (IPEX) syndrome
IPF-1/PDX-1	13q12.1	AR	Pancreatic agenesis, cerebellar hypoplasia, cardiac septal defects, absent gall bladder
PTF1A	10p12.2	AR	Pancreatic agenesis, cerebellar hypoplasia, central respiratory dysfunction

DEND: Developmental delay, epilepsy and neonatal diabetes

will be positive for at least one autoantibody, though its absence cannot completely rule out the possibility of T1D. HbA1c values are variable and less predictive in diagnosing diabetes in infants <6 months of age. Therefore, genetic testing should be strongly considered for infants with persistent hyperglycemia to confirm the diagnosis of NDM. Further, treatment and prognosis for monogenic forms of NDM are influenced to a significant extent by the specific gene defect.

Genetic testing

Comprehensive next-generation sequencing (NGS) is the best approach for early molecular genetic diagnosis. It must be performed for all infants with NDM presenting before 6 months and those 6–12 months who are negative for islet cell antibodies or extra-pancreatic features such as gastrointestinal anomalies or congenital defects, unusual family history, or even the development of multiple autoimmune disorders

at a young age [9]. Genetic diagnosis shall guide specific treatment and facilitate prognostication.

MANAGEMENT

The management of neonatal hyperglycemia should aim to identify and address risk factors for hyperglycemia. The glucose infusion rate should be titrated and reduced to physiologic glucose requirements for optimum growth and nutrition. Underlying sepsis should be treated. Discontinue or decrease the dose of drugs that can result in hyperglycemia, like epinephrine, dopamine, or glucocorticoids.

The management of hyperglycemia along with dehydration and/or electrolyte imbalance or ketoacidosis involves resuscitation with intravenous fluids and electrolytes with close monitoring of vitals. Intravenous insulin infusion should be started at a low dose @0.02-0.05 units/kg/hr (as in DKA) to treat persistent hyperglycemia, with hourly monitoring of blood glucose levels [10]. Once the child is stable and fit to take orally, he/she is transitioned to subcutaneous insulin therapy using multiple daily injections (MDI) or continuous subcutaneous insulin infusion (CSII). MDI of subcutaneous insulin using a combination of long-acting insulin glargine (@0.2 to 0.4 unit/kg per day in 1–2 divided doses) and rapid-acting analogs (lispro or aspart or glulisine @0.1–0.15 units/kg per dose before each main feed usually 3–4 injections/day) is recommended to optimize blood glucose control [11,12]. Infants are susceptible to hypoglycemia because of relatively low insulin requirements in NDM's. Smaller doses (0.1 units) using a dilution of the U-100 insulin (100 units/mL) to up to one-tenth of the original concentration may be prepared with a compatible diluent, keeping in mind the shelf life of the insulin. CSII via an insulin pump offers the advantage of delivering smaller doses with accuracy relative to the MDI regimen [13]. Short-acting and intermediate-acting insulin (regular and NPH, respectively) should be avoided due to the increased risk of hypoglycemia and variable glycemic control compared with rapid and long-acting insulin.

Oral sulfonylurea (SU) therapy can be effectively used for glycemic control in NDM due to mutation in the KATP channel. It promotes the closing of the KATP channel and releases insulin from the beta-cells. Oral glibenclamide is the most commonly used SU in the treatment of NDM [14]. It is started at a low dose @ 0.2mg/kg/day in 2 divided doses before a feed. The dose is gradually increased by 0.1 mg/kg/dose up to 1mg/kg/day (occasionally 2–3mg/kg/d) with close monitoring of blood glucose and insulin dose titration and discontinuation, if possible. In 90–95% of children with mutations in KATP channel can be weaned off insulin after starting SU therapy. Further, glibenclamide significantly improves neurological and neuro-psychological abnormalities in individuals with NDM due to *KCNJ11* or *ABCC8* mutations [9]. The dose of glibenclamide depends on the age of initiation and the specific gene mutation. Infants with TNDM due to *ABCC8* and *KCNJ11* mutation need a much lower dose of glibenclamide (0.05 mg/kg/day) than PNDM. Most cases of TNDM do not need insulin as they have some degree of endogenous β-cell function, and may respond to oral SU; while other may spontaneously undergo remission without any therapy [15].

Long-term treatment for neonatal DM requires the involvement of a multidisciplinary team. Regular self-monitoring of blood glucose monitoring and screening for complications is recommended. Children with pancreatic aplasia/hypoplasia will also require exocrine pancreatic supplements. Appropriate genetic counselling is provided to affected families.

SUMMARY

Neonatal diabetes mellitus is a rare monogenic disorder that usually presents within first six months and the diagnosis is considered until 12 months of age with antibody negativity. Two forms have been delineated based on the duration of Insulin dependence i.e. transient and

permanent NDM. Children with NDM should have their molecular genetic testing to define their subtype, plan management and guide prognosis. Most children with NDM due to mutation in the KATP channel can be transitioned to sulfonylurea therapy.

REFERENCES

1. De Franco E, Flanagan SE, Houghton JA, Allen HL, Mackay DJ, Temple IK, et al. The effect of early, comprehensive genomic testing on clinical care in neonatal diabetes: an international cohort study. Lancet. 2015;386(9997):957–63.
2. Greeley SA, Tucker SE, Naylor RN, Bell GI, Philipson LH. Neonatal diabetes mellitus: a model for personalized medicine. Trends Endocrinol Metab. 2010;21(8):464–72.
3. Grulich-Henn J, Wagner V, Thon A, Marg W, Kapellen TM, Haberland H, et al. Entities and frequency of neonatal diabetes: data from the diabetes documentation and quality management system (DPV). Diabet Med. 2010;27(6):709–712.
4. Nansseu JRN, Ngo-Um SS, Balti EV. Incidence, prevalence and genetic determinants of neonatal diabetes mellitus: a systematic review and meta-analysis protocol. Syst Rev. 2016;5(1):188.
5. De Franco E, Ellard S. Genome, exome, and targeted next-generation sequencing in neonatal diabetes. Pediatr Clin North Am. 2015;62(4):1037–1053.
6. Hattersley AT, Greeley SAW, Polak M, Rubio-Cabezas O, Njostad PR, Mlynarski W, et al. ISPAD clinical practice consensus guidelines 2018: the diagnosis and management of monogenic diabetes in children and adolescents. Pediatr Diabetes. 2018;19(Suppl 27):47–63.
7. Letourneau LR, Carmody D, Wroblewski K, et al. Diabetes Presentation in Infancy: High Risk of Diabetic Ketoacidosis. Diabetes Care 2017; 40:e147.
8. Lemelman MB, Letourneau L, Greeley SAW. Neonatal diabetes mellitus: an update on diagnosis and management. Clin Perinatol. 2018;45(1):41–59.
9. Greeley SAW, Polak M, Njølstad PR, Barbetti F, Williams R, Castano L, et al.. ISPAD Clinical Practice Consensus Guidelines 2022: The diagnosis and management of monogenic diabetes in children and adolescents. Pediatr Diabetes. 2022 Dec;23(8):1188-1211.
10. Bottino M, Cowett RM, Sinclair JC. Interventions for treatment of neonatal hyperglycemia in deficient birth weight infants. Cochrane Database Syst Rev. 2011;10:CD007453.
11. Park JH, Shin SY, Shim YJ, Choi JH, Kim HS. Multiple daily injections of insulin regimen for a 10-month-old infant with type 1 diabetes mellitus and diabetic ketoacidosis. Ann Pediatr Endocrinol Metab. 2016;21(2):96–98.
12. Hwang MJ, Newman R, Philla K, Flanigan E. Use of insulin glargine to manage neonatal hyperglycemia in an ELBW infant. Pediatrics. 2018;141(Suppl 5):S399–S403.
13. Beardsall K, Pesterfield CL, Acerini CL. Neonatal diabetes and insulin pump therapy. Arch Dis Child Fetal Neonatal Ed. 2011;96(3):F223–224.
14. Bowman P, Sulen A, Barbetti F, Beltrand J, Svalastoga P, Codner E, et al. Effectiveness and safety of long-term treatment with sulfonylureas in patients with neonatal diabetes due to KCNJ11 mutations: an international cohort study. Lancet Diabetes Endocrinol. 2018;6(8):637–646.
15. Bonfanti R, Iafusco D, Rabbone I, Diedenhofen G, Bizzari, C, Patera PI, et al. Differences between transient neonatal diabetes mellitus subtypes can guide diagnosis and therapy. Eur J Endocrinol. 2021;184(4):575-585.

Newborns and Infants of Mothers with Diabetes Mellitus

DR DEEPA ANIRUDHAN[1], DR KARTHIK BALASUNDARAN[2]
Associate Professor of Pediatrics[1] and Assistant Professor of Pediatrics,[2]
Government Medical College, Thrissur, Kerala

INTRODUCTION

Diabetes in pregnancy is associated with an increased risk of fetal, neonatal, and long-term complications in the offspring [1]. Maternal diabetes may be pre-gestational (i.e. type 1 or type 2 diabetes diagnosed before pregnancy with a prevalence rate of approximately 1.8 percent) or gestational (i.e. diabetes diagnosed during pregnancy with a prevalence rate of approximately 7.5 percent). The outcome is generally related to the onset and duration of glucose intolerance during pregnancy and the severity of the diabetes in the mother. The complications are more likely to occur in pre-gestational diabetes compared to gestational diabetes. The risk of congenital malformations in gestational diabetes is only slightly increased compared to the general population, since the duration of diabetes is less and hyperglycemia occurs later in gestation (typically >25 weeks) [2].

PATHOPHYSIOLOGY

Hyperglycemia in in the mother results in hyperglycemia in the fetus, which in turn causes hypertrophy of the fetal pancreatic islets and beta-cells and thereby increased insulin secretion by the fetus. Chronic fetal hyperinsulinemia leads to an increase in the metabolic rate and oxygen consumption, leading to relative hypoxemia, as the placenta is unable to meet the increased metabolic demands [3]. This in turn results in an increase in the synthesis of erythropoietin and an increase in red blood cell mass and polycythemia. This also promotes catecholamine production, which may result in hypertension and cardiac hypertrophy; and may contribute to the 20 to 30 percent rate of stillbirths seen in poorly controlled pregnancies with diabetes [4]. As the fetal red cell mass increases, iron redistribution results in iron deficiency in developing organs like brain and heart, which may also contribute to cardiomyopathy and altered neurodevelopment [5]. Hyperinsulinemia causes increased fetal growth, particularly of insulin-sensitive tissues (i.e. liver, muscle, cardiac muscle, and subcutaneous fat), resulting in macrosomia, defined as a birthweight (BW) ≥4000 g or greater than the 90th percentile for gestational age (GA).

Hyperinsulinemia has been shown to suppress the production of surfactant in the lung and thus predispose to respiratory distress syndrome after birth. There is excessive accumulation of glycogen in the liver with increased activity of hepatic enzymes involved in lipid synthesis, and accumulation of fat in adipose tissue. These metabolic effects might contribute to long-term metabolic complications in the offspring (Figure 1).

FETAL EFFECTS

In the first trimester maternal hyperglycemia may cause diabetic embryopathy, resulting in major birth defects (neural tube defects, cardiac defects and caudal regression)

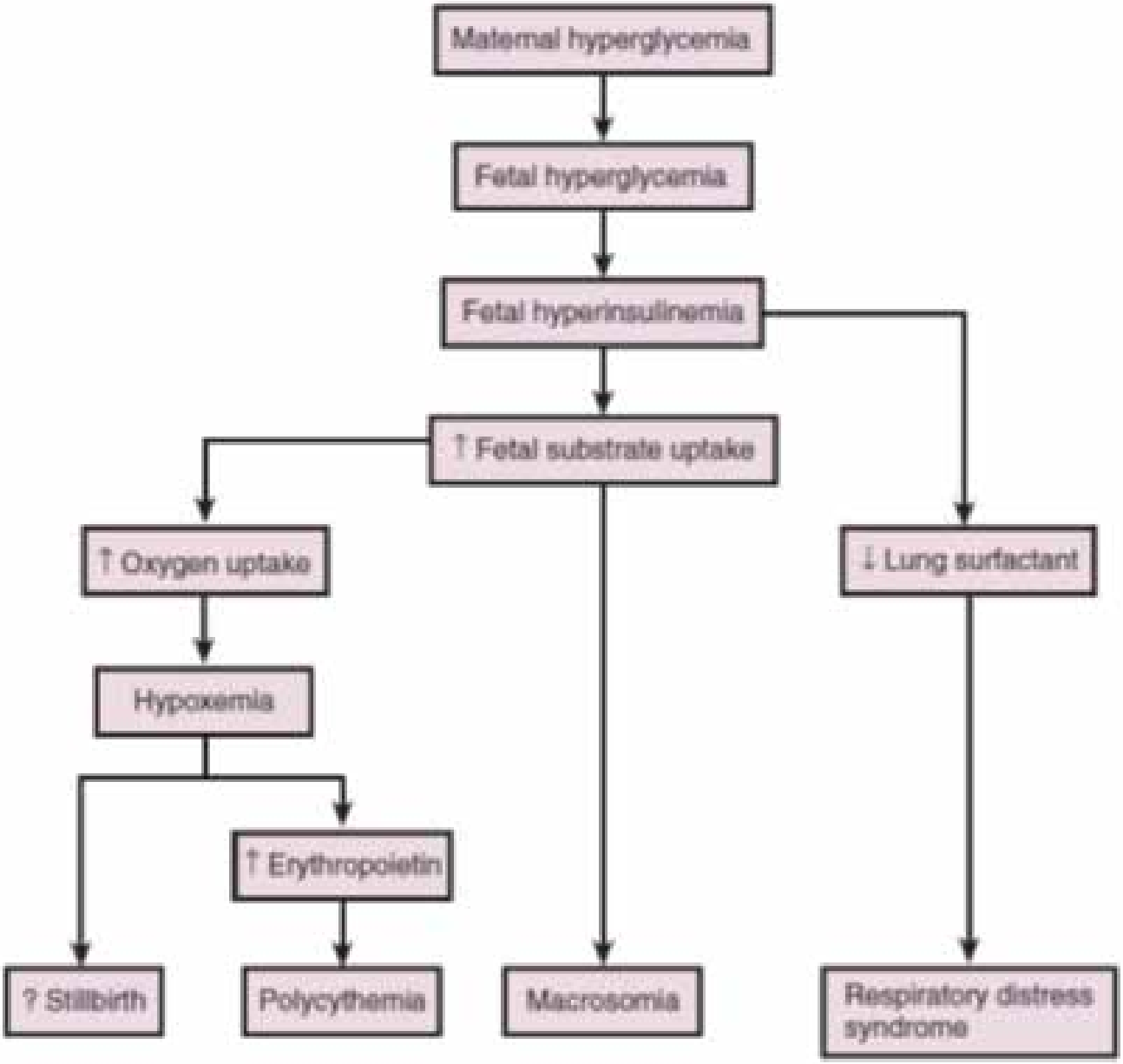

Figure 1: Pathogenesis of fetal and neonatal morbidity in infants of mothers with diabetes

Adapted from: Devaskar SU, Garg M. Diseases of the Fetus and Infant. Disorders of carbohydrate metabolism in neonates. In: Martin RJ, Fanaroff AA, Walsh MC (Eds). Fanaroff and Martin's Neonatal-Perinatal Medicine, 10th Edition. Elsevier Mosby, Philadelphia, 2015; pp. 1439-1444 [1].

and spontaneous abortions especially when the maternal HbA1c is more than 7%. Diabetic fetopathy occurs in the second and third trimesters, resulting in fetal hyperglycemia, hyperinsulinemia, and macrosomia.

NEONATAL EFFECTS

Infants of mothers with diabetes are at increased risk for congenital anomalies, mortality and morbidity compared with neonates born to a mother without diabetes. The perinatal mortality ranges between 0.6 to 4.8 percent. The neonatal complications are listed in Table 1 [6].

Table 1: Neonatal complications in infants of mothers with diabetes

- Congenital anomalies
- Prematurity
- Perinatal asphyxia
- Macrosomia, which increases the risk of birth injury (e.g. brachial plexus injury)
- Respiratory distress
- Metabolic complications: hypoglycemia, hypocalcemia, hypomagnesemia
- Hematologic complications: polycythemia, hyperviscosity
- Low iron stores
- Hyperbilirubinemia
- Cardiomyopathy

Congenital anomalies

There is a four-fold increase in major congenital anomalies due to maternal hyperglycemia at the time of conception and during early gestation (Table 2) [7,8]. Congenital malformations account for approximately 50 percent of the perinatal deaths in infants of mothers with diabetes [9]. The risk can be reduced by strict glycemic control during the pre- and peri-conceptional period (HbA1c < 6.5%). Two-thirds of the anomalies involve either the cardiovascular or the central nervous system.

Preterm delivery

Spontaneous as well as medically indicated preterm delivery occurs more frequently in diabetics than non-diabetic pregnancies.

Macrosomia

At birth these babies are large for gestational age (>4000 g or >90th centile weight for gestational age), plethoric and show evidence of excessive fat as well as visceromegaly in the form of a large liver, spleen, and heart. As the growth of the brain and possibly the kidney is not dependent on insulin, these two organs are normal.

Perinatal asphyxia and birth injury

Macrosomia predisposes these infants to shoulder dystocia. This may lead to brachial plexus

Table 2: Common congenital anomalies in infant of mothers with diabetes [7,8]

System	Manifestations
Neurologic	Anencephaly with or without herniation of neural elements, arrhinencephaly, microcephaly, holoprosencephaly, neural tube defects (meningomyelocele and other variants).
Cardiovascular	Transposition of the great vessels with or without ventricular septal defect (VSD), VSD, coarctation of the aorta with or without VSD or patent ductus arteriosus, atrial septal defect, single ventricle, hypoplastic left ventricle, pulmonic stenosis, pulmonary valve atresia, double outlet right ventricle truncus arteriosus.
Gastrointestinal	Duodenal atresia, imperforate anus, anorectal atresia, small left colon syndrome, situs inversus.
Genitourinary	Ureteral duplication, renal agenesis, hydronephrosis.
Skeletal	Caudal regression syndrome (sacral agenesis), hemivertebrae.
Other	Single umbilical artery, cleft palate.

Adapted from: Tyrala EE. Obstet Gynecol Clin North Am 1996;23:221-41[7] and Reece EA, Homko CJ. Semin Perinatol 1994;18(5):459-69 [8].

injury, clavicular or humeral fractures, perinatal asphyxia, and, less often, cephalohematoma, subdural hemorrhage, or facial palsy [10,11].

Respiratory Distress Syndrome (RDS)

Respiratory distress syndrome occurs due to surfactant deficiency. This is possibly related to an antagonistic effect of insulin on the stimulation of surfactant synthesis by cortisol, leading to a delay in lung maturation. In addition to RDS, other causes of respiratory distress in infants of mothers with diabetes include transient tachypnea of the newborn, polycythemia, asphyxia or cardiomyopathy.

Metabolic complications

Infants of mothers with diabetes have higher incidence of hypoglycemia, hypocalcemia and hypomagnesemia.

Hypoglycemia

Hypoglycemia occurs in around 25–50 percent of infants of mothers with pre-gestational diabetes and 15–25 percent of infants of mothers with gestational diabetes [2]. This occurs as a result of hyperinsulinemia with a lack of a counter-regulatory hormonal response. Significantly lower blood glucose (BG) concentrations occur even in infants of mothers with rigorous glycemic control. As fatty acids are not mobilized from adipose tissue, the circulating levels of free fatty acids (FFA) and ketone bodies remain low [1]. Hypoglycemia can persist for 72 hours and rarely up to 7 days. Hypoglycemia is treated with frequent feedings, although some infants require intravenous (IV) dextrose.

Hypocalcemia

Hypocalcemia becomes apparent between 48 and 72 hours after birth unlike hypoglycemia in 5–30 percent of babies. Plasma calcium concentrations of lower than 7 mg/dL are usually seen. The mechanisms of hypocalcemia are probable failure of the infants of mothers with diabetes to mount an appropriate parathyroid hormone (PTH) response, persistently high levels of calcitonin, and possible alterations in vitamin D metabolism [1].

Hypomagnesemia

Hypomagnesemia, defined as serum magnesium concentration less than 1.5 mg/dL (0.75 mmol/L), occurs in up to 40 percent of infants of mothers with diabetes within the first three days after birth [12]. It has been proposed that low neonatal levels are due to maternal hypomagnesemia caused by increased urinary loss secondary to diabetes. Prematurity may be a contributing factor. It is usually transient and asymptomatic and, thus not treated usually. However, hypomagnesemia can reduce both PTH secretion and PTH responsiveness. As a result, in some neonates with hypocalcemia and hypomagnesemia, the hypocalcemia may not respond to treatment until the hypomagnesemia is corrected [13].

Polycythemia and hyperbilirubinemia

Polycythemia defined as central venous hematocrit>65 percent may lead to hyperviscosity syndrome, including vascular sludging, ischemia, and infarction of vital organs. Hyperviscosity is thought to contribute to the increased incidence of renal vein thrombosis seen in infants of mothers with diabetes. To detect polycythemia, the hematocrit should be measured within 12 hours of birth. Polycythemia leads on to hyperbilirubinemia. Hyperbilirubinemia occurs in 11 to 29 percent of infants of mothers with diabetes, especially in preterm infants [6]. In addition to prematurity, other factors associated with neonatal jaundice include poor maternal glycemic control, macrosomia, and polycythemia.

Low iron stores

The combined erythrocyte and storage iron pools are lower in infants of mothers with diabetes. The degree of low iron stores at birth is inversely related to the degree of polycythemia, suggesting a shunting of fetal iron into the red cell mass [14].

Cardiomyopathy

There is an increased risk of transient cardiomyopathy in infants of mothers with diabetes. Hyperinsulinemia increases the synthesis and deposition of fat and glycogen in the myocardial cells. The most prominent change is thickening of the interventricular septum (IVS) with reduction in the size of the ventricular chambers, resulting in potential obstruction of left ventricular outflow. Although most infants are asymptomatic, 5–10 percent of babies develop cardiac failure. Symptomatic infants typically recover after two to three weeks of supportive care, and echocardiographic findings resolve within 6 to 12 months [15]. Supportive care for symptomatic infants includes increased IV fluid administration and propranolol. Inotropic agents are contraindicated because they may decrease ventricular size and further obstruct cardiac outflow.

TREATMENT

Serious perinatal outcomes in infants of mothers with diabetes can be decreased by early and appropriate treatment of gestational diabetes (diet, glucose monitoring, metformin, and insulin therapy as needed). The fasting BG should be kept below 80 mg/dL and post-prandial values below 120 mg/dL to minimize the risks [1]. It is desirable to express breastmilk before the birth of the baby (≥36 weeks of gestational age) to provide an immediate supply of milk to the newborn in order to prevent hypoglycemia [2].

NEONATAL MANAGEMENT

Infant of a mother with diabetes should be monitored closely after birth. The baby should be fed within 1 hour of birth and BG levels monitored closely. The aim is to keep BG >40 mg/dL in babies <24 hours and >50 mg/dL in babies <48 hours. Thereafter BG should be kept around >60 mg/dL by frequent feedings. If the baby is still jittery or has seizures, serum calcium and magnesium should be obtained and appropriate correction may be given if found to be low.

Asymptomatic hypoglycemia can be managed initially with feeding. Oral or gavage feeding with breastmilk or formula can be given. Oral dextrose gel is an alternative for prophylaxis of hypoglycemia. If the hypoglycemia is recurrent, repeated feedings or IV glucose may be needed. Infants with BG levels persistently <25 mg/dL during the first 4 hours after birth and <35 mg/dL at 4–24 hours after birth should be treated with IV glucose, especially if symptomatic with a small bolus of 200 mg/kg of dextrose (2 mL/kg of 10% dextrose). This should be followed by a continuous IV glucose infusion at a rate of 4–8 mg/kg/min to avoid rebound hyperglycemia [2].

The respiratory and cardiac status should be assessed and baby should be evaluated for any congenital anomaly. Polycythemia and hyperbilirubinemia should be managed as per standard protocols.

LONG-TERM OUTCOME

Metabolic risks

Diabetes mellitus

Infants of mothers with diabetes have an increased risk of developing diabetes, both type 1 and type 2 which is, partially genetically determined. The lifelong risk of type 1 diabetes is 2 percent in offsprings of a mother with type 1 diabetes, 6 percent in siblings, and 65 percent by age 60 years in identical twins (versus 0.3 to 0.4 percent in subjects with no family history) [8].

Obesity and impaired glucose intolerance

Infants of mothers with either pre-gestational diabetes or gestational diabetes are at risk for obesity and impaired glucose metabolism in later life as a result of intrauterine exposure to hyperglycemia. Although macrosomic infants of mothers with diabetes revert to normal body composition in early childhood, during adolescence they tend to be obese with increased adipose tissue mass. In later life there is increased

risk of obesity, insulin resistance, as well as type 2 diabetes (5 to 10 times higher) [1].

Neurodevelopmental outcome

The evaluation of the neurodevelopmental outcome of infants of mothers with diabetes is confounded by the contribution of perinatal events such as perinatal asphyxia and metabolic acidosis in addition to chronic metabolic insults and iron deficiency. Electrophysiologic studies have demonstrated a long-lasting impact on memory, indicating the effect of prenatal iron deficiency on the neuronal development. Although the long-term impact on intellectual functioning is not much affected in infants of mothers with diabetes as compared to normal babies, it is affected in babies with malformations or those who have early growth delay in utero [1].

Mortality and morbidity

The perinatal mortality and morbidity in infants of mothers with diabetes has reduced considerably in recent years as a result of improvements in antepartum care, fetal monitoring, rigorous control of maternal metabolism, and maternal education.

SUMMARY

Diabetes in pregnancy is associated with an increased risk of fetal, neonatal, and long-term complications in the offspring. The management of infants of women with diabetes includes anticipation and treatment of any complication associated with maternal hyperglycemia and also to provide routine neonatal care. Infants of mothers with diabetes should have a thorough physical examination that assesses the respiratory and cardiac status of the infant and identifies any major congenital anomaly. Laboratory evaluation includes measurement of the infant's BG and hematocrit, and screen for hypoglycemia, hypocalcemia, polycythemia and hyperbilirubinemia. Long-term outcome data suggest that prenatal exposure to hyperglycemia increases the risk of postnatal metabolic complications (e.g. diabetes, increased body mass index [BMI], and impaired glucose metabolism) and may also negatively impact neurodevelopmental outcome.

REFERENCES

1. Devaskar SU, Garg M. Diseases of the Fetus and Infant. Disorders of carbohydrate metabolism in neonates. In: Martin RJ, Fanaroff AA, Walsh MC (Eds). Fanaroff and Martin's Neonatal-Perinatal Medicine, 10th Edition. Elsevier Mosby, Philadelphia, 2015; pp. 1439-1444
2. Sheanon NM, Muglia LJ. The Fetus and the Neonatal Infant. The Endocrine system. Chapter 127.1 The infants of diabetic mothers. In: Kliegman R, St. Geme J (Eds.). Nelson Textbook of Pediatrics, 21st Edition; Elsevier Saunders; New York, 2019; pp 983-85.
3. Philips AF, Dubin JW, Matty PJ, Raye JR. Arterial hypoxemia and hyperinsulinemia in the chronically hyperglycemic fetal lamb. Pediatr Res 1982;16(8):653-8.
4. Kitzmiller JL. Sweet success with diabetes. The development of insulin therapy and glycemic control for pregnancy. Diabetes Care 1993;16 Suppl 3:107-21.
5. Nold JL, Georgieff MK. Infants of diabetic mothers. Pediatr Clin North Am 2004;51(3):619-37.
6. Cordero L, Treuer SH, Landon MB, Gabbe SG. Management of infants of diabetic mothers. Arch Pediatr Adolesc Med 1998;152(3):249-254.
7. Tyrala EE. The infant of the diabetic mother. Obstet Gynecol Clin North Am 1996;23:221-41.
8. Reece EA, Homko CJ. Infant of the diabetic mother. Semin Perinatol 1994;18(5):459-69.
9. Weintrob N, Karp M, Hod M. Short- and long-range complications in offspring of diabetic mothers. J Diabetes Complications 1996;10(5):294-301.
10. Weindling AM. Offspring of diabetic pregnancy: short-term outcomes. Semin Fetal Neonatal Med 2009;14(2):111-8.
11. Gregory KD, Henry OA, Ramicone E, Chan LS, Platt LD. Maternal and infant complications in high and normal weight infants by method of delivery. Obstet Gynecol 1998;92(4):507-13.

12. Rosenn B, Miodovnik M, Tsang R. Common clinical manifestations of maternal diabetes in newborn infants: implications for the practicing pediatrician. Pediatr Ann 1996;25(4):215-22.

13. Banerjee S, Mimouni FB, Mehta R, Llanos A, Bainbridge R, Varada K, et al. Lower whole blood ionized magnesium concentrations in hypocalcemic infants of gestational diabetic mothers. Magnes Res 2003;16(2):127-30.

14. Georgieff MK, Landon MB, Mills MM, Hedlund BE, Faassen AE, Schmidt RL, et al. Abnormal iron distribution in infants of diabetic mothers: spectrum and maternal antecedents. J Pediatr 1990;117(3):455-61.

15. Way GL, Wolfe RR, Eshaghpour E, Bender RL, Jaffe RB, Ruttenberg HD. The natural history of hypertrophic cardiomyopathy in infants of diabetic mothers. J Pediatr 1979;95(6):1020-5.

IAP Navi Mumbai

NAVI MUMBAI IAP BRANCH REPORT – DECEMBER 2022 (Till 25th Dec)

ACADEMIC –

1. 1st Dec 2022 – **Journal Journey** – MahaIAP, IAP Raigad, IAP Andhra Pradesh
   Guest Of Honor – Dr Niman Mohanty
   https://youtu.be/Ko_9Ui7she8

2. 2nd Dec 2022 - **IAP PG Teaching Sessions**
   Experts – Prof Dr S Balasubramanian, Prof Dr Srinivasan
   Convenors – Dr V N Yewale, Dr Snehal Mallakmir, Dr Jeetendra Gavhane, Dr Satish Shahane
   https://us02web.zoom.us/j/87234744551?pwd=WDEvR012QllvcXhPUG9aTlVxdTJrUT09

3. 8th Dec 20222 – **Measles Outbreak & Vaccination – IAP Raigad**
   Panelist – Dr V N Yewale
   https://us02web.zoom.us/j/81670982530?pwd=djkwbEZyZzM0OTAxWUlxNmhnMFkvUT09

4. 10th Dec 2022 – **Non-Communicable Disease Prevention – Begin from womb**
   Dignitary – Dr Upendra Kinjawdekar

5. 15th – 18th Dec 2022 – **ICAAICON - 56th Annual conference of Indian College of Allergy, Asthma and Applied Immunology**
   Topic – Allergy Practice in real world Setting
   Panelist – Dr Vikram Patra

6. 16th Dec 2022 - **IAP PG Teaching Sessions**
   Experts – Prof Dr S Balasubramanian, Prof Dr Srinivasan
   Convenors – Dr V N Yewale, Dr Snehal Mallakmir, Dr Jeetendra Gavhane, Dr Satish Shahane
   https://us02web.zoom.us/j/86252701148?pwd=SjhSa21abEpZaUJ5c2JHZjAxWHdnUT09

7. 18TH Dec 2022 **– Interdisciplinary CME with pediatricians, obstetricians & ENT surgeons**
   Topic - Universal Newborn Hearing Screen: Let's Catch Them Young!
   Speakers – Dr Amit Saxena, Dr Leena Deshpande, Dr Gargi Bangar
   Panelist – Dr Satish Shahane
   MOC – Dr Asmita Patil
   Moderator – Dr Mumtaz Sharif

8. 21ST Dec 2022 – IAP Raigad
   Topic: UTI in Office Practice
   Expert – Dr Pankaj Deshpande
   https://us02web.zoom.us/j/83591861067?pwd=OG1LSkUxQVR3WHZWUTE5a2REQmlOUT09.

IAP Navi Mumbai

SOCIAL –

1. **Dr Jeetendra Gavhane**, President NMIAP was invited to be a member of the task force by the Navi Mumbai Municipal Corporation Health Dept. for handling the Measles Outbreak in Navi Mumbai.
2. **Dr Uddhav Talnikar & EB NMIAP member Dr Madhavi Ingale** were instrumental in starting Autism Screening at NMMC Hospital Navi Mumbai
3. A **Walkathon** was organized on International Day of the Disabled by NMMC Staff in order to spread awareness regarding Importance Early Newborn Hearing Screening.
4. On International day for the Disabled, **Dr Jeetendra Gavhane**, President NMIAP was invited as a **Chief Guest** to address the NNMC official s and Hospital Staff on the topic of newborn hearing screening. The event was graced by NMMC Commissioner Dr A. Bangar & Addl. Commissioner Mrs. Sujata Dhole.
5. **Pediatrician Author & blogger Dr Shilpa Aroskar** shares tips from her personal experiences on work life balance in the 2nd episode of her podcast. https://open.spotify.com/episode/2VdYHtJYtdSM016L0ezBaF?si=MSnRTHiTQ4-AEEwpKDn1fQ&utm_source=whatsapp
6. **Dr Pravin Gaikwad & Dr Arti Gaikwad** won 4 gold medals in Maharashtra masters athletics at Aurangabad, qualified for nationals in Running 5 k and 10 k and 2 Swimming events.
7. **Dr. V N Yewale addressed the gathering at National Expert Consultation on NAP – AMR 2.0 (2023 – 2027)** talking about the IAP work in last 10 years in AMR right from the Mission AAA to IAP ICMR Call to Action, Id surv. platform to collect data on resistance, training workshops with RAP, WAR. He also conveyed how the IAP and TB division are working together. He extended support of any for AMR through a well- established network of 30000 + pediatricians and 400 branches across the country. IAP modules will be utilized by the government.
8. **Dr V N Yewale** was quoted in Times of India on the safety profile of cough syrups in pediatric population.

IAP Navi Mumbai

IAP Kerala

Antimicrobial awareness week - IAP Pathanamthitta

IAP Kerala

Newborn week observation IAP Pathanamthitta

IAP Kerala

IAP Kerala OB meeting with DMO Measles outbreak at Malappuram

IAP Wayanad medical camp for SAM children

IAP Kerala

Adolescent health class - IAP Trivandrum

IAP Kerala

IAP Wayanad medical camp for SAM children

IAP Kerala

IAP Wayanad medical camp for SAM children

IAP Kerala

IAP Wayanad medical camp for SAM children

IAP Kerala

IAP Kannur Family meet

IAP Kerala

CALPEDICON INAUGRATION