Child India

January 2025

Monthly e-Newsletter of Indian Academy of Pediatrics

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DR KALPANA DATTA (CHATTERJEE)
DR DIBYENDU RAYCHAUDHURI
BRIG. (DR) SHUVENDU ROY (Service)
DR MIHIR SARKAR (Org. Secretary, Pedicon 2026)

CONTENT

Editor's Note

Dear friends,

New year greetings to you all through the 1st issue of Child India for 2025.

On behalf of all of you we congratulate Dr Basavaraja GV and his team - HSG Dr Yogesh Parikh, OB and EB 2024 for all the good work done and wish IAP President 2025, Dr Vasant Khalatkar and his team - HSG Dr Yogesh Parikh, OB and EB 2025 who, we are certain that under the able leadership of our energetic IAP President, will work in full earnest to ensure that the IAP flag flies high. We also congratulate Dr Neelam Mohan President Elect and wish her the very best.

World Braille Day is celebrated on January 4th each year, to celebrate the birth anniversary of the French educator Louis Braille, who invented the Braille, inspired by a military night-writing code invented by a French army man, Charles Barbier de la Serre. The system was further refined for use in various purposes like literature and mathematics. Louis Braille lost his sight at a very young age due to a mishap. The theme for World Braille Day 2025 was "Celebrating Accessibility and Inclusion for the Visually Impaired" and it was created to raise awareness about the importance of braille as the best communication medium for blind people. By celebrating this day, teachers, caregivers, and parents can tell their children about how braille is extremely helpful for people who need it.

In 1984 the Government of India decided to celebrate the birthday of great Swami Vivekananda, i.e. 12 January, as National Youth Day (Rashtriya Yuva Diwas) every year. The philosophy of the Swamiji and the ideals for which he lived and worked is considered a great source of inspiration for the Indian youth. The day is dedicated to inspiring young minds, promoting education, and encouraging community service. The theme for National Youth Day 2025 is "Youth Empowerment for Nation Building", focusing on empowering young people to recognize their capabilities and contribute to the nation's development.

World Leprosy Day is observed on the last Sunday of January, every year. In2025 it is on Jan 26th and the theme for 2025 is "Unite. Act. Eliminate.". The theme is a call to action aiming to raise awareness of leprosy, highlight the challenges faced by persons affected by leprosy, and inspire collaborative action to eliminate leprosy.

This issue that deals with Pediatric Dermatology is thanks to submissions from Dr Vijaybhaskar and the OB, EB 2025 and me are extremely grateful to him.

Happy reading,

Dr Jeeson C Unni
Editor-in-Chief

President's Address

Dear Colleagues,

Warm greetings from CIAP as your President for 2025!

I am truly humbled and grateful for the trust and confidence you have placed in me. It is a profound honor to lead our esteemed organization, and I assure you of my unwavering commitment to this responsibility. My approach will always be collaborative—we will walk side by side, not ahead or behind, as we work to elevate IAP to new heights.

Reflections on Pedicon 2025: A Benchmark of Excellence

Looking back at Pedicon 2025 in Hyderabad, I can't help but feel a deep sense of pride. It was nothing short of a masterpiece—a conference that surpassed all expectations and left an indelible mark on us all.

From the majestic chariot welcome to the spectacular inauguration, every detail reflected the passion and dedication of our colleagues from Hyderabad and Telangana. The scientific sessions, workshops, CME programs, and TOT sessions were executed flawlessly, showcasing not only meticulous planning but also the true spirit of volunteerism. That it was all managed by Hyderabad's senior members without professional event managers is a testament to their commitment to IAP's ideals.

The hospitality, grand banquets, and thoughtful touches like the mementos made this event a memorable one. Special thanks to Dr. Surendranath, Dr. Neeli, and their exceptional team for their relentless efforts. You have set a new benchmark for future Pedicons. Three cheers to you!

A Call to Action: Preparing for the Unexpected – Tackling HMPV in Children

Now that we are back to our workplaces after the phenomenal Pedicon, it's time to address a pressing concern that emerged during our discussions: Human Metapneumovirus (HMPV).

HMPV, while not new, has been causing an alarming surge in cases recently. This respiratory virus, especially dangerous for children under five, can lead to severe illnesses like bronchiolitis and pneumonia, often requiring hospitalization. While sporadic cases have been around since its identification in 2004, the current spike has the potential to overwhelm pediatric infrastructure across the country.

As pediatricians, we have a duty to act - not just react. Let's focus on a unified, multi-faceted approach:

President's Address

Key Focus Areas for Addressing HMPV

1. Enhanced Surveillance:

Strengthening monitoring systems to detect outbreaks early and respond swiftly.

2. Infection Control:

Establishing and following strict infection control protocols in hospitals, clinics, and communities to reduce transmission.

3. Research and Innovation:

Encouraging studies to deepen our understanding of HMPV, develop effective vaccines, and improve treatment options.

4. Infrastructure and Preparedness:

Revisiting our pediatric infrastructure post-COVID to ensure it meets the demands of our growing population. This includes adequate hospital beds, oxygen supplies, trained personnel, and public education.

5. Government Collaboration:

Advocating for policies that prioritize funding for research, public awareness campaigns, and equitable vaccine distribution.

6. Public Awareness:

Educating communities on prevention, early symptom recognition, and timely medical intervention.

The Role of IAP

The Indian Academy of Pediatrics has already initiated steps by forming a dedicated HMPV Task Force. This team will provide expert guidance and collaborate with the government to strengthen preparedness. Together, we aim to create robust strategies to protect our children from the worst impacts of HMPV.

A Collective Effort: The Role of All Stakeholders

- Healthcare Professionals:
- Stay updated with the latest guidelines and research.
- Follow infection control measures rigorously.
- Educate families about prevention and early recognition of symptoms.
- Advocate for necessary policy changes.
- Families and Communities:

President's Address

- Practice good hygiene, like regular handwashing and respiratory etiquette.
- Stay informed about HMPV and its prevention.
- Support vaccination initiatives.
- Seek medical care promptly if symptoms persist.
- Government and Policymakers:
- Ensure funding for research, surveillance, and response strategies.
- Promote policies that prioritize child health.
- Drive public awareness campaigns for better preparedness.

The Road Ahead

As pediatricians, we stand as guardians of the youngest and most vulnerable members of society. But addressing a challenge like HMPV requires teamwork. Together—with healthcare professionals, families, policymakers, and our wider community—we can create a safer future.

Let us channel the spirit of collaboration and excellence demonstrated at Pedicon 2025 to tackle this challenge and many more ahead. With your continued support, I am confident we will rise to every occasion, setting new milestones for IAP.

Warm regards,

Dr. Vasant Khalatkar
President, CIAP 2025

Secretary's Message

Respected Seniors and Dear Friends,

"No one can whistle a symphony. It takes a whole orchestra to play it."

January is the month of planning activities to be implemented in the year 2025. I am extremely happy to inform you that this month had a pleasant start with the Welcome 2025 - "Happy New Year" Meeting held on 01st of January 2025 via zoom platform to welcome all the OB and EB members of 2025. Allotment of 08 IAP Action Plan Modules allotment was sent on 01st and 02nd of January 2025 to 63 branches.

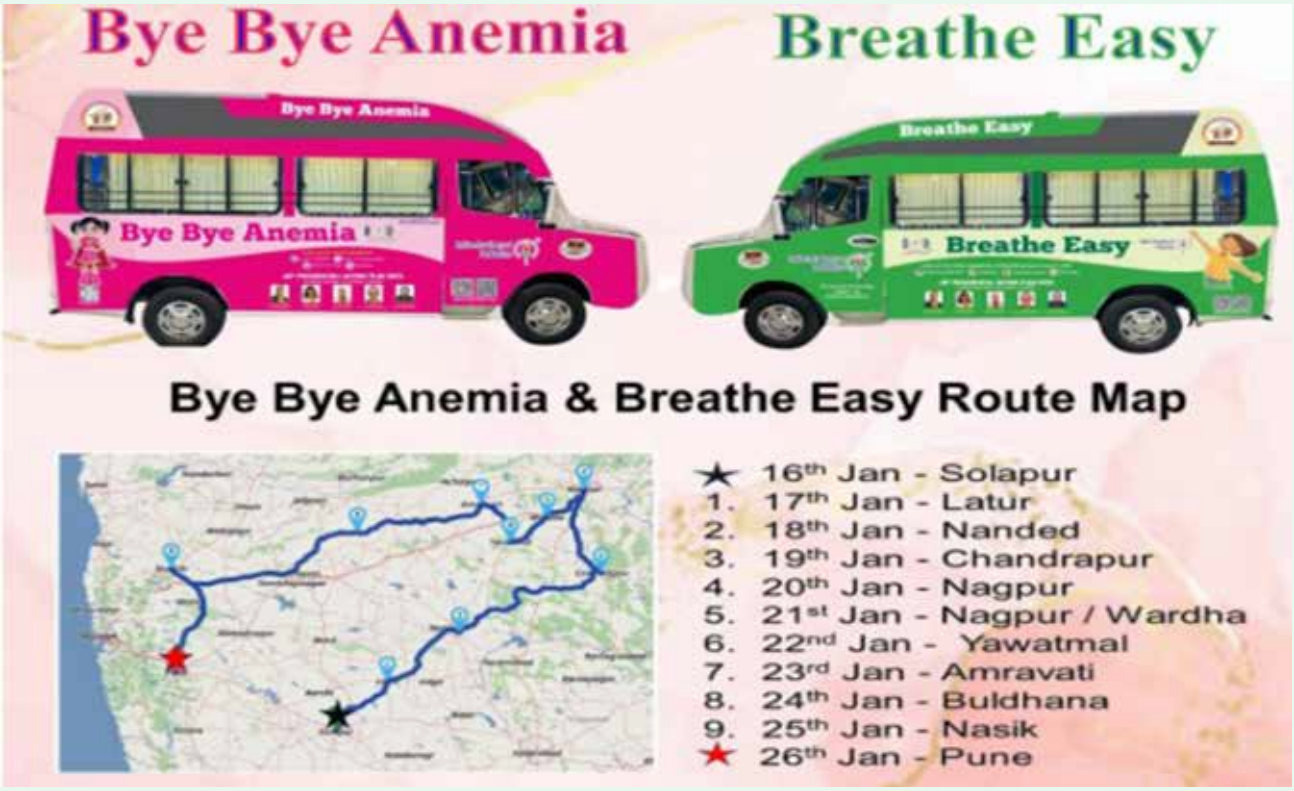
Also our Iconic 62nd Pedicon 2025 was held from 08th to 12th January 2025 at HICC Novotel Convention Centre Hyderabad. Padma Vibhushan Dr Nageshwara Reddy Gastroenterologist, inaugurated the conference. Also Mr. Nitin Gadkari – Minister of Road Transport and Highways of India and Dr Harsh Vardhan – Minister of Health and Family Welfare gave their virtual message for Pedicon 2025. This event brought together pediatricians, researchers, and healthcare professionals worldwide to discuss the latest advancements in pediatric care, research, and education. Over 7000 distinguished guests graced the conference, including renowned Speakers and Editors.

We have also successfully conducted various "PHYSICAL" meetings during IAP Pedicon 2025 at Hyderabad such as IAP Office Bearer Meeting on 06th of January; IAP Executive Board Meeting on 07th January; NRP ToT/Meeting on 08th January; Meeting with IP & IJPP on 09th January along with the Inaugural Function; Shantilal C Seth Oration, meeting with State/District Branches and Annual General Body Meeting on 10th January; Meeting with IAP Sub Specialty Chapters and Meeting with ICP on 11th January followed by Award paper Presentation and PG Quiz; UG Quiz, other Awards and Valedictory Function on 12th January.

Secretary's Message

Innaugration of "Bye Bye Anaemia Drive" and "Breathe Easy Busyatra Campaign" under the IAP Presidential Action Plan 2025 on 09th January 2025

Secretary's Message



Along with this we have also conducted various "VIRTUAL" meetings such as Launch of EEE Education by Essence of Experience on 01st January; Tribunal Committee Meeting on 01st as well as 02nd January; Bus Yatra Meeting on 04th January; OMAG Annual Meeting 2025 on 12th January; Anemia Bus Meeting on 14th January; Yuva Pedicon on 16th and 20th of January; Meeting of Chicken Pox – GSK and Meeting with IAP State and City Branch Office Bearers on 22nd January; Income Tax - Vivaad Se Vishwas on 23rd January; IAP-Tribunal Meeting on 24th and 25th January; Blood Donation and Organ Donation Webinar on 25th January; IAP - BAHRAIN Webinar on "Updates in Neonatology" on 26th January;

A virtual Release of film "DISHAA" was done under the flagship of IAP ki Baat Community Ke Sath on 26th January 2025

Various "Branch Installation" Ceremony were held as below:

- 4th January - Hubli Dharwad District
- 5th January – Hyderabad Branch
- 19th January - Bangalore and Indore Branch
- 25th January – Nashik Branch
- 26th January – Pune Branch

Secretary's Message

Secretary's Message

Secretary's Message

IAP-ICP-recognized certificate courses were successfully launched in various pediatric specialties. These courses have already commenced, and additional programs in advanced ventilation, pulmonology, learning disabilities, early childhood care, bio-ethics, mental health, and gastroenterology are set to begin soon, spearheaded by distinguished experts

- Behavioral Disorders & Autism, and Pediatric Infectious Diseases (IAP-PIDC)
- Pediatric Emergency (IAP-PECC)
- Allergy & Asthma (IAP-PAAC)
- IAP Vaccinology (IAPVAC)
- Pediatric Infectious Disesases (IAP-PIDCC)

Along with this, IAP conducted 11 National ToTs on the following modules under the Presidential Action Plan 2025 on 08th January 2025:

1. CARE: Cough Assessment and Respiratory Evaluation
2. CUTIS: Clinical Update for Tackling Issues of Skin
3. EASE: Encephalitis Awareness, Study and Evaluation
4. HIT: History, Investigation & Treatment - Typhoid & Beyond
5. ONE HEALTH: Fighting Outbreaks and Challenges of Zoonotic Diseases in Pediatrics
6. PINK FOOTSTEP: Proactive Interventions for Nutrition and Knowledge, 'A footstep taken to prevent Anemia'
7. POEM: Pediatric Office Emergency Medicine
8. VAC: Vaccinology Awareness for Clinicians
9. WYI: Well Youth Initiative Mpower Yuva Module
10. PEER: Pediatric Endocrinology Evaluations and Recommendations Module
11. NURSE: Nursing Advocacy in Pediatrics Module

Also January month has recorded successful conduction of total of 62 NRP courses as follows

- Basic NRP courses - 55
- Advance NRP courses – 6 + 1 Regional ToT = 7

Secretary's Message

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 Yogesh N Parikh
Secretary General, IAP 2024 & 2025

Please join us by clicking on IAP official social media accounts links below:-

IAP official Facebook page :-(https://www.facebook.com/iapindiaofficial)

IAP official Twitter page :-(https://twitter.com/i/flow/login?redirect_after_login=%2Fiapindia)

IAP official Instagram page :-(https://www.instagram.com/indianacademyofpediatrics/)

IAP official YouTube channel :-(https://www.youtube.com/channel/UCVI2bRlWIrm4Rz3rSmmE3XQ)

IAP official WhatsApp channel :-(https://whatsapp.com/channel/0029VaO0bMA35fM0TC9z9c03)

Use Hashtags :- #IAPkiBaat #IAP #indianacademyofpediatrics #anemia #muktbharat

President's Engagements

President Medal Exchange

Executive Board Meeting

President's Engagements

President's Engagements

President's Engagements

President's Engagements

IAP Bengaluru – Inaugural and Installation Cerermony

IAP Indore Branch Installation Ceremony

Bye Bye Anemia and Breathe Easy Program – Nasik

President's Engagements

NRP – FGM Workshop at Hyderabad

IAP Indore Branch Installation Ceremony

IAP Pune Branch Installation

IAP Nasik Branch Installation

President's Engagements

Bye Bye Anemia and Breathe Easy Program – Kolhapur

President's Engagements

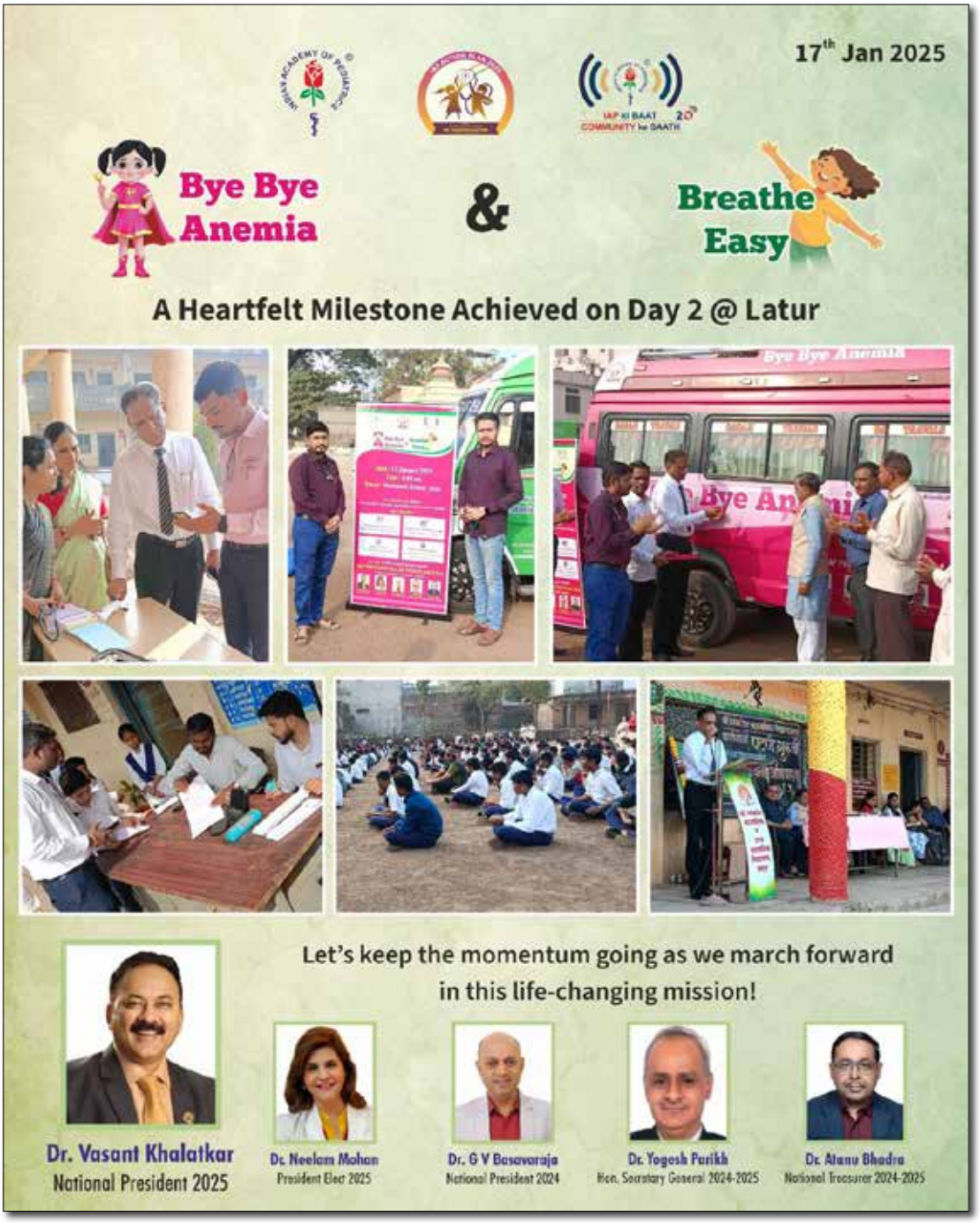
Presidential action plan 2025 by President CIAP Dr Vasant Khalatkar "Bye Bye Anemia" on wheels and Breathe easy was Flagg off by honourable transport minister Mr Nitinji Gadkari in Nagpur.

Dr Vasant khalatkar discussed on this project and importance of awareness in India to educate on Anaemia & Asthma by CIAP.

Mr Nitin ji Gadkari appreciated this noble concept of screening of needy kids in various school across India Mr Gadkariji given his best wishes to Dr Vasant Khalatkar for his various presidential action plans in child health.

The program was grace by AOP Nagpur patron Dr Uday Bodhankar, MAHAIAP President Dr Sanjay Pakhmode, MAHAIAP 25 Convenor Dr Sanjay Deshmukh, AOP President Dr Shilpa Hazare, Secretary Dr Kailash Vaidya, AHA President Dr Meena Deshmukh, President Elect AOP Nagpur Dr Sandeep Mogre, NNF Nagpur President Dr Milind Mandalik, Dr Anil raut, Dr Pravin Pagey, Dr Mohib Haque, Dr Girish Charde, Dr Pranoti Jadhav, Dr Arachana Jaiswal, Dr Vivek Shivhare, Dr Vinit Wankhede

President's Engagements



A Heartfelt Milestone Achieved at Latur! The second stop of our transformational journey, 'Bye Bye Anemia' & 'Breathe Easy Yatra' touched hearts and lives in Latur! With an overwhelming response and enthusiastic participation, we united to combat anemia and spread awareness about lung health. From empowering sessions to impactful health drives, the spirit of Laturians shone bright as we took bold steps toward a healthier tomorrow. Together, we are making a difference—one city, one life at a time. A big THANK YOU to all the incredible individuals, healthcare heroes, and partners who made this stop a resounding success. Let's keep the momentum going as we march forward in this life-changing mission!

National Awards presented at PEDICON 2025 Hyderabad

Shantilal Seth Oration

Dr Naveen Thacker MD, FIAP

Director of Deep Children Hospital & Research Centre at Gandhidham-Kutch, Gujarat, India. He is the President of the International Pediatric Association (IPA) for 2023-2025. He serves as Vice-chair of the Steering Committee of the International Collaboration on Advanced Vaccinology Training (ICAVT)

The Most Eminent Teachers Award were presented to :

NAME	CITY	STATE
DR GJ KASLIWAL	SOLAPUR	MAHARASHTRA
DR APURBA KUMAR GHOSH	KOLKATA	WEST BENGAL
DR SHALLY AWASTHI	LUCKNOW	UTTAR PRADESH
DR VEENA KALRA	NEW DELHI	DELHI
DR S THANGAVELU	CHENNAI	TAMIL NADU

The **Lifetime Achievement Award** were presented to

NAME	CITY	STATE
DR SHASHI N VANI	AHMEDABAD	GUJARAT
DR M INDRA SHEKHAR RAO	SECUNDERABAD	TELANGANA
DR S SRINIVASAN	PUDUCHERRY	PUDUCHERRY
DR MAYA MUKHOPADHYAY	KOLKATA	WEST BENGAL
DR KARUNA THAPAR	AMRITSAR	PUNJAB

The **Social Champion Award** were presented to

NAME	CITY	STATE
DR UDAY BODHANKAR	NAGPUR	MAHARASHTRA
DR JS TUTEJA	INDORE	MADHYA PRADESH
DR KIRAN AGGARWAL	DELHI	DELHI
DR K JAYOJI RAO	BENGALURU	KARNATAKA
DR SUJOY CHAKRAVARTY	HOWRAH	WEST BENGAL

Presidential Excellence Award were presented to

NAME	CITY	STATE
DR AJEET M GOPCHADE	NANDED	MAHARASHTRA
DR SATISH B DEOPUJARI	NAGPUR	MAHARASHTRA
DR RAJIV PK	ERNAKULAM	KERALA
DR SHRINATH B MUGALI	DHARWAD	KARNATAKA
DR P RAMACHANDRAN	CHENNAI	TAMIL NADU

Honorary Fellowship

Dr Shobhna Gupta

Deputy Commissioner & Incharge of Child Health Division & RBSK programme of Ministry of Health and Family Welfare, Government of India

Dr. Arun Kumar Neopane

Nepal Pediatric Association as President, Director General, Medical Services (DGMS), Head of different Medical Operations

IAP Fellowship Award were presented to

SR No.	Member Name	City	State
01	DR UPENDRA S KINJAWADEKAR	NAVI MUMBAI	MAHARASHTRA
02	DR A CHENTHIL	CUDDALORE	TAMIL NADU
03	DR A YASHOWANTH RAO	HYDERABAD	TELANGANA
04	DR ADARSH E	BENGALURU	KARNATAKA
05	DR AMAR SINGH THAKUR	BILASPUR	CHHATTISGARH
06	DR ANIL SHRIRAM RAUT	NAGPUR	MAHARASHTRA
07	DR ARJIT MOHAPATRA	KHURDA	ODISHA
08	DR ASHOK V BADAKALI	BAGALKOT	KARNATAKA
09	DR BASAVARAJ M PATIL	GULBARGA	KARNATAKA
10	DR CHANDRAKALA BS	BENGALURU	KARNATAKA
11	DR DEEPAK CE	TARIKERE	KARNATAKA
12	DR DINESH KUMAR SINGH	GORAKHPUR	UTTAR PRADESH
13	DR DINESHKUMAR P CHIRLA	HYDERABAD	TELANGANA
14	DR GEETANJALI SETHY	CUTTACK	ODISHA
15	DR GHANSHYAM CHAUDHARY	JHANSI	UTTAR PRADESH
16	DR JAI SINGH	CHITTORGARH	RAJASTHAN
17	DR JYOTI KUMAR GUPTA	KANPUR	UTTAR PRADESH
18	DR K KESAVULU	ANANTAPUR	ANDHRA PRADESH
19	DR KHURSHID AHMED WANI	SRINAGAR	JAMMU AND KASHMIR
20	DR M VENKATACHALAPATHY	CHIK BALLAPUR	KARNATAKA
21	DR MANISH GUPTA	NEW DELHI	DELHI
22	DR MANMEET KAUR SODHI	AMRITSAR	PUNJAB
23	DR MINHAJ A SHEIKH	MUMBAI	MAHARASHTRA
24	DR NEHAL PATEL	AHMEDABAD	GUJARAT

25	DR P ASHOK KUMAR	KOCHI	KERALA
26	DR PANKAJ AGARWAL	BARMER	RAJASTHAN
27	DR PRASHANT V KARIYA	SURAT	GUJARAT
28	DR R RAMAKRISHNA PARAMAHAMSA	RAJAHMUNDRY	ANDHRA PRADESH
29	DR RAJENDRA GOVINDRAO PATIL	NAGPUR	MAHARASHTRA
30	DR RAMESH B DAMPURI	SECUNDERABAD	TELANGANA
31	DR RAMESH M BAJANIA	SURENDRA NAGAR	GUJARAT
32	DR RASHNA DASS HAZARIKA	GUWAHATI	ASSAM
33	DR RIAZ I	THIRUVANANTHAPURAM	KERALA
34	DR SANJEEV KUMAR DIGRA	JAMMU	JAMMU AND KASHMIR
35	DR SHAMIK GHOSH	KOLKATA	WEST BENGAL
36	DR SUBHASISH BHATTACHARYYA	KOLKATA	WEST BENGAL
37	DR SUSRUTA DAS	KHURDA	ODISHA

Post Graduate Quiz Winner

DR. RAMYA .S DR. SAKTHIVIGNESH	**1st Prize**	INSTITUE OF CHILD HEALTH , MADRAS MEDICAL COLLEGE , CHENNAI

DR ANNAPOORNA DR SRAVANI	2nd Prize	GANDHI MEDICAL COLLEGE, HYDERABAD

Under Graduate Quiz Awards

DR AJINKYA NAIK DR MAHESH BANSAL	1st Prize	AIMMS, NEW DELHI

DR BASUDHA RAY DR MALCOLAM JOSEPH CASTELINO	2nd Prize	KALINGA INSTITUTE OF MEDICAL SCIENCES, BHUBANESWAR

Dr. James Flett Endowment Award

1st Prize - Dr Badavath Naresh, Warangal

2nd Prize - Dr Srijan Sinha – Ranchi

Dr. S. T. Achar Endowment Award

1st Prize - Dr Hamsika V, Puducherry

2nd Prize - Dr Jyothi Lakshmi Sorna Mariappan, Thiruvallur

Dr. S. S. Manchanda Neonatology Research Award

1st Prize - Dr Revant Kumar, New Delhi

2nd Prize - Dr M Jeeva Sankar, New Delhi

Dr. V. Balagopal Raju Endowment Award

1st Prize - Dr Supriya Kushwah, Dakshina Kannada

2nd Prize - Dr Ravi Teja Meda, Anantapur

Branches and Chapters Awards – 2024

STATE	
(Category: State with 1 - 100 members)	
STATE	**PRIZE**
IAP Tripura State Branch	**First Prize**
IAP Meghalaya State Branch	**Second prize**
IAP Manipur State Branch	**Third Prize**
IAP Mizoram State Branch	**Appreciation**
(Category: State with 101 - 200 members)	
STATE	**PRIZE**
IAP Goa State Branch	**First Prize**
IAP Himachal Pradesh State Branch	**Second prize**
(Category: State with 201 – 350 members)	
STATE	**PRIZE**
IAP Uttarakhand State Branch	**First Prize**
(Category: State with 351 - 600 members)	
STATE	**PRIZE**
IAP Chhattisgarh State Branch	**First Prize**
(Category: State with 601 – 1000 members)	
STATE	**PRIZE**
IAP Odisha State Branch	**First Prize**
IAP Punjab State Branch	**Second prize**
(Category: State with 1001 - 2000 members)	
STATE	**PRIZE**
IAP Delhi State Branch	**First Prize**
IAP Madhya Pradesh State Branch	**First Prize**
IAP Haryana State Branch	**Second prize**
IAP Bihar State Branch	**Appreciation**
(Category: State with 2001 - 3000 members)	
STATE	**PRIZE**
IAP Andhra Pradesh State Branch	**First Prize**
IAP West Bengal State Branch	**First Prize**
IAP Uttar Pradesh State Branch	**Second prize**
IAP Telangana State Branch	**Third Prize**
IAP Gujarat State Branch	**Appreciation**

(Category: State with >3001 members)	
STATE	**PRIZE**
IAP Karnataka State Branch	**First Prize**
IAP Kerala State Branch	**First Prize**
IAP Maharashtra State Branch	**First Prize**
IAP Tamil Nadu State Branch	**Second prize**

CITY/DISTRICT

(Category: City / District / Local with 1 - 100 members)	
CITY/DISTRICT	**PRIZE**
IAP Yavatmal Branch	**First Prize**
IAP Aligarh Branch	**Second prize**
IAP Chhindwara Branch	**Second prize**
IAP Murshidabad Branch	**Second prize**
IAP Palghar District Branch	**Second prize**
IAP Tellicherry Branch	**Second prize**
IAP Gadag Branch	**Third Prize**
IAP Morbi Branch	**Third Prize**
IAP Osmanabad Branch	**Third Prize**
Datia Academy Of Pediatrics	**WTD**
IAP Hissar Branch	**WTD**
IAP Raigad Branch	**NBW**
IAP Tiruvannamalai District Branch	**AAD**
IAP Wayanad Branch	**AAD**
Academy Of Pediatrics Mayurbhanj	**Appreciation**
IAP Badagara Branch	**Appreciation**
IAP Balasore Branch	**Appriciation**
IAP Banaskantha Branch	**Appreciation**
IAP Buldhana Branch	**Appreciation**
IAP Chandrapur Branch	**Appreciation**
IAP Durg-Bhilai Branch	**Appriciation**
IAP Hingoli Branch	**Appreciation**
IAP Kamareddy Branch	**Appriciation**
IAP Kasargod Branch	**Appriciation**
IAP Keonjhar Branch	**Appreciation**
IAP Rohtak Branch	**Appreciation**
IAP Siliguri Branch	**Appreciation**
IAP Theni-Cumbam-Bodi Branch (Tcb)	**Appreciation**
IAP Vidisha Branch	**Appreciation**

(Category: City / District / Local with 101 - 200 members)	
CITY/DISTRICT	**PRIZE**
IAP Pathanamthitta Branch	**First Prize**
IAP Kolhapur Branch	**Second prize**
IAP Erode Branch	**Third Prize**
Jalandhar Academy Of Pediatrics	**Third Prize**
IAP Kanpur Branch	**Third Prize**
IAP Amritsar Branch	**WOD**
IAP CTKK Branch (TT Branch)	**AAD**
IAP Gwalior Branch	**DD**
IAP Howrah Branch	**ORS**
IAP TTKPN Branch	**CPR**
IAP Agra Branch	**Appreciation**
IAP Amravati Branch	**Appreciation**
IAP Burdwan Branch	**Appreciation**
IAP Cuttack Branch	**Appreciation**
IAP Davangere Branch	**Appreciation**
IAP Faridabad Branch	**Appreciation**
IAP Guwahati Branch	**Appreciation**
IAP Jabalpur Branch	**Appreciation**
IAP Karimnagar Branch	**Appreciation**
IAP Raipur Branch	**Appreciation**
IAP Shimoga Branch	**Appreciation**

(Category: City / District / Local with 201 - 400 members)	
CITY/DISTRICT	**PRIZE**
IAP Kannur Branch	**First Prize**
IAP Kottayam Branch	**First Prize**
IAP Gurgaon Branch	**Second prize**
IAP Navi Mumbai Branch	**Second prize**
IAP Mysore Branch	**Third Prize**
IAP Rajkot Branch	**Third Prize**
Bhopal Association Of Pediatricians (IAP Bhopal Branch)	**Appreciation**
IAP Bhubaneswar City Branch	**Appreciation**
IAP Dakshina Kannada Branch	**Appreciation**
IAP Indore Branch	**Appreciation**
IAP Kozhikode (Calicut) Branch	**Appreciation**
IAP Krishna Branch	**Appreciation**
IAP Kurnool Branch	**Appreciation**
IAP Madurai Branch	**Appreciation**
IAP Nasik Branch	**Appreciation**
IAP Thane Branch	**Appreciation**
IAP Thrissur Branch	**Appreciation**
IAP Vadodara Branch	**Appreciation**

(Category: City / District / Local with 401 - 600 members)	
CITY/DISTRICT	**PRIZE**
IAP Nagpur Branch	**First Prize**
Surat Pediatric Association Charitable Trust	**First Prize**
IAP Jaipur Branch	**Second prize**
IAP Kochi (Cochin) Branch	**Second prize**
IAP Lucknow Branch	**Second prize**
IAP South Delhi City Branch	**Third Prize**
IAP Thiruvananthapuram Branch	**WOD**
IAP East Delhi City Branch	**Appreciation**
IAP Pune Branch	**Appreciation**
IAP Thiruvananthapuram Branch	**Appreciation**
IAP West Delhi City Branch	**Appreciation**

(Category: City / District / Local with >1001 members)	
CITY/DISTRICT	**PRIZE**
IAP Bengaluru Branch	**First Prize**
IAP Mumbai Branch	**First Prize**
IAP Chennai Branch	**Second prize**
IAP Twin Cities Branch	**Second prize**

CHAPTERS	**PRIZE**
IAP Non-Communicable Diseases Chapter	**First Prize**
Medicolegal Chapter	**First Prize**
Infant & Young Child Feeding Chapter	**Second Prize**
Hematology-Oncology Chapter	**Second Prize**
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Neurology Chapter	**Third Prize**
Environment & Child Health Chapter	**Third Prize**


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Neonatal Herpes Simplex

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Introduction

Neonatal herpes simplex virus (HSV) infection is a rare but potentially severe condition with high mortality and morbidity. First reported in the 1930s, its spectrum and treatment have since evolved significantly. HSV, particularly type 2, is most often transmitted during delivery through an infected maternal genital tract, though transplacental and nosocomial transmission can also occur.[1] Early antiviral therapies such as vidarabine demonstrated efficacy but were associated with toxicity, leading to the adoption of acyclovir as the standard treatment. Studies have shown that higher doses and prolonged administration of acyclovir improve outcomes, reducing the devastating effects of the infection.[2] Advancements in diagnostics, particularly polymerase chain reaction (PCR), have improved early detection and treatment monitoring, significantly enhancing neonatal prognosis.

Epidemiology

Neonatal herpes simplex virus (HSV) infection is a rare but serious condition, with an incidence in the United States estimated between 1 in 3,200 to 1 in 10,000 live births, accounting for approximately 1,500 cases annually.[3] In India, while specific incidence data is limited, the high prevalence of maternal HSV infections and restricted access to prenatal care suggest a potentially higher burden. Globally, the incidence varies based on healthcare infrastructure, with an estimated 10 per 100,000 live births.[4]

Transmission

HSV disease of the newborn is acquired during one of three distinct time intervals: intrauterine (congenital), peripartum (perinatal), and postpartum (postnatal). The overwhelming majority (~85%) of neonatal HSV infections occur during the peripartum period, typically via exposure to infected genital secretions during vaginal delivery. An additional 10% of cases are acquired postnatally, usually from caregivers with active HSV lesions, while only ~5% occur in utero, resulting in congenital HSV infection.[5]

Perinatal Infection (~85%)

Perinatal HSV transmission occurs when the newborn is exposed to the virus during vaginal delivery in the presence of maternal genital HSV infection. The risk is highest when the mother acquires a primary HSV infection late in pregnancy, as she lacks protective HSV-specific antibodies. Additional factors influencing transmission include:

- Duration of ruptured membranes (prolonged rupture increases risk).
- Use of fetal scalp monitors (provides an entry point for the virus).
- Mode of delivery: While cesarean section reduces transmission risk, it does not completely eliminate it.

Intrauterine Infection (Congenital HSV, ~5%)

Congenital HSV occurs when the virus infects the fetus at any point during pregnancy. The affected newborn often presents at birth or within 48 hours of life with a characteristic triad of symptoms:

- Cutaneous manifestations: Vesicular skin lesions, rash, aplasia cutis, and areas of hypo- or hyperpigmentation.
- Ocular involvement: Chorioretinitis, keratoconjunctivitis, and optic nerve atrophy.
- Central nervous system (CNS) abnormalities: Intracranial calcifications, microcephaly, encephalopathy, or hydranencephaly.

Severe cases may also present with placental infarcts, hydrops fetalis, or stillbirth, often leading to significant neurodevelopmental impairment or death

Postnatal Infection (~10%)

Postnatal transmission occurs when the neonate contracts HSV from close contact with an infected caregiver, such as a family member with active herpes labialis (cold sores). Neonates may also be exposed through breast lesions, contaminated hands (herpetic whitlow), or exposure in the nursery. Unlike infants born to mothers with prior HSV infections, neonates exposed postnatally do not benefit from transplacental maternal antibodies, making them more susceptible to severe disease.

Disease classifications

Neonatal HSV infections acquired peripartum or postpartum are further categorized into three clinical presentations:

1. Skin, Eye, and Mouth Disease: Accounts for ~45% of neonatal HSV cases. It is the most localized form, characterized by vesicular skin lesions, conjunctivitis, and oropharyngeal involvement. If left untreated, SEM disease can progress to CNS or disseminated forms.
2. Central Nervous System (CNS) Disease: Encephalitis with or without SEM involvement occurs in ~30% of neonatal HSV cases, often presenting with seizures, irritability, or lethargy.
3. Disseminated Disease: The most severe form, affecting multiple organs, including the CNS, lungs, liver, adrenal glands, skin, eyes, and mouth, and accounts for ~25% of cases. This form has the highest mortality

The timing of symptom onset varies by disease classification. Disseminated and SEM disease typically present at 10 to 12 days of life, whereas CNS disease manifests later, around 16 to 19 days of life.[6-8]

Clinical features

Skin, Eyes, and/or Mouth Disease

Skin, eyes, and mouth disease is the most common clinical manifestation of neonatal HSV infection, accounting for approximately 45% of cases. This form is typically marked by vesicular lesions that appear on the skin, eyes, or mucous membranes, often on areas of trauma, such as the scalp, eyelids, or over the presenting body part during delivery. Lesions typically arise within the first 1–2 weeks of life but can also be seen earlier, especially in cases where there was prolonged rupture of membranes. These vesicles usually have an erythematous base and contain clear or slightly cloudy fluid. Eye involvement can be subtle and often asymptomatic initially, which necessitates early ophthalmologic examination if any symptoms appear.[9]

Although SEM disease is rarely fatal if confined to the skin and mucous membranes, it can progress to more severe forms such as disseminated disease or CNS infection if left untreated. Infants with untreated SEM lesions are at risk of developing significant neurological impairments, including microcephaly, spastic quadriplegia, or sensory loss by 12 months.[9]

Central Nervous System (CNS) Disease

Around 30% of neonates with HSV infection develop CNS disease, which may or may not be accompanied by SEM involvement. Infants typically present between 10 days to 4 weeks of age with signs such as fever, temperature instability, poor feeding, and irritability, progressing to seizures, a bulging fontanel, and focal neurological signs. CSF analysis typically shows a pleocytosis (50–100 cells/mm^3), elevated protein levels, and low to normal glucose. Although the presence of skin lesions can aid diagnosis, around 60-70% of CNS cases will develop vesicular lesions at some point.[10]

Disseminated Disease

Disseminated disease accounts for about 25% of neonatal HSV infections, with a particularly poor prognosis in preterm infants. Symptoms typically emerge within the first 14 days of life and can present like sepsis, with respiratory distress, jaundice, hepatomegaly, and coagulopathy. Seizures, meningitis, and respiratory failure are also common. Interestingly, up to 50% of cases may not show skin vesicles at any point during the illness, complicating diagnosis. Mortality in untreated cases can be as high as 90%, even with antiviral therapy, although survival rates improve significantly with early intervention.[11]

Intrauterine Infection

Intrauterine HSV infection is rare, occurring in approximately 5% of neonatal cases. It is typically diagnosed when neonates present with a triad of symptoms: cutaneous lesions (e.g., scarring, active lesions, hypo- and hyperpigmentation, or erythematous rashes), ocular findings (such as microphthalmia, retinal dysplasia, and optic atrophy), and neurological manifestations (e.g., microcephaly, encephalomalacia, intracranial calcifications). These infants may also present with hydranencephaly, placental infarcts, or in utero death, resulting in significant neurodevelopmental impairment. Intrauterine HSV infection typically leads to severe manifestations immediately at birth or within 48 hours.

Postnatal Infection

Postnatal HSV infections, which occur in about 10% of cases, typically arise from close contact with caregivers who have an active HSV infection, such as herpes labialis. Transmission can also occur via breast lesions or herpetic whitlow in the nursery. Infants born to mothers without prior HSV exposure are more vulnerable to severe outcomes since they lack protective maternal antibodies. These infections are often less common but can still lead to severe disease, especially if not recognized early.

Diagnosis

Polymerase Chain Reaction (PCR)

Polymerase Chain Reaction (PCR) is considered the gold standard for diagnosing neonatal herpes simplex virus (HSV) infections, particularly in cases of Skin, Eyes, and Mouth (SEM) disease. PCR is highly sensitive and specific, detecting HSV DNA even in very low viral loads. It is preferred because it provides rapid results and can be performed on various specimens, including skin lesions, eye swabs, cerebrospinal fluid (CSF), and blood. This makes it invaluable not only for diagnosing SEM disease but also for detecting disseminated forms of HSV. In SEM cases, PCR is often conducted on swabs taken from skin lesions or eye discharge, enabling clinicians to confirm the presence of the virus early and differentiate HSV from other pathogens that may cause similar clinical presentations, such as bacterial infections. PCR's high sensitivity allows detection of the virus even before the appearance of visible symptoms, making it a crucial tool in the management of neonatal HSV.[2]

Viral Culture

Historically, viral culture was widely used for diagnosing HSV infections, including SEM disease, but it is now considered less sensitive

than PCR and takes longer to yield results. Despite these limitations, viral culture can still provide important diagnostic information and is sometimes used when PCR is unavailable. In cases of SEM disease, viral culture can help identify the specific strain of HSV (whether HSV-1 or HSV-2), which may have implications for treatment and prognosis. However, since viral culture has a lower sensitivity and can be slower, it is generally used as a supplementary test to PCR rather than as the primary diagnostic tool.2

Direct Fluorescent Antibody (DFA) Testing

Direct Fluorescent Antibody testing is a rapid diagnostic method that uses labeled antibodies to detect HSV antigens directly in clinical specimens. Although DFA can be performed quickly, it is less commonly used than PCR due to the latter's superior sensitivity and specificity. DFA can still be useful in certain clinical situations, particularly when rapid results are needed, and is often applied to skin or eye lesions in cases of SEM disease. Despite its utility, DFA is not as widely favored because of its lower sensitivity compared to PCR and its limited ability to detect the virus in asymptomatic or early-stage infections.[12]

Tzanck Smear

The Tzanck smear involves scraping the base of a lesion and examining it under a microscope for the presence of multinucleated giant cells, which are characteristic of viral infections, including HSV. However, this test is not specific to HSV and can also detect other viral infections that cause similar cellular changes, such as varicella-zoster virus. Due to its nonspecific nature and relatively low diagnostic accuracy, the Tzanck smear is no longer widely used for diagnosing neonatal HSV infections, particularly SEM disease. While it can provide an initial clue that a viral infection is present, it is largely replaced by more specific and sensitive tests, such as PCR, in modern clinical practice.[13]

Serological Tests

Serological tests, which measure the presence of HSV-specific antibodies, are not typically used in the neonatal diagnosis of HSV, especially in SEM disease. This is because newborns often do not produce detectable levels of antibodies, particularly in the early stages of infection, and maternal antibodies can interfere with the results. Serological testing might be more relevant in older infants or in assessing the immune status of a child, but it is not helpful for diagnosing SEM disease. In SEM cases, where the primary focus is on identifying the virus through direct detection methods such as PCR, serological tests play a limited role. Moreover, these tests are not useful for differentiating between active and past infections in neonates.2

Cerebrospinal Fluid (CSF) Analysis

CSF analysis is performed when CNS involvement is suspected, especially in cases where SEM disease may progress to more severe forms of HSV infection, such as HSV encephalitis. A lumbar puncture is performed to obtain CSF, which is then analyzed for signs of infection, including elevated white blood cells (pleocytosis), elevated protein levels, and low glucose levels, which suggest viral encephalitis. Although CSF analysis is primarily used for diagnosing CNS or disseminated HSV infections, PCR testing of CSF is highly valuable for confirming HSV as the causative agent. In SEM disease, CNS involvement is less common but still possible, so CSF analysis may be performed if symptoms such as lethargy or seizures develop in conjunction with skin, eye, or mouth lesions. PCR testing of CSF in these cases would provide definitive evidence of HSV infection and help guide treatment decisions.2

Management

Skin, Eye, and Mouth (SEM) Disease:

Neonates with SEM disease should receive intravenous acyclovir 20 mg/kg every 8 hours

for 14 days. If HSV DNA is detected in CSF or there are abnormal CSF parameters, treatment should be extended to 21 days. Suppressive therapy with oral acyclovir (300 mg/m^2/dose, three times daily for 6 months) is recommended to reduce skin recurrences. Ocular involvement requires additional topical antiviral therapy under ophthalmologic supervision (e.g., trifluridine or vidarabine). Regular neutrophil count monitoring is essential due to the risk of neutropenia.[14]

CNS or Disseminated Disease:

Acyclovir 20 mg/kg IV every 8 hours for 21 days, followed by oral suppressive therapy for 6 months in CNS disease cases.14

Supportive Care:

Infants should receive adequate IV fluids, respiratory support if needed, and correction of clotting abnormalities.

Prognosis

Skin, Eye, and Mouth (SEM) Disease:

The prognosis for SEM disease is excellent with early and appropriate treatment. Mortality is extremely rare, and neurological outcomes remain unaffected if the infection remains localized. However, without treatment, SEM disease can progress to CNS or disseminated disease, significantly worsening outcomes. Suppressive oral acyclovir therapy for 6 months reduces skin lesion recurrences but does not impact neurological development.[15]

CNS Disease:

Despite IV acyclovir therapy (60 mg/kg/day for 21 days), mortality is around 4%, and neurological impairment is common, with up to 70% of survivors experiencing developmental delay, seizures, or motor deficits.

Disseminated Disease:

The most severe form, with 30% mortality despite treatment. Survivors often have significant multi-organ complications and long-term neurodevelopmental disabilities.

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Collodion Baby

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Introduction

Collodion baby (CB) is a rare congenital dermatological disorder first described by Hallopeau and Watelet.[1] It is characterized by a tight, shiny, parchment-like membrane encasing the entire body at birth, resembling oiled parchment or cellophane. CB is not a distinct disease but rather a transient phenotype of various congenital keratinization disorders, primarily autosomal recessive ichthyosis, including lamellar ichthyosis (LI) and non-bullous congenital ichthyosiform erythroderma (NBCIE).[2]

Due to an impaired skin barrier, affected neonates are prone to complications such as dehydration, hypothermia, infections, and respiratory distress. The membrane typically cracks and sheds within the first few weeks of life, revealing either normal or persistently abnormal ichthyotic skin. While some infants (10%) experience complete resolution of symptoms (self-healing collodion baby), approximately 75% develop lifelong ichthyosis, and the remaining 15% exhibit other dermatological or systemic disorders.[3,4]

Epidemiology

The estimated incidence of CB ranges between 1 in 50,000 to 1 in 100,000 live births, though underreporting may lead to inaccuracies in prevalence data.[5] The condition occurs worldwide with no significant ethnic or gender predilection. However, a higher prevalence has been observed in populations with increased consanguinity due to the autosomal recessive inheritance pattern.

Etiology and Genetic Basis

Collodion baby is a genetically heterogeneous condition, primarily inherited in an autosomal recessive pattern. It represents a phenotypic manifestation of several congenital disorders of keratinization.

CB is caused by mutations in genes responsible for epidermal differentiation, lipid metabolism, and cornification. The most commonly implicated genes[6] include:

- TGM1 (Transglutaminase 1) – Accounts for 40% of cases. Located on chromosome 14q11.2, this gene encodes transglutaminase-1, an enzyme crucial for cross-linking proteins in the epidermal cell envelope, ensuring skin barrier integrity. Mutations in TGM1 disrupt this function, leading to ichthyotic skin changes.
- NIPAL4 (Ichthyin) – Plays a role in epidermal lipid transport and keratinization.
- ALOX12B and ALOXE3 – Involved in epidermal lipid metabolism, essential for normal desquamation. Mutations in ALOX12B and TGM1 are specifically implicated in NBCIE, leading to a milder phenotype compared to LI.
- ABCA12 – Encodes a lipid transporter protein crucial for epidermal barrier formation. Severe

mutations in ABCA12 result in Harlequin ichthyosis, a more severe keratinization disorder.

- CYP4F22, PNPLA1 (OMIM 615024), and CERS3 (OMIM 615023) – These genes regulate ceramide synthesis and lipid metabolism in the epidermis. Disruptions lead to defective skin barrier function and ichthyotic features.
- In NBCIE, mutations in ALOX12B and TGM1 result in defective transglutaminase-1, impairing epidermal differentiation and leading to a less severe clinical presentation compared to LI.

Underlying Disorders:

Collodion baby is most commonly associated with the following congenital ichthyoses:

- Lamellar Ichthyosis (LI) – Severe form with generalized scaling and lifelong symptoms.
- Non-bullous Congenital Ichthyosiform Erythroderma (NBCIE) – Characterized by erythroderma and fine white scaling, often less severe than LI.

Other less common causes of CB include:[7]

- Ectodermal Dysplasia – Group of disorders affecting skin, hair, and sweat glands.
- Sjögren-Larsson Syndrome – Ichthyosis with neurological and intellectual impairment.
- Comèl-Netherton Syndrome – Ichthyosis associated with atopic dermatitis and hair shaft abnormalities.
- Gaucher Disease Type 2 – Lysosomal storage disorder with ichthyosis and systemic involvement.
- Hay-Wells Syndrome – Ectodermal dysplasia with skin and craniofacial abnormalities.
- Trichothiodystrophy – Ichthyosis with brittle hair and developmental delay.
- Neutral Lipid Storage Disease – Metabolic disorder leading to ichthyosis and lipid accumulation.

Pathophysiology

The fundamental abnormality in CB is disordered cornification, leading to the formation of the collodion membrane. This results in:

- Impaired epidermal barrier function – Leading to increased transepidermal water loss (TEWL) and susceptibility to dehydration and infections.
- Abnormal lipid metabolism – Defects in ceramide synthesis and lipid transport contribute to ichthyosis and skin dysfunction.
- Defective keratinocyte differentiation – Mutations in TGM1 and related genes impair normal epidermal maturation, leading to hyperkeratosis and scaling.

Clinical Features of Collodion Baby

Neonatal Presentation

- Collodion membrane: A tight, shiny, parchment-like covering over the entire body at birth.
- Ectropion: Outward turning of the eyelids, leading to corneal exposure, dryness, and risk of ulceration.
- Eclabium: Eversion of the lips, often fixing them in an 'O' shape, affecting feeding and oral closure.
- Flattened nose and ears: Due to membrane-induced tightness, leading to hypoplastic nasal and auricular cartilage.
- Hypotrichosis: Scanty or absent hair, though hair may perforate the horny covering over time.
- Pseudocontractures: Restricted limb and digit movements, leading to distal limb ischemia, edema, and digital necrosis in severe cases.
- Poor sucking reflex: Due to restricted lip and jaw movement, resulting in feeding difficulties.

- Respiratory distress: Can occur in severe cases due to chest restriction and hypoventilation.

Systemic Complications

- Severe transepidermal water loss (TEWL): More than six times higher than in normal neonates, leading to hypernatremic dehydration, electrolyte imbalances.[8]
- Temperature instability: High risk of hypothermia due to impaired thermoregulation.
- Secondary infections: Fissures in the collodion membrane predispose to bacterial and fungal infections, commonly caused by Candida, Staphylococcus aureus, and Pseudomonas species.
- Ophthalmologic complications: Corneal ulceration and scarring due to ectropion and lagophthalmos (inability to close eyelids).
- Hearing loss: Due to accumulation of scales in the external auditory canal.
- Aspiration pneumonia: A potential life-threatening complication.
- Progression to chronic ichthyosis: 75% of cases progress to autosomal recessive congenital ichthyosis (ARCI), requiring lifelong dermatologic care.3

Severe Spectrum: Harlequin Ichthyosis

At the extreme end of congenital ichthyosis, Harlequin ichthyosis presents with thick, armor-like hyperkeratotic plates that are deeply fissured, creating a characteristic appearance.[9] This condition severely restricts movement and leads to respiratory distress due to the tight, rigid skin, which limits chest expansion. Feeding difficulties are also common, often resulting from eclabium (outward turning of the lips) and orofacial restriction, further complicating nutritional intake. The neonatal mortality rate for Harlequin ichthyosis is high, primarily due to infections, dehydration, and respiratory failure, making early and intensive medical intervention crucial.

Diagnosis

The diagnosis of Collodion Baby (CB) is primarily clinical, based on the presence of a characteristic collodion membrane at birth, along with associated features such as ectropion, eclabium, hypoplastic nasal and auricular cartilage, and restricted limb movements. In cases where the diagnosis is uncertain, genetic testing can confirm mutations in ichthyosis-related genes, aiding in prognosis and genetic counseling. Although histopathology is rarely needed, a skin biopsy may be performed in atypical cases, revealing hyperkeratosis, thickened stratum corneum, and abnormal epidermal differentiation. Additionally, enzyme activity of transglutaminase can be analyzed on culture or via immunofluorescence on a skin biopsy specimen to further aid in diagnosis.

A prognostic scoring system has been developed to assess the severity and outcomes of CB.[10] This system includes parameters such as:

- Extent and severity of collodion membrane – Complete versus partial body involvement.
- Degree of ectropion and eclabium – Assessing the impact on eye and oral function.
- Severity of transepidermal water loss (TEWL) – Risk of dehydration and electrolyte imbalance.
- Respiratory complications – Presence of chest restriction and hypoventilation.
- Risk of infections – Presence of skin fissures and secondary bacterial or fungal infections.
- Neurological and systemic involvement – Including feeding difficulties, limb ischemia, and hearing impairment.

This scoring system helps predict short-term survival, the likelihood of developing autosomal recessive congenital ichthyosis (ARCI), and the need for intensive neonatal care.

However, it is not yet widely standardized in clinical practice.

Management

The management of a collodion baby requires a multidisciplinary approach, involving dermatologists, neonatologists, ophthalmologists, ENT specialists, and geneticists. The primary focus is supportive care to prevent complications associated with the condition, which primarily includes maintaining the skin barrier, controlling infections, ensuring adequate hydration and nutrition, and preventing dehydration and electrolyte imbalances.

Neonatal Care:

Environmental Control: A humidified incubator is essential to reduce trans-epidermal water loss (TEWL) and prevent dehydration. The recommended humidity levels typically range from 40% to 60%, though some advocate for higher levels of 90% to 100%, despite limited evidence to support the latter. Care in the incubator is typically maintained for at least four weeks or until membrane desquamation, although transitioning to an open crib may occur sooner, depending on clinical condition. The incubator's temperature should be carefully monitored, ideally between 32–34°C, to prevent overheating due to impaired sweating, ensuring that the neonate's body temperature is regulated.[2]

Skin Care: The collodion membrane should not be manually removed, as it will shed naturally within two to four weeks. Frequent bathing with water, sometimes with a mild cleanser, is recommended, and liberal application of emollients (such as petroleum jelly, vitamin E acetate, glycerol, sodium chloride, and urea-based moisturizers) should be done to prevent excessive skin dryness and maintain hydration. It is crucial to avoid the use of keratolytics, including salicylic acid, glycolic acid, and N-acetylcysteine, due to the risk of systemic absorption and toxicity.

Hydration and Nutrition: Adequate hydration is vital to prevent dehydration and TEWL. Intravenous (IV) fluids are administered to correct fluid and electrolyte imbalances, while tube feeding is necessary if oral intake is compromised or inadequate. Close monitoring for signs of dehydration and electrolyte disturbances is essential.

Infection Control: Strict aseptic precautions must be followed to prevent infections, particularly sepsis, which can be a significant cause of morbidity and mortality in collodion babies. Early administration of antibiotics is crucial if secondary infections occur, and continuous surveillance for signs of infection is required.

Ophthalmologic Care: Collodion babies often suffer from ectropion (outward turning of the eyelids), which can expose the cornea to drying, leading to corneal ulceration. Artificial tears and lubricants should be used to prevent corneal damage. If ectropion persists, surgical correction may be needed, or periocular retinoids like tazarotene may be used to address the condition.

ENT Care: Regular cleaning of the external auditory canal is important to prevent potential hearing loss, especially since malformed ears and poor development of the nostrils are common in these neonates.

Pharmacologic Therapy: Oral retinoids, such as isotretinoin or acitretin (1 mg/kg/day), are often used to reduce scaling and prevent hyperkeratosis, both of which are hallmark features of congenital ichthyosis in collodion babies. Topical retinoids may also aid in restoring the skin barrier function. Mild topical steroids can be used to reduce secondary inflammation.

Genetic and Prenatal Diagnosis:

Collodion babies most commonly present with autosomal recessive congenital ichthyosis, and thus, genetic counseling is recommended for affected families. Prenatal diagnosis is possible through various methods, including genomic PCR using chorionic villous samples around the

10-12th weeks of gestation, or amniocentesis at 15-18 weeks. At 18-20 weeks, fetoscopy and fetal skin biopsy can be performed to detect ultrastructural abnormalities. The TGM1 gene is typically the first to be analyzed, followed by other genes like ALOX12B, ALOXE3, and NIPAL4 if necessary.[11]

Prognosis of Collodion Baby

The prognosis for a collodion baby depends largely on the severity of the condition, the presence of complications, and the management provided in the neonatal period.

Survival Rate: Mortality in collodion babies is primarily attributed to complications like dehydration, sepsis, respiratory failure, and electrolyte imbalances. With appropriate and timely neonatal care, including humidified incubators, skin moisturization, and infection control, the survival rate has improved, with reports indicating a survival rate of about 81% if the neonate survives the initial period.

Skin Recovery: The collodion membrane typically sheds within 2-4 weeks, and the skin gradually recovers with the use of emollients and retinoids. If the neonate survives this early period, the skin can improve significantly, although long-term management of ichthyosis-related symptoms may be required.

Long-Term Health: If the neonate survives the neonatal period, long-term prognosis is generally determined by the underlying cause of the collodion presentation. In cases of autosomal recessive congenital ichthyosis, long-term care focuses on skin hydration, preventing infections, and managing associated complications. However, if systemic complications (such as sepsis or electrolyte imbalances) are well-controlled, long-term survival can be good, with many children leading relatively normal lives.

Developmental and Neurological Outcomes: While the condition itself may not directly impact neurological function, complications such as hypoxia, infections, and severe dehydration can contribute to developmental delays. With proper treatment, most collodion babies can achieve normal developmental milestones, although follow-up care is essential.

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Juvenile Dermatomyositis

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Introduction

Juvenile dermatomyositis (JDM) is a rare, idiopathic inflammatory myopathy primarily affecting children under the age of 18. It is characterized by progressive muscle weakness and distinctive cutaneous manifestations, including heliotrope rash and Gottron's papules.[1] JDM is an autoimmune condition, with immune-mediated vascular damage playing a central role in its pathogenesis. Although the exact etiology remains unclear, genetic susceptibility, environmental triggers, and dysregulated immune responses contribute to disease onset. The clinical presentation varies from mild muscle weakness to severe systemic involvement, impacting quality of life and long-term prognosis. Early diagnosis and appropriate management are crucial in preventing complications such as calcinosis, contractures, and cardiorespiratory dysfunction.

Epidemiology

The mean age at onset is approximately 7 years, with earlier onset observed in girls. About 25% of affected children present before the age of 5. The time from the appearance of initial symptoms to diagnosis typically averages around 6 months but can range from as short as 5 weeks to as long as 2 years.[2]

The incidence of JDM varies geographically. In the United States, it ranges from 2.5 to 4.1 cases per million children annually, while in the United Kingdom, the reported incidence is slightly lower, at approximately 1.9 to 3.2 cases per million children. Racial differences have been observed in the United States, with an estimated annual incidence of 3.4 per million in White children, 3.3 per million in Black children, and 2.7 per million in Hispanic children.[3-5]

JDM exhibits a strong female predominance, with a female-to-male ratio of approximately 2.3:1 in the United States and a higher ratio of 5:1 in the United Kingdom. The median age of onset is around 6.8 years in girls and 7.3 years in boys. The median time to diagnosis is typically 3–4 months, though this may vary.[4,5]

The exact incidence of juvenile dermatomyositis (JDM) in India remains largely undocumented due to limited epidemiological studies. However, recent research has provided valuable insights into its prevalence and clinical characteristics. A study involving 43 children diagnosed with JDM reported that 81.4% had at least one myositis-specific or myositis-associated autoantibody, suggesting a significant burden of the disease within the Indian pediatric population (Joseph et al., 2024).[6]

Etiology

The exact cause of JDM remains unclear, but it is thought to result from a combination of genetic susceptibility and environmental triggers. A key factor in the pathogenesis is the involvement of the immune system, including both the innate and adaptive immune responses.

Genetic Factors:

JDM is associated with several genetic

markers, particularly within the human leukocyte antigen (HLA) region. Alleles like HLA-DQA1*0501 and HLA-DR3 are linked with an increased risk of developing the condition. Other genetic loci related to proinflammatory cytokine genes and lymphocyte signaling also contribute to disease susceptibility.[7]

Environmental Triggers:

- Infectious agents such as Coxsackie B virus, Parvovirus B19, Enteroviruses, and Streptococcus species are suspected to trigger the onset of JDM, likely through mechanisms like molecular mimicry or breaking self-tolerance.[8]
- Non-infectious triggers include D-penicillamine, vaccinations, and bone marrow transplants.
- Ultraviolet radiation (UVR) exposure may also play a role in exacerbating or triggering the condition, especially in the context of specific autoantibodies.

Immune Mechanisms:

- Type I interferon-alpha/beta genes are overexpressed in JDM, indicating an activated immune response. These interferons can upregulate MHC class I expression, promote T-cell survival, and induce pro-inflammatory cytokines.[9]
- Autoantibodies directed against proteins involved in protein synthesis or gene regulation (such as tRNA synthetases and histidyl RNA synthetase) are often found in JDM patients and may contribute to muscle inflammation.[10]

Pathophysiology

Juvenile dermatomyositis (JDM) is characterized by both humoral (antibody-mediated) and cell-mediated immune responses, leading to vascular injury and muscle inflammation. Autoantibodies targeting endothelial antigens contribute to vascular injury, microangiopathy, and ischemia, which in turn result in muscle damage and inflammation. The activation of the complement system, particularly the deposition of the membrane attack complex (MAC), further exacerbates vascular damage and muscle pathology.

Immune cell infiltration plays a crucial role in disease progression, with CD4+ T cells, B cells, plasmacytoid dendritic cells (pDCs), and macrophages accumulating around blood vessels in affected muscle tissues. TH17 cells, a subset of CD4+ T cells, produce IL-17, which stimulates muscle cells to express MHC class I and pro-inflammatory cytokines like IL-6, amplifying the inflammatory response.

Interferon activation is another key mechanism in JDM pathogenesis. Plasmacytoid dendritic cells produce excessive amounts of interferon-alpha and interferon-beta, leading to endothelial and muscle fiber damage. This persistent interferon signaling contributes to the chronic inflammation observed in JDM.

Genetic and cytokine factors also influence disease severity and progression. TNF-alpha polymorphisms, particularly TNF-α-308A, have been linked to prolonged disease courses, calcinosis (calcium deposits in tissues), and ulcerative skin lesions. Additionally, IL-1 receptor antagonist polymorphisms may affect disease severity, further highlighting the role of genetic predisposition in JDM pathophysiology.

Clinical Features

Juvenile Dermatomyositis (JDM) is characterized by a combination of progressive muscle weakness and distinct dermatological manifestations. The onset is typically insidious, with systemic symptoms often appearing before noticeable muscle involvement.

Muscle Weakness and Functional Impairment

The primary clinical feature of JDM is progressive, symmetrical, bilateral muscle weakness, particularly affecting proximal muscle

groups, including the deltoids, quadriceps, pelvic girdle, and shoulder girdle. The severity varies, ranging from mild muscle pain to profound weakness. As the condition progresses, children experience difficulty performing daily tasks such as climbing stairs, combing hair, dressing, or rising from a seated position. A Gower's sign may be observed, where affected individuals use their hands to support themselves while rising from the floor. Pharyngeal and esophageal muscle involvement can lead to dysphagia, dysphonia, and dyspnea, with severe cases requiring intensive care management.

Dermatological Manifestations

JDM presents with characteristic skin findings, often appearing before muscle involvement.

- Heliotrope Rash: A purplish (violaceous) or dusky erythematous discoloration around the eyelids, sometimes accompanied by periorbital edema.
- Gottron's Papules and Plaques: Raised, violaceous, scaly papules occurring over bony prominences such as the metacarpophalangeal, proximal interphalangeal, and distal interphalangeal joints, as well as the elbows, knees, and ankles. These lesions typically spare the spaces between the joints.
- Facial and Photosensitive Rashes: A malar rash affecting the cheeks with sparing of the nasolabial folds, resembling the rash seen in systemic lupus erythematosus (SLE). Erythematous patches may be seen on the V-neck area, shoulders, and anterior chest wall.
- Periungual Changes: Capillary abnormalities in the nailfold region, including periungual erythema, telangiectasias, cuticular hypertrophy, and nailfold infarcts. Chronic nailfold changes may persist despite muscle improvement.
- Poikiloderma: A combination of hypo-pigmentation, hyperpigmentation, telangiectasia, and skin atrophy, commonly affecting sun-exposed areas.
- Mechanic's Hands: Hyperkeratosis and fissuring along the lateral and palmar aspects of the fingers, often associated with myositis-specific autoantibodies and interstitial lung disease.

Vascular and Systemic Manifestations

- Raynaud's Phenomenon: Vasomotor instability causing episodic pallor, cyanosis, and erythema in response to cold or stress.
- Livedo Reticularis: A net-like, violaceous discoloration of the skin due to impaired blood flow.
- Cutaneous and Mucosal Ulcerations: More severe disease may present with painful ulcerations resulting from complement deposition and microvascular injury.

Calcinosis Cutis

Calcinosis, a manifestation of dystrophic calcification, occurs in 20-40% of JDM cases, typically appearing within three years of diagnosis, though it may develop up to 20 years later. It presents as firm, white or flesh-colored subcutaneous nodules containing calcium hydroxyapatite deposits. Types of calcinosis include, Superficial calcareal masses, Deep calcareal masses, Linear deposits, Extensive subcutaneous encasement of the torso. The most commonly affected sites include elbows, knees, and extremities. Calcinosis may regress spontaneously or ulcerate, increasing the risk of secondary infections and functional impairment. Early intervention with high-dose corticosteroids and bisphosphonates may reduce its incidence.

Gastrointestinal Involvement

Juvenile dermatomyositis (JDM)-related vasculopathy can affect various organ systems, including the gastrointestinal tract, leading to abdominal pain and melena due to intestinal

vasculitis, and in rare, severe cases, pneumatosis intestinalis or colonic perforation.

Other systemic manifestations

Pulmonary and cardiovascular manifestations are also common, with interstitial lung disease often developing, typically associated with myositis-specific autoantibodies. Respiratory muscle weakness can result in hypoventilation and respiratory failure. Myocardial involvement may lead to ventricular arrhythmias, cardiomyopathy, and an increased risk of cerebrovascular events. Metabolic and lipodystrophic changes, such as lipodystrophy, can occur after prolonged disease progression, manifesting as fat loss in the upper trunk, sometimes accompanied by hirsutism, hepatomegaly, and acanthosis nigricans. These changes increase the risk of cardiovascular and metabolic comorbidities, including insulin resistance and dyslipidemia. Early systemic symptoms may include fatigue, asthenia, fever, weight loss, irritability, anorexia, and generalized pain or malaise, which often precede the muscle and skin manifestations and may persist throughout the disease course.

Diagnosis and Evaluation

The diagnosis of juvenile dermatomyositis (JDM) is based on a combination of clinical findings, laboratory tests, imaging studies, and histopathology. The primary manifestations include proximal muscle weakness, characteristic cutaneous signs (heliotrope rash, Gottron's papules), and elevated muscle enzyme levels. While the traditional Bohan and Peter criteria remain a reference, modern practice emphasizes imaging and autoantibody testing over routine electromyography (EMG) and muscle biopsy.

Laboratory Findings

Markers of inflammation and autoimmunity play a critical role in diagnosis. Erythrocyte sedimentation rate (ESR) is often elevated but is nonspecific. Muscle enzymes such as creatine kinase (CK), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), and aldolase may be elevated early in the disease course, though CK levels may remain normal in 10–40% of cases initially and normalize within months of disease onset. Antinuclear antibodies (ANA) are positive in approximately 50% of cases, but extractable nuclear antigens (SSA, SSB, Sm, RNP, DNA) are usually negative.[11]

Myositis-specific autoantibodies (MSA), found in about 10% of patients, correlate with distinct disease phenotypes:

- Aminoacyl-transfer RNA synthetases (e.g., Jo-1, PL-12, PL-7) are linked to a spring onset of myositis, interstitial lung disease, arthritis, Raynaud's phenomenon, and mechanic's hands.
- Anti–signal recognition particle (SRP) autoantibodies are associated with autumn onset, severe refractory myositis, and in adults, high mortality with cardiac involvement.
- Anti-Mi-2 antibodies are present in 5% of cases and indicate a milder disease course.
- Anti-p155/140 kDa autoantibodies are found in 29% of JDM patients and correlate with extensive cutaneous involvement, ulcerations, and edema.

Imaging Studies

Magnetic resonance imaging (MRI) is the preferred modality for detecting muscle edema and inflammation, particularly using T2-weighted fat suppression and STIR sequences. The degree of T2 hyperintensity correlates with disease severity and helps guide muscle biopsy selection. Ultrasound can show increased echogenicity and reduced bone surface echo, though it is less specific.

Electromyography (EMG)

EMG findings in JDM typically include short, small-amplitude motor unit action potentials, fibrillation potentials, and complex repetitive discharges, though 19% of cases may have normal EMG findings.

Muscle Biopsy and Histopathology

Muscle biopsy is reserved for ambiguous cases and demonstrates classic JDM features:

- Perifascicular atrophy, perivascular mononuclear infiltrates, and membrane attack complex deposition.
- Capillary damage with endothelial swelling, capillary dropout, and infarcts.
- Muscle fiber necrosis, regeneration, and connective tissue fibrosis.
- CD4+ T cells and B cells in perimysial and perivascular areas, differentiating JDM from polymyositis (which primarily has CD8+ T cell-mediated endomysial inflammation).

An international consensus scoring system has been developed to assess muscle biopsy findings in juvenile dermatomyositis, evaluating four key domains: inflammatory markers, including CD3+ and CD68+ infiltrates; vascular changes, such as capillary dropout, arterial abnormalities, and infarction; muscle fiber pathology, which includes MHC-I overexpression, perifascicular atrophy, and signs of muscle degeneration or regeneration; and fibrosis, with particular focus on endomysial and perimysial fibrosis. This scoring system is currently being studied for its prognostic value in understanding disease progression and its potential to guide clinical management.

Assessment scales

Clinical evaluation plays a crucial role in monitoring disease severity and response to treatment in juvenile dermatomyositis (JDM). Several standardized tools have been developed to assess different aspects of the disease. The Childhood Health Assessment Questionnaire (CHAQ) is a validated 30-item scale that evaluates functional ability across eight domains, including dressing, hygiene, and walking. The Childhood Myositis Assessment Scale (CMAS) is a 14-point scale that measures muscle strength, endurance, and functional capacity, with tasks such as timed neck flexion and sit-ups. The Disease Activity Scale assesses muscle and skin involvement, while the Cutaneous Assessment Tool (CAT) is a 21-item test validated for evaluating cutaneous disease severity and damage in JDM. Two major international groups, the Paediatric Rheumatology International Trials Organization (PRINTO) and the International Myositis Assessment and Clinical Studies Group (IMACS), have developed core assessment tools that include physician and patient global assessments, functional ability scores (CHAQ, CMAS), laboratory markers (CK, LDH, ESR), as well as the Myositis Disease Activity Assessment Tool (MDAAT) and Myositis Damage Index (MDI) to provide a comprehensive evaluation of disease activity and damage.[12]

Treatment and Management

Systemic Therapy

Corticosteroids remain the mainstay of treatment, with oral prednisone recommended at 1–2 mg/kg/day. Intravenous methylprednisolone (30 mg/kg/day, max 1 g/day for 3 days) is preferred in severe cases, particularly when gastrointestinal involvement leads to malabsorption due to vasculitis. Long-term corticosteroid therapy is required for remission, necessitating gradual tapering to minimize side effects. Adjunctive calcium and vitamin D supplementation is essential to mitigate the risk of osteoporosis.

For steroid-resistant or severe cases, methotrexate (10–20 mg/m^2/week, oral or subcutaneous) with folic acid (1 mg/day) is widely accepted as a steroid-sparing agent. It may be introduced upfront in moderate-to-severe weakness or vasculopathy, or within six weeks if corticosteroids fail.

Other immunosuppressive agents include:

- Cyclosporine A, either alone or combined with methotrexate, as an alternative steroid-sparing agent.

- Intravenous immunoglobulin (IVIG) (1–2 g/kg monthly for six months) for steroid-resistant or steroid-dependent cases.
- Cyclophosphamide (0.5–1 g/m^2 monthly) for severe complications like ulcerations or respiratory disease.
- Azathioprine and mycophenolate mofetil as additional second-line agents.

Biologic therapies are emerging options for refractory cases:

- Anti-TNF agents (Infliximab, Etanercept)—mixed results in clinical trials.
- Rituximab (anti-CD20 monoclonal antibody)—shows promise but requires further validation.

Management of Skin Disease

- Photosensitive rashes worsen with UV exposure, requiring sun protection (UVA/UVB sunscreen, avoidance of direct sun). Treatment includes:
- Topical corticosteroids for localized lesions.
- Hydroxychloroquine (5–7 mg/kg/day), though its role is still under study.
- Topical tacrolimus (0.1%), now declining in use.
- Methotrexate, which also benefits skin manifestations.

Calcinosis treatment

- Diltiazem (240–480 mg/day or 3–6 mg/kg/day)—variable response.
- Bisphosphonates, aluminum hydroxide, probenecid, and corticosteroid injections—limited success.
- Surgical excision—reserved for significant pain, disfigurement, or impaired function, though it risks recurrence and complications.

Rehabilitation and Supportive Care

- Physical therapy should begin once symptoms are controlled to restore muscle strength, endurance, and prevent contractures.
- Passive range of motion exercises during active disease.
- Isometric and isotonic exercises once stabilized.
- Intensive resistance training may be safe and beneficial.
- For dysphagia and dysphonia, referrals are necessary:
- Speech therapy for voice and swallowing issues.
- Occupational therapy for food consistency modification and proper positioning.

Multidisciplinary Approach and Long-Term Monitoring

- Regular follow-up every 3–6 months to monitor muscle strength and enzyme levels.
- Referral to tertiary care centers for specialized management.
- Long-term steroid tapering with immunosuppressants reduces complications.
- Remission is defined as normalized muscle function and enzymes for six months.

Differential diagnosis

The differential diagnosis of juvenile dermatomyositis (JDM) includes various inflammatory, infectious, metabolic, and genetic conditions that present with muscle weakness, skin manifestations, or systemic involvement. Polymyositis and other idiopathic inflammatory myopathies (IIMs), such as overlap myositis, necrotizing myopathy, and antisynthetase syndrome, share features with JDM but often lack its characteristic rash. Infectious myositis,

caused by viral (e.g., influenza, enteroviruses), bacterial, or parasitic infections, can present with acute muscle weakness and elevated muscle enzymes. Metabolic myopathies, including mitochondrial disorders, glycogen storage diseases (e.g., McArdle disease), and lipid metabolism defects, typically manifest with exercise intolerance, myoglobinuria, or progressive weakness. Dystrophinopathies (Duchenne and Becker muscular dystrophy) cause progressive proximal muscle weakness but lack inflammatory markers. Congenital myopathies and myasthenia gravis should be considered in cases with early-onset weakness or fluctuating symptoms. Systemic lupus erythematosus (SLE) and scleroderma can mimic JDM with vasculopathy and skin involvement, while vasculitic syndromes (e.g., Kawasaki disease, polyarteritis nodosa) may present with systemic inflammation and cutaneous findings. Drug-induced myopathy (e.g., due to statins, corticosteroids, hydroxychloroquine) and hypothyroid myopathy should also be excluded.

Prognosis

The prognosis of juvenile dermatomyositis (JDM) is highly variable, with disease progression following a monocyclic, polycyclic, or chronic course. In recent years, advancements in therapeutic strategies have significantly improved outcomes, with early intervention and close monitoring playing a crucial role in disease control. However, in developing countries, mortality rates remain high due to limited healthcare access. The major causes of death include severe muscle weakness, superadded infections, gastrointestinal vasculitis with a risk of bowel perforation, myocardial failure, and respiratory distress. One of the most debilitating complications is calcinosis cutis, which affects approximately 30% of patients and contributes to significant morbidity. In addition to poor cosmesis, calcinosis can cause pain, limit mobility, and lead to contractures, ulcerations, and nerve entrapment. Historically, before the advent of corticosteroids, one-third of patients experienced spontaneous remission, one-third developed a chronic course, and one-third succumbed to the disease. Unlike the adult form of dermatomyositis, JDM is not typically associated with malignancies.

Complications

Potential complications of JDM include dysphagia, which results from esophageal muscle involvement and can lead to weight loss and malnutrition. Swallowing difficulties may also cause aspiration pneumonia, increasing the risk of respiratory infections. Respiratory compromise can occur if the disease affects chest or thoracic muscles, leading to dyspnea and respiratory distress. Additionally, as the disease progresses, calcium deposits may develop in the muscles, connective tissues, and skin, further contributing to functional limitations and long-term disability.

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FAMILY MEET

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ORGAN AND BLOOD DONATION

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REPUBLIC DAY

IAP Kerala

IAP Thiruvananthapuram - National Girl Child Day

IAP Kottayam - Charity day/Blood donation day

IAP Pathanamthitta -
CUTIS: Skin module national TOT

IAP Cochin - Awareness on Blood Donation

IAP Kerala

IAP Thrissur - Teachers training program on development assessment in children

IAP Madhya Kerala - National Girl Child Day

IAP Wayanad - : Installation ceremony/GB meeting/ Family get-together

IAP Palakkad - Monthly CME at KPM Regency on Jan 24th 2025

IAP Kerala

IAP Malappuram - Class on 'Lifestyle diseases in children' for High School students Vettathur, Malappuram by Dr Mohammed Shafeeq, Assistant Professor, MES Perinthalmanna.

IAP Vadakara - Free Eye Screening camp for teachers and children & Health Awareness talk

IAP Kozhikode - Training for RBSK Nurses

IAP Kozhikode - IGNITE - PAP 2025, BLS Training