5th National Conference of HAE Society of India (HAESICON)

21st - 22nd February 2026

Sheraton Grand Bangalore Hotel at Brigade Gateway, Bengaluru, India

3 Hrs
CME Credits by Karnataka Medical Council

Organized by **Hereditary Angioedema Society of India (HAESI)** in collaboration with **Bangalore Dermatological Society (BDS)**, **Indian Rheumatology Association Karnataka Chapter**, and in association with **Manipal Hospital, Old Airport Road, Bengaluru**

www.haesi.in haesocietyofindia@gmail.com

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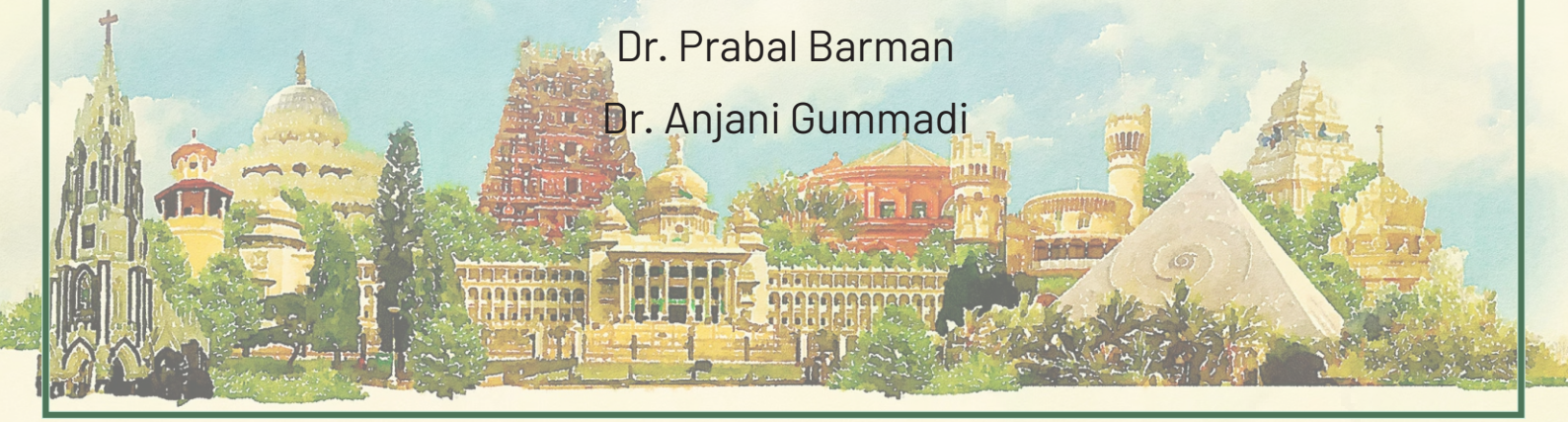

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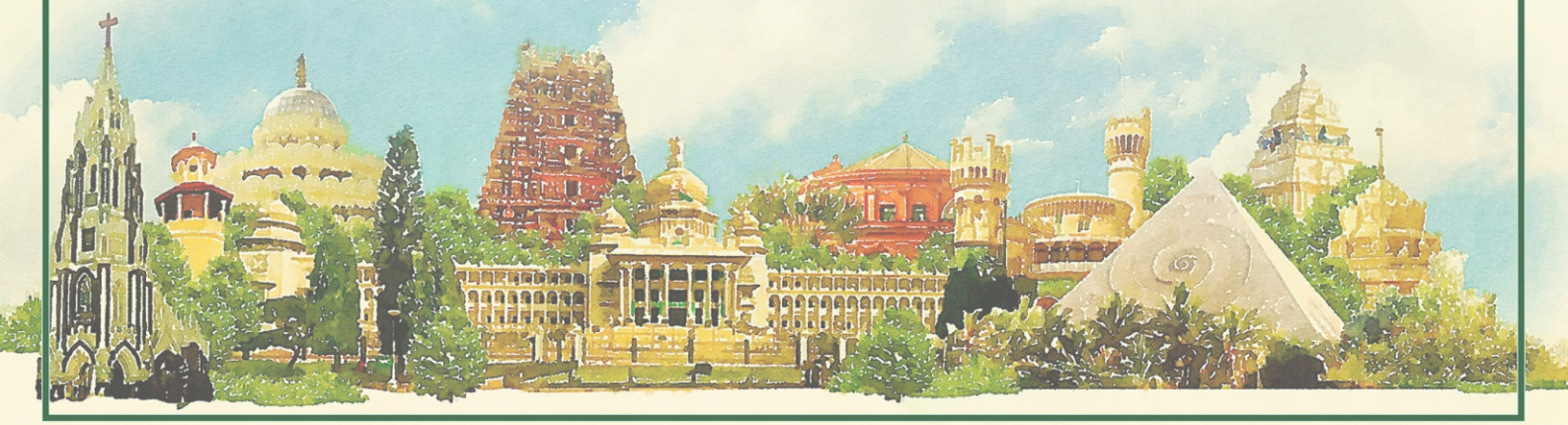

INVITATION

Greetings from the Hereditary Angioedema Society of India (HAESI)!!

HAESI is pleased to invite you to participate in the **5th National Conference of the HAE Society of India (HAESICON 2026)**, scheduled to be held on **21st –22nd February 2026** at **Sheraton Grand Bangalore Hotel, Brigade Gateway, Bengaluru.**

We warmly welcome **residents, research fellows, consultants, and practising clinicians** with a special interest in Urticaria, Angioedema and Hereditary Angioedema to join us as delegates.

HAESICON 2026 continues HAESI's commitment to advancing knowledge, improving clinical practice, and fostering collaboration in the fields of Angioedema, Urticaria, and Hereditary Angioedema (HAE). This conference brings together national and international experts, clinicians, researchers, and trainees to share the latest scientific developments, practical insights, and real-world experiences.

We are honoured to host distinguished international faculty and eminent national experts, and we look forward to vibrant academic discussions, networking opportunities, and collaborative learning over the two days.

We look forward to welcoming you to **Bengaluru, India**, for an enriching scientific and academic experience.

Best Wishes

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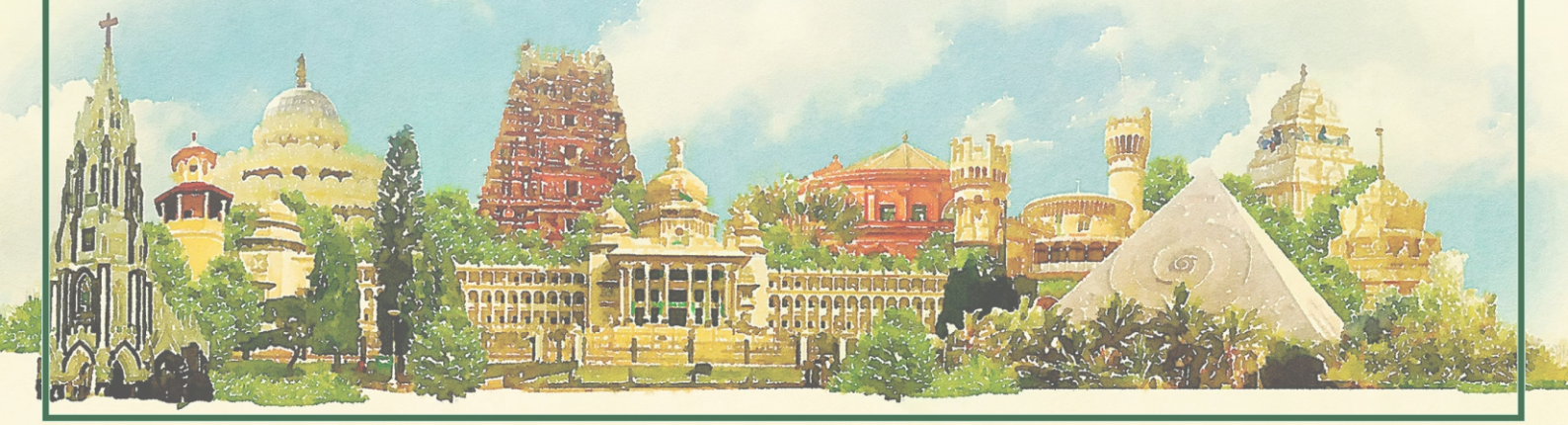
MEET THE FACULTY



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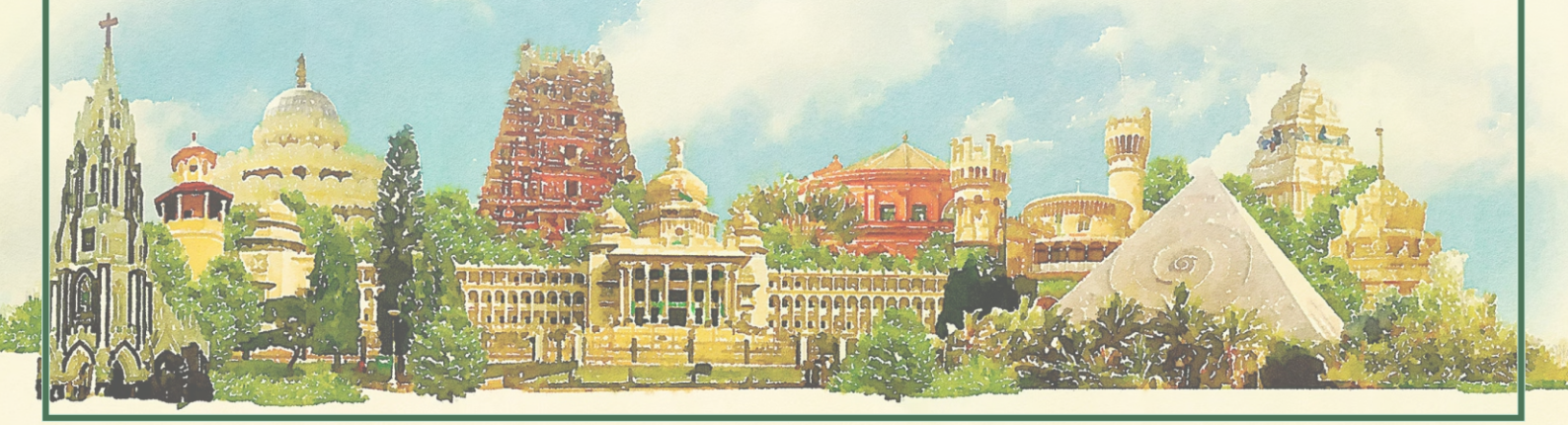

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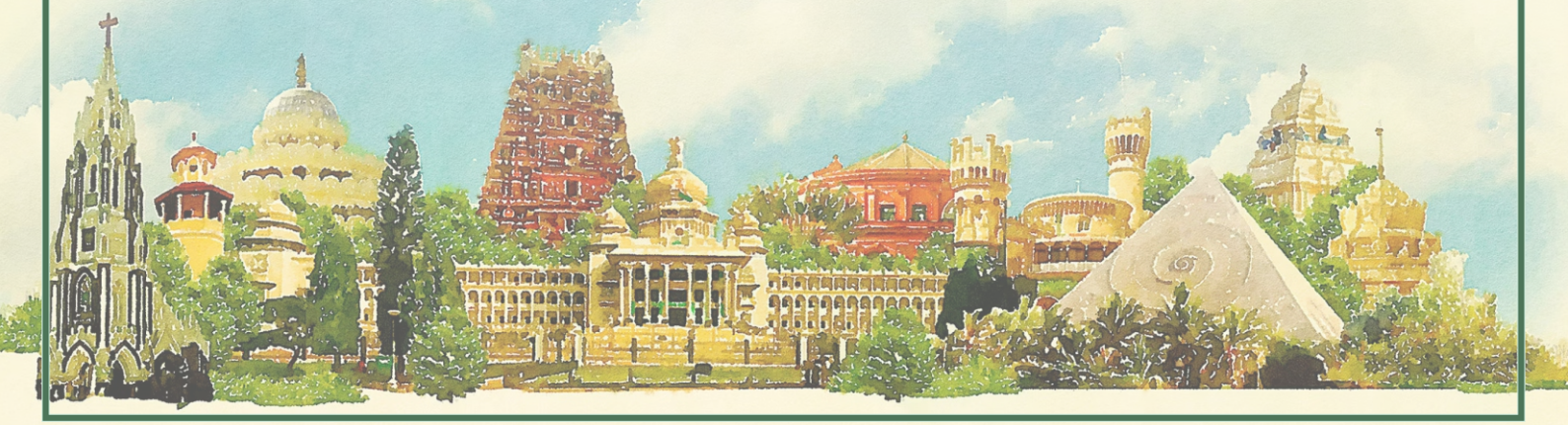
SCIENTIFIC PROGRAMME



Scientific Programme

DAY 1: February 21, 2026 (Saturday)

Time	Title	Speaker	Chairperson
13:00 – 14:00	**Registration and lunch**		
14:00 – 14:15	**Introduction and Welcome**	Sunil Dogra M Sendhil Kumaran	
14:15 – 15:15	**Session 1: Clinical manifestations and diagnosis of angioedema**		
14:15 – 14:40	**An update on clinical manifestations and diagnosis of Hereditary Angioedema**	Anjani G	Praveen Kumar Chandrika Bhat
14:45 – 15:10	**An update on clinical manifestations and diagnosis of mast cell mediator mediated angioedema and urticaria**	Ankur Jindal	
15:15 – 15:35	**Coffee Break**		
	Session 2: HAE in the Asia Pacific		
15:35 – 16:00	**HAE in the Asia Pacific region: where do we stand in Circa 2026**	Philip Li	Vineeta Shobha Dharshini Sathishkumar
	Session 3: Case discussions		
16:00 – 16:25	**Challenging cases (Urticaria and Angioedema): 5 presentations, 5 minutes each**		Priyanshu Mathur Divya Gupta
	Silent for Decades, Malignant in the End: Hepatocellular Carcinoma After 27 Years of Danazol in Hereditary Angioedema	Dushyanth Patel	
	When Wheals Refuse to Settle: A Case of Refractory Chronic Spontaneous Urticaria	Sowbarnika Subramanian	
	The Silent Swelling: Hereditary Angioedema Unmasked by Recurrent Abdominal Pain	Reetika Ramanathan	
	RES IPSA LOQUITUR – THE GENEALOGY CHART	Riya Bijoy	
	Not All Angioedema Is Histaminergic: A Case of Steroid-Refractory Facial Swelling	Mahendra Kumar	
16:30 – 17:10	**How will you approach?** **Grand Rounds and Case Discussions:**	**Presenters:** Rachna Shanbag Mohite Vinay K **Experts:** Rashmi Sarkar Balachandra BV Ravishankar Marpalli Srikanta Basu Indrasis Poddar AS Sanjana	KM Mahendranath Prabhakar Sangolli

Scientific Programme

DAY 1: February 21, 2026 (Saturday)

Time	Title	Speaker	Chairperson
17:10 – 17:40	**Patient support groups for HAE**	Fanny Schappler Pravalika M	Prasanna Shiroi Fiona Wardman
17:40 - 17:55	**Acquired and drug induced angioedema: When to suspect and how to evaluate?**	Abhishek Patil	Roohi Rasool Sujala Sacchidanand
18:00 – 19:00	**Poster walk**	**Moderated by** Henriette Farkas Sendhil M Kumaran Vineeta Shobha Daria Fomina Philip Li Rachna Shanbag Mohite	
Poster 1	**Steroid-Dependent Angioedema in Juvenile SLE: An Early Warning Sign of Disease Reactivation and its therapeutic challenges.**	Nagashri BS	
Poster 2	**Bradykinin-Mediated Angioedema With Normal Complement Levels: A Diagnostic Challenge**	Gurrevala Sharvani	
Poster 3	**REFRACTORY URTICARIAL VASCULITIS: OVERCOMING MULTIMODAL TREATMENT FAILURE WITH TNF APLHA INHIBITION**	Goolla Akhila	
Poster 4	**Urticarial Vasculitis with Angioedema in a one year old: A Rare Pediatric case report**	Priya Kamboj	
Poster 5	**Endotype Distribution and Therapeutic Stratification in Chronic Spontaneous Urticaria with Angioedema (CSU): A Real-World Analysis of 52 patients**	Goolla Akhila	
Poster 6	**Case series on chronic urticaria in children- diagnostic and therapeutic challenges**	Revathy S	
Poster 7	**Urticaria as a Window into Inborn Errors of Immunity**	Neha Singh	
Poster 8	**Autosomal Dominant Hereditary Angioedema in Two Families: A Genetically Confirmed Case Series of Six Patients**	Sowmya Nagarajan	

Scientific Programme

DAY 1: February 21, 2026 (Saturday)

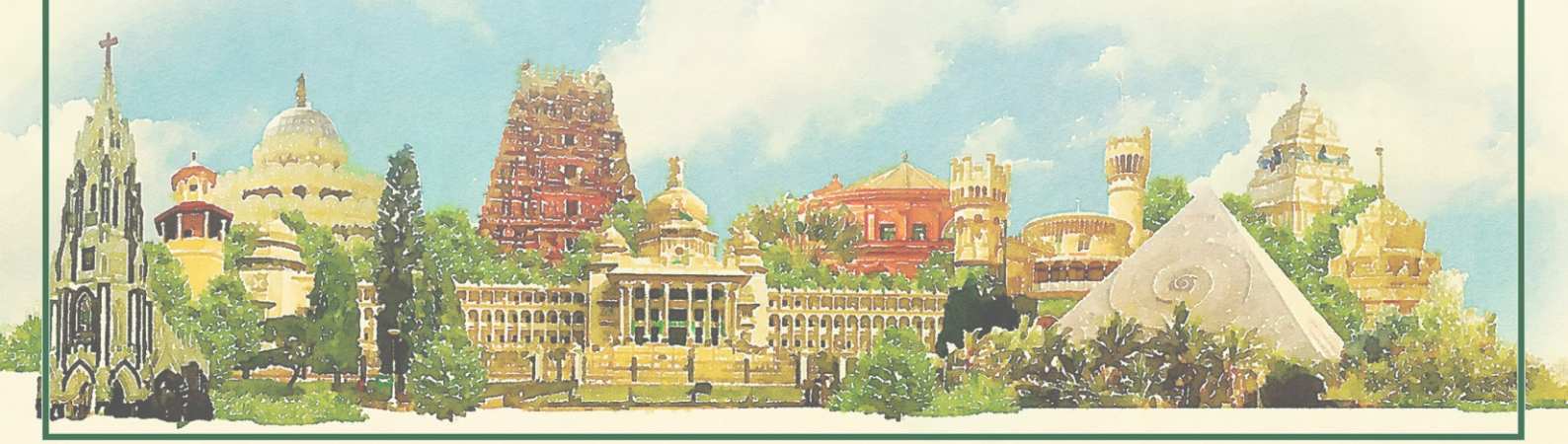
Time	Title	Speaker	Chairperson
Poster 9	**Clinical Profile and Outcomes of Hereditary Angioedema in North East India: A Retrospective Study.**	Pratap Patra	
Poster 10	**Profile of 38 patients with chronic spontaneous urticaria from a tertiary care centre in Eastern India**	Bharat Kumar	
Poster 11	**Clinical Profile, management and outcome of patients with hereditary angioedema type 1: our experience from a single centre in South India.**	Uma Tejaswi	
Poster 12	**Autoimmune diseases and autoantibodies in patients with Hereditary Angioedema: A Preliminary study.**	Navjot Kaur	
Poster 13	**ANGIOEDEMA : A MYSTERY BOX**	Smitha J N Singh	
Poster 14	**Recurrent Angioedema Without Urticaria: A Case of Hereditary Angioedema Type I**	Sannitha S Kapatral	

Gala Dinner on February 21, 2026 (Saturday): 19:00 onwards

Scientific Programme

DAY 2: February 22, 2026 (Sunday)

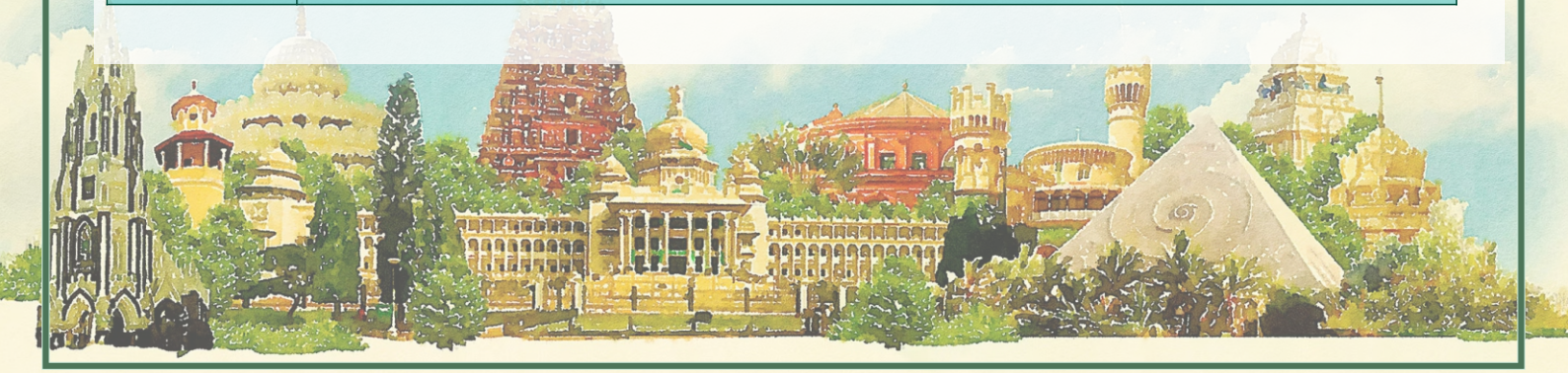
Time	Title	Speaker	Chairperson
08:30 – 09:00	**Session 4: Tug of War**		
	Debate: **Genetic testing and antenatal screening in HAE are a 'must'** **Genetic testing and antenatal screening in HAE are 'optional'**	Kunal Chandwar Chengapppa KG	Ashwin Dalal Sathish Loganathan
09:00 – 09:30	**Session 5: Award Paper presentations**		
	Genetic profile and genotype-phenotype correlation in a single centre cohort of patients with hereditary angioedema from India	Sanghamitra Machhua	BG Dharmanand Keerthi Vardhan Pratap Patra
	Clinical Profile Of Patients With Hereditary Angioedema From A Tertiary Care Centre In South India	Anunya Augustine	
	Interplay Of FCεRIα Genetics And Basophil Activation Driven Immune Dysregulation In Chronic Urticaria	Ayaz Gull	
	Session 6: Oration		
09:30 – 10:30	**4th Dr. Hilary Longhurst Oration** **Title: My experience with normal C1-INH HAE at Moscow, Russia**	Daria Fomina	M Sendhil Kumaran Ankur Jindal
10:30 – 11:00	**High Tea**		
	Session 7: Management of Urticaria and Angioedema		
11:00 - 11:20	**Debate:** **Icatibant vs plasma derived C1-inhibitor for on demand treatment of acute attacks**	Archan Sil Sagar Bhattad	Karthik Nagesh Chetan Ginigeri Manoj Parida
11:20 – 11:30	**An update on 'Rare disease policy of the Government of India' for patients with hereditary angioedema**	Suprit Basu	
11:30 – 12:10	**Discussion on Indian Guidelines for Diagnosis and Management of HAE**	Ankur Jindal Samipa Mukherjee Archan Sil Rachna Shanbag Mohite	



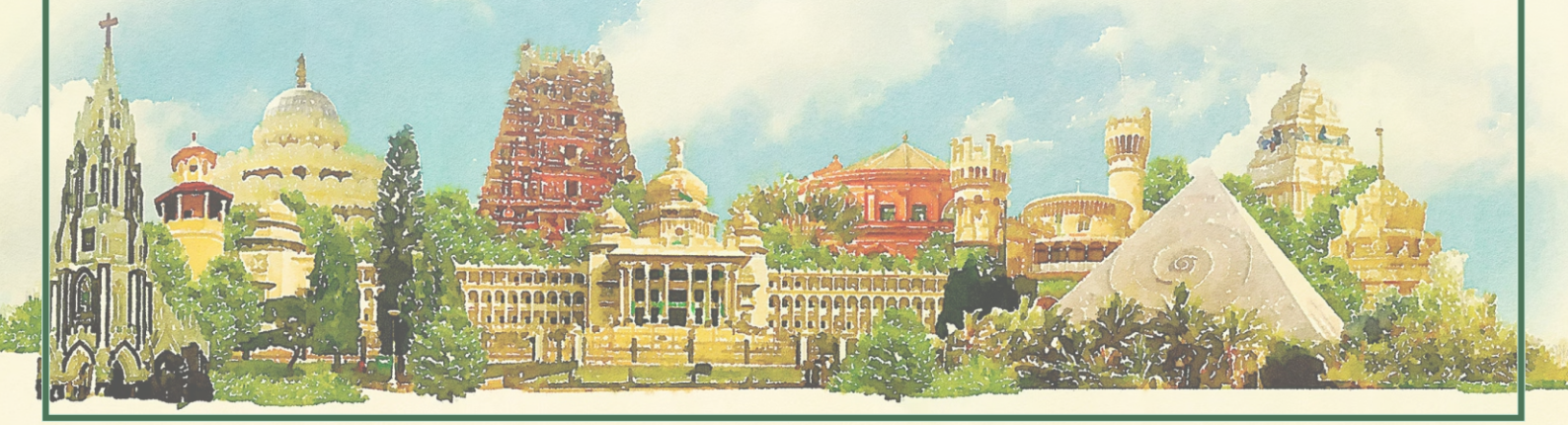
Scientific Programme

DAY 2: February 22, 2026 (Sunday)

Time	Title	Speaker	Chairperson
12:10 – 12:25	**New era in the management of urticaria**	Kiran Godse	M Sendhil Kumaran Neeraj Gupta
12:25 – 12:55	**New era in the acute treatment of HAE**	Henriette Farkas	
12:55 – 13:15	**How I manage mast cell mediator mediated angioedema and chronic spontaneous urticaria? (A real-life experience)**	Payal Chauhan Shehanaaz Begum	Neha Singh Harshini AS
13:15 – 14:15	**Lunch**		
	Session 8: Short talks		
14:15 – 15:15	**Short talks** 1. Angioedema mimics 2. Urticaria and angioedema- Do's & Don'ts 3. Special situations in HAE- Pregnancy and Procedures 4. Breaking the News- Counselling in HAE 5. Pediatric HAE: Special Considerations 6. Future directions for HAE in India	Samipa Mukherjee Hitaishi Mehta Soumya Narula Philip Li Henriette Farkas Ankur Jindal	Jagdish Chinappa Bhaskar Shenoy Murugesh Shamanur
15:15 - 16:00	**Open House (Urticaria, Angioedema and Hereditary Angioedema)**	**Moderator:** Sunil Dogra M Sendhil Kumaran **Panellists:** Henriette Farkas Daria Fomina Ankur Jindal Philip Li Kiran Godse	
16:00 - 16:15	**Announcement of results, prize distribution and valedictory**	Sunil Dogra M Sendhil Kumaran	
16:15 onwards	**High Tea**		



CHALLENGING CASES



CC1: Silent for Decades, Malignant in the End: Hepatocellular Carcinoma After 27 Years of Danazol in Hereditary Angioedema

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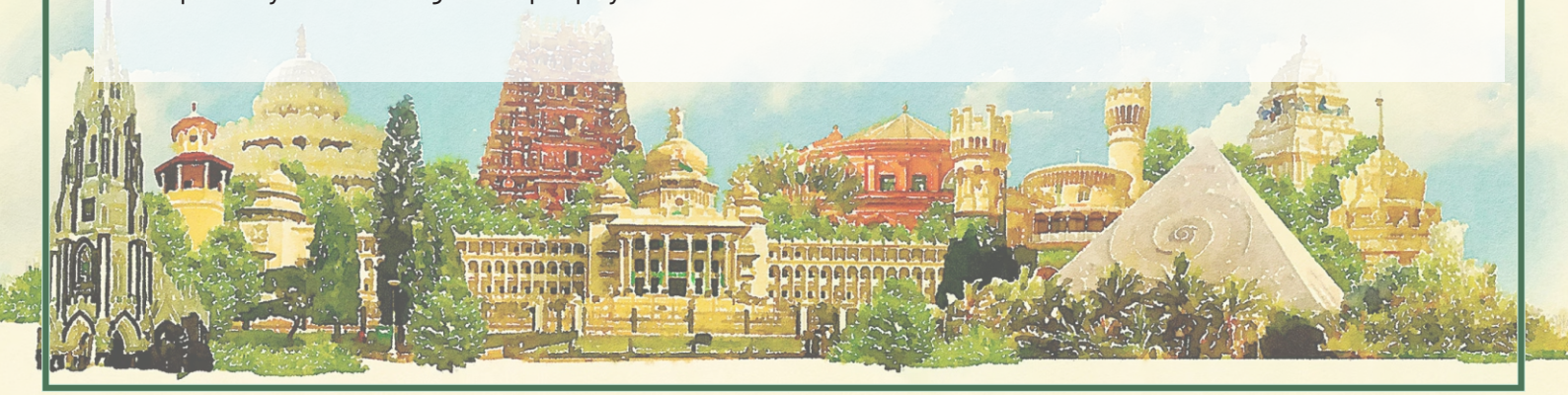
ABSTRACT

Background: Hereditary angioedema (HAE) is caused by C1-esterase inhibitor deficiency, leading to recurrent episodes of subcutaneous and/or submucosal swelling. Danazol, an attenuated androgen, is used for long-term prophylaxis in patients with HAE, especially when none of the first line treatment options are available. However, prolonged danazol use is associated with hepatic toxicity, including adenomas and, rarely, hepatocellular carcinoma (HCC), usually after many years of therapy. We report one such case.

Case Presentation: A 53-year-old male with type 1 HAE diagnosed at 28 years of age. His C4 was 0.24 g/L, C1-esterase inhibitor was 0.22 g/L, and WES showed *SERPING1* gene mutation. He was treated with danazol 200 mg/day since 1998. He had complete control of his disease and the dose reduced to 100 mg/day from 2020 onwards.
In 2025, he was incidentally found to have an abdominal mass. There was no history of viral hepatitis, significant alcohol use, or other risk factors for chronic liver disease.
CT Abdomen revealed a large left-lobe liver mass measuring 9.2x12.7x13.4 cm arising from left lobe of liver (segments II, III, IVB), corresponding to an FDG-avid subcapsular mass on PET-CT, with maximal dimension 11.5 × 10.9 × 14.5 cm. The mass was excised and biopsy confirmed moderately differentiated (G2) conventional-type HCC; PIVKA-II was markedly elevated (6000), and the liver was non-cirrhotic. Danazol was discontinued and he was initiated on tranexamic acid (1500 mg per day) and was also advised to use plasma-derived C1 inhibitor concentrate as on-demand treatment.

Conclusion: This case highlights the risk of hepatic complications including HCC after prolonged danazol use in patients with HAE. This may develop despite preserved liver function and absence of other major risk factors. The long duration and high cumulative androgen exposure in this patient suggest a cumulative dose–dependent hepatic toxicity that may have contributed to malignant transformation.
It is important to do a serial monitoring for these complications in patients with HAE who are on long-term androgen therapy. It is also important to minimize the use of attenuated androgens and there is an urgent need to bring first line treatment options for HAE in India especially for the long-term prophylaxis.



CC2: When Wheals Refuse to Settle: A Case of Refractory Chronic Spontaneous Urticaria

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ABSTRACT

Background: Chronic spontaneous urticaria (CSU) is a mast cell–driven disease characterized by a relapsing–remitting course and at times refractory to treatment. Most patients demonstrate either type 1 or type 2b autoimmune mechanisms, while a subset exhibit features of both. CSU associated with type 2b autoimmunity often fail to respond to conventional treatment. We report one such case.

Case Description: A 17-year-old girl presented with a 9-month history of wheals (painful, pruritic) involving trunk and extremities with recurrent angioedema of the eyelids, lips, hands, and feet. Wheals persisted for more than 24 hours and healed with hyperpigmentation. She also had a history of allergic rhinitis and conjunctivitis. Before referring to us, she was managed elsewhere as CSU with angioedema, given antihistamines (bilastine 80 mg/day, desloratidine 10mg/day), montelukast, hydroxychloroquine, omalizumab (300 mg once fortnightly) and prednisolone (tapering doses).

On examination, she had multiple urticarial wheals over the trunk and extremities (Figure-1), with residual hyperpigmentation (Figure-2) and angioedema involving upper lip (Figure-3). Rest of the examination was unremarkable.

Laboratory investigations are mentioned in Table 1.

A clinical possibility of refractory CSU with angioedema with type 1 and 2b autoimmunity was considered. She was initiated on cyclosporine 150 mg/day (later increased to 250 mg per day), bilastine (80 mg per day) and ranitidine. Prednisolone was tapered and stopped. She showed some improvement, however in view of persistent rashes, she was started on omalizumab 300 mg s.c every 4 weeks.

Despite treatment, she had persistent rashes, itching and occasional pain in the limbs. Hence, she was given first dose of dupilumab 600 mg s.c. recently. The response to dupilumab is yet to be assessed.

Conclusion: Endotyping of CSU is important, as patients with combined type 1 and 2b autoimmune endotype are refractory to omalizumab and cyclosporine; dupilumab may be a suitable therapeutic option.

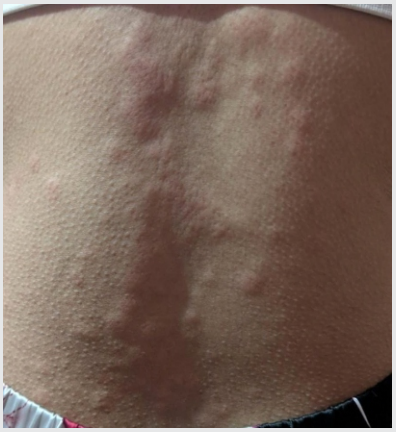


Figure - 1

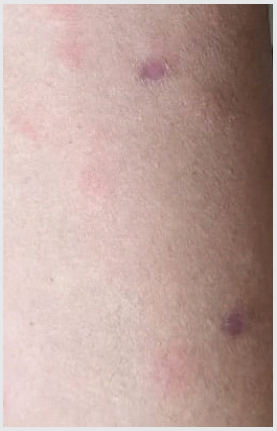


Figure – 2

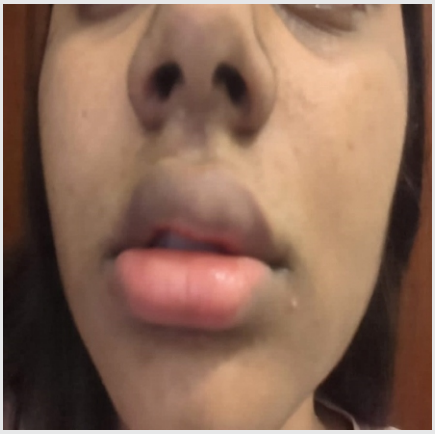
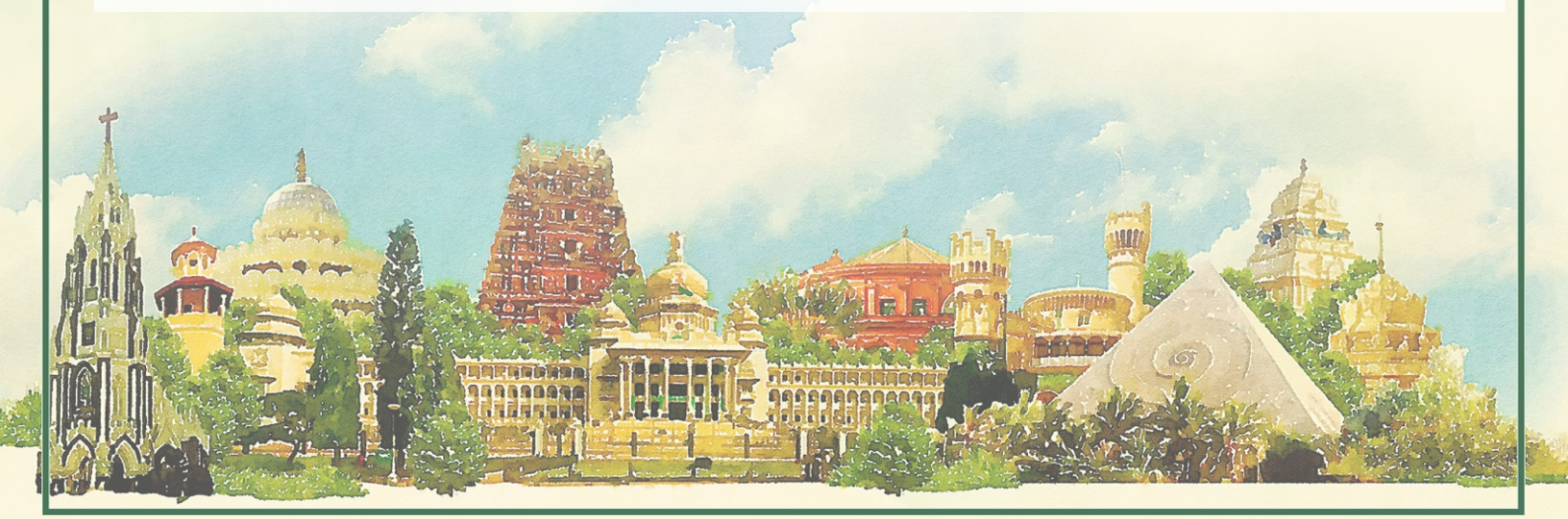


Figure - 3

Table 1: Laboratory Investigations of the index case

Parameter (Unit)	Result	Reference Range
Erythrocyte Sedimentation Rate (mm/hr)	17	0 – 20
C-Reactive protein (mg/dL)	1.35	< 0.5
Absolute Eosinophil Count (AEC, cells/µL)	86	50 – 500
Skin Biopsy	Normal	Normal
Total Serum IgE (IU/mL)	2328	< 100
Anti-ds DNA	Negative	Negative
ANA Profile - nRNP/Sm	Positive (+++)	Negative
Anti-Thyroglobulin (IU/mL)	47.46	< 4.0
Anti-Thyroid Peroxidase (IU/mL)	128.77	< 9.0
Thyroid stimulating hormone (µIU/mL)	3.019	0.5 – 5.0



CC3:The Silent Swelling: Hereditary Angioedema Unmasked by Recurrent Abdominal Pain

AUTHORS:

Dr. Reetika Rama Nathan, Dr. Abhishek Patil

AFFILIATIONS:

Department of Rheumatology, Manipal Hospitals HAL, Bangalore

ABSTRACT

Background: Hereditary angioedema (HAE) is a rare autosomal dominant disorder caused by C1-INH gene mutations. While cutaneous and laryngeal edema are classical manifestations, rarely gastrointestinal involvement can be the sole presentation, often leading to delay in diagnosis and treatment.

Case Description: We report a 20-year-old male who presented with recurrent episodic abdominal pain and vomiting since childhood, with a noticeable increase in frequency over the preceding two years. The episodes were crampy, periumbilical, self-limiting, and occasionally associated with intermittent wrist swelling. There was no history of urticaria, fever, altered bowel habits, or prior abdominal surgeries.

Initial evaluation revealed a weakly positive ANA by immunofluorescence (1:80), low serum C4 levels (0.09 g/L), and normal C3 levels. Upper gastrointestinal endoscopy and colonoscopy were unremarkable, and fecal calprotectin was within normal limits. Repeated contrast-enhanced CT scans of the abdomen demonstrated segmental jejunal wall thickening with mesenteric edema, prominent vasa recta, and mild ascites. Rectal biopsy showed nonspecific findings.

A detailed family history revealed similar recurrent abdominal symptoms in the patient's brother and a maternal history suggestive of fatal laryngeal edema. Subsequent evaluation showed markedly reduced C1 esterase inhibitor levels, confirming the diagnosis of HAE with gastrointestinal involvement.

Conclusion: Recurrent, self-limiting abdominal pain with transient bowel wall edema and normal inflammatory markers should prompt evaluation for HAE. Low serum C4 can serve as a simple and effective diagnostic clue. Early recognition is crucial to avoid unnecessary interventions and prevent potentially fatal complications such as laryngeal edema.

References:

1. Marrawani M, Alatawneh M, Asafra F, Salloum OH, Abuayash AM, Samamra M. Hereditary angioedema presented as isolated ascending and transverse colon swelling mimicking acute abdomen. SAGE Open Med Case Rep. 2024;12:2050313X241272574.
2. Cao Y, Liu S, Zhi Y. Recurrent and acute abdominal pain as the main clinical manifestation in patients with hereditary angioedema. Allergy Asthma Proc 2021; 42: 131–135.

CC4: RES IPSA LOQUITUR – THE GENEALOGY CHART

AUTHORS:

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AFFILIATIONS:

Department of Pediatrics, Christian Medical College, Vellore

ABSTRACT

Case Description: Mrs. R, a 27-year-old female presented with recurrent diffuse abdominal pain over 5 years, occurring every 1–2 weeks. The pain was moderate, and cramping, associated with non-bilious vomiting, with no relation to meals, and no weight loss, no vaginal discharge, TB contact, or bowel/urinary symptoms. It only subsided after receiving intravenous analgesics from the local clinic. Past and menstrual history were unremarkable. The general and systemic examination was non-contributory. She was been extensively evaluated with several ultrasonogram, endoscopies, and blood investigations, urinalysis, at several occasions with following differentials: Peptic ulcer disease; Recurrent urinary tract infection; Ureteric calculi; IBD; Diverticulitis; Abdominal TB; Porphyria; Pelvic inflammatory disease; Heavy metal toxicity.

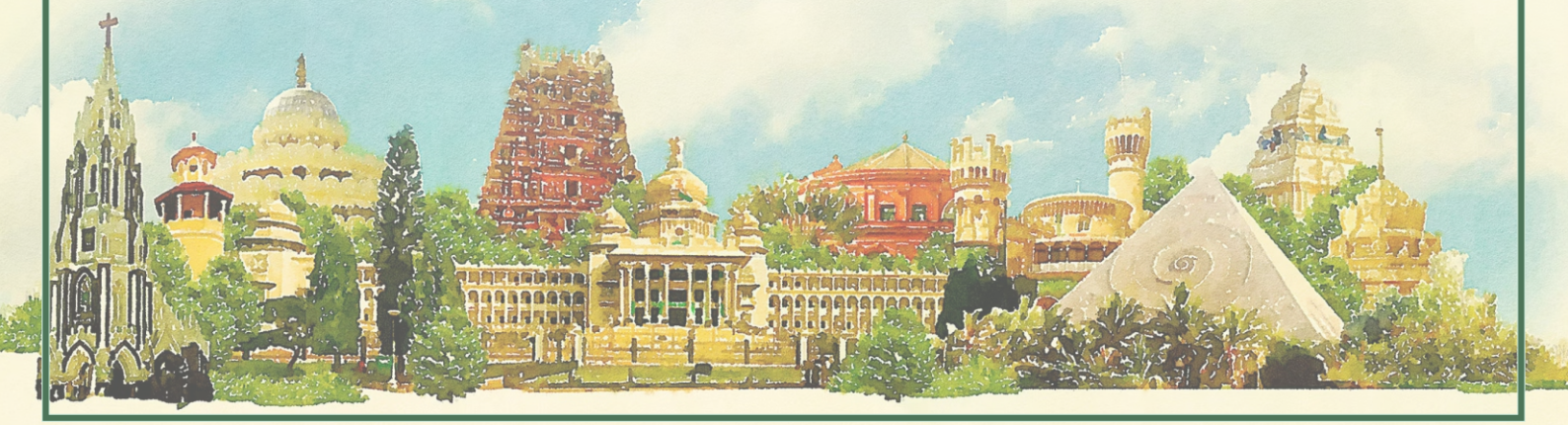
The missed lead and Diagnostic Clinch

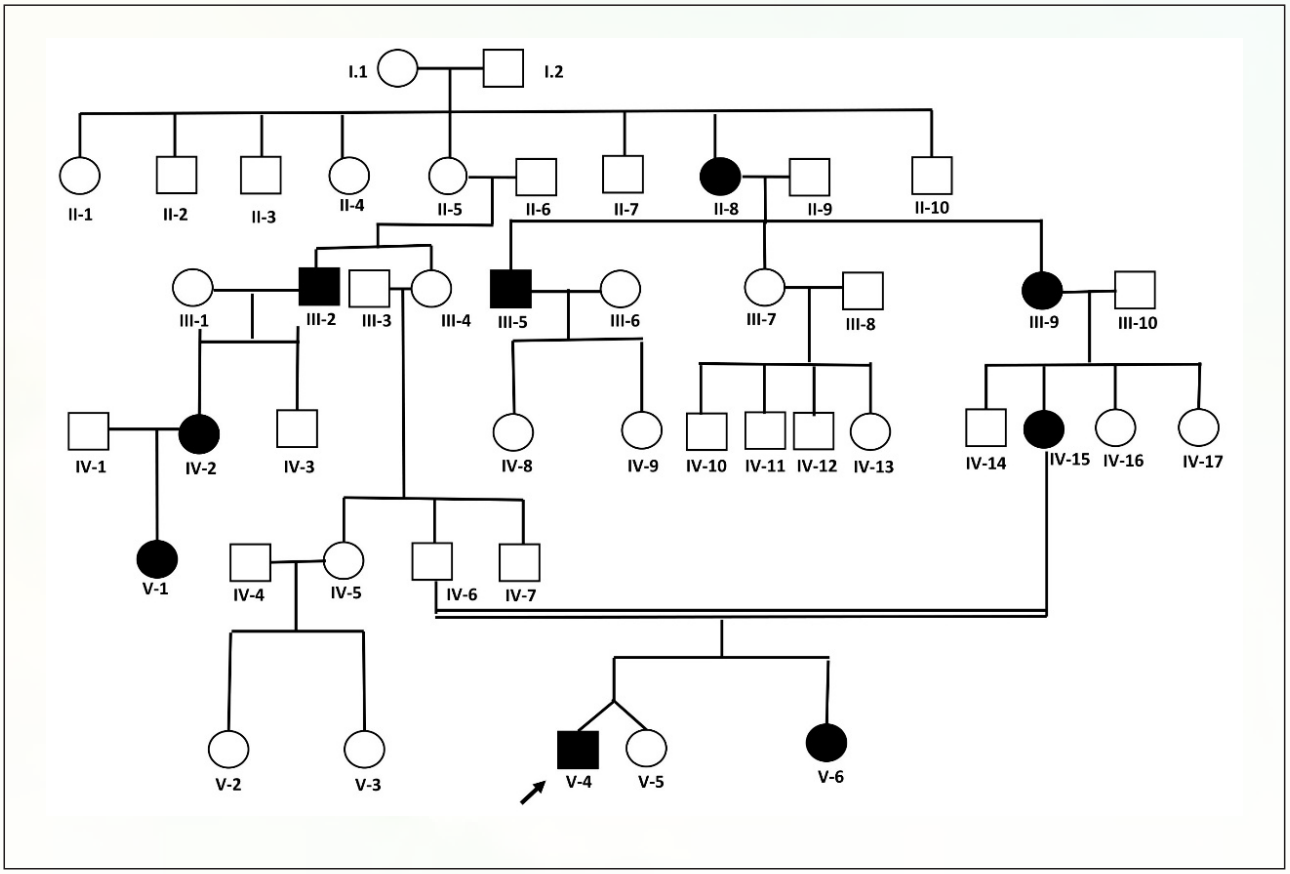
The patient's 5 year and 6 month-old son had

Two episodes of recurrent facial and upper airway swelling, and was diagnosed with Hereditary angioedema(HAE).

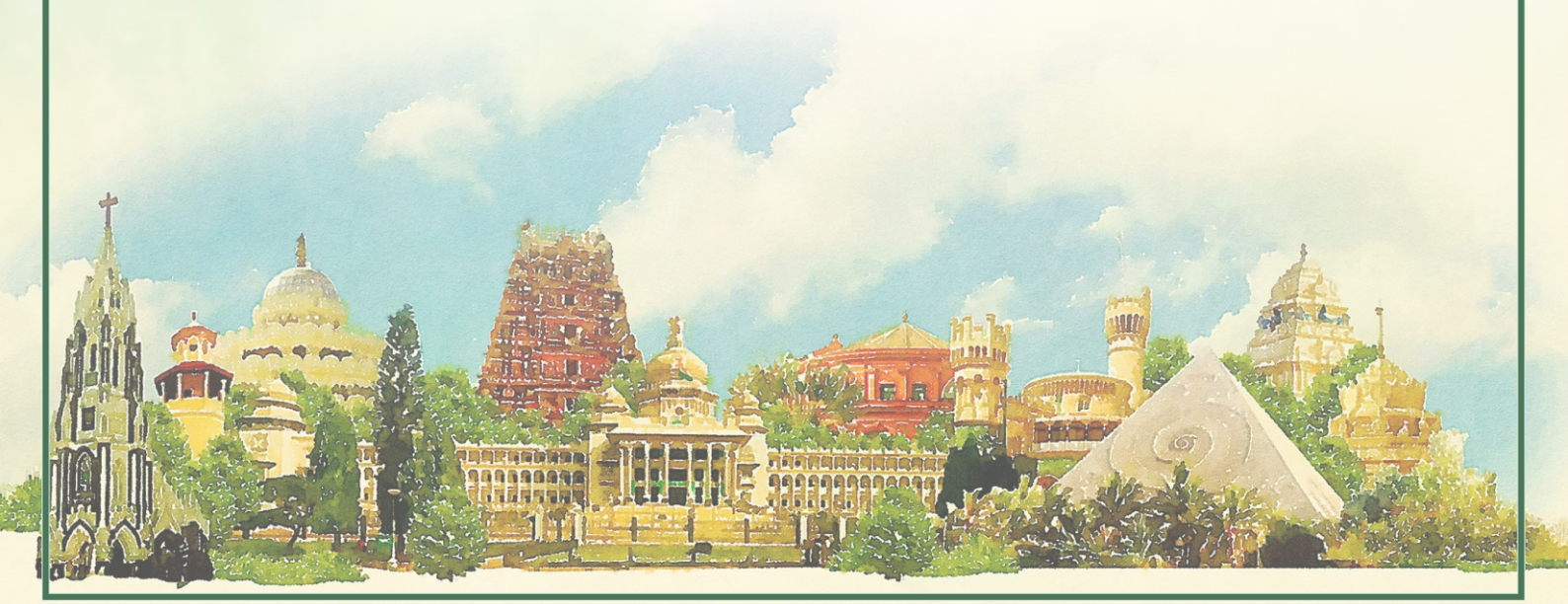
The detailed family analysis revealed a total of 9 affected family members [Image] with clinical symptoms. The age of presentation, delay in diagnosis, and investigations of the 5-generation of family is given in the table.

Pedigree of Family members





Clinical Characteristics and Laboratory Investigations in the Index Family



Patient	Delay in Diagnosis (years)	C1 Esterase Inhibitor Levels (g/L; N: 0.16–0.33)	Serum C4 Levels (mg/dL; N: 10–40)
II-8	10	0.0548	< 6.93
III-2	32	0.0468	< 5.6
III-9	8	0.0587	< 7.58
IV-2	10	0.0449	< 6.15
IV-15	5	0.0438	< 7.58 [The Index patient, Mother]
V-1	1	0.0417	< 7.4
V-4	No delay	0.06	< 7.54
V-6	No delay	0.0761	< 7.58

Discussion: HAE-I involving GI tract only was made in the index female and she was administered with oral tranexamic acid (2g/d), with significant improvement. This case portrays the myriad presentation in a single family, and also sensitises the gut-only presentation of HAE.

CC5: Not All Angioedema Is Histaminergic: A Case of Steroid-Refractory Facial Swelling

AUTHORS:

Dr. Harshvardhan Kuri[1], Dr. Mahendra Kumar[1], Dr. Rajavardhan Rangappa[1], Dr. Ankur Jindal[2]

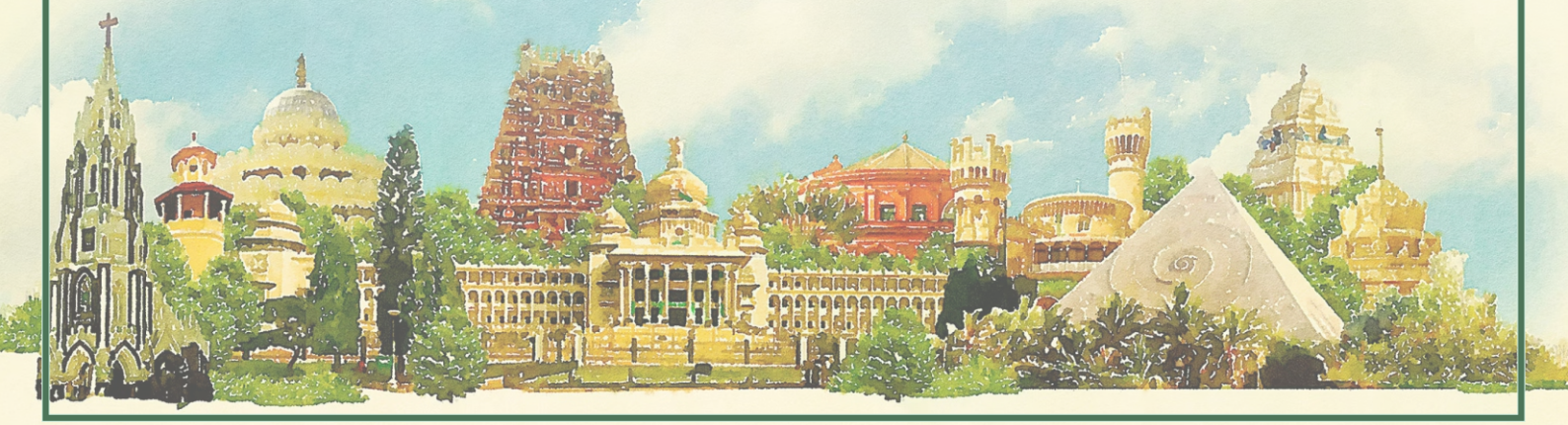
AFFILIATIONS:

[1] Department of Critical Care Medicine, Manipal Hospital, Sarjapur Road, Bengaluru, Karnataka, India [2] Pediatric Clinical Immunology and Rheumatology Division, Department of Pediatrics, Manipal Hospital, Old Airport Road, Bengaluru, Karnataka, India

ABSTRACT:

The patient presented with acute-onset facial angioedema following a dental procedure on 10/01/2026, during which local anaesthesia with lignocaine and adrenaline was administered at multiple sites. Approximately 2—2.5 hours after the procedure, she developed progressive swelling and numbness of the upper lip, cheeks, and eyelids, without associated breathlessness, hoarseness of voice, tongue swelling, abdominal pain, or desaturation. She was admitted to Manipal Hospital, Sarjapur, from 11/01/2026 to 15/01/2026. Initial emergency management with intramuscular adrenaline, intravenous hydrocortisone, antihistamines, and steroids showed minimal clinical response. Due to progressive and steroid-refractory swelling, bradykinin-mediated angioedema was suspected. She received Inj. Cinryze (C1 esterase inhibitor) 1000 IU IV on 12/01/2026, following which eyelid opening and lip swelling improved. But swelling extended to the neck region, hence a repeat dose of Cinryze 1000 IU was administered on 13/01/2026, resulting in further improvement, and the patient thereafter showed a gradual resolving trend. Investigations revealed low complement levels (C3 0.78, C4 0.12), low C1-INH level (189; reference 210—390) with negative ANA (IIF) and ANA profile. CBC showed mild anaemia and mild thrombocytopenia with otherwise stable parameters. Past history revealed a single episode of limb swelling at 11 years of age following a bee sting, and a known sulpha drug allergy; father had history of allergy to sulpha drugs. The overall clinical picture was suggestive of bradykinin-mediated angioedema, possibly hereditary or acquired. At discharge, swelling had nearly resolved with minimal residual lip edema. She was advised to repeat complement and C1-INH levels after one week, and outpatient follow-up for further evaluation.

AWARD PAPER



AP1: Genetic profile and genotype-phenotype correlation in a single centre cohort of patients with hereditary angioedema from India

AUTHOR:

Sanghamitra Machhua[1], Ankur Kumar Jindal*[1,2], Shania[1], Suprit Basu[1], Bijaya Kumar Padhi[3], Prabal Barman[1], Ridhima Agarwal[1], Reva Tyagi[1], Archan Sil[1], Gurjit Kaur[1], Saniya Sharma[1], Manpreet Dhaliwal[1], Anuradha Bishnoi[4], Vinay K[4], Rakesh Kumar Pilania[1], Pandiarajan Vignesh[1], Deepti Suri[1], Ravinder Garg[1], M Sendhil Kumaran[4], Sunil Dogra[4], Amit Rawat[1], Surjit Singh[1]

AFFILIATIONS:

1. Allergy Immunology unit, Department of Paediatrics, Advanced Paediatrics Centre, Postgraduate Institute of Medical Education and Research, Chandigarh (India)
2. Pediatric Clinical Immunology and Rheumatology Division, Department of Pediatrics, Manipal Hospitals, Old Airport Road, Bengaluru (India)
3. Department of Community Medicine and School of Public Health, Postgraduate Institute of Medical Education and Research, Chandigarh (India)
4. Department of Dermatology, Venereology and Leprology, Postgraduate Institute of Medical Education and Research, Chandigarh (India)

Introduction: Hereditary angioedema (HAE) is an uncommon autosomal dominant genetic disorder, affecting approximately 1 in 50,000 people. Deficiency in the C1-inhibitor (C1-INH) protein, resulting from pathogenic variants in the Serpin family G member 1 (*SERPING1*) gene, represents the most frequent pathophysiological anomaly in HAE patients.

Objectives: To report the spectrum of *SERPING1* gene mutations and analyse genotype–phenotype correlations in patients with C1-INH-HAE from India.

Methods: We enrolled patients diagnosed to have C1-INH-HAE, based on characteristic clinical manifestations, along with laboratory findings such as low levels of complement C4, C1- INH and/or normal C1-INH with reduced functional activity. The *SERPING1* gene was tested by targeted next-generation sequencing, Sanger sequencing, and/or multiplex ligation-dependent probe amplification for the detection of large deletions. The clinical phenotypes were correlated with the specific *SERPING1* variants and the specific exon involved.

Results: This study included 190 patients diagnosed with HAE (91 females, 99 males) from 68 different families. Type-I HAE was identified in 76.84% patients, while 23.16% patients were diagnosed with Type-II HAE. We found 41 different pathogenic variants, 16 of which were novel (Figure 1). However, no variant was detected in 45 patients. Missense mutations (43%) constituted the majority of mutations, followed by splicing defects (13%), nonsense mutations (9%), frameshift mutations (6%) and deletions (5.26%) (Figure 2). Exon 8 was observed as the most affected exon in our cohort (Figure 2).
Missense mutations, predominantly involving exon 8, were significantly associated with lower extremity involvement and tongue swelling. Splicing defects showed a negative correlation with triggers. Furthermore, frameshift mutations showed a significant negative association with disease occurrence among family members (Figure 3).

Conclusion: This study delineates the genetic landscape of C1-INH-HAE in India and demonstrates clinically relevant genotype–phenotype correlations, underscoring the importance of genetic testing for personalised diagnosis, prognostication, risk assessment and disease management.

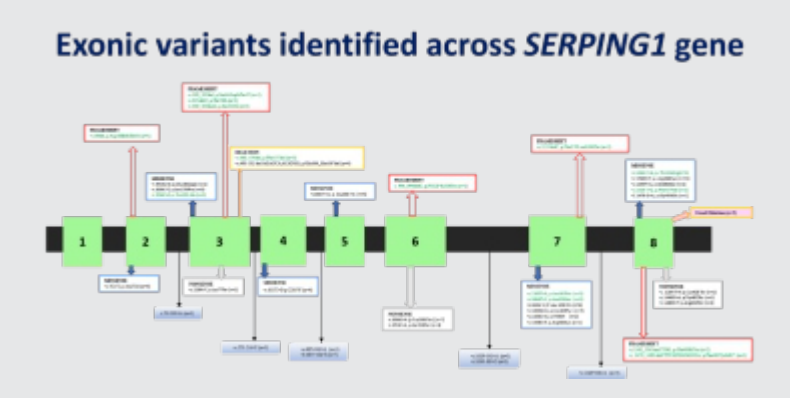


Figure - 1

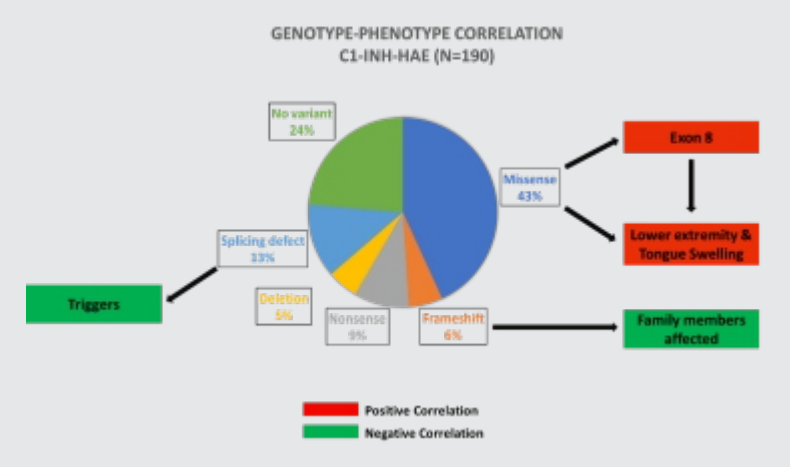


Figure - 2

AP2: CLINICAL PROFILE OF PATIENTS WITH HEREDITARY ANGIOEDEMA FROM A TERTIARY CARE CENTRE IN SOUTH INDIA

AUTHORS:

Dr. Anunya Augustine, Dr. Ankur Kumar Jindal

AFFILIATIONS:

[1]Paediatric Clinical Immunology and Rheumatology Division, Department of Paediatrics, Manipal Hospitals, Old Airport Road, Bangalore

Background: Hereditary angioedema (HAE) is a rare genetic disorder characterised by recurrent episodes of swelling involving the skin, gastrointestinal tract, and upper airway. There is a paucity of literature on HAE from South India. We report a case series on HAE from a tertiary care centre in South India.

Objective: To report the clinical profile of patients with HAE managed at a tertiary care centre in South India.

Patients and Methods: Retrospective, observational study carried out in the Angioedema Clinic, Paediatric Clinical Immunology and Rheumatology Division, Department of Paediatrics, Manipal Hospital, Bangalore, over a period of 8 months (April – November, 2025). Medical records were reviewed for demographic and clinical details, laboratory findings, treatment received, and outcomes.

Results: A total of 44 patients with HAE were included in this study. Of these, 95.45% were diagnosed with type 1 HAE, while one patient each was diagnosed with type 2 HAE and type 3 HAE. Male-to-Female ratio was 1:1. Median age of onset of symptoms was 15 years, median age at diagnosis was 28 years, and median delay in diagnosis was 15 years. Family history was positive in 72.7% of patients, and 38.63% reported deaths among family members due to laryngeal edema. One patient was asymptomatic. Among symptomatic patients, swelling of extremities (83.7%) was the most common manifestation, followed by facial edema (79%), abdominal symptoms (69.76%), laryngeal edema (58.13%), genital edema (46.5%) and tongue swelling (18.6%). Acute episodes were treated with plasma-derived C1 esterase inhibitor in 18.18%, fresh frozen plasma in 11.36%, and Icatibant in 4.54% of patients. For long-term prophylaxis, 31.81% were treated with danazol, 27.27% with tranexamic acid, 2.27% with stanozolol, and 18.18% with both (androgens and tranexamic acid). No mortality was reported over a period of 206 patient months of follow-up.

Conclusion: We report the clinical profile and outcome from a single-centre cohort of patients with HAE from South India. A long diagnostic delay remains a critical hurdle for patients with HAE in India, putting them at risk of mortality. Hence, there is an urgent need to improve awareness and access to first-line treatment options for patients with HAE in the country.

AP3: INTERPLAY OF FCεRIα GENETICS AND BASOPHIL ACTIVATION DRIVEN IMMUNE DYSREGULATION IN CHRONIC URTICARIA

AUTHOR INFORMATION:

Dr. Ayaz Gull, Prof. Roohi Rasool

AFFILIATION / DEPARTMENT:

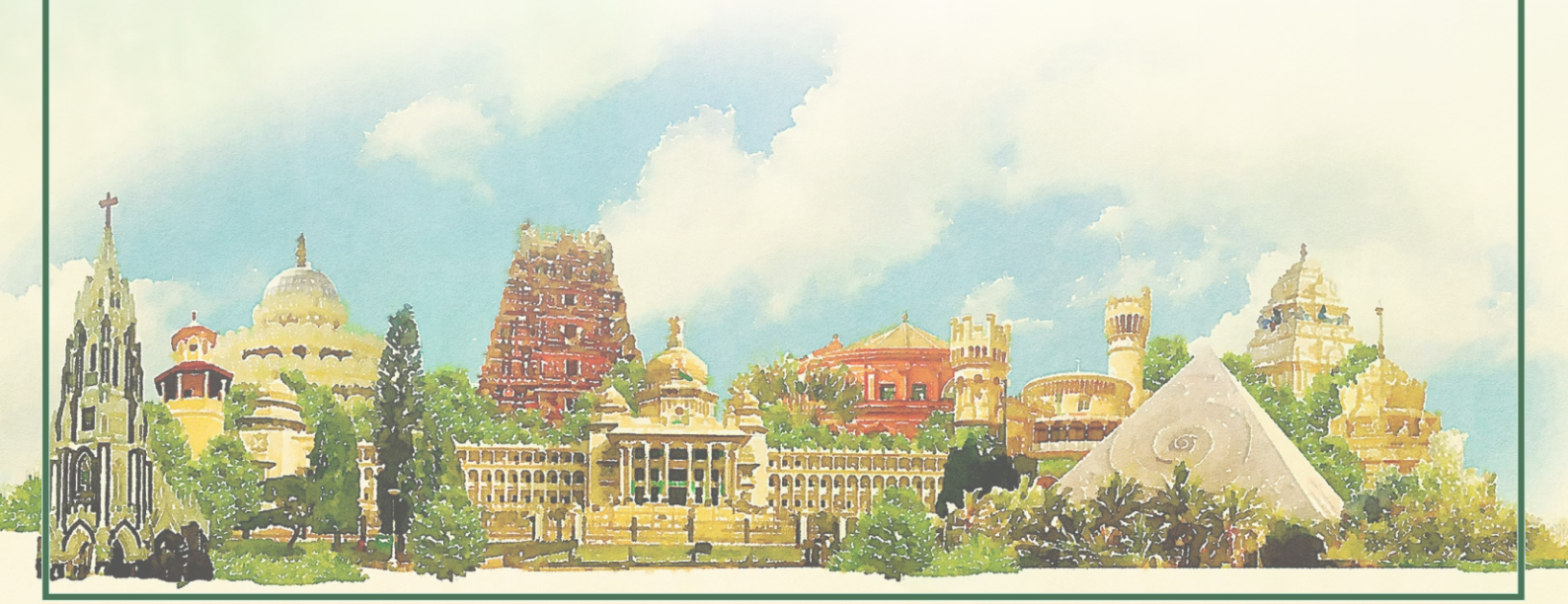
[1]Allergy and Immunology, SKIMS, Srinagar, J&K

Background: Chronic Urticaria (CU) is an immune-mediated disorder characterized by recurrent wheals and/or angioedema lasting over six weeks. The high-affinity IgE receptor (FcεRI) is central to mast-cell and basophil activation. FcεRIα promoter polymorphisms may alter receptor expression, influencing CU susceptibility and severity.

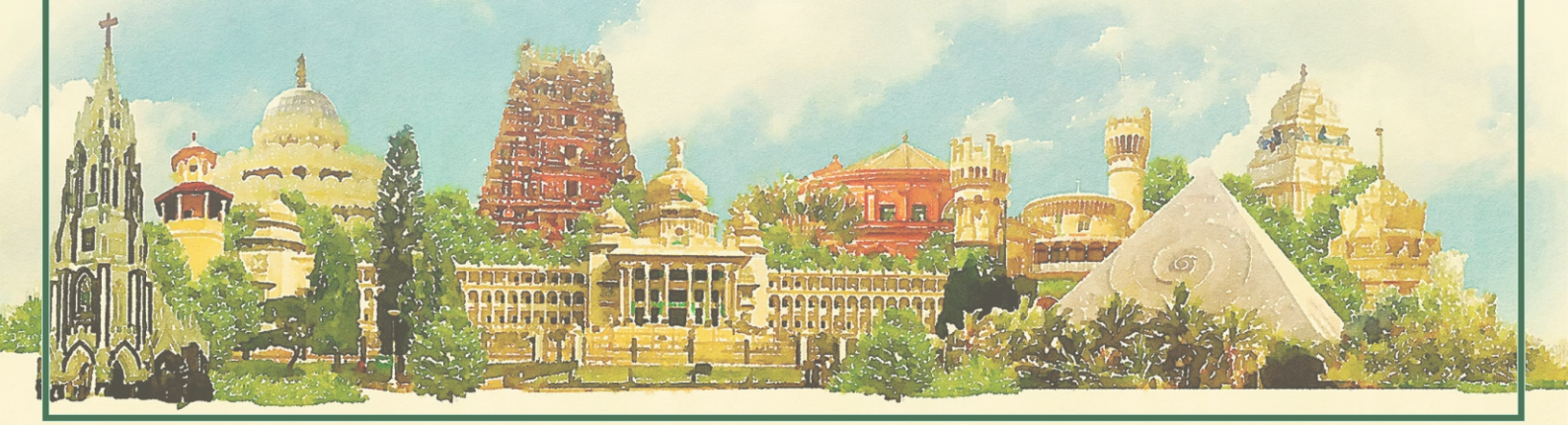
Methods: A case control study was performed on 100 CU patients and 100 healthy controls. FcεRIα promoter polymorphisms (rs2427827, rs2241746) were genotyped by PCR-RFLP and mRNA expression was quantified using real-time PCR. Serum total IgE, HDM-specific IgE, thyroid autoantibodies, and vitamin D were measured by ELISA. Basophil activation test (BAT) was performed by flow cytometry assessing CD203c expression following allergen stimulation. Statistical correlations with clinical and immunological parameters were analyzed.

Results: The rs2427827 CC genotype was significantly associated with CU risk (OR = 3.9, p = 0.002). FcεRIα mRNA expression was upregulated ($p < 0.0001$), correlating positively with total IgE, HDM-specific IgE, and ASST positivity but inversely with vitamin D levels ($p < 0.0001$). BAT showed enhanced basophil activation in HDM-sensitized patients ($p < 0.0001$), correlating with FcεRIα expression and autoimmune markers.

Conclusions: FcεRIα polymorphisms, heightened receptor expression, and basophil hyperactivation contribute to CU pathogenesis. Vitamin D deficiency amplifies immune dysregulation. Targeting FcεRI pathways and correcting vitamin D levels may improve CU outcomes.



POSTER ABSTRACTS



PA1: Steroid-Dependent Angioedema in Juvenile SLE: An Early Warning Sign of Disease Reactivation and its therapeutic challenges

AUTHOR INFORMATION:

Dr. Nagashri B S[1], Dr. Ramya J[2]

AFFILIATION / DEPARTMENT:

1. Senior Resident, Clinical immunology and Rheumatology, St Johns medical college and hospital, Bangalore.
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Case Abstract

Acquired angioedema is a rare complication of SLE and may be a presenting manifestation, typically bradykinin-mediated and mostly related to C1-inhibitor inactivation by autoantibodies. Persistent angioedema is exceptionally uncommon in SLE, and treatment is often challenging, with frequent refractoriness to standard immunosuppressive regimens. We present a child in whom persistent angioedema served both as the initial manifestation of SLE and, later, as a subtle precursor of a major flare

A 5-year-old girl had 8 months of waxing–waning periorbital swelling which evolved into juvenile-onset SLE with multiorgan involvement, including class 2 nephritis, mononeuropathy multiplex, bicytopenia and hypocomplementemia, responding to high-dose steroids and cyclophosphamide, with sustained clinical remission on azathioprine, HCQ and low-dose prednisone (2.5mg) for 2.5 years with mild persistent hypocomplementemia throughout. After 2.5 years, persistent periorbital swelling recurred; CH50 and C1-INH levels were low, consistent with acquired angioedema, and whole-exome sequencing excluded hereditary defects. This swelling was strikingly steroid dependent, recurred on taper, and remained refractory to azathioprine; switching to MMF was ineffective. Rituximab was given, guided by literature, but yielded only a partial response. This isolated manifestation with hypocomplementemia continued for 1.5 years amid multiorgan remission. Ultimately, this seemingly benign, chronic angioedema heralded a major SLE flare with polyserositis even after rituximab therapy.

This case highlights acquired angioedema as a subtle but important warning sign of impending systemic reactivation, underscoring the need for vigilant clinical and laboratory monitoring. It also illustrates that angioedema in juvenile SLE can pose major management challenges, as it may be steroid dependent and show incomplete responses to agents such as cyclophosphamide, azathioprine, MMF and rituximab in some cases, rather than consistently responding to standard immunosuppressive therapy.

PA2: Bradykinin-Mediated Angioedema With Normal Complement Levels: A Diagnostic Challenge

AUTHOR INFORMATION:

Dr. Gurrevala sharvani[1], Dr. Ankur Kumar Jindal[1]

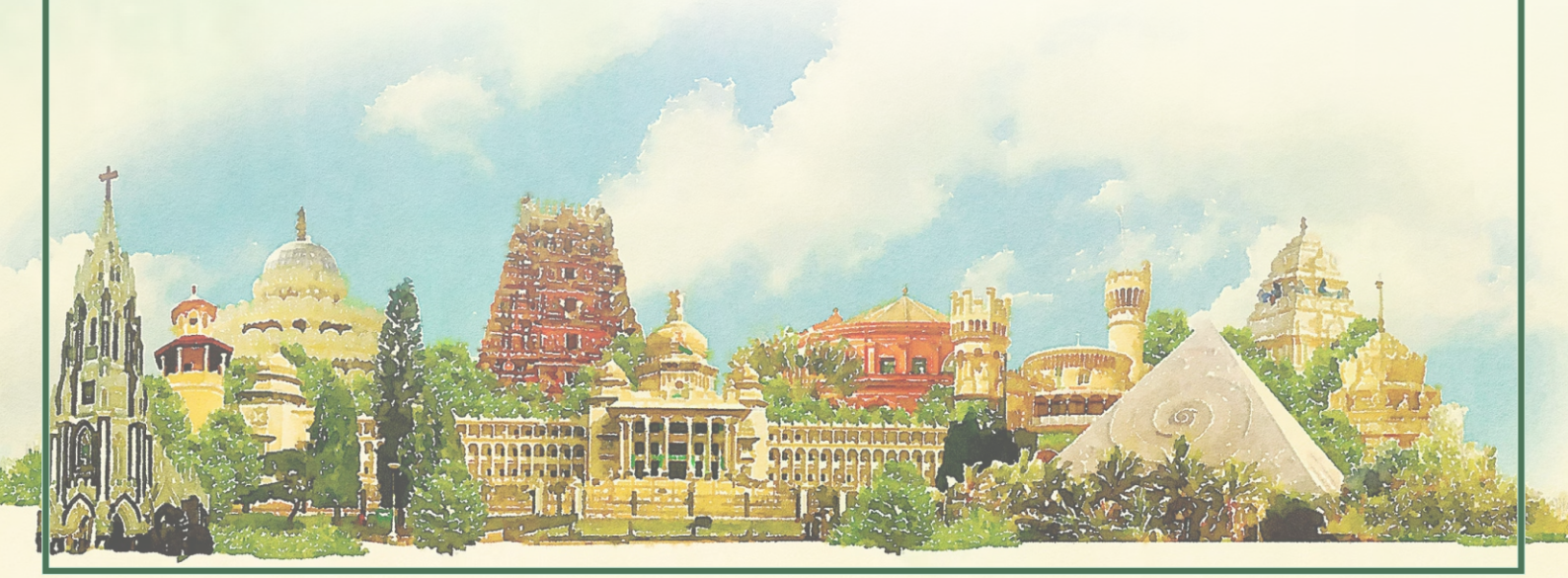
AFFILIATION / DEPARTMENT:

Paediatric Clinical Immunology And Rheumatology Division, Department of Paediatrics, Manipal Hospitals Old Airport Road Banglore

Background: Angioedema is characterized by recurrent episodes of swelling involving subcutaneous and sub mucosal tissue. Angioedema is broadly classified into mast cell mediated and bradykinin mediated. Hereditary angioedema (HAE) caused by deficiency of C1-INH protein is the most common type of bradykinin mediated angioedema. HAE due to normal C1INH (HAE-nl-C1INH) is extremely uncommon. We report one such case.

Case Presentation: A 17-year-old boy presented with recurrent episodes of angioedema for the past two years, predominantly involving the face, lips, and periorbital region. Episodes occurred once every 3–4 months, lasting 2–7 days, without associated urticaria, gastrointestinal symptoms, or laryngeal involvement. Attacks were frequently precipitated by cold environmental exposure. There was no family history of angioedema. Initial response to conventional antihistamines was inadequate.
Investigations revealed C1 esterase inhibitor levels of 0.28 g/L (reference: 0.21–0.39 g/L) and normal C4 levels of 0.29 g/L (reference: 0.10–0.40 g/L),I excluding classical HAE. Whole-exome sequencing (WES) identified a heterozygous missense variant in the PLG gene (Exon 4, c.341C>T; p. Thr114Met), classified as a variant of uncertain significance (VUS).
The patient was initially managed using bilastine 40 mg per day. He had no angioedema in last 2 months. Hence, he was not initiated on any other therapy.

Conclusion: Mutations in the PLG gene have been implicated in rare forms of bradykinin-mediated angioedema due to altered plasminogen activity and kallikrein–kinin system activation. The clinical profile in the index case suggest a phenotype consistent with bradykinin mediated angioedema and he was found to have a variant in PLG gene. However, on a short follow-up of 2 months, he has no recurrence of angioedema while taking bilastine. His further course needs to be seen and he may require treatment for PLG gene mutation.



PA3: REFRACTORY URTICARIAL VASCULITIS: OVERCOMING MULTIMODAL TREATMENT FAILURE WITH TNF APLHA INHIBITION

AUTHOR INFORMATION:

Dr. Goolla Akhila[1], Dr Ankur Jindal[1], Dr. B.G Dharmanand[2]

AFFILIATION / DEPARTMENT:

Paediatric Clinical Immunology And Rheumatology Division, Department of Paediatrics, Manipal Hospitals Old Airport Road Banglore

Background: Angioedema is characterized by recurrent episodes of swelling involving subcutaneous and sub mucosal tissue. Angioedema is broadly classified into mast cell mediated and bradykinin mediated. Hereditary angioedema (HAE) caused by deficiency of C1-INH protein is the most common type of bradykinin mediated angioedema. HAE due to normal C1INH (HAE-nl-C1INH) is extremely uncommon. We report one such case.

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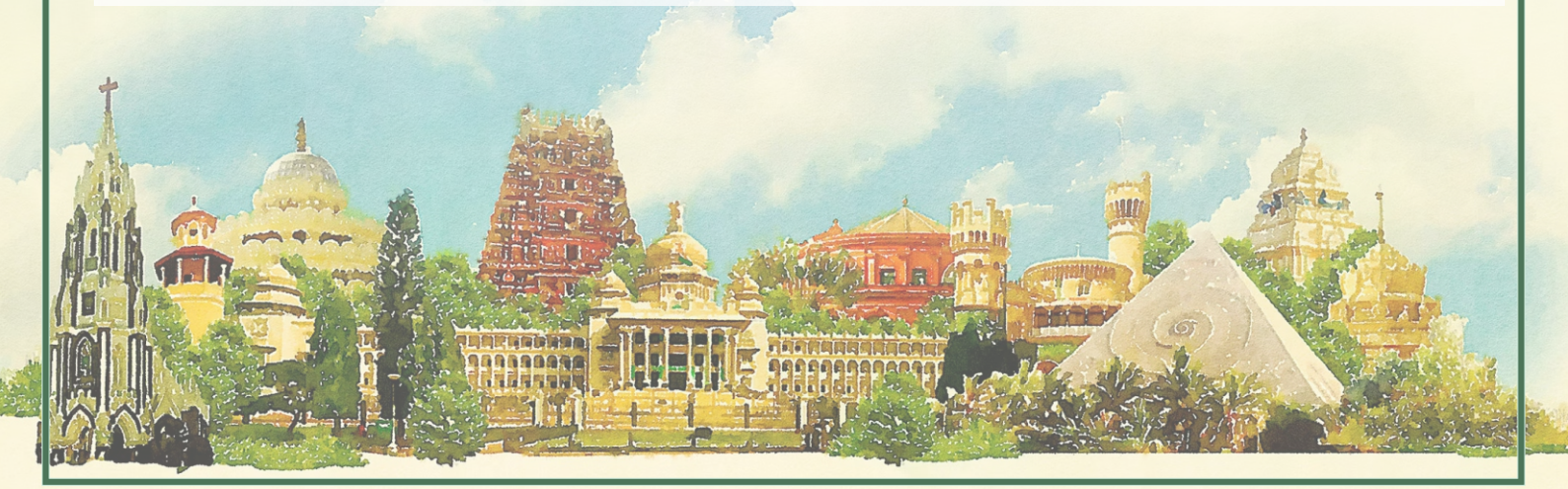
Conclusion: Mutations in the PLG gene have been implicated in rare forms of bradykinin-mediated angioedema due to altered plasminogen activity and kallikrein–kinin system activation. The clinical profile in the index case suggest a phenotype consistent with bradykinin mediated angioedema and he was found to have a variant in PLG gene. However, on a short follow-up of 2 months, he has no recurrence of angioedema while taking bilastine. His further course needs to be seen and he may require treatment for PLG gene mutation.



PA4: Urticarial Vasculitis with Angioedema in a one year old: A Rare Pediatric case report

AUTHOR INFORMATION:

Dr. Priya Kamboj, Dr. Palak Mittal

AFFILIATION / DEPARTMENT:

Govt. Medical College, Amritsar

Introduction: Urticarial vasculitis is an uncommon form of cutaneous small-vessel vasculitis characterized by urticarial lesions persisting for more than 24 hours. Its occurrence in infancy is rare, and association with angioedema further complicates diagnosis, warranting thorough evaluation to exclude complement-mediated and systemic causes.

Case Report: A one-year-old female child presented with recurrent erythematous annular plaques involving the face, trunk, and bilateral upper and lower limbs for two months, associated with mild to moderate pruritus. The lesions were persistent, non-evanescent, and remained at fixed sites for more than 24 hours. Recurrent swelling of the lips and periorbital region suggestive of angioedema was noted. The onset followed a febrile illness, and worsening of angioedema occurred after administration of cefixime for typhoid fever, raising suspicion of a drug-related trigger. Cutaneous examination revealed well-defined erythematous annular plaques with central clearing, some tender and non-blanchable. Mucosae, palms, soles, hair, and nails were spared. Systemic examination was unremarkable. A provisional diagnosis of urticarial vasculitis with angioedema was made.

Investigations: Laboratory evaluation including complete blood counts, liver and renal function tests, complement levels (C3, C4), C1 esterase inhibitor levels with functional assay, and thyroid profile were within normal limits, thereby excluding hereditary angioedema, hypocomplementemic urticarial vasculitis, and autoimmune associations.

Management and Outcome: The child was treated with antihistamines, a short course of systemic corticosteroids, and avoidance of beta-lactam antibiotics, with significant clinical improvement.

Conclusion: Urticarial vasculitis should be considered in infants presenting with persistent urticarial lesions and angioedema. Early diagnosis and systematic evaluation are essential for appropriate management.

Keywords: Urticarial vasculitis; Angioedema; Infant; Pediatric dermatology

PA5: Endotype Distribution and Therapeutic Stratification in Chronic Spontaneous Urticaria with Angioedema (CSU): A Real-World Analysis of 52 patients

AUTHOR INFORMATION:

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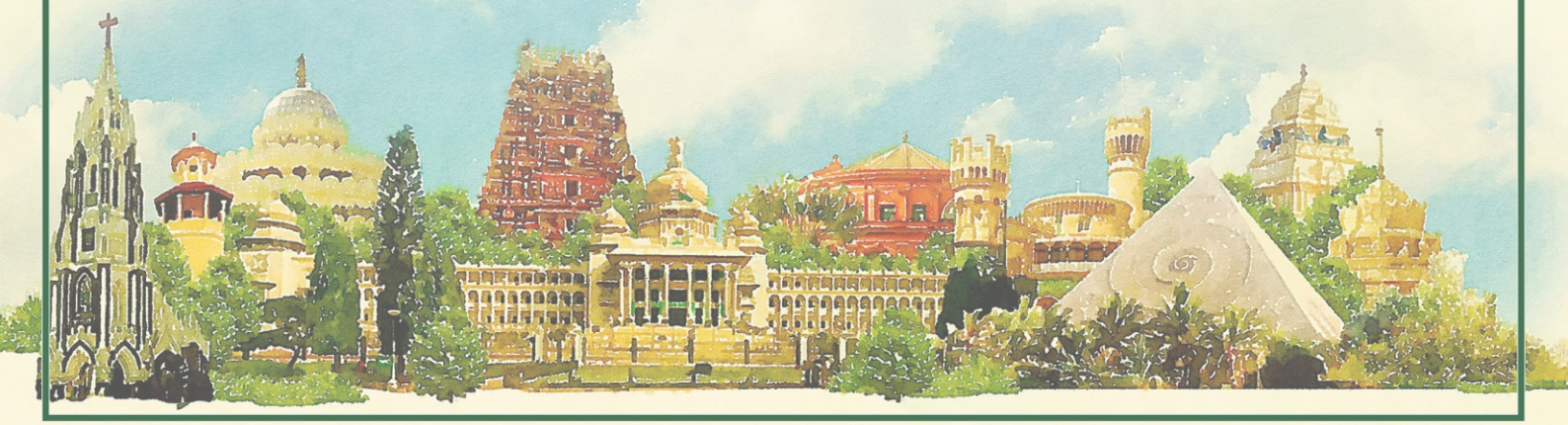
AFFILIATION / DEPARTMENT:

[1]Paediatric Clinical Immunology and Rheumatology Division, Department of Paediatrics,

Background: CSU, frequently associated with angioedema, is a mast-cell–driven disease with recurrent wheals or swelling persisting >6 weeks.[1] Immunologic profiling identifies two principal endotypes: Type I is characterized by high total IgE responding well to antihistamines ±omalizumab. Type IIb is driven by IgG autoantibodies targeting IgE or FcεRI with lower IgE and greater resistance to antihistamines and/or omalizumab.[1] Juliana et al from Brazil reported 38% type I, 9% type IIb, and 51% mixed type I/IIb endotypes, <2% non-type I/IIb in their cohort of 394 patients.[2] Despite well-defined endotypes, similar data for Indian CSU patients remain sparse, necessitating this study.

Methods: A retrospective analysis of all patients with CSU ± angioedema was carried out in the Angioedema Clinic, Paediatric Clinical Immunology and Rheumatology division, Manipal Hospital, Bengaluru. Clinical data, laboratory investigations, treatment details and outcome were collated. Patients were assigned to 1 of the 4 endotypes: type I with 1 of the following: allergic disease, high IgE. Type IIb with presence of autoimmunity, anti TPO positivity, low IgE. Mixed type with overlap of type I/IIb endotypes. Non type I/IIb with none of the markers of type I or IIb.[2]



Results: Of total 65 patients registered in the clinic between April and December 2025, we included 52 patients in whom complete clinical data were available. Mean age of this cohort was 23.7±16.2 years and mean age of symptom onset was 20.2 ± 15.5years with female preponderance (59.6%). Endotyping of the cohort showed type I as the most common endotype (n=30, 57.7%) followed by non-type I/IIb (n=15, 28.8%), Type 1/IIb (n=5, 9.6%), and type IIb (n=2, 3.8%). Statistical analysis showed no significant differences across endotypes for mean age (p=0.242), age of symptom onset (p=0.415), gender distribution (p=0.298). Allergic manifestations were present in 21.2% (n=11), predominating in type I. Autoimmune associations included SLE (Type IIb, n=1) and autoimmune thyroiditis/ANA positivity (Type I/IIb, n=1)
All patients received antihistamines while omalizumab was used in 4 patients [type I (n=1), type I/IIb (n=1) and non-type I/IIb (n=2)]. Cyclosporine was used in patients with type IIb and type I/IIb (1 each), and Dupulimab in one patient with type I/IIb.

Conclusion: This cohort demonstrates predominance of type I and non-type I/IIb endotype in patients with CSU. Baseline CSU endotyping is essential for guiding clinical management. Patients with type I endotype tend to respond better to antihistamines and/or omalizumab as compared to any other endotype.

Key words: Chronic spontaneous urticaria, angioedema, IgE, autoimmune, antihistamines

References:

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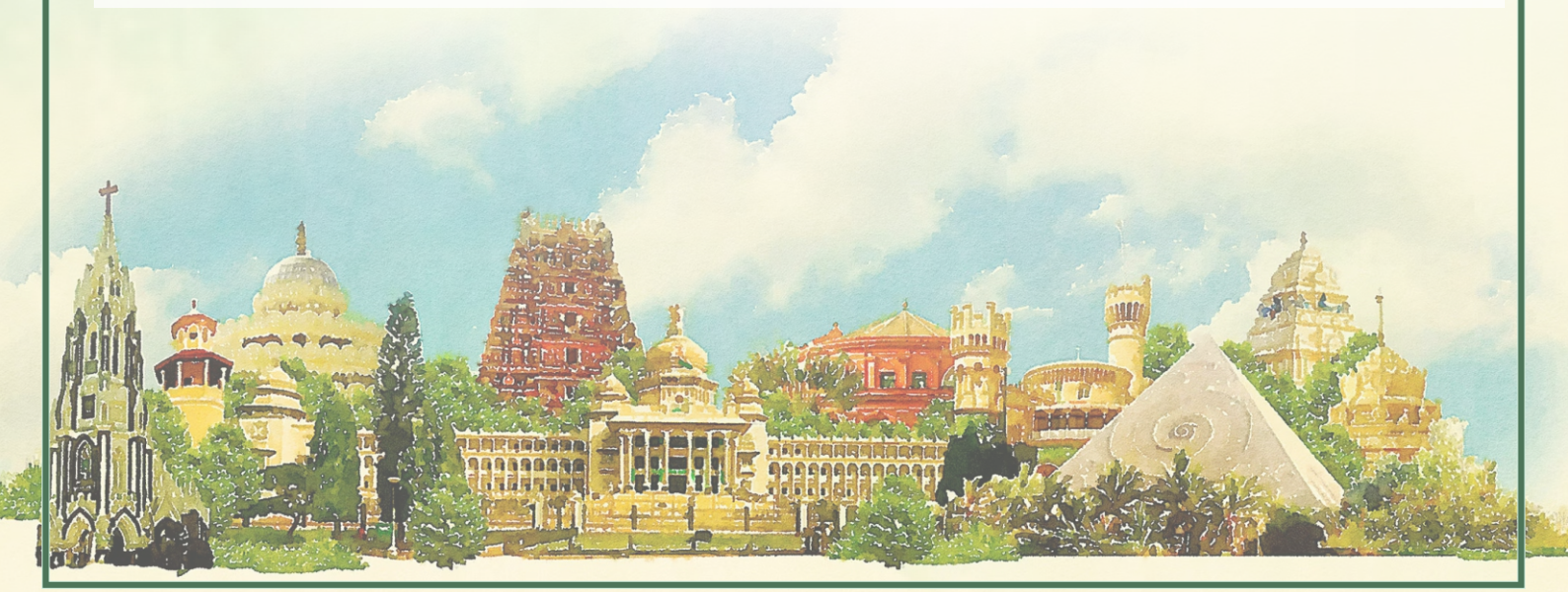

PA6: "Case series on chronic urticaria in children- diagnostic and therapeutic challenges"

AUTHOR INFORMATION:

Dr. Revathy. S

AFFILIATION / DEPARTMENT:

[1]Assistant professor, department of paediatrics and consultant Allergist in Akash Institute of medical sciences and research centre, Bengaluru.

Introduction: Chronic urticaria is defined as occurrence of wheals, angioedema or both for more than 6 weeks of duration. It can be spontaneous, inducible or in some cases both can coexist. Recent advances classify cause of chronic spontaneous urticaria into autoimmunity type-1 and autoimmunity type-2b. Investigations such as autologous serum skin test, total serum IgE, anti TPO IgG, CBC, ESR help in understanding the type and prognosis [(1)]. This case series describes 3 cases of difficult to treat chronic urticaria in children.

Case description:

Case-1: 16year old adolescent male presented with symptoms of chronic inducible urticaria in the form of symptomatic dermographism and allergic rhinitis since last 1 year. Lab investigation showed raised serum IgE and specific IgE by ELISA method showed significant high titres to pollen and house dust mite group of aeroallergens. Treated with intranasal corticosteroids for allergic rhinitis which improved after 1 month. Patient is on tab. Bilastine20mg OD along with HISTAGLOB injection for symptomatic dermographism at present and his Urticaria Activity Score has reduced.

Case-2: 15year old adolescent female presented with chronic spontaneous urticaria with angioedema of eyelids only with history of 2 such episodes in previous 2 years and did not respond to high dose antihistamines. Patient responded to oral prednisolone given for 10 days. Autologous serum skin test was positive significantly. Total serum IgE was raised and Anti- TPO levels highly positive. She was initiated on autologous serum injection 1 dose/ week for 8weeks followed by 1 dose every fortnight for next one month, followed by monthly once dosage. Currently she is on monthly once dosage and she is in complete remission since last one month.

Case-3: 7year old female child presented with chronic spontaneous urticaria with angioedema of eyelids, lips and at times even tongue. Patient has experienced 4 episodes per month since last 6months. Her height and weight are below 3rd centile for age. Bone age is less than chronological age. Autologous serum skin test was significantly positive. Skin prick testing for aeroallergens was negative. Total serum IgE levels were raised. Lab workup for serum C4 levels, Anti TPO, anti TTG IgA, ANA profile, HBsAg, Anti-HCV and stool for ova cyst were reported to be negative. Currently she is on autologous serum injection 5th week.

Conclusion: Difficult to treat chronic urticaria in children pose diagnostic as well as therapeutic challenge. Symptomatic dermographism is seen to be coexisting with some form of atopic disease in most patients [(2)]. Chronic spontaneous urticaria of autoimmune type need thorough evaluation to rule out underlying causes.

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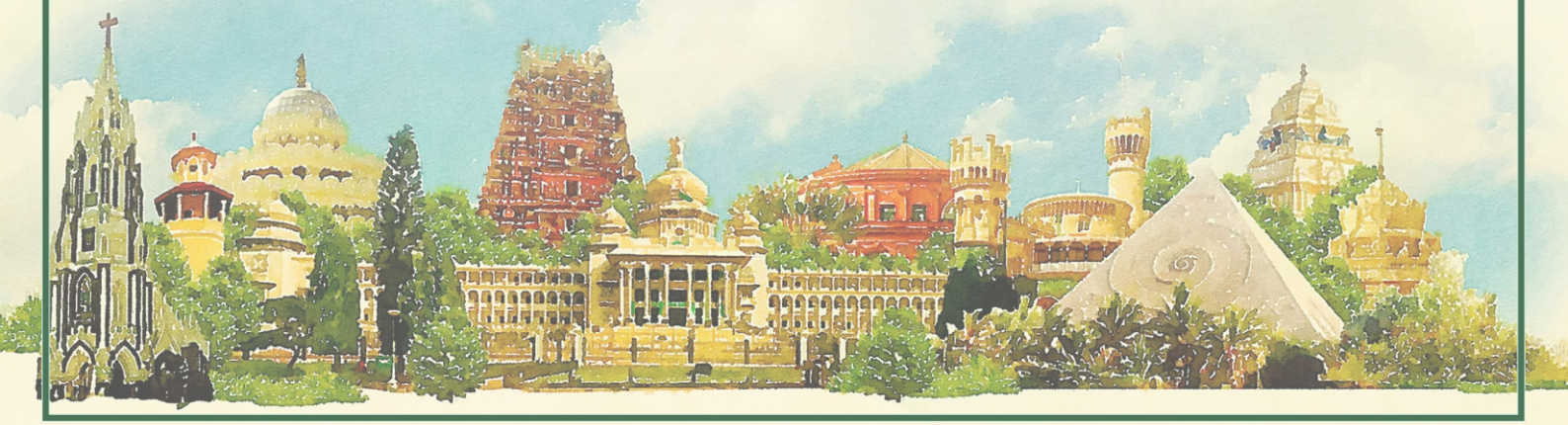

PA7: Urticaria as a Window into Inborn Errors of Immunity

AUTHOR INFORMATION:

Dr. Neha Singh[1], Dr. Bharat Kumar[2], Dr. Rajesh Kumar[3], Dr. Sougata Mahato[3], Dr. PNS Munda[3], Dr. Krishna Kumar[3]

AFFILIATION / DEPARTMENT:

ESIC Medical College & Hospital, Ranchi Panda Multispeciality Care Hospital & Research Centre, Bokaro Steel City Rani Hospital, Ranchi

Objectives: To analyse the profile of patients diagnosed with an IEI presenting with urticaria

Methods: Between May 2024 and July 2025, **36 patients** were diagnosed with primary immunodeficiency and autoinflammatory disorders.

Results: Eight patients presented with urticaria, including PRKCD defect (n=1), **STAT1** defect (n=1), STAT3 defect (n=3), **leukocyte adhesion defect** (n=1), and prolidase defect (n=1). The mean age of urticaria onset was 23 months and mean age of diagnosis was 30 months. Urticaria preceded the onset of infections in 3/7 patients. All patients had chronic urticaria. Management strategies were individualised based on underlying pathology. The majority of patients received antihistaminics, antibiotics, and antifungal prophylaxis. Systemic corticosteroids were utilised in 3 patients, while JAK inhibitor therapy was utilised in 2 patients. One patient received monthly immunoglobulin therapy. Six patients had remission while one patient succumbed to the primary immunodeficiency.

Conclusion : This experience highlights the diversity and complexity of paediatric immunological disorders and emphasises the need for a multidisciplinary, precision-based approach for optimal diagnosis and management.

PA8: Autosomal Dominant Hereditary Angioedema in Two Families: A Genetically Confirmed Case Series of Six Patients

AUTHOR INFORMATION:

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ABSTRACT

Background: Hereditary angioedema (HAE) is a rare, autosomal dominant disorder characterized by recurrent episodes of non-pitting, non-pruritic angioedema occurring in the absence of urticaria. Attacks may involve the skin, gastrointestinal tract, or upper airway and can be life-threatening. Failure to recognize isolated angioedema episodes often results in significant diagnostic delay and inappropriate management, particularly in resource-limited settings.

Methods: We describe a case series of six affected individuals from two unrelated families evaluated at tertiary care centers in India. Clinical evaluation focused on recurrent angioedema episodes without wheals, abdominal symptoms, laryngeal involvement, precipitating factors, and family history. Laboratory investigations included serum complement C4 levels and quantitative and functional C1 esterase inhibitor (C1-INH) assays. Genetic testing was performed to confirm pathogenic variants consistent with autosomal dominant hereditary angioedema. Clinical response following diagnosis and initiation of prophylactic therapy was assessed during follow-up.

Results: All six patients presented with recurrent angioedema episodes in the absence of urticaria, with abdominal involvement observed in four patients and peripheral or facial swelling in five. Two patients had undergone unnecessary surgical or emergency interventions prior to diagnosis. Biochemical evaluation revealed persistently reduced serum C4 levels and low functional C1-INH activity in all cases. Genetic analysis confirmed disease-causing mutations in both families, establishing autosomal dominant inheritance. Institution of long-term prophylactic therapy resulted in a significant reduction in the frequency and severity of angioedema attacks across all patients.

Conclusion: This case series underscores the critical importance of recognizing recurrent isolated angioedema, particularly in the absence of urticaria, as a key clinical feature of hereditary angioedema. Early biochemical screening and genetic confirmation are essential to prevent diagnostic delay, avoid iatrogenic morbidity, and facilitate timely, effective management. Enhanced clinician awareness is vital to improving outcomes in patients with hereditary angioedema.

Keywords: Hereditary angioedema, isolated angioedema, C1 esterase inhibitor deficiency, autosomal dominant, genetic confirmation, abdominal angioedema

PA9: Clinical Profile and Outcomes of Hereditary Angioedema in North East India: A Retrospective Study.

AUTHOR INFORMATION:

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AFFILIATION / DEPARTMENT:

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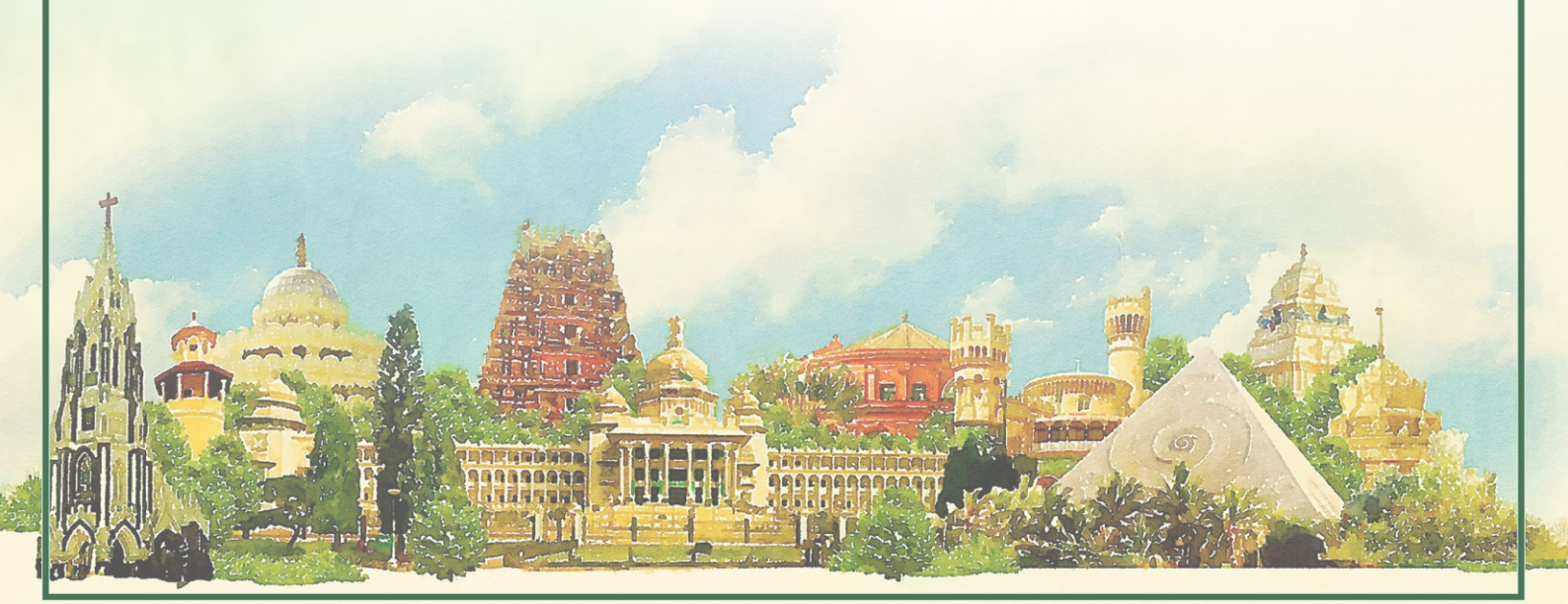
Background: Hereditary angioedema (HAE) is a rare autosomal dominant disorder characterized by unpredictable outcomes. Due to limited clinical awareness, it often remains undiagnosed for years, leading to high mortality rates. This report analyzes the clinico-epidemiological features and outcomes of HAE patients at a tertiary care institute in North East India.

Methods: We conducted a retrospective record review of 13 patients diagnosed with HAE between 2021 and 2025. Data regarding clinical presentation, epidemiology, management strategies, and patient outcomes were analyzed.

Results: The cohort comprised 13 cases (7 males, 6 females) with ages ranging from 8 to 52 years. Frequency of angioedema episodes varied from one to four per month, with the face being the most common site of involvement. Pathogenic variants in the *SERPING1* gene were confirmed in two patients. Notably, one patient's family history revealed the undiagnosed deaths of both a father and grandfather due to similar symptoms. Long-term prophylaxis consisted of tranexamic acid for all patients, while acute "on-demand" episodes were managed with fresh frozen plasma (FFP) and C1-esterase inhibitor supplements.

Conclusion: In North East India, HAE remains underdiagnosed due to insufficient clinical suspicion, contributing to significant morbidity and preventable mortality. Increasing healthcare provider awareness is critical for early diagnosis and improving patient survival.



PA10: Profile of 38 patients with chronic spontaneous urticaria from a tertiary care centre in Eastern India

AUTHOR INFORMATION:

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AFFILIATION / DEPARTMENT:

Department of Critical Care and Medicine, Panda Multispeciality Care Hospital & Research Centre, Bokaro Steel City ESIC Medical College & Hospital, Ranchi

Objectives: To analyse the profile of 38 patients diagnosed with chronic spontaneous urticaria (CSU) at a tertiary care centre in Eastern India

Methods: Between April 2024 and December 2025, 38 patients were diagnosed with chronic spontaneous urticaria at our hospital. The data was analysed and entered in a pre-designed excel sheet. The clinical features, and laboratory findings, including IgE levels, treatment & outcome details, were analysed.

Results: The mean age of presentation was 24.5 years(2 – 56 years) and the mean duration of disease was three months(1 – 8 months), respectively, at the time of presentation. Our cohort included 20 adult and 18 pediatric (<18 years) patients. Male-to-female ratio was 1:2. Twenty patients presented with wheals only, and 18 had wheals and angioedema. Eleven patients had allergic rhinoconjunctivitis, and four patients had bronchial asthma.

All patients received antihistaminics. Eighteen patients received bilastine, eleven patients cetirizine, five patients hydroxyzine and four patients fexofenadine as first-line treatment. Five patients failed treatment (Cetirizine=4, Fexofenadine=1). Systemic corticosteroids were used in five patients. Mean IgE level was 456 (IU/mL). Of these, two (Cetirizine=2) responded to a doubling dose of treatment, and three were shifted to Bilastine. Mean treatment duration was 16 weeks (12–30 weeks). With treatment, all were in remission.

Conclusion: Our experience highlights the diversity and complexity in management of CSU in our setting.

PA11:Clinical Profile, management and outcome of patients with hereditary angioedema type 1: our experience from a single centre in South India.

AUTHOR INFORMATION:

Dr. Uma Tejaswi [1], Dr. Jayanth B Nair[1], Dr. Sagar Bhattad[1]

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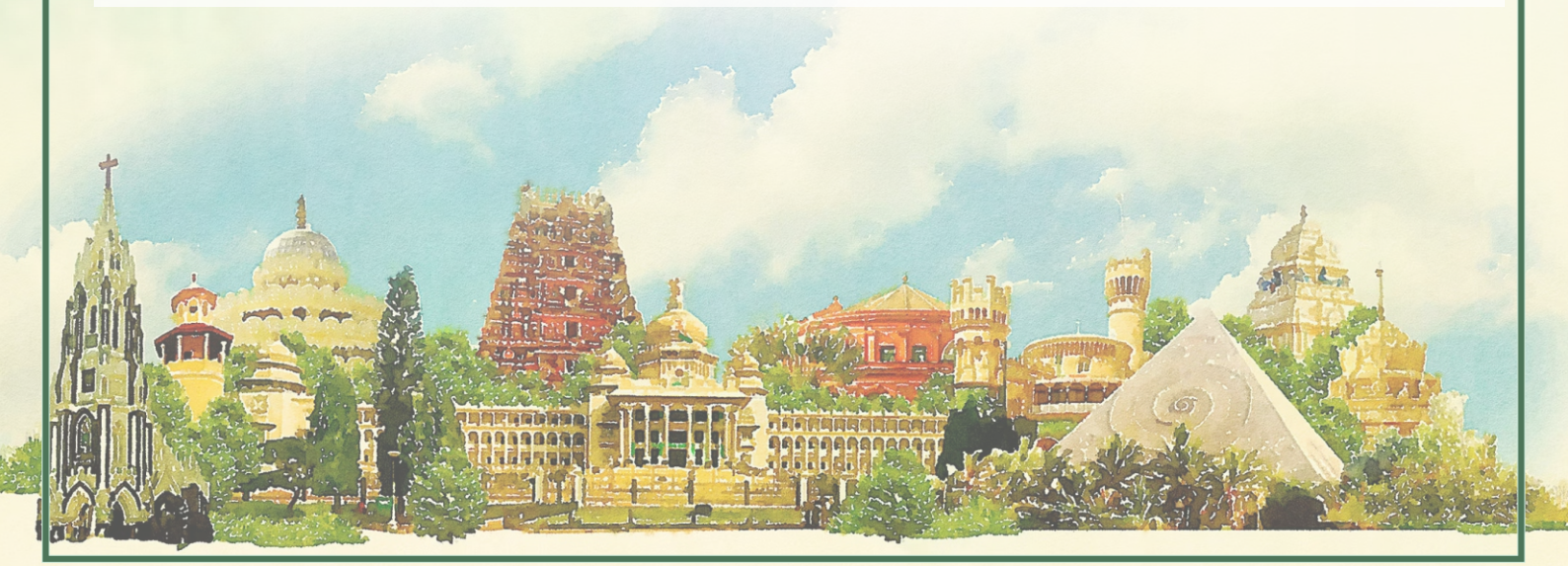
Division of Paediatric Immunology, Rheumatology and Bone Marrow Transplant, Manipal Hospital - Yelahanka, Bangalore, India.

Introduction: Hereditary angioedema (HAE) is an uncommon genetic disorder caused by deficiency or dysfunction of the C1 esterase inhibitor. HAE is characterised clinically by recurrent episodes of non-itchy subcutaneous and/or submucosal swellings. Early recognition and management of laryngeal oedema is lifesaving in HAE patients. Although clinical symptoms of HAE can manifest at any age; most of them become symptomatic before the age of 18 years.

Methods: Data of patients who presented to us and diagnosed with HAE type 1 were retrieved from the Outpatient records from Sept 2017 – Nov 2025. Of the total 11 patients diagnosed with HAE, clinical and laboratory data were available for 8 patients. Male: female ratio was 1.2:1. Median age at onset of symptoms and diagnosis were 14 years (range 3-35 years) and 23 years (range 3-67 years) respectively. Median delay in diagnosis was 5 years (range 0-42 years). Facial and extremity swelling was the most common presentation (8/8), followed by abdominal symptoms (5/8) and laryngeal oedema (1/8). Genital swelling noted in one of them. One patient developed sagittal sinus thrombosis with large right frontal venous infarct and bleed. A positive family history was present in 5/8 patients. Diagnosis was established by low C4 and C1-INH levels. 5/8 of them had low C4 levels and 3/8 of them had very low C1-INH levels. Whole-exome sequencing detected a pathogenic variant in SERPING1 gene in one of the two tested patients. Most patients received fresh frozen plasma as on demand therapy. Long term prophylaxis included attenuated androgens (1/8) and tranexamic acid (8/8). Number of attacks significantly reduced in patients on long-term prophylaxis. No deaths were reported during the study period. Limitations of our study include a small sample size and its retrospective nature.

Conclusion: HAE is a life threatening disease and a timely diagnosis is crucial to improve outcomes. A significant delay in diagnosis was observed in our cohort, highlighting the need to raise awareness about the disease amongst physicians and primary caregivers.



PA12: Autoimmune diseases and autoantibodies in patients with Hereditary Angioedema: A Preliminary study.

AUTHOR INFORMATION:

Dr. Navjot Kaur[1], Dr. Ankur Kumar Jindal[2], Dr. Amit Rawat[3], Dr. M Sendhil Kumaran[4], Dr. Saniya Sharma[5]

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2. Department of Pediatrics, Advanced Pediatrics Centre, Postgraduate Institute of Medical Education and Research, Chandigarh, India.
3. Department of Pediatrics, Advanced Pediatrics Centre, Postgraduate Institute of Medical Education and Research, Chandigarh, India.
4. Department of Dermatology, Venereology and Leprology, Postgraduate Institute of Medical Education and Research, Chandigarh, India.
5. Department of Pediatrics, Advanced Pediatrics Centre, Postgraduate Institute of Medical Education and Research, Chandigarh, India.

Background: Hereditary angioedema (HAE) is a rare inherited disorder characterized by recurrent, non-pruritic episodes of subcutaneous, gastrointestinal, and upper airway edema due to deficiency or dysfunction of C1 inhibitor (C1-INH). Emerging evidence suggests an increased risk of autoimmune diseases in patients with HAE, possibly related to complement dysregulation and impaired immune complex clearance. However, data from the Indian subcontinent are lacking.

Objectives: This study aimed to evaluate the risk of autoimmune diseases and the presence of antinuclear antibodies (ANA) in patients with HAE and to compare these findings with healthy controls.

Methods: A cross-sectional observational study was conducted from July 2023 to December 2024 at a tertiary care center in North India. Fifty-five patients with hereditary angioedema (HAE) type 1 or type 2 and fifty-two age- and sex-matched healthy controls were enrolled. Participants were evaluated using a structured questionnaire focusing on autoimmune manifestations and an eight-point fatigue severity score. Serum antinuclear antibodies (ANA) were assessed using indirect immunofluorescence, and samples with ANA positivity of ≥2+ were further analyzed using an ANA immunoblot assay. Non-parametric statistical tests were applied.

Results: The median age of symptom onset among cases was 8 years, while the median age at diagnosis was 20 years. Questionnaire positivity with higher fatigue scores was significantly more common in patients with HAE (24%) compared to controls (2%). ANA positivity was detected in 32.7% of HAE patients and 15.3% of controls, though this difference was not statistically significant. A significant correlation was observed between questionnaire positivity and ANA positivity, supporting immune dysregulation in HAE.

Conclusion: Patients with HAE demonstrate a higher prevalence of autoimmune manifestations and autoantibody positivity compared to healthy individuals. Routine screening for autoimmune features may facilitate early identification and comprehensive management of these patients. Larger, longitudinal studies are required to further elucidate this association.

PA13: ANGIOEDEMA : A MYSTERY BOX

AUTHOR INFORMATION:

Dr. Smitha J N Singh

AFFILIATION / DEPARTMENT:

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Introduction : Malfunction of the immune system is known in three main conditions - Immunodeficiency , Allergy & Autoimmunity.

Case description : A 81 year old lady, retired teacher hailing from Bangalore came with the C/O multiple joints pain mainly in the hands, knees, foot B/L since 10 yrs. H/O swelling of tongue with difficulty in breathing on and off since 30 yrs after consuming certain types of food. H/O running nose, cold, headache with frequent sinusitis since 1 month. No H/O wheezing or eye symptoms or H/O skin rashes with itching. H/O lactose intolerance since childhood.Personal history - Vegetarian with
normal appetite, bowel and bladder habits with good sleep.H/O Allergy to Penicillin, Aspirin, Analgesics, few antibiotics except Azithromycin and Norflox, as well as food items like Nuts, Sea food, Fruits, Groundnut oil.

Treatment history - Antihistamines- Cetirizine SOS

-Past History : HTN since 20 yrs, DM since 15 yrs Joints pain since 10 yrs

-Gynae History : ML -54 yrs, P3 L3 A3, Menopause at the age of 55 yrs

-Family History : Father : HTN, DM, Mother : HTN, Brother : HTN

GPE - Normal with Vitals stable

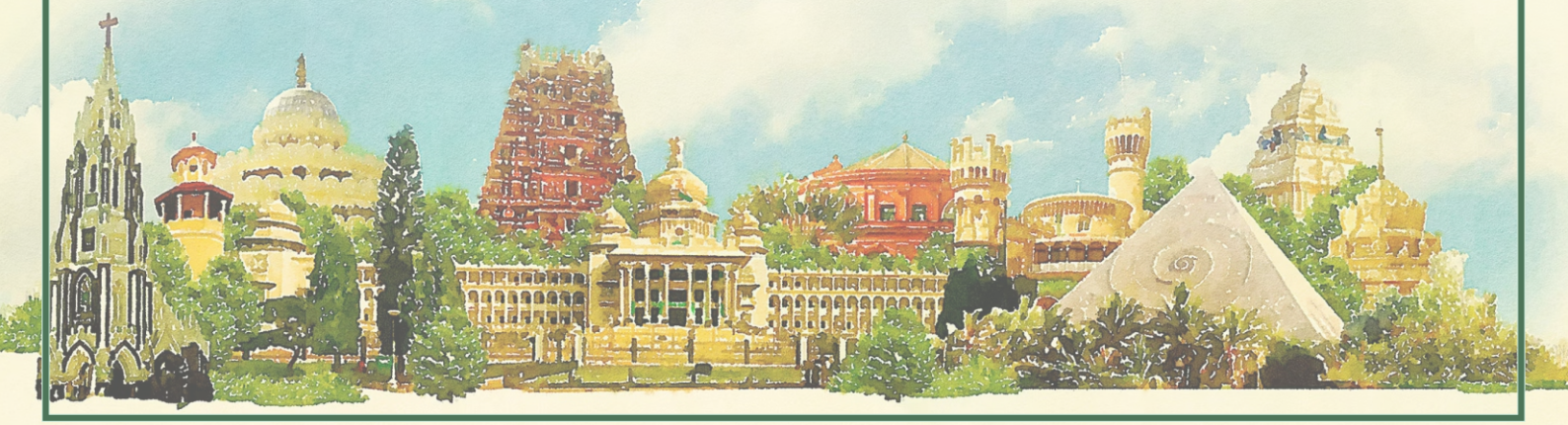
Systemic examination -NAD,

Investigations :
-Hb : 12.6 gm/dl - Tc : 6930 cells/cumm
- Dc : N-61%, L-31.7%, B-0.1%, E-1.9%, M- 5.2% -Plt count : 2.91 Lakhs/cumm, - ESR : 50 mm/hr, - LFT : Normal,
RBS : 86 , RF - 18, Serum creatinine : 0.95, Anti CCP - Negative,
- CRP-2.9 , ANA (IF) - 2+ Nuclear homogenous pattern, Serum IgE levels - 84.88, - HIV - Negative, - HCV - Negative, C3 Levels - 140, C4 Levels - 46,
ANA Profile - Negative, C1q antibody - Negative 2.0 units, Allergy panel - Negative,
X-Ray both hands - OA changes

Final diagnosis: UCTD / ANA associated urticaria with OA with ? Allergy

With the above findings and diagnosis in mind she was started on Hydroxychloroquine, calcium supplements, Glucosamine, Vitamin D supplements as well as antihistamines.She came for follow up after 6 weeks with reduction in her joints pain by 30%, no H/O allergies except for mild swelling of tongue occasionally.Next visit was after 2 months where she C/O on and off swelling of throat associated with cough and mild breathing difficulty after taking coffee and tea, was advised to avoid triggering factors and continue same treatment. Again she came after a month with H/O taking Covid vaccination - 2 days later, she had developed nasal block, facial oedema, and had similar complaints like swelling and discomfort in the throat. This time she was asked to take a chest X- ray which revealed a mass in the right side of the chest which finally turned out to be Adenocarcinoma Lung - Stage 4. She was treated for the same at a different center.

Discussion : Initially the whole scenario looked like some type of Arthritis or Allergy or CTD but with in 6 months of Patient's visit, it turned out to be Malignancy where the symptoms were absolutely clueless Hence this case is an eye opener that every visit patient has to be evaluated with all other un common possibilities so that early diagnosis helps in better prognosis in life threatening diseases.



PA14: Recurrent Angioedema Without Urticaria: A Case of Hereditary Angioedema Type I

AUTHOR INFORMATION:

Dr. Sannitha S Kapatral, Dr. Shivanand D R, Dr. Amruthavalli P

AFFILIATION / DEPARTMENT:

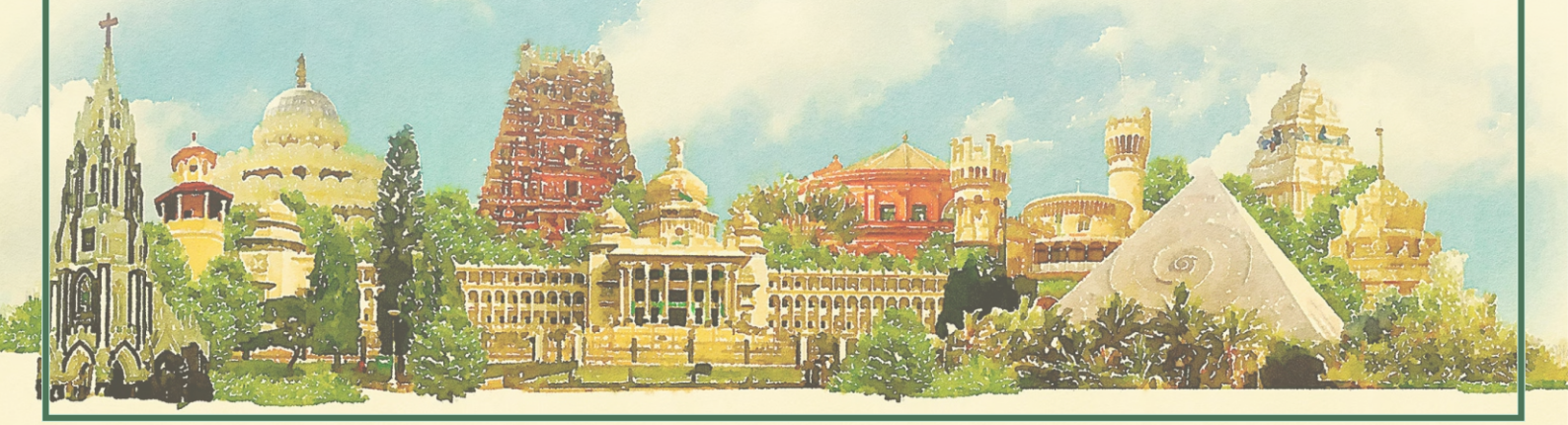
Sri Siddhartha Medical College, Tumkur

INTRODUCTION: Hereditary angioedema (HAE) is a rare, potentially life threatening disorder characterized by recurrent, non-pitting, non-pruritic swelling of the skin and mucosa due to C1 esterase inhibitor (C1-INH) deficiency, leading to bradykinin overproduction. Unlike histamine-mediated angioedema ,HAE episodes occur without urticaria and are unresponsive to antihistamine or corticosteroids. Delayed recognition may result in significant morbidity, particularly if laryngeal involvement occurs. Early diagnosis through clinical suspicion and complement testing is therefore crucial

DETAILS OF CASE REPORT: Here is a 43 year old woman presenting with her third episode of gradual facial angioedema involving the eyelids, lips and face. The swelling was non-pruritic, non pitting and not associated with urticaria. There was no identifiable drug triggers, including ACE inhibitors and no history suggestive of allergy o autoimmune diseases.
Previous episodes were treated with antihistamines and corticosteroids without improvement and resolved spontaneously within 2-5 days.
Complement studies revealed a markedly reduced C1-INH level:0.03g/L;ref(0.21-0.39), confirming the diagnosis of Hereditary Angioedema type I.

CONCLUSION: This case underscores the importance of distinguishing bradykinin mediated angioedema from allergic angioedema. Prompt diagnosis through complement studies enables appropriate management with C1- INH replacement therapy and prophylactic strategies, significantly reducing morbidity and preventing life- threatening airway compromise.

ACADEMIC PARTNERS



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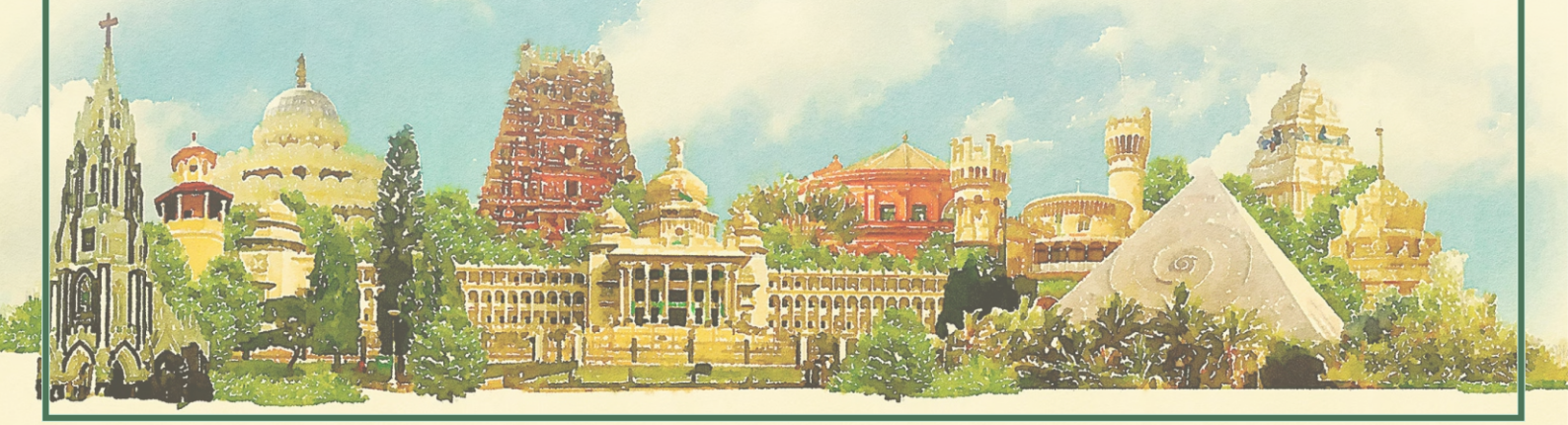
HOW TO BECOME MEMBER OF HAE SOCIETY OF INDIA



Hereditary Angioedema Society of India (HAESI)

Membership Application Form

Photograph

Name..... Gender (M/F).....

Age..... Date of Birth.....

Nationality.....

Qualification (Send a self-attested scanned copy of the highest degree).....

Speciality.....

Current position.....

Name of Hospital/Institute.....

Address for correspondence.....

.....

Mobile number.....

Email ID: .....

Type of membership: Founder/Life/Associate/Corporate/ Institutional

Medical Registration number.....

Details of the payment (Cheque/Demand draft/NEFT).....

Membership fee: Founder members: INR 5,000; Life members: INR 3,000
Associate members: INR 500/year; Corporate members: INR 50,000/year

I hereby apply for the membership of Hereditary Angioedema Society of India (HAESI). I agree to abide by the rules and regulations of the society. I certify that the details submitted by me are true and any false information may entail cancellation of my membership from the society.

Signature.....

Date.....

Please send the filled Membership form to
haesocietyofindia@gmail.com

Name of Account: Hereditary Angioedema Society Chandigarh HASC
Account Number: 40149338861
Name of Bank: State Bank of India
Branch: Medical Institute Branch
Address: Sector-12, Chandigarh
IFSC code: SBIN0001524

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