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Goa Branch

HAESI
HEREDITARY ANGIOEDEMA SOCIETY OF INDIA

4th NATIONAL CONFERENCE OF HAE SOCIETY OF INDIA

10th November 2024

Hyatt Centric Candolim Goa

Organized by the

Hereditary Angioedema Society of India

In collaboration with the

Indian Association of Dermatologists, Venereologists and Leprologists (IADVL), Goa Branch

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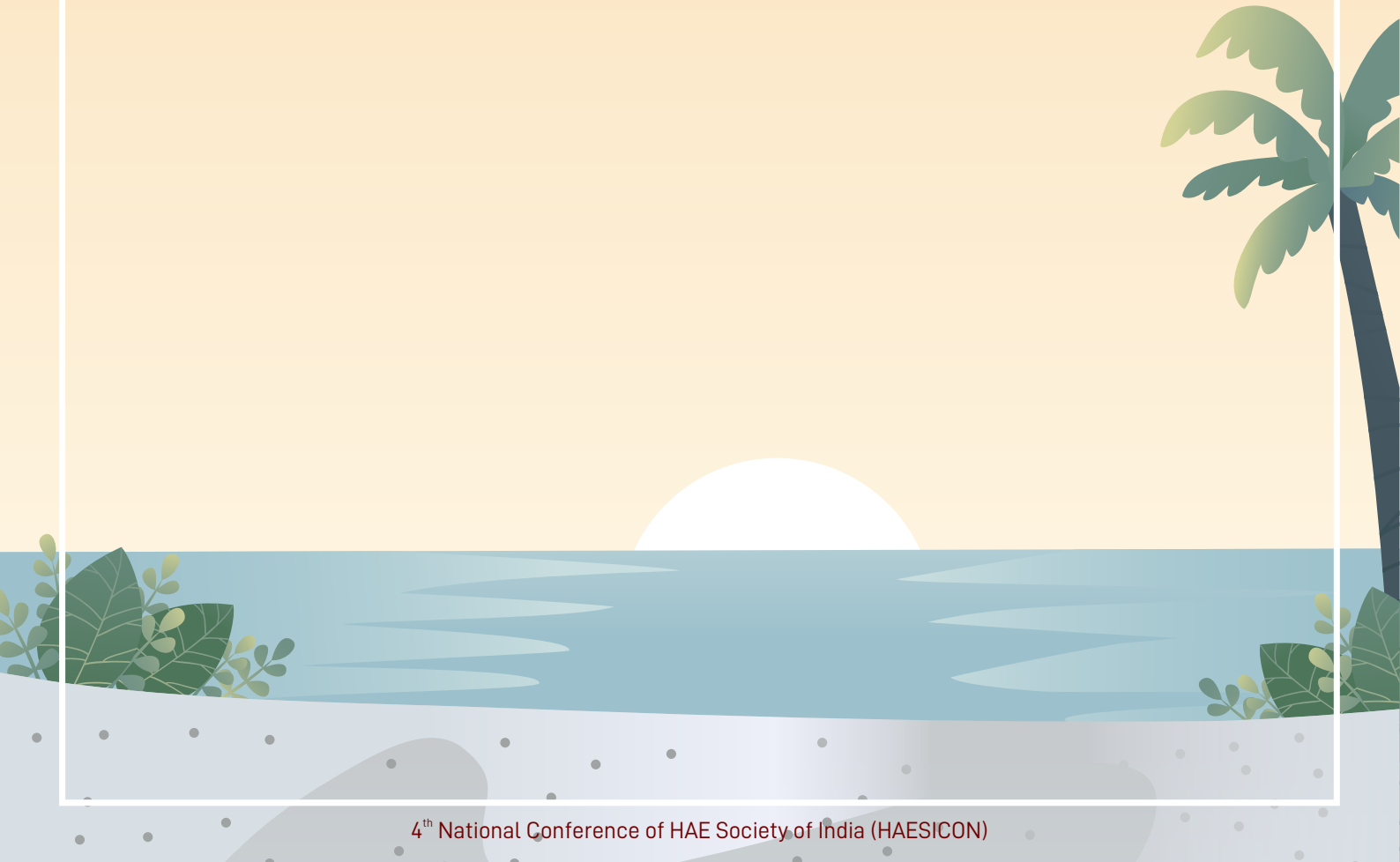
WELCOME MESSAGE



Invitation

Dear Colleagues,

Welcome to the **4th National Conference of HAE Society of India (HAESICON)!**

It is an honour for us to welcome the global community of professionals united by a commitment to tackling hereditary angioedema **(HAE)**. This conference is a vital platform for the exchange of pioneering research, clinical insights, and patient-centred approaches that have the potential to redefine HAE care and transform patient outcomes.

The Hereditary Angioedema Society of India (HAESI) was founded in February 2021 with an aim to increase awareness and to promote knowledge about HAE amongst physicians, to improve quality of life of patients and bring better treatment options for HAE in India. Over the last 3 years, society has made a substantial contribution towards these goals.

We are now hosting the **4th National Conference of HAE Society of India (HAESICON)** in **Goa, India** on Nov 10, 2024. We are honoured to have **Dr Hilary Longhurst** (New Zealand), **Dr Markus Magerl** (Germany), **Dr Thomas Buttgereit** (Germany), and **Dr Jane Wong** (Hong Kong) along with national experts on HAE as faculty for this conference. To encourage young talent, we have invited **award paper presentations** from fellows, students and rising star faculty.

"Welcome to **Goa – A rich blend of Portuguese and Indian heritage**, where vibrant culture meets the serene beauty of India's western coast! This incredible location is more than just sun and sand; it's a place where diverse ideas converge and creativity thrives. As we come together here, let's embrace the spirit of exploration, collaboration, and open-mindedness that Goa embodies. Here's to inspiring discussions, meaningful connections, and memorable moments.

Welcome to this academic event with opportunities of research collaborations & networking and let's make this conference a milestone in the journey toward progress and hope in **HAE!**

Prof. Sunil Dogra
Organizing Chairperson

Dr. Naguesh Pai Kakode
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Dr. Ankur Jindal
Organizing Secretary

Dr. Chetna Khemani
Co-organizing Secretary

Dr. Diksha Ghanshyam Phadke
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SCIENTIFIC PROGRAM

Scientific Programme

Time	Title	Speaker	Chairperson
0800 - 0830 hrs	**Registration (2nd Floor)**		
0830 - 0900 hrs	**Postgraduate Quiz**	Samipa Samir Mukherjee	
0900 - 0915 hrs	**Introduction and Welcome**	Sunil Dogra Markus Magerl Ankur Jindal	
0915 - 1015 hrs	**Epidemiology of Hereditary Angioedema in the Asia Pacific** **1. Hong Kong** **2. India** **3. Nepal** **4. Malaysia** **5. Sri Lanka**	 Jane Wong Ankur Jindal Sudip Prajauli Adli Ali Chandima Jeewandara	 Sendhil Kumaran Naguesh Kakode Vaishali Joshi
1015 - 1045 hrs	**High Tea**		
1045 - 1105 hrs	**Approach to Angioedema: A clinican's perspective**	Markus Magerl	Yasmeen J Bhat Kiran Godse
1110 - 1130 hrs	**Family screening in HAE**	Jane Wong	Pankaj Shukla Jagdish Goyal
1135 - 1155 hrs	**Laboratory diagnosis of HAE including upcoming biomarkers**	Thomas Buttgereit	Sheela Nampoorthiri Amit Rawat
1200 - 1230 hrs	**Angioedema Potpouri** **1. An overview of ACARE and its activities** **2. HAE International** **3. HAE India**	 Thomas Buttgereit Fiona Wardman Shaibal Guha	Bikash Ranjan Kar Pravalika M Dharvinder Kumar
1230 - 1330 hrs	**3rd Dr. Hilary Longhurst Oration** **Title: "My journey into angioedema with Prof. Marcus Maurer"**	Markus Magerl	Sunil Dogra Ankur Jindal
1330 - 1430 hrs	**Lunch (restaurant on the ground floor)** **Poster walk, Moderated by:** **Markus Magerl, Jane Wong, Aman Gupta, Pratap Patra**		
1430 - 1500 hrs (5+2 minutes each)	**Award paper presentations (4 abstracts)**	Shania Vij Jyothi Janardhanan Sathish L Sanghamitra Machhua	Satyaki Ganguly Neeraj Gupta Sankar J
1500 - 1520 hrs	**Androgen therapy in HAE**	Markus Magerl	Chetna Khemani Manobalan K

Scientific Programme

Time	Title	Speaker	Chairperson
1525 - 1545 hrs	**Coffee Break**		
1545 - 1605 hrs	**Challenging case discussion** **1. A case of urticaria with angioedema** **2. A case of hereditary angioedema** **3. A case of acquired angioedema**	Sendhil M Kumaran Ahmed Zaid Jamal Dev Desai	Binay Biswas Roohi Rasool Dhanesh Volvoikar
1610 - 1630 hrs	**Debate:** • FFP should be the first line on-demand treatment for patients with HAE in India • FFP should *not* be the first line on-demand treatment for patients with HAE in India	Archan Sil Himanshi Chaudhary	Sankar VH Hima Gopinath
1635 - 1655 hrs	**C1-inhibitor replacement therapy in HAE: when and how?**	Anjani Gummadi	Dipti Shripad Pujari Diksha Phadke
1700 - 1720 hrs	**Are we looking for a potential cure for HAE?: Results from the kallikrein gene knockout study**	Hilary Longhurst (virtual)	Aaqib Banday Sitesh Roy
1720 - 1730 hrs	**Announcement of results of PG Quiz and prize distribution and valedictory**	Sunil Dogra Sendhil M Kumaran Samipa Samir Mukherjee	

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ORAL ABSTRACTS

OA1: Mutation spectrum of *SERPING1* gene in patients with hereditary angioedema from India

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

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ABSTRACT

Introduction: Hereditary angioedema (HAE) is an uncommon genetic disorder characterized by recurrent episodes of swelling affecting subcutaneous and/or submucosal sites. It is caused by pathogenic variants in the Serpin family G member 1 (*SERPING1*) gene, which encodes the C1-inhibitor (C1-INH) protein.

Objectives: In this study, we aim to report the spectrum of *SERPING1* gene mutations in a large cohort of patients with HAE in 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. *SERPING1* gene was tested by targeted next generation sequencing, sanger sequencing, and/or multiplex ligation-dependent probe amplification for detection of large deletions.

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 1a). However, no variant was detected in 45 patients. Missense mutations (56.55%) constituted the majority of mutations, followed by splicing defects (12.6%), nonsense mutations (12.41%), frameshift mutations (7.58%) and deletions (5.26%) (Figure 1b). Exon 8 was observed as the most affected exon in our cohort, whereas exon 4 was the least affected.

Conclusions: This study highlights the variability in genetic profiles of HAE in India, highlighting the importance of genetic testing in diagnosis and management.

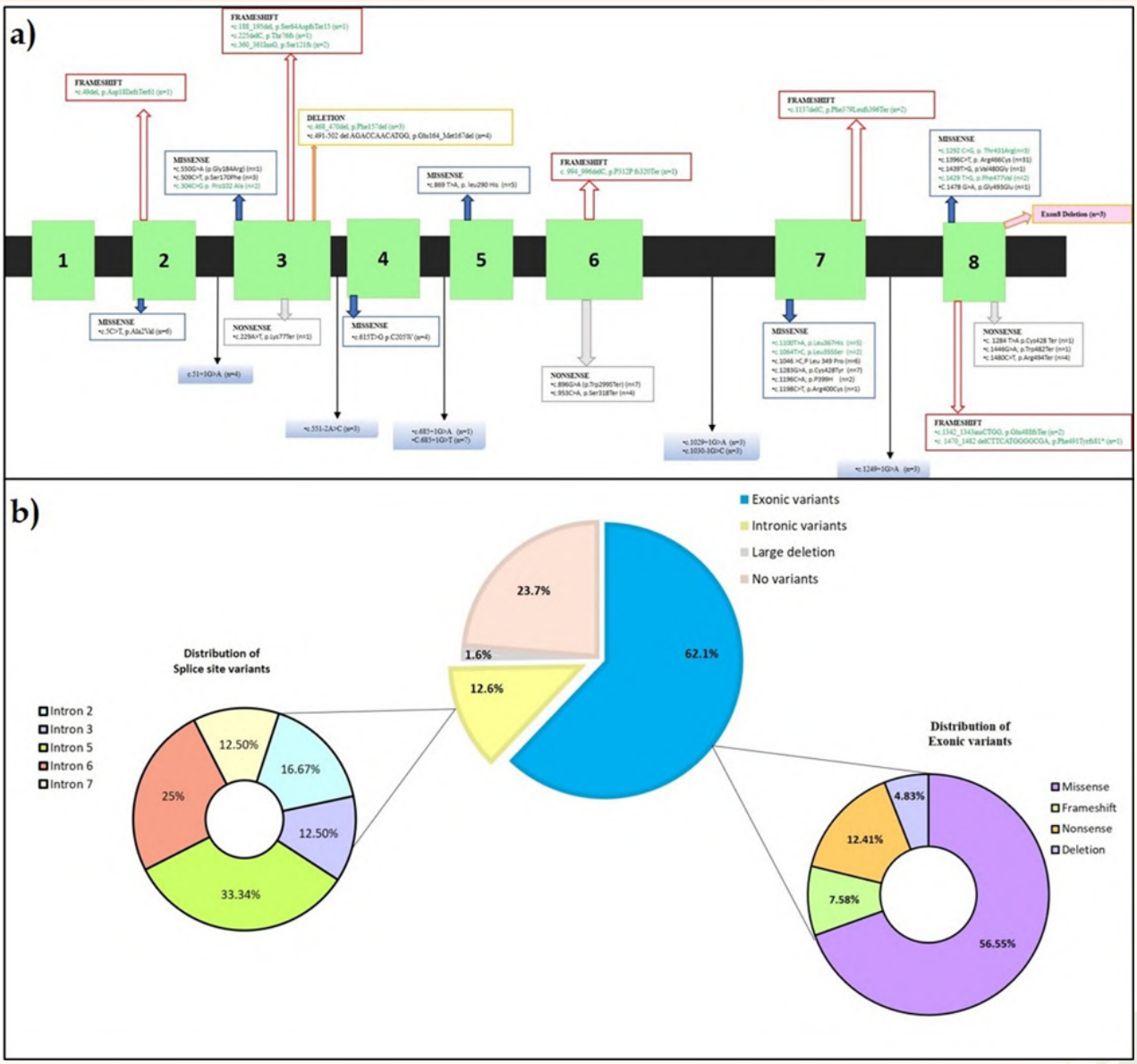

Figure 1. a) Different mutations identified in coding exons of *SERPING1* gene; b) Mutation spectrum of *SERPING1* gene identified in Indian cohort (n=190)

OA2: A case series of Hereditary Angioedema from a tertiary care center in South India

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Department of Pediatric Immunology and Rheumatology, Aster CMI Hospital, Bengaluru, India

Abstract

Background: Hereditary angioedema (HAE) is an autosomal dominant condition that is characterized by the local development of edema of the skin, subcutaneous tissue, and the walls of almost any organ, including the gastrointestinal and upper respiratory tracts.
Objective: To describe the clinical characteristics of seven patients with hereditary angioedema at a tertiary care center in South India.

Patients and methods: Case record files of patients registered in our hospital's Pediatric Rheumatology and Immunology clinic during the study period (February 2017 to September 2024) were studied. Seven patients were diagnosed with HAE during this period. Data regarding demographics, the onset of disease, time to diagnosis, frequency of attacks per year, and disease treatment were collected.

Results: Seven patients were diagnosed with HAE during the study period. The mean age at diagnosis was 29.5 years with M: F ratio of 4:3. The Mean delay in the diagnosis was 11.9 years (range: 6 months - 42 years). All patients had typical features of HAE with recurrent angioedema. A positive family history was noted in 42 % of these patients. Recurrent abdominal pain was noted in 50% of patients. However, one patient developed cavernous sinus thrombosis. The mean frequency of attacks at presentation was 12 per year (range:8-14). C1 esterase inhibitor levels were low in all patients. Genetic testing (exome sequencing) was done in three patients which was normal in two patients and P5 had a pathogenic mutation in the *SERPING1* gene. Four patients attained disease control with tranexamic acid, whereas two patients had a reduced attack frequency after tranexamic acid initiation. One patient (P1) required multiple FFP transfusions, danazol, and tranexamic acid.

Conclusions: We present the clinical and laboratory profiles of seven patients with HAE. Tranexamic acid has been of some benefit in most patients. The availability of C1 inhibitors may change the future outlook for these patients in our setup.

Parameters	P1	P2	P3	P4	P5	P6	P7
Age at onset (in years)	30	6	25	12	3	35	12
Age at diagnosis (in years)	37	6.5	67	14	8	58	16
Sex	Male	Male	Male	Female	Female	Male	Female
No. of episodes (per year)	10-12	8-10	10-12	10-14	10-12	10-12	8-10
Family history	Nil	Nil	Yes	Yes	No	Yes	No
C1 esterase levels(g/L)	<0.05	0.32	0.65	0.03	0.04	0.02	0.02
Genetic test (WES)	Negative	Negative	Not done	Not done	SERPING1	Not done	Not done
Treatment	Tranexamic acid, danazol, FFP	Tranexamic acid	Tranexamic acid	Tranexamic acid	Tranexamic acid	Tranexamic acid	Tranexamic acid
No. of attacks after initiation of treatment (per year)	Nil	Nil	Nil	1-2	Nil	1-2	LF

WES: whole exome sequencing; LOF: lost to follow up

OA3: A rare thrombotic complication in hereditary angioedema

AUTHORS:

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Introduction

Hereditary angioedema (HAE) typically presents with episodes of unpredictable subcutaneous and submucosal swelling. Thrombotic complications are rarely associated with hereditary angioedema. HAE patients also have evidence of systemic activation of coagulation including elevated plasma prothrombin fragments, thrombin antithrombin complexes and D-dimers. The proposed pathophysiology is C1INH is a multifunctional serine protease inhibitor that functions as a major endogenous negative regulator of the kallikrein-kinin, contact pathway of coagulation and complement systems.

Abstract

A 56-year-old gentleman, was evaluated for recurrent abdominal pain from 24 years of age. He consulted gastroenterology and suspected pancreatitis, gastroscopy showed multiple ulcers and the biopsy showed features of helicobacter pylori infection and treated for the same. His abdominal pain was recurrent, and not resolved with proton pump inhibitors, H2 blockers and managed symptomatically. At 38 Years of age, he had pain in the right hand for 2 months followed by discoloration of the right index finger for 2 days. Differentials of vasculitis, homocystinuria, and cervical rib were considered and evaluated, blood investigations showed normal homocysteine levels and lipid profiles. Connective tissue disease workup showed ANA-Negative, Anticardiolipin, lupus anticoagulant, Rheumatoid factor, C-ANCA/P-ANCA and cryoglobulins were normal. Complements levels showed low C4 (<5.6; N:10-40 mg/dl) and normal C3(118; N: 90-180mg/dl). Ultrasound doppler showed thrombotic occlusion of the proximal ulnar artery with poor distal reformation by collaterals. He was started on oral corticosteroids and dose-tapered over six months. Anti-coagulation heparin was started initially and switched to warfarin later, doses were titrated according to the pro-thrombin time. Disease-modifying agents methotrexate, sulfasalazine, and hydroxychloroquine were added because mixed connective tissue disease was considered clinically. He lost to follow-up. His family history is very significant, the index patient's daughter, granddaughter, and aunt had similar symptoms and C4 levels were low, and diagnosed hereditary angioedema probable type 1. He is non-smoker and non-alcoholic and developed stroke15 years later evaluated elsewhere, currently alive and on regular rehabilitation.

Here, we report one of the rare thrombotic complication associated with hereditary angioedema.

Conclusion

Venous thromboembolism is rarely associated with Hereditary angioedema. This case, we highlight the importance of rare presentation and neglected HAE diagnosis and stress the importance of early recognition and prompt treatment.

OA4: *SERPING1* gene variants and phenotypic correlations in patients with hereditary angioedema: a single centre cohort study from India

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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. Previous reports have shown that the distribution of SERPING1 gene variants and their clinical implications may differ among various ethnic populations. To date, no study from India has investigated these genotype-phenotype correlations in HAE patients.

Objectives: To analyse *SERPING1* gene variants and their correlations with clinical phenotypes in patients with C1-INH-HAE.

Methods: In this prospective study, we enrolled 190 patients with C1-INH-HAE from the Pediatric Immunodeficiency Clinic of a tertiary care referral hospital in North India between January 1996 and June 2024. The mutation spectrum in the *SERPING1* gene was analysed using Sanger sequencing, targeted next-generation sequencing (NGS), and multiplex ligation-dependent probe amplification (MLPA). The clinical phenotypes were correlated with the specific *SERPING1* variants and the specific exon involved. Multivariate logistic regression was employed to identify significant genotype-phenotype correlations, with statistical significance at $p<0.05$.

Results: Pathogenic variants in the *SERPING1* gene were identified in 76.31% (n = 145) of the patients. A significant positive correlation was found between prodromal symptoms in the patients affected with missense mutations (Odds ratio (OR): 3.147; 95%CI: 1.31-7.555; p=0.01) as well as with those affected with nonsense mutations (OR: 3.289; 95%CI: 0.996-10.856; p=0.05). Mutations in exon two were significantly associated with genital involvement (OR: 5.88; 95% CI: 0.97–35.66; p = 0.05), while mutations in exon eight were associated with upper extremity involvement (OR:3.48; 95% CI: 1.09–11.11; p = 0.035) and a higher likelihood of experiencing triggers (OR: 4.20; 95% CI: 1.44–12.20; p =0.008). Patients with splicing defects are 0.28-fold less likely to experience triggers than patients with other variants (OR:0.287; 95%CI:0.096-0.854; p=0.025). Our findings also show a significant negative correlation between the presence of a frameshift mutation and the occurrence of the disease in family members (OR:0.103; 95%CI:0.186-0.577; p=0.010). In addition, a significant positive

correlation was observed between missense variants and HAE Type 2 mutations (OR:5.773; 95%CI:1.85-18.001; p=0.003).

Conclusions: The observed genotype-phenotype correlations provide valuable insights into the clinical variability observed in HAE and may facilitate the development of personalized approaches to diagnosis, treatment, and risk assessment.

OA5: A case report of a patient of Type 1 Hereditary Angioedema with Gynaecomastia – Is Stanozolol the Culprit?

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Senior Resident, Allergy Immunology Unit, Advanced Pediatrics Center, PGIMER, Chandigarh.

Introduction: Hereditary angioedema (HAE) due to C1 inhibitor (C1INH) deficiency is an autosomal dominant disease characterized by discrete, recurrent episodes of non-urticarial angioedema that can involve the face, oropharynx, extremities or abdomen. Long term prophylaxis (LTP) is individualized and is commonly provided with anabolic androgens (danazol, stanozolol), tranexamic acid or a combination of both.

Case report: A 19-year-old boy, apparently well till 04 years of age, presented to us at 12 years of age with complaints of recurrent episodes of swelling predominantly involving the perioral, periorbital areas and throat (s/o laryngeal edema), with each episode lasting for about 3-4 days and were self-resolving. He was accordingly, suspected and evaluated for HAE. Both his C4 and C1INH levels were very low. ANA was normal. Targeted NGS confirmed presence of SERPING1 pathogenic mutation. He was thus diagnosed to have HAE Type 1. Initially, he was started "on demand" treatment for acute attacks of angioedema with FFP, which on follow up was switched over to Plasma derived-C1-INH therapy, when it became available in India. He was also started on LTP with Tab Stanozolol and tranexamic acid. Following this, the severity and number of acute attacks reduced.

Around 6 months back, the parents complained of localised swellings around both the nipples. On examination, he was confirmed to have bilateral gynaecomastia. The child at the time was on Stanozolol (2 mg/day) and Tranexamic Acid (20 mg/kg/day). A Paediatric Endocrinologist opinion was also taken and he was evaluated for gynaecomastia. His FSH, LH levels were normal but his 17 β Estradiol levels were found to be elevated - **45.3 pg/ml (7.03 – 42.6)**. In absence, of any other cause, his gynaecomastia was considered to be due to Stanozolol causing Hyperestrogenemia.

Discussion: Multiple studies documenting the adverse effect profile of anabolic androgen use in patients of HAE have been published. Amongst the common adverse effects attributed to chronic usage of androgens are weight gain, virilization and menstrual irregularities in females. However, any adverse effects specific to males have not been reported. Furthermore, Stanozolol may lead to dose dependant increase in aromatase expression resulting in increased conversion to estrogens. However, upon review of literature, LTP with Stanozolol in HAE has not been reported to cause gynaecomastia secondary to Hyperestrogenemia and thus we report this case.

Conclusion: Androgens such as Stanozolol have been effective drugs for LTP in HAE but associated with an ever-increasing adverse effect profile. It is imperative to identify these adverse effects promptly and modify therapy accordingly.

OA6: Acquired Angioedema - a case series

AUTHORS:

Dev Desai[1], Vinay K[2], Amit Rawat[1], Samipa Samir Mukherjee[3], M Sendhil Kumaran[2], Sunil Dogra[2], Ankur Kumar Jindal[1]

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Dr. Samipa's Skin Hair and Nail Clinic and Cloud Nine Hospital, Bangalore, India

Introduction:

Acquired angioedema (AAE) is a form of bradykinin-mediated angioedema with onset in adulthood, usually beyond the fourth decade of life. Many cases of AAE are associated with underlying lymphoproliferative or autoimmune disorders. AAE if not diagnosed and treated timely, can lead to significant inconvenience to the patient and can rarely cause fatalities by suffocation due to laryngeal involvement. Here we describe the epidemiology and clinical manifestations of 4 AAE patients who were evaluated and managed at our centre.

Methodology:

Retrospective review of patient records.

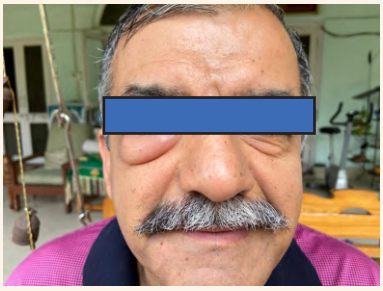
Case series:

2/4 patients were male. The onset of symptoms in all 4 cases was seen in the fourth or fifth decade of life. All patients had episodes of swelling of hands and feet, 3 patients presented with episodes of abdominal pain, 2 patients each had periorbital and perineal (scrotal and vulval) involvement, 3 patients had laryngeal involvement and 1 patient had required intubation in three episodes. Serum C4 was found to be low in 2/3 patients in which it was tested while serum C1 estrase inhibitor was low in all patients. A workup for underlying lymphoproliferative or autoimmune disorders was done for all the 4 cases- patient 1 was found to have a B cell non-Hodgkin lymphoma, no underlying etiology was found in patient 2, patient 3 had ANA 3+ positivity but no clinical symptoms suggestive of any autoimmune disorder, while patient 4 was found to have benign monoclonal gammopathy. Sanger sequencing for SERPING1 gene showed no pathogenic variant in patient 2, while results are awaited for the other 3 patients. All 4 patients were started on Tranexamic acid and on demand replacement of plasma-derived C1 esterase inhibitor (pdC1inh). Patient 1 also received Rituximab for the underlying B-cell NHL.

Conclusion:

AAE should be considered as a differential diagnosis for patients who have onset of non-urticarial angioedema in adulthood. Underlying lymphoproliferative and autoimmune disorders should be screened for in all suspected AAE cases. Education of patients and their primary healthcare providers about the nature of the disease and management of acute attacks can prevent morbidity and mortality in these patients.

Figure 1: Angioedema involving face and feet in patient 1



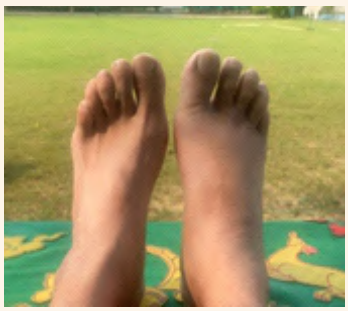


Figure 2: Angioedema involving face and lips in patient 2

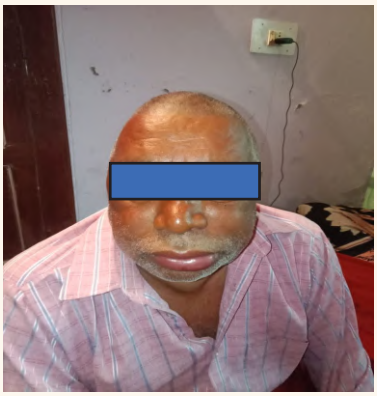
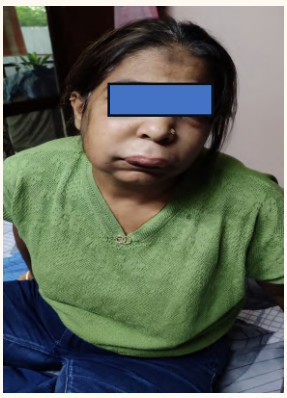


Figure 3: Angioedema involving face and hands in patient 3



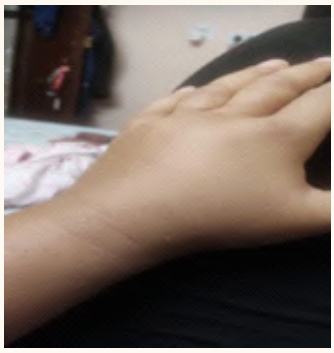
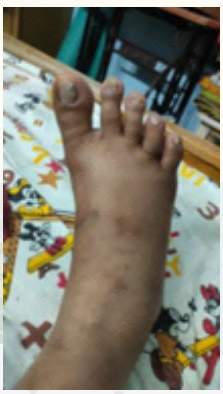


Figure 4: Angioedema involving hands and feet in patient 4



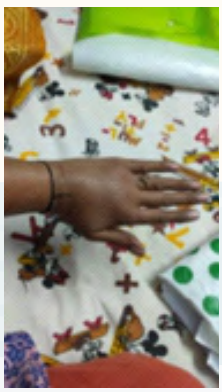


Table 1:
Epidemiology and clinical features of 4 AAE cases.

Patient	1	2	3	4
Gender	M	M	F	F
Current age	59	61	44	51
Age at onset	55	45	44	46
Symptoms	Swelling of hands and feet, periorbital, pain abdomen	Swelling of hands, feet, periorbital area and scrotum	Swelling of hands, and feet, hoarseness of voice, abdominal pain	Pain abdomen, swelling of hands and arms, swelling of the perineal region
Need for intubation	No	3 episodes	No	No
C4 (mg/dL)		7.9 (10-40)	7.1 (10-40)	10.37 (9-36)
C1 INH (mg/L)	95 (195-354)	68 (210-390)	90 (190-370)	160 (250-380)
C1 INH function		7% (>68%)		
Secondary cause workup	B cell NHL (monoclonal lymphocytosis), normal Ig profile, normal SPE	ANA neg, CBC normal	ANA 3+, SPE normal, no lymphadenopathy	Benign monoclonal gammopathy
Genetics	Awaited	No pathogenic variant in SERPING1 gene	Awaited	Awaited
Management	pdC1INH, Danazol, Tranexamic acid, Rituximab	Tranexamic acid, pdC1inh	Tranexamic acid, pdC1inh	Tranexamic acid, pdC1inh

HAESI
HEREDITARY ANGIOEDEMA SOCIETY OF INDIA

POSTER ABSTRACTS

PA1: Living with Hereditary Angioedema: Unseen Struggles and Barriers to Care in India

AUTHOR:

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Introduction:

Hereditary angioedema (HAE) is characterized by unpredictable and potentially life-threatening attacks, placing a significant physical and emotional burden on patients and their families. There is limited data available on the quality of life (QoL) in the HAE patient population from the Indian subcontinent.

Objective:

To assess the psychological impact and identify factors contributing to inadequate or delayed treatment in patients with HAE.

Methods:

A questionnaire was distributed to eight HAE patients who visited Ankura Hospitals for Women and Children, Hyderabad, between April 2022 and September 2024. The responses were recorded and analyzed.

Results:

Out of the seven patients who responded, six were male. Swelling of the extremities occurred in 42.9% of patients, while 14.3% experienced episodes involving the face, neck, and abdominal pain. Airway involvement was reported in 28.6% of patients. Hospitalization was required for 71.5% of patients, with one patient requiring intubation. The average attack duration was 2-3 days for 85% of the patients. Despite this, 58% preferred to wait and observe their symptoms rather than seeking immediate medical consultation. Although 85% of patients believed that antihistamines, hydrocortisone, or steroids provided symptom relief, none had used C1 esterase inhibitor replacement for treatment or prophylaxis. Financial constraints were identified as a barrier to treatment by 71% of the patients. A majority (85.7%) reported impaired QoL, with 57% citing social stigma, leading them to conceal their condition. Furthermore, 85.7% experienced anxiety, depression, or fear of traveling. All patients expressed concerns about facial disfigurement, fear of future attacks, and the negative impact of the disease on their career and educational opportunities.

Conclusion:

This study highlights the significant burden of HAE, including the social stigma and psychosocial impact experienced by patients, as well as the financial barriers to effective treatment

PA2: Clustering of cases of Hereditary Angioedema in two families: An experience from a tertiary care center of Eastern India

AUTHORS:

Deblina Patro[1], Habib Mostofa[1], Ritajyoti Ghosh[1], Ankita Debgupta[1], Fahad Siddiqui[1], Taraknath Ghosh[2], Kanai Lal Barik[3], **Archan Sil**[4]

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Background: Hereditary angioedema (HAE) is an autosomal dominant disorder, mainly caused by pathogenic variants in the *SERPING1* gene. The genetic profile of HAE may vary in different countries.

Objective: To report the clinical, laboratory, and genetic profile of patients with HAE from two families in West Bengal, India.

Methods: We reviewed records of patients diagnosed to have HAE in our unit. There are 7 patients from 2 families, residing in two different districts of West Bengal. Clinical, laboratory, and molecular characteristics of these patients were studied in detail.

Results: There were 7 patients with HAE from 2 families. Median age at onset of symptoms and median age at diagnosis were 29.5 (range: 12-55 years) and 35.5 years (range: 17-67 years), respectively. Median delay in diagnosis was 6 years (range: 5-12 years) with a male-to-female ratio of 1:1. Predominant sites of involvement were face, eyelids, lips, tongue, back, hands, feet, and genitalia. While a history suggestive of laryngeal oedema was elicited from 1/7 (14.3%) patient, abdominal symptoms were present in 4/7 (57.1%) patients. Prodromal symptoms varied from tingling and itching to pain at the local site. Trauma was the commonest trigger of acute attacks, followed by emotional stress. The C4 level was reduced in all the patients with a mean level of 5.36 mg/dL. C1-INH level was reduced in 6 patients, indicative of type-I HAE. One patient was diagnosed as type-2 HAE based on the C1-INH function. Pathogenic variants were identified in the *SERPING1* gene in both the families. No link could be established between these 2 families. One patient received on demand treatment with C1-INH and rest were on long term prophylaxis with tranexamic acid and danazol.

Conclusion: High index of suspicion is required to diagnose this disorder in all patients presenting with angioedema without urticaria. We report 6 cases of type 1 HAE and 1 patient with type 2 HAE from two families in Eastern India.

PA3: Hereditary Angioedema in 3 generations in a family in Eastern India

AUTHORS:

Dr. Debasis Patro[1], Dr. Smita Satapathy[2], Dr. J. Bikrant Ku Prusty[3], Dr. Rachita Sarangi[3]

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2. Consultant Pediatric Allergy and Immunology, IMS and SUM Hospital, Bhubaneswar.
3. Professor, Department of Pediatrics, IMS and SUM Hospital, Bhubaneswar.

Hereditary Angioedema (HAE) is an uncommon disorder with a global prevalence of approximately 1 in 10,000 to 1 in 50,000 population. The prevalence in India is underestimated due to a lack of awareness or diagnostic facilities. Here we present where this disease runs in a family affecting 3 generations.

A 9-year-old female from Odisha presented with swelling of the face, lips, neck, and around the eyes associated with mild difficulty in breathing and swallowing after being accidentally injured while walking. She responded fairly to anti-histaminics, a short course of steroids, and adrenaline nebulization. Similar history in the past after trivial trauma, which was first noticed at the age of 4 years and spontaneously resolved after 24-48 hours. After careful history taking, it was noticed that the mother had her first symptoms during pregnancy during the first trimester, which spontaneously resolved. Maternal uncle also has intermittent swelling of hands and foot after trivial trauma associated with pain in the abdomen which resolves spontaneously. Maternal grandmother, along with 3 other female siblings, also has similar complaints. History of death of one of the maternal aunts at the age of 30 years due to possible laryngeal edema. C1 esterase inhibitor levels in the proband were done, which was low (0.06 g/L) along with low C4. Suspecting HAE type 1, parents were counseled about the disease process and the need for fresh frozen plasma (FFP) as they are not affordable for C1 inhibitors in an emergency or before any dental /surgical procedure. They were also counseled about the need for family screening. Currently, the child is on long-term prophylaxis with anti-fibrinolytic, and she had a minor flare-up after a trivial trauma recently.

This case highlights the need for proper history-taking and pedigree analysis with family counseling in a child suffering from angioedema with or without urticaria. Simple screening tests like C4 levels during the attack along with C1 esterase inhibitor level could help us in suspecting this rare entity in those who fairly respond to 1st line management of urticaria.

PA4: HEREDITORY ANGIOEDEMA IN 10 YEARS OLD CHILD (Case Report)

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Dr. Dhanesh Volvoikar

AFFILIATIONS:

Consultant: Allergy Clinic, Goa

Introduction: Hereditary Angioedema (HAE) is rare disease having estimated prevalence of 1:10,000 to 50,000. In a state of Goa with15 lakhs population we should detect about 30 cases. But this is probably the only known worked up case from this state.

Case Report: This child has classical history with onset at 2 years of age. He gets lip swelling and eyelid swellings which usually, increase over 2 /3 days and then decrease over the next 3 days. Since then, a minimum of 2/3 similar episodes occur every year and subside on its own. He also gets once in 3/4 months pain in abdomen episodes lasting for 2/3 days. For one such episode child was admitted but hospital could not diagnose and child had spontaneous recovery. Serum C4 was 2.8 (decreased markedly from normal of 12-43 mg/dl), C1 Esterase Inhibitor was 0.05 is also markedly decreased from the normal range of 0.21-0.38g/L. Both these tests and clinical scenarios are diagnostic of HAE. Child's parents are now counseled and child is advised long term prophylaxis of Tranexamic acid. Emergency plan is written and efforts are being made to register this child under rare disease program to get appropriate support since parents cannot afford C1 INH enzyme concentrate. Child is also advised to join HAESI.

Conclusion: Because of lack of awareness these rare diseases remain undetected and risk of life threatening laryngeal angioedema always remains possibilities. Although the onset was at 2 years it took so many years for correct diagnosis and to place the child under standard treatment protocol which will now help him in leaving normal healthy life.

PA5: Rare type of hereditary angioedema in young child

AUTHORS:

Sathish Kumar Loganathan[1], Dharshini. S[2], Vivek Sugadev[1], John Jude[3]

AFFILIATIONS:

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2. Department of Dermatology and Venereology, Unit-1, Christian Medical College, Vellore, Tamilnadu
3. Department of Microbiology, Christian Medical College, Vellore, Tamilnadu

Introduction

Plasminogen gene mutation causing hereditary angioedema (HAE) is very rare in Pediatric population. Plasmin plays its role in bradykinin production via activation of factor XII. This mutation leads to change in Kringle 3 domain which alters the structure of the wild type protein. The mutant protein leads to increased production of the bradykinin thereby resulting in HAE. In this mutation, patients tend to have higher chance of head and neck swellings compared to extremity oedema and swelling of the genitals.

Case details

An 8-year 10-month-old female child, first born to non-consanguineous parents presented with recurrent swelling of lips, tongue, and eyes for the past 2 years. She was evaluated elsewhere, and blood investigations showed elevated immunoglobulin E- 2161 IU/L, diagnosed with allergic urticaria, and managed with ketotifen. Her swelling episodes and duration were frequent, visited our centre for further evaluation. We suspected hereditary angioedema and investigated further, investigations revealed normal C3/C4 and C1 esterase inhibitor (C3-125; N:90-180 mg/dl, C4-37.1; N: 10-40 mg/dl and C1INH-0.281; N:0.16-0.33g/l). A next-generation sequencing revealed a heterozygous mutation in plasminogen gene/exon 6. She was initiated on oral tranexamic acid, and symptoms improved.

Conclusions

Plasminogen gene mutation in HAE is rare in Pediatric population and here we report rare mutation in Indian child.

PA6: The Hidden Threat of Angiotensin Converting Enzyme Inhibitors in Angioedema

AUTHORS:

[1]Dr Vatsal Patidar, [2]Dr Kiran Godse, [3]Dr Sharmila Patil

AFFILIATIONS:

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[2]Professor of Dermatology, Department of Dermatology, D Y Patil School of Medicine, Navi Mumbai

[3]Professor and Head of the Department of Dermatology, Venereology & Leprosy, D Y Patil Medical College & Hospital & Research Centre

INTRODUCTION:

The rare yet, serious threat of Angiotensin Converting Enzyme inhibitors in causing Angioedema gets frequently overlooked by physicians. Angiotensin Converting Enzyme inhibitors and Angiotensin receptor blockers cause the accumulation of Bradykinin, which facilitates the symptoms of Angioedema. The poor response to conventional treatment modalities in these cases make it difficult to treat. Awareness amongst clinicians is needed to withdraw the offending drug promptly in order to treat ACEi induced Angioedema.

CASE REPORT:

A 52 year old male came to the casualty with complains of swellings over the face since 1 day. Patient was given antihistamines for the same by a doctor but was not responding to the treatment. There was no known food or drug allergies but was a known case of hypertension and had been taking Angiotensin Converting Enzyme inhibitors for the same. Resolution was achieved after stopping the ACEi and symptomatic treatment with corticosteroids and antihistamines.

CONCLUSION:

Angiotensin Converting Enzyme Inhibitors are a fairly commonly used medication in hypertensives. These patients may develop Angioedema and fail to respond to antihistamines and corticosteroids. Angioedema can be triggered by from 2 weeks to years after starting therapy with Angiotensin Converting Enzyme inhibitors.

Prompt withdrawal of these medications in cases of Angioedema is an important step in its management.

Awareness about the potential severe side affects of Angiotensin Converting Enzyme inhibitors and rarely Angiotensin Receptor Blockers is an important aspect of treating Angioedema.

PA7: SECONDARY ANGIOEDEMA AS PRESENTING FEATURE OF CHILDHOOD LUPUS

AUTHORS:

DR. RAKESH MONDAL, DR.SUBHASHREE DAS, DR.JAUHAR IQBAL

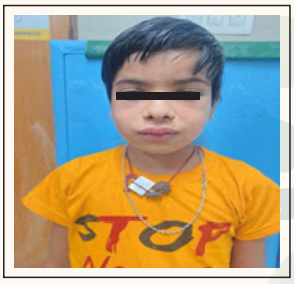
AFFILIATIONS:

Division of Paediatric Rheumatology unit, Dept. of Paediatric Medicine, Medical College and Hospital, Kolkata

Introduction: Angioedema is an immune mediated inflammation of skin or subcutaneous tissue that can be life threatening if it compromises the airway and it can be a rare presenting feature of systemic lupus erythematosus (SLE).

Case summary: 7 years 10 months old girl born out of non-consanguineous marriage with no significant birth history and past history presented to us with complaint of swelling of the lips and face along with distension of abdomen and intermittent pain abdomen for 2 years. Before presenting to us, the child was taken to many health facilities and many investigations were done. Initially she was diagnosed as a case of nephrotic syndrome and treated with tablet prednisolone which was stopped by parents abruptly. The initial lab reports showing microcytic, hypochromic anemia with normal iron profile, non-reactive serology, and mild ascites with multiple mildly enlarged lymph nodes in porta hepatis and peripancreatic area. Bone marrow biopsy report showed hypercellular marrow with negative markers of malignancy. Tuberculosis was also ruled out and further diagnosis of chronic liver disease was made due to deranged Liver function test and coagulation profile. Also, autoimmune hepatitis and Wilson disease were ruled out. Further liver biopsy was planned but due to financial constraints she was lost to follow up.

When the child presented to us, we found facial flushing, periorbital puffiness, edema around perioral area, b/l cervical lymphadenopathy and b/l parotid swelling with hepatosplenomegaly, ascites, and reduced air entry at lower zone of both lungs on auscultation. Further investigations showed parotitis on USG neck, bilateral pleural effusion, ascites 2+ with raised ESR, CRP and microcytic anemia with lymphopenia. As the presentation was quite surprising with no evidence of infection and malignancy and non responsive to treatment so we further proceeded to rule out autoimmune diseases. There was also no history of intake of drugs like ACE inhibitors or ARBs.



Surprisingly ANA and anti dsDNA titer came positive with hypocomplementemia (C3:49mg/dl, C4: 06mg/dl). As the clinical course and lab parameters were suggestive of SLE, we diagnosed the child as a case of childhood lupus where angioedema was a striking presentation.
After diagnosis she was given tablet HCQ along with prednisolone and the symptoms started to improve. Now the child is clinically stable and on follow up.

Conclusion: The definitive pathophysiology of acquired angioedema in SLE is still not much clear but there is an increasing concern about angioedema in SLE as well. SLE patients have demonstrated a significantly high number of acetylated modifications of C1-inhibitor (C1-INH) and high autoantibody titres for C1-INH while another study suggests that C1-INH levels can be normal in SLE individuals, but significantly less reactive to C1s and C1r with no identified autoantibodies or mutations. So timely diagnosis and management of angioedema which can be a rare but first presentation of lupus reduce the morbidity or fatal complications.

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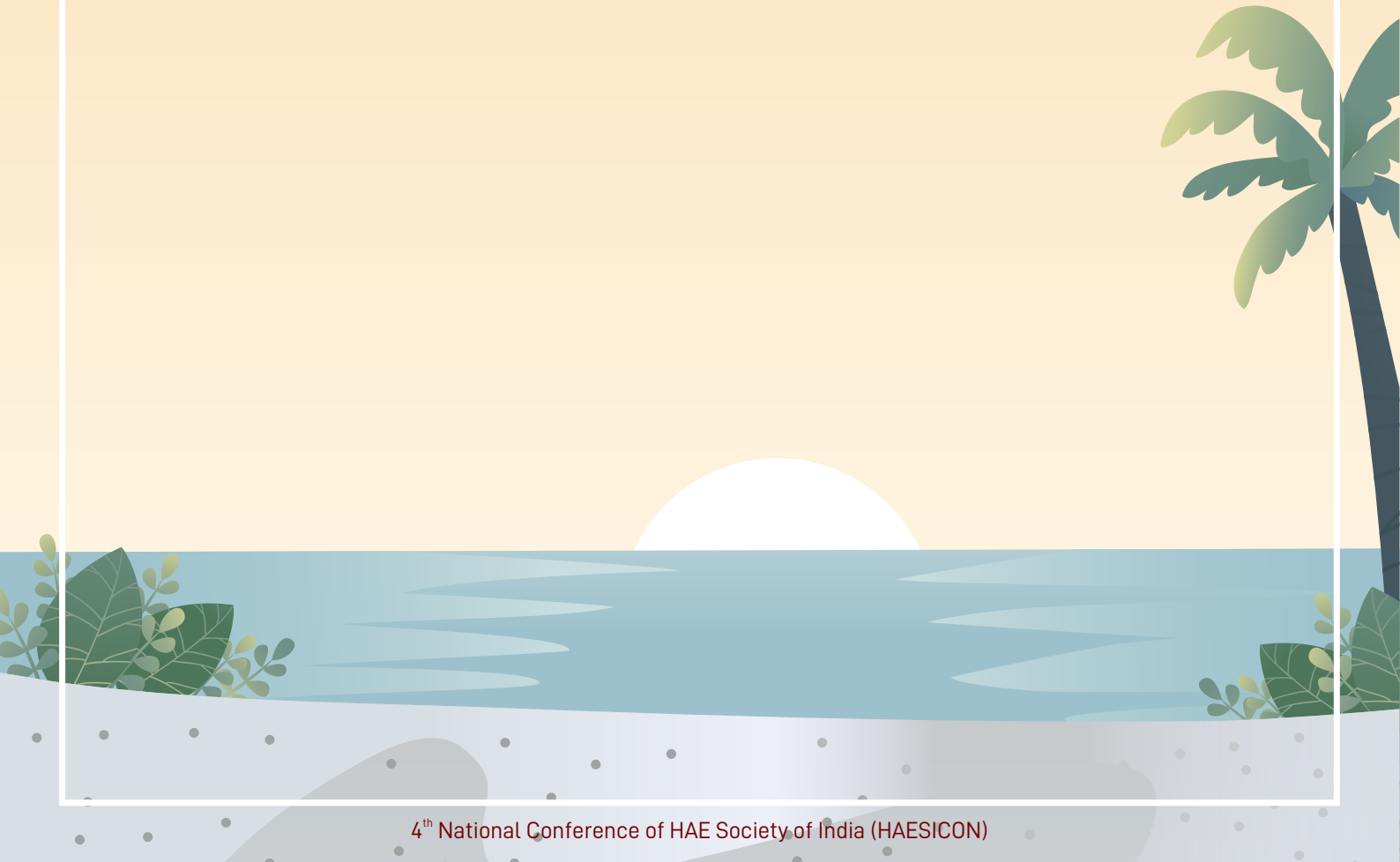
HOW TO BECOME MEMBER OF HAE SOCIETY OF INDIA



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 Demand Draft/Cheque to following address:
Dr. Ankur Kumar Jindal, Room no. 4116, 4th Floor A Block, Allergy Immunology Unit, Department of Pediatrics, Advanced Pediatrics Centre, PGIMER, Sector-12, Chandigarh- 160012,
Mobile: +91-95920-65559
Email: ankurjindal11@gmail.com;
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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