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Comprehensive Guide to URTICARIA

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CHAPTER
7

Angioedema

Rohit Batra, Anushruti Aggarwal, Sanjeev Gupta

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INTRODUCTION

Angioedema is swelling of the deep dermis, subcutaneous, or submucosal tissue due to vascular leakage. Acute episodes often involve the lip, eyes, and face; however, angioedema may affect other body parts, including respiratory and gastrointestinal (GI) mucosa. This condition can be acute or chronic and life-threatening if it involves the airway. Unlike urticaria, which affects the superficial dermis, angioedema involves the deeper dermis and subcutaneous tissues, leading to more pronounced swelling.

HISTORICAL PERSPECTIVE

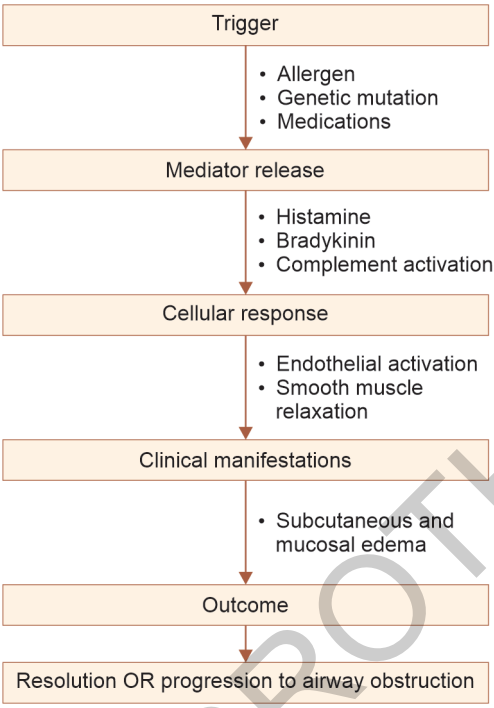
The term "angioedema" was coined by Heinrich Quincke in 1882, who described cases of recurrent, nonpitting edema of the skin and mucous membranes.

EPIDEMIOLOGY

While hereditary angioedema (HAE) is a rare genetic disorder, the more commonly seen subtypes are acquired and drug-induced forms. Angioedema can occur at any age, but hereditary forms often present in childhood or adolescence. Also, HAE is reported to be more common in females.

PATHOPHYSIOLOGY

Angioedema is due to a sudden increase in local vascular permeability in subcutaneous or submucosal tissue. Histamine and bradykinin are the most commonly involved vasoactive mediators. Bradykinin-mediated angioedema is primarily seen in hereditary and angiotensin-converting enzyme (ACE) inhibitor-induced forms, while histamine-mediated angioedema is typically related to allergic reactions. Other mechanisms, including prostaglandins, leukotrienes, and the complement system, also play a role in certain types of



FLOWCHART 1: Pathophysiology of angioedema.

angioedema. Understanding these mechanisms is crucial for effective diagnosis and management **(Flowchart 1)**. Let us discuss these mechanisms one by one.

Mast Cell-mediated Angioedema

Mast cell-mediated angioedema is usually associated with allergic reactions where histamine is released from mast cells and basophils in response to an allergen, leading to vasodilation and increased vascular permeability. It is the most common type of angioedema. Stress can be a trigger for this type.

Bradykinin-mediated Angioedema

Bradykinin-mediated angioedema results from the excessive production or impaired degradation of bradykinin, a potent vasodilator. Bradykinin increases vascular permeability and leads to fluid leakage into surrounding tissues, causing swelling. C1-inhibitor is a regulator of the complement pathway. If there is a deficiency of the C1-inhibitor, it leads to uncontrolled production of kallikrein leading to proteolysis of high molecular weight kininogen and bradykinin, leading to edema by increased vascular permeability.

Hereditary Angioedema

Hereditary angioedema is a genetic disorder characterized by recurrent episodes of severe swelling. It is further classified into three types. Type I HAE is due to a

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deficiency in C1 inhibitor (C1-INH), a protein that regulates the complement and contact systems. The mutated gene is the *SERPING1* gene. In type 2 HAE, the C1-INH levels are normal or elevated but the protein secreted is dysfunctional. Type 3 HAE often affects women and is influenced by estrogen levels. It is associated with mutations in the *F12* gene, which affects the factor XII protein involved in the production of bradykinin.

Acquired Angioedema

Acquired angioedema (AAE) develops later in life, often associated with other conditions. Type I is due to consumption of C1-INH by autoantibodies or associated with malignancies such as lymphoproliferative disorders. Type II AAE is associated with the production of autoantibodies against C1-INH.

Drug-induced Angioedema

Angioedema can be triggered by medications, particularly those affecting the bradykinin pathway.

- *ACE inhibitors*: They reduce the breakdown of bradykinin and blood and tissue levels of bradykinin rise. It presents with swelling of eyelids, lips, tongue, and airway can also be involved.
- *Nonsteroidal anti-inflammatory drugs* (*NSAIDs*): They can trigger angioedema through various mechanisms, including shifting the arachidonic acid pathway toward leukotriene production, which can cause mast cell degranulation. Angioedema typically involves the face, lips, and eyelids, often with urticaria.

Idiopathic Angioedema

Angioedema is said to be idiopathic only after ruling out all other causes by thorough evaluation. No allergic etiology is present, complement levels are normal and family history is mostly absent. It presents with recurrent episodes of angioedema without identifiable triggers.

Other Mechanisms

Other less common mechanisms include involvement of the prostaglandins, leukotrienes, and complement pathway. Prostaglandins and leukotrienes can increase vascular permeability and contribute to angioedema. Activation of the complement pathway can lead to the release of anaphylatoxins (C3a, C5a) that increase vascular permeability. Research shows that complement activation can play a role in some cases of idiopathic and autoimmune-related angioedema.

CLINICAL PRESENTATION OF ANGIOEDEMA

The hallmark of angioedema is localized swelling of the deeper dermis and subcutaneous tissue. Episodes of swelling usually develop over minutes to hours

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and can last for 24–72 hours. The clinical presentation can vary depending on the type and underlying cause of the angioedema.

Specific Clinical Features by Type of Angioedema

- *HAE*:
  - Recurrent episodes of swelling involving the skin and mucous membrane, occur often without an identifiable trigger. Gastrointestinal symptoms such as abdominal pain, nausea, vomiting, and diarrhea may be associated due to swelling of the intestinal walls.
  - Laryngeal edema can occur causing potentially life-threatening airway obstruction. Episodes may occur spontaneously or be triggered by trauma, stress, hormonal changes, or certain medications.
- *AAE*: It involves recurrent episodes affecting the skin, gastrointestinal tract, and respiratory system. It can be associated with autoimmune disorders or lymphoproliferative diseases. Age of onset is late as compared to hereditary angioedema.
- *Drug-induced angioedema*:
  - *ACE inhibitors*: Swelling typically affects the face, lips, tongue, and airway. It can occur at any time during treatment, even after years of use of the drug. Prompt recognition is important.
  - *NSAIDs*: Swelling may be associated with urticaria and often involves the face, lips, and extremities.
  - *Clinical course*: Symptoms usually resolve upon discontinuation of the offending drug. Treatment with epinephrine, antihistamines, or corticosteroids might be required in the management of drug-induced angioedema.
- *Idiopathic angioedema*:
  - *Presentation*: Recurrent episodes of angioedema without a known cause. Affected areas include the face, extremities, and genitals.
  - *Duration*: Episodes can vary in frequency and severity, often lasting for several days.
- *Allergic angioedema*:
  - *Symptoms*: Often occur with urticarial and can be triggered by allergens such as foods, insect stings, medications, and environmental factors.
  - In severe cases, it can be part of an anaphylactic reaction, characterized by systemic symptoms such as hypotension, bronchospasm, and shock.
  - *Management*: Immediate treatment with antihistamines, corticosteroids, and epinephrine is essential.

Special Considerations

As with other disorders, management of angioedema needs specific considerations in special groups like children, pregnancy, and elderly.

- *Pediatric angioedema:*
  - Clinical features are similar as in adult angioedema.
  - Infections, vaccinations, and trauma are common triggers in children.
  - Avoiding known triggers and close monitoring during episodes is required.

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- *Angioedema in pregnancy:*
  - *Hormonal influence:* Pregnancy can exacerbate hereditary angioedema due to hormonal changes.
  - *Treatment considerations:* Certain treatments may be contraindicated during pregnancy, necessitating careful management.
- *Angioedema in the elderly*:
  - *Age-related factors*: Comorbidities and polypharmacy can complicate diagnosis and management.
  - *Presentation*: Similar to younger adults, but with increased risk of drug-induced angioedema.

Comparison of Types of Angioedema

See **Table 1**.

Differential Diagnosis of Angioedema

See **Table 2**.

TABLE 1: Types of angioedema.

Type	Cause	Clinical features	Laboratory findings	Treatment
Allergic angioedema	Allergens (food, drugs, insect stings)	Urticaria, rapid onset swelling	Normal C4, elevated tryptase	Antihistamines, corticosteroids, epinephrine
Hereditary angioedema	Genetic mutation (C1-INH deficiency)	Recurrent, non-pruritic swelling, no urticaria	Low C4, low C1-INH levels	C1-INH replacement, bradykinin inhibitors
Angiotension-converting enzyme (ACE) inhibitor-induced	ACE inhibitors	Facial swelling, no urticaria	Normal C4, normal C1-INH	Discontinuation of ACE inhibitor, bradykinin inhibitors
Idiopathic	Unknown	Recurrent, episodic swelling	Normal C4, normal C1-INH, normal tryptase	Antihistamines, corticosteroids

TABLE 2: Differential diagnosis of angioedema.

Condition	Morphology of lesion	Distinguishing features
Angioedema	Swelling without redness, affects deep layers	Often asymmetric, can involve airway
Urticaria	Raised, red, itchy wheals, affects superficial skin	Transient wheals
Cellulitis	Red, warm, tender swelling, often with fever	Involves infection, spreads over time
Dermatitis	Itchy, red, inflamed skin, may blister	Often related to contact or irritant exposure
Lymphatic obstruction	Chronic, nonpitting swelling	Often due to cancer or infection

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DIAGNOSTIC APPROACH TO ANGIOEDEMA

Clinical History and Examination

- Detailed history of the symptoms, frequency and duration of episodes need to be taken. The sites affected including face, lips, tongue, throat, extremities, and gastrointestinal tract need to be noted. Any associated symptoms such as abdominal pain, difficulty breathing, urticaria, itching, or systemic reactions need to be taken into consideration.
- Triggering factors such as foods, medications, physical trauma, stress, hormonal changes, or recent infections need to be asked. Check for a family history of angioedema or related conditions to assess for hereditary forms.
- History of medicines, especially ACE inhibitors, NSAIDs, and estrogens. Consider underlying conditions such as autoimmune diseases, infections, or malignancies.

While performing clinical examination, assessment of affected areas for the extent, consistency, and presence of urticaria should be carried out. It is important to check for signs of airway compromise, such as stridor, hoarseness, or difficulty breathing. Any signs of gastrointestinal involvement, such as tenderness or distension, should be evaluated. Also, it is necessary to rule out any signs of systemic illness or malignancy.

Laboratory Investigations

Basic Tests

- *Complete blood count (CBC)*: To identify signs of infection or hematologic abnormalities
- *Electrolytes and liver function tests*: Important to rule out systemic causes
- *Thyroid function tests*: To check for thyroid abnormalities

Specific Tests for Angioedema

- *C1 esterase inhibitor (C1-INH) levels and function:*
  - *Type I HAE*: Low C1-INH levels and low C4 levels
  - *Type II HAE*: Normal or elevated C1-INH levels with dysfunctional protein and low C4 levels
- *C4 levels*: Typically low during and between attacks in HAE
- *C1q levels*: Decreased in acquired angioedema due to lymphoproliferative disorders
- *Bradykinin levels*: Elevated in bradykinin-mediated angioedema
- *C1-INH autoantibodies*: Present in acquired angioedema due to autoimmune mechanisms

Allergy Testing

- *Skin prick tests or serum-specific IgE*: To identify allergen triggers in cases of suspected allergic angioedema
- *Oral challenge tests*: Performed under medical supervision to confirm specific food or drug triggers

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Imaging Studies

Ultrasound or CT Scan

- *Abdominal imaging*: Useful in patients with gastrointestinal symptoms to assess for bowel wall edema
- *Airway imaging*: To evaluate the extent of laryngeal or pharyngeal involvement in severe cases

Genetic Testing

Genetic Analysis

- *SERPING1 gene mutations:* To confirm hereditary angioedema types I and II
- *F12 gene mutations*: To identify type III hereditary angioedema

Functional Tests

Bradykinin Pathway Assessment

- *Kallikrein activity*: To measure the activity of kallikrein, which is involved in bradykinin production
- *Bradykinin receptor function*: To assess the responsiveness of bradykinin receptors

Differential Diagnosis

Exclude Other Causes

- *Urticaria*: Differentiate between angioedema and urticaria by assessing the depth and nature of swelling.
- *Cellulitis*: Consider bacterial infection presenting as localized swelling and erythema.
- *Thrombophlebitis*: Assess for venous thrombosis presenting as unilateral limb swelling.
- *Lymphatic obstruction*: Rule out causes such as malignancy or infection leading to localized edema.

Provocative Tests

Provocation with Known Triggers

- *Physical triggers:* Cold, pressure, or vibration tests to identify physical urticarias that may present with angioedema
- *Medication withdrawal and rechallenge:* To confirm drug-induced angioedema under controlled conditions

MANAGEMENT OF ANGIOEDEMA

The management of angioedema depends on the underlying cause, the severity of the symptoms, and whether the condition is acute or chronic.

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General Principles

Initial assessment includes evaluating the severity of symptoms, especially if there are signs of airway compromise. Conduct a thorough history and physical examination to identify potential triggers and underlying causes.

Immediate Management

- *Airway management*: Ensure the airway is patent. In cases of laryngeal edema, intubation or tracheostomy may be needed.
- *Symptomatic relief*: Provide symptomatic relief for pain and swelling.

Specific Management based on Type of Angioedema

Hereditary Angioedema

Acute Attacks

In HAE types I and II, the treatment of choice in acute attacks consists of infusion of C1-INH concentrate (20U/kg) or ecallantide. Another option is icatibant.

- *C1 Inhibitor concentrate*: C1-INH concentrates are available which are derived from pooled plasma (Berinert, Cinryze) and recombinant C1 INH (Ruconest). It is approved for the treatment of acute HAE attacks. The dose is 20 U/kg body weight intravenously. Side effects such as infections and dysgeusia can occur. Cinryze can be given every 3–4 days in a dose of 1,000 units.
- *Bradykinin receptor antagonist (icatibant)*: It is a selective bradykinin 2 receptor antagonist, approved by the Food and Drug Administration (FDA) for use in patients 18 years and more. A 30-mg dose is given subcutaneously, which can be repeated after 6 hours. Maximum it can be given up to three times in 24 hours. Few side effects such as nausea, rash, headache, and fever can occur.
- *Kallikrein inhibitor (ecallantide)*: Use to reduce bradykinin production. It can be given to patients 12 years or older. Three subcutaneous injections of 10 mg each are given. It should only be given by healthcare professionals as risk of anaphylactoid reaction is present. The side effect profile is similar to icatibant.

Long-term Prophylaxis

- *C1-INH concentrate*: Regular prophylactic administration in patients with frequent or severe attacks
- *Androgens (danazol)*: Used in low doses (200 mg oral danazol) to increase hepatic synthesis of C1-INH, though their use is decreasing due to side effects
- *Antifibrinolytics (tranexamic acid)*: Consider in patients who cannot tolerate C1-INH or androgens

Acquired Angioedema related to Angiotensin-converting Enzyme Inhibitor

Treatment is with antihistamines, epinephrine, and glucocorticoids. Care should be taken to stop the offending ACE inhibitor, and the patient should be not rechallenged with any of the ACE inhibitors in the future.

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Acquired C1 Inhibitor Deficiency Angioedema

Majority of cases are asymptomatic and respond to immunochemotherapy. Treatment of acute attacks with icatibant and plasma-derived C1 inhibitor concentrate, and prophylaxis is with rituximab with or without chemotherapy and splenectomy.

CONCLUSION

Angioedema is a complex condition marked by episodic swelling of deeper skin and mucosal tissues, requiring precise diagnosis and tailored treatment. With varied pathophysiology involving mast cell degranulation, bradykinin production, or autoimmune processes, hereditary, drug-induced, and idiopathic types each need distinct management. Early recognition and prompt treatment are critical, especially for cases affecting the airway.

Advances in understanding molecular pathways have led to targeted therapies, enhancing patient outcomes. Continued research is necessary to clarify uncommon mechanisms and develop improved treatments. Clinicians should remain vigilant in assessing recurrent swelling, particularly when conventional treatments fail.

Overall, effective management of angioedema relies on patient education, trigger evaluation, and appropriate diagnostics, integrating research insights with clinical expertise for personalized care.

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Comprehensive Guide to URTICARIA

Salient Features

- This book is a concise guide to understanding and treating urticaria.
- All different types of urticaria are very well explained and covered in separate chapters.
- Clinical images, flowcharts and tables have been added in each chapter to make this book clinically oriented and user-friendly.
- Separate chapters on prick test, oral desensitization and urticaria in special situations like pregnancy, children, elderly, etc., have been added.
- This book is very useful for dermatologists, postgraduates, internists and family practitioners.



Sanjeev Gupta MD DNB MNAMS is working as Professor and Head, Department of Dermatology, MM Institute of Medical Sciences and Research, MMDU, Mullana, Ambala, Haryana, India. He is a member of most national and international academic societies. He has around 170 publications, 4 books and 17 book chapters to his credit. He has invented many dermatological techniques and instruments to simplify the procedures and reduce the cost. Twenty-six of such inventions have been published in one of the most prestigious journals of dermatology—Journal of the American Academy of Dermatology USA. He was awarded with "Making a Difference" award by the American Academy of Dermatology, USA. He is the first Indian to be honoured with this prestigious award. He has served his professional association [Indian Association of Dermatologists, Venereologists, and Leprologists (IADVL)] as Executive Committee Member at the state level as Treasurer, Secretary, Vice President, and President and also at the National level as Joint Secretary and Vice President IADVL. He was honoured with the LN Sinha Award by IADVL in 2013 and the ACSI Innovation Award 2021 by the Association of Cutaneous Surgeons of India (ACSI). He is the recipient of Dr BM Ambady Oration awarded at DERMACON 2025 Jaipur.



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