

URTICARIA



— Prof. Ahmed Bouhamidi —

Educational objectives

By the end of this course, the student should be able to:

**1**

Define urticaria



Identify the clinical triad of urticarial papules and distinguish urticaria from angioedema.

2

Differentiate acute from chronic



Set the 6-week threshold and distinguish between spontaneous urticaria and inducible urticaria.

3

Investigate an etiology



Conduct a rational, stepwise diagnostic approach without routine prescription of additional tests.

4

Recognize emergencies



Identify anaphylaxis and bradykinin-mediated angioedema, two potentially life-threatening conditions.

**5**

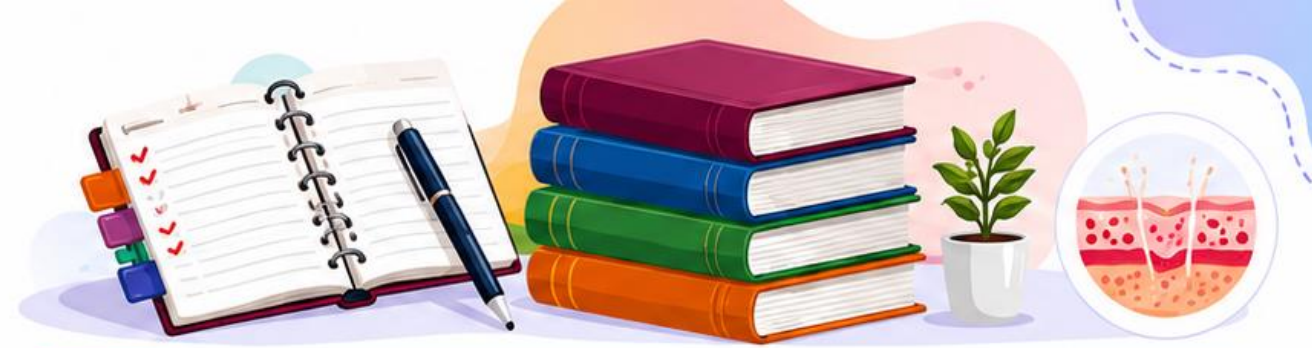
Implement treatment



Apply the 2026 international therapeutic algorithm, from step 1 to step 4.



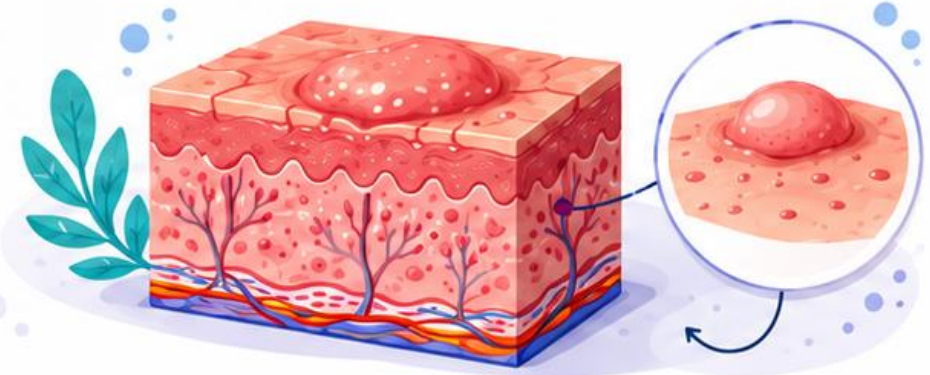
Course plan



01	Definition		06	Inducible urticarias	
02	Pathophysiology		07	Angioedema	
03	Classification		08	Diagnosis	
04	Acute urticaria		09	Treatment	
05	Chronic urticaria		10	Clinical cases	
11	Key messages				

The urticarial wheal: a triad

According to the international definition EAACI/GA²LEN (Zuberbier et al., Allergy 2026), urticaria is characterized by **wheals**, **angioedema**, or both.



1

Central wheal



Superficial dermal swelling, Well circumscribed, variable in size and shape, almost always surrounded by a reflex erythematous halo.

2

Pruritus



Sensation of itching, sometimes intense, almost constant — this symptom is the most bothersome for the patient.

3

Evanescent



Spontaneous resolution within 30 minutes to 24 hours, with return to normal skin appearance, without residual lesions or scarring.



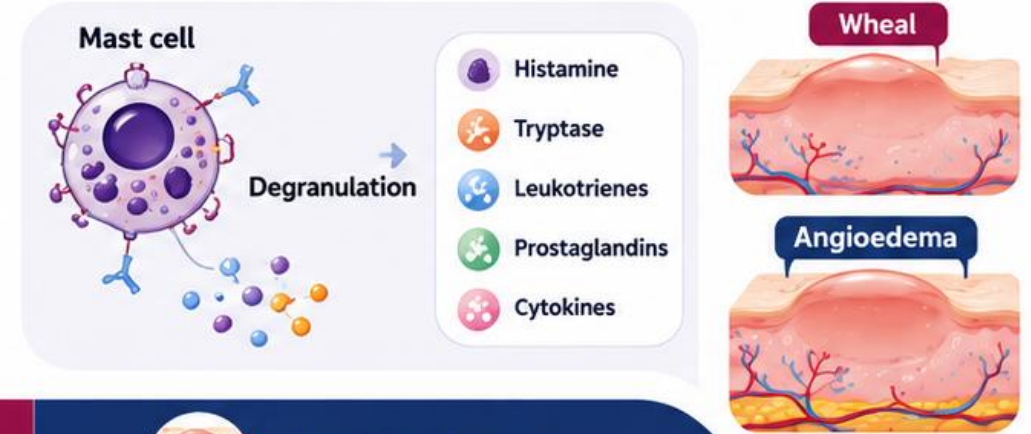
PEDAGOGICAL POINT

An isolated urticarial wheal lasting more than 24 hours, leaving residual pigmentation or not pruritic, should suggest **urticarial vasculitis**, not ordinary urticaria.



Wheal vs angioedema:

two expressions, a mast cell–mediated mechanism



Feature	Wheal	Angioedema
Site	Superficial skin (epidermis and upper dermis)	Deep dermis / hypodermis / mucous membranes
Sensation	Itching, sometimes burning	Painful tension, burning — rarely itching
Color	Erythematous	Normal skin color or erythematous
Resolution	30 minutes – 24 hours	Slower: up to 72 hours
Topography type	Diffuse, all over the integument	Face, lips, tongue, larynx, extremities, genital organs



KEY TAKEAWAY

The same patient may present isolated wheals, isolated angioedema, or both — this combination already suggests a different diagnosis (see slide 26).



1 DEFINITION

A common disorder, an underestimated burden



20%

cumulative lifetime prevalence
of **acute** urticaria
during the lifetime



0,5–1%

prevalence of spontaneous
chronic urticaria
at a given time



> 50%

of patients seen in tertiary
centers also report
angioedema



~50%

of patients remain symptomatic
despite standard-dose
anti-H1 therapy

IMPACT



Quality of life comparable to rheumatoid arthritis or insulin-treated diabetes



Sleep, sexual life, schooling and work performance **impaired**



Frequent psychiatric comorbidities: **anxiety, depression**



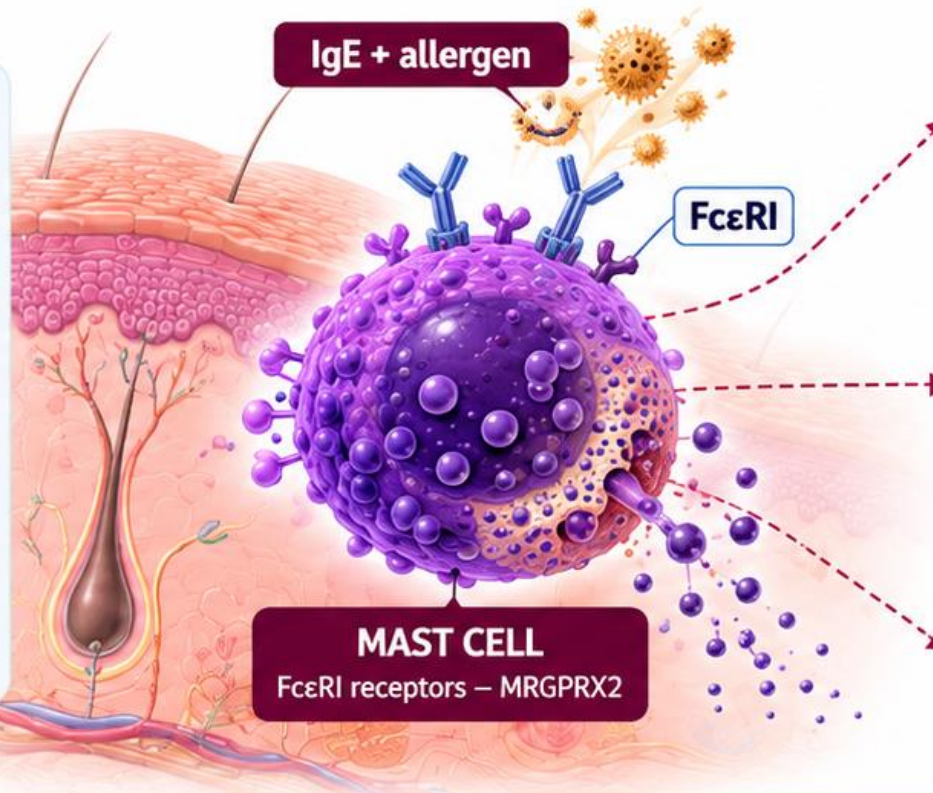
Increased mortality, largely related to associated **suicidal risk**



The cutaneous mast cell, the orchestra conductor

A sentinel cell at the crossroads of innate and adaptive immunity

- ✓ **Central role** in urticaria
- ✓ **Integration** of dependent and independent immune signals
- ✓ **Interface** between innate and adaptive immunity



Histamine

- Pruritus, vasodilatation, increased vascular permeability
- Erythema and oedema

PAF (platelet-activating factor)

- Amplifies oedema
- Maintains inflammation

Cytokines / chemokines

- Recruitment of inflammatory cells
- Maintenance of the immune response

T lymphocyte



Eosinophil



Basophil



Neutrophil

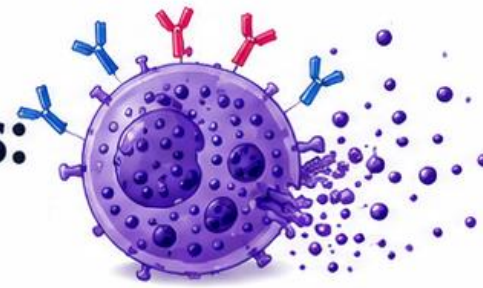


IN SYNERGY

Other cells amplify the response: T lymphocytes (Th1/Th2/Th17), B lymphocytes (auto-antibodies), eosinophils, basophils, macrophages and cutaneous sensory nerves.



In more than 50% of tertiary care centers: an autoimmune mechanism



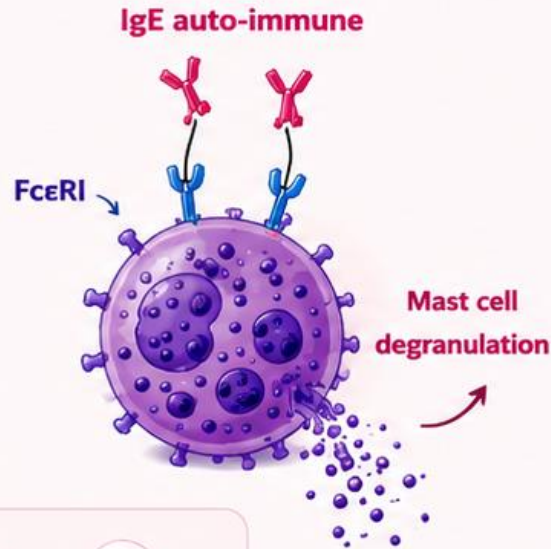
Predominant in
chronic spontaneous
urticaria (CSU)

TYPE I

“auto-allergy”

IgE auto-immune

IgE directed against **self-antigens** (e.g. thyroperoxidase, IL-24) bind to the **FcεRI** receptor and activate the **mast cell** directly, as in classic allergic reactions.



Examples of auto-antigens



Thyroperoxidase



IL-24



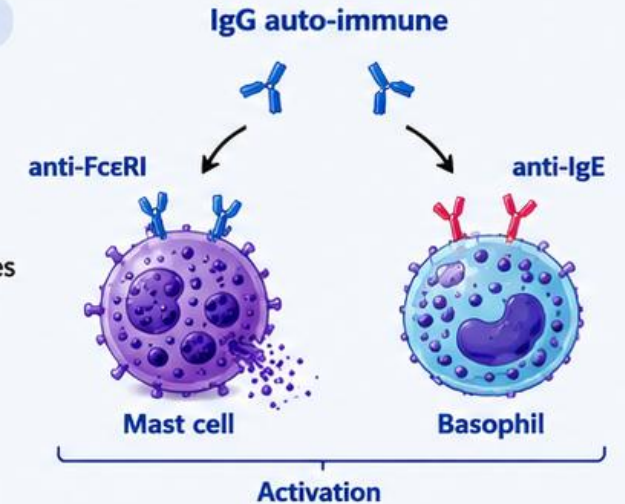
Other auto-antigens

TYPE IIb

classic auto-immunity

IgG anti-FcεRI / anti-IgE

IgG (often IgG1/IgG3) directed against the FcεRI receptor or against IgE themselves activate the mast cell and the basophil — a biological signature: **high CRP**, **low eosinophils** and **basophils**.



Typical biological signature



High CRP



Low
eosinophils



Low
basophils



CLINICAL UTILITY

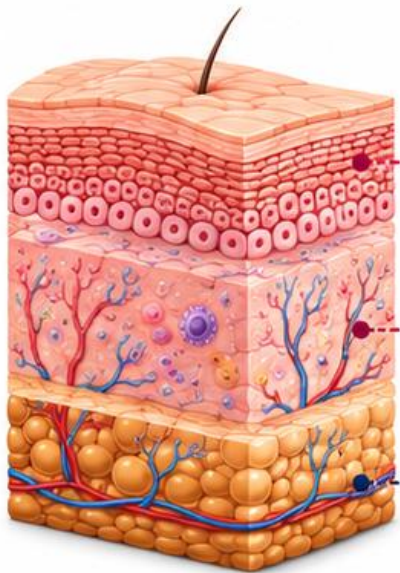
A low total IgE level associated with high anti-TPO IgG levels suggests a type IIb autoimmune CSU, often less responsive to standard-dose omalizumab.



The same process, two different depths



Same immunologic cascade,
but retention according to the skin level involved.



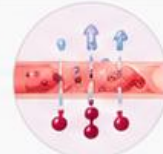
LEVEL INVOLVED	CLINICAL EXPRESSION
EPIDERMIS	 Papillary dermis → Urticarial wheal
RETICULAR DERMIS	 Dilation and increased permeability of post-capillary venules and lymphatic vessels
HYPODERMIS	 Deep dermis / hypodermis → Angioedema



Mast cell
activation



Release of mediators
(histamine, PAF, cytokines...)



Vasodilation and
↑ vascular permeability



Same mechanism,
different location
= different clinical
expression



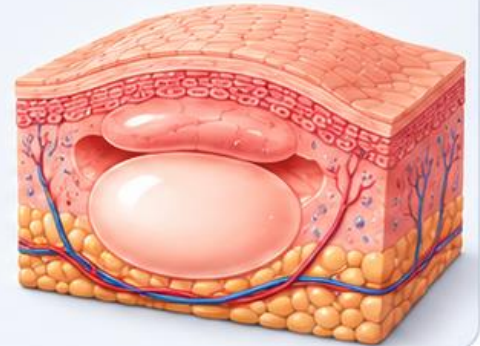
WHEEL

Superficial dermal oedema,
dilatation and increased
permeability of post-capillary venules,
lymphatic vessels, and infiltration
of inflammatory cells
(T cells, eosinophils, basophils).



ANGIOEDEMA

Same modifications, located
in the deep dermis and hypodermis —
which explains the pain,
the longer duration of symptoms,
and the slower resolution.



KEY POINT

The absence of partial vessel necrosis:
the reverse of urticarial vasculitis
(see slide 28).

Two axes, two questions to ask



These two axes can be combined freely — the same patient can present several subtypes.

AXIS 1

« Since when? »



Acute urticaria

≤ 6 weeks

Brief episode, often linked to a triggering factor.



Chronic urticaria

> 6 weeks

daily or recurrent

Persistence or recurrences almost daily for more than 6 weeks.

AXIS 2

« Is there a specific triggering factor? »



Spontaneous

No identifiable triggering factor.



Inducible

Defined and specific triggering factor:
cold, pressure, exertion...

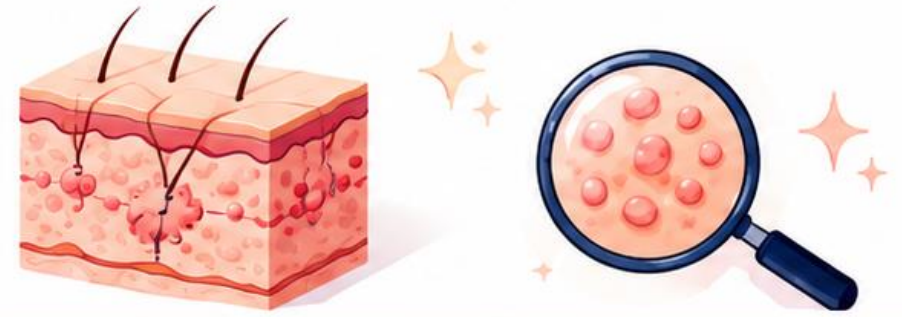


Combination of the two axes: examples of urticaria subtypes

Duration	Trigger	Example of subtype
 Acute (≤ 6 weeks)	 Spontaneous	 Acute spontaneous urticaria (most common)
 Chronic (> 6 weeks)	 Spontaneous	 Chronic spontaneous urticaria (CSU)
 Chronic (> 6 weeks)	 Inducible	 Chronic inducible urticaria (CIndU)



Subtypes of chronic urticaria



Chronic Spontaneous Urticaria (CSU)

- **CSU of known cause**

Autoimmune type I or IIb, identifiable cause.

- **CSU of unknown cause (CSUuc)**

Mechanism not yet elucidated.



May evolve through episodes interspersed with complete **remissions** lasting several months to several years.



Chronic Inducible Urticaria (CIndU)



Symptomatic dermographism



Cold urticaria



Delayed pressure urticaria



Solar urticaria



Heat urticaria



Vibratory angio-oedema



Cholinergic urticaria



Contact urticaria



Aquagenic urticaria

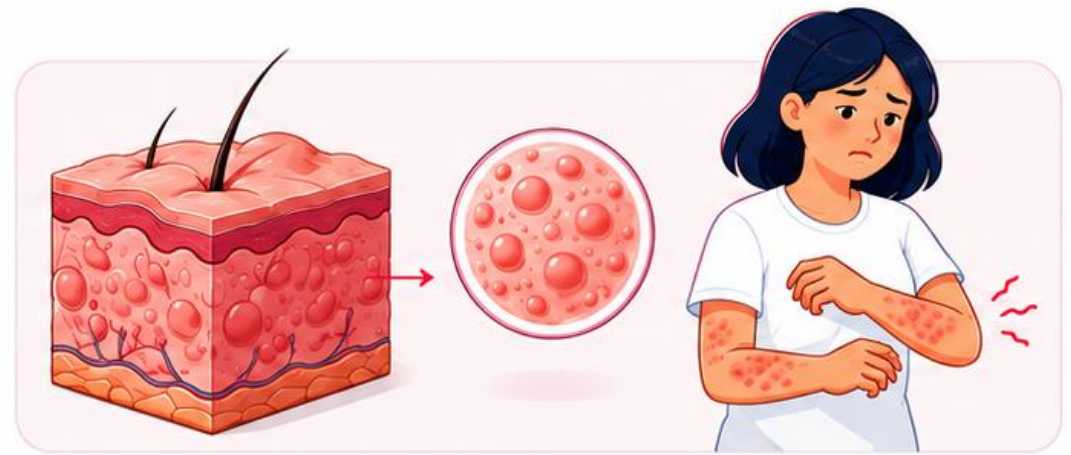


Usual grouping: dermographism, cold, pressure, solar and heat = “**physical urticarias**”.

Acute urticaria: benign, but not always innocuous

Wheals and/or angioedema evolving for ≤ 6 weeks.

This is the most frequent reason for dermatology consultation related to urticaria.



1		Infections	Most frequent cause, particularly viral upper respiratory tract infections (common cold, COVID-19)	
2		Medications	Beta-lactams (IgE-mediated), NSAIDs (non-IgE-mediated)	
3		Foods	In sensitized individuals — suspect an IgE-mediated allergy if suggestive history/context	
4		Insect stings / contact	Latex, venoms, direct contact with an allergen	
5		Idiopathic	No cause found in a significant proportion of cases	

Management: **simple** in the vast majority of cases



STRONG RECOMMENDATION

No systematic work-up in acute spontaneous urticaria

(EAACI/GA²LEN 2026 — consensus, recommendation ↓↓↓)



Targeted history is sufficient to look for a recent triggering factor (infection, medication, food).



Exception: suspected IgE-mediated food allergy or drug hypersensitivity (especially NSAIDs) → targeted allergy work-up.



Infection



Medication



Food



NSAIDs



Others



The vast majority of acute urticaria cases resolve spontaneously with simple treatment and good information for the patient.



SYMPTOMATIC TREATMENT



Second-generation H1-antihistamines at standard dose, daily



Possible dose up-dosing up to 4x the standard dose if needed (except in pediatric patients)



Short course of oral corticosteroids (3–5 days) if severe flare or angioedema



Avoid identified triggering factor



Patient education: advise to seek medical care if signs of anaphylaxis



Never underestimate anaphylaxis



Acute urticaria can be the first cutaneous sign of anaphylaxis.

 **Systematically** look for:

RESPIRATORY



- Dyspnea
- Wheezing
- Stridor
- Laryngeal edema

CARDIOVASCULAR



- Hypotension
- Tachycardia
- Malaise
- Syncope

GASTROINTESTINAL



- Abdominal pain
- Nausea / Vomiting
- Diarrhea

CONTEXT

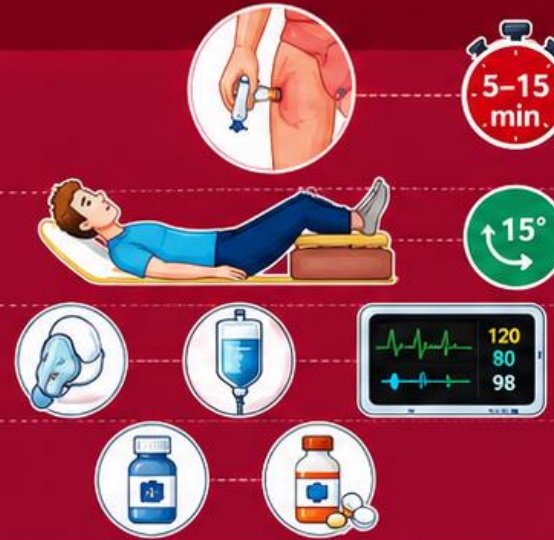


Brief delay after exposure to an allergen (food, sting, medication)



IMMEDIATE MANAGEMENT

- 1** IM adrenaline IM in the anterolateral thigh
0.01 mg/kg (max 0.5 mg in adults) — treatment of 1st choice, repeat if needed in 5–15 min
- 2** Prolonged supine position with legs elevated (or sitting position if dyspneic), call EMS
- 3** Oxygen, IV fluid administration, continuous monitoring
- 4** H1-antihistamines and corticosteroids = adjuvant treatments only, never as 1st choice



KEY POINTS



Act quickly



Adrenaline = 1st choice



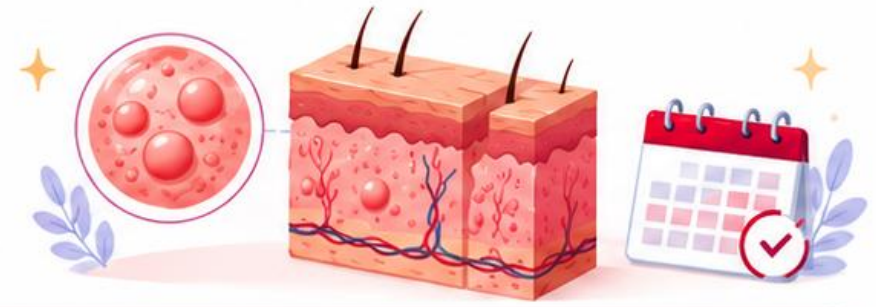
Monitor



Document and inform the patient



Chronic spontaneous urticaria (CSU)



DEFINITION spontaneous appearance of wheals, angioedema, or both, lasting **more than 6 weeks**, without an identifiable physical trigger — occurring almost daily or recurrently.



1 Unpredictable course



Remissions may occur after months or years — some patients achieve complete remission.

2 Cause often unknown



CSU of unknown cause (CSUuc) occurs in a large proportion of patients despite a thorough work-up.

3 Frequent association



Inducible urticaria may coexist with CSU — the history should always investigate for all types.



DO NOT CONFUSE: “chronic urticaria” does not mean “chronic urticaria continues” — some patients have episodes of wheals separated by symptom-free days.



Measure to better treat: UAS7 and UCT

 **UAS7 — Urticaria Activity Score over 7 days (disease activity)**

Score	Wheals / 24 h		Itch	
0		None		None
1		< 20 wheals		Mild, not bothersome
2		20–50 wheals		Moderate, somewhat affecting sleep/activity
3		> 50 wheals or large confluent areas		Severe, markedly affecting sleep or activity



Daily score 0–6, summed over 7 days → UAS7 from 0 to 42.



UCT — Urticaria Control Test

4 questions, recall over 4 weeks
(UCT7 version over 7 days available).
Score 0–16.



≥ 12 / 16
well-controlled disease



IN PRACTICE

Daily self-assessment (paper or an app, e.g., CRUSE) over 4 weeks to monitor fluctuations and guide therapeutic decisions at each consultation.



When **angioedema** predominates: AAS and AECT



AAS — Angioedema Activity Score



5 dimensions scored from 0–3:



Time



Physical
impairment



Impact on
daily activities



Aesthetic
impact



Overall
severity

➤ Daily AAS	0–15
➤ AAS7 (weekly)	0–105
➤ AAS28 (4 weeks)	0–420



Daily score 0–6, accumulated over 7 days
→ AAS7: 0–42.



AECT — Angioedema Control Test



4 questions, similar to the UCT —
recall period of 4 weeks or 3 months.



Well-controlled disease threshold
≥ 10 / 16



Indication

For patients with isolated angioedema or
angioedema associated with wheals;
to be used in addition to the UCT
when both coexist.



IN PRACTICE

AAS measures angioedema activity and its impact.
AECT assesses disease control.



Aggravating factors — without being the cause



! NSAIDs

Aggravate up to **25% of CSU** through a non-IgE-mediated mechanism; **paracetamol** and **COX-2** inhibitors are better tolerated.



! Stress

Up to **one third** of patients identify it as an aggravating factor; stress management approaches can help.



! Infections

Infections of the upper airways may precede or worsen a flare of CSU.



! Foods (pseudo-allergens)

Rarely a true cause; an elimination diet for **2-3 weeks** may be proposed without delaying treatment.



Angioedema: mast cell-mediated or bradykinin-mediated?

A key question: these two mechanisms **do not respond to the same treatment**.



 Features	 Mast cell-mediated (histaminergic)	 Bradykinin-mediated (HAE / AAE)
 Association with wheals	Frequent 	Never 
 Itching	Possible 	Absent (no itching or pain) 
 Onset of symptoms	Minutes to hours 	Hours (progressive) 
 Duration	< 24–72 h 	2 to 5 days 
 Response to H1-antihistamines / epinephrine	Good 	Absent 
 Response to corticosteroids	Partial 	Absent 
 Specific treatment	H1-antihistamines, omalizumab 	C1-INH concentrate, icatibant 



CLINICAL TAKEAWAY: Isolated and recurrent angioedema, resistant to H1-antihistamines and epinephrine, → **always** raise the possibility of a **bradykinin-mediated cause**.



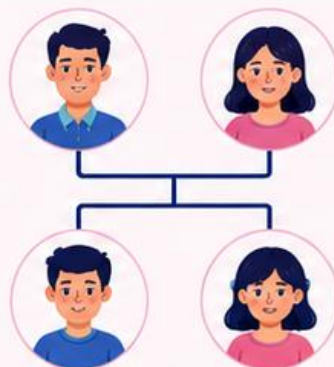
Bradykinin-mediated angioedema: don't miss HAE



Evocative background



Family history of recurrent angioedema, onset in **childhood** or **adolescence**.



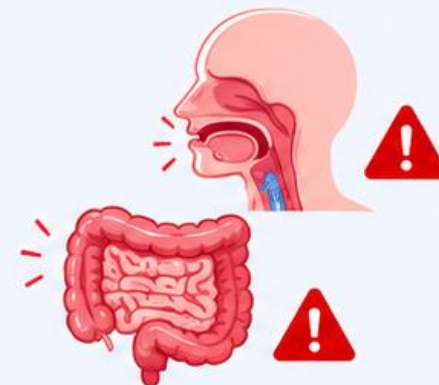
At-risk locations



Face, tongue, **larynx** (risk of asphyxia)



Digestive tract (pseudo-surgical abdomen).



Triggering factors

- Minor trauma
- Stress
- Surgery
- **Estrogens** (pregnancy, contraception)
- **ACE inhibitors** (acquired C1-INH deficiency)



Biologic confirmation

- **C4** and functional **C1-inhibitor** level (antigenic and functional)
- Search for **anti-C1-INH** antibodies if acquired.



HAE types I/II (C1-INH deficiency or dysfunction) and HAE with normal C1-INH (mutations in FXII, plasminogen, angiotensin-1, etc.).



Bradykinin angioedema in crisis: **what to do?**



1

Assess the airway



Laryngeal involvement = vital emergency → transfer to ICU, consider early intubation.



2

Specific treatment



IV C1-inhibitor concentrate, icatibant (bradykinin B2 receptor antagonist), or ecallantide depending on availability.



3

Ineffective



Adrenaline, anti-H1 and corticosteroids: no proven efficacy on this mechanism — do not delay specific treatment when used alone.



4

Prevention



Short-term tranexamic acid prophylaxis (e.g., before dental procedure); long-term management at a reference center (CREAK).



Un trigger défini, un test de provocation dédié



 Sous-type	 Déclencheur	 Test diagnostique
 Dermographisme symptomatique	 Friction / grattage cutané	 Dermographie (FricTest)
 Urticaire au froid	 Exposition au froid	 TempTest (élément Peltier), test au glaçon
 Urticaire retardée à la pression	 Pression soutenue	 Test au poids suspendu, dermatographe
 Urticaire solaire	 UV / lumière visible	 Photo-provocation à longueurs d'onde croissantes
 Urticaire à la chaleur	 Contact chaud	 TempTest
 Angio-œdème vibratoire	 Vibration	 Test au vortex-mixer
 Urticaire cholinergique	 Effort, chaleur, stress	 Épreuve d'effort graduée (ergométrie)
 Urticaire de contact / aquagénique	 Contact direct / eau	 Test cutané de provocation

Spotlight: **dermographism** and cold urticaria



Symptomatic dermographism



- The most common form of inducible urticaria
- Linear wheals appear within minutes after scratching or rubbing
- Symptomatic dermographism (pathologic): lifetime prevalence $\approx$ 5.9% (point prevalence $\approx$ 3.2%); simple form, non-pruritic, non-pathologic $\approx$ 5.4% lifetime prevalence — two distinct entities, should not be confused
- Treatment: **anti-H1**, usually at higher doses



Key message : recognizing these two entities helps avoid misdiagnosis and unnecessary treatments.



Cold urticaria



- Wheals within minutes after rewarming of a previously cold-exposed area
- **Major risk:** immersion in cold water $\rightarrow$ risk of generalized anaphylactoid shock due to systemic vasoplegia, syncope
- Rule out secondary causes (cryoglobulinemia, infection) before an atypical or delayed form
- **Essential education :** avoid cold water immersion



Safety alert : inform the patient about the risks of cold water exposure and warning signs.

Spotlight: cholinergic urticaria and delayed pressure urticaria



Cholinergic urticaria



Small pinpoint papules, **very itchy**, triggered by an increase in core body temperature (exercise, stress, hot bath)



More commonly affects **adolescents** and **young adults**



Activity score: Cholinergic Urticaria Activity Score (**CholUAS**)



Differential diagnosis: **exercise-induced anaphylaxis**



Delayed pressure urticaria



Painful, deep wheals, appearing **4 to 8 hours** after sustained pressure (backpack strap, belt, chair, standing station)



Often associated with **severe, persistent chronic spontaneous urticaria (CSU)**



May respond to **antihistamines** alone: often partial response; higher doses or multiple agents usually required



Key message: recognizing these subtypes helps guide the diagnosis, tailor management, and improve patients' quality of life.





The diagnostic approach in 3 steps

1

History taking



- An essential and first step: onset, frequency, duration of lesions, triggers, associated symptoms, ongoing treatments, personal and family history, recent stress (sleep, work, etc.).

2

Physical examination



- If lesions are fugacious, they may have disappeared by the consultation: request **recent photographs taken by the patient** during the flare.

3

Targeted basic workup



- A limited set of complementary tests, guided by the **suspected subtype** — always starting with a systematic screening and cost-effective tests (see slide 27).



7 objectives of the workup in CSU (mnemonic “the 7 C’s”)



Confirm



Cause



Contributing factors



Comorbidities



Consequences



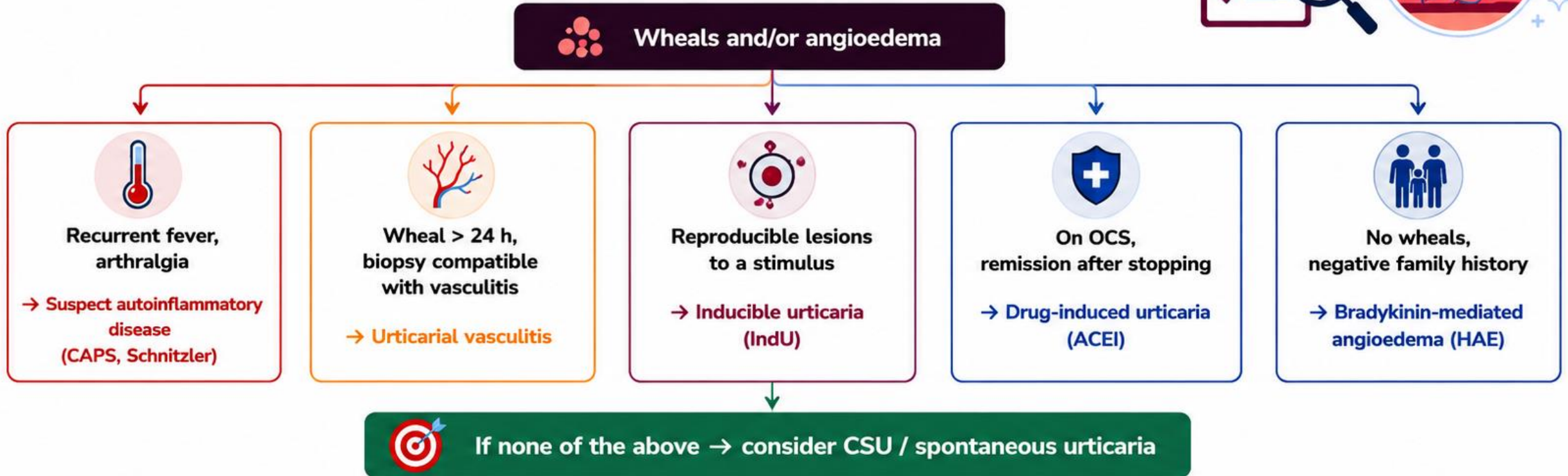
Components (biomarkers)



Course (prognosis)



Diagnostic algorithm (EAACI/GA²LEN, Allergy 2026)



Reading the algorithm is based on the clinical history. Each “red flag” points to a specific differential diagnosis and its dedicated tests — in their absence, CSU/CIU becomes the most likely diagnosis.

Adapted from: Zuberbier T. et al., “The International Guideline for the Definition, Classification, Diagnosis and Management of Urticaria”, Allergy 2026 (Figure 1, simplified algorithm for educational purposes).





Basic work-up vs extended work-up

 Subtype	 Basic work-up (systematic)	 Extended work-up (guided by clinical suspicion)
 Acute spontaneous urticaria	None	Targeted allergy testing if food- or drug-related allergy is suspected
 Chronic spontaneous urticaria (CSU)	CBC, ESR and/or CRP (+ total IgE, anti-TPO IgG in specialist settings)	Infectious serologies, functional autoantibodies (ASST/APST), thyroid assessment, tryptase, biopsy if atypical, C4/C1-INH if isolated angioedema
 Inducible urticaria	Provocation testing + threshold assessment	CBC/ESR-CRP for cold urticaria; investigation for secondary causes if atypical



Key message: A broad, non-targeted work-up does not improve patient management and may lead to anxiety-provoking false-positive results.



Differential diagnoses **not to miss**



**Urticarial
vasculitis**



**Cutaneous
mastocytosis**



**Mast cell
activation syndrome**



**Bradykinin-mediated
angioedema
(HAE/AAE)**



**Auto-inflammatory
syndromes
(CAPS, Schnitzler)**



**Exercise-induced
anaphylaxis**



**Bullous pemphigoid
(pre-bullous phase)**



**Adult-onset
Still's disease**



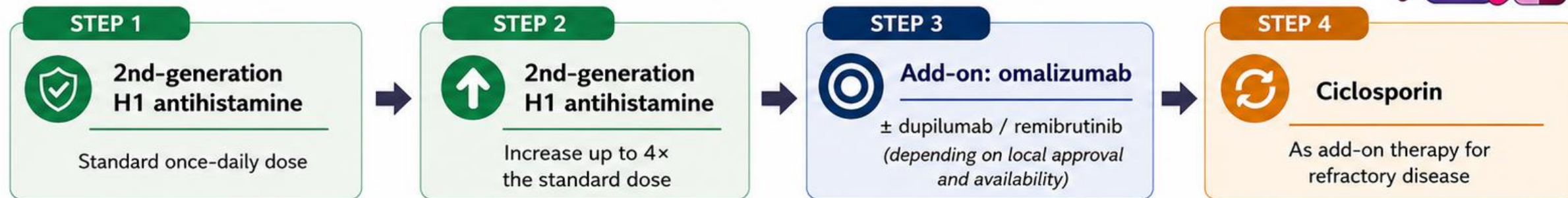
**MwS /
Gleich syndrome**



These conditions may present with urticaria, wheals and/or angioedema, but involve distinct physiopathological mechanisms and require specific treatments.



The 2026 International Treatment Algorithm



i If treatment failure or intolerance occurs at a step after 2–4 weeks (earlier in severe disease), move to the next step as add-on therapy — **never as a replacement for H1 antihistamines**.

Step	Strength of recommendation	Level of evidence
1–2 2nd-generation H1 antihistamine (up to 4× dose)	↑↑ Strong recommendation in favour	✓ High-quality evidence
3 Omalizumab (add-on)	↑↑ Strong recommendation in favour	✓ High-quality evidence
3 Dupilumab / remibrutinib (add-on)	↑ Suggested	★ Emerging evidence (2025–2026)
4 Ciclosporin (add-on)	↑ Suggested	⚠ Off-label; reserved for refractory disease
💊 Long-term corticosteroids	↓↓ Strongly discouraged	⊕ Short rescue course only (3–5 days)

Adapted from Zuberbier et al., Allergy 2026 (updated according to the EAACI/GA²LEN/EuroGuiDerm/APAAACI consensus conference of December 6, 2024).



Steps 1–2: 2nd-generation H1 antihistamines



Preferred molecules

Bilastine • Cetirizine • Desloratadine • Ebastine
Fexofenadine • Levocetirizine • Loratadine • Rupatadine



Why 2nd generation? 1 dose/day (long half-life), excellent safety profile, minimal central nervous system penetration (non-sedating).



Points to avoid

Combining several different H1 antihistamines, and switching to a 1st-generation H1 antihistamine (sedating) — not recommended by guidelines.



The principle of up dosing (×4)

If no adequate control after 2–4 weeks at the standard dose (or earlier, if severe):



Increase the dose of the same H1 antihistamine, up to 4 times the standard dose.

Off-label practice in some countries depending on local approval — assess benefit/risk.



Reassess using UAS7/UCT at each step before moving to the next step.



Palier 3 : les biothérapies add-on



 Molécule	 Cible	 Posologie usuelle	 Statut 2026
 Omalizumab	Anti-IgE 	 300 mg SC / 4 sem. ; « updosing » possible (raccourcir l'intervalle et/ou augmenter la dose)	 Recommandation forte (↑↑), traitement de référence
 Dupilumab	Anti-IL-4Rα (bloque IL-4/IL-13) 	 Selon schéma de l'AMM locale	 Suggesté (↑), preuves émergentes
 Remibrutinib	Inhibiteur oral de BTK 	 25 mg per os, 2 prises/jour	 Suggesté (↑) ; approuvé (FDA, 30 sept. 2025)



Prédicteurs de réponse à l'omalizumab

- IgE totales élevées, basophiles > 20/μL, absence d'hypothyroïdie associée : meilleure réponse à dose standard.



Comorbidités bonus

- L'omalizumab et le dupilumab agissent aussi sur l'asthme associé ; le dupilumab, sur une dermatite atopique concomitante.



Step 4: ciclosporin — and the (limited) role of corticosteroids



Ciclosporin A



Indication

Refractory CSU in steps 1–3
(failure or inaccessibility of omalizumab)



Dosage

Low doses (1–3 mg/kg/day),
in association with H1 antihistamines



Monitoring

Blood pressure, renal function,
magnesemia, kalemia — off-label treatment
in urticaria



Alternative in case of failure

Switch to dupilumab/remibrutinib
or switch between biologic therapies



Systemic corticosteroids



Strong recommendation AGAINST prolonged use
or delayed-onset forms



Only acceptable use short rescue course,
3 to 5 days, in severe exacerbation



Why avoid long-term use? undesirable cumulative effects
(metabolic, bone, infectious) without proven benefit
in long-term control of CSU



Special populations



Children

The same therapeutic algorithm as in adults, adapted to age and weight.

Omalizumab is the only biologic approved from **12 years** of age in CSU refractory to anti-H1.



Pregnancy

2nd-generation H1 antihistamines are the preferred and usually safe option in pregnancy.

Avoid **ciclosporin** and **corticosteroids** long term.



Breastfeeding

The same principles of prudence apply; individual decisions should be made on a case-by-case basis, with patient discussion.



Key message:

The 2026 guideline recommends using the same algorithm in these populations, with proven safety and individualised adaptation — just as in adult patients.



Key messages



-  1 Urticaria associated with wheals (< 24 h) and/or angioedema (up to 72 h) — **duration > 6 weeks** defines chronicity.
-  2 Acute spontaneous urticaria does not require any systematic work-up: **the history is key.**
-  3 All urticaria must be assessed for signs of anaphylaxis — **IM adrenaline** remains the **first-line treatment.**
-  4 Isolated angioedema without wheals and resistant to H1 antihistamines should suggest a **bradykinin-mediated mechanism (HAE).**
-  5 The treatment of CSU follows a **stepwise algorithm**: standard-dose H1 antihistamine → up-dosing up to 4× → add-on (omalizumab as first-line add-on) → ciclosporin.
-  6 Long-term systemic corticosteroids **have no role** in chronic urticaria.



Bibliography



- 1 | Zuberbier T, Abdul Hamed Ansari Z, Abdul Latiff AH, et al. *The International Guideline for the Definition, Classification, Diagnosis and Management of Urticaria*. **Allergy**. 2026;0:1–51. doi:10.1111/all.70210
- 2 | Zuberbier T, Abdul Latiff AH, Abuzakouk M, et al. *The international EAACI/GA²LEN/EuroGuiDerm/APAAACI guideline for the definition, classification, diagnosis, and management of urticaria*. **Allergy**. 2022;77(3):734–766. doi:10.1111/all.15090
- 3 | Maurer M, Casale TB, Saini SS, et al. *Dupilumab in patients with chronic spontaneous urticaria (LIBERTY-CSU CUPID): two randomized, double-blind, placebo-controlled, phase 3 trials*. **J Allergy Clin Immunol**. 2024;154(1):184–194. doi:10.1016/j.jaci.2024.01.028
- 4 | Kaplan A, Ledford D, Ashby M, et al. *Omalizumab in patients with symptomatic chronic idiopathic/spontaneous urticaria despite standard combination therapy*. **J Allergy Clin Immunol**. 2013;132(1):101–109. doi:10.1016/j.jaci.2013.05.013
- 5 | Magerl M, Altrichter S, Borzova E, et al. *The definition, diagnostic testing, and management of chronic inducible urticarias — EAACI/GA²LEN/EDF/UNEV consensus recommendations, 2016 update*. **Allergy**. 2016;71(6):780–802.
- 6 | Bernstein JA, Cremonesi P, Hoffmann TK, Hollingsworth J. *Angioedema in the emergency department: a practical guide to differential diagnosis and management*. **Int J Emerg Med**. 2017;10(1):15. doi:10.1186/s12245-017-0141-z
- 7 | Cicardi M, Aberer W, Banerji A, et al. *Classification, diagnosis, and approach to treatment for angioedema — HAWK consensus report*. **Allergy**. 2014;69(5):602–616. doi:10.1111/all.12380
- 8 | Kulthanan K, et al. *The prevalence of symptomatic dermographism: results of the international UCARE PREVALENCE-D study*. **Allergy**. 2025/2026. doi:10.1111/all.70047

