CASE REPORT

Angioedema and lip filler: 4 years-same patient case report and literature analysis

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ABSTRACT

Background: Hyaluronic acid (HA) fillers are widely used in aesthetic medicine for minimally invasive facial enhancement due to their favorable safety profile. However, rare but potentially significant adverse reactions such as angioedema can occur, with clinical and therapeutic implications.
Objective: To describe a case of repeat angioedema following repeated HA lip filler sessions in a patient with nickel hypersensitivity, and to review current evidence regarding pathophysiology, risk factors, and management strategies.
Case Report: A 53-year-old woman with a history of nickel allergy presented recurrent episodes of localized angioedema after several lip filler procedures performed by different clinicians, each involving various HA products and injection techniques. Despite premedication with corticosteroids in later sessions, angioedema recurred variably, with laboratory testing excluding hereditary forms. The potential contribution of nickel exposure from the needles, procedural factors, and the underlying allergy was considered.
Discussion: Angioedema is a rare but clinically relevant complication of HA fillers, particularly in patients with allergic predisposition. Comprehensive pre-procedural screening, patient education, and prompt management protocols are essential. Current literature supports the overall safety of HA fillers, though practitioners should remain vigilant for immune-mediated responses, especially when performing procedures in at-risk individuals.
Conclusion: This report presents, to our knowledge, the only documented case of recurrent angioedema after hyaluronic acid lip filler in a nickel-allergic patient, with extended follow-up across multiple procedures and practitioners. Our findings provide novel insight into this rare association, emphasizing the importance of thorough allergy screening and multidisciplinary management in aesthetic practice.

Key words: hyaluronic acid (HA) fillers, recurrent angioedema, nickel allergy, aesthetic procedure complications

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Background

In aesthetic medicine nonsurgical and preferentially noninvasive techniques are used in order to rejuvenate and resolve disharmonies. Hyaluronic acid (HA) fillers are the most performed procedure after botulinum toxin[1]. They allow the correction of folds, rhytids and other aesthetic or medical conditions, and their effectiveness and versatility explain the growing use. In addition, their safety profile is such as to make them perfect for worldwide use. However, adverse reactions can occur.

The incidence of complications is low, and most adverse events are mild. We can classify filler complications by severity (mild, moderate, or severe) or by time (early or late)[2–4]. Recently an Italian consensus report on filler complications reported a classification based on severity[5].

Among others, angioedema is an immediate/early adverse event of varying severity. Angioedema presents itself as edema, localized and self-limiting, of the skin or mucous membranes, which is due to the presence of fluids in the interstice due to the loss of integrity of the vascular walls. Angioedema can be isolated or accompanied with urticaria or any indication of anaphylactic condition. Fluids in angioedema collect asymmetrically in areas where the vascular system is altered – not always downward.

This adverse effect has been already reported in different studies and overviews[2-6]. Foreign materials, HA fillers can trigger immune responses; sensitized patients may experience reactions upon re-exposure, ranging from mild to severe, potentially lasting several weeks[7]. Nonetheless, angioedema is still a rare event and only few cases have been reported[8].

Pathophysiology and classification of angioedema

Angioedema is a well-defined clinical entity characterized by localized, transient swelling of the skin and mucosa resulting from increased vascular permeability. In aesthetic medicine, understanding the underlying mechanisms is essential for an accurate diagnosis and optimal management of complications such as angioedema following dermal filler injections.

Current literature identifies several distinct pathways.

- *Histaminergic angioedema (mast cell-mediated)*

  The pathophysiology mirrors that of urticaria, although it takes place in the deep dermis and subcutaneous tissue, often involving the mouth and face. Degranulation of mast cells involves various mediators of inflammation: histamine, heparin, leukotriene C4, and prostaglandin D2, that cause vasodilation of the dermal venules and increase their own permeability.

  It is possible that acute angioedema, with or without symptoms of an allergic reaction, is caused by food, drugs, latex, exercise, insect bites, and other uncommon allergens. The classic allergic reaction is mediated or type IgE. The reaction typically occurs within a few minutes up to two hours after exposure to the trigger. Isolated angioedema without urticaria is uncommon in IgE-mediated reactions but can occur.

  Vasodilation is precisely due to its pro-inflammatory action. The concentration of bradykinin can be increased both because of its increased production and because of its reduced degradation. It is driven by mast cell and basophil degranulation, leading to the release of histamine and other mediators (e.g., tryptase, leukotrienes), it responds to antihistamines and corticosteroids.
- *Bradykinin-mediated angioedema*

  Mast cells are not involved; therefore, pruritus and urticaria are typically absent. In such cases, adrenaline, antihistamines, and glucocorticoids are generally ineffective, as the condition is mediated by increased bradykinin levels leading to marked vascular permeability. This is typically not associated with urticaria or pruritus. It is often related to hereditary or acquired C1-inhibitor deficiency or triggered by medications (such as ACE-inhibitors). It does not respond to antihistamines; therapies include bradykinin antagonists and C1-inhibitor replacement.

- *Idiopathic or other mechanisms:*
  Encompasses forms with unknown or complex etiology (e.g., idiopathic, physical, autoimmune, or contact-related). May present without classical allergic or bradykinin features.

Case report

A 53-year-old patient, previously treated by other practitioners and known to me from prior procedures (see Figure 3 for a detailed timeline of the sessions), had a medical history significant for hypercholesterolaemia and nickel allergy.

Three different operators performed the same procedure: HA lip filler. It is important to know that the three different operators had also widely different experience in the aesthetic medicine field, suggesting a various ability in performing the procedure (number of entrance point with the needle, injection technique and tissue handling, etc.).

The patient's clinical history includes an allergy to nickel, as well as reporting several cases of angioedema limited to the area of the lips, even sporadic. These events were studied, and the case of hereditary angioedema was excluded, relating the events to the dietary nickel.

A written informed consent was obtained from the patient prior to each procedure, including specific consent for clinical photography. Due to the retrospective nature of this report and the time elapsed between treatment sessions, additional laboratory testing and product batch identification were not available.

Session 1

The first session in 2018 (operator "A"), using HA 20mg/ml (with BDDE 1μg/mL), developed edema 2–3 hours later of both labial districts that requires betamethasone administration (up to 10 mg over the next 6 hours) and monitoring. The operator attributed the cause of edema to the HA and BDDE (Figure 1).

Session 2

The second session in 2019 (operator "B") involved both upper and lower lip. Infraorbital nerve blocks were performed using 1% lidocaine 1 cc both sides. The procedure was performed using a HA 20mg/mL and 3mg/mL of lidocaine crosslinked with BDDE from a different manufacturer to the first session. Prior to the session the patient took 2 mg of betamethasone, and 2mg after completion. The patient did not report any side effects, thus suggesting to the operator a possible implication of the previous hyaluronic acid.

Session 3

For the third session in 2020 (operator "C"), the same product used in session 2 was used in both upper and lower lip fillers with a total volume product of 0.6 mL. Infraorbital nerve blocks were performed with 1% lidocaine, 1 mL per side. The patient followed the same pharmacological routine: 2 mg of betamethasone before and 2 mg immediately after the session.

Two hours later, an asymmetric edema of the upper lip developed. The patient was immediately

Figure 1. Angioedema of the lips following fillers with hyaluronic acid.

examined, excluding alarm signs and symptoms, and treated with intramuscular betamethasone 4mg/2mL and chlorphenamine 10mg/1mL. She was then observed for a few hours.

During the following hours, the patient was consistently monitored, and instructed on how to recognize early signs of edema of the oral or laryngeal cavity.

After 6 hours the edema definitively stopped spreading and remained localized to the upper lip (Figure 2).

Blood tests at 36 hours showed elevated total IgE. All the results are as follows (Table 1):

Session 4

The fourth session occurred in 2022 (operator "C"), using the same product in sessions 2 and 3 in both upper and lower lip fillers with a total volume product of 0.6 mL. An infraorbital nerve block was performed using 1% lidocaine 1 cc both sides. The patient followed the same pharmacological routine: 2 mg of betamethasone before and 2 mg immediately after.

No edema of the upper lip developed.

The patient had been monitored consistently, and instructed how to recognize early signs of edema of the oral or laryngeal cavity in the following hours.

Following a more detailed interview, the patient reported several cases of angioedema limited to the area of the lips, including sporadic manifestations. The patient revealed having undergone a few hematic examinations prescribed from her clinician, to study the events (Table 2). However, at that time (2012) her clinician excluded the possibility of a bradykinin type of angioedema, considering the cause of the reactions due to dietary nickel. For context, prior testing showed a C1-inhibitor concentration reported as 160 mg/L (laboratory reference 195–345 mg/L), suggesting a reduced level; units and functional activity need clarification[9,10].

Discussion

Angioedema as an adverse event following hyaluronic acid (HA) lip fillers distinctly highlights a convergence of immunological, procedural, and patient-specific factors. This discussion explores the state-of-the-art scientific literature regarding angioedema in aesthetic medicine, focusing on incidence, pathophysiology, risk factors, and implications for clinical management, while linking these findings to the reported case and relevant references.

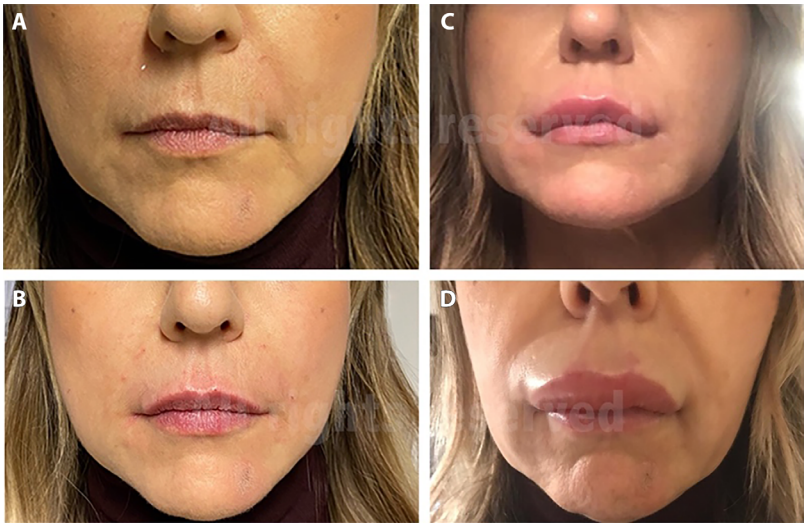

Figure 2. A) pre-procedure B) immediate post-procedure C) After 2 hours D) After 4 hours.

Table 1. Laboratory results at 36 hours (complete panel, units, reference ranges).

Type	Outcome	Reference value
Erythrocytes	4.41 10(12)/L	4,31-5,10
Hemoglobin (Hb)	129g/L	123-153
Hematocrit	0,416	0,380-0,450
Average globular volume	94,4 fL	80,0-96,0
Average globular Hb	29,3 pg	26,0-33,0
Average globular Conc. Hb	311	322-360
RDW	12,30%	11,5-14,5
WBC – Leucociti	9.2 10(9)/L - 47.4%	4,4-11,0
Lymphocytes	4.4 10(9)/L - 44.4%	1,8-7,8
Monocytes	4.1 10(9)/L - 6.7%	1,1-4,8
Eosinophilic	0.1 10(9)/L - 0.8%	0,0-0,5
Basophiles	0.1 10(9)/L - 0.5%	0,0-0,2
PLT	288 10(9)/L	
S-IgE Totals (PRIST)	*147,0 UI/mL*	*Adults < 100. 00*

Table 2. Immunological blood profile from previous test in 2012.

Type	Outcome	Reference Value
VES	17	0-20
TAS	33 UI/mL	0-200
C3c	1.13 g/L	0.9-1.8
C4	0.17 g/L	0.1-0.4
C1 inhibitor	160 mg/L	195-345
PRIST (total IgE)	24.2 UI/mL	
ANA	< 20 UI/mL	

Classification and incidence of filler-related adverse events

Recent consensus papers and international guidelines (supported by the World Allergy Organization (WAO)[11], European Academy of Allergy and Clinical Immunology (EAACI), and recent registry studies in Europe and North America) have introduced a more nuanced classification system for angioedema, addressing both acquired and hereditary forms, as well as mixed and secondary variants. While this expanded framework is highly relevant in allergology and immunology, we find that its clinical utility in the context of aesthetic medicine remains limited. For most aesthetic procedures, it is sufficient for the practitioner to recognize the acute, histaminergic forms most encountered, rather than to differentiate between the full spectrum of hereditary and secondary subtypes. Acknowledging the broader classification underscores the complexity of angioedema, however, given its rare incidence and distinct presentation in aesthetic practice, a detailed discussion may not be warranted in this setting. Routine screening for allergy history and vigilance for acute reactions remains the cornerstones of safe practice.

Along the exponential rise in non-surgical procedures for facial enhancement, HA-based fillers remain central due to their proven safety and reversible nature. Large clinical series and systematic reviews indicate that the majority of adverse reactions after lip fillers are mild and transient: swelling, erythema, ecchymosis, and nodules[12–14]. However, systematic reviews and recent prospective studies confirm that more severe reactions - such as angioedema, hypersensitivity, granuloma formation, and vascular compromise- are rare but clinically significant, often requiring

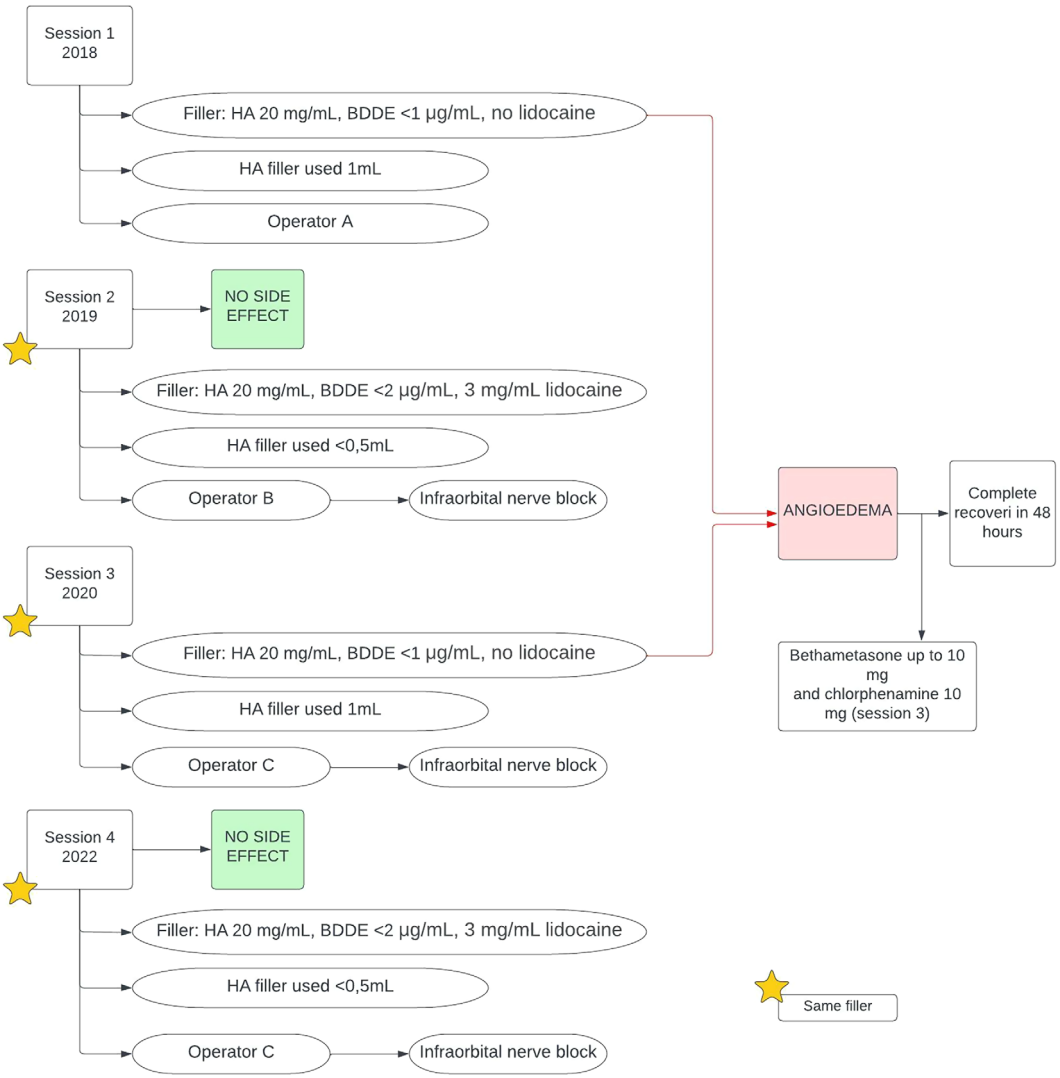

Figure 3. Timeline of sessions (2018–2022), products, doses, techniques, onset time, management, and outcomes.

urgent pharmacologic intervention, including hyaluronidase[15]. Substantial displays of angioedema following HA fillers are exceedingly rare; prospective series report very low rates of delayed hypersensitivity-like reactions (e.g., ~0.21% for a specific product in a 1-year multicenter study), and most reported adverse events are mild and transient[16]. Delayed inflammatory reactions, including angioedema following triggers like viral illness or vaccination, remain rare but are increasingly documented[6].

Pathophysiology of angioedema post-filler

Angioedema is a self-limited, localized swelling of the skin or mucosa that may manifest within minutes to hours post-injection. Distinguishing simple transient swelling from true angioedema requires careful clinical evaluation, as treatment and prognosis differ significantly. Anaphylaxis and hereditary angioedema must be ruled out, and, in allergic patients, the complication may occur at various time points due to different underlying mechanisms.

Nickel allergy and other immunological risk factors

Hypersensitivity to nickel is believed to involve 8% to 19% of the adult population[17]. Prevalence in female sex is 3-10 times higher than in men. In most cases nickel allergy is not due to occupational exposure[18]. In addition, nickel hypersensitivity is very often accompanied by hypersensitivity to other metals (e.g. chromium and cobalt).

Nickel hypersensitivity is a delayed cell-mediated reaction (type IV immune-mediated reaction) due to the presence of nickel ions. Ions penetrate through the skin and bind to Toll-like receptor 4 (TLR4) of dendritic cells (DCs), which migrate to the lymph nodes where nickel is presented to the naïve T cells through the major histocompatibility complex (MHC) II. If DCs activation is sufficient, allergen-specific T cells and memory T cells proliferate and migrate into the blood, to the skin, leading to sensitization. In the absence of adequate dendritic cell activation, immune tolerance is induced.

Some aspects on the subject are not yet clear, for example the mechanism of activation of CD8+ T cells in nickel allergy is still debatable, as is the role of tissue-resident T cells in lowering the threshold value for a local reaction[17].

Subsequent exposure to the antigen involves the activation of the related T cells which, overturned in the circulatory stream, show clear signs of hypersensitivity at a distance of 48-72 hours[19].

It is now clear that there is a second class of nickel-related disorders known as systemic contact dermatitis or Systemic Nickel Allergy Syndrome (SNAS) in which both Th2 and Th1 related immunological mechanisms are involved[20–22].

At this point it is necessary to consider that the needles used in all the procedures to which the patient underwent are needles of two common manufacturers containing nickel stainless steel AISI 304, probably the most widespread on the market. In stainless steel AISI 304, nickel content ranges from approximately 8% to 11%. Nickel skin penetration is hindered by stratum corneum which can lead to a delay of 50 hours, and this is believed to predispose a cumulative effect. It is evident that barrier disfunction (e.g. iatrogenic, as the needle usage) may augment metal skin absorption: while stainless steel (e.g., 304) contains ~8–10% Ni, available data suggest very low Ni release under sweat/contact conditions; nonetheless, mechanical disruption and repeated microtrauma may increase exposure in sensitized individuals. Nickel-reduced devices or protective coatings may be considered in patients with high levels of sensitization[23].

It is important to point out that the only apparent difference between session 2 and 3 (beyond the operator, his experience and geographical location), reported by the patient, is the duration of the procedure, contact of the needle with the labial area and the quantity of the product (it was more in session 3).

To the best of our knowledge, in the literature there is only few cases of allergy reactions to nickel in aesthetic medicine[24,25]. However, it is worth recalling the existence, although not frequent, of type IV hypersensitivity reactions in dentistry, in particular in the use of oral prostheses[26] and angioedema of the face as a result of prolonged contact with nickel[27], and in other fields of medicine[22,28].

Key recommendations from international and expert guidelines on angioedema management

It is important to distinguish angioedema in the anaphylactic reaction due to its severity and the different treatment that it involves, for example, the use of adrenaline. Anaphylactic reaction symptoms include urticaria, itching, hyperemia, bronchospasms, hypotension and angioedema.

Consensus statements and emergency care studies indicate that most angioedema cases (histamine-mediated) resolve spontaneously or with antihistamines ± short courses of corticosteroids. In cases with airway involvement, epinephrine administration and urgent medical attention are mandatory. Bradykinin-mediated forms do not respond to antihistamines/steroids[29]; life-threatening manifestations are extremely uncommon[30–32]. Nevertheless, clinicians should remain vigilant for complications, particularly in individuals with pre-existing allergy risk factors, repeated filler exposure, or recent

infections. Ongoing surveillance and the introduction of new cross-linked HA fillers have not notably increased angioedema rates, supporting the overall safety of these products[33,34].

Discussion

Recent prospective studies and systematic reviews consistently demonstrate that the overwhelming majority of local adverse events following hyaluronic acid (HA) filler injections - such as swelling, discomfort, and minor ecchymoses - are mild in nature and typically resolve spontaneously within a few days. Swelling is known to be a side effect of HA fillers as well as anaphylactic reactions[42]. BDDE-crosslinked HA fillers demonstrate a favorable biocompatibility profile without evidence of clinically significant toxic byproducts in available reviews[43].

The incidence of true angioedema, which is characterized by distinct, asymmetric, and prolonged swelling often accompanied by systemic symptoms, remains exceedingly low, being reported at well under 1% in the most recent series. It is now well established that allergic or immune-mediated reactions are seen more frequently in patients with relevant medical histories, including those with multiple drug allergies, atopic predisposition, or known sensitivities to metals. These patients may benefit from individualized pre- and post-procedural pharmacological prophylaxis. Notably, the introduction of newer-generation HA products - including cross-linked and rheologically tailored gels—does not appear to increase the rate of severe immune-mediated reactions; indeed, data from multicenter trials confirm a robust safety profile overall[43].

Angioedema following HA lip fillers, while uncommon, demands close attention due to its potential severity and impact on patient safety and satisfaction. The present literature, including large multicenter studies, systematic reviews, and rare case reports, supports the overall safety of these procedures but identifies clear risk categories that warrant heightened vigilance.

The present case, and a limited body of literature, also draw attention to nickel allergy as a possible cofactor in angioedema after lip fillers. Needles for filler delivery almost universally contain nickel alloys, and skin barrier disruption may increase percutaneous absorption and immune activation in sensitized individuals. There is indirect evidence of nickel-induced adverse reactions from both dermatology (systemic nickel allergy syndrome) and dentistry, but only a few reports document Ni-related reactions in aesthetic dermatology (notably with microneedling rather than filler injection)[24].

The proper management of emergencies within aesthetic medicine clinics remains an important element[44-46], so we wanted to share our approach algorithm. This is important because training and systematic approaches are fundamental to proper management of emergency[47-49].

To date, only two clinical cases have been reported in the literature describing nickel-related hypersensitivity and angioedema following aesthetic filler procedures. Our case report adds new evidence to this rare association, presenting the first documented instance of recurrent angioedema after hyaluronic acid lip filler in a nickel-allergic patient with extended follow-up across multiple procedures and operators. This longitudinal approach allows for a more detailed assessment of the interplay between patient-specific immunological factors, device composition, and technique.

The lack of complete laboratory data and detailed product specifications represents a limitation in this report; however, all available medical and procedural information was verified through patient records and clinician notes. This case contributes to the evolving understanding of immune-mediated reactions in aesthetic medicine, particularly in the context of concomitant nickel allergy. Continued research to refine patient selection, procedural protocols, and management algorithms (Table 3) - together with the detailed reporting of rare but significant complications - will further mitigate risk and inform best practices.

Table 3. Management in the aesthetic clinic setting.

	Action	**Management**
Initial Assessment	Rapidly assess airway, breathing, and circulation (ABCDE)	Rapidly assess airway, breathing, and circulation. Always prioritize airway protection for any signs of respiratory distress, voice change, or stridor.
Differentiation of Angioedema Types	Distinguish between:	
	histaminergic (allergic/urticaria-associated),	**First-line:** non-sedating, second-generation H1-antihistamines (increased dose if required). **Severe or refractory cases:** short course of systemic corticosteroids is recommended as adjunct. Epinephrine (adrenaline) is indicated if there are features of anaphylaxis[11,29,32,35].
	bradykinin-mediated (hereditary/acquired),	Antihistamines and corticosteroids are generally ineffective. Generally self-limitant, **if not or severe:** centralize and specific therapy - bradykinin receptor antagonists (e.g., icatibant), C1-inhibitor concentrate, fresh frozen plasma (for HAE) [29,32,35–37].
	and idiopathic forms	Corticosteroids, anti H1 histamine and epinephrine in the presence of laryngeal involvement[29,31,35,38,39].
	for every form	Oxygen, intravenous fluids as indicated, monitoring for progression, and rapid emergency department transfer for airway involvement[37,40].
Referral and Follow Up:		Refer patients with recurrent, unexplained, or severe angioedema to allergy/immunology specialists for further evaluation and consideration of underlying causes (e.g., C1-INH deficiency, drug-induced forms) [11,31,32,36].
Education and Prevention:		Counsel patients about avoidance of known triggers, medication review (especially ACE-inhibitors), and importance of carrying emergency medications for severe allergy-prone patients[9,11,41].

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