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Case Report

Histamine Angioedema in Children: A Rare Entity Not to be Misunderstood

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Abstract

Angioedema is a common symptom with different etiologies, diagnostic orientation is based on the associated signs. The most common are histamines, non-IgE-mediated, mild and often associated with hives. Anaphylaxis angioedema and bradykinin angioedema are rare but potentially severe due to upper airway involvement. We report the case of a 14-year-old patient with a history of bilateral orbital swelling associated with deep urticaria, and a brother followed for angioedema. Presenting with bilateral palpebral and jugal edema, associated with urticaria tasks in the trunk and back. The diagnosis of histamine angioedema was made and confirmed by the response to corticosteroids and antihistamines, and a biological assessment ruling out differential diagnoses. Treatment was based on crisis management and the implementation of substantive treatment for recurrent and disabling seizures. Education measures associated with this drug treatment are also essential.

Keywords: *Angioedema; Histamine; Bradykinin; Children; Antihistamine*

Abbreviation

AE: Angioedema

Introduction

Angioedema is a common condition, characterized by localized and transient swelling of subcutaneous or submucosal tissues. Three etiological mechanisms can be at the origin of its constitution: histamine, bradykinin and idiopathic. Through this study, we report the observation of a 14-year-old girl with histamine angioedema after free and informed consent from her parents.

Case Report

This is a 14-year-old girl from an unrelated marriage. She has a history of hospitalization for bilateral orbital swelling associated with urticarial lesions, treated as orbital cellulite stage I, her brother is followed for angioedema put on antihistamine with healing at the age

of 18 years. It is present for bilateral palpebral and jugal edema, of brutal installation, associated with erythematous, not pruriginous, not confluent, not inflammatory at the trunk and back (Figure 1), without reaching the mucous membranes, without breathing. The interrogation ruled out the notion of exposure to a known allergen, or taking medication. In addition, the patient presents mictional burns for 3 days, all evolve in context of apyrexia and conservation of the general condition. The biological assessment showed a CRP at 28 mg/l, without hyperleukocytosis or hypereosinophilia. With hyperleukocytology on the cytobacteriological examination of the urine. She was given hydrocortisone 5 mg/kg/6h haemisuccinate with cetirizine 10 mg/d antihistamine and Ceftriaxone 50 mg/kg/d for urinary tract infection. The evolution was marked by the complete disappearance of edema and urticarial lesions without scarring 12 hours after the start of treatment (Figure 2).

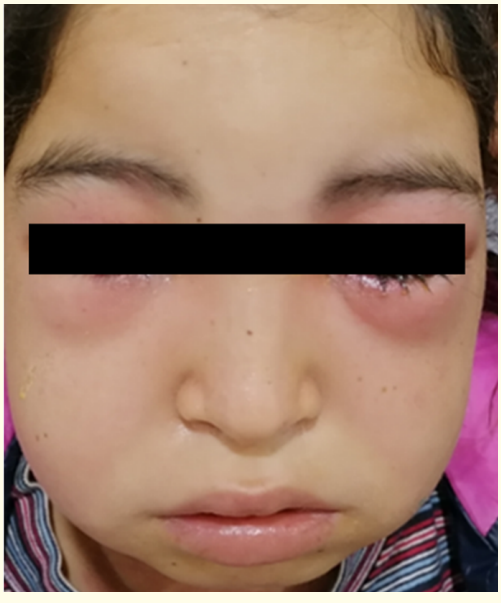

Figure 1A: *Palpebral and jugal edema.*

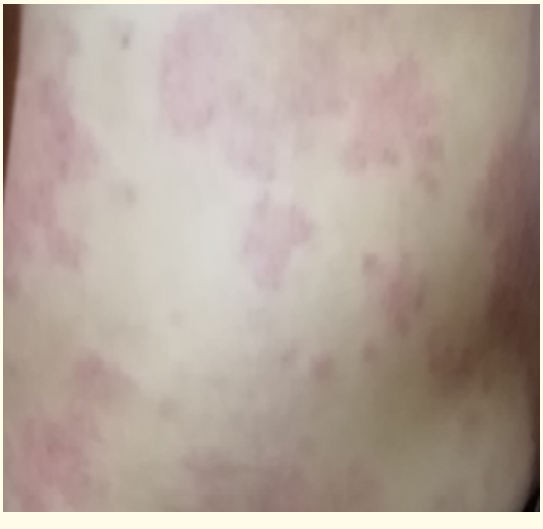

Figure 1B: *Urticarian lesions in the trunk.*

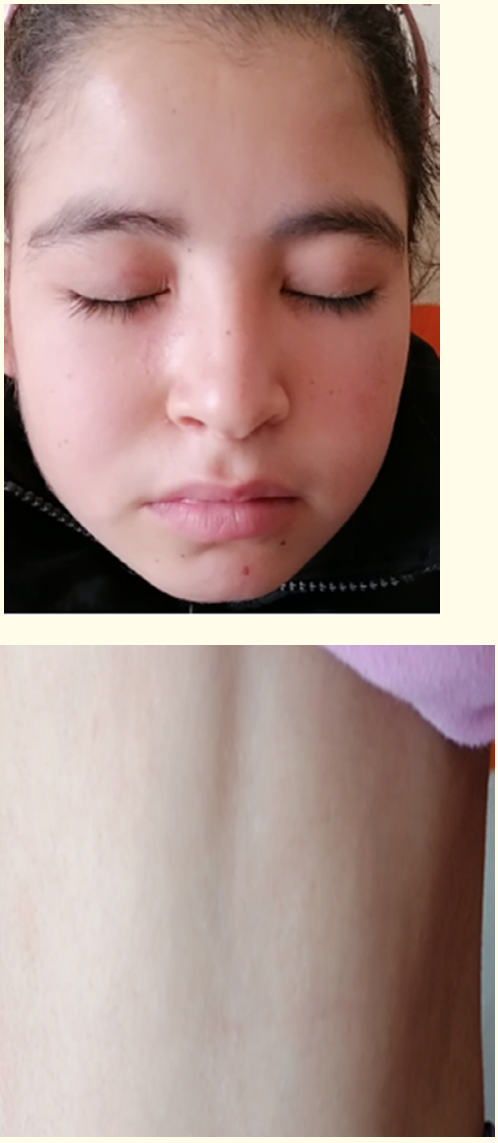

Figure 2: *Removal of edema and lesions after 12 hours of treatment.*

Before this symptomatology, we evoked an allergic reaction, a hereditary bradykinin angioedema, a histamine angioedema, and a disease of the system including vasculitis or lupus. For this a biological assessment showed a normal C1 inhibitor level at 0.35 g/l, Assay of C3 at 1.25 g/L, and C4 at 0.279 g/L normal, and an immunological assessment comprising: Anti SSB/ SSA/ Ac Anti DNA native/ Ac Antiphospholipids IgM and IgG/ Fc Rheumatoid, were negative. Skin test revealed cockroach sensitization.

The diagnosis of histamine angioedema decompensated probably by the urinary tract infection was retained before: the response to corticosteroids and antihistamines, the etiological assessment ruling out other diagnoses. The patient was treated with Cetirizine 10 mg/d antihistamine for 3 months, combined with corticosteroids at 1 mg/kg/d for 15 days with progressive degression, combined with adjuvant treatment and avoidance of cockroach contact. The evolution after 3 months was marked by the total disappearance of the symptoms without relapse.

Discussion

The exact incidence and prevalence rates of pediatric angioedema are not well known, and any reported rates will clearly vary depending on whether the angioedema is considered with or without hives, or on a specific cause of angioedema. Two of the studies indicated that the prevalence of non-hereditary angioedema was 4.9 and 7.4%. Other studies have indicated that 15% to 25% of the population at some point in their lives will suffer from urticaria/angioedema [1]. For histamine angioedema, it is difficult to judge the exact prevalence. It has been estimated that the lifetime prevalence of all types of hives is 8.8% to 10.8% [2]. The main cause of angioedema is histamine, via dependent IgE mast cell activation, or not dependent IgE [3,4].

Histamine angioedema is similar to hives, it is the clinical manifestation of a deep hives affecting the hypodermis or deep dermis [5]. Normal skin colour, or discreetly pink, of variable, non-pruriginous consistency. It can be associated with a feeling of subcutaneous tension, or even pain or burning. It disappears without sequelae but can recur. It is of sudden installation and of short duration, as a rule less than 24 hours. Some episodes have been described as lasting up to 72 hours. This is superimposed on our study where we have a total regression of edema and hives 12 hours after starting treatment without sequelae or scarring.

In the presence of a patient with angioedema in the emergency department, there is no rapid diagnostic examination to determine the histamine or bradykinin mechanism. It will be necessary to use clinical elements seen previously. In our observation, the patient had a brother followed for angioedema, which may be in favor of hereditary bradykinin origin, but the response to corticosteroids and antihistamines pointed to histamine origin. The rest of the balance should include examinations to rule out other differential diagnoses and look for the triggering factor. This is the assay of the C4 fraction of the complement, the functional and weight assay of C1 Inh during the seizure, the assay of C1q [6]. In our case, all the results were normal, which is in favor of the histamine origin.

Treatment is based on conditioning, checking the release of the upper airways, Methylprednisolone bolus 15 to 30 mg/kg (or 1 mg/m^2), in case of respiratory distress, adrenaline is indicated in the emergency treatment, in hospitals, it is used at the dose of 0.01 mg/kg intravenous or intramuscular. The background treatment is based on antihistamines for 3 months, preferably second generation which have less sedative and anticholinergic effects. The molecules used are Loratadine or Cetirizine, from 2 years and over 30 kg, at a dose of 10 mg/d, or cetirizine at a dose of 2.5 mg*2/d from 2 to 6 years and 5 mg*2/d from 6 to 12 years [7]. In our case we put the patient on cetirizine 10 mg/d, for its affordable price in our context, in combination with corticosteroids 1 mg/kg/d for 15 days with progressive degression over a total duration of 4 to 6 weeks and adjuvant treatment.

Conclusion

Histamine angioedema in children is a rare pathology, to be recognized in front of a deep hives of acute installation, responding to corticosteroids and antihistamines. Indeed, the substantive treatment associated with the education of parents is essential in the long-term management.

Conflict of Interest

None.

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Author Contributions

EF K wrote the manuscript. CF, RN, DG supervised the findings of this work, MB approved the findings of this work. All the authors have read the final version of the manuscript.

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