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Skin Pruritus from Atopic Dermatitis Revealing Thrombopathy

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Abstract

Atopic dermatitis is a very frequent dermatosis with an estimated incidence of 10% in young children, in some individuals the disease can persist until adulthood.

We report in our observation a case of atopic dermatitis that reveals after many complications a platelets abnormality.

To our knowledge, such an association has never been described in the literature.

Keywords: Atopic dermatitis; Glanzmann thrombasthenia; Hemorrhage syndrome

Introduction

Atopic dermatitis is a chronic, recurrent and common inflammatory skin condition, which mainly affects young children with an incidence of 10% [1]. This is an IgE-mediated response to minimal amount of common environmental proteins such as pollen, house dust, house dust mites and food allergens.

Glanzmann's Thrombasthenia (TG) is an inherited thrombopathy, linked to an abnormality of the platelet membrane receptor αIIbβ3 (GPIIb-IIIa) which can be qualitative or quantitative. The absence or marked decrease in platelet aggregation is the main feature of the disease [2].

Case Presentation

This is a child of 2 years and 6 months, from first-degree consanguineous parents, with a history of atopic dermatitis in the siblings of spontaneous resolution around adolescence and a chronic eczema rash since birth. The patient had been admitted with a febrile rash for 15 days.

Clinical examination found erythematous, itchy skin lesions with vesicles, scabs and pustules associated with very severe cutaneous xerosis leadingto "kaposi juliuesberg" diagnosis (Figure 1, 2 and 3), the patient responded well to antibiotics treated with amoxicillin and clavulanic acid, intravenous aciclovir, emollients and topical corticosteroids (Figure 4).

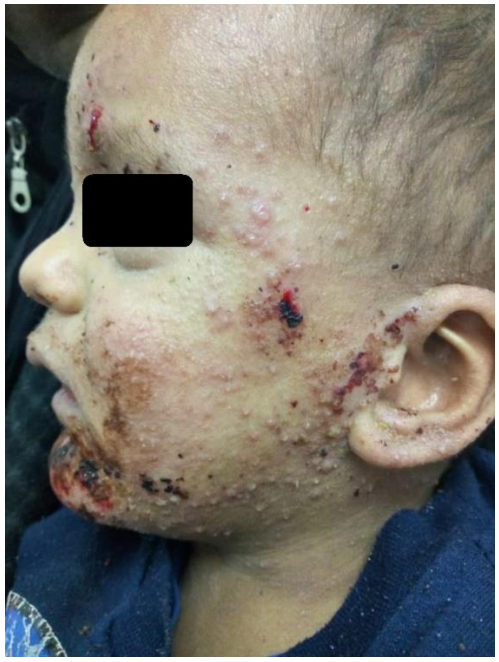

Figure 1: Lesions in the face.

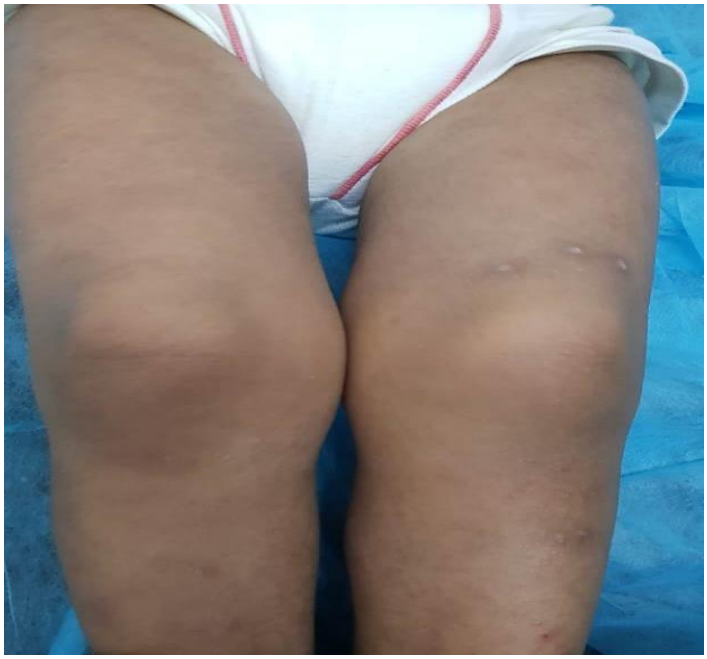

Figure 2: Lesions in the lower limbs.

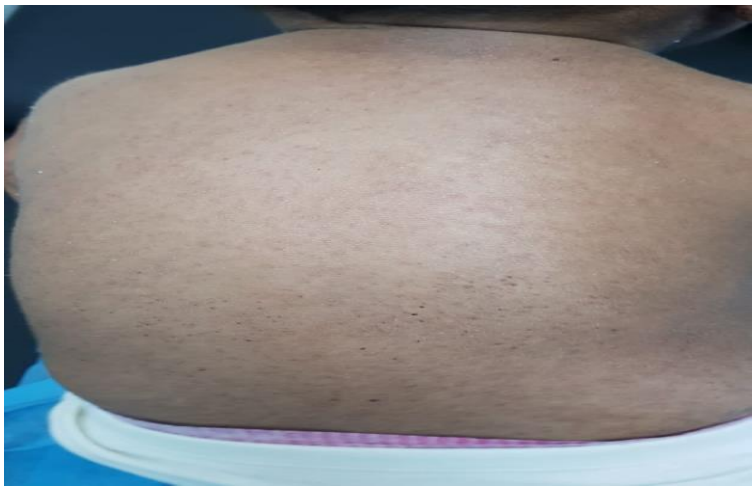

Figure 3: Cutaneous xerosis on the back.

Figure 4: Disappearance of lesions after treatment.

In addition, the patient presented an extended bleeding time at the scratching lesions. The resumption of questioning revealed recurrent gingivorrhagia.

Paraclinical examinations showed eosinophilia at 4100/mm^3 and IgE at 32802 IU/L, the immunoglobulin profil and lymphocyte subtyping were normal, the anti-vaccine antibodies were normal.

The AD-HIES score is 27; A skin biopsy taken was in favor of atopic dermatitis, Haemostatic screening tests were normal, moreover, functional platelet tests were in favor of glanzmann thrombasthenia (Figure 5).

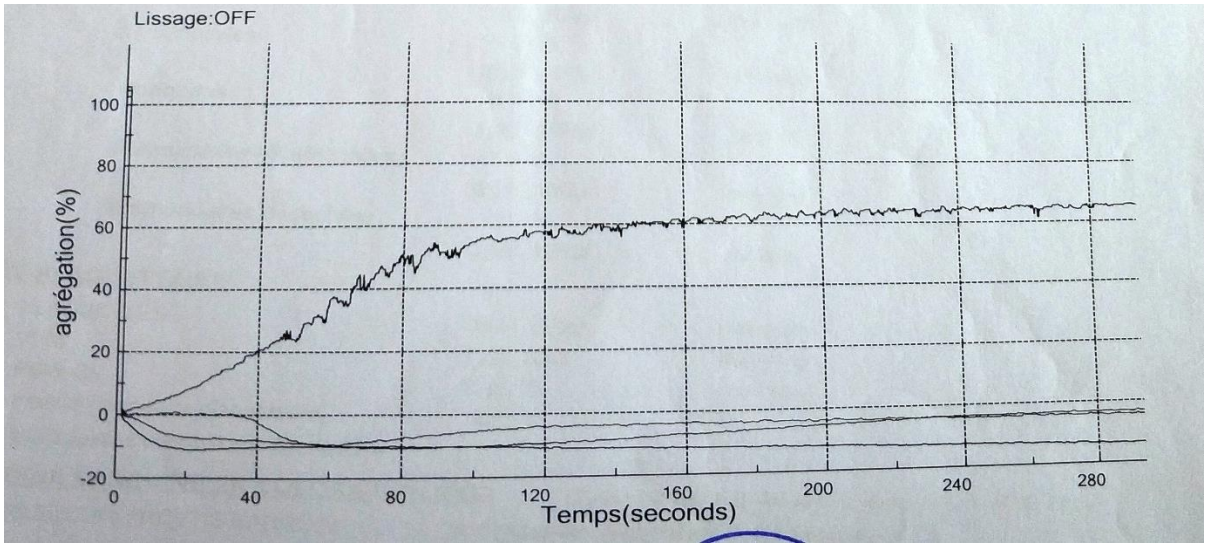

Figure 5: Result of the platelet aggregation test.

Discussion

Atopic dermatitis is a chronic, recurrent and common inflammatory skin disorder, which mainly affects young children with an incidence of 10% [1].

The etiology of atopic dermatitis is still under study, both genetic and environmental risk factors are involved, the etiopathogenesis remains multifactorial involving immunological processes of IgE-mediated hypersensitivity and specific T lymphocytes and mechanisms linked to skin barrier dysfunction [3].

Glanzmann's thrombasthenia is a disease affecting the megakaryocyte lineage characterized by a defective platelet aggregation which clinically manifests as cutaneous-mucous hemorrhagic syndrome. It is linked either to an absence of formation of the IIb/IIIa complex or to an abolition of the integrin function of the IIb/IIIa complex, however normally formed secondary to the destruction of a disulfide bridge in the EGF region of the GPIIIa protein [4,5].

Genetically, atopic dermatitis is linked to several mutations in the gene encoding filaggrin, located on chromosome 1q21 [6]. These mutations are semi-dominant due to the total absence of symptoms in some heterozygous patients, their penetrance is incomplete [7], whereas Glanzmann's disease is transmitted in an autosomal recessive manner with several mutations in the ITGA2B (Integrin α2β) genes encoding for the GPIIb glycoprotein, and ITGB3 (Integrin β3) encoding the GPIIIa glycoprotein, located on chromosome 17 at position 17q21.32 [5].

In our observation, close clinical monitoring of complications of atopic dermatitis led to the discovery of another underlying pathology; The coexistence of these two pathological entities with a different pathophysiological mechanism and genetic substratum is most likely only a sporadic case since these mechanisms are not linked to each other.

Conclusion

The association between Glanzmann's thrombasthenia and atopic dermatitis has never been described before in the literature; Since the loci and mechanism of genetic transmission are different, this may be a sporadic case in the literature.

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