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Bilateral Eyelid Swelling Associated With Acute Hypothyroidism[☆]

Edema palpebral bilateral asociado a crisis hipotiroidea

The eyelid is subject to considerable deformity owing to the particular laxity of the subcutaneous cellular tissue, which is caused by infiltration of the interstitial spaces. The degree of swelling can range from simple tumefaction of the ciliary margin to a considerable increase in periocular tissue volume, which in some cases can lead to narrowing, or even closure, of the palpebral fissure. Differential diagnosis of eyelid swelling is complex. It requires a meticulous physical examination and inclusion of both inflammatory and non-inflammatory causes. The main noninflammatory cause is acute hypothyroidism, as in the present case.¹

We describe the case of a 52-year-old woman who presented with a 1-week history of sudden-onset bilateral eyelid swelling that was neither painful nor pruriginous. Her personal history was remarkable for autoimmune hypothyroidism (Hashimoto thyroiditis) that first appeared 5 years previously and for which she was receiving hormone replacement therapy (levothyroxine 75 µg/d). She also had a 1-year history of autoimmune hepatitis, which was treated with prednisone (7.5 mg/d po), and hypertension, which was treated with enalapril (20 mg/d po). She attended our clinic with bilateral eyelid swelling that had begun 24 hours earlier. The patient had not ingested new drugs, experienced injury, received insect bites or stings, or applied cosmetic products to the area. Examination revealed soft bilateral edema that was more pronounced on the right side with discrete erythema and no crepitus, vesicles, or desquamation (Figs. 1 and 2). Her visual acuity and eye movement were unaltered, and she had no skin lesions at other sites. The results of a full laboratory workup were normal, although thyroid-stimulating hormone (TSH) was 50.3 mIU/L and free thyroxine was diminished (0.5 ng/dL). The dose of levothyroxine was increased considerably (500 µg/d), as was that of oral prednisone (15 mg/d), to reduce inflammation. Seven days after the first visit, her TSH level had fallen to 35 mIU/L, thyroid hormone levels had returned to normal, and the swelling had improved considerably. A week later, the skin



Figure 1 Eyelid swelling 24 hours after onset. The lesion was neither warm to the touch nor painful and was more pronounced on the right.



Figure 2 Bilateral palpebral swelling involving the nasal root 72 hours after onset.



Figure 3 Swelling resolved after improvement of acute hypothyroidism.

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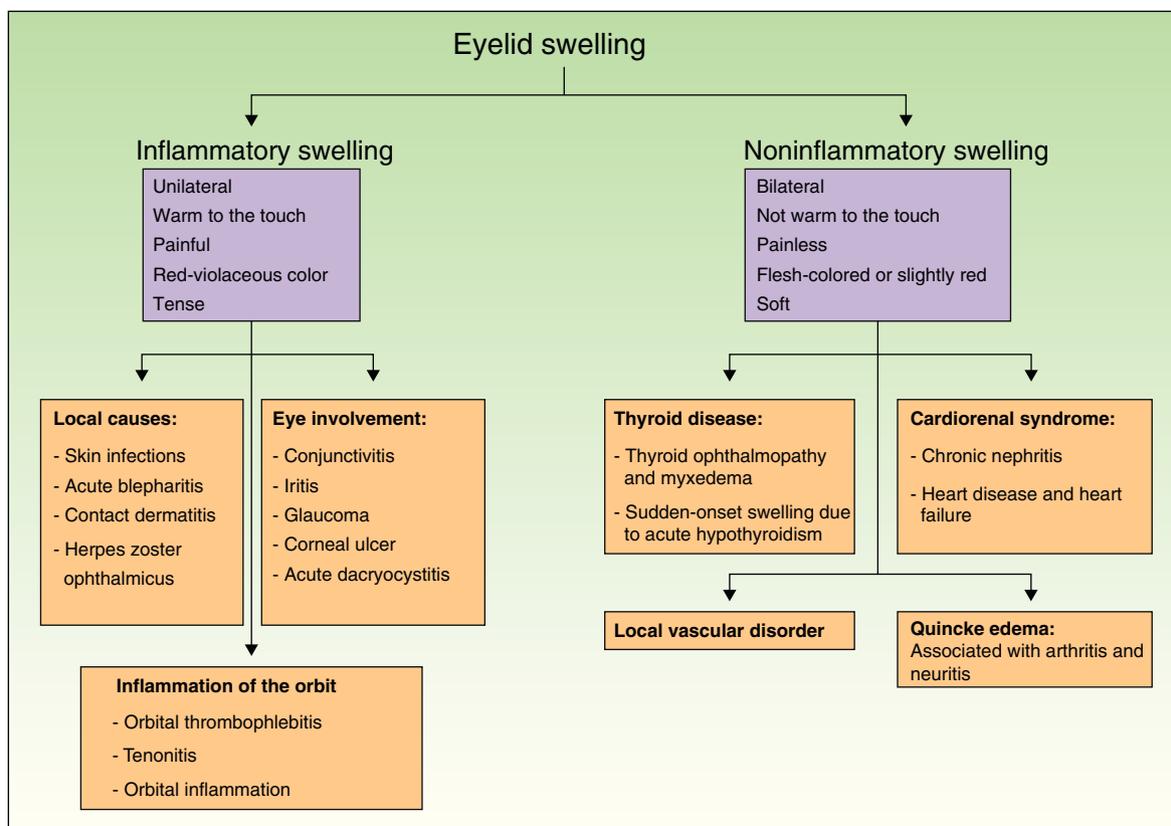


Figure 4 Diagnostic algorithm for palpebral swelling.

complaint was in total remission (Fig. 3) and the TSH concentration had fallen to 20 mIU/L.

The characteristics of the lesion and the clinical course confirmed a diagnosis of bilateral eyelid swelling associated with acute hypothyroidism.

The differential diagnosis of eyelid swelling should include various clinical entities (Fig. 4). A careful evaluation should be made of the intensity, location (one or both eyelids), and coloring of the swelling. The initial examination first makes it possible to rule out swelling caused by trapped air, with its characteristic crunching snow sensation, and blood pooling in the case of hematoma. Finally, it is necessary to define whether the swelling is inflammatory or noninflammatory and analyze the possible causes.

Eyelid swelling associated with hypothyroidism is uncommon and can occasionally mimic other conditions such as angioedema, bites and stings, and contact dermatitis.² In the case we describe, the absence of induration, vesicles, and pruritus make a diagnosis of eczema or angioedema unlikely; in addition, the patient's clinical condition improved considerably when the acute hypothyroidism resolved. The causes of swelling are not well known, although mucopolysaccharide deposits play a role. Case reports show that eyelid swelling is a clinical sign of severe acute hypothyroidism. It is sometimes more subtle, is associated with thinning of the eyebrows,³ and, in some cases, can manifest as palpebral redundancy resulting from laxity of the eyelid.⁴ The swelling resolves gradually with treatment of hypothyroidism.^{4,5}

Eyelid swelling is not the only dermatologic manifestation associated with hypothyroidism, and other more common presentations sometimes go unnoticed. In more than 80% of cases the epidermis is thin, dry, rough, hyperkeratotic, and covered with fine superficial scales.⁶ The hair is opaque, rough, and brittle, partly owing to reduced secretion of sebum. Alopecia has been reported in up to 50% of patients and can be diffuse or partial; the nails are thin and fragile with striations and horizontal and longitudinal ridges.⁷

Finally, in the case of sudden-onset asymptomatic bilateral soft swelling in which dermatitis and angioedema can be ruled out, systemic causes such as hypothyroidism should be suspected. Therefore, eyelid swelling can be a sign of numerous local and systemic diseases and requires a meticulous clinical workup in order to determine the cause and enable appropriate treatment to be started.

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Dyshidrosiform Linear Immunoglobulin A Dermatitis[☆]

Dermatitis Inmunoglobulina A lineal dishidrosiforme

Dyshidrosiform eruptions have a broad differential diagnosis.¹ Linear immunoglobulin (Ig) A dermatitis is a rare, autoimmune blistering disease that can be idiopathic or drug-induced,² and is characterized by a linear deposit of IgA in the epidermal basement membrane.^{3,4} We describe a case of linear IgA dermatitis that began as a dyshidrosiform eruption.

A 56-year-old woman presented at the emergency room with palmoplantar blisters and vesicles (Fig. 1) that were very painful and itchy and had appeared 24 hours previously. She was diagnosed with dyshidrosis. Several days later she developed blisters on the trunk and extremities, without mucosal involvement. A biopsy of one of the lesions was performed. Three weeks previously the patient had been treated for a urinary tract infection with oral fosfomicin, and had experienced diarrhea secondary to the antibiotic treatment. Laboratory tests, including indirect immunofluorescence analysis of antinuclear, antitransglutaminase, anti-intercellular adhesion, and anti-basement membrane antibodies, were normal. Histopathology revealed subepidermal blisters that contained a papillary dermal infiltrate consisting of abundant neutrophils and formed noneosinophilic microabscesses at the tips of the papillary ridges. Direct immunofluorescence (DIF) revealed linear

IgA deposition in the epidermal basement membrane, but no deposits of IgG or C3 (Fig. 2). Based on these findings the patient was diagnosed with linear IgA dermatitis. She responded well to treatment with oral prednisone with no recurrence within 6 months of stopping treatment.

While the initial appearance of palmoplantar vesicles and blisters suggested a diagnosis of dyshidrotic eczema, the subsequent appearance of lesions on the trunk and extremities was indicative of a blistering disease. Histopathology and the results of the DIF led to a diagnosis of linear IgA dermatitis.

Linear IgA dermatitis can be clinically and histologically similar to bullous pemphigoid and dermatitis herpetiformis.³ These 3 entities are characterized by subepidermal blister formation, an inflammatory infiltrate, and immunoglobulin deposition in the epidermal basement membrane. They can be distinguished by histological analysis and DIF. In dyshidrosiform pemphigoid the inflammatory infiltrate is composed mainly of eosinophils, and DIF shows a linear deposition of IgG and C3.⁵ Cases of IgA pemphigoid involving linear deposition of IgA and C3⁶ have also been reported. In dermatitis herpetiformis abundant neutrophils are observed at the tips of the dermal papillae and DIF shows granular deposition of IgA.⁵ In linear IgA dermatitis the inflammatory infiltrate is composed mainly of neutrophils, and DIF reveals linear IgA deposition in the epidermal basement membrane.⁵

It should be noted that although dyshidrosiform linear IgA dermatitis is rare, several cases have been reported.^{7,8}



Figure 1 Palmoplantar vesicles of 24 hours duration.

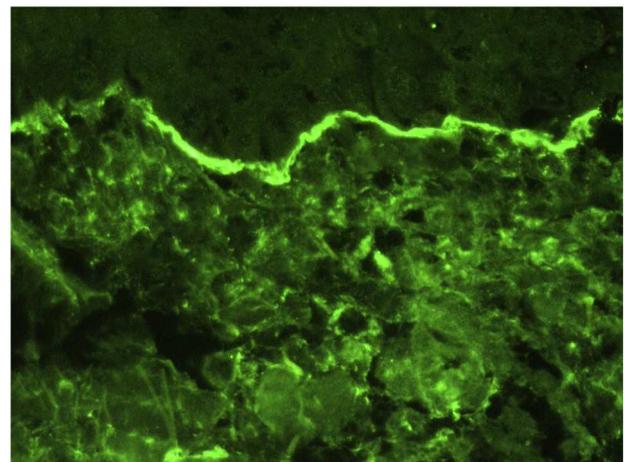


Figure 2 Direct immunofluorescence showing linear and homogeneous immunoglobulin A deposition.

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