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A variant of idiopathic angioedema presenting as persistent facial swelling over 1 year

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Key words: angioedema; facial swelling; skin biopsy.

Angioedema is characterized by swelling of the deeper layer of the skin and/or the submucosa. It is usually localized,

sudden, and transient, lasting from several hours to a few days and is often recurrent. The swellings develop

from extravasation of plasma in the affected areas because of a transient increase in endothelial permeability caused by vasoactive substances produced locally (1). Histopathologically, it presents as localized edema with separation of collagen fibers and blunting and widening of the rete pegs, along with the presence of mast cells and eosinophils (2). We describe here a patient with chronic angioedema that persisted for over 1 year.

A 32-year-old woman visited the allergy clinic for prolonged facial swell-

Idiopathic angioedema presenting as persistent facial swelling over 1 year.

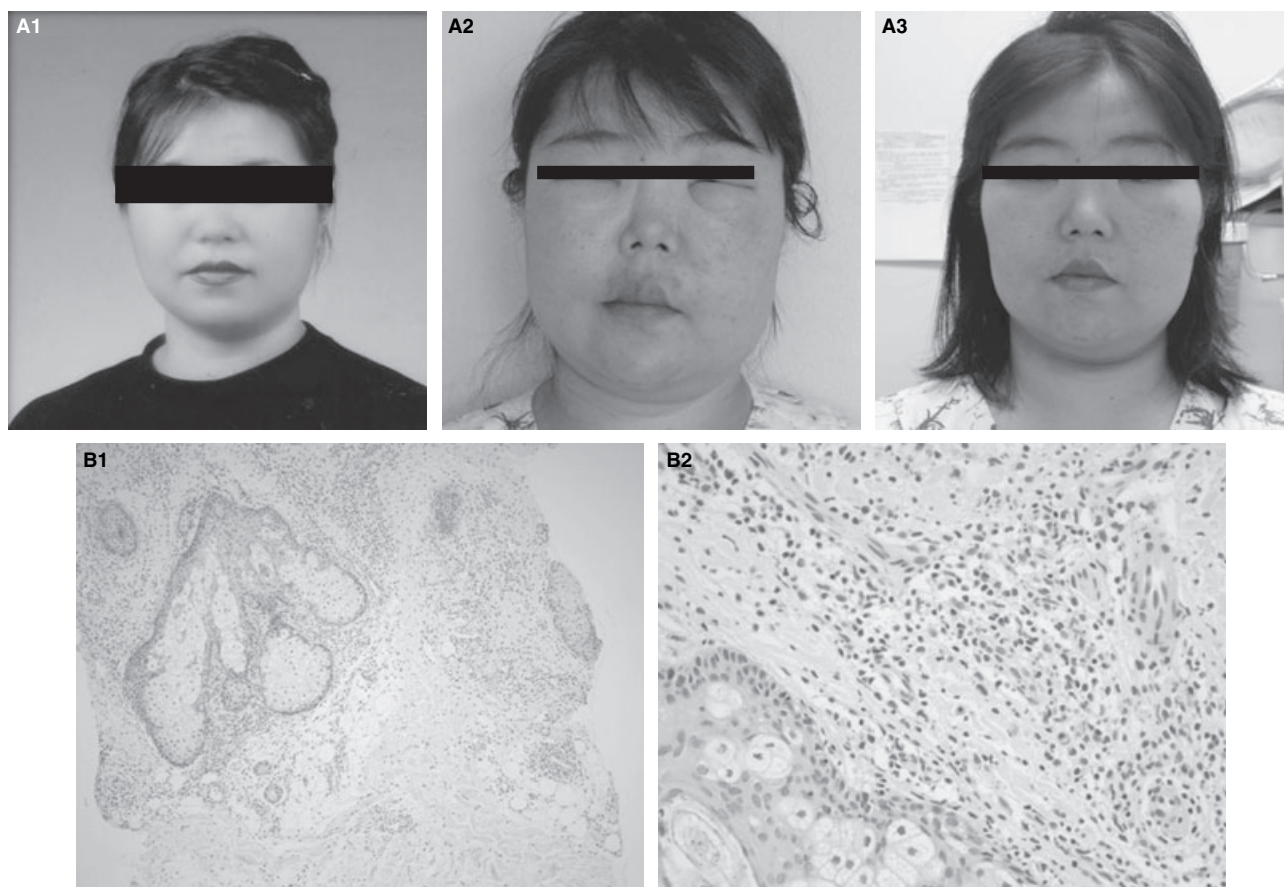


Figure 1. (A) A 32-year-old woman with prolonged facial swelling lasting over 1 year. (A-1) Before development of the disease. (A-2) At time of admission. (A-3) After treatment with high dose steroids. (B) A skin biopsy specimen showing dense lymphocytic infiltration in the deep dermis, particularly in the periappendageal and perivascular areas. (B-1) Hematoxylin–eosin staining; original magnification $\times 100$. (B-2) Hematoxylin–eosin staining; original magnification $\times 400$.

ing, lasting over 1 year and so severe that she was unable to open her eyes (Fig. 1). The swelling was nonpitting, nontender and not warm. The patient had no special familial history and no known exposure to drugs, foods, or chemicals that would cause this swelling. Her vital signs were stable and physical examination was unremarkable. Laboratory assays showed normal erythrocyte sedimentation rate, blood chemistry, thyroid function, and urinalysis, but there was mild leucopenia and anemia. She was negative for antinuclear antibodies and rheumatoid factor, and her serum complement level was also normal. Her chest X-ray, chest and abdomen computed tomography, and carotid artery Doppler were within the normal range. Skin biopsy showed dense lymphocyte infiltration in the deep dermis, particularly in the peri-appendageal and perivascular areas, and increased mast cells and eosinophils. She was treated with systemic corticosteroid therapy (methylprednisolone 30 mg three times a day), which reduced her swelling within a few days. After 1 week, the swelling disappeared almost completely.

In attempting to diagnose the cause of her facial swelling, we considered other conditions that mimic angioedema. Her symptoms and laboratory findings were not compatible with allergic contact dermatitis, facial cellulitis, facial lymphedema, hypothyroidism, superior vena cava syndrome, Melkersson-Rosenthal syndrome or any tumors. Skin biopsy findings were slightly different from those of angioedema and were indicative of lupus erythematosus (LE), Jessner's lymphocytic infiltration, polymorphous light eruption, pseudolymphoma, lymphoma or chronic urticaria. Histopathological findings of LE-specific skin disease included hyperkeratosis, atrophy of the epidermis and infiltration of mononuclear cells into the perivascular and perifollicular areas (3). This patient showed an increase in eosinophils, which was not compatible with LE. Jessner's lymphocytic infiltration consists of red papules and plaques, often annular or arciform, on the upper part of the body, with histopathological findings of dense lymphocytic 'sleeve-like' perivascular infiltrate of the dermis (4), but no eosinophils. Polymorphous light eruption

(PLE) is a common idiopathic photosensitivity disorder with nonscarring pruritic erythematous papules, vesicles or plaques that develop on light-exposed skin. It is accompanied by perivascular lymphocytic infiltrates in the dermis, subepidermal edema and variable epidermal changes (5). The suspected triggers of pseudolymphoma include foreign agents, such as tattoo dyes, insect bites, scabies, arthropod venom, contact agents and hyposensitive infectious agents; in this condition, the skin becomes very itchy and hard (6). The condition of our patient differed from pseudolymphoma because she showed dense lymphocytic infiltration in the dermis as well as other differentiating clinical manifestations. Her pathology results were compatible with chronic urticaria and angioedema, in that there was infiltration of mast cells and eosinophils. The extent of infiltration was probably because of chronic inflammation, persisting for over 1 year.

In conclusion, we report a variant case of constant chronic idiopathic angioedema, persisting for over 1 year and characterized by dense infiltration of lymphocytes as well as mast cells and eosinophils.

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Is house dust mite monosensitization associated with clinical disease?

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Key words: allergy; asthma; atopy; house dust mite.

Controversy still exists on the role of dust mites in atopic diseases. While earlier, mostly cross-sectional studies indicated that exposure to house dust mites (HDM) is closely linked with asthma (1), recent prospective studies have challenged this view (2, 3). Current data, on the other hand, are rather consistent in showing that exposure is associated with specific sensitization.

Most asthmatics are sensitized to many allergens, and the relative risk of one allergen in the development of the disease in such a situation is impossible to evaluate. Very few studies to date have

House dust mite monosensitization is a marker of exposure rather than associated with atopic disease.

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