

Radioiodine as a possible triggering agent for pemphigus foliaceus

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Dear Editor,

Pemphigus foliaceus (PF) is an autoimmune blistering disease presenting in endemic and sporadic forms. The typical presentation is recurrent shallow erosions in a seborrheic distribution¹. PF has also been found in association with autoimmune diseases such as autoimmune thyroid disease (e.g. toxic nodular goiter and Hashimoto's thyroiditis)^{2,3}. In some patients, PF appears to be triggered by radioiodine therapy.

A 54-year-old woman presented with a 2-month history of crusted and superficial erosions on the face, chest and upper back. She denied any photosensitivity, and there was no mucosal involvement. She had history of multinodular toxic goiter since three years ago which was treated with radiolabeled iodine; one month after administration of radiolabeled iodine, cutaneous lesions appeared (Figure 1). All laboratory tests, including cell blood count, liver and renal function tests were within the normal limits except for the thyroid function tests. Of note, antinuclear antibody and anti-double-stranded DNA tests were negative. Two punch biopsies (one lesional and one perilesional skin) were taken. Microscopic examination revealed intraepidermal blister in the upper granular layer containing acantholytic cells (Figure 2). The results of direct immunofluorescence showed IgG and complement 3 (C3) in the intercellular spaces of the epidermal cell surface. These findings were consistent with the clinical diagnosis of PF. According to impaired thyroid function tests which was compatible with the diagnosis of post ablative hypothyroidism, levothyroxine was started for our patient. Intravenous pulse therapies with 1gr/day of methylprednisolone were started in 3 consecutive days to induce remission. Four days after treatment, the cutaneous lesions improved dramatically, then oral prednisolone 40 mg/day and azathioprine 100 mg/day were started and the patient was followed one month after initiation of



Figure 1. Photograph shows crusted and superficial erosions on the face and chest.

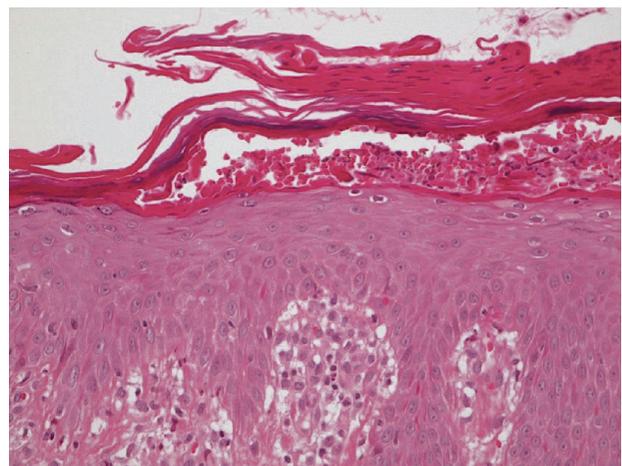


Figure 2. A subcorneal blister with acantholytic cells (H&E, x40)

the therapy, with no residual lesion.

Pemphigus foliaceus is an autoimmune blistering disorder with characteristic lesions that are scaly and crusted, often on an erythematous base. Mucosal involvement is absent, even with widespread disease¹. Autoantibodies against epidermal components mediating cell-cell adhesion result in

acantholytic blisters within the epidermis in the pemphigus group diseases⁴ and in PF. The most common target of autoantibodies are the desmoglein 1 (Dsg1) glycoproteins in the desmosomes. The autoantibody response primarily involves IgG (IgG4 subclass). The effect of the antibodies and the immunological pathway are the most likely mechanisms. A variety of factors are possible triggers for PF. Fogo selvagem, a form of endemic pemphigus foliaceus (rural areas of Brazil,) is likely due to a combination of environmental factors and possibly the patient's genetic susceptibility⁵. Ultraviolet exposure, drugs, nutrition (thiol-containing foods such as garlic and onions), radiation, and infections have also been associated with some cases of PF⁶. Pemphigus has been described in association with various autoimmune disorders. Its occurrence with autoimmune thyroid diseases such as Hashimoto's thyroiditis, primary hypothyroidism, diffuse toxic goiter and autoimmune thyroiditis has been reported. There is strong circumstantial evidence that pemphigus has an autoimmune etiology^{2,3}. Multinodular goiter and toxic multinodular goiter are presumably autoimmune disorders that result from a group of antibodies, known as thyroid-stimulating immunoglobulins, which bind and activate the thyroid receptor sites for thyroid-stimulating hormone³ (pemphigus foliaceus coexisting with toxic multinodular goiter). In some patients, PF appears to be triggered by radioiodine therapy, and bullous pemphigoid has been described in patients treated with radioiodine⁷.

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