

Clinically, eyebrow involvement in FFA is reported as non-inflammatory and nonscarring, with no perifollicular erythema, scaliness or papules.⁴ Among our cases, two had diffuse erythema and two complained of local pruritus.

Dermoscopic features of eyebrow loss in patients with FFA have been described previously. Red and grey dots were considered a favourable prognostic factor for local regrowth, while loss of follicular openings was seen in advanced disease.⁵ The most important finding in our patients was the presence of few black dots and short, curved and broken hairs. No exclamation mark hairs were seen. On dermoscopy without immersion fluid, no peripilar casts or scaling were noticed.

The histology of the eyebrows of our cases showed features of lichenoid inflammation or interface changes over the perifollicular epithelia, similarly to previously published eyebrow biopsies.⁴ Direct immunofluorescence of the eyebrows in FFA has never been reported. Our cases showed variable findings, one negative and one with IgG and IgM deposits in the basement membrane zone.

There is no current standardized treatment for FFA. As there is no cure, a number of treatments have been proposed to control symptoms and halt disease progression. Regarding the eyebrow hair loss, Donovan *et al.*⁶ reported the benefit of intralesional triamcinolone acetonide at a concentration of 10 mg mL⁻¹, with no cases of atrophy. In our case, the injections led to improvement of hair regrowth but were responsible for atrophy. We believe that a 2.5 mg mL⁻¹ concentration is possibly a better choice for these patients.

The differential diagnosis of eyebrow loss includes several diseases ranging from local dermatological disorders to scarring and nonscarring alopecia and systemic diseases.⁷ Isolated eyebrow involvement due to FFA should be considered in all cases of madarosis in women. The presence of few black dots and dystrophic hairs on eyebrow dermoscopy may suggest the diagnosis of FFA. As the eyebrow involvement can be the initial presentation, we encourage dermatologists to perform eyebrow biopsies to diagnose FFA, as early diagnosis can avoid permanent disfiguring hair loss.

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Increased total serum IgE levels in moderate-to-severe hidradenitis suppurativa

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DEAR EDITOR, Hidradenitis suppurativa (HS) is a chronic, recurrent, inflammatory disease affecting mainly the axillary, inguinal and perineal areas. HS commonly presents as painful inflamed nodules that can progress to abscesses, fistulas, odorous discharge and scarring.¹ HS is considered a systemic chronic disease associated with multiple comorbidities.²

Although it was previously considered an infectious disease, the concept of HS as an immunological disease has been gradually accepted.³ It is noteworthy that the inflammatory burden in these patients is prominent, with elevated levels of interleukin (IL)-1 β , tumour necrosis factor- α and IL-10 in HS skin at even greater levels than in psoriatic skin.⁴ On the other hand, serum levels of IgA, IgG and IgM were not raised in a short communication of seven patients with HS.⁵

To the best of our knowledge, studies on total serum IgE levels in HS have not been previously published. Elevated IgE levels are usually related to atopic diseases, but many other disorders such as helminth infections, immunodeficiency syndromes and inflammatory diseases are also associated with high IgE levels. We sought to assess the total serum IgE levels in moderate-to-severe HS and to explore the patient and clinical characteristics associated with high IgE levels.

We conducted a transversal multicentre study in three Spanish hospitals with dedicated HS clinics. As part of a broader multicentre collaboration, and following a pre-established protocol, total IgE, IgA and IgG serum levels were systematically measured in all consecutive patients with HS (Hurley stages II and III exclusively) between July 2014 and November 2015. Demographic, clinical and laboratory data were retrieved

through case note review. IgE was considered elevated if it was above the laboratory's upper normal level ($> 100 \text{ IU mL}^{-1}$).

In total, 99 patients were included. Out of the 99 patients, 50 (51%) were male, the mean $\pm$ SD age was 40.6 ± 13.3 years and the mean duration of disease was 16.0 ± 13.5 years. The axillary region was the most commonly affected (88%). Seventy (71%) of the subjects were smokers. Thirty-six (36%) were overweight and 39 (40%) obese. Sixty-seven (68%) had an axillary-mammary phenotype. Only one patient had a diagnosis of adult atopic dermatitis (with normal IgE levels) and no patient had a diagnosis of asthma.

The mean total IgE level was 186.4 IU mL^{-1} , with a maximum value of 2379 IU mL^{-1} . Thirty-seven patients (37%) with HS had elevated IgE levels (defined by total IgE levels $\geq 100 \text{ IU mL}^{-1}$). Four patients had a concomitant elevation of IgE and IgA, and another three had an increase in IgE, IgA and IgG levels. The mean total IgE was higher in smokers (mean 238.9 vs. 59.4 IU mL^{-1} , $P = 0.02$), men (267.2 vs. 103.9 IU mL^{-1} , $P = 0.04$) and those whose HS was not located on the groin (233.8 vs. 146.8 IU mL^{-1} , $P = 0.03$). A stratified analysis of these three variables did not show any association between them, nor any tendency to confusing variables. No other patient or clinical variables (age, body mass index, duration of disease, number of affected areas, high-sensitive C-reactive protein, Dermatology Life Quality Index, pain, pruritus, Hurley stage, clinical phenotype or pilonidal cyst) showed an association with an increase in IgE levels.

In short, our study showed an increase in serum levels of IgE in about one-third of patients with moderate-to-severe HS. Prior studies have shown that the mean total IgE levels in the general population range from 32 to 41 IU mL^{-1} .^{6,7} The mean in our population was higher, at $> 180 \text{ IU mL}^{-1}$. Higher levels of IgE in male patients have also been previously reported,⁶ and our results are in-keeping with this finding.

van der Zee et al. demonstrated a heavy inflammatory infiltrate in chronic HS skin lesions with a marked increase of CD20^+ and CD79a^+ B cells and CD138^+ plasma cells.⁸ An increased number of tryptase-positive mast cells was also found in early and chronic lesions and in normal-appearing perilesional skin. The T helper (Th) subset in HS is mixed, and does not fit into a classic Th1, Th2, Th17 or Th22 profile.^{8,9}

IL-10 is a major regulatory cytokine of inflammation that is elevated in HS lesions. It is well known that IL-10 enhances B-cell IgE synthesis by promoting differentiation into plasma cells.¹⁰ We hypothesize that elevated IL-10 in HS promotes B-cell differentiation to plasma cells that can produce the IgE detected in HS skin, and can increase the serum IgE levels. This enhancement of IgE production and a dense infiltration of mast cells in HS could trigger degranulation of these cells, releasing histamine and causing pruritus, a frequent symptom

in HS. It remains to be determined whether the heavy inflammatory infiltrate of plasma cells in the HS lesions promotes elevated IgE levels in patients with HS.

Limitations of this research study include the small number of patients and the lack of a healthy control group. Further studies with larger numbers of patients are required to support these results.

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