¿Qué esconde la alopecia? Alopecia universalis como síntoma de inicio de un paciente seropositivo al VIH

What is hidden by alopecia? Alopecia universalis as an onset symptom of an HIV seropositive patient

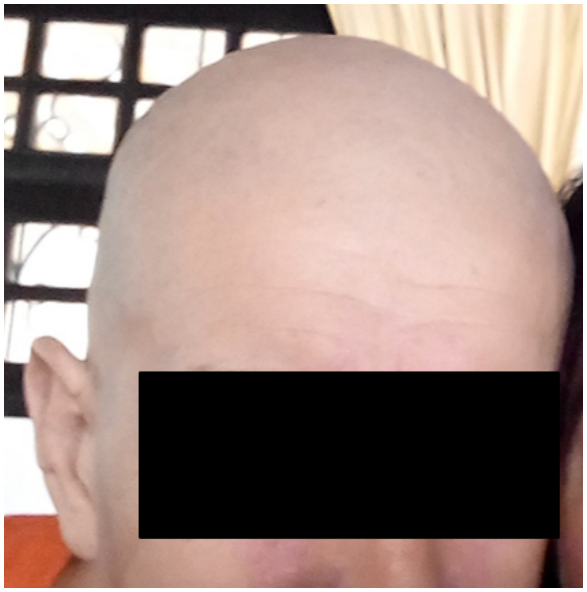
CASE REPORT

Acute and symptomatic HIV infection can be associated to a wide range of symptoms. Up to 90% of HIV-seropositive patients have dermatologic manifestations during their illness course. Seborrheic dermatitis is the most common cutaneous eruption associated to HIV-1 and pruritus is also a usual symptom, sometimes in earlier stages of the disease[1-2]. With regard to hair changes, the most typical in these patients are lengthening of the eyelashes and scalp hair loss[3]. Alopecia areata (AA) is a nonscarring hair loss which has been reported as a rare symptom in HIV-seropositive patients [2-9] . A less common variant in these patients is alopecia universalis, which consists on a total body hair loss[9].

We present a clinical case of a 37-year-old man, caucasian, with medical history of allergic rhinitis, without any regular medication, who presented to the Emergency Room due to angioedema and pruritic rash two weeks after taking nonsteroidal anti-inflammatory drug and amoxicillin-clavulanate because of a zygomatic bone fracture. He had face, neck, arms and legs oedema and itch lesions on his arms and legs. Neck ultrasonography demonstrated a soft tissue oedema. Laboratory examination revealed high white blood cells count (11600 $x10^{12}$/L) and eosinophilia (1500 cells/µL). He was medicated with prednisolone and hydroxyzine with mild oedema improvement.

The patient was referred to the Internal Medicine appointment. He mentioned a predominantly nocturnal pruritic rash and non-quantified weight loss. Physical examination revealed patchy bald spots on the scalp associated to sparse hair in the whole body, itch lesions on his arms and legs, erythematous pruritic papules at his trunk, seborrheic dermatitis on his face and painless axillary and inguinal lymphadenopathy. Laboratory findings showed even higher white blood cells count (14500$x10^{12}$/L) associated to eosinophilia (3000 cells/µL). Antineutrophil cytoplasmic antibodies and specific IgE to penicillin and amoxicillin were normal. Serologic testing for syphilis was negative, as well as hepatitis B and C serologies. However, HIV-1 serology was positive, with a CD4 lymphocyte count of 505 cells/mm , CD4/CD8 ratio of 0,21[3] and viral load of 12700x10 copies/mL. Antiretroviral[3] resistance test was negative and the patient didn't have the HLA B*5701 allele. In three months, patient's alopecia progressed to alopecia *universalis* simultaneously with a decrease in CD4 lymphocyte count (201 cells/mm) and CD4/CD8 ratio (0,18), and[3] an increase of the viral load (18200x10 copies/mL). He started treatment with[3] emtricitabine / rilpivirine / tenofovir (200mg/25mg/245mg daily). In less than one month he noticed hair growth and appetite increasement. Two more months after treatment started, he was already asymptomatic. CD4 lymphocyte count increased to 304 cells/mm , CD4/CD8 ratio was 0,27 and viral load was almost undetectable (0,049x103 copies/mL).[3]

Figure 1. Scalp showing diffuse alopecia, previous to the treatmen



There are several skin manifestations in HIV-seropositive patients, which are not only associated to the terminal stage but can also be seen in the earlier stages of the disease[1].

Seborrheic dermatitis, that our patient had, is one of the most common cutaneous symptoms in HIV-1 patients, being reported in 20 to 40% of them. Besides that, our patient also had pruritus, which is another common complaint, especially in the earlier stages[1,2]. A cause for this pruritus could be eosinophilic folliculitis, which is an unique HIV-1 condition that could justify our patient pruritus and peripheral eosinophilia, although he had CD4 lymphocyte count higher than 300/mm3 at diagnosis, when he had more pruritus complaints. We would need a biopsy specimen of the scalp to confirm it.

On the other hand, AA in HIV-seropositive patients is not common[9], and alopecia universalis is an even rarer symptom. AA is an autoimmune disorder that targets hair follicles, conditioning a nonscarring hair loss, with a worldwide incidence of around 2%. It can be caused by genetic predisposition and environmental factors[9]. Barcaui *et al*[7] showed that alopecia related to HIV-1 infection is the result of a hair cycle disruption. According to this data, there is a higher proportion of telogen follicles with apoptotic follicular stem cells, mainly with less CD4 lymphocytes count at late disease stages. Besides that, there was an architectural disorder of the perifollicular collagen bundles. They even suggested that this apoptosis is caused by virus proteins which interact with cell-cycle.

After treatment start, our patient's CD4 lymphocyte count increased and viral load decreased, associated to hair regrowth and alopecia improvement. Thus, this is in agreement with what Stewart *et al*[6] showed in their study: when the patient's CD4 lymphocyte count and CD4/CD8 ratio was low, the patient had active hair loss and after treatment there was an increase of this ratio and CD4 lymphocyte count simultaneously with hair regrowth. For this reason, we can conclude that alopecia universalis recovery is probably correlated to patient's immune system improvement.

To our knowledge, this is the first case of alopecia universalis in HIV-positive patients reported in Portugal.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest in this work.

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There was no funding.

ETHICAL ASPECTS

The patient signed a written informed consent.

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