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Comment on: Diffuse Large B-Cell Lymphoma with Axillary Cutaneous Invasion in a HIV Positive Patient

Comentário a: Linfoma Difuso de Grandes Células B com Invasão Cutânea Axilar num Doente com Infecção VIH

Keywords: HIV Infections; Lymphoma, Large B-Cell, Diffuse
Palavras-chave: Infecções por HIV; Linfoma Difuso de Grandes Células B

Dear Editor,

We read with great interest the article published by Dias *et al.*¹ The authors report the case of a 34-year-old man with untreated HIV-1 infection that was admitted to the hospital with obstructive jaundice and progressive swelling of the left axillary region.¹ An imaging study by computed tomography scan revealed an expansive 3.2 cm pancreatic mass and multiple hypodense liver lesions. Hepatic and pancreatic biopsy confirmed the presence of stage IV diffuse large B-cell lymphoma.

Additionally, on dermatological examination, the presence of multiple erythematous papules and nodules of variable size, upon an area of nontender swelling, in the left axillary region was also evident, which the authors identified as cutaneous invasion by diffuse large B-cell lymphoma.

The cutaneous manifestations of systemic lymphomas can be varied and are often non-specific. Their diagnosis relies on correlation with histopathological examination and immunohistochemical staining of an appropriate skin biopsy.² This procedure would be a necessary step to establish a definitive diagnosis of skin infiltration by diffuse large B-cell lymphoma.

Notably, a broad spectrum of infectious, inflammatory, and neoplastic skin conditions may develop in the setting of HIV infection,³ particularly in severely immunosuppressed patients, who often have mixed infections or combined infectious–neoplastic or inflammatory–neoplastic lesions.³ The reported patient had a CD4⁺ T-cell count of 133

cells/mm³ (11.8%), which is classified as WHO clinical stage 4 HIV infection (the severely symptomatic stage) and can encompass all the AIDS-defining illnesses.³ In this setting, the differential diagnosis of multiple erythematous papules and nodules is broad and includes entities such as Kaposi sarcoma, cutaneous tuberculosis and non-tuberculous mycobacterial skin infections, fungal infections (for example, chromoblastomycosis, coccidioidomycosis or histoplasmosis) and cutaneous leishmaniasis.⁴

With this comment, we wish to draw attention to the importance of clinical-histopathological correlation for an accurate diagnosis of cutaneous manifestations of systemic diseases.

AUTHORS CONTRIBUTION

JB: Draft of the paper.

MC: Critical review, approval of the final version.

PROTECTION OF HUMANS AND ANIMALS

The authors have followed the protocols of their work center on the publication of data. The data was anonymized and none of the authors had access to patient identification. The study was conducted in accordance with the Helsinki Declaration updated in 2013.

DATA CONFIDENTIALITY

The authors declare having followed the protocols in use at their working center regarding patients' data publication.

COMPETING INTERESTS

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Swift Reversal of Hypercortisolism-Related Dermatological Features Following Treatment in a Patient with Cushing's Disease

Reversão Rápida das Manifestações Cutâneas Associadas ao Hiper cortisolismo após Tratamento da Doença de Cushing

Keywords: Alopecia/etiology; Cushing Syndrome/complications; Skin Diseases/etiology; Skin Manifestations/drug therapy

Palavras-chave: Alopecia/etiologia; Doenças da Pele/etiologia; Manifestações Cutâneas/tratamento farmacológico; Síndrome de Cushing/complicações

Cushing's syndrome (CS) is a rare disorder caused by exposure to high levels of glucocorticoids. Dermatological manifestations are common and often discriminatory of this condition.¹ We report the case of a female with an adrenocorticotrophic hormone (ACTH)-dependent CS who had prominent dermatological features at presentation, which remarkably improved after treatment of the hypercortisolism.

A 74-year-old female presented with prominent and troublesome hair loss and easy bruising of the skin for nine months. She also complained about proximal muscular weakness and had recently been diagnosed with hypertension and diabetes. She denied exposure to exogenous corticosteroids. On clinical examination, she had severe frontovertical alopecia, with the scalp crown practically devoid of hair, suggestive of type III female pattern alopecia (Ludwig classification) (Fig. 1A). She also had reduced skin-fold thickness and exuberant ecchymosis in her upper limbs (Fig. 1B). Facial plethora, hirsutism, purple striae and skin

hyperpigmentation were absent. Her laboratory work-up confirmed endogenous hypercortisolism, with an elevated 24h-urinary free cortisol at 2300 µg/24h (reference range: 124 - 581; urinary volume of 2 liters) and an unsuppressed cortisol level of 35.4 µg/dL (normal < 1.8 µg/dL) following a 1 mg-overnight dexamethasone suppression test. ACTH was elevated at 377 pg/mL (reference range: 7.2 - 63.3) and her serum potassium level was normal at 4.3 mmol/L (reference range: 3.5 - 5.1). The diagnosis of an ACTH-dependent CS was established and a magnetic resonance imaging (MRI) test revealed a large 3 cm-pituitary macroadenoma invading the right cavernous sinus, the sphenoidal sinus and the skull base with significant clival destruction, but with no optic chiasm compression.

The patient started on metyrapone achieving eucortisolemia within a month, after which she underwent bilateral adrenalectomy, as curative pituitary surgery had been deemed unfeasible at our multidisciplinary team meeting. Within three months, her CS-related dermatologic manifestations improved remarkably (Figs. 1C and D).

Skin manifestations in CS patients are frequent and reflect the hypercatabolic effects of cortisol excess on collagenous subcutaneous fibers.^{2,3} Excessive levels of cortisol also reduce the synthesis of proteins, such as hyaluronan and proteoglycans. These proteins are important skin components, but also elements of the hair follicles and are essential for a normal hair follicle cycling and growth.⁴ High levels of ACTH can also lead to overproduction of adrenal androgens, which in turn may further contribute to the CS-related alopecia. Thus, hypercortisolism may impact the fine-tuned mechanisms of hair follicles resulting in hair growth disruption and hair loss.⁴



Figure 1 – Female patient with ACTH-dependent Cushing's syndrome (A) with alopecia (B) and skin ecchymosis, (C, D) which improved remarkably within three months after the treatment of hypercortisolism