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Case for diagnosis. Multiple infiltrated plaques in a patient with human immunodeficiency virus and hepatitis C co-infection: lichen myxedematosus[☆]



Dear Editor,

This report describes the case of a 45-year-old male patient, smoker, diagnosed with human immunodeficiency virus (HIV) and hepatitis C virus (HCV) infection three years before. He was undergoing regular treatment with antiretroviral therapy (ritonavir, tenofovir and atazanavir), and had an undetectable viral load, with a CD4 cell count of 534 cells/mm³, but without treatment for hepatitis C. He complained of cutaneous lesions with two years of evolution, and significant worsening in the last months, with local pruritus. On examination, erythematous, infiltrated papules and plaques were observed in the gluteal region bilaterally, as well as in the left abdominal, cervical and upper dorsal regions (Figs. 1–3). He underwent laboratory tests that showed AST (aspartate aminotransferase) of 82 U/L, ALT (alanine aminotransferase) of 115 U/L, GGT (gamma-glutamyl transferase) of 131 U/L, alkaline phosphatase of 83 U/L, total bilirubin of 1.28 mg/dL, and fasting glucose of 103 mg/dL. Other laboratory tests within normal limits included: Hb, 15.3 g/dL; leukocytes,

5800 mm³; platelets, 205,000 mm³; TSH, 2.51 IU/mL; free T4 1.08 µg/dL; Cr 0.79 mg/dL; non-reactive ANA (antinuclear antibody), non-reactive rheumatoid factor, proteinogram with no monoclonal peaks. A skin biopsy was performed, which showed abundant mucin deposits in the upper and middle dermis (Fig. 4).



Figure 1 Erythematous, confluent plaques with an infiltrated appearance, in the gluteal region, bilaterally.



Figure 2 Erythematous plaque with a papular center on the left abdominal region.



Figure 3 Erythematous, whitish papules on the upper back and posterior cervical regions.

What's your diagnosis?

- Lichen amyloidosis;
- Lichen myxedematosus;

[☆] Study conducted at the Sanitary Dermatology Outpatient Clinic, Secretaria de Saúde do Estado do Rio Grande do Sul, Porto Alegre, RS, Brasil.

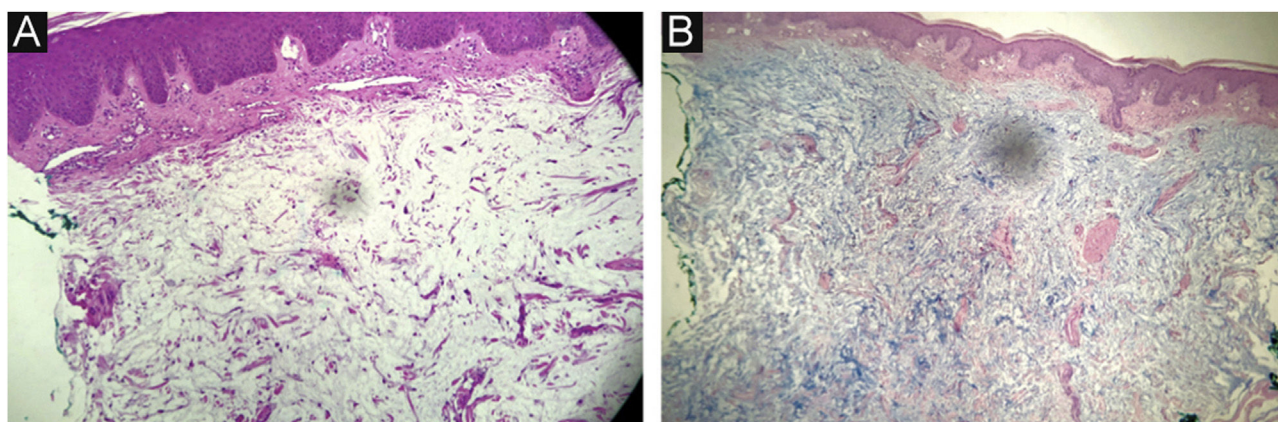


Figure 4 (A) Mucin deposits in the upper and middle dermis, with no associated fibroblastic proliferation; absence of amyloid (Hematoxylin & eosin, $\times 40$). (B) Special staining highlights mucin deposition in the dermis (Alcian Blue $\times 40$).

- c) Eruptive collagenoma;
- d) Granuloma annulare.

Discussion

The diagnosis of lichen myxedematosus was confirmed through clinical-pathological correlation. The patient was instructed to maintain antiretroviral therapy and to start treatment for hepatitis C.

Lichen myxedematosus (LM) is a rare, chronic subtype of mucinosis that clinically manifests as papules, nodules, or plaques restricted to the skin. It is characterized by fibroblast proliferation, with varying degrees of fibrosis, and mucin deposition in the dermis, in the absence of thyroid disease.¹⁻³ Its etiopathogenesis is unknown; however, it is known that a variety of clinical conditions have been associated with LM, such as HIV infection, HCV, and exposure to chemicals such as L-tryptophan.²⁻⁷

The current classification of mucinoses was proposed by Rongioletti et al. and divides the papular mucinoses into scleromyxedema, a variant with systemic involvement and associated with paraproteinemia, and localized papular LM. Localized LM is divided into 5 subtypes: discrete papular mucinosis, persistent acral papular mucinosis, self-healing cutaneous mucinosis, juvenile papular mucinosis, and nodular papular mucinosis.^{1,6} Atypical cases with the overlapping of subtypes and distinct characteristics may occur.^{1,6} The patient in the present case can be classified as having localized papular LM of the mild papular mucinosis subtype.

Diagnostic criteria include papular rash, mucin deposition, and variable degree of fibroblast proliferation on histopathological examination, as well as the absence of gammopathy, thyroid disease, or systemic involvement.⁴ Histopathology shows mucin deposition, predominantly in the middle and upper dermis.³ The differential diagnosis of LM includes granuloma annulare, lichen amyloidosis, lichenoid eruptions, lichen planus, and eruptive collagenoma.⁴

There are no well-defined treatments reported in the literature, and the recommended approach is clinical observation alone.^{3,4} In general, the prognosis is good, even without specific treatment, and in rare cases, spontaneous

resolution may occur.^{3,8} To date, there is no description of the evolution of localized conditions to scleromyxedema. Topical corticosteroids and calcineurin inhibitors are used to relieve symptoms.³

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Authors' contributions

Nathalia Hoffmann Guarda: Design and planning of the study; drafting and editing of the manuscript; collection, analysis, and interpretation of data; effective participation in research orientation; intellectual participation in the propaedeutic and/or therapeutic conduct of the studied cases; critical review of the literature; critical review of the manuscript.

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Renata Heck: Approval of the final version of the manuscript; design and planning of the study; effective participation in research orientation; intellectual participation in the propaedeutic and/or therapeutic conduct of the studied cases; critical review of the literature; critical review of the manuscript.


Conflicts of interest

None declared.

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Papulopustular infantile acne treated with oral isotretinoin[☆]



Dear Editor,

Infantile acne is considered when it occurs between one and 16 months of age.¹ Topical retinoids, benzoyl peroxide at low concentrations, and oral antibiotics (except tetracyclines) are used in the treatment of children.²

This report describes the case of a two-month-old boy who presented papules, pustules, and a cyst on the malar region, bilaterally, as well as closed and open comedones, compatible with the diagnosis of infantile acne (Fig. 1). The laboratory hormonal evaluation of the child and mother (who also had severe acne) was normal. Initially, oral erythromycin was used for two months, oral cephadroxyl for another two months, as well as the fixed combination of adapalene and benzoyl peroxide associated with non-comedogenic emollients.

Despite the prolonged use of oral antibiotics and topical medications, progression of lesions and scar formation occurred. At seven months of age, oral isotretinoin was started at a dose of 0.5 mg/kg/day (target dose 960–1200 mg). The 10 mg capsule was frozen and half of the tablet was administered to the child in the milk.¹

After reaching the 150 mg/kg dose nine months later and with gradual adjustment according to weight gain (up to ¾ of the tablet), there was no disease activity (Fig. 2) throughout a 12-month follow-up. During treatment, the patient had mild cheilitis and xerosis, without laboratory alterations. As post-isotretinoin maintenance therapy, the fixed combina-

tion of adapalene and benzoyl peroxide was prescribed, as well as non-comedogenic emollients.

The androgenic hormonal laboratory investigation is mandatory in cases of refractory infantile acne, although most cases are not related to underlying endocrine diseases.^{1,3}

Oral isotretinoin, as well as topical therapy, are off-label treatments at this age; however, the many recently published cases demonstrate not only important clinical improvement in refractory cases but also their safe use in infants.^{3,4}

Acitretin is used in recessive congenital ichthyosis throughout life, since birth, being the confirmation test of retinoid safety in childhood. Early closure of epiphyses in children treated with oral retinoids is a rare event, associated with previous diseases, use of high doses, or prolonged treatment.² In the meantime, oral isotretinoin, when prescribed for refractory infantile acne, is a short-term treatment that requires low doses.⁴

The oral isotretinoin dose for infantile acne varies among publications between 0.2 and 2.0 mg/kg/day, with a total treatment period of five up to 14 months.¹ According to the latest acne consensus, the cumulative dose of isotretinoin should be the one in which complete clearing of lesions is attained, with drug maintenance for two more months, in contrast to the strict recommendation of reaching 120–150 mg/kg in all patients.⁵

Delay in the diagnosis of infantile acne is mainly due to the rarity of the disease at this age, as well as undertreatment and delay in the introduction of oral isotretinoin in these children.¹ It is therefore important that infants with severe, chronic acne, refractory to conventional treatment, be evaluated for underlying endocrinological disorders, not delaying drug use when there is resistance to oral antibiotics as well as the formation of scars.

[☆] Study conducted at the Hospital de Doenças Tropicais Dr. Anuar Aua, Goiânia, GO, Brazil.