Erythema Elevatum Diutinum, Vasculitis, and Fungoid Mycosis.[1,3]

A histopathological skin exam is essential to establish the proper diagnosis. This exam commonly shows interstitial inflammatory infiltrate, comprised of epithelioid histiocytes that at times appear in palisading form, with areas of degeneration of the collagen, with almost no mucinous material. In addition, it is common to see neutrophils and eosinophils in the infiltrate, which can also contain multinucleated and even atypical histiocytes.[1,3,5] When associated with medication, it may be histopathologically distinguishable through the presence of vacuolar interface dermatitis, exocytosis of lymphocytes, and the absence of neutrophils.[3]

The proposed treatments for IGDA are still not well-defined. Treatment can include the topical or systemic use of corticosteroids, non-steroid anti-inflammatory drugs, antimalarial drugs, cyclosporine, methotrexate, dapsone, cyclophosphamide, and anti-TNK alpha.[3,4,5] In cases in which the drug is the causal factor, this drug must be discontinued. When the diagnosis of the subjacent disease is proven, it should be treated, which can bring about a concomitant improvement in the skin. The cutaneous lesions present a spontaneous resolution, but they may also present some form of resistance to the treatment.[3,4]

In conclusion, the IGDA is a rare dermatosis that can be secondary to other diseases or to the use of certain drugs, and for this reason, dermatologists and rheumatologists should act together in their diagnoses and in their research on subjacent diseases in their initial stages. ❑

REFERENCES

1. Crowson AN, Magro C. Interstitial granulomatous dermatitis with arthritis. Hum Pathol. 2004;35:779-80.
2. Romite R, Valente NYS. Dermatite intersticial granulomatosa com artrite. Variante do granuloma anular? An Bras Dermatol. 1999;74:149-52.
3. Veronez IS, Dantas FL, Valente NY, Kakizaki P, Yasuda TH, Cunha Tdo A. Interstitial granulomatous dermatitis: rare cutaneous manifestation of rheumatoid arthritis. An Bras Dermatol. 2015;90:391-3.
4. Worsnop FS, Ostlere L. Interstitial granulomatous dermatitis with arthritis presenting with the rope sign. Clin Exp Dermatol. 2013;38:564-5.
5. Antunes J, Pacheco D, Travassos AR, Soares-Almeida LM, Filipe P, Sacramento-Marques M. Autoimmune thyroiditis presenting as interstitial granulomatous dermatitis. An Bras Dermatol. 2012;87:748-51.

Mailing address:
Juliana Lacerda Reis Ucelli
Rua Francisco Manuel, 126 - Benfica
20911-270 - Rio de Janeiro -RJ - Brazil
E-mail: julacerdareis@gmail.com

How to cite this article: *Ucelli JLR, Rudolph FSB, Obadia DL, Dionello CF. Interstitial granulomatous dermatitis with arthritis. An Bras Dermatol. 2017;92(3):434-6.*

Eczema *craquelé* associated with antiviral treatment for chronic hepatitis C*

Edla Polsinelli Bedin Mascarin do Vale[1]
Carlos Henrique Lima Rodrigues[1]
Fábio Esper Kallas[2]
Patricia da Silva Fucuta[1]

DOI: http://dx.doi.org/10.1590/abd1806-4841.20174464

Dear Editor,

Interferon-based therapy has many side effects, often leading to the premature cessation of therapy.[1,2] We report two patients who developed severe eczema craquelé during interferon-based therapy for a chronic hepatitis C virus (HCV) infection. *Case 1*. a 56-year-old female patient with HCV liver cirrhosis was submitted to antiviral treatment with pegylated interferon alfa-2a and ribavirin. The patient evolved to deep fissures and flaking skin along the trunk and lower limbs, with intense pain and bleeding. Therapy was discontinued at week 9, and she was treated with prednisone, sunflower oil enriched with vitamins, and intense skin hydration.

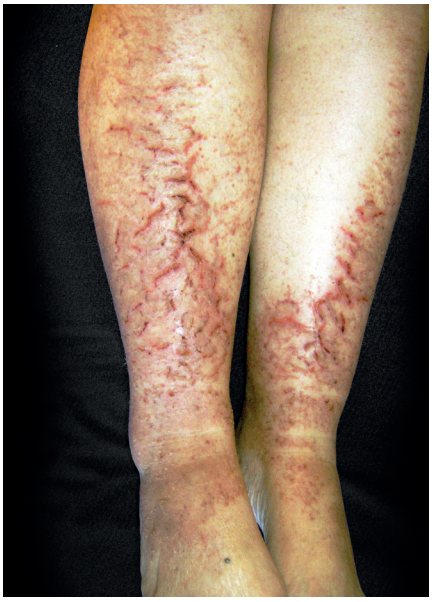

Figure 1: Patient 1 = lesions affecting the lower limbs at week 15 of antiviral therapy

Received on 15.02.2015
Approved by the Advisory Board and accepted for publication on 09.04.2015
* Work performed at Hospital de Base da Faculdade de Medicina de São José do Rio Preto (FAMERP) – São José do Rio Preto (SP), Brazil.
Financial support: None.
Conflict of interest: None.

[1] Gastroenterology and Hepatology Unit - Hospital de Base da Faculdade de Medicina de São José do Rio Preto (FAMERP) – São José do Rio Preto (SP), Brazil.
[2] Department of Clinical Surgery - Santa Casa de Misericórdia de Passos – Passos (MG), Brazil.

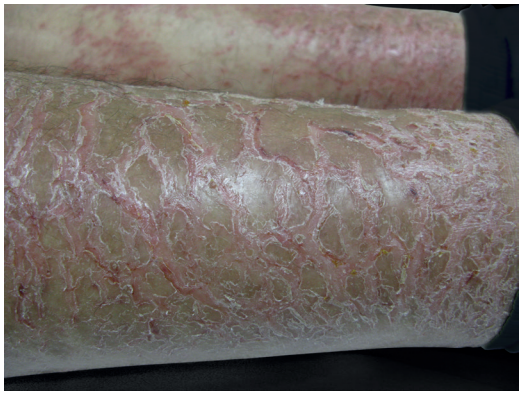

Figure 2: Patient 2 = detail of the right lower limb at week 14 of antiviral therapy with peginterferon and ribavirin

After 3 weeks, with partially improved lesions, peginterferon was reintroduced and it was tolerated until week 15, when it was permanently discontinued due to worsening skin lesions. The diagnosis was eczema craquelé (Figure 1). The skin lesions improved after discontinuation of antiviral therapy, but the HCV viral load relapsed. *Case 2*. a 49-year-old male patient with HCV liver cirrhosis was submitted to antiviral treatment with pegylated interferon alfa-2a and ribavirin. The patient evolved to scaly lesions on the lower limbs associated with itching and dry skin, which were managed with topical ketoconazole. The patient's clinical condition progressively worsened, and the lesions spread to the dorsal region of the thorax. He was diagnosed with eczema craquelé at week 14 of treatment (Figure 2). The skin lesions were treated with anionic emollient and sunflower oil, topical dexamethasone twice a day, and oral fexofenadine hydrochloride for itchiness as needed. Despite these comorbid symptoms, the patient chose to continue antiviral treatment, which was tolerated until week 24 and discontinued due decompensated cirrhosis. Eczema craquelé gradually improved after discontinuation of antiviral treatment. The present study has shown a clear association between the development of eczema craquelé and interferon-based therapy for HCV. Besides, severe lesions that did not respond to the standard management led to discontinuation of antiviral therapy in a patient with liver cirrhosis, resulting in recurrence of the virus. Adverse reactions lead to great complexity in treating chronic hepatitis C. Among these, dermatological reactions constitute a significant number of cases and can even contribute to the discontinuation of therapy.[3] Lesions such as hives, psoriasis, peeling eczema, alopecia, lichen planus, pigmented lesions, cutaneous pseudolymphoma, blisters, and skin necrosis have been widely described as adverse effects. Asteatotic eczema, or eczema craquelé is rarely described in the general literature. It appears as a characteristic extensive lesion that makes the patient uncomfortable and is potentially serious. The lesion resembles barnacles that cover the affected area. Extensive fissures and porcelain skin, mainly affecting the lower limbs, may also reach the hands and arms, which may be a sign of malignant processes in the internal organs. The lesions are caused by the loss of natural moisture of the stratum corneum associated with reduced lipids in the cells of this dermal layer. This condition can be associated with erythematous pruritic lesions, which are responsible for the itching and excoriations that arise later, even leading to bleeding.[4] Eczema craquelé commonly occurs in dry, cold climates, in patients with dry skin or who habitually take hot showers daily or use soaps and detergents without further skin hydration. Other predisposing factors are malnutrition (zinc deficiency), prolonged corticosteroid use, anti-androgen therapy, prolonged diuretic use, atopy, myxedema, and malignancy.[5] Treatment for eczema craquelé is based on suspending the causal factor, using emollients to hydrate the skin, reducing the use of soaps and detergents, and showering at room temperature. In more severe cases or in those unresponsive to this therapy, topical corticosteroids are applied on the lesions. Appropriate skin care and eczema craquelé early recognition are of fundamental importance during interferon-based therapy for chronic hepatitis C. ❑

REFERENCES

1. Chopra A, Klein PL, Drinnan T, Lee SS. How to optimize HCV therapy in genotype 1 patients: management of side-effects. Liver Int. 2013;33:30-4.
2. Fried MW, Shiffman ML, Reddy KR, Smith C, Marinos G, Gonçales FL Jr, et al. Peginterferon alfa-2a plus ribavirin for chronic hepatitis C virus infection. N Engl J Med. 2002;347:975-82.
3. Mistry N, Shapero J, Crawford RI. A review of adverse cutaneous drug reactions resulting from the use of interferon and ribavirin. Can J Gastroenterol. 2009;23:677-83.
4. Piérard GE, Quatresooz P. What do you mean by eczema craquele? Dermatology. 2007;215:3-4.
5. Sparsa A, Boulinguez S, Liozon E, Roux C, Peyrot I, Doffoel-Hantz V, et al. Predictive clinical features of eczema craquele associated with internal malignancy. Dermatology. 2007;215:28-35.

Mailing address:
Edla Polsinelli Bedin Mascarin do Vale
Av. Brigadeiro Faria Lima, 5544
15090-465 - São José do Rio Preto – SP
Brazil
E-mail: edlapbedin@gmaill.com

How to cite this article: *Vale EPBM, Rodrigues CHL, Kallas FE, Fucuta PS. Eczema craquelé associated with antiviral treatment for chronic hepatitis C.. An Bras Dermatol. 2017;92(3):436-7.*