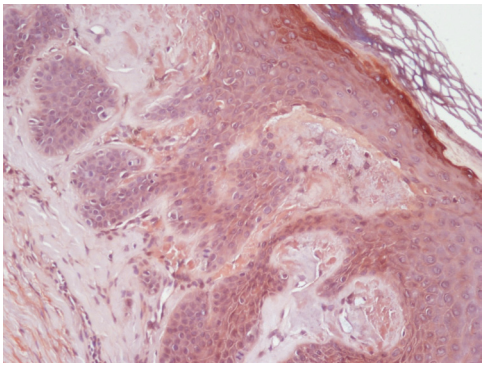

Figure 3: Original, magnified 200X – Congo red: Orange coloration. Absence of birefringence when submitted to polarized light

tion is in fact a new subtype of primary cutaneous amyloidosis or a variant of lichen amyloidosis.[1,5] The histopathological analysis consists of a nodular deposit of amorphous, eosinophilic, and homogenous material in the papillary dermis. The overlaying epidermis is atrophic and can partially encompass the amyloid material, in thin collarettes, and can present hyperkeratosis. There is some conflicting evidence in the literature regarding the nature of the deposits. The first case reports suggest a collagenous nature of the material, as they were stained with Verhoeff-van Gieson and Periodic acid–Schiff (PAS) and not with Congo red, and presented electronic microscopy consistent with collagen deposit, and the lesions were called collagen papules of the auricular concha.[4] However, the majority of the reported cases demonstrated that the material was stained with Crystal Violet and became orange-colored with Congo red, generally presenting positive birefringence when submitted to polarized light, thus suggesting the amyloid origin of such deposits.[1,2,3,4] According to the author of the largest case study on this issue, they could represent two distinct entities, though clinically similar, which could have been clarified if immunohistochemistry had been used in the first cases.[4] The material deposited in our patient presented a negative birefringence, which does not exclude the diagnosis of primary cutaneous amyloidosis, based on the clinical and histopathological findings. It is suggested that the amyloid material has an epidermal origin, since the immunohistochemical profile is positive for CK 34beta32, which corresponds to cytokeratins of high molecular weight, such as CK 1, 5, 10, and 14, which react with the squamous epithelium, including the epidermis.[4] There is no specific treatment capable of removing the amyloid deposits. As the papules of the auricular concha are mostly asymptomatic, localized, and superficial, then electrocoagulation, curettage, and excision seem to be sufficient for a good aesthetic result.[2,4]❑

REFERENCES

1. Shimauchi T, Shin JH, Tokura Y. Primary cutaneous amyloidosis of the auricular concha: Case report and review of published work. J Dermatol. 2006;33:128-31.
2. Neff AG, McCuin JB, Mutasim DF. Papular amyloidosis limited to the ears. J Am Acad Dermatol. 2010;62:1078-9.
3. Hicks BC, Weber PJ, Hashimoto K, Ito K, Koreman DM. Primary cutaneous amyloidosis of the auricular concha. J Am Acad Dermatol. 1988;18:19-25.
4. Wenson SF, Jessup CJ, Johnson MM, Cohen LM, Mahmoodi M. Primary cutaneous amyloidosis of the external ear: a clinicopathological and immunohistochemical study of 17 cases. J Cutan Pathol. 2012;39:263-9.
5. Bakos L, Weissbluth ML, Pires AK, Müller LF. Primary amyloidosis of the concha. J Am Acad Dermatol. 1989;20:524-5.

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Interstitial granulomatous dermatitis with arthritis*

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Dear Editor,

Interstitial granulomatous dermatitis is a rare, idiopathic disease with typical histopathological characteristics and with a variable clinical expression.[1] In 1993, Ackerman *et al*. proposed the term interstitial granulomatous dermatitis with arthritis (IGDA), to describe the association of the cutaneous cords with changes in the musculoskeletal system.[2,3] However, other cutaneous lesions have also been described, such as erythematous or hyperchromic papules, subcutaneous plaques, and lesions with annular shapes, which generally affect the side walls of the thorax, armpits, abdomen, and medial surface of the thighs.[3-5]

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A 47-year-old female patient presented annular asymptomatic lesions in her armpits over the past year. The lesions had increased in size over the past four months. Upon ectoscopic examination, the lesion presented a hyperchromic macula with clearly defined edges and erythematous-purple plaques, with infiltrated edges and an annular shape in her armpits (Figure 1). A direct mycological exam and culture to test for fungi were performed, both of which proved to be negative. The histopathological exam of the right armpit lesion presented perivascular and interstitial inflammatory infiltrate superficial and deep, comprised of histiocytes that in some areas are palisading and in other areas, such as the interstice, in addition to groups of neutrophils and eosinophils (Figure 2). After two months, the patient returned and reported the appearance of painful and erythematous nodules on the medial surface of the thighs for the past week. The patient also reported the appearance of polyarthralgia in the fingers, wrists, and knees bilaterally.

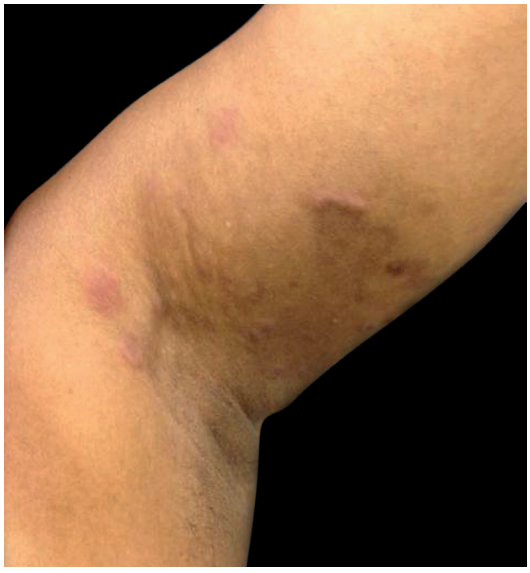

Figure 1: Left armpit lesions. Hyperchromic macula with defined edges and erythematous-violet plaques, with infiltrated edges and an annular shape

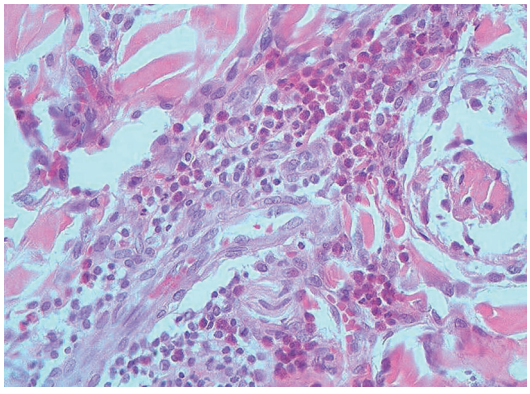

Figure 2: Histopathological exam under Hematoxylin & eosin x200(HE) staining. Details of the interstitial infiltrate, comprised of histiocytes, neutrophils, and eosinophils

The initial rheumatological evaluation did not show arthritis, despite the joint pain, and the musculoskeletal system exam presented no significant synovitis or functional impotence. Laboratory exams were requested to investigate autoimmune diseases, the results of which were normal; however, some inflammatory functional results were high (C-reactive proteins (CRP) and Erythrocyte Sedimentation Rate (ESR)). To combat the patient's rheumatology, Clobetasol Proprionate creme was prescribed for seven days, to be applied to the armpit lesions and Venlafaxine, 37.5mg/day, to combat rheumatology.

Due to persistent joint pain, changes in the inflammatory results, and a lack of response to Venlafaxine, magnetic resonance (MR) exams of the hands and wrists were requested. The radiologist's analysis of the exams, in which a Gadolinium exam was performed, identified synovitis by hypercaptation in the sequence considered in T1 in some joints, such as the wrists, and proximal metacarpophalangeal and interphalangeal joints (Figure 3). Prednisone and methotrexate were prescribed, with excellent clinical and laboratory response. Prednisone was later discontinued.

Systemic involvement in IGDA appears as migratory arthritis or polyarthritis of small and large joints, which can appear before, during, or after its cutaneous manifestation.[1,4,5] IGDA mainly affects middle-aged women, the rheumatoid factor may be positive or not, and it is associated with a wide range of diseases, such as systemic erythematous lupus, antiphospholipid antibody syndrome, auto-immune thyroiditis, autoimmune hepatitis, Churg-Strauss syndrome, Behcet's disease, pulmonary paracoccidioidomycosis, pulmonary silicosis, chronic uveitis, and paraneoplastic syndrome.[1-5] However, the most common association is with rheumatoid arthritis.[3]

There are reports that some drugs are capable of causing IGDA, such as the angiotensin-converting enzyme inhibitors, calcium canal blockers, beta-blockers, hypolipidemic drugs, and alpha-blockers.[3-5] In this patient's case, joint involvement was investigated, thus excluding, initially, the diagnosis of autoimmune disease and drugs as causal factors.

The differential diagnosis should be performed with Annular Granuloma, Sarcoidosis, Centrifuge Annular Erythema, Migratory Chronic Erythema of Lyme Disease, Multiform Erythema,

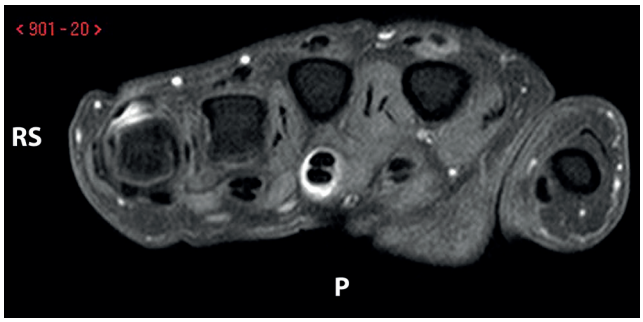

Figure 3: Nuclear magnetic resonance of the right hand. Liquid distension in the sheaths of the flexor tendons of the third finger, at the height of the middle phalanx, and in the tendinous sheath of the extensors of the second and fifth fingers, which is more accentuated at the level of the metacarpus, compatible with tenosynovitis.

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.

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

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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.

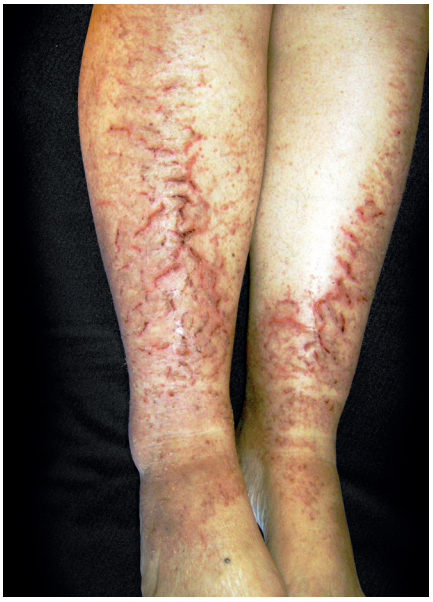

Figure 1: Patient 1 = lesions affecting the lower limbs at week 15 of antiviral therapy

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