The lesions tend to resolve spontaneously after a mean period of 11 months and do not leave a scar.[3] In general, antibiotic therapy is ineffective, although some cases have shown a good response to oral macrolides or topical metronidazole.[7]

We must include IFAG in the differential diagnosis of acquired facial nodules in children. The medical history and the clinical, microbiologic, and ultrasound findings enable us to make an early diagnosis and to avoid unnecessary aggressive procedures.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

References

1. Léaute-Labrèze C, Maleville J, Taïeb A. Dermatoses bactériennes. In: Saurat JH, Grosshans E, Laugier P, Lachapelle JM, editors. Dermatologie et maladies sexuellement transmissibles. 3rd ed. París: Masson; 1999. p. 114–21.
2. Roul S, Léaute-Labrèze C, Boralevi F, Bioulac-Sage P, Maleville J. Idiopathic aseptic facial granuloma (pyodermite froide du visage): A pediatric entity. Arch Dermatol. 2001;137:1253–5.
3. Boralevi F, Léaute-Labrèze C, Lepreux S, Barbarot S, Mazereeuw-Hautier, Eschard C, et al. Idiopathic facial aseptic granuloma: A multicentre prospective study of 30 cases. Br J Dermatol. 2007;156:705–8.
4. Satta R, Montesu MA, Biondi G, Lissia A. Idiopathic facial aseptic granuloma: Case report and literature review. Int J Dermatol. 2016. Epub ahead of print.
5. Zitelli KB, Sheil AT, Fleck R, Schwentker A, Lucky AW. Idiopathic facial aseptic granuloma: Review of an evolving clinical entity. Pediatr Dermatol. 2015;32:e136–9.
6. Prey S, Ezzedine K, Mazereeuw-Hautier J, Eschard C, Barbarot S, Boralevi F, et al. IFAG and childhood rosacea: A possible link. Pediatr Dermatol. 2013;30:429–32.
7. Neri I, Raone B, Dondi A, Misciali C, Patrizi A. Should idiopathic facial aseptic granuloma be considered granulomatous rosacea. Report of three pediatric cases. Pediatr Dermatol. 2013;30:109–11.
8. González-Rodríguez AJ, Jordá-Cuevas E. Idiopathic facial aseptic granuloma. Clin Exp Dermatol. 2015;40:298–300.
9. García-Martínez FJ, Muñoz-Garza FZ, Hernández-Martín A. Ecografía en dermatología pediátrica. Actas Dermosifiliogr. 2015;106:76–86.
10. Hiraldo-Gamero A, Vera-Casaño A, Sanz-Trélles A. Idiopathic facial aseptic granuloma. Actas Dermosifiliogr. 2013;104:635–6.

I. Vázquez-Osorio,* C.C. Álvarez-Cuesta, L. Rodríguez-González, E. Rodríguez-Díaz

Servicio de Dermatología, Hospital Universitario de Cabueñes, Gijón (Asturias), España

* Corresponding author.
E-mail address: rogivaos@gmail.com (I. Vázquez-Osorio).

1578-2190/

Lymphangioma-like Kaposi sarcoma☆

Sarcoma de Kaposi de tipo linfangiomatoso

To the Editor:

Lymphangioma-like Kaposi sarcoma (LLKS) is a rare histologic variant of Kaposi sarcoma that can present as any of the 4 known clinical variants. LLKS is a vascular neoplasm that develops secondary to infection by human herpesvirus type 8 (HHV-8), which is also known as the Kaposi sarcoma virus. Clinically, it can present with the usual manifestations, namely, patches, plaques, or nodules. However, in some cases, it presents as blisters that may be confused with bullous skin disease.

Case Description

The patient was an 80-year-old man whose history was unremarkable. He presented with raised erythematous, oval plaques measuring 1-3 cm in diameter that had first appeared 5 years previously. The plaques occasionally coalesced and were found on the upper and lower limbs and lower back. The lesions had gradually increased in number and size, although they were neither painful nor pruriginous. The physical examination revealed flaccid blisters (1 cm) containing serum (Fig. 1). Treatment with various topical options had been unsuccessful.

A complete laboratory workup including complete blood count and biochemistry revealed iron-deficiency anemia. Serology testing for HIV was negative.

Histopathology revealed that the epidermis was conserved and highlighted a proliferation of anastomosed vascular spaces that occupied the complete thickness of the dermis, dissected the collagen bundles, and surrounded cutaneous muscles and adnexa. No blood was identified in these structures. Clusters of lymphocytes and plasma cells were common in the stroma. Closer examination revealed that the vascular channels were lined with a layer of flattened endothelial cells and that there was no atypia or mitosis. Immunohistochemistry showed that tumor proliferation was positive for the endothelial markers CD31 and CD34 and for the lymphatic marker D2-40. It also showed clear nuclear staining for latent nuclear antigen 1 of HHV-8 (Fig. 2).

The patient was diagnosed with LLKS and referred to the medical oncology department. Given the poorly aggressive clinical course and the patient's age, it was decided—after

☆ Please cite this article as: Martínez-Ortiz F, Avivar MG. Sarcoma de Kaposi de tipo linfangiomatoso. Actas Dermosifiliogr. 2017;108:268–271.

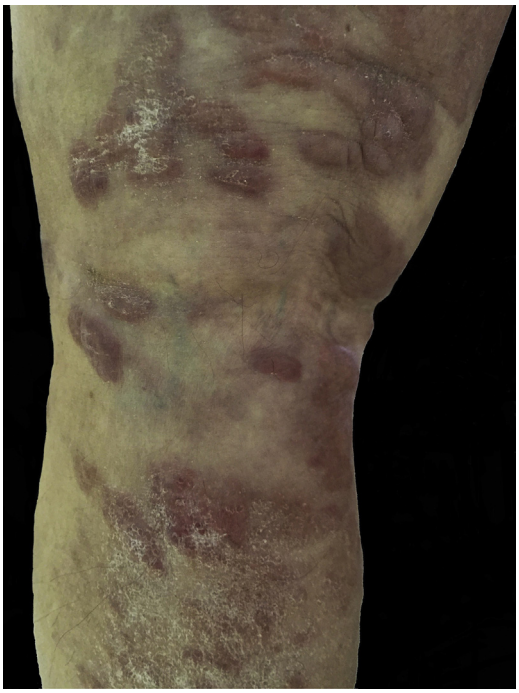

Figure 1 Coalescent erythematous-squamous plaques and blisters measuring 1 to 3 cm on the lower limbs.

evaluation and with the patient's agreement—to adopt a wait-and-see approach.

Discussion

LLKS is a malignant vascular neoplasm with evidence of lymph and blood vessel differentiation. It is a rare histologic variant of Kaposi sarcoma (5% of cases)[1] and can present in any of the 4 existing epidemiologic variants. Etiology and pathogenesis are controversial, as is the question of whether the disease should be classified as reactive or neoplastic.[2] The discovery of HHV-8, which is present in 100% of cases of Kaposi sarcoma, irrespective of the subtype in question, is a major finding in our knowledge of the etiology and pathogenesis of the condition.[3] Similarly, the question of cell differentiation is controversial, since cells express both specific blood and lymph markers.[4,5]

Clinical Manifestations

The most common presentation of LLKS is blisters,[6] although it can also appear in the more classic form of plaques or nodules. LLKS can also present as a mucocutaneous lesion, affecting mainly the legs and arms. The condition is more

[b]

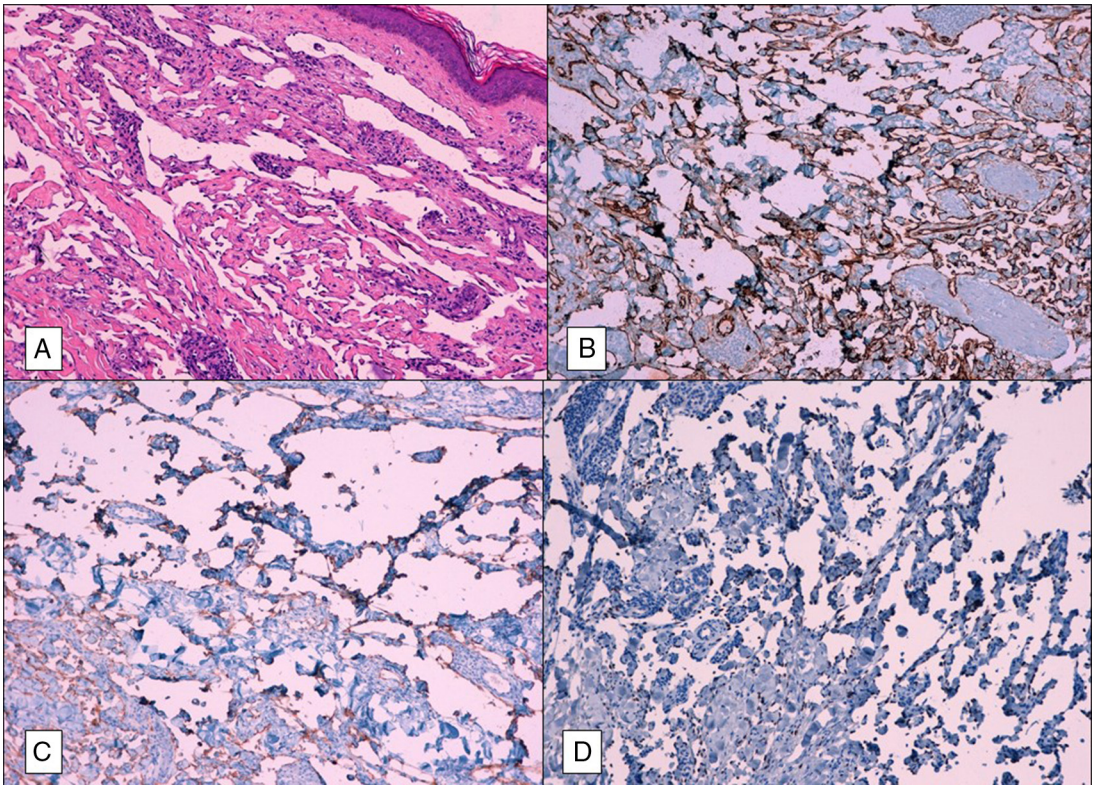

Figure 2 A, Anastomosed, small-caliber vascular channels with no content dissecting collagen bundles (hematoxylin-eosin, original magnification, ×10). B, Expression of CD34 (×10). C, Expression of D2-40 (×10). D, Expression of HHV-8 (×10).