Correspondence

Scalp necrosis as a manifestation of temporal arteritis

An 85-year-old man with a history of hypertension and glaucoma was referred to us with a 2-month history of pain and skin necrosis on both temporoparietal regions (Fig. 1), which had been diagnosed as herpes zoster. The patient reported occasional fever below 38 °C, asthenia, jaw claudication, pain on swallowing, weakness of scapular and pelvic waists, an unspecified weight loss, and general malaise. The physical examination showed extensive bilateral necrosis with serpiginous borders and a dirty and sphacelus appearance. The necrotic area was spontaneously highly painful and even more painful on palpation. The ophthalmologic examination was normal. The analytical study showed an elevated erythrocyte sedimentation rate (ESR) (70 mm in 1°h) and normochromic normocytic anemia.

Temporal arteritis was suspected and a biopsy of the temporal artery confirmed the diagnosis (Fig. 2).

Treatment was started with boluses of 6-methylprednisolone (500 mg × 3) and continued with oral steroids and oral methotrexate. The evolution was favorable. The areas of necrosis were debrided and were treated daily with antimicrobial cream to achieve granulation tissue and enable the area to be covered with skin grafts. The patient died soon after from an acute myocardial infarction.

Temporal arteritis, giant cell arteritis, or Horton's arteritis is characterized by a multisystem granulomatous vasculitis of the medium and large vessels, preferentially involving the external carotid area and, most frequently, the temporal artery. It is a rare disease, which appears in approximately 25 of every 100,000 individuals. It almost exclusively affects individuals aged over 55 years, females, and the white population, although cases have been reported in young people.[1]

It clinically manifests as cephalea, associated or not with a temporal artery of thickened, nodular appearance that is pulsatile and painful to palpation. It is usually accompanied by fever, general malaise, fatigue, anorexia, weight loss, jaw and tongue claudication, and musculoskeletal and ophthalmologic symptoms. It can also be associated with polymyalgia rheumatica syndrome.[2] Cases of myocardial infarction, aorta dissection, strokes, and other infarctions of visceral organs have been reported in these patients.

The diagnosis is simple in an elderly patient with the classic complex of fever, cephalea, and anemia with an increased ESR, and is confirmed by a biopsy of the temporal artery.[3] Recent studies have proposed magnetic resonance angiography as a noninvasive method to aid in the clinical diagnosis and to select the optimal site for taking the biopsy, given that the involvement of the artery is not uniform.

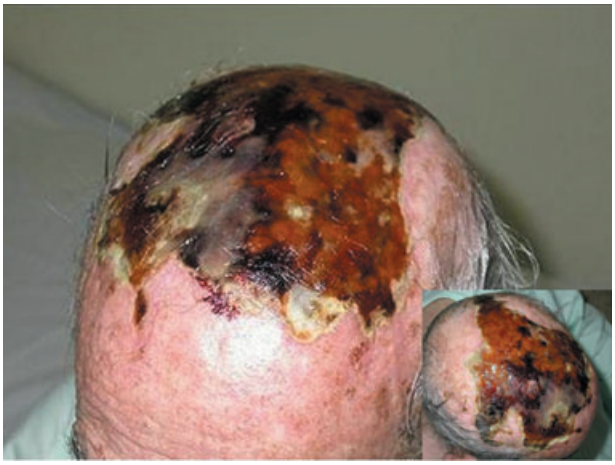

Figure 1 Skin necrosis in both temporoparietal regions. The necrotic area shows serpiginous borders and a dirty and sphacelus appearance

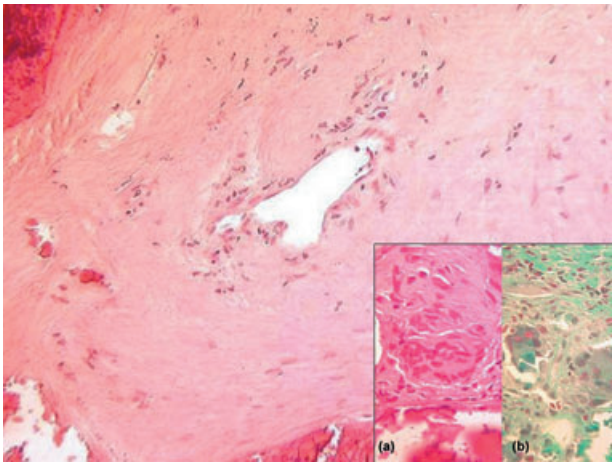

Figure 2 Histopathologic findings in temporal arteritis (×10). Reduction of the vascular lumen and dystrophic foci. (a) Hematoxylin and eosin (×40). (b) Trichromic (×40). Foci of calcification and presence of multinucleated giant cells

Pathology reveals a granulomatous arteritis that involves the inner layer with abundant multinucleated giant cells. It is associated with a proliferation of fibroblasts and muscle cells of the intima, with small changes in the middle layer,

fragmentation of the elastic lamina, and an inflammatory infiltrate with a variable amount of lymphocytes, histiocytes, and even eosinophils. In individuals of advanced age, it may be associated with signs of arteriosclerosis.

Skin manifestations are uncommon. They consist of ischemia and necrosis, resulting from a total or partial reduction in the blood supply to the territory irrigated by the affected artery. In milder cases, there can be alopecia, hyperpigmentation, and sensitivity on the skull.[4] Cases with an initial diagnosis of herpes zoster have been reported.[5]

The first two cases of scalp necrosis as a manifestation of temporal arteritis were described by Cooke *et al*. in the 1940s. Since then, only 26 cases have been reported in the literature.[6] Patients with scalp necrosis are older, with a greater risk of visual alterations and gangrene of the tongue,[5] and a worse prognosis.[7–9]

This is a severe disease, especially because of the complications; however, most cases are now reversible and the mortality rate is low.[4,10]

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Significant improvement in ulcerative necrobiosis lipoidica with hydroxychloroquine

Necrobiosis lipoidica (NL) is an inflammatory granulomatous disease of the skin, coexisting with diabetes in about 0.3% of cases.[1] Typical lesions of NL occur on pretibial skin as ovoid plaques with a violaceous border and a yellow central atrophic area with telangiectasia. Ulceration is a major complication of NL and may result in local dysesthesia, pain, infection, or cosmetic impairment. In addition, rare reports indicate an increased incidence of squamous cell carcinoma arising in previous ulcerative NL lesions.[2] The treatment of ulcerative NL is difficult. We present a patient with type-II diabetes mellitus and treatment-resistant chronic ulcerative NL who was treated with hydroxychloroquine.

A 62-year-old woman with type-II diabetes mellitus was referred with a 3-year history of NL on both shins. She was treated with systemic antimicrobials and topical steroids with no improvement. She had bilateral, well-defined, atrophic, yellowish-pink plaques with telangiectases on the anterior and lateral surfaces of both lower legs with no neuropathic sensory symptoms. Several variably sized ulcers with sloughy, cribriform bases and ragged margins were present (Fig. 1). Hematology and biochemistry profiles were normal, except for the presence of long-standing hyperglycemia with a blood glucose level of 176 mg/dL. An autoantibody screen, serum immunoglobulins, and chest x-ray were also normal. The histology of a skin biopsy from one of the ulcers showed ulceration on the surface, a dermal inflammatory infiltrate with foreign body giant cells, and areas of necrobiosis of collagen; a perivascular lymphocytic infiltrate, plasma cells, thickening of blood vessel walls with proliferation of endothelial cells, and thickened fibrous septa were seen in the deep dermis and subcutis (Fig. 2).

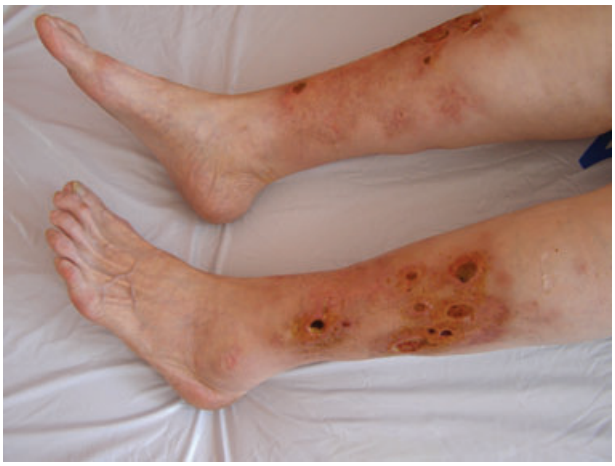

Figure 1 Ulceration of right and left leg before treatment with hydroxychloroquine