To the Editor:

Giant cell arteritis or temporal arteritis (TA) is the most common systemic vasculitis in adults.¹ Histologically there is a lymphocytic-monocytic panarteritis with the formation of granulomas and giant cells. There is patchy involvement of medium and large arteries, particularly the extracranial branches of the carotid artery and, more specifically, the temporal artery.

The clinical presentation of TA is variable, with multiple, nonspecific symptoms in elderly patients, particularly women, who initially present fever, asthenia, weight loss, headache, unpredictable joint and muscle pain, stiffness, and polymyalgia rheumatica.¹⁻⁷ Up to a third of patients develop visual symptoms. Loss of vision, sometimes bilateral and irreversible, is the main complication of this condition and it can sometimes be the first symptom. The alterations in the blood tests in TA can also be relatively nonspecific, with normocytic-normochromic anemia, elevated alkaline phosphatase, and a raised erythrocyte sedimentation rate (ESR).¹⁻⁷ Up to 40% of patients develop atypical clinical manifestations such as respiratory symptoms, pyrexia of unknown origin, aortic aneurysms, or digestive tract, neurological, or skin disorders.¹

We present the case of a 91-year-old woman with a past history of systemic hypertension, congestive cardiac failure, and atrioventricular block for which a pacemaker had been implanted. She came to the emergency department with a 48-hour history of bilateral loss of vision, with no accompanying symptoms.

On physical examination there was asymmetric, unreactive, bilateral mydriasis with changes suggestive of ischemic optic neuritis on funduscopy.

The blood test performed in the emergency department revealed a normocytic anemia (hemoglobin, 10.7 g/dL) and an ESR of 23 mm/h. Cerebral computed tomography showed no relevant abnormalities. On a suspicion of TA, biopsy of the temporal artery was performed and treatment was started with intravenous methylprednisolone at a dose of 250 mg every 6 hours.

Histological study of the artery showed a thickened wall with a moderate chronic inflammatory infiltrate in the media and adventitia of the vessel, composed of lymphocytes, histiocytes, occasional eosinophils, and occasional images suggestive of giant cells (Figure 1).

Two days after starting the treatment, the patient presented intense pain in the tongue, leading to difficulty moving the tongue, swallowing, and speaking. On examination, there was a large, well-defined, deep, excavated ulcer with a clean base and that was not infiltrated on palpation (Figure 2); it was very painful.

On a repeat blood test, the ESR had risen to 88 mm/h, with no other abnormalities.

Biopsy of the tongue lesion showed a deep ulcer that reached the skeletal muscle tissue, with fibrosis and images of myocyte necrosis, suggestive of ischemia (Figure 3).
severity, and it occurs in a subgroup of older patients with a higher incidence of loss of vision and a mortality of up to 40%. Still less common is involvement of the tongue, in the form of pain, stiffness, ulceration, or extensive necrosis.6,7

When the diagnosis is suspected, TA must be treated rapidly to avoid irreversible complications, particularly complete loss of vision. TA usually responds well to high doses of corticosteroids. The ischemic disorders, including those of the skin, can appear at any time in the course of the disease, particularly during the tapering of steroid treatment; these patients must therefore remain on treatment for long periods, sometimes for years or even for life.

TA must be included in the differential diagnosis of tongue disorders in elderly patients with heterogeneous clinical presentations with multiple, nonspecific symptoms with no other apparent cause.

Correspondencia:
Susana Córdoba Guijarro
Servicio de Dermatología
Hospital de Fuenlabrada
C/ Camino del Molino, 2
28942 Fuenlabrada, Madrid, España,
scordoba.hlfr@salud.madrid.org

Conflicts of interest
The authors declare no conflict of interest

References