



Letter to the Editor

Progression of an untreated case of giant cell arteritis

Evolución de un caso de arteritis de células gigantes sin tratamiento

To the Editor:

Giant cell arteritis (GCA) affects blood vessels of medium and large calibre, with a preference for extracranial aortic arteries. It generally appears in persons older than 50 years.

The patient was a 77-year-old male who had quit smoking 16 years previously and suffered arterial hypertension and benign prostatic hyperplasia. Three years earlier, he had been explored due to headaches, and a cranial CT scan was carried out, as well as a biopsy of the temporal artery. He was diagnosed with grade I secretory meningioma and GCA (Figure a). A surgical intervention was performed for the meningioma and he underwent treatment with dexamethasone 3 mg/24 h for 11 days. Apart from treatment with dexamethasone for 11 days, the GCA was not treated and no controls were carried out. The patient remained asymptomatic. He continued treatment with enalapril 5 mg/24 h, lysine carbocysteine 2.7 g/24 h, and tamsulosin 0.4 mg/24 h.

The patient attended consultation due to tiredness and weight loss of 15–20 kg with one and a half months of evolution. The physical exploration was normal.

Leukocytes in the blood count were $1.50 \times 10(9)/l$, neutrophils 27.9%, and haemoglobin 12.7 g/dl. Biochemistry was normal. C-reactive protein was 15.3 mg/dL; rheumatoid factor was negative, TSH, CEA, and PSA were normal; serology for hepatitis B and C virus was negative. The thoracic-abdominal CT scan was normal. Colonoscopy was normal. The electromyogram showed signs of predominantly

chronic bilateral L5-S1 radicular involvement. Temporal artery biopsy (Figure b) showed an arterial wall without inflammatory changes, compatible with arteritis in the healing phase. The bone marrow biopsy was diagnostic of acute myeloid leukaemia. With all this information, the patient was diagnosed with acute myeloid leukaemia and GCA in remission.

The arteritis showed a good response to corticoid treatment. A dosage of 40–60 mg/24 h of prednisone or equivalent was started and progressively lowered, depending on the degree of control of the disease. The duration of this treatment is usually 1 or 2 years. This patient followed corticoid treatment for only 11 days as an anti-oedema measure after brain tumour surgery had been performed. According to some authors, corticoid treatment improves symptoms but it does not alter the course of the disease.¹ In this case, the patient was asymptomatic from the diagnosis of GCA until the onset of symptoms due to haematological disease, so it could have either been a silent form of GCA² or it could have modified its course from treatment with dexamethasone.

The healing of lesions could be observed in the second biopsy, which was carried out 3 years after the diagnosis of GCA and which also showed healing of the disease. There are cases of healing without treatment described in medical literature³; in a series of autopsied patients, it was observed that 1.7% of cases showed histological lesions of GCA without activity and that 25% of patients had not been diagnosed with GCA during their lifetime,⁴ suggesting that in some cases it may not be noticed due to its relatively few symptoms.⁵

Mortality rates with GCA are low, probably because early diagnosis and treatment take place in most cases.⁶ The life expectancy of patients with GCA does not significantly differ from the general population.^{6–8}

An increase in the incidence rate of neoplastic processes has been observed in some series of patients with GCA. However, it is unlikely

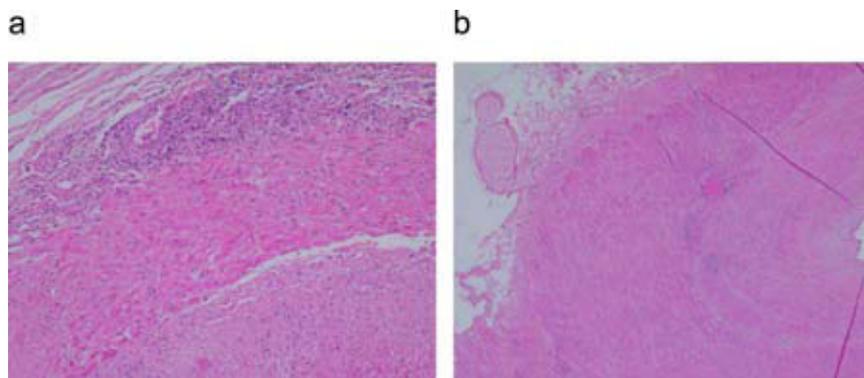


Figure. a) Acute and chronic inflammation with presence of giant cells and fragmentation of internal elastic tissue can be observed. b) Fragment of arterial wall with absence of inflammation, along with fibrosis and neovascularization of the middle layer.

that GCA is a paraneoplastic sign, based on the long interval between the diagnosis of GCA and the onset of neoplastic disease.⁹

References

1. Latorre J, Romero JM, Sánchez I, Surcel P, Davins M, Viver E. Arteritis de células gigantes. *An Cir Card Cir Vasc.* 2005;11:188-200.
2. Gonzalez-Gay MA, Garcia-Porrúa C, Amor-Dorado JC, Llorca J. Giant cell arteritis without clinically evident vascular involvement in a defined population. *Arthritis Rheum.* 2004;51:274-7.
3. Hernández-Rodríguez J, García-Martínez A, Espígol-Frigolé G, Grau JM, Collado A, Cid MC. Sustained spontaneous clinical remission in giant cell arteritis: report of two cases with long-term followup. *Arthritis Rheum.* 2006;55:160-2.
4. Gonzalez-Gay M. Giant cell arteritis and polymyalgia rheumatica: two different but often overlapping conditions. *Semin Arthritis Rheum.* 2004;33:289-93.
5. Gonzalez-Gay MA, Garcia-Porrúa C, Amor-Dorado JC, Llorca J. Giant cell arteritis without clinically evident vascular involvement in a defined population. *Arthritis Rheum.* 2004;51:274-7.
6. González-Gay MA, Blanco R, Abraira V. Giant cell arteritis in Lugo, Spain, is associated with low longterm mortality. *J Rheumatol.* 1997;24:2171-6.
7. Matteson EL, Gold KN, Bloch DA, Hunder GG. Long-term survival of patients with giant cell arteritis in the American College of Rheumatology giant cell arteritis classification criteria cohort. *Am J Med.* 1996;100:193-6.
8. Nordborg E, Bengtsson BA. Death rates and causes of death in 284 consecutive patients with giant cell arteritis confirmed by biopsy. *BMJ.* 1989;229:549-50.
9. Gonzalez-Gay MA, Lopez-Diaz MJ, Martinez-Lado L, Peña-Sagredo JL, Lopez-Agreda H, Miranda-Fillooy JA, et al. Cancer in biopsy-proven giant cell arteritis. A population-based study. *Semin Arthritis Rheum.* 2007;37:156-63.

Francisco José Nicolás-Sánchez,* Pilar Gallel-Vicente,
José María Peña-Porta, and Rosa María Sarrat-Nuevo

Servicio de Medicina Interna, Hospital de Santa María, Lleida, Spain

*Corresponding author.

E-mail address: fnicolas@comll.cat (F.J. Nicolás-Sánchez).