# Giant cell arteritis with normal inflammatory markers

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## Introduction

Giant cell arteritis (GCA) is a granulomatous medium to large vessel vasculitis. It typically occurs in older people and is associated with polymyalgia rheumatica (PMR).[1] The condition usually manifests with involvement of the extracranial branches of the carotid artery. This results in the classical symptoms of a headache, temporal tenderness, jaw claudication and associated constitutional symptoms (such as fever, lethargy and malaise). The most serious manifestation is permeant visual loss that occurs due to optic nerve ischaemia.[2]

In the UK, the incidence of GCA is approximately 2.2 per 10,000 patient-years.[3] A full-time general practitioner is likely to see a new case every 1–2 years.[4]

## Case presentation

An 86-year-old man presented to hospital with sudden onset visual loss in his right eye. His past medical history included chronic lymphocytic leukaemia, benign prostate hypertrophy and macular degeneration. Initial blood tests revealed a C-reactive protein (CRP) that was less than 5 mg/L, an erythrocyte sedimentation rate (ESR) of 30 mm/hour and a platelet count of $260 \times 10^9$/L.

He did not have any of the typical features GCA, eg headache, scalp tenderness, jaw claudication or constitutional symptoms. There was no associated PMR symptoms. An ophthalmology review was sought. Ophthalmological assessment revealed right eye anterior ischaemic optic neuropathy. This was followed by a temporal artery ultrasound showing widespread bilateral halo sign consistent with inflammation. This raised a strong suspicion of GCA, and the patient was subsequently treated with 3 days of intravenous methylprednisolone followed by prednisolone. This was given to protect the left eye from optic nerve ischaemia.

Temporal artery biopsy performed at a later stage showed adventitial chronic inflammation of uncertain significance.

The combination of ischaemic neuropathy and halo sign on ultrasound sufficed for a diagnosis of GCA to be made.[5]

## Discussion

The inflammatory nature of GCA typically results in raised inflammatory markers. Therefore, the American College of Rheumatology include an ESR of >50 mm/hr as one of its five classification criteria.[6] CRP is a more sensitive marker than ESR for a positive temporal artery biopsy, which is diagnostic of GCA. In clinical practice, both tests are performed to evaluate for GCA.[7]

This case is unusual as, at the time of presentation, the inflammatory markers were low to normal. Furthermore, the patient did not exhibit the classical symptoms of headache, scalp tenderness, jaw claudication or PMR-related symptoms. Rather, the patient presented directly with visual loss.

Notable learning points drawn from this case are that normal inflammatory markers should not exclude the diagnosis of GCA. If suspicion remains high, inflammatory markers should be repeated. It should also be noted that GCA may not present with the typical symptoms of headache, temporal tenderness or jaw claudication; instead, it may present directly with visual symptoms.

Early treatment with steroids is essential to prevent progression of visual symptoms and to protect the contralateral eye.[8] ■

## References

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