

Case notes

Giant cell arteritis

Patient details

Initials: AR

Age: 79 years

Gender: Male

Lifestyle: Retired, driver, non-smoker

Hobbies: Family, walking

Reason for visit: Vague symptoms of possible 10-minute loss of vision LE three times over the previous fortnight. Now has headache over left temple and poor VA LE at distance and near for 2 days.

Refraction: RE: -0.25/-0.25x75

LE: 0.00/-0.50x60

Distance Acuity: RE: 6/7.5, LE 6/36

Reading add: RE & LE: +2.25D

General health: High blood pressure and high cholesterol

Medication: Ramipril, amlodipine, simvastatin, aspirin

Ocular history: Bilateral pseudophakia 2022, uncomplicated surgery

Family ocular history: Nil

Examination

Slit lamp & undilated Volk 90D

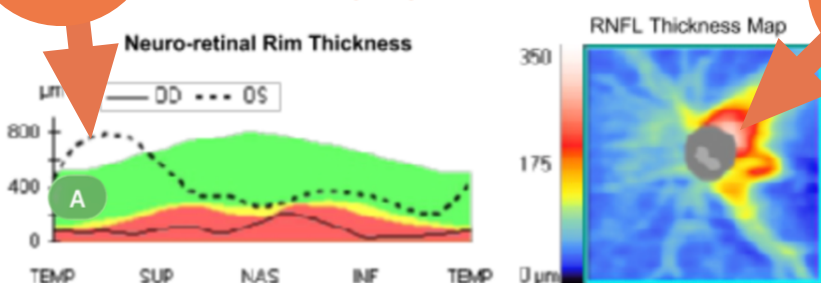
Right eye	Structure	Left eye
Dermatochalasis	Lids & lashes	Dermatochalasis
Clear	Cornea	Clear
G1 injection	Conjunctiva	G1 injection
Quiet	Anterior chamber	Quiet
IOL	Lens	IOL
Nil PCO		Nil PCO
C:D 0.4	Disc	C:D 0.10
Healthy rim		Disc swelling superiorly
Few drusen, ERM	Macula	Few drusen, ERM
No holes/tears/detachment	Periphery	No holes/tears/detachment
Normal	Pupil reactions	Left RAPD

(IOL = intra-ocular lens; PCO = posterior capsular opacification; ERM = epi-retinal membrane; G = Grade)

Right eye	NCT	Left eye
11mmHg	@15:00	12mmHg
(mean of 3 readings)		

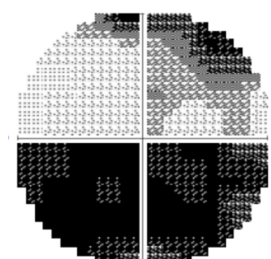
Disc swelling

OCT LE



Disc swelling

Field LE



Diagnosis

AR presented to his optometrist with a history of several episodes of possible *amaurosis fugax* - temporary obscuration of vision - in his left eye. He then started to suffer with left-sided temporal headache and jaw pain and noticed reduced vision on the same side.

On examination, the supero-temporal aspect of the left optic disc was swollen (evident on the OCT neuro-retinal rim thickness and heat maps) and there was a visual field defect (inferior > superior). Visual acuity was reduced to 6/36 and there was a left-sided relative afferent pupil defect.

This pattern of symptoms, including the prodromal episodes of *amaurosis fugax*, is strongly suggestive of giant cell arteritis (GCA).

Giant cell arteritis

GCA is a condition that generally affects older people, where the lining of the medium and large-sized arteries becomes inflamed.

Typically, the blood vessels of the scalp, neck and arms can be involved and particularly those at the side of the head - the temples - which is why the condition is also known as temporal arteritis.

The inflammation causes a narrowing or blockage of the blood vessels, which interrupts blood flow to the tissues that are supplied by the artery. In the eye, inflammation of the short posterior ciliary arteries causes arteritic anterior ischaemic optic neuropathy, where profound and irreversible damage to the optic nerve can occur.

Symptoms of GCA include visual loss, head pain (particularly in the temple), scalp tenderness, jaw pain when chewing, fever, fatigue, malaise and weight loss.

Clinical manifestations in the acute episode include swollen optic disc(s), relative afferent pupil defect, reduced visual acuity and visual field defect. Patients who are suspected to have GCA require emergency referral on account of the sight- and life-threatening implications of the condition.

The visual loss from GCA can progress very rapidly and can often involve both eyes.

1: College of Optometrists (2023). <https://www.college-optometrists.org/clinical-guidance/guidance-annexes/annex-4-urgency-of-referrals-table>

Referral

The College of Optometrists recommends that for cases of suspected temporal arteritis, an emergency referral (which they define as ASAP) should be made.¹

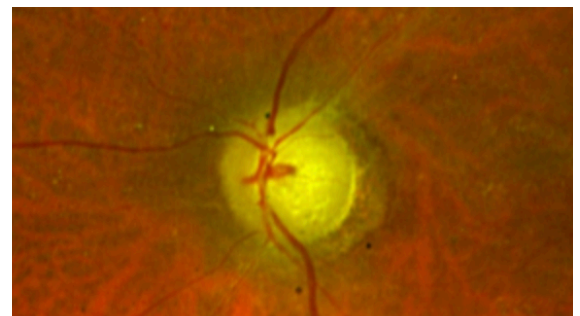
A differential diagnosis for GCA is non-arteritic anterior ischaemic optic neuropathy, where there is insufficient perfusion of the optic disc that leads to optic disc ischaemia. If a patient presents with symptoms suggestive of GCA, it is always wise to err on the side of caution and make an emergency referral due to the potential severity of GCA.

Hospital management

When AR was examined in A&E, his C-reactive protein was measured, which is a marker of inflammation. His erythrocyte sedimentation rate was also measured. He was admitted to hospital under the care of the rheumatology department.

A biopsy of the temporal artery was taken. A 2cm specimen was requested, as there can be 'skip' lesions, so a smaller biopsy does not always show that the relevant inflammatory changes.

AR was treated with high-dose intra-venous methylprednisolone (steroid). The laboratory results were strongly suggestive of GCA. Unfortunately, the acuity in his left eye continued to deteriorate and, with time, the optic disc became pale and atrophic (see below) and the acuity did not improve beyond 2/60.



Acknowledgment

Ms Katherine Smyth, Consultant Ophthalmologist, Bolton NHS Foundation Trust.

Produced by
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on behalf of FODO


The Association for
Eye Care Providers