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SHORT COMMUNICATION

Case report

Giant cell arteritis mimicking idiopathic orbital inflammatory disease

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PURPOSE. *To report an unusual presentation of giant cell arteritis, referred from primary care, mimicking orbital apex syndrome.*

CASE REPORT. *A 72 year old woman was referred with a two week history of pyrexia, dull right eye ache, 2mm of right proptosis, mild conjunctival chemosis and restriction of right eye movements.*

RESULTS. *An erythrocyte sedimentation rate (ESR) was 90 and fluorescein angiography showed almost complete choroidal non-perfusion suggestive of giant cell arteritis. Temporal artery biopsy confirmed the diagnosis.*

CONCLUSIONS. *Giant cell arteritis (GCA) typically presents with anterior ischemic optic neuropathy (AION), choroidal ischemia, central retinal artery occlusion, infrequently manifesting as an ocular motility problem, but has rarely been known to mimick idiopathic orbital inflammatory disease. Prompt recognition and therapy can minimize the chance of ipsilateral ocular involvement and protect the fellow eye. (Eur J Ophthalmol 2003; 13: 392-4)*

KEY WORDS. *Central retinal artery occlusion, Giant cell arteritis, Orbital apex syndrome.*

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INTRODUCTION

The central retinal artery, short posterior ciliary arteries and muscular branches of the ophthalmic artery can all be sequentially affected by arteritis. We describe an unusual presentation of giant cell arteritis, referred from primary care, mimicking orbital apex syndrome.

Case report

A 72 year old woman was referred with a two week history of pyrexia, dull right eye ache and right sided frontal headaches. Examination revealed 2mm of right proptosis, mild conjunctival chemosis and restriction of right eye movements on adduction, upgaze and abduction. Right visual acuity (RVA) was 6/9, left 6/6, with no relative afferent pupil defect, normal fields to confrontation, and the right optic disc was mildly swollen. The working diagnosis was right orbital apex syndrome. An urgent CT head scan showed non-specific inflammatory changes of the right apical orbital fat and around both optic nerves, normal extraocular muscles and paranasal sinuses and excluded the presence of a retro-orbital abscess.

When seen by us the following morning, eye movements were unchanged (Fig. 1), RVA was perception to light (PL) and there was a cherry red spot, optic disc swelling and retinal artery cattle trucking (Fig. 2a). An erythrocyte sedimentation rate (ESR) was 90 and fluorescein angiography showed almost complete

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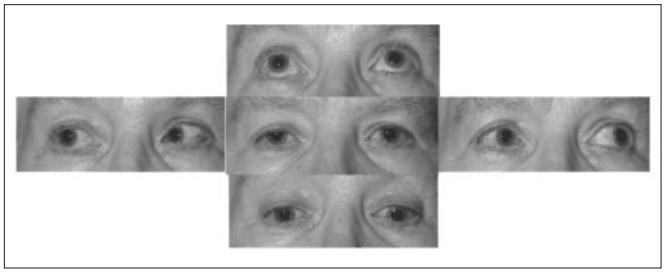

Fig. 1 - *Restricted eye movements on presentation.*

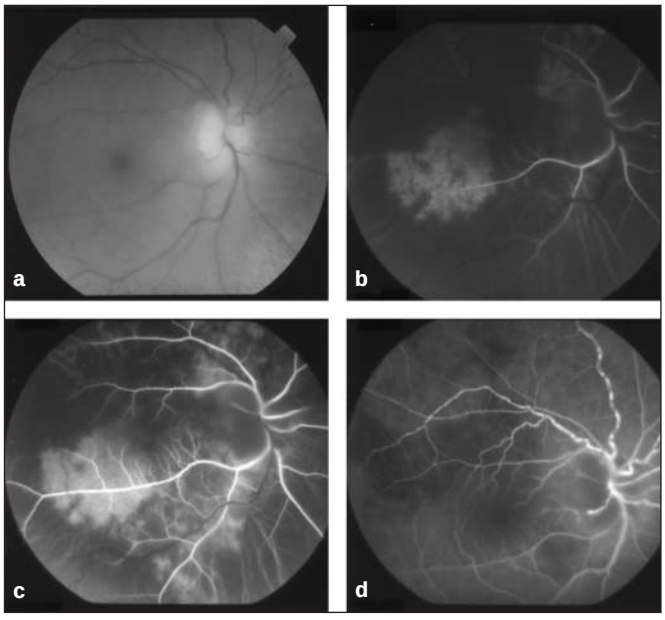

Fig. 2 - a) *Right swollen optic disc and cherry red spot.*
b) *Delayed arterial phase. Minimal choroidal perfusion at 24 seconds.*
c) *Persistent delayed choroidal perfusion 8 seconds later at 32 seconds.*
d) *Late venous phase. Cattle trucking at 3 minutes.*

choroidal non-perfusion (Fig. 2b) suggestive of giant cell arteritis (GCA). Intravenous methylprednisolone (1g) was administered for three consecutive days. Temporal artery biopsy confirmed the diagnosis of GCA. The pyrexia, lid swelling, right dull eye ache, frontal headaches and eye movements resolved entirely within three days, but the RVA improved only to counting fingers (CF).

DISCUSSION

GCA typically presents with anterior ischemic optic neuropathy (AION), choroidal ischemia, (1) central retinal artery occlusion, infrequently manifesting as an ocular motility problem, but has rarely been known to mimick idiopathic orbital inflammatory disease (2, 3). This presumably occurs due to swelling of the orbital tissues in response to infarction or ischemia of the branches of the ophthalmic artery. Abnormal eye movements are reported in GCA. The incidence of diplopia with GCA is estimated at 10 to 17% (4), although presentation of manifest ophthalmoplegia is rare and is believed to be due to infarction of a cranial nerve trunk (5), or ischemic necrosis of the extraocular muscle (6). The CT scan changes did not suggest frank orbital inflammatory disease or abscess and there was no significant enlargement of the extraocular muscles.

This lady had lack of gross visual field deficit, in particular altitudinal field loss, which can occur in AION. In retrospect there was no history of polymyalgia rheumatica nor signs of scalp tenderness. There was no jaw claudication, which is pathognomonic for AION. The picture (Fig. 2) of rapidly developing retinal changes suggested the diagnosis of GCA, which was subsequently confirmed by an ESR and temporal artery biopsy. The well recognised changes of impaired central retinal artery perfusion combined with optic disc swelling were indicative of more than simple central retinal artery obstruction. Fluorescein angiogram confirmed significant non-perfusion of the choroidal circulation, reflecting occlusive changes in the ophthalmic artery due to the arteritis. Hayreh has previously noted massive choroidal non-perfusion from occluded posterior ciliary arteries and stated that this was “almost diagnostic” of arteritic disease (7).

It is important to recognise this unusual presentation of giant cell arteritis at an early stage so that prompt therapy can be initiated to minimize the chance of ipsilateral ocular involvement and to protect the fellow eye.

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