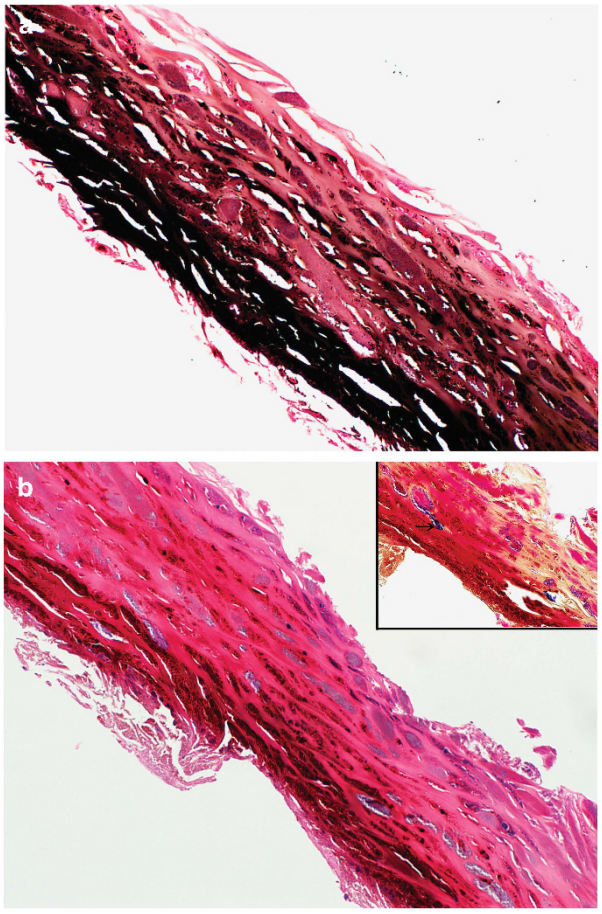

Figure 2 (a) Fontana Masson stain of corneal biopsy demonstrating pigmented granules that stain positively, indicating the presence of adrenochrome (× 20). (b) Hematoxylin and eosin stain corneal biopsy demonstrating pigmented, acellular intrastromal material (× 20). Epithelium and Bowman's membrane are absent, consistent with clinical defect. Gram stain demonstrates diffuse intralamellar Gram-positive cocci (insert, arrow).

Fontana Masson, but negatively for iron (Figure 2). At this point, the ibopamine drops were discontinued.

Comment

Bacterial organisms can produce condition that promotes adrenochrome autoxidation. An acidic environment is created by the formation of lactic acid as the major metabolic end-product of carbohydrate fermentation by *S. viridans*. In our patient, the concurrent infection with *S. viridans* may have facilitated the oxidation of ibopamine into its degradation products, resulting in the rapid pigment deposition in the cornea. It is also possible that the deposition would have occurred even without the favorable environment created by the infectious keratitis. Animal models have shown that for pigment deposition to occur, oxidized adrenochrome and a susceptible corneal surface must be present,[6] a condition satisfied by our patient's persistent epithelial defect. Thus, based on our observations, patients using topical ibopamine eye drops should be carefully monitored for pigment deposition in the cornea, particularly in the presence of a compromised epithelial surface.

Conflict of interest

The authors declare no conflict of interest.

Acknowledgements

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SJ Bhosai[1], CC Lin[1,2,3], J Greene[1], MM Bloomer[1] and BH Jeng[1,2,3]

[1]Department of Ophthalmology, University of California San Francisco, San Francisco, CA, USA
[2]Francis I. Proctor Foundation, University of California San Francisco, San Francisco, CA, USA
[3]Department of Ophthalmology, San Francisco General Hospital, San Francisco, CA, USA
E-mail: jengb@vision.ucsf.edu

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Sir,

How common is inflammatory marker-negative disease in giant cell arteritis?

Giant cell arteritis is an inflammatory vasculitis affecting medium- and large-sized arteries and can result in arteritic anterior ischaemic optic neuropathy. C-reactive protein

level and erythrocyte sedimentation rate are commonly used to aid diagnosis; however, inflammatory-marker negative disease does occur.

Case report

A 67-year-old Finish woman presented with a 2 day history of left visual loss. She had a history of polymyalgia rheumatica for which steroid treatment had been stopped 12 months prior.

On presentation best corrected visual acuity was 6/36 in the left eye. Clinical examination revealed a left relative afferent papillary defect and reduced colour vision. On dilated examination a pale, swollen left disc was observed. Erythrocyte sedimentation rate (ESR) was 62 mm/h and C-reactive protein (CRP) was normal (<8 mg/l).

The patient was admitted for intravenous Methylprednisolone and a temporal artery biopsy was consistent with a diagnosis of giant cell arteritis (GCA; Figure 1).

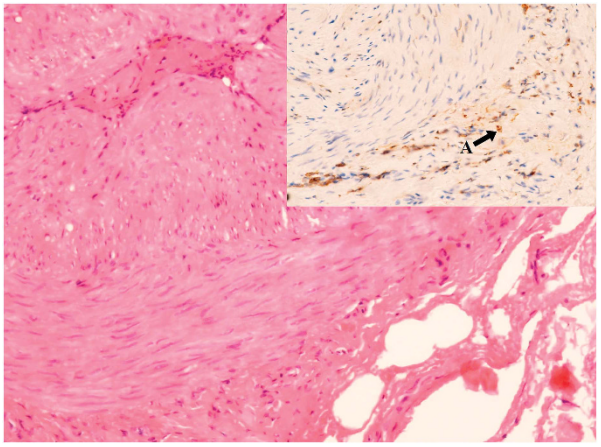

Figure 1 A composite histology slide showing one cross-section through the temporal artery biopsy (TAB). The larger image shows a haematoxylin and eosin stain of the artery wall, and the inset is stained with leucocyte common antigen (CD45) highlighting the lymphocytic infiltrate of the media (A). Fragmented internal elastic lamina was also seen.

Comment

The annual incidence of GCA in the Scandinavian populations is reported to be as high as 15–35/100 000 in those over the age of 50,[1] higher than the incidence in the standard European population. Inflammatory markers are commonly used to aid diagnosis of GCA. CRP has been reported to be a more sensitive predictor of the disease than ESR (97.5–100% for CRP vs 76–92% for ESR).[2,3] Our case is unique in that it belongs to the rarer group of CRP-negative disease and highlights four pertinent facts about the pathophysiology of GCA:

(1) ESR and CRP together have a superior sensitivity than either test alone.
(2) Polymyalgia rheumatica and GCA are two closely related inflammatory syndromes.
(3) The disease rarely burns out spontaneously and cessation of steroid therapy carries a high risk of reactivation or progression from polymyalgia rheumatica to full-blown GCA.
(4) Scandinavian ancestry increases pretest probability.[4]

Including our case there are only three published cases of isolated CRP-negative GCA and only two cases of simultaneous ESR and CRP negativity (see Table 1).

Our review of the current literature demonstrates that simultaneous ESR- and CRP-negative disease is rare. Both parameters together offer a good safety net through which GCA will only rarely slip. It is important to consider medications affecting inflammatory markers. Hegg *et al*[6] report that both nonsteroidal anti-inflammatory drugs and statins were associated with a lower ESR in biopsy-proven GCA. Fibrates have recently been shown to reduce CRP levels,[7] however, our patient was not on fibrates and therefore her CRP negativity remains unexplained. Contrary to Hayreh *et al*'s observation[2] ESR was more sensitive to detect GCA-related inflammatory activity in the presented case.

Table 1 Summary of current literature

	ESR-negative disease (%)	*CRP-negative disease (%)*	*ESR- and CRP-negative disease (%)*
Parikh *et al*[3]	14.3	1.7	0.8
Poole *et al*[5]			1 Case report
Levy *et al* (current study)		1 Case report	

Conflict of interest

The authors declare no conflict of interest.

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SL Levy[1], AD Bull[2] and AR Nestel[3]

[1]Department of Ophthalmology, Royal Hallamshire, South Yorkshire, Sheffield, UK
[2]Department of Histopathology, North Devon District Hospital, Devon, UK
[3]Department of Ophthalmology, North Devon District Hospital, Devon, UK
E-mail: sarahfoster@doctors.org.uk

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Sir,

An unusual case of orbital cellulitis due to Panton Valentine Leucocidine producing *Staphylococcus aureus*

We report an unusual case of orbital cellulitis due to Panton Valentine Leucocidin (PVL) producing *Staphylococcus aureus* bacteraemia secondary to a furuncle. Both the orbital cellulitis and the secondary pulmonary involvement resolved completely with linezolid and clindamycin.

Case report

A 68-year-old Asian male presented with rapid onset right upper lid swelling, redness and pain. The symptoms started 24 h after a small boil on the tip of his nose and then progressed to a full blown orbital cellulitis the next day. He had poorly controlled type 2 diabetes mellitus (HbA1c 6.9). On examination visual acuity was light perception and he had axial proptosis, a very tense orbit, severe chemosis and ophthalmoplegia (Figure 1). He was apyrexial and did not have any known immune deficiency or compromise. Blood culture was taken and intravenous flucloxacillin, ceftriaxone and metronidazole were administered.

After 48 h of treatment there was no clinical improvement. He developed pleural effusion (Figure 2). Results of blood culture yielded PVL-positive *Staphylococcus aureus* with leukocytosis of 40 000 cells/ml and CRP was 187 mg/l. Treatment was switched to Linezolid and clindamycin. Pulmonary involvement and orbital cellulitis resolved after a 2-week course of new regime.

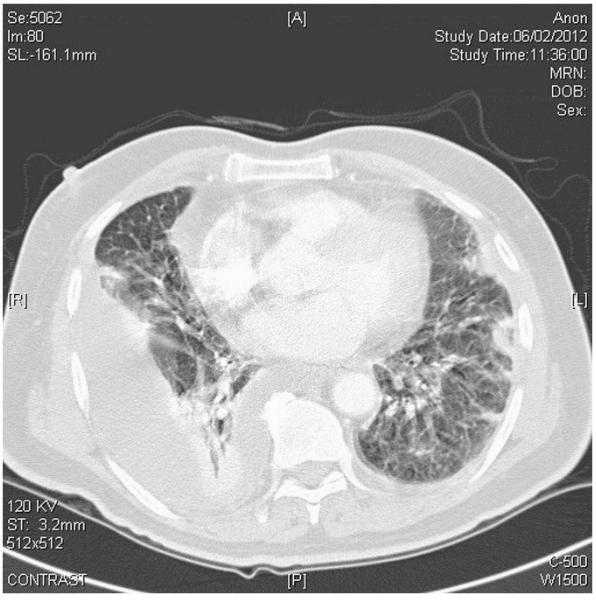

Figure 2 The pulmonary CT angiogram shows left-sided dependent pleural effusion and a greater right-sided loculated effusion with a paraspinal pleural collection.

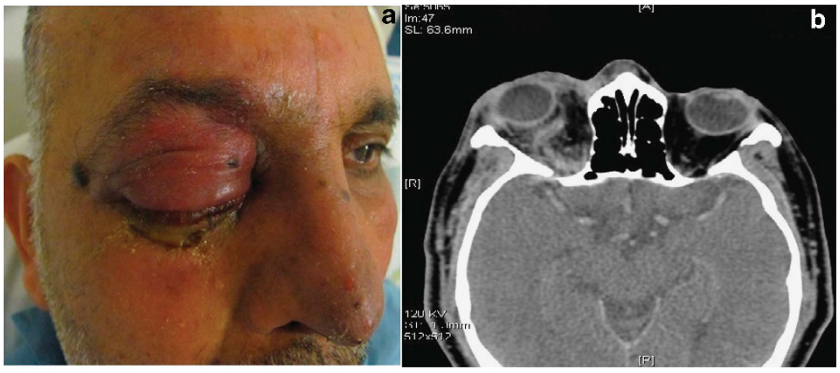

Figure 1 (a) A photo of the patient. (b) Axial CT scan of the orbit, showing several patchy areas of inflammatory change within the cone area.