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Sir,

Wegener's granulomatosis masquerading as upper lid chalazion

A 79-year-old gentleman presented with a left upper lid lump clinically typical of a chalazion. Incision and curettage was performed twice, and each time followed by recurrence. The second time, the lid lesion had grown to a very large size and filled 3/4 of the upper lid and produced a considerable ptosis (Figure 1a). The lesion was incised via a skin incision and curettings were sent for histology together with a tarsal plate biopsy. The histology showed extensive tissue necrosis associated with a heavy eosinophil infiltrate (Figure 1b). Neither active vasculitis nor granulomatous inflammation was identified, but the appearances were considered to reflect tissue involvement in Wegener's granulomatosis (WG), particularly in view of similar findings in a previous prostatic biopsy. Relevantly, the past medical history included previously active WG, which presented as prostatic enlargement. Circulating anti-neutrophil cytoplasmic antibody (cANCA) was high, but there was no other evidence of clinically active WG elsewhere in the body. The patient was commenced on 30 mg prednisolone and azathioprine. With continuing systemic immunosuppression, over the next 4 months, complete resolution of the lesion followed (Figure 1c).

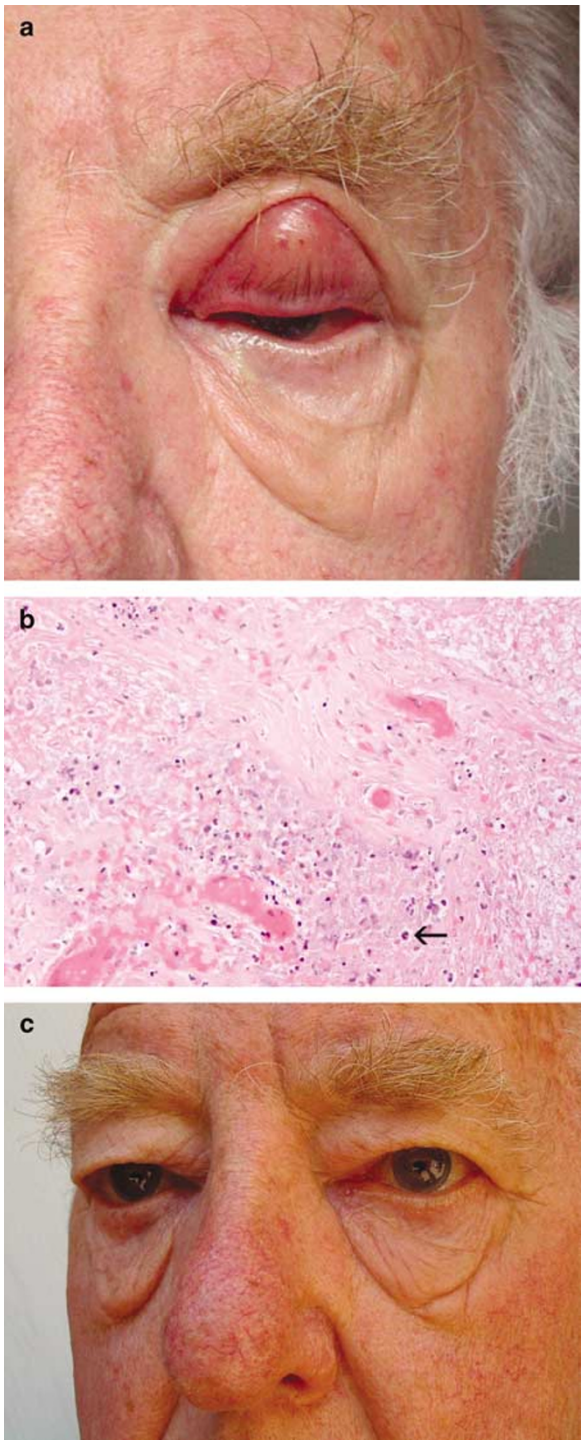

Figure 1 (a) Large left upper lid mass causing secondary ptosis. (b) Histology from tarsal plate biopsy, showing an area of stromal necrosis associated with eosinophilic polymorphs (arrow). (c) Complete resolution of left upper lid lesion.

Comment

Chalazion is the most common inflammatory lesion of the eyelid,[1] but can occasionally be clinically confused

with other benign or malignant lesions—sebaceous gland carcinoma being the most frequently misdiagnosed malignancy.[2] WG is a multisystem disease of unknown aetiology characterized by granuloma formation, necrosis and variable degrees of small to medium-sized vessel vasculitis.[3] Clinical presentation is most common with initial upper or lower respiratory tract, renal, or orbital symptoms.[4] The main orbital signs of the disease are proptosis, scleritis, and lid inflammation,[5] but WG mimicking the presentation of a chalazion has not been reported in the literature. Biopsy results in cases of WG with orbital involvement can be difficult to interpret as there are a spectrum of histopathological features, and the classic triad of vasculitis, tissue necrosis, and granulomatosis inflammation may only be present in about one-half of cases.[6] A positive assay for cANCA with specificity for proteinase-3 or myeloperoxidase provides strong evidence if WG is suspected. Diagnosis of orbital WG therefore requires correlation of the clinical signs, use of the cANCA test and comparison with extraorbital biopsies, if available. This case highlights an atypical presentation of WG to the ophthalmologist, with resolution after conventional systemic treatment of the underlying disorder.

References

1 Aurora AL, Blodi FC. Lesions of the eyelids: a clinicopathological study. *Surv Ophthalmol* 1970; **15**: 94–104.
2 Ozdal PC, Codere F, Callejo S, Caissie AL, Burnier MN. Accuracy of the clinical diagnosis of chalazion. *Eye* 2004; **18**: 135–138.
3 Klippel JH, Dieppe PA. The vasculitides—Wegener's granulomatosis. *Rheumatology* 1994; 6.19.1–6.19.10.
4 Hoffman GS, Kerr GS, Leavitt RY, Hallahan CW, Lebovics RS, Travis WD *et al*. Wegener's granulomatosis. An analysis of 158 patients. *Ann Intern Med* 1992; **116**(6): 488–498.
5 Perry SR, Rootman J, White VA. The clinical and pathologic constellation of Wegener granulomatosis of the orbit. *Ophthalmology* 1997; **104**: 683–694.
6 Kalina PH, Lie JT, Campbell RJ, Garrity JA. Diagnostic value and limitations of orbital biopsy in Wegener's granulomatosis. *Ophthalmology* 1992; **99**(1): 120–124.

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Sir,

Hospital cataract surgery volume and postoperative endophthalmitis

The use of population-based linked administrative data as in Fang *et al*'s[1] recent paper is one of the best ways to investigate cataract surgery outcomes. It provides adequate power for statistical analysis, and eliminates issues of sampling and ability to generalise the results associated with smaller cohort studies and controlled trials. In particular, it represents the 'real life' of the treatment provided to that population.[2,3]

We read with interest Fang *et al*'s[1] description of an association between cataract surgery volume and the risk of postoperative endophthalmitis. Their quoted 2-year incidence postoperative endophthalmitis of 0.84% seems high compared to most other recent reports. Using the same population-based linked data approach, we have looked at cataract outcomes in Western Australia over a 21-year period.[3–6] In our own study, we undertook a comprehensive process of validation of the endophthalmitis cases reported in the database. We found that the International Classification for Diseases codes for endophthalmitis were frequently misused for other eye conditions.[4] As it does not appear that Fang *et al* validated the coded diagnosis of endophthalmitis, could the apparently high rate in Taiwan and the associations with hospital volume be spuriously inflated due to misclassification of other ocular infections as endophthalmitis?

Notwithstanding, their finding that the technique of cataract extraction did not alter endophthalmitis risk was consistent with our own studies.[5,6] This reinforces the importance of multifactorial risk reduction rather than just concentrating on surgical technique to prevent postoperative endophthalmitis.

Using unadjusted and multivariate adjusted Cox regression analyses, Fang *et al* reported that the risk of endophthalmitis was higher at low volume compared to high volume hospitals. Although they discussed the possibility that the differences may be due to referral bias or difference in expertise and systems of care, they did not discuss nor analyse another potential confounding effect, the length of stay in hospital. There were four times as many in-patient cataract operations in high volume compared to low volume hospitals. We previously found that same-day admissions for cataract surgery posed a higher risk for postoperative endophthalmitis. After