

Table 1 Patient demographics for chlamydial positive swabs

Age (years)	n = 300 % of patients with positive chlamydial swabs
0–9	12 (3/26)
10–19	17 (9/52)
20–29	29 (27/94)
30–39	11 (6/57)
40 and over	1 (1/71)
Total	15 (46/300)

such as preauricular lymphadenopathy and follicles, which would be apparent in a primary care setting.⁵ Up to 54% of men and 74% of women would be expected to have concurrent genital infection when presenting with chlamydial conjunctivitis,² although the majority of cases would be asymptomatic.⁵

We would therefore wish to encourage GP's using the Edinburgh Red Eye Diagnostic Algorithm, seeing apparent infective conjunctivitis cases, to consider taking swabs for chlamydia based on patient demographics, history and clinical features. This will minimize delay in diagnosis, or avoid entirely missing the opportunity to pick up chlamydial conjunctivitis, and prevent the systemic and public health implications of an untreated asymptomatic genital infection.

Conflict of interest

The authors declare no conflict of interest.

References

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Sir, Reply: 'The accuracy of the Edinburgh Red Eye Diagnostic Algorithm'

We thank Drs Soomro and Buchan for their interest in our article and for their very pertinent comments regarding the risk of chlamydial conjunctivitis going undiagnosed when clinicians use our diagnostic algorithm to help assess patients presenting with red eye(s). We will add a footnote to the diagnosis of 'infective conjunctivitis' to alert the user to consider chlamydia if the patient is in one of the at-risk groups; <40 years of age or has other suggestive symptoms (genital discharge) or signs such as pre-auricular lymphadenopathy or as per our original advice if symptoms persist despite treatment with topical chloramphenicol.

Conflict of interest

The authors declare no conflict of interest

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Sir, Comment on 'Topiramate maculopathy secondary to dose titration: first reported case'

It was with much interest that we read the recent article by Severn *et al*¹ regarding the first documented report of topiramate maculopathy secondary to an increase in dose. In contrary, in 2014 we shared our experience of acute myopia syndrome secondary to topiramate discontinuation, a dose decrease.² Therefore, we assume that an increase as well as decrease of dosing may have a common physiological mechanism through which both ocular adverse effects are mediated.

We previously hypothesized that the change in topiramate plasma levels was the likely culprit of the ocular effects rather than the administration itself—a theory that corroborates findings of Severn *et al*. A sudden change in plasma levels of topiramate may result in abnormal carbonic anhydrase activity and subsequent fluid accumulation within the uveal tissue, suprachoroidal space and the vitreous body. The anticipated accumulation of H⁺ in the uveal tissue and consequently altered permeability of choriocapillaris could also account for the etiology of choroidal folds. Interestingly, we also found choroidal folds as a feature of advanced acute myopia syndrome suggesting that topiramate maculopathy may be one of the early signs or an incomplete manifestation of acute myopia syndrome.

We believe that the crux for ocular adverse effects of topiramate could be in its pharmacokinetics, which is known to be unbalanced as it accumulates