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.

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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

disproportionately in erythrocytes in blood.[3] Therefore, we suspect that the fluctuation in dosing could be responsible for abrupt changes in plasma levels and thus, disproportionate carbonic anhydrase activity in susceptible individuals. Similarly, it implies that any insult to the red cells could result in an unexpected change of topiramate plasma levels.

In our opinion, the sudden changes in the plasma levels of topiramate should be avoided if possible and its mode of titration be reviewed in conjunction with both neurologists and pharmacologists.

Conflict of interest

The authors declare no conflict of interest.

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

We agree with Kozner *et al* that the maculopathy reported in our case[1] likely represents a mild form of the idiosyncratic reaction causing choroidal effusion, induced myopia, and angle-closure glaucoma that has been reported with topiramate and other sulfur-containing medications. In these cases, it is a consistently reported feature that the reaction generally occurs within days following commencement of the drug or an increase in dose.[2] In Kosner's case,[3] the reaction occurred 3 days after discontinuing topiramate and a sulfur-containing antibiotic, but the patient had commenced the medication 10 days prior to presentation. It is therefore difficult to be sure of the precise trigger in this case.

The important clinical point is that discontinuation of the drug leads to resolution of the symptoms. In some reports, resolution has possibly been hastened following the administration of IV methylprednisolone.[2,3]

Conflict of interest

The authors declare no conflict of interest.

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Sir,
TLP: a premature concept

Siaudvytyte *et al*[1] present a literature review and meta-analysis regarding translaminar pressure difference (TLPD) in open-angle glaucoma. Five studies that demonstrated a higher TLPD in open-angle glaucoma have been analyzed in detail.

TLP defined as intraocular pressure (IOP) minus cerebrospinal fluid pressure (IOP – CSF-p)[2] is an interesting mechanical concept. However, there are some critical considerations concerning the interpretation and implementation of TLP we would like to mention.

First, as discussed by Siaudvytyte *et al*[1] both IOP and CSF-p used in the equation for TLPD are dynamic and fluctuate independently over time with numerous variables affecting both intracranial pressure and intraocular pressure measurements. Therefore, the estimation of TLPD would be best done by a simultaneous measurement of both IOP and CSF-p. None of the presented studies did it and thus all TLPD studies are dealing with two non-dependent and ever-changing variables to a definite time leaving us without a correlation that would fulfill the requirements of the simple physical equation of pressure, which is force over area at a definite time.

Second, in four out of the five analyzed studies, CSF-p has been measured by lumbar puncture and the lumbar CSF-p was extrapolated to the retrolaminar CSF-p. The assumption the lumbar CSF-p might equal the retrolaminar CSF-p only holds if the CSF-p is homogenous distributed in all CSF spaces, inclusively the subarachnoid space of the