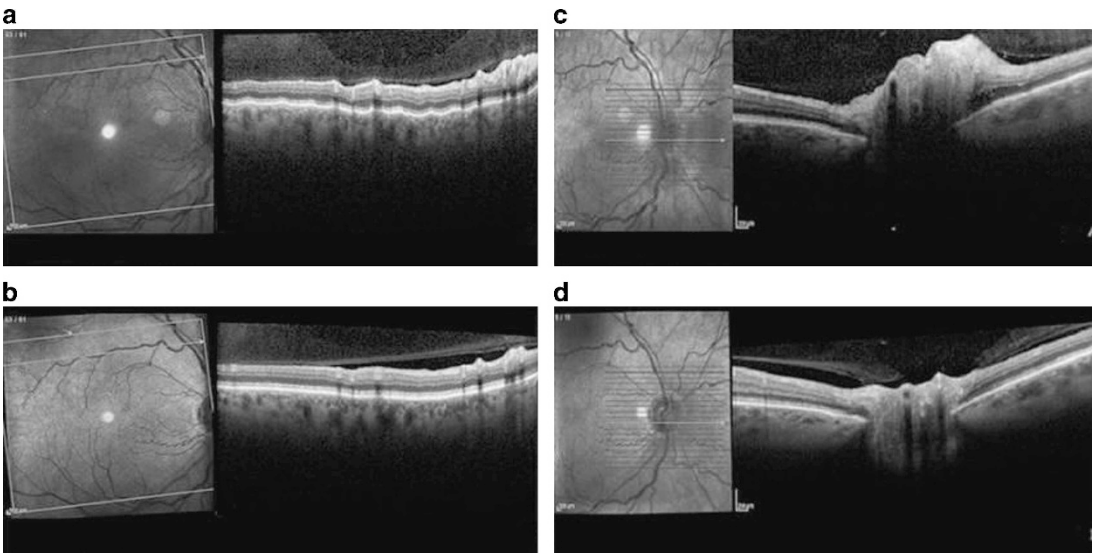

Figure 2 High-resolution OCT images of the posterior pole during acetazolamide administration with retinal folds (a) and after the resolution of the effusion (b). High-resolution OCT images of the posterior pole during acetazolamide administration with papillary oedema (c) and after the resolution of the effusion (d).

manifestations after the drug administration was discontinued.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Spontaneous haemorrhage in an eyelid hidrocystoma in a patient treated with clopidogrel

Hidrocystomas are benign cystic tumours of the sweat glands that frequently occur in the periocular region. Rarely, more serious pathology such as basal cell carcinoma or malignant melanoma may resemble and be mistaken for hidrocystoma.[1,2] We report a very unusual presentation of spontaneous bleeding within a hidrocystoma, mimicking a malignant melanoma.

Case report

A 62-year-old woman presented with a lesion on the medial aspect of the left upper lid, which was slowly enlarging over 2 years and darkened 6 months earlier. She admitted to having excessive sun exposure in the past, and her past medical history included hypertension, type II diabetes, and peripheral vascular disease. Her regular medications were insulin, metformin, simvastatin, ramipril, and clopidogrel.

Examination revealed a curious lesion on the medial aspect of the left upper lid (Figure 1a). The lesion, which appeared cystic and lobulated, was mostly skin coloured with surface telangiectasia and purple–blue discoloration inferiorly. Although a benign lesion was suspected, the unusual appearance prompted excisional biopsy. Histopathological examination showed a papillary hidrocystoma with apocrine differentiation, featuring small lakes of blood within the myxoid material occupying the lumen (Figure 1b).

Comment

Apocrine hidrocystomas usually present as slow-growing, solitary, or multiple small, tense,

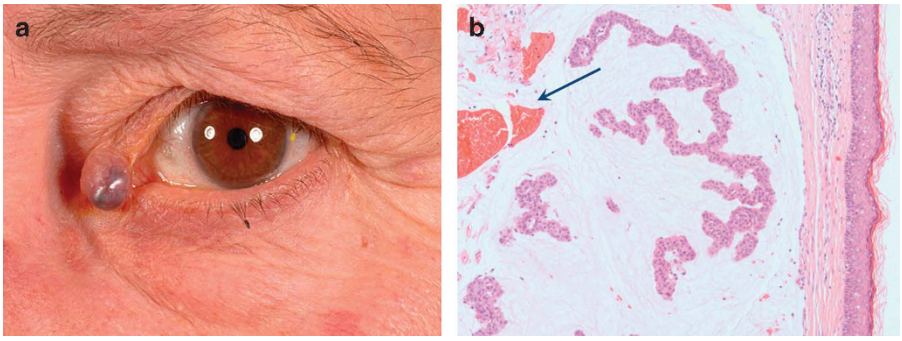

Figure 1 (a) Clinical appearance of the cystic lesion on the medial aspect of the left upper lid with telangiectatic vessels on the surface and localised purple–blue discolouration inferiorly. (b) Subcutaneous cyst lined predominantly by papillary apocrine and conventional double-layered hidrocystoma epithelium, and containing small lakes of blood (arrow) within the luminal basophilic myxoid material (H&E ×10).

dome-shaped, thin-walled cysts. They are usually translucent, skin-coloured lesions, but are occasionally lightly or deeply pigmented. Histologically, they are characterised by a unilocular or multilocular cystic space in the dermis with a lining consisting of a double layer of epithelial cells.[1] Although there is a single case report of a giant apocrine hidrocystoma presenting as a tense haematoma of the scalp,[3] to the best of our knowledge there are no published reports of eyelid hidrocystomas presenting with spontaneous bleeding within the lesion. A possible predisposing factor for intralesional bleeding in our case was regular use of clopidogrel, an antiplatelet agent that has been associated with spontaneous haematomas at other sites.[4,5]

Conflict of interest

The authors declare no conflict of interest.

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Sir,

Purtscher retinopathies: Are we aiming at the wrong target?

We welcome, and read with interest, the systematic review of Purtscher retinopathies by Miguel *et al*,[1] who described the systemic aetiologies underlying Purtscher's, the clinical features, the efficacy of corticosteroid therapy, and visual outcomes.

The discovery of effective therapies for Purtscher-spectrum retinopathies requires identification of the mechanism underlying Purtscher-related microvasculopathy. Despite the sporadic treatment of Purtscher's with corticosteroids, only 8 of the 17 Purtscher-associated aetiologies identified by the authors are primary inflammatory disorders, 5 of which necessitate systemic steroid therapy (Table 1). Ocular inflammation is not a feature of Purtscher retinopathies.

We observed that 14 Purtscher-associated aetiologies were recognized precipitants of thrombotic microangiopathy (TMA) (Table 1)—a systemic syndrome that triggers widespread microvascular thrombosis in response to a range of primary disorders.[2] Ocular features of Purtscher's include cotton wool spots, retinal haemorrhages, Purtscher-flecken, and arteriolar obstruction with late leakage on fluorescein angiography.[1] Thrombotic microangiopathy may account for all features through terminal arteriole and capillary hyaline thrombosis—pathological hallmarks identified in patients as early as 1924.[2]

The molecular pathophysiology of TMA is heterogeneous, varying according to systemic aetiology. TMA involves a complex, interdependent dysregulation of haemostatic, thrombotic, and complement cascades with endothelial dysfunction and inflammation—