ophthalmoplegia in the left eye appeared (Figure 1b). There was only a nasal island of visual field in the right eye, whereas a small relative central scotoma was detected in the left at that time (Figure 3b). Another episode of ophthalmoplegia in the right eye occurred on 3 September, 1997. He also suffered from bilateral episcleritis, and nodular erythema and thrombophlebitis at extremities at different time points. These events always accompanied the bowel symptoms such as diarrhoea and abdominal pain as well as the elevated platelet counts and CRP level, which completely recovered immediately after steroid treatment (Figures 1c and 5). Systemic and ocular symptoms were silent from October 1997, until the last visit in January 2001.

Comment

Patients with UC have aberrant coagulative and fibrinolytic conditions leading to the vascular endothelial damage and sometimes manifest cerebral vascular complications. [1,2,5] Nelson *et al*[2] demonstrated necrotizing vasculitis in a frontal brain biopsy specimen taken from a 19-year-old male patient with UC. Fibrinoid necrosis accompanying acute inflammatory cells involved meningeal and cortical blood vessels.[4] Optic neuritis and ischaemic optic neuropathy were also reported in patients with UC.[6] To the best of our knowledge, however, this is the first attempt to show a UC patient manifesting total ophthalmoplegia.

Disease activity of UC is correlated with several inflammatory and coagulation markers, which include the platelet counts and CRP level.[1,2,5] Platelets release several kinds of chemical mediators, such as platelet-activating factor and thromboxane, increasing inflammatory cells in and permeability of the vessel walls. Serum levels of the circulating soluble intercellular adhesion molecule-1 paralleled with the CRP dynamics.[5] Taken together with the absence of space-occupying lesions in the brain, microcirculatory disturbance of the cranial II–VI nerves at the orbital apex region due to the aberrant coagulative and fibrinolytic status associated with UC was presumably the pathogenesis of ocular symptoms in the current case.

Acknowledgements

Proprietary interest: None

References

1 Schneiderman JH, Sharpe JA, Sutton DM. Cerebral and retinal vascular complications of inflammatory bowel disease. *Ann Neurol* 1979; **5**: 331–337.
2 Nelson J, Barron MM, Riggs JE, Gutmann L, Schochet Jr SS. Cerebral vasculitis and ulcerative colitis. *Neurology* 1986; **36**: 719–721.
3 Ellis PP, Jentry JH. Ocular complications of ulcerative colitis. *Am J Ophthalmol* 1964; **58**: 779–785.
4 Saida N, Matsutani A, Ogawa K. Optic neuritis in a patient of ulcerative colitis. *Jpn J Ophthalmol* 1996; **50**: 1301–1306.
5 Collins CE, Rampton DS. Platelet dysfunction: a new dimension in inflammatory bowel disease. *Gut* 1995; **36**: 5–8.
6 Sedwick LA, Klingele TG, Burde RM, Behrens MM. Optic neuritis in inflammatory bowel disease. *J Clin Neuro-ophthalmol* 1984; **4**: 3–6.

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Sir,

Tubular apocrine adenoma—an unusual eyelid tumour

A 63-year-old woman presented to an eye department with a lower lid cutaneous lesion present for a period of 6 months. She previously had a lesion excised from the same site 2 years before, for which no, histopathological records were available. The present lesion, which measured 6 mm in diameter, was not adherent to deeper structures and was not associated with regional lymphadenopathy. The lesion was excised (with a 2 mm margin of normal skin) with preservation of underlying orbicularis oculi muscle. Histopathological examination of the excised lesion demonstrated a benign skin adnexal neoplasm. It contained gland-like structures with a predominantly tubular pattern and a fibrous stroma (Figure 1). Columnar cells lined the ductal system with an outer cuboidal layer. There was also evidence of decapitation secretion in the columnar cells lining the ducts (Figure 2). Immunohistochemical studies (Figure 3) revealed an epithelial phenotype consistent with apocrine differentiation (EMA and cytokeratin 7

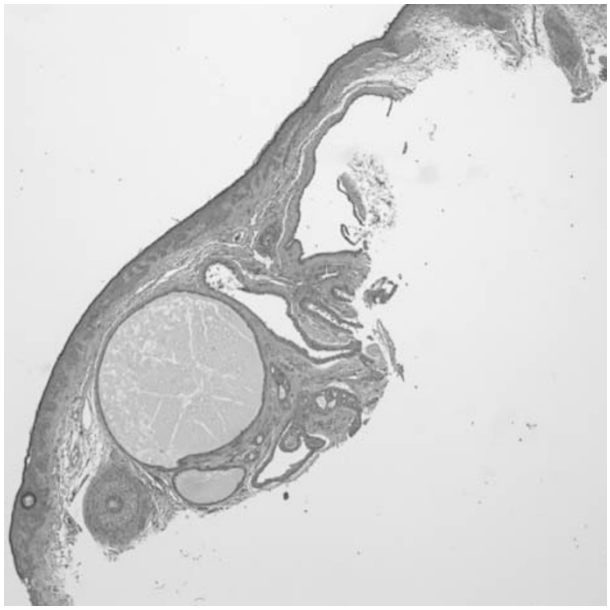

Figure 1 Tubular structures diffusely infiltrating stroma (H + E x40).

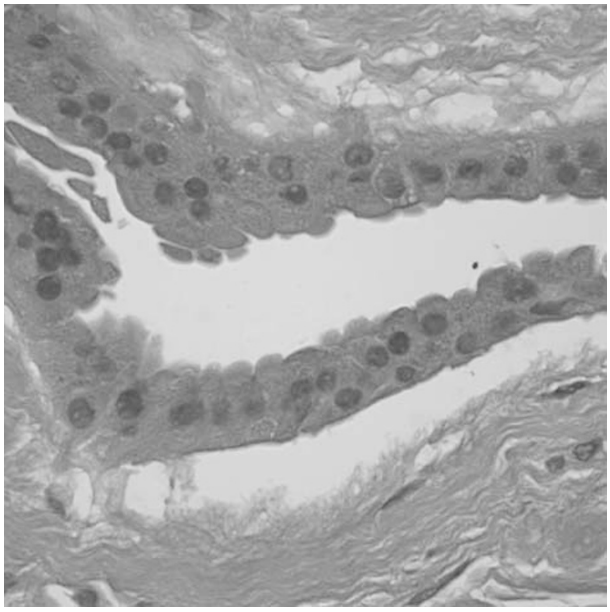

Figure 2 Decapitation secretion seen within the tubular structures (H + E x400).

immunoreactive), and peripheral myoepithelial cells were demonstrated by smooth muscle actin immunoreactivity. In addition, the tumour cells were oestrogen receptor positive (Figure 3). Proliferation index was assessed with Ki67 immunoreactivity and was very low. These features are consistent with a tubular apocrine adenoma.

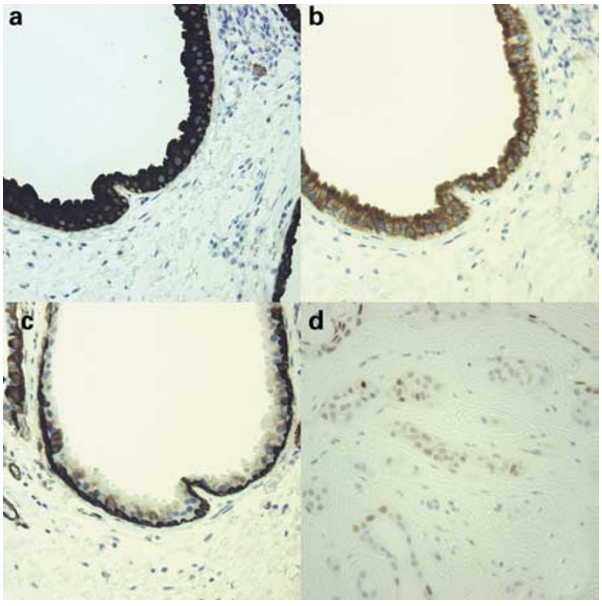

Figure 3 Immunohistochemical profile. The lining epithelial cells were both cytokeratin 7 (a) and EMA (b) immunoreactive. Peripheral myoepithelial cells were highlighted by smooth muscle actin (c) and there was variable nuclear oestrogen receptor immunoreactivity (d). A, B and C x200, D x400.

Tubular apocrine adenoma is a rare apocrine neoplasm first described by Landry and Winkelmann.[1] They are nodular, frequently large, slow growing, and are found most commonly on the scalp. Histologically, the tumour is characterised by lobules of well-differentiated tubular structures located in the dermis. The tubules are lined by an inner layer of tall columnar cells, which show decapitation secretion, and frequently show an outer layer of cuboidal cells. Comedo-like channels that extend into the epidermis and connect with some of the tubular structures are occasionally seen.[1] The stroma is composed of fibrous tissue with few inflammatory cells. This is in contrast to syringocystadenoma papilliferum, which contains numerous inflammatory cells in the stroma.[2] While it is most commonly seen in the scalp region tubular apocrine adenomas have been described in other areas such as the skin of the chest.[3]

This tumour has also been reported to occur within a mammary adenoid cystic carcinoma.[4] A number of reports have described tubular apocrine adenoma, with areas within the tumour showing features of syringocystadenoma papilliferum.[3,5]

Immunoperoxidase studies have shown that the tumour cells contain cytokeratin. Human milk protein and carcinoembryonic antigen have been localised to the apical region of the cell.[5] Oestrogen receptor immunoreactivity is, not unsurprisingly, seen in benign apocrine tumours.

Electron microscopic studies of these tumours have shown luminal cells to have vacuoles and lipid granules and microvilli that project into the lumen of the tubules.[6] We have presented a case of tubular apocrine adenoma, a rare tumour with a predilection for the scalp region, on the eyelid margin, which recurred following initial excision. To our knowledge, this tumour has not previously been reported in the eyelid skin.

References

1 Landry M, Winkelmann RK. An unusual tubular apocrine adenoma. *Arch Derm* 1972; **105**: 869–879.
2 Weedon D. The skin: systemic pathology. 3rd Ed. Vol 9. in Symmers WStC (Ed.) Churchill Livingstone: London, 1992; 836.
3 Epstein BA, Argenyi ZB, Goldstein G, Whitaker D. An unusual presentation of a congenital benign apocrine hamartoma. *J Cutan Pathol* 1990; **17**: 53–58.
4 Prioleau PG, Santa Cruz DJ, Buettner JB, Bauer WC. Sweat gland differentiation in mammary adenoid cystic carcinoma. *Cancer* 1979; **43**: 1752–1760.
5 Ansai S, Watanabe S, Aso K. A case of tubular apocrine adenoma with syringocystadenoma papilliferum. *J Cutan Pathol* 1989; **16**: 230–236.
6 Umbert P, Winkelmann RK. Tubular apocrine adenoma. *J Cutan Pathol* 1976; **3**: 75–87.

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Sir,

Extranasal nasal glioma

A 1-week-old girl was referred for an opinion on a possible dacryocoele (Figure 1) by her paediatrician. The lesion was fluctuant but not mobile and approximately 2 cm in diameter. Because of the paracentral location, a dermoid cyst was considered the most likely diagnosis. MRI scanning excluded intracranial extension of the lesion (Figure 2).

2 months before the planned surgery, the oculoplastic team was consulted. It was felt that because of the midline location, a small encephalocoele should be considered. Thin slice CT sagittal scans were performed, which excluded any small bony defects (Figure 3). The scans were examined by a paediatric neuroradiologist who felt that a dermoid cyst was the most likely cause.

Excision biopsy was performed. The lesion appeared to be firmer than a typical dermoid cyst and was removed *en masse* without rupture. There was attachment via a thin stalk to a small depression on the anterior surface of the frontal process of the maxilla. There was no leakage of CSF from the cut end of the attachment when severed.

Histological analysis suggested extranasal nasal glioma (Figure 4). The child remains well with no evidence of local recurrence.

Discussion

Congenital midline nasal masses (CMNMs) are rare, presenting in 2–5 per 100,000 live births.[1] The differential

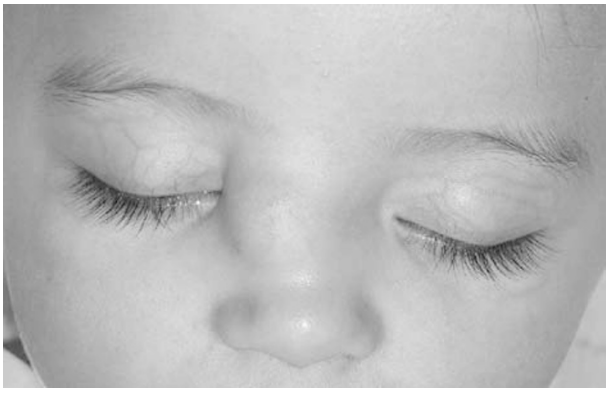

Figure 1 Photograph of the congenital midline nasal mass.

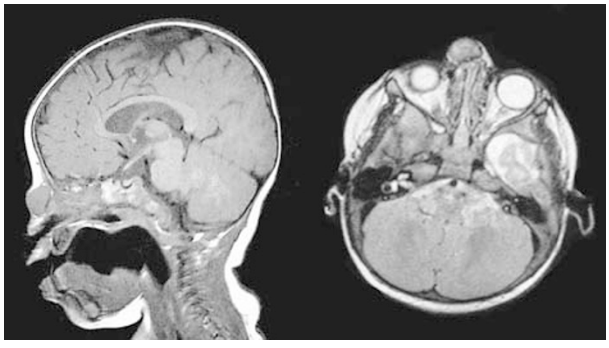

Figure 2 Sagittal MRI T1 weighting (left) and axial MRI T2 weighting (right) scans: the brain is normal. The lesion shows an isointense signal on T1 and a slight hyperintense signal on T2 weighting.