be made with regard to neurological symptoms prior to accepting the diagnosis of pseudopapilloedema.

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Sir,

Alternate total ophthalmoplegia and optic neuropathy associated with ulcerative colitis

Ulcerative colitis (UC) is an idiopathic, nonspecific inflammatory bowel disease involving primarily the mucosa and submucosa of the colon and occasionally manifests extraintestinal complications.^{1–4} In the ophthalmic field, conjunctivitis, episcleritis, and iridocyclitis as well as optic neuritis and retinal vasculitis

have been reported.^{3,4} Here, we present a case of alternate total ophthalmoplegia and optic neuropathy presumably due to vasculitis and ischaemia associated with UC.

Case report

A 50-year-old Japanese man was referred to our hospital on 30 October, 1994, because of a sudden onset of retrobulbar pain on 5 August, visual loss on 8 October, and ophthalmoplegia on 29 October, in the right eye. He had an 18-year history of UC, which was histopathologically confirmed at the right hemicolectomy in 1975, and had been treated with salazopyrin. He had no past history of migraine, varicella-zoster infection, Behcet's disease, and collagen diseases other than UC. The right eye showed blepharoptosis, total ophthalmoplegia, and decreased corneal sensitivity (Figure 1a). Best-corrected visual acuity was hand motion in the right eye and 20/20 in the left. Anterior segments and media were normal except for fixed mydriasis of the right pupil. Findings of the optic disc and retina were unremarkable (Figure 2a). Goldmann's perimetry

Figure 1 Diagnostic positions of gaze. (a) On 30 October 1994, blepharoptosis and total ophthalmoplegia in the right eye are shown. (b) On 28 January 1995, ophthalmoplegia is apparent in the left eye. Note that the right ocular movement is normal. (c) On 16 February 1995, 18 days after steroid therapy, eye movement is bilaterally normal.

revealed a large, absolute central scotoma perforating into the temporal periphery in the right eye and a normal visual field in the left (Figure 3a). CT and MRI demonstrated no intraorbital, cavernous sinus, and intracranial lesions (Figure 4). Laboratory work-up showed an increase in the platelet counts ($69.5 \times 10^4/\mu\text{l}$; normal, $13.0\text{--}30.0 \times 10^4/\mu\text{l}$) and CRP level (8.72 mg/dl ; normal, $<0.3 \text{ mg/dl}$), and positive rheumatoid factor (Figure 5). Antineutrophil cytoplasmic autoantibody,

Figure 2 Fundoscopic findings. (a) On 30 October 1994, there is no apparent abnormality. (b) On 16 February 1995, optic disc is pale and good in colour in the right and the left eye, respectively.

Figure 3 Goldmann's perimetry. (a) On 30 October 1994, an absolute central scotoma perforates into the temporal periphery of visual field in the right eye, whereas the left eye shows normal visual field. (b) On 28 January 1995, the right eye has only a nasal island of visual field, while the left eye demonstrates a small relative central scotoma.

Figure 4 (a) T1W and (b) Gd-DTPA-enhanced SPIR MRI findings on 30 October 1994. There is no space-occupying lesion in the cavernous sinus (left) and in the orbital apex region (right).

Figure 5 Schematic diagram of the relationship between disease course and changes in the platelet counts and CRP levels. Shaded squares represent platelet counts and closed circles represent CRP levels.

anticardiolipin antibody, and lupus anticoagulant were negative. Additional neurological deficit was undetectable by a neurologist. Methylprednisolone pulse therapy (1000 mg/day for 3 days), and subsequent tapered oral predonine administration immediately diminished ocular pain and ophthalmoplegia with the normalization of the platelet counts and CRP level (Figure 5), whereas visual loss remained unchanged and the optic disc became atrophic (Figure 2b). On 28 January, 1995, visual disturbance ($12/20$) and total

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.

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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