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Sir,

Bilateral jugular vein thrombosis: a rare cause of papilloedema

Papilloedema is often the presenting feature of serious intracranial pathology. Commoner causes include space occupying lesions and benign intracranial hypertension. Impaired cerebral venous drainage is also known to increase intracranial pressure and result in papilloedema. Jugular vein thrombosis is uncommon but may rarely cause papilloedema. We present the case of such a patient.

Case report

A 77-year-old man was seen in the ophthalmic clinic with a 6-month history of headache and a 1-month history of deteriorating vision especially in the left eye.

Past medical history included hypertension and previous myringotomy and T-tube insertion for a right middle ear effusion.

Visual acuity was 6/36 left and 6/12 right. There was no afferent pupillary defect and the ocular media were clear. Fundoscopy revealed bilateral optic disc swelling. He underwent an urgent magnetic resonance imaging (MRI) scan; however, no significant pathology was evident.

A neurological opinion was sought and the patient underwent a lumbar puncture. The CSF opening pressure was elevated at 28 cm H_2O. Brain imaging did not reveal intracerebral venous thrombosis; therefore formal angiography was carried out. This showed slow flow within the venous sinuses and was discovered to be due to a complete occlusion of the right jugular vein and severe narrowing of the left jugular vein (Figure 1). Computerised tomography (CT) scanning of the chest did not show any compressive intrathoracic lesion.

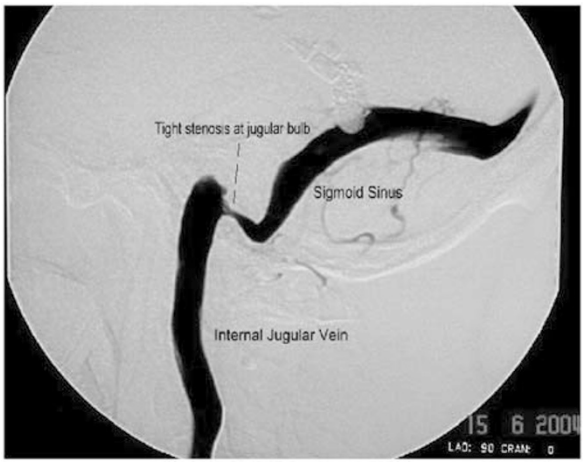

Figure 1 Left jugular venogram showing a narrowing at the jugular bulb.

A full range of blood test were performed including blood count, urea and electrolytes, inflammatory markers, tumour markers, immunological screen, and a thrombophilia screen. These investigations revealed no abnormality apart from a mild anaemia with haemoglobin of 12.9 g/dl and a normal mean corpuscular volume.

The patient was initially heparinised for 6 days to see if that would improve the cerebral venous blood flow, especially if the thromboses were fresh. However, upon repeating the lumbar puncture the cerebro-spinal fluid (CSF) pressure remained elevated at 33 cm H_2O. A neurosurgical opinion was sought and the patient underwent a lumbo-peritoneal shunt procedure. On discharge his visual acuity had improved to 6/9 right and 6/12 left. He has been reviewed in the ophthalmic clinic over the past 6 months and his condition is stable, although he has lost visual field in both eyes due to optic nerve damage secondary to papilloedema. We have still not found an underlying cause for the bilateral jugular vein thrombosis.

Comment

Spontaneous internal jugular vein thrombosis is a rare vascular disorder. In the pre-antibiotic era this condition was a well-known complication of head and neck infection.[1] The leading cause at present is trauma; this can include catheterisation of the internal jugular vein,[2] intravenous drug abuse[3] or head and neck surgery.[4] Other causes include malignancy,[5] thrombophilic[6] states and use of the oral contraceptive pill.[7]

Papilloedema secondary to jugular vein occlusion is rare, and to our knowledge has only been described in two previous cases. One patient developed papilloedema

due to a skull base metastasis compressing the left jugular vein.[8] The second had complex intra-cranial vascular malformations which resulted in bilateral jugular vein occlusion and left optic atrophy suspected to be due to papilloedema.[9] Our patient had long standing symptoms and interestingly presented with a reduction in visual acuity due to optic nerve dysfunction. Although his acuity improved once the intracranial hypertension was controlled, he did develop permanent optic nerve dysfunction as evidenced by visual field testing. All investigations in our patient were negative and we are unable to explain why he developed spontaneous bilateral jugular vein thrombosis. This condition is rare, but should be considered in patients with papilloedema and a negative MRI scan.

Acknowledgements

Competing interests: none.

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Sir,

Corneal endothelial deposits associated with rifabutin treatment in Crohn's disease

Abstract

Rifabutin is a broad spectrum antibiotic used to treat mycobacterial infection in HIV patients, and more recently as a treatment for Crohn's disease. Corneal endothelial deposits have been described in association with long-term rifabutin treatment in HIV patients, but have not previously been reported in patients treated for Crohn's disease. We report a case of endothelial corneal deposits associated with 3 years treatment of rifabutin in Crohn's disease. The underlying process resulting in corneal deposits is still poorly understood. The occurrence of corneal deposits in a patient treated for Crohn's disease, with no other significant past medical history, would support the theory that they could be due to the systemic toxicity of rifabutin.

Case report

A 20-year-old female was referred by her optician with corneal endothelial deposits. She had been taking rifabutin for Crohn's disease for 3 years. There was no other significant past medical history. Her visual acuity on presentation in each eye was 6/9 unaided, improving to 6/6 with glasses. She had no visual symptoms. Ophthalmological examination revealed yellowish brown corneal endothelial deposits evenly distributed circumferentially in both cornea (Figure 1). There was no history or signs suggestive of primary uveitis. Ocular examination including colour vision and visual fields were otherwise within normal limits. On review 4 months later, her deposits remained static. Following this visit, the patient discontinued her treatment with rifabutin against medical advice. On review at 4 months following discontinuation of treatment her deposits persisted and showed no change in colour or distribution. However, she remained asymptomatic. Although rifabutin has been known to accumulate in the anterior part of the lens[1] and may be associated