mains poorly defined, however, a structural weakness of the meninges may predispose patients to developing a CSF leak following trivial trauma.[6] Occasionally meningeal diverticula are identified, also attributed to structural weakness of the meninges (e.g. in Marfan's syndrome[6]). Extradural pathology is a rare cause, but a few patients with vertebral osteophytes causing CSF leak have been described.[1,2,7–9]

With advances in neuroimaging SIH has become increasingly accurately diagnosed. Of the limited patients with osteophyte-related SIH reported,[1,2,7–9] the majority are due to cervical osteophytes[2,8] and have been managed conservatively or with an epidural blood patch. To our knowledge, there are two reported patients with thoracic osteophytes causing SIH. Winter et al.[9] reported a 42-year-old woman with SIH due to a calcified T7/8 disc protrusion treated with a local autologous epidural blood patch. Binder et al.[1] reported a 55-year-old woman with SIH due to a large ventral T2/3 osteophyte treated via median sternotomy and anterior T2/3 discectomy, allowing primary dural repair.

Our patient demonstrates the difficulties that can arise in the diagnosis of SIH, particularly with presenting features of reduced level of consciousness and imaging showing bilateral subdural haematomas – an uncommon presentation of SIH and a marker of disease severity.

Unlike previously reported patients, we successfully adopted a posterior approach to excise the calcified disc responsible for the CSF leak. Whilst an anterior approach was necessary to remove the T2/3 osteophyte described by Binder et al., via a posterior approach we adequately visualized and excised the disc prolapse causing a dural defect. Whilst the defect was not directly visualised, the site of CSF leakage was identified and successfully patched.

In summary, we describe a patient with SIH presenting with reduced level of consciousness and bilateral subdural haematomas resulting from a thoracic CSF leak caused by a calcified disc breaching the dura. Due to ongoing symptoms despite conservative management, a T6/7 hemilaminectomy and costotransversectomy was performed to allow excision of a calcified thoracic disc prolapse and dural repair. This led to an early and sustained improvement in symptoms.

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Progression of choroid plexus papilloma

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ABSTRACT

Choroid plexus papillomas are rare neoplasms that arise from choroid plexus epithelium. The World Health Organization classification describes three histological grades. Grade I is choroid plexus papilloma, grade II is atypical choroid plexus papilloma and grade III is choroid plexus carcinoma. Progression between grades is rare but documented. We present two adult cases, a 53-year-old female and a 70-year-old male, who demonstrated clear interval histological progression from grade I choroid plexus papilloma to higher grades.

1. Introduction

Choroid plexus tumours are rare, comprising 0.3–0.6% of all intracranial tumours. Histologically, 80% are choroid plexus papillomas (CPP), 15% are atypical papillomas, and less than 5% are carcinomas.[1] Progression from a benign CPP to a higher grade atypical CPP or choroid plexus carcinoma (CPC) is rare.[2] We report two patients with interval histological progression.

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2. Case reports

2.1. Patient 1

A 53-year-old woman presented in 2005 with a history of headaches. Imaging showed a left lateral ventricular lesion (Fig. 1a, b). She underwent total resection via an interhemispheric transcallosal approach. Histopathology and immunohistochemistry were consistent with World Health Organization (WHO) grade I CPP (Fig. 2a). Yearly imaging between 2005 and 2009 showed no recurrence. In 2009, she re-presented with recurrent headaches. Imaging showed four new intraventricular lesions (Fig. 1c, d). She

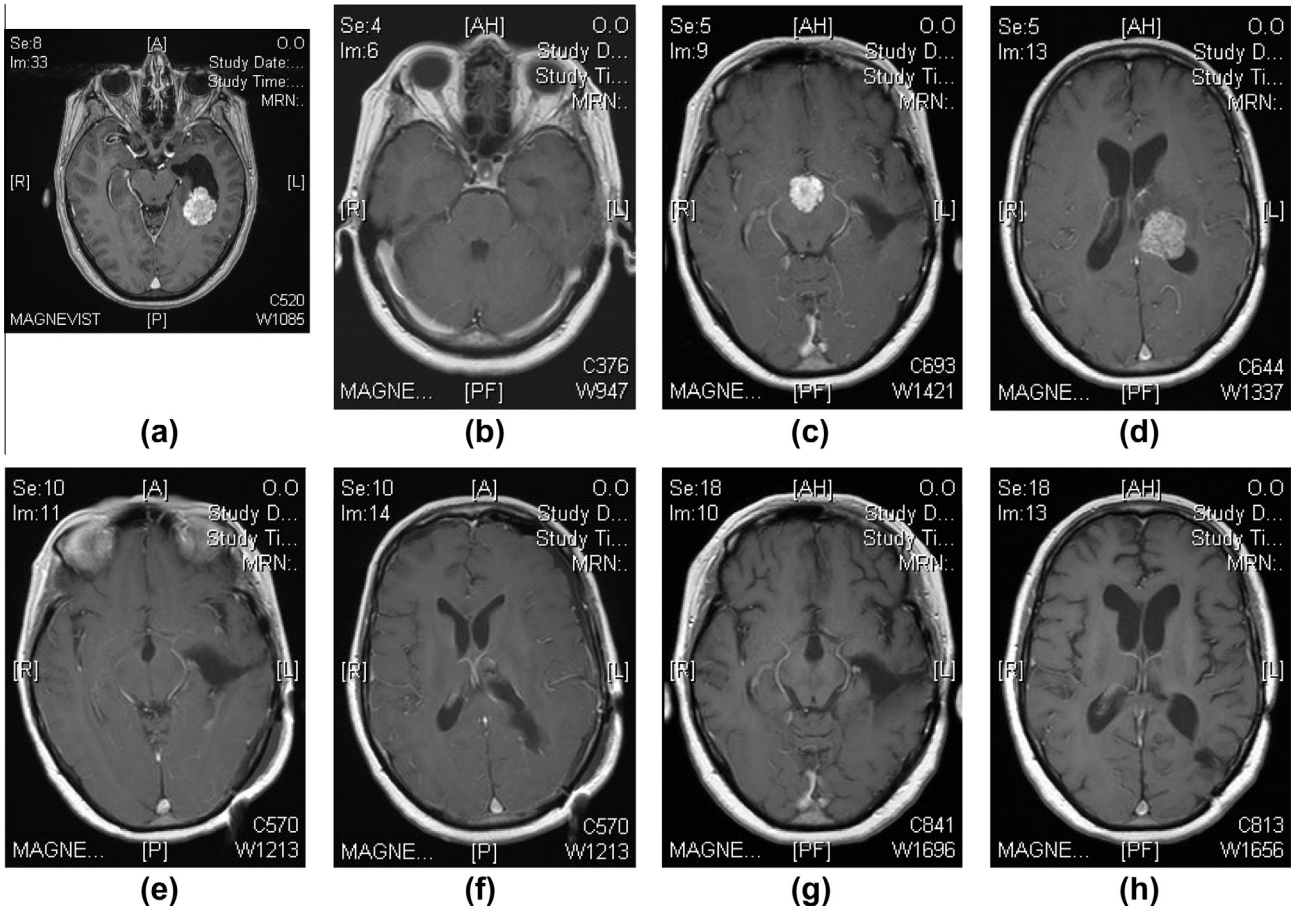

Fig. 1. T1-contrast enhanced axial MRI (a) August 2005, showing lesion in temporal horn of the left lateral ventricle. (b) February 2006. Postoperative image showing removal of the left temporal horn lesion and resolution of ventriculomegaly. (c) January 2009, showing new lesion in the third ventricle. (d) January 2009, showing new lesion in posterior horn of the left lateral ventricle. (e) February 2009, showing removal of the third ventricular lesion. (f) February 2009, showing removal of lesion in posterior horn of the left lateral ventricle. (g) November 2010, showing no interval change since the February 2009 MRI. (h) 2010, showing no interval change since the February 2009 MRI.

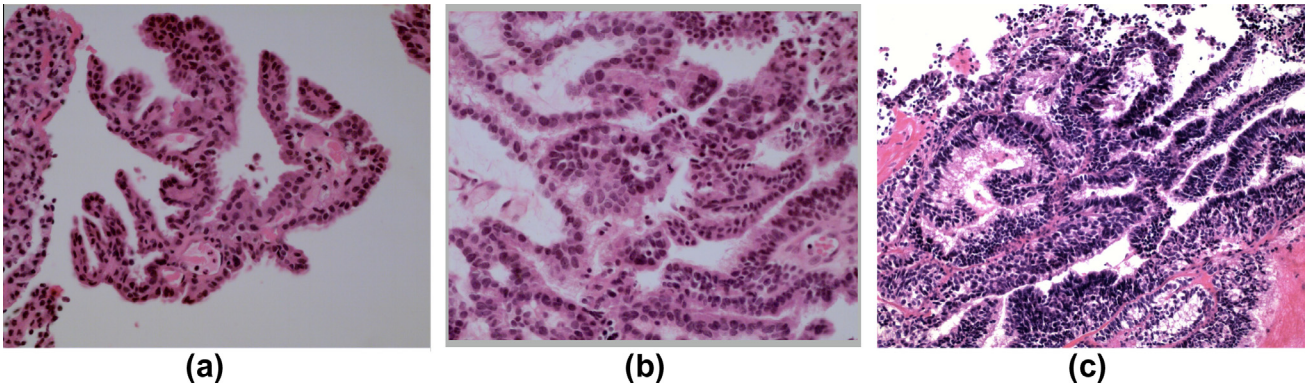

Fig. 2. (a) High power photomicrograph of choroid plexus papilloma (CPP) showing papillary architecture with bland epithelial cells (haematoxylin and eosin [H&E], original magnification × 400). (b) High power photomicrograph of atypical CPP showing papillary architecture with mitotic figures in epithelial cells. (H&E, original magnification × 400). (c) Medium power photomicrograph of choroid plexus carcinoma showing high cellularity, nuclear pleomorphism and mitotic figures. (H&E, original magnification × 200).

underwent a two stage resection involving a transcallosal and an inter-parietal approach via two separate craniotomies (Fig. 1e, f). Histopathology compared to 2005 showed an increase in nuclear pleomorphism and previously absent mitotic figures, consistent with atypical CPP, WHO grade II (Fig. 2b) and she underwent adjuvant radiotherapy. Her 12 month follow-up revealed no clinical or radiological recurrence (Fig. 1g, h). In March 2011 she re-presented with symptomatic diabetes insipidus and imaging showed a third ventricular recurrence. She underwent uncomplicated redo surgery and was discharged home with plans for adjuvant radiotherapy. Histopathology compared to 2009 showed increased nuclear hyperchromatism, mitosis and previously unseen focal necrosis consistent with CPC (Fig. 2c). The Ki67 proliferation index increased from <1% in the CPP to 12% (Fig. 3). P53 immunohisto-