Spontaneous intracranial hypotension: use of unenhanced MRI

Abstract We report a case of spontaneous intracranial hypotension diagnosed with unenhanced cranial MRI, showing laminar subdural fluid and engorgement of the hypophysial and perisellar sinuses. Cerebrospinal fluid pressure was low. MRI was normal after resolution of symptoms. Prior reports emphasise the enhancing pachymeninges seen in this syndrome. We maintain that, when subdural collections and perisellar engagement are detected on unenhanced MRI in the proper clinical setting, contrast enhancement may not be necessary for the diagnosis.

Key words Meninges · Hypotension, spontaneous intracranial · Magnetic resonance imaging

Introduction

The syndrome of spontaneous intracranial hypotension [1-7], also named low-pressure headache [8], or syndrome of orthostatic headache and diffuse pachymeningeal gadolinium enhancement [9], has a myriad of clinical presentations. It has been well known in the neurological literature for years [10], but use of MRI was first described in an abstract in 1991 [8], in which it was claimed that the hallmark was diffusely enhancing pachymeninges. Since then, other communications have appeared in the neurosurgical, neurological and radiological literature, and the most characteristic radiographic finding is indeed the enhancing pachymeninges; other imaging features have been described [2, 6, 7, 9].

To our knowledge, there has been no description of spontaneous intracranial hypotension on unenhanced MRI, although Schievink et al. [2] included a patient with bilateral subdural collections seen on unenhanced images in a series of 11 patients. We report a case in which diffuse subdural fluid and engorgement of the sella turcica and perisellar sinuses led us to the diagnosis. These abnormalities resolved completely after the patients symptoms subsided.

Case report

A 32-year-old woman was referred for MRI by her neurologist to exclude multiple sclerosis as the cause of a right abducens palsy. She also had mild nuchal headache during the day. The rest of the neurological examination was normal, and the neck was mobile. The patient reported a viral syndrome with malaise and myalgia a fortnight previously, but had no fever or other infectious symptoms. A history of a traffic accident some years before was elicited, without loss of consciousness or any other neurological symptoms.
Fig. 1  
(a) Axial T2-weighted MRI: no abnormality in cerebral parenchyma. A homogeneously high-signal subdural collection is seen along the cerebral convexities and falx. 
(b) Coronal T1-weighted image: the subdural layer over the cerebral convexities is isointense with cerebral cortex. The perisellar dural sinuses are engorged and the diaphragma sellae is bowed upwards. 
(c) Axial T2-weighted image: the tentorial and anterior temporal duramater shows a high-signal subdural collection. The orbits are normal.

Fig. 2a, b  
MRI 3 months later, when the patient was asymptomatic: on the axial T2-weighted image the subdural collections have disappeared. 
(b) The diaphragm sellae is horizontal and the perisellar sinuses have decreased in size.

On MRI (Fig. 1), extensive subdural collections 1–2 mm thick were seen over the convexities, the interhemispheric duramater and tentorium. Their signal was homogeneously high on T2- and isointense with brain cortex on T1-weighted images. The brain parenchyma was normal. The diaphragma sellae bulged up into the chiasmatic cistern, and the perisellar dural sinuses were engorged, with bowing of the lateral walls of the cavernous sinuses. The cerebellar tonsils were not distorted and lay above the foramen magnum. No haemorrhage or descent of other cranial structures was observed. The craniocervical junction was normal, although the duramater at the foramen magnum posteriorly was more prominent than usual. We advised the referring neurologist of our suspicion of intracranial hypotension, and decided not to administer contrast medium. The patient was admitted for lumbar puncture and treatment. The opening pressure was less than 60 mm H2O. The cerebrospinal fluid (CSF) was clear and had 8 WBC/mm3, mainly lymphocytes, protein 48 mg/dl, and normal glucose and electrolytes.

The patient’s headache improved in 24 h with bedrest, oral rehydration and analgesics. The abducens palsy resolved completely in the following 2–3 days. Because of the history of cranial trauma an outpatient contrast-enhanced cisternogram and skull base CT was performed, which did not show any cranial CSF leak. Clinical follow-up has been 3 months during which the patient remained asymptomatic. MRI at 3 months (Fig. 2) demonstrated complete resolution of the dural engorgement and fluid, a normal position of the diaphragma sellae and normal cavernous sinus walls.

**Discussion**

The syndrome of spontaneous intracranial hypotension is attributed to CSF leakage. Where a leak is found, it is