

Subdural haematoma upon straining

Sir: Atraumatic ("spontaneous") subdural haematomas may occur in individuals with coagulopathies, brain metastasis or with negative cerebrospinal fluid pressures after ventricular shunting procedures.^{1,2,3} A form of acute, atraumatic, often life threatening, subdural haematoma of cortical arterial origin is recognised as well as the development of subdural collections after aneurysmal rupture.^{4,5} Minor, perhaps unrecognised, cranial trauma may precipitate this condition in elderly subjects with cerebral atrophy. We describe a patient with acute spontaneous subdural haematoma apparently produced by straining and manifested by persistent severe headaches.

A 65 year old male had recently changed jobs. In his new occupation he was required to do unaccustomed heavy lifting. His past medical history was negative and he only had rare headaches relieved by common analgesics. There was no history of head trauma. Two weeks prior to his admission to hospital, he developed acute right sided headaches and burning dysaesthesias immediately following lifting of a heavy weight. He was seen initially by his family physician, who found him mildly hypertensive for which he prescribed accordingly. The patient's headache worsened as the days passed. Preliminary neurological examination, full blood count, blood chemistry, coagulation profile, chest radiograph and electrocardiogram were normal. Blood pressure was 160/100 mmHg. Computed tomography (CT) of the brain revealed a large right isodense subdural haematoma that was subsequently drained through multiple burr holes and craniectomy. No arterial cortical source of active bleeding was identified at surgery. He had a prompt and uneventful recovery and was released from hospital 9 days after admission.

This case is unusual because the patient had no apparent predisposing factor for the development of a spontaneous subdural haematoma. We believe his illness was unrelated to his hypertension since it was mild, he had no prior history, and the location of the haematoma was not typical of hyperactive bleeds (basal ganglionic or capsular). His symptoms mimicked "benign exertional headache" or "effort headache", a syndrome present in migraineurs, and by definition of good prognosis.⁶ We suspect that rupture of bridging dural veins while performing the Valsalva manoeuvre on heavy lifting caused the bleed. Individuals with a history of acute headaches upon straining are candidates for neurodiagnostic

investigation, when the exceptional presence of atraumatically-formed subdural haematomas may be revealed.

DANIEL E JACOME
GONZALO F YANEZ

Department of Medicine,
Palmetto General Hospital and
Hialeah Hospital, and
Department of Neurology,
University of Miami School of Medicine,
Miami, Florida 33016, USA

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Rapid development of occlusion hydrocephalus by intraventricular fat possibly derived from a ruptured dermoid cyst

Sir: A previously healthy 46 year old woman was admitted to our hospital in December 1986 with a 5 day history of severe headaches, nausea and vomiting. Cranial CT had disclosed enlarged lateral and third ventricles and a low attenuation area in the left frontal horn. On admission, blood pressure was 120/70 mm Hg and pulse rate sometimes dropped to 30 beats/minute. Neurological examination was normal. The patient was alert, but slightly disoriented. As the occasionally slow heart rate suggested raised intracranial pressure (ICP), an external ventricular drainage system was inserted immediately. Continuous measuring of ICP demonstrated multiple B waves and rapidly increasing ICP values (up to 45 mm Hg) when the drainage system was closed, as well as signs of obstruction of the aqueduct of Sylvius on a spinal infusion test. Lumbar and ventricular

CSF analysis gave normal protein values and no pleocytosis. In two samples of ventricular CSF there were macrophages containing phagocytosed lipid material. As normal CSF circulation could not be restored a ventriculoperitoneal shunt (Raimondi unisystem) was inserted 3 weeks after admission. Eleven months later the patient had completely recovered. CT showed normal size ventricles with unchanged intraventricular and intracisternal fat deposits.

CT demonstrated supratentorial ventricular dilatation and dispersed hypodense droplets in the subarachnoid space of the quadrigeminal cistern. Additionally, there was a fluid level in the anterior horn of the left lateral ventricle with negative absorption values of -60 Hounsfield units (HU), clearly below those of CSF (3 to 14 HU), indicating intraventricular fatty material, which was freely movable when the patient changed position. MR imaging (performed by Dr W Keil, Würzburg; Siemens Magnetom 1.0T) confirmed the CT findings with fat deposits close to the upper vermis and a band-like accumulation of fat sharply outlining the roof of the left lateral ventricle (fig).

The occurrence of free, fluctuating fat within the ventricles or the subarachnoid space is a rare finding. The diagnosis of this material was greatly assisted by density measurement during cranial CT. In the few cases with intraventricular or subarachnoid fat reported so far, the material originated from ruptured dysontogenetic tumours such as epidermoid¹ or dermoid cysts² and teratomas.³ Owing to their chemical composition, absorption values of epidermoid



Fig Cranial MR image (sagittal section) delineating hypointense material in the upper vermis and a band-like accumulation of similar signal intensity at the roof of the left lateral ventricle (arrows).