

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.

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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 unit-system) 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).

cysts are similar to those of CSF. Dermoid cysts, however, exhibit negative absorption values markedly below CSF, spread along the base of the skull and tend to be located in the midline, whereas epidermoid cysts usually occur in lateral positions.⁴ Intraventricular locations in both types of cyst have been noted⁷ sometimes leading to ventricular obstruction.⁵ Both tumours may rupture spilling fatty components into the subarachnoid space or the ventricular system.² Because of their negative absorption values, fat droplets are easily detected by CT, although initially they may be misinterpreted as intraventricular air.² Clinical symptoms include seizures, raised ICP or aseptic meningitis¹ which may occur after spontaneous rupture or postoperatively.^{2,5} Chambers⁵ described a patient with communicating hydrocephalus requiring a shunt. In contrast to our patient, a solid tumour mass was identified by either CT, encephalography or histological investigation in all cases previously reported. In our case, we suggest that the intraventricular fat was derived from a ruptured dermoid cyst because of the negative absorption values (-60HU) and the location of residual fat deposits near the midline (upper quadrigeminal cistern). The occlusion of the aqueduct of Sylvius was confirmed in the sagittal MR images. We suggest that it was due to a granulomatous reaction caused by the lipid material in the neighbourhood of the aqueduct. The clinical symptoms of our patient included severe headaches, vomiting and, as a sign of raised intracranial pressure, a slowed heart rate. The rapid development of symptoms required a temporary external drainage of the lateral ventricles and followed by implantation of a ventriculo-peritoneal shunt. To our knowledge, this is the first case reported where an occlusion hydrocephalus was caused by free intraventricular fatty material possibly derived from a ruptured dermoid cyst.

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Cerebral venous thrombosis in hereditary protein C deficiency

Sir: Protein C is a vitamin K-dependent serine protease which together with its cofactor protein S and antithrombin III is probably the most important inhibitor of plasma coagulation.¹⁻³ It is a plasma zymogen that is activated by thrombin coupled with an endothelial cofactor named thrombomodulin and inactivated by a specific plasma protease inhibitor. The anticoagulant effects of protein C are achieved through cleavage of factors Va and VIIIa of the intrinsic pathway. Clot lysis results from interaction of protein C with the inhibitor of plasminogen activator.⁴

Homozygous protein C deficiency has low or even undetectable antigenic levels and is mostly lethal through massive venous thrombosis during the neonatal period.^{5,6} The heterozygous form is an autosomal dominant disorder with an incidence of 1 per 16,000 individuals. It usually presents during early adulthood as recurrent superficial and deep thrombosis of pelvic and leg veins sometimes with subsequent pulmonary embolism in several members of a given family.⁷

Diagnosis is made by repeated measurements of decreased protein C levels, while all other vitamin K-dependent factors are normal and an acquired protein C deficiency is excluded.⁸ The latter occurs in liver diseases, where protein C can already be lowered yet with a normal thromboplastin time. An acquired deficiency is also observed in disseminated intra-vascular coagulation, during coumarin treatment, and in the postoperative period.⁹

We observed a 30 year old housewife with common migraine since age 18. Since then she had been smoking five cigarettes a day

and had been regularly taking an oral contraceptive. At age 25 she had suffered from two episodes of pulmonary embolism which had been treated by coumarin for several months.

In March 1986 she had sudden prickling headache bitemporally, recurrent vomiting and blurred vision. Two days later numbness of the left arm occurred, and memory deficits were noted.

On admission the obese patient had left homonymous hemianopia, a weak left arm, and a moderate paresis of the left leg. Deep tendon reflexes were brisk, moderately elevated on the left. The patient had a slight meningism, she was drowsy with diminution of concentration and attention, but fully orientated. Routine blood examination revealed a slightly elevated activity of Gamma-GT (53 U/L). Serologic search for infectious and autoimmune disorder was unproductive. Cerebrospinal fluid was clear with a protein content of 0.38 g/l. There were two cells/mm³ in the cell count; cell sedimentation showed few leucocytes and monocytes, but no erythrocytes or lymphocytes. CT on admission revealed abnormally increased density along the superior and the straight sinus which showed a slight enhancement after contrast medium application. There was no empty triangle sign. Patchy hyperdense areas in both parietal areas and right occipitally were interpreted as either congested blood vessels or petechial haemorrhages. Subsequent carotid angiography revealed lack of filling of the sinuses mentioned above with dilatation of some collateral cortical veins, thus confirming sinovenous thrombosis.

Intravenous heparin treatment was started at a daily dose of 24,000 U. Additionally sorbitol and dexamethason were given.

In spite of this treatment somnolence increased, vision in the previously intact right hemianopic fields blurred, there was an intermittently dilated right pupil, and a choked right optic disc. Two days later short-lasting focal motor seizures of distal left arm were observed. EEG showed a moderate diffuse abnormality and a delta focus in the right temporo-occipital region with recurrent focal sharp-slow-waves.

CT four days after admission revealed a patchy haemorrhagic infarction in the right parieto-occipital region with some small haemorrhages in both parietal areas. The latter were thought to be due to anticoagulant medication, and heparin treatment was immediately stopped. From the sixth day on the patient's condition gradually improved. She intermittently reported formed hallucinations in the left