(A and B) Axial CT when the patient was 8 years old, showing a small arachnoid cyst in the left middle fossa. (C and D) Axial CT when the patient was 18 years old, showing that the cyst in the left middle fossa had grown considerably, now reaching well above the sphenoidal wing.

discovered that he had a relatively small arachnoid cyst in the left temporal fossa. The cyst did not reach above the sphenoidal wing or the pyramid (figure A and B). As we at that time were not yet aware of the cognitive impairment caused by most temporal cysts,¹ we refrained from surgical decompression.

Ten years later, he was referred to us again, now complaining of a strong, episodic, frontal headache that had developed over the past year. The strength of the headache varied with body position; it increased when he was leaning forward, and was relieved when he was resting on his back. New CT (figure C and D) showed that the cyst had grown considerably. At the age of 8, the cyst volume was 6.5 ml, as calculated from the films. During the next 10 years, the cyst volume nearly tripled, measuring 18.3 ml when he was 18 years of age.

The total intracranial volume was calculated to be 1485 ml on both occasions, with no skull growth occurring between the ages of 8 and 18 years. Also the size of the two middle cranial fossae remained practically

unchanged during the same period. The size of each fossa was calculated from the films, using a standardised technique based on the bony landmarks surrounding it. The right fossa was calculated to contain 19 ml on both occasions, and the somewhat larger left fossa showed a non-insignificant increase, from 24 ml to 24.7 ml.

The patient was operated on under general anaesthesia, with craniotomy, removal of cyst membranes, and fenestration to the basal cisterns. Postoperatively, the left temporal lobe expanded and completely filled the space that had been occupied by the cyst. Thus, the cyst disappeared, and so did his headache. He has now been followed up for 23 months after surgery, without reproduction of the cyst or the headache.

The patient presented here displayed a considerable growth of a temporal arachnoid cyst as seen on two scans 10 years apart. We do not know exactly when the growth took place, but it is tempting to correlate it with the increased symptoms (mainly headache) evolving over the past year before the second CT. In retrospect it is possible, or

even probable, that the patient's headache at the age of 8 also was caused by the cyst, and not a sinusitis. The increase in cystic volume must have occurred after the neurocranium had become relatively rigid, as the patient was already 8 years old before the documented growth started, and the total intracranial volume as well as the volume of the left middle fossa remained constant over the 10 years. Therefore, the cyst growth must have occurred at the expense of brain tissue or the CSF compartment, and cannot be ascribed to a soft, yielding skull allowing an unrestrained growth of the underlying cyst, as we may occasionally see in infants, and as was reported by Kumagai *et al.*³

Thus, the present finding may be taken as an indication that temporal arachnoid cysts are not merely passive accumulations of fluid, and that the cystic growth in this patient must have been caused by an increased intracystic pressure that was sufficient to widen the sylvian fissure, and to dislocate the temporal lobe. In previous studies, it has been shown that this effect on the temporal lobe is sufficient to cause cognitive dysfunction, with postoperative improvement when the cyst is surgically decompressed.¹⁻⁵ In our opinion, such findings alone may constitute a sufficient indication for surgery, but only if the complication rate can be kept at a negligible level.

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Pituitary apoplexy presenting as massive subarachnoid haemorrhage

Pituitary apoplexy is an uncommon, but well recognised clinical syndrome, which usually results from ischaemic or haemorrhagic necrosis of a pituitary adenoma.¹ It is characterised by the abrupt onset of severe headache, visual impairment, ophthalmoplegia and often, a deteriorating level of consciousness.² Signs of meningeal irritation are frequent accompaniments, which may confuse the clinical picture with that of aneurysmal subarachnoid haemorrhage (SAH). We report a case of massive SAH caused by pituitary apoplexy and present the CT and MRI findings.

A 72 year old woman, with nothing in her history to suggest pituitary dysfunction, presented with the abrupt onset of severe headache, vomiting, and gradually deteriorating level of consciousness. A CT examination of the brain without contrast enhancement

showed an extensive basal subarachnoid haemorrhage that had diffused into both sylvian fissures and into the sulci over the convexity of the cerebral hemispheres. In addition there was a rounded heterogeneous density mass in the suprasellar cistern (fig 1 A). After the administration of contrast material the lesion displayed heterogeneous central enhancement (fig 1 B). A ruptured anterior communicating aneurysm was suspected but was excluded by normal cerebral angiography.

Brain MRI confirmed haemorrhage within a pituitary tumour (fig 2 A). It could also be seen that the haemorrhage had ruptured through a defect in the tumour capsule into the subarachnoid space (fig 2 B). Despite the administration of 100 mg hydrocortisone intravenously every 6 hours and cardiopulmonary support her condition progressively worsened and she died 3 days after admission.

The clinical syndrome of pituitary apoplexy evolves within hours to days.² The symptoms vary from mild to severe and can progress rapidly to coma and death. Because many patients are unaware that they harbour a pituitary tumour, pituitary apoplexy is often unrecognised at presentation. In pituitary apoplexy, blood and necrotic tumour tissue are enclosed and compressed within the confined space of the sella turcica, a region that is in close anatomical proximity to the basal cisterns. When the pressure gradient within the sella exceeds the resistance of the surrounding structures, blood is expelled into the subarachnoid space producing a clinical picture that may be indistinguishable from aneurysmal SAH.³ Thus pituitary apoplexy must be included in the differential diagnosis of "angiographically negative" SAH.⁴ Brain CT is the modality of choice in the initial investigation of SAH. It will show the spread and severity of the haemorrhage within the

subarachnoid space, as well as any extension into the brain.¹ When pituitary apoplexy is the cause, CT will usually show the pituitary tumour as well as any recent haemorrhage. Brain MRI is superior to CT in identifying the tumour as well as the associated haemorrhage or infarction. In the case presented here, the enhanced MRI also displayed the defect in the tumour capsule which represented the site of rupture. The management of pituitary apoplexy includes the immediate administration of high dose corticosteroids to combat adrenal insufficiency. When there is rapid deterioration of vision or a progressively worsening level of consciousness, urgent surgery in the form of trans-sphenoidal decompression should be carried out.

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Figure 1 (A) Cranial CT without contrast, showing a heterogeneous but mostly high density mass in the suprasellar cistern (arrowheads). There is also extensive subarachnoid haemorrhage. (B) Cranial CT with contrast, showing central enhancement within a heterogenous high density mass (arrowheads).

Figure 2 (A) Coronal T1 weighted MRI, showing suprasellar extension of a sellar mass. The area of high intensity signal (arrow) represents methaemoglobin within clotted blood. Note also the hyperintense tumour rim. (B) Contrast enhanced sagittal MRI T1 weighted image, showing that the bulk of the mass is isointense with brain. There is a defect posteriorly in the superior aspect of the capsule representing the site of rupture (arrow).

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Early detection of non-compliance in Wilson's disease by consecutive copper determination in cerebrospinal fluid

A 41 year old technical employee was diagnosed with Wilson's disease in 1982, 2 years after onset of dysarthria, diplopia, visual deficits, ataxia, and concentration deficits. The patient improved rapidly with *D*-penicillamine and a copper free diet, and has since returned to normal neurological function. Penicillamine was stopped in 1989 and he changed his diet from copper free to a normal diet in 1987. In 1990 he was put on 800 mg zinc a day. During the past 17 years he underwent 20 determinations of copper concentration in CSF for follow up of treatment (figure) as copper concentrations in CSF can be used as an indicator of brain copper concentration in the cerebral manifestation of Wilson's disease.¹⁻⁵

Copper was measured by flameless atomic absorption (Perkin Elmer, HGA 500, Überlingen, Germany). The main resonance line was 324.7 nm with deuterium background compensation and argon as a protective gas; the sample volume was 20 µl using an autosampling system. The CSF was measured undiluted. The patient was now again routinely admitted for monitoring of the efficiency of the treatment. He was free of complaints and the neurological examination as well as a neuropsychological test battery and visually evoked potentials were normal. He explicitly denied any change in diet or drug therapy (zinc, 800 mg a day). We found, however, an almost threefold increase of his CSF copper concentration compared with the previous values (figure). Serum