Figure 2 Anatomical study. Top: T1 weighted MR axial images of the distal right upper arm across the lesional site; (A): proximal; (B): intermediate; (C): distal section. Left: surgical findings (D); brachialis muscle fibres covering the median nerve have been sectioned; note the anomalous deep course of the median nerve trunk (small white arrow) apart from the vascular bundle (large white arrow) and through the brachialis muscle belly (black arrow). See text for details.

lumbrical or thenar muscles⁶; similar cases may involve the ulnar and the superficial radial nerves.

The pathophysiology of this type of lesion is mainly mechanical: a repeated and prolonged compression may develop either directly by the contracting muscle itself or by fixation against more rigid osteoligamentous tissues; the movements performed by our patient consisted in repeated and alternated elbow flexions and extensions under loading: in this setting the contracting muscle could also have reduced or blocked the longitudinal sliding of the nerve along its bed, with subsequent stretching and friction of the nerve trunk.⁷

In conclusion, this case emphasises that rare anatomical variations should be considered in the differential diagnosis of proximal median mononeuropathies at uncommon lesional sites.

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Documented growth of a temporal arachnoid cyst

Most arachnoid cysts are probably present at birth, or develop soon after. Once they are formed, they are thought to remain stable, apparently in some kind of equilibrium with the rest of the intracranial space. Not infrequently, cysts have been reported to disappear spontaneously (for references see Wester and Hugdahl¹) and a minority may grow with increasing age, although rather slowly.² Occasionally, cysts in infants have been reported to grow to a substantial size. Kumagai *et al.*³ reported on a newborn boy who developed a temporal fossa cyst between the age of 2 and 4 months, which later increased considerably before it was removed when the patient was 8 months old.

Except for the very smallest, most arachnoid cysts display radiological signs indicating an increased intracystic or intracranial

pressure. For example, during infancy and early childhood, cysts may influence the shaping of the adjacent skull bone, in the case of middle fossa cysts resulting in an enlarged fossa, often with a bulging of the overlying bone. Moreover, middle fossa cysts may also dislocate the temporal lobe posteriorly, and larger cysts regularly cause a midline shift, thus indicating expansive forces.

This notion of raised intracystic and intracranial pressures is supported by the common intraoperative finding of a cyst wall bulging out of the dural incision. However, the relative paucity of associated symptoms, the moderate radiological displacement, the absence of perifocal oedema in the adjacent brain parenchyma, and, finally, the total intraoperative impression, all indicate that the pressure is only moderately increased.

It is a common clinical experience that the symptoms caused by an arachnoid cyst may first present after many years, and that the symptoms also may vary over time. Why this is so is not known, but it is tempting to suggest variations in the intracystic pressure as one explanation.

The demonstration of a cyst enlargement over time may be of some importance, as it would provide additional evidence of an increased intracystic pressure, and that arachnoid cysts not merely represent passive accumulations of fluid as implied by the term "the temporal lobe agenesis syndrome".⁴ With the exceptions mentioned above, results from very few patients have been published that show growth of an arachnoid cyst. We hereby report on one such patient.

An 18 year old man had CT at the age of 8 because of complaints thought to be caused by a sinusitis (a moderate transitory headache) and no other symptoms. It was then

(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