Subarachnoid haemorrhage and acoustic neuroma

Sir: We wish to add our two cases of subarachnoid haemorrhage (SAH) in association with acoustic neuroma to the literature in which we have located only two proven case reports.

These two cases represent the only two with SAH in a total of seventy acoustic tumours operated on by one of us (RHS).

Case 1, a labourer (AK) aged 37 years who presented with a coma-producing SAH from which he made a full recovery. The four vessel angiographic study was normal, but on discharge home he was suspected of being a little deaf, to which he admitted on direct questioning. Deafness did not progress but during routine follow-up two years later he presented with ataxia and visual failure, features which led to renewed investigations and the discovery of a large mass in the right cerebello-pontine (CP) angle. Surgical removal was undertaken but because of tumour size and vascularity, this was effected in two stages. The acoustic origin of the tumour was proven histologically. The patient returned to his former employment.

The second case (RM) aged 66 years presented with deafness, ataxia and sensory change over the left trigeminal territory. Relevant investigations showed a large left CP angle tumour but operation was refused by the patient until he was re-admitted ten months later with a proven subarachnoid haemorrhage and an increase in his signs which were now accompanied by a left facial weakness. The CT scan indicated blood in the region of the tumour. At operation it was noteworthy that during exposure of the bone, placement of the self-retaining retractors induced bouts of bradycardia, these bouts reverting to sinus rhythm once the retractor was released. This occurred several times and did not correspond with any fluctuation of the ICP which was monitored throughout. The tumour was covered in recent blood clot which filled the CP angle cistern. There was also older looking clot within the tumour. Postoperative recovery was uneventful and he returned to his former employment. Histological proof of acoustic origin was obtained.

In adding these two acoustic tumours with associated SAH to the literature which contains numerous references to other neoplasms associated with SAH we wish to indicate certain features of interest and also discuss some comments which we have encountered. As in Fine's case, both our tumours were large (RM 3 cm, AK "large", size not recorded). Curiously, hearing loss was non-progressive in the second case and was not the first symptom, whilst the first patient only admitted to hearing loss when questioned directly. In both cases, neurological progression was very rapid once deterioration began.

The first case presented as a "typical SAH" against a background of good health; and when four vessel angiographic study did not reveal a cause for the haemorrhage, routine follow-up was carried out. This concurred with our routine policy for all patients presenting with SAH and having normal angiograms. These events clearly highlight the limitations of vertebral angiography in cases of laterally placed posterior fossa tumours. Accordingly, in our opinion, Fine's statement that "angiography is the final means of diagnosis" is incorrect. Verbiest in his paper on AVM in the posterior fossa intimated that haemorrhage excluded tumour from the diagnostic possibilities in two of his patients with eighth nerve dysfunction and ataxia. Such does not accord with our experience. Indeed, we would submit that a normal four vessel study in a patient with SAH who may also be a little deaf should justify at least plain films of the internal acoustic meati, and isotope scanning.

We would finally like to point out that we consider it to be of practical importance when blood clot is seen in the cerebello-pontine angle on EMI scan, since it may carry implications for retractor manipulation during surgery, as in our second case. Probably the total size of the mass, that is tumour plus clot is the important factor here. Such a mass by producing congestion at the foramen magnum would displace CSF from and then completely occupy the cisterna magna. The loss of the cushioning effect of the CSF would render the medulla vulnerable to transmitted pressure changes. Such changes could be induced by skull movements and produced by adjustments to the self-retaining retractors.

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