syndrome with Guillain-Barré features (limb weakness and distal paresthesias), and during a relapse one year later, exhibited purely Miller Fisher features.

Our patient had a classic manifestation of Miller Fisher syndrome with ataxia, areflexia and ophthalmoplegia followed by a recurring polyneuropathy eight weeks later. We believe this is the first reported case of this association. Most often the course of Miller Fisher syndrome is one of spontaneous recovery. This patient reinforces the need for continued observation of these patients, even after initial improvement of their neurological deficit.

DR KAISER-SMITH
Department of Medicine,
Section of General Internal Medicine,
University of Medicine and Dentistry of New Jersey,
School of Osteopathic Medicine,
301 S. Central Plaza,
Suite 3100,
Stratford, NJ 08084
DR BARONE
Department of Medicine,
Section of Neurology,
University of Medicine and Dentistry of New Jersey,
School of Osteopathic Medicine
PO Box 330
Voorhees, NJ 08043, USA

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Spontaneous haemorrhage in craniopharyngioma

Sir: While chemical meningitis caused by the extravasation of oily cystic fluid from a ruptured craniopharyngioma is known, it usually occurs after surgery. Spontaneous rupture is more rare[1] and proven haemorrhage has not been documented in this tumour. We describe a patient who presented with subarachnoid haemorrhage secondary to haemorrhage in a craniopharyngioma.

A 59 year old housewife, who had been suffering from sciatic pain and mild headache for two months developed severe headache and nausea on the day before admission. She had no contributory previous medical or family history. On examination the patient was alert, cooperative but was in some distress. The detected abnormalities were mild fever and pain bilaterally upon straight leg raising. Her neck was supple.

Lumbar puncture yielded deeply xanthochromic, turbid cerebrospinal fluid (CSF) with the opening pressure of 22 cm H_2O. Other than the uncounted erythrocytes, the cell number of the CSF was 250 per mm^3 with 59% polymorphonuclear leukocytes, 38% lymphocytes, and 3% monocytoid cells; the protein was 125 mg/dl, and the glucose 38 mg/dl. There were no detectable cholesterol crystals. The CSF cytology was negative. CT revealed a high density suprasellar mass (fig A) which was diffusely enhanced by contrast medium. Plain radiographs of the skull demonstrated calcification in the suprasellar region corresponding to the mass in CT. The cerebral angiogram was unremarkable. The tentative diagnosis was a haemorrhage either into a craniopharyngioma or a pituitary adenoma. Laboratory examination of the peripheral blood was unremarkable except for the mildly elevated prolactin level.

Repeat CT without contrast medium revealed a gradual decrease in the density of the juxtasellar portion of the mass (fig B).

Two months after admission, subtotal removal of the suprasellar tumour was performed via the right fronto-temporal approach. The arachnoid membrane was brownish and thick. Behind the optic chiasm was a partly dark coloured, partly calcified mass which did not yield any fluid. Histological examination of the specimen demonstrated typical craniopharyngioma consisting of islands of epidermoid tissue in the adamanitinomatous pattern, squamous cells, keratinisation, calcification, and microcyst formation. In addition, there were blood clots and numerous haemosiderin-laden macrophages. Some of the blood vessel walls in the tumour and the surrounding connective tissue stroma showed hyaline or fibrous thickening. The postoperative course was uneventful, except for transient diabetes insipidus.

The clinical manifestations of craniopharyngioma include visual field deficit, hypopituitarism, increased intracranial pressure caused by compression of the surrounding structures and chemical meningitis due to extravasation of cystic fluid. However, haemorrhage has not been confidently proved to occur in craniopharyngioma as it has, rarely, in pituitary adenoma.[2]

As in the present case, the CT density of some of craniopharyngiomas has been reported to vary with time.[3,4] In such a case the CT change was attributed to the protein concentration, not to haemorrhage, in the cyst.[3] In another case haemosiderin deposition was found in the connective tissue stroma of the cyst wall. The authors suggested haemorrhage in the tumour although there had been no sudden episode of clinical deterioration or bloody cyst fluid at surgery.[4] The CSF and preoperative CT findings of

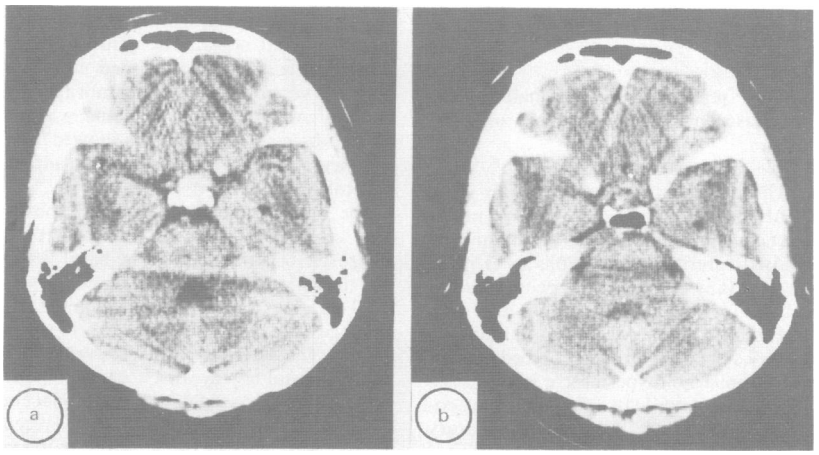

Fig *CT without contrast medium, showing a juxtasellar high density mass (A) 1 day and (B) 42 days after the onset of symptom.*

our patient indicated a haemorrhagic tumour which was confirmed at surgery. The histological findings suggest that some of the blood vessel walls in the tumour and connective tissue stroma underwent degenerative changes and ruptured. We suggest that craniopharyngioma be added in the differential diagnosis of parasellar haemorrhagic masses.

TORU YAMAMOTO*
SHUNICHI YONEDA†
NOBORU FUNATSU†
*Department of Neurology, Kitano Hospital and Neurological Center, Tazuke-Kofukai Medical Research Institute,**
Nipponbashi Neurosurgical Hospital,† Osaka, Japan.

Address for correspondence: Toru Yamamoto, M.D., Department of Neurology, Kitano Hospital and Neurological Center, Tazuke-Kofukai Medical Research Institute, 13-3 Kamiyama-cho, Kita-ku, Osaka 530, Japan.

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Accepted 1 January 1989

Normal pressure hydrocephalus and cerebrovascular disease: findings of post-mortem

Sir: The clinical distinction between some patients with Alzheimer's disease, multi-infarct dementia and normal (or intermittently raised) pressure hydrocephalus is often difficult. Many demented old people show a combination of changes typical both of Alzheimer's disease and cerebral infarcts at necropsy, hence the term "combined dementia".[1,2] Similarly, hypertensive cerebrovascular disease and hydrocephalus may coexist but there is a paucity of confirmatory neuropathological data in the literature.[3,4] We report the clinical and pathological features in a patient whose dementia initially resolved following insertion of a shunt for hydrocephalus but who subsequently deteriorated despite a functioning shunt. At necropsy he was found to have multiple small infarcts, particularly in the white matter of the cerebral hemispheres, in addition to periventricular gliosis.

This 66 year old man with mild hypertension presented in 1981 with a 2 year history of clumsiness, falling and memory loss. CT revealed enlargement of all four ventricles with no periventricular lucency. Intraventricular pressure monitoring showed B waves for 43% of the 21 hour period of measurement. Following insertion of a ventriculo-atrial shunt with a low pressure valve in May 1981 the patient improved sufficiently to become independent when walking and managing his financial affairs. CT at this time showed that the ventricles had returned to normal size. He began to deteriorate in 1983 when repeated CT again showed normal sized ventricles. He continued to deteriorate. By 1984 CT showed recurrent ventricular enlargement, now with periventricular lucency. However an isotope shuntogram confirmed that not only was the shunt patent but that there was a normal clearance of isotope through it. The patient subsequently died following a protracted illness that included removal of a small right frontal intracerebral haematoma, and repeated chest and urinary tract infections.

At necropsy (October 1984) the heart was normal in weight (320 g), with no left ventricular enlargement. Moderate atherosclerosis was seen in the coronary arteries with no myocardial infarction. There was thrombus around the distal end of the shunt catheter but the cateheter itself was patent. Atherosclerosis was minimal in the thoracic aorta but ulcerated with mural thrombosis in the abdominal aorta. The carotid arteries showed little atherosclerosis and no ulceration. The vertebral arteries were normal. The kidneys contained multiple acute pyelonephritic abscesses and the lungs showed bronchopneumonia. There was a small adrenal cortical adenoma.

The brain weighted 1355 g and showed little cerebral atrophy. A minor degree of atherosclerosis was seen in the basal vessels and the right vertebral artery was hypoplastic. There was no obvious thickening of the meninges and the foramina of Luschka were patent. The lateral ventricles were widely dilated. Histology revealed no senile plaques or neurofibrillary tangles in the hippocampus or frontal cortex. Multiple old and recent infarcts were present in the white matter of the left frontal lobe accompanied by widespread moderately severe arteriosclerosis in the blood vessels in the same area. There were small numbers of macrophages containing iron pigment in the perivascular spaces. État criblé was present. An old cavity was seen in the right frontal lobe with surrounding gliosis where the haematoma had been removed. Small numbers of old haemorrhages were seen in the right thalamus well posterior to the damaged frontal lobe. A moderate amount of periventricular gliosis was seen which was probably due to the hydrocephalus. There was a mild degree of degeneration of both lateral corticospinal tracts in the spinal cord, more on the right than the left. There was also some mild degeneration of the gracile tracts.

When confronted with a patient with dementia, gait dyspraxia and incontinence we suggest that it is more realistic to look for a remediable hydrocephalic component to the illness than to consider that patients must have either Alzheimer's disease, multi-infarct dementia or normal pressure hydrocephalus alone. This concept certainly helps when counselling both the patient and his relatives. It is an interesting question whether successful treatment of such hydrocephalus will retard progression of the accompanying damage due to cerebrovascular disease.

DR H NEWTON
JD PICKARD
RO WELLER
Southampton General Hospital, Southampton SO9 4XY, UK

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Hypergeusia as the presenting symptom of a posterior fossa lesion

Sir: Hypergeusia (increased sensitivity of taste) is rarely a manifestation of