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 cranioopharyngioma be added in the differential diagnosis of paranasal 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.

References

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 Similarly, hypertensive cerebrovascular disease and hydrocephalus may co-exist but there is a paucity of confirmatory neuropathological data in the literature.2 3

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 catheter itself was patent. Atherosclerosis was minimal in the thoracic aorta but ulcerated with mural thrombosis in the abdominal aorta. The carotid arteries showed slight 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. Etat 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 cortical and tectospinal 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 a hydrocephalus will retard progression of the accompanying damage due to cerebrovascular disease.

References

Hypergeusia as the presenting symptom of posterior fossa lesion

SIR: Hypergeusia (increased sensitivity of the taste) is rarely a manifestation of...