Fluctuating dementia and rhinorrhoea

SIR,

CSF rhinorrhoea is a common condition which is usually due to trauma, congenital malformation, tumour, or spontaneous. Non-traumatic CSF rhinorrhoea is rare; there have been previous case reports of this secondary to colloid cyst of the third ventricle.1 Aetiological such non-traumatic CSF leaks are classified in the high pressure group described by Ommaya.2 It has been proposed that the CSF leak acts as a safety valve, preventing the development of raised intracranial pressure, the leak allowing the tumour to grow while remaining clinically silent.

A 79-year-old woman, resident at a local nursing home, was admitted via Neurology outpatients. She was normally able to walk, incontinent of urine, and fully dependent on nursing staff for normal daily activities. It was noted that after a minor fall she developed a profuse watery nasal discharge following which, over a period of weeks, she became rational, lucid, able to read and write, and regained urinary continence. She would then slowly revert to her former self, until another bout of rhinorrhoea, whence she would again improve, the above cycle of events repeating itself several times before she was seen in our out-patients clinic. On examination she was wheelchair-bound, had an indwelling urinary catheter, but could move all limbs spontaneously. Reflexes were brisk but symmetrical, with bilateral extensor plantar responses. Neuropsychological assessment revealed severely impaired cognitive function, with a WAIS-R IQ of 64 (predicted 98), and gross impairment of memory function such that the memory quotient could not be calculated.

A brain CT scan, with contrast (figure), demonstrated a benign looking mass in the fourth ventricle, causing considerable hydrocephalus. A ventriculo-peritoneal shunt was inserted, following which she became self-caring, and regained urinary continence. A repeat scan one week post-operatively showed the shunt in the right lateral ventricle, ventricles being of normal size. She was able to mobilise with the help of a Zimmer frame, and discharged back to her nursing home. Two months later repeat neuropsychological yielded a verbal IQ of 91, not significantly below predicted. Her memory quotient of 114 was higher than her predicted IQ, scores on the majority of subtests falling above the mean for the age group. She continues to make excellent progress and is now walking unaided one year post-discharge.

Our patient was unfit for a major operative procedure and thus insertion provided a satisfactory outcome. This case illustrates the clinical importance of cerebrospinal rhinorrhoea as a clinical sign, and the importance of recognising it in the differential diagnosis of vasomotor rhinitis.

Figure Brain CT scan showing benign-looking mass in the fourth ventricle

Causes of rhinorrhoea and dementia

- old age ‘emile rhinorrhoea’
- CSF rhinorrhoea due to: trauma; fracture cribriform plate
- high pressure leak:
  - tumour (direct/indirect)
  - hydrocephalus (obstructive)
  - communicating
- normal pressure: subarachnoid: osteomyelitis
- focal atrophy (olfactory/ intrasellar)

A negative skin biopsy for malignancy does not exclude the possibility of subsequent fatal disease

SIR

Lentigo maligna (LM) is a benign melanocytic skin lesion that affects the elderly population. It is widely regarded as having an excellent prognosis and as such, is often ignored by both patient and physician alike. We report a fatal case of regressed primary lentigo maligna melanoma (LMM), which is the invasive counterpart of LM. Since the risk of progression from LM to LMM is considered small and because they present in the elderly as a large lesion in a cosmetically difficult area, treatment tends to be less aggressive than in other forms of melanoma. The absence of invasion on histology seemingly further justifies a conservative approach.

In March 1988 an 85-year-old female presented with LM on her right cheek. In March 1993 she was re-referred because the lesion had gradually increased in size, shape, and pigmentation. Histology of the lesion from a biopsy taken at this time showed evidence of a largely regressed melanocytic lesion. Four months later the patient was admitted to hospital as an emergency in status epilepticus. Investigation revealed widespread malignant disease. At autopsy multiple melanocytic deposits were seen within the lungs and brain which on histology had the appearance of metastatic spindle-cell malignant melanoma. No other primary site was identified other than the lesion on the right cheek.

The histological appearances of the early lesion had the characteristics of a benign melanocytic naevus. However, in LMM the neurofibromatous component is prominent and is typically the site of malignant change. In this case histological analysis confirmed the diagnosis of malignant melanoma. On examination there was a high proportion of subcutaneous melanocytes, with minimal stromal invasion. There was no sign of regression, ulceration, or recurrence of the lesion. No other dermatological abnormalities were found.

The natural history of malignant melanoma is characterised by a biphasic pattern of disease. The early clinical stage of tumour progression is characterised by a period of indolence during which the lesion exhibits no signs of regression or growth. When the lesion finally regresses, it passes through a period of nodular growth. The second phase is marked by the development of metastatic disease, usually lymphadenopathy. In this case there was no evidence of lymphadenopathy.