Letters

Subdural metastases from prostatic adenocarcinoma

Sir: Bony metastases on skull radiographs and/or bone scan are frequent in patients with metastatic prostatic carcinoma, though these lesions are seldom of clinical importance.¹ When the cranial dura mater is affected, it is usually the result of direct extension from calvarial deposits. Prostatic subdural metastases without evidence of direct invasion from calvarial lesions has only been described once.² A similar case with CT scan findings is presented.

A 63-year-old man was found to have a poorly differentiated adenocarcinoma of the prostate (Stage D) in 1975. He underwent an orchiectomy and received radiotherapy to the sacrum. Unrelenting bone pain prompted administration of various chemotherapeutic agents without success. In February 1982 he experienced a focal motor seizure that began in the left hand and became generalised. Neurological examination was normal except for diffuse hyperreflexia, bilateral extensor plantar responses, and a mild quadriparesis. Because of complaints of postictal neck pain, radiographs of the cervical spine were done revealing a pathological fracture of the C5 vertebral body without subluxation. The patient was treated with bedrest, a cervical collar, and phenytoin. No bony

lesions were seen on skull radiographs or radionuclide bone scan, but there were multiple rib and spinal metastases. An enhanced CT scan of the head revealed multiple lesions consistent with subdural metastases (fig). He received whole brain irradiation without change in his neurological status and died one month later of sepsis. Permission for necropsy was refused.

Penley *et al*² described the only case of prostatic subdural metastases without evidence of direct invasion from calvarial lesions. Their patient had characteristic osteoblastic metastases on skull radiographs and the CT scan showed bilateral enhancing subdural lesions without midline shift. Pathological findings included a smooth inner table of the skull with the outer surface of the dura mater intact, yet multiple subdural metastatic deposits were found without evidence of carcinomatosis or direct extension from calvarial metastases. Though necropsy was not performed in the present case, the absence of skull lesions on the radiographs and bone scan make calvarial metastases very unlikely. In support of the lesions being subdural, the CT scans of both the previous and this patient were strikingly similar, revealing multiple extracerebral crescentic or biconcave lesions without oedema or midline shift. Naheedy *et al*,¹ in a review of 50 patients suspected of having subdural or epidural metastases, stressed that uniform enhancement of the lesion with contrast is a distinctive feature of subdural metastases. Most of the lesions on CT scan in the present case enhanced uniformly. These CT features coupled with the absence of skull lesions by radiographs and bone scan in a patient with metastatic prostatic adenocarcinoma support the diagnosis of subdural metastases unassociated with calvarial lesions. The subdural lesions on CT scan must have arisen by a mechanism other than direct extension. Metastatic seeding into the subdural space along the perineural lymphatics or vascular embolisation are unlikely causes. A more plausible mechanism for metastatic deposits involving only the dura is invasion of dural veins via Batson's venous plexus.³

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Subacute sclerosing panencephalitis presenting as transient homonymous hemianopia

Sir: Disburbance of visual function is a well recognised feature of subacute sclerosing panencephalitis (SSPE),^{1,2} a disease thought to be due to persistent infection of the central nervous system with measles virus.³ The case described here is remarkable in that a transient homonymous hemianopia was the presenting feature of the disease, and was followed by an asymptomatic period of nearly two years before the onset of other features.

A 19-year-old man had a normal birth and developmental milestones, but had been slightly backward at school. Clinical measles occurred at the age of four years. He was the third of four children. He was completely well until January 1980 (aged 17 years) when he noticed that he tended to bump into objects on the left side. He was found to have a complete left homonymous hemianopia without other abnormalities. A CT brain scan was normal and an EEG showed some right occipital slow waves. Within six weeks the hemianopia had completely resolved.

He remained asymptomatic until November 1981 when his parents noticed a personality change; he became withdrawn, depressed and showed an aversion to loud noises. His vision rapidly deteriorated and he frequently bumped into furniture and doors. Positive findings in December 1981 were inappropriate behaviour, poverty of speech, severe loss of visual acuity, bilateral grasp reflexes in the hands, and clumsiness of all limbs. He continued to deteriorate and by January 1982 hardly spoke, his right arm was held in a fixed rigid position and he developed myoclonic jerking of the right arm and leg. By March 1982 he had become mute, bed-bound and incontinent of urine and faeces. He lay in bed mute without any response to verbal or painful stimuli. The optic fundi were normal but he appeared to be completely blind. He had roving eye movements in all directions of gaze. All limbs were spastic, especially those on the right side. Myoclonic movements of the right arm and leg

Figure Enhanced CT scan reveals multiple subdural metastases.