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

PEYTON DELANEY

Department of Medicine,
Division of Neurology,
University of Texas Health Science Center,
San Antonio, Texas 78284,
USA

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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.

Letters

occurred every three or four seconds associated with eye blinking. All tendon reflexes were pathologically brisk with bilateral extensor plantar responses.

EEG showed stereotyped repetitive slow wave disturbances which were linked in time to the myoclonic jerks, characteristic of SSPE. CT scan showed areas of poorly defined low attenuation in white matter of both frontal lobes, the left temporal horn and near the trigones. Cerebrospinal fluid (CSF) analysis revealed protein 690 mg/l; normal IgG concentration and oligoclonal bands were detected on polyacrylamide gel electrophoresis. Measles antibody titres (complement fixation test) were raised in the CSF (1:64) and serum (1:1024).

He had been treated with clonazepam, phenytoin and dexamethasone, and subsequently with isoprinosine, but his condition has continued to deteriorate. He is now (May 1982) unresponsive. The myoclonic jerks have become less marked.

This patient developed classical clinical features of SSPE with progression from the stage of insidious personality change to that of visual failure, pyramidal rigidity with myoclonic jerking of the limbs, proceeding to a state of mutism, incontinence and complete paralysis. There was a history of measles infection in childhood. The onset of SSPE at the age of 17 years is later than usual although well recognised.¹ The investigations were typical of those found in SSPE.² It seems reasonable to assume that the earlier symptom of transient homonymous hemianopia was part of the same illness, especially in view of the fact that his initial visual symptoms in November 1981 were very similar to those which had occurred nearly two years previously. As far as we are aware, the presentation of SSPE with transient homonymous hemianopia has not been described previously, and the delay of nearly two years between the onset of the hemianopia and the subsequent development of classical SSPE symptoms is remarkable. The disease is usually fatal within one or two years of clinical onset,³ although some patients have survived for much longer periods.^{4,5} The total duration of this patient's illness is not yet known. In this case the visual loss which occurred subsequently was probably due to cortical blindness, which is consistent with observations that neuronal loss in SSPE is very marked in the occipital lobes.⁶ If this pathological process had indeed occurred at the onset of the illness in January 1980, it remains unclear why it should have been arrested for a period as long as two years.

PGE KENNEDY*

C GARDNER-THORPE†

RS KOCEN*

*Department of Neurology,
National Hospital for Nervous Diseases,
Queen Square, London WC1N 3BG

†Department of Neurology,
Royal Devon and Exeter Hospital
(Wonford),
Barrack Road, Exeter EX2 5DW

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Sagittal sinus thrombosis and occult malignancy

Sir: We describe a patient with sinus thrombosis diagnosed in life, the cause at necropsy being intravascular metastases from a carcinoma of the gall bladder. In contrast to previously reported cases, there was no manifestation of malignancy in life.

A lady, aged 58 years, had been treated in 1972 with radio-iodine for thyrotoxicosis. The proptosis present at the time did not resolve. In 1980 she presented with a two month history of headache, temporal pain and malaise. She was admitted to her District General Hospital where, over a week, she became drowsy, confused and complained of double vision. An isotope brain scan suggested a subdural collection and she was transferred to the regional neurology unit on dexamethasone therapy. At transfer she appeared pale and unwell. The blood pressure was 210/90, there was a soft systolic ejection murmur at the left sternal edge without radiation or signs of

endocarditis. She was clinically euthyroid, had bilateral proptosis, sixth nerve palsy and papilloedema. Hearing was reduced by a bilateral conductive defect. There was no pyramidal signs and there was no meningism. After four days, hypoaesthesia to light touch and pin prick was noted on the lower lip. The proptosis became worse, periorbital oedema and chemosis appeared bilaterally. She developed a flaccid left hemiparesis, bilateral extensor plantar responses and continued to deteriorate, bleeding from venepuncture sites; she died on the tenth day of admission.

Serial blood cultures were sterile, serology normal, ESR raised at 70 mm/h and temporal artery biopsy normal. Left ventricular hypertrophy was evident on the ECG; radiographs showed a small left pleural effusion and generalised degenerative changes of the spine on skeletal survey. Cranial computed tomography demonstrated an old frontal lobe infarction and no evidence of a subdural collection. The cerebrospinal fluid was under increased pressure at 280 mm and was clear. The cell count, electrophoresis and cytocentrifuge were normal and the protein 48 g/l. Cavernous sinus thrombosis and possibly sagittal sinus thrombosis was suspected but angiography could not be performed because of disseminated intravascular coagulation.

At necropsy the dura was inflamed and densely adherent to the skull. The sagittal sinus was thrombosed and the carotid arteries inflamed in the cavernous sinus. Anaplastic cell nests were present in the artery walls, in the organising thrombus in the sagittal sinus and in the sphenoidal sinuses. Tumour was also present in the pontine veins. The gall bladder wall was thickened and haemorrhagic; the capillaries were packed with anaplastic tumour. Similar changes were seen in the liver, pancreas and para-aortic lymph nodes. The cells were of biliary origin and the primary site thought to be gall bladder.

Sagittal sinus thrombosis may be associated with infection, dehydration, head injury, leukaemia, malignancy and obstetric problems such as abruptio placentae.¹ As in this case headache, papilloedema, ophthalmoplegia and paraesthesiae suggest the diagnosis. Additional features include nausea, vomiting, convulsions, hemiparesis, paraparesis, visual field defects and nuchal rigidity.² The cerebrospinal fluid is under increased pressure and contains excess protein and a raised white cell count. The peripheral white cell count is often raised.³ CT scanning may be