Letters

Although occlusion of the basilar artery has been well studied and documented in adult patients, particularly after the work of Kubik and Adams, it is very uncommon in childhood. It is characterised by sudden and sometimes associated with peripheral neuropathy or involvement of the central nervous system. The skull bones are often involved but intracranial complications are rare. We report an adult male with multiple plasmacytoma who presented with raised intracranial pressure.

A 40-year-old man presented with severe occipital headache for 10 days and diplopia on looking to the right. He gave a 3 month history of low back pain. Examination revealed bilateral papilloedema, partial right lateral rectus palsy and limited straight leg raising on both sides. A CT scan of the head was normal. Opening pressure at lumbar puncture was over 400 mm CSF; protein, glucose, and gamma globulin content were normal and no cells were seen. The blood urea was elevated at 10 mmol/L with normal electrolytes. Bone alkaline phosphatase, calcium, plasma protein electrophoresis, plasma protein and albumin were normal. The ESR was 10 mm in the first hour, haemoglobin 12 g/dl, white cell count was 4.5 x 10^9/L (normal distribution) with normal platelet count. A 24 hour urine collection revealed normal creatinine clearance and protein loss of 3-2 g.

A radiograph of the sacrum showed a large osteolytic lesion. Small “punched-out” lytic lesions were seen on radiographs of the skull and left clavicle and multiple defects in sacrum, pelvis, ribs and spine were evident on the bone scan. Two bone marrow aspirations gave normal appearances and bone marrow trephine showed no evidence of metastases or myeloma. Biopsy of the sacrum revealed plasmacytoma. One month after admission, free kappa light chain globulins were found in the urine and accounted for 3-9 g in a 24 hour protein loss of 5-2 g.

Soon afterwards, plasma protein electrophoresis revealed kappa chains plus a significant fall in plasma immunoglobulin levels. CSF electrophoresis and plasma viscosity were normal. CT scan of the head at this stage revealed no intracranial abnormality; however, there was a large lytic lesion in the left temporal bone.

Despite mephalan, dexamethasone, alkalinisation, purinol and radiotherapy, he deteriorated over the course of the following year with numerous pathological fractures and infections. He developed severe hypercalcaemia and later succumbed to a chest infection.

The diagnosis of multiple plasmacytoma was based on biopsy and radiological evidence of multiple bone lesions without

Multiple plasmacytoma presenting as raised intracranial pressure

Sir: Plasma cell tumours may cause compression of the spinal cord or nerve roots and are sometimes associated with peripheral neuropathy or involvement of the central nervous system. The skull bones are often involved but intracranial complications are rare. We report an adult male with multiple plasmacytoma who presented with raised intracranial pressure.

A 40-year-old man presented with severe occipital headache for 10 days and diplopia on looking to the right. He gave a three month history of low back pain. Examination revealed bilateral papilloedema, partial right lateral rectus palsy and limited straight leg raising on both sides. A CT scan of the head was normal. Opening pressure at lumbar puncture was over 400 mm CSF; protein, glucose, and gamma globulin content were normal and no cells were seen. The blood urea was elevated at 10 mmol/L with normal electrolytes. Bone alkaline phosphatase, calcium, plasma protein electrophoresis, plasma protein and albumin were normal. The ESR was 10 mm in the first hour, haemoglobin 12 g/dl, white cell count was 4.5 x 10^9/L (normal distribution) with normal platelet count. A 24 hour urine collection revealed normal creatinine clearance and protein loss of 3-2 g.

A radiograph of the sacrum showed a large osteolytic lesion. Small “punched-out” lytic lesions were seen on radiographs of the skull and left clavicle and multiple defects in sacrum, pelvis, ribs and spine were evident on the bone scan. Two bone marrow aspirations gave normal appearances and bone marrow trephine showed no evidence of metastases or myeloma. Biopsy of the sacrum revealed plasmacytoma. One month after admission, free kappa light chain globulins were found in the urine and accounted for 3-9 g in a 24 hour protein loss of 5-2 g.

Soon afterwards, plasma protein electrophoresis revealed kappa chains plus a significant fall in plasma immunoglobulin levels. CSF electrophoresis and plasma viscosity were normal. CT scan of the head at this stage revealed no intracranial abnormality; however, there was a large lytic lesion in the left temporal bone.

Despite mephalan, dexamethasone, alkalinisation, purinol and radiotherapy, he deteriorated over the course of the following year with numerous pathological fractures and infections. He developed severe hypercalcaemia and later succumbed to a chest infection.

The diagnosis of multiple plasmacytoma was based on biopsy and radiological evidence of multiple bone lesions without

Multple plasmacytoma presenting as raised intracranial pressure

Refereces


Accepted 25 May 1986
cytological change in the bone marrow.\(^1\)

Multiple extramedullary plasmacytoma is a rare tumour with an incidence of about 1% of all plasma cell tumours.\(^2\) Cranial and intracranial plasma cell tumours are also extremely rare. Cushing\(^3\) found that among 2000 intracranial tumours, only four were intracranial myelomas but he did not record the details.

Clarke\(^4\) reviewed 24 cases of cranial and intracranial plasma cell tumours from the literature and added four of his own. He distinguished three separate syndromes; multiple cranial nerve palsies, intracranial tumour formation, and a constellation of signs due to invasion of the orbit by plasma cell tumour. Infracranial abnormalities are thought to be due to plasmacytomas arising in the base of the skull.\(^5\) Alternatively it has been suggested that increased intracranial pressure could be due to abnormal globulin production inducing a hyperviscosity syndrome with protein deposition in the central nervous system.\(^5\) In our patient, CT scan showed that there was no significant mass lesion and plasma viscosity was normal. Dural involvement around the left mastoid may have lead to sinus thrombosis and consequent raised intracranial pressure.

We are grateful to Miss C Mackay for typing the manuscript and to Mr G Neil of the Blood Transfusion Service, Royal Infirmary of Edinburgh for organising the protein electrophoresis strip.

A COLLIERT
B ASHWORTH
Department of Medical Neurology
University of Edinburgh,
and Northern General Hospital,
Edinburgh EH5 2DQ, UK

References

Accepted 20 July 1986

Fig High resolution agarose gel electrophoresis showing an abnormal protein band in patients serum and urine. (PAP3 = normal control pool serum sample).

Abducent palsy after rapid shrinkage of a prolactinoma

Sir.—The sixth cranial or abducent nerve, by virtue of its anatomical course, is particularly susceptible to damage in pathological conditions affecting the cavernous sinus.\(^1\) An expanding pituitary tumour may produce a sixth nerve palsy by lateral displacement and stretching of the cavernous portion of the nerve, often with accompanying third and fourth nerve damage.\(^2\) We report the occurrence of a transient sixth nerve palsy in association with the rapid reduction in size of a pituitary prolactinoma induced by bromocriptine, a relationship not previously documented.

A previously well 31 year old man presented with a two-month history of blurring of vision of the left eye, and diminished libido and potency. He had no associated headache, diplopia or other symptoms. Examination of the left eye revealed a visual acuity of 6/12, an upper temporal quadrantic field loss and pallor of the optic disc. The right eye was normal, as was the remainder of neurological and the general physical examination.

Skull radiographs showed massive expansion of the pituitary fossa, with destruction of the dorsum sellae and almost complete replacement of the adjacent sphenoid sinus. A large, partly cystic pituitary tumour, with suprasellar, cavernous sinus and sphenoidal sinus extension was delineated by axial and coronal CT scanning (fig 1). Visual evoked potentials showed bilateral prolongation of latencies with attenuation of the major positive peaks, these abnormalities being more severe on the left. The serum prolactin, as measured by radioimmunoassay, was 11,300 ng/ml (normal less than 25). Other parameters of endocrine function were normal, including serum TSH, thyroxine, FSH, LH, growth hormone, cortisol and testosterone levels. Bilateral carotid and left vertebral angiograms confirmed significant suprasellar extension, with elevation of the A\(_1\) segment of the anterior cerebral artery, and lateral displacement of the cavernous portions of the internal carotid arteries. No evidence of intracranial aneurysm formation or of arterial encasement was present.

Oral bromocriptine was commenced at a dosage of 2.5 mg nocte, gradually increasing to 15 mg per day. The patient’s condition remained unchanged until 4 weeks after the institution of treatment, at which time he developed increasing horizontal diplopia over 4 days. There was no associated headache or changes in visual acuity. Exam-