Although our patient did not have any response to accommodation, the pupillary denervation hypersensitivity demonstrated would appear to indicate that he did have tonic pupils. This case further illustrates that patients treated with what had previously been thought to be adequate antibiotic therapy for primary or secondary syphilis may still develop chronic progressive CNS syphilis (although in this and other reported cases of neurosyphilis a re-infection cannot be absolutely excluded). Patients with dilated tonic pupils (rather than only Argyll Robertson pupils) should have serological testing for syphilis and lumbar puncture when appropriate. This would help ensure that a CNS syphilis infection that may effectively be treated does not remain undiagnosed.

References


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Parkinsonism due to corpus callosum astrocytoma: case report

Sir: Brain tumour is an uncommon cause of the Parkinson syndrome. In the most recent review by Polyzois et al in 1985, 49 verified cases with supratentorial tumours were collected from the literature. The majority of the cases were of supratentorial extra-axial benign mass lesions. We report the case of a patient with Parkinsonism due to a corpus callosum astrocytoma.

The presenting illness of this 79 year old man began in November 1983 with difficulty in gait associated with progressive weakness of the entire right side. Tremor of the left hand appeared 2 months later.

At the time of examination on 8 February, 1984, he was unable to stand alone and he had urinary incontinence. The findings were as follows: blood pressure was 150/70 mm Hg, pulse 88 beats/min and temperature 36.0°C with normal general examination. On neurological examination he showed no insight into the nature of his problems. His attention span was brief and he presented dressing apraxia. No papilloedema was present. A resting tremor (4–6 Hz) was found in the left arm. This was associated with marked cog-wheel rigidity. Tremor was present but less intense in the left leg. Voluntary movements were slow and his face was mask-like. Sucking and pal-momental reflex were detected. He also had retropulsion. There was a mild right-sided hemiparesis with hyperreflexia and positive Babinski reflex. There were no sensory deficits or cerebellar signs. Routine tests results were normal. The computed tomography brain scan showed a large medially situated tumour (figure). This extended into both parietal lobes through the posterior portion of the corpus callosum. Stereotaxic biopsy revealed that the tumour was an astrocytoma.

Extrapyramidal symptoms characterised by rigidity, bradykinesia and tremor, have...
been reported as a rare complication of brain tumour.\textsuperscript{1-4} Only one case, described by Sciarra \textit{et al},\textsuperscript{2} as corpus callosum glioma with Parkinsonian symptoms was found by us in the literature. When symptoms of Parkinsonism do result, it is not uncommon to make an incorrect diagnosis of Parkinson's disease.\textsuperscript{2} However, symptoms of increased intracranial pressure or mental change and signs of cortico-spinal tract and sensory involvement eventually develop, suggesting the diagnosis of tumour. Any combination of Parkinsonian signs may be present, but most authors reported contralateral static tremor and rigidity.\textsuperscript{1,2,6}

The exact aetiology of the development of the Parkinsonian syndrome in our patient is not well understood and several mechanisms can be postulated: (a) mechanical pressure on basal ganglion nuclei could be caused directly by the tumour; (b) indirectly by torsion or compression of midbrain and tentorial herniation; (c) the deeply situated glioma may directly involve the basal ganglia.

In this patient, the assumption of a causal relationship between Parkinsonism and the tumour is based on the lack of any associated precipitating factors (such as ingestion of drugs or poisoning) and the onset of tremor shortly after the contralateral hemiparesis had appeared. The mechanism by which Parkinsonian symptoms are produced contralaterally to cortico-spinal tract symptoms, may be explained by the medial localisation of the tumour and is due probably also to the direct involvement of the left internal capsule whose normal functioning would have been essential in order for tremor and rigidity to appear.

Early recognition of an intracranial tumour as a cause for Parkinsonism is therefore very important if further neurological deficit is to be prevented. It would seem desirable to obtain a CT scan in any Parkinsonian patient with other associated neurologic manifestations, and may also be indicated in patients with hemiparkinsonian symptoms, essentially with tremor.

\textbf{Letters}

\textbf{Accepted 17 January 1986}

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\section*{Fig 1 Plain computed tomography (CT) scan showing a huge low density area in the posterior cranial fossa extending to the squiggles middle cranial fossae.}

\section*{Huge epithelium-lined cyst: report of two cases}

Sir: Epithelium-lined cysts of the central nervous system have been reported with various names as "neuroepithelial cyst, ependymal cyst, parapharyngeal cyst, choroid plexus cyst, and colloid cyst."\textsuperscript{11-12} However, their exact origin is uncertain and the pathogenesis of these cysts is still controversial.\textsuperscript{5,8,12}

The following case reports describe two cases of huge epithelium-lined cysts in the posterior cranial fossa with extension to the middle cranial fossa. Although a review of the literature disclosed many reports concerning the location of the cysts,\textsuperscript{11,12,16-13} such cases as described here appear never to have been reported. We present details of these cases, with clinico-pathological features of these cystic lesions.

Case 1 was a 2-month-old male infant with an increase in head circumference and horizontal nystagmus. He was admitted to our department for diagnostic workup and treatment. Neurological examination recorded only horizontal nystagmus. A computed tomography (CT) scan revealed a huge low density area in the posterior cranial fossa extending to the bilateral middle cranial fossa (fig 1). Metrizamide CT cisternography revealed no communication between the cyst and the ventricular system. Vertebral angiography demonstrated marked bowing and displacement of the basilar artery and an avascular area between clivus and pons. Suboccipital craniectomy was performed; during the operation, the cyst wall was exposed and was found to consist of tough membrane with many capillaries. Facial and acoustic nerves were stretched posteriorly over the cyst wall. The right cerebellar hemisphere was displaced to the left. The cyst wall was widely opened and partially removed to establish a communication with the subarachnoid space. The cyst cavity was found to extend from the preoptiopital region to the bilateral middle cranial fossa, beyond the incisura of the tent. No abnormalities of the cerebellum or brain stem were noticed, and there was no communication between the cyst and the ventricular system. A ventriculo-peritoneal shunt was performed 2 weeks after the craniectomy, because hydrocephalus did not resolve following the partial removal of the cyst. The fluid obtained from the cyst contained a protein level of less than 0.1 g/l. Microscopic examination revealed that the wall consisted of a single layer of ciliated cuboidal and columnar epithelial cells with an underlying basement membrane. The wall was supported in part by connective tissue (fig 2).

The postoperative course was uneventful, and he was discharged 2 weeks after the second operation.

Case 2 was a 10-month-old female baby with arrested development. CT scan demonstrated a huge low density area in the posterior cranial fossa which extended to the right middle cranial fossa. CT cisternography did not show communication between the cyst and the ventricular system. Cranietomy was