Stuttering pituitary apoplexy resembling meningitis

In the first clear description of pituitary apoplexy Brougham *et al*[1] suggested that the syndrome should be considered as the abrupt development of headache, amblyopia, diplopia, drowsiness, confusion or coma. These clinical features have now become well established.[2,3] However, Brougham *et al* mention one case of haemorrhage into a pituitary tumour simulating rupture of an intracranial aneurysm where ophthalmoplegia was delayed several days.[4] We have seen two cases where delay in diagnosis occurred as a result of the subacute onset of symptoms. The clinical picture was further confused by the finding of a highly cellular cerebrospinal fluid (CSF).

The first case was a 50 year old warehouse attendant who had a five day history of generalised headache and vomiting with two days of double vision. His relatives had noticed an intermittent droop of the right eyelid. He had previously been in good health apart from the drainage of a "cold abscess" in early childhood. He drank about 20 pints of beer each week.

Examination revealed a slight pyrexia (37·5°) with limitation of neck flexion but no neck stiffness. He had a partial left third nerve palsy with a dilated but reactive left pupil and a partial left ptosis. There was no field defect and extra-ocular movements appeared full. Power and tone was normal in the limbs. The biceps reflexes could be elicited on reinforcement but otherwise tendon reflexes were all absent. Plantar responses were flexor and sensory testing was normal. An unenhanced CT scan was performed to exclude an abscess. Lumbar puncture revealed opalescent CSF under 19 cms pressure which contained a concentration of 77 mg% of protein with 52 white cells (5% polymorphs, 91% lymphocytes, 4% macrophages). No organisms were seen and the concentration of sugar was normal. An EEG showed an excess of mainly posteriorly distribute slow waves.

A provisional clinical diagnosis of a subacute meningoencephalitis was made and treatment was started with intravenous Acyclovir and thiamine. Over the next three days there was a deterioration in his clinical state with a rising pyrexia to 39°C. He developed bilateral ptosis with impaired adduction and elevation of both eyes. Visual acuity in the right eye was reduced to counting fingers at one metre and a right altitudinal field defect developed which extended below the horizontal meridian.

A high resolution enhanced CT scan of the orbits revealed a pituitary adenoma with some suprasellar extension. Treatment with Prednisolone was started immediately with rapid improvement of visual acuity to 6/7·5 in the effected eye. Endocrine studies revealed a normal prolactin (< 65 units) but low TSH (0·2 uU/L) and free T4 (5·5 pm/L). He subsequently had an uneventful transphenoidal hypophysectomy and pathological examination of the specimen was consistent with the eventual diagnosis of infarction in a pituitary adenoma.

The second case was a 45 year old printer's assistant who presented with symptoms of the carpal tunnel syndrome bilaterally. It was noted, a year later, that he had large hands and other features of acromegaly. A pituitary adenoma was confirmed on CT scan. Whilst awaiting a transphenoidal hypophysectomy he awoke with sudden onset frontal headache and vomited. The following day he was a little better but three days later he became confused and complained of visual impairment. He was admitted to a local hospital where he was found to have neck stiffness and a temperature of 40°C. A lumbar puncture revealed CSF which was xanthochromic and under increased pressure with raised protein concentration and a mixed pleocytosis. The following day he developed peri-orbital and subconjunctival oedema and bilateral sixth nerve palsies. His deep tenden reflexes were absent and his conscious level began to deteriorate. He was transferred to a specialist unit where he was found to be disoriented in time and place and pyrexial. Periorbital oedema, bruising around the left eye and marked neck stiffness were noted. Visual acuity was 3/60 on the right and 6/18 on the left and he was unable to identify any of the Ishihara test plates. There was a temporal hemianopia to colour in the left eye and a dense central scotoma on the right on confrontation. There was mild disc swelling with absent venous pulsation. Lateral rectus weakness was complete and bilateral and upward movement of the right eye was limited. The only other abnormal finding was total areflexia.

The full blood count was normal as was the urea although the serum sodium was low at 126 mm/L. The CSF contained 1·2 G/L of protein with a pressure of 30 cms of CSF and a white cell count of 900 (10^6/L) of which 79% were polymorphs and the rest lymphocytes. There were 20 (10^6/L) red cells and a normal CSF glucose. Urine and serum osmolarities suggested inappropriate secretion of ADH. A plain skull radiograph showed an enlarged pituitary fossa and opacification of the sphenoid sinus. CT scan revealed a suprasellar mass with lateral extension. Serum cortisol was low (60 mu/L) as were levels of total thyroxine, TSH, prolactin, FSH and LH confirming panhypopituitarism. A random estimation of growth hormone was slightly elevated (16·6 mu/L). In view of the gradual evolution of symptoms and the cellular CSF a provisional diagnosis of meningitis was made by the referring hospital. The CSF proved to be sterile and field defects and cranial nerve signs resolved when antibiotics were withdrawn and he was treated with intravenous steroids and thyroxine.

The presence of inflammatory changes in the CSF in some cases of pituitary apoplexy is well documented in the literature. Bjerre and Lindholm[5] have recently emphasised the occurrence of "sterile meningitis" as an important feature of pituitary apoplexy and report six cases where CSF examination revealed an elevated leucocyte and or polymorph count. Four further cases in the literature are cited. The presence of CSF pleocytosis usually presents little diagnostic concern in patients with a typical acute onset where the diagnosis is confirmed by urgent CT scanning. The subacute or stuttering onset in our cases made diagnosis difficult and prompted this report. In the first case the absence of a field defect on presentation compounded the difficulty in diagnosis. In the second case the lack of awareness of this mode of presentation of pituitary apoplexy delayed establishing an accurate diagnosis despite the previous diagnosis of a pituitary tumour.

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Intracranial haemorrhages occurring in the idiopathic hypereosinophilic syndrome

The hypereosinophilic syndrome (HES) can produce a range of neurological disorders. Three major patterns of neurological involvement have been described: encephalopathy, sensory polyneuropathy, and central nervous system thrombo emboli.

We describe an elderly woman with this syndrome who presented with right temporal lobe and left cerebellar hemisphere haemorrhages.

An Indian woman aged 72 years first presented to our hospital in 1980 with general malaise and a nocturnal cough. Examination at that time revealed a mild expiratory wheeze. Her haemoglobin was normal but the white cell count was elevated at 46 000 × 10^9/l (78% eosinophils and 15% neutrophils). Her platelets, coagulation screen and auto antibodies were normal. A chest radiograph showed old calcified apical foci with some ill defined shadowing in the left mid zone. A bone marrow examination revealed eosinophilia with both the red and white cell series severely depressed. There was no excess of blasts and no abnormal cells.

A diagnosis of HES was made and she was started on a reducing dose of prednisolone. On discharge her white cell count was 10·7 × 10^9/l with 1% eosinophils. Symptomatically she had improved and her nocturnal cough had disappeared.

She remained well on a small dose of prednisolone and continued to have a normal eosinophil count over the next five years. She was lost to follow up in 1985 but according to her general practitioner she continued to take prednisolone until 1987. She was admitted in February 1989 after collapsing at home. According to her son the event was sudden and without warning. There was no history of head trauma or of a seizure. She was a non smoker, non drinker and was not on any medication. Systems enquiry, by proxy, was normal.

On examination she was unconscious (Glasgow coma scale Grade IV), apyrexial but with no neck stiffness. General examination was normal, BP 170/80.

On neurological examination, her pupils were mid point and fixed. Fundoscopy was normal and she had no obvious facial asymmetry. Her eye movements were difficult to assess and she had a depressed gag reflex.