Stuttering pituitary apoplexy resembling meningoencephalitis

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, amnesia, diplopia, drowsiness, confusion or coma. These clinical features have now become well established.2 However, Brougham et al. mention one case of haemorrhagic apoplexy into a pituitary tumour simulating rupture of an intracranial aneurysm where ophthalmoplegia was delayed several days.3 We have seen two cases where delay in diagnosis occurred as a result of 7–8 days. A lumbar puncture immediately after the onset of symptoms in one 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 eye which had previously been good 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°C) 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 no bitemporal hemianopia apparent. 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 to pin prick extensor. Treatment with an enhanced CT scan was performed to exclude an abscess. Lumbar puncture revealed opalescent CSF under 19 cm 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 distributed slow waves.

A clinical diagnosis of subacute meningoencephalitis was made and treatment was started with intravenous Acyclovir and thiamine. Over the next three days the patient deteriorated 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 temporal 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 affected eye. Endocrine studies revealed a normal prolactin (< 65 units) but low TSH (0.2 uU/L) and free T4 (9.5 pmol/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 cerebral type (coma to head injury). It was noted, a year later, that he had had amnesia 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 haemorrhages and bilateral sixth nerve palsies. His deep tendon 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. He was drowsy, Peribulbar oedema, brusising around the left eye and marked neck stiffness were noted. Visual acuity was 6/60 on the right and 6/18 on the left with no light perception on the left side of the Ishihara test plates. There was a temporal hemanopia to colour in the left eye and a dense central scotoma on the right on confrontation. There was mild disc swelling with absent venous pulsations in the left eye. Venous congestion 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 mmol/L. The CSF contained 1.2 g/L of protein with a pressure of 30 cm of CSF and a white cell count of 900 (10^6/L) of which 79% were polymorphs, 13% lymphocytes and 8% monocytes. There were 20 (10^6/L) red cells and a normal CSF glucose. Urine and serum osmolalities 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 μU/mL) as were levels of thyroid stimulating hormone, thyroxine, prolactin, TSH, FSH and LH confirming panhypopituitarism. A random estimation of growth hormone was slightly elevated (16 μU/mL). In view of the gradual evolution of symptoms and the normal CSF a presumptive diagnostic isolation 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 Lindholm1 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 pituitary apoplexy. The acute onset where the diagnosis is confirmed by urgent CT scanning. The subarachnoid or subdural onset in our cases made diagnosis difficult and prompted this report. In the first case the absence of any CSF 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 obtaining an accurate diagnosis despite the previous diagnosis of a pituitary tumour.

We thank Professor WI McDonald and Dr MFT Yealand for permitting us to report cases under their care.

Intracranial haemorrhages occurring in the idiopathic hyper eosinophilic syndrome

The hyper eosinophilic 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 thromboemboli.

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 × 10^3/L (78%, eosinophils and 15%, neutrophils). Her platelets, coagulation screen and auto antibodies were normal. A chest radiograph showed old calcified apical focus with some ill defined shadowing. In 1983, 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 × 10^3/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. Symptoms enquire, 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.
Meningitis and spinal subdural empyema as a complication of sinusitis

Spinal subdural empyema is a rare event. We describe the case of a young man who developed sinusitis while recovering from a routine operation followed by a dramatic meningitic illness with formation of a spinal subdural empyema.

A previously fit 23 year old man had an elective left temporal mandibular joint meniscectomy for Conner's syndrome. Two days later he developed a mild headache with a slightly purulent nasal discharge. Within the following 24 hours his headache became bifrontal and more severe. He developed visual hallucinations though he remained orientated and there was marked neck stiffness, severe low back pain with very limited straight leg raising and a high fever. There was no previous history of headache or immune suppression. His lumbar puncture showed turbid green cerebrospinal fluid (CSF) with 284 polymorphs and 134 lymphocytes with a CSF protein of 1-5 gms per litre and a low CSF glucose.

Plain skull radiographs showed thickening of the right maxillary sinus with a small gas bubble in the supra-stella cistern. A CT scan confirmed these findings and showed a completely opacified right ethmoid. Cultures from his CSF grew streplococcus milleri (a micro-aerophilic haemolytic streptococcus) and a bacteroides of the corrodens type. Both these organisms are compatible with an origin in the upper respiratory tract. Amoxicillin, metronidazole and gentamicin were given intravenously. His sinuses were drained and a considerable amount of green pus was obtained. His temperature gradually settled through his hospitalisation and his tendon jerks in both lower limbs became depressed. A cervical myelogram was performed and this showed somewhat irregular subarachnoid space in the lower cervical region and a complete block at the level of D7. A subsequent CT scan of the same region confirmed a posteriorly situated mass causing forward displacement of the cord but no bony erosion. He had a laminectomy from D7 to D10. A subdural mass with granulation tissue was identified and suitable decompressive measures were undertaken. Following this he made a slow but progressive recovery although he suffered with low back pain and was discharged home after completion of a month of antibiotic therapy.

The association between sinusitis and intracranial empyema is well established. Spinal subdural empyema is a much rarer occurrence and has not been previously described as a complication of sinusitis.

In a 1973 review of the literature only ten cases were described. Five of these patients died, three were left with significant neurological deficits and only two fully recovered. Prompt surgical drainage and antibiotics have obviously improved prognosis, and more recently eight additional cases have been reported. Seven of these patients made full or fair recoveries.

Typically, spinal subdural empyema presents with a fever, backache, and radicular symptoms and signs. There have been claims that the absence of percussion tenderness helps to distinguish it from an epidural collection of pus19 which is usually associated with vertebral osteomyelitis or oncostasis. The diagnosis has recently been facilitated with the introduction of spinal CT.
Intracranial haemorrhages occurring in the idiopathic hypereosinophilic syndrome.

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