Posterior ischaemic optic neuropathy after a spontaneous extradural haematoma

Most extradural haematomas (EDH) occur after head injury but cases of "spontaneous" EDH have been reported. These are a result of either local sepsis, a dural arteriovenous malformation or an abnormality of clotting. We report the case of a spontaneous EDH resulting in posterior ischaemic optic neuropathy as a consequence of tentorial herniation.

A 15 year old boy developed a headache which built up over a period of 30 minutes. In the evening he was seen by his general practitioner who diagnosed a viral infection as the headache was associated with some fever. On the second day he vomited in the morning. By the morning of the third day he had fever and was unable to get up because of headache and giddiness. His mother saw him at approximately 1.00 am on the fourth day of his illness when he seemed to be sleeping normally. By 7.00 am the same morning he was found unconscious and was admitted to the District General Hospital on 17 March 1988. There was no other predisposing illness or head injury.

In the accident and emergency department, he was noted to be cyanosed with vomit in his nose and throat. He was in coma with a Glasgow Coma Score of 5. Both pupils did not respond to light and the right was 6 mm, the left 3 mm. His auxiliary temperature was 40°C, a regular pulse of 110, blood pressure 120/55 mmHg and normal heart sounds. Auscultation of his chest revealed added sounds in the left base suggesting an aspiration pneumonia which was confirmed on chest X ray. His blood results were as follows: Hb 1.5 g/dl, white cell count 11.7 x 10^9/l, platelet count 180 x 10^9/l, sodium 140 mmol/l, potassium 4.2 mmol/l and urea 4.1 mmol/l. Clotting was within normal limits with an INR 1.0 and KCCT (control 48).

Before being transferred, the patient was intubated, ventilated, paralysed, sedated and given 100 ml mannitol 20%. On arrival in the neurosurgical unit on 17 March 1988, a CT

Figure CT scan showing a 3 cm extradural haematoma of mixed density in the right frontal region.

was performed (figure) which showed a 3 cm maximum density EDH over the right frontal convexity which was of mixed density. There was no radiological evidence of head injury. In view of his condition and the radiological findings, an immediate right frontal craniotomy was performed. A moderately sized EDH was found. The haematoma was considered to be of some days duration. No evidence was found of either a fracture or a dural arteriovenous malformation.

On the evening of operation, the patient developed a transient diuresis which resolved and there were no further episodes. The patient was extubated four days postoperatively and his conscious level continued to improve. Initially his vision was very restricted and he could see only a few objects. Examination showed that the pupils, although unequal, were reacting to light. He appeared to have a left homonymous hemianopia very early after surgery. This was not demonstrable subsequently as he has remained completely blind since then. He was also noted to have a mild left hemiparesis (Medical Research Council grade 4). One month later he was discharged and his conscious level had returned to normal, his hemiparesis had resolved but he was blind.

On review in the department three months later, his vision was unchanged with no light perception in either eye. He had put 19 kg in weight. He had polydipsia and, according to his mother, prolonged and excessive periods of sleeping. On examination it was alert and orientated with no limb weakness. His pupils did not react to light and fundoscopy revealed bilateral optic atrophy.

Visual evoked responses showed no response. A CT scan showed moderate atrophy of the right frontal lobe and mild dilatation of the ventricular system compared with the previous scan. The orbits, sella and suprasellar cisterns appeared normal. MRI showed slender optic nerves consistent with atrophy and confirmed the frontal atrophy. There was no evidence of hypothalamic abnormality on the scan. He had an endocrine assessment six months postoperatively which showed an isolated defect of growth hormone production.

One year postoperatively the patient remains blind with fixed pupils and the previous excess of sleeping has resolved and there is no longer excessive thirst. His appetite remains normal and there has been no change in his personality.

The cause of the precuneal damage shown by the optic atrophy is not well known, though it is felt to be due to compression of the optic nerves against the skull base by the overlying brain. This has been shown radiologically in the case of a patient with a falx meningioma where the visual disturbance was reversed postoperatively. It had not, however, been confirmed pathologically.

In our patient, the CT scan on presentation showed evidence of raised intracranial pressure with obliteration of the basal cisterns and an absent third ventricle. There was no evidence of occult infarction radiologically on a later scan. We suggest that the cause of this atrophy is a result of infarction secondary to raised intracranial pressure. His visual deterioration was unusually, appeared to have been progressive. In this patient there was a suggestion of an endocrinopathy with an initial diuresis, polydipsia, weight gain and hyperphagia. This was, however, not borne out by endocrine assessment which at six months showed an isolated defect in growth hormone production. This finding is of unknown clinical significance but must be recent as the patient is of normal height.

We believe that the cause of the haematoma was truly spontaneous. There was no evidence of other causes for a posterior fossa extradural haematoma or a dural arteriovenous malformation or sepsis at surgery. There was evidence of normal blood coagulation on admission and no history of recent head injury was found after very careful retrospective inquiry.

We would recommend a new classification of EDHs which recognises their cause. Primary EDHs are those haematomas where no cause can be elucidated and secondary EDHs where a cause is demonstrated.

CJ GERBER G NIELISKER P KENNEDY

Wessex Neurological Centre, Southampton General Hospital, Tremona Road, Southampton, UK

Correspondence to: Dr Gerber.


A pilot study of N-Methyl-D-Aspartate (NMDA) antagonist in Parkinson's disease

Recent experimental studies have suggested that agents which antagonise glutamatergic transmission could exert anti-Parkinsonian activity. For example, it has been shown that NMDA antagonists potentiate the effects of levodopa and could thus act as a complementary agent with MPTP "neurotoxicity" in rats. Injection of the NMDA antagonist MK 801 within the medial pallidum reverses Parkinsonian symptoms in MPTP-treated monkeys. The mechanism of action remains unclear but it has been suggested that blockade of NMDA receptors would result in a facilitation of dopamine action by preventing the glutamate-induced dephosphorylation of DARPP-32, a dopamine- and cAMP-regulated phosphoprotein. However, no clinical data are yet available in Parkinson's disease (PD). We investigated in PD patients the effects of an add-on therapy with ifenprodil, a non competitive antagonist of the NMDA receptor at the polyamine modulatory site, which inhibits [3H] MK 801 binding. This drug is, as far as we know, the sole NMDA antagonist currently available on the market. Ifenprodil also possesses alpha-adrenoceptor blocking properties and is already in clinical use as a cerebral and peripheral vasodilator.

Two groups of patients with idiopathic PD were studied after informed consent: the first one included nine non-fluctuating patients, mean age (SD), 67 (4); mean duration of PD 9 (1) years, mean (SD) duration of levodopa therapy: 7 (1) years,
Central pontine myelinolysis in a patient with AIDS

Central pontine myelinolysis (CPM) has been associated with rapid correction of hyponatraemia as well as an acute rise in serum sodium even from normonatraemic levels. CPM has also been linked with hyperosmolality. The rate of change in the osmolality, the length of time that this rate of change persists, and the clinical condition of the patient are all important factors. We describe a case of CPM in a 49 year old HIV positive homosexual man who had a normal serum sodium and osmolality throughout his illness.

The patient had been HIV positive for at least six years. In 1989 he developed increasing hepatosplenomegaly and uncontrollable thrombocytopenia. A splenectomy was performed and histology showed only non-thrombocytopenic thrombocytopenia. A plaque was formed in the portal and showed normal erythrocytes and platelets. The spleen was not involved.

Seven months later he presented with a short history of dysphagia, and fever. The only abnormal finding was a pericardial rub. Investigations showed that he was mildly anaemic, and hypoalbuminaemic with a serum albumin of 19 g/l. Liver function tests and clotting studies were normal. Initial investigations were all negative apart from demonstrating a pericardial effusion and a small pleural effusion. On rescreening for pericardioceles, however, the effusion had resolved. A small pleural effusion was aspirated and found to contain numerous lymphocytes suggesting a lymphoma. Immunocytochemistry was unhelpful. Bone marrow examination and a liver biopsy revealed normal liver.

The patient had no gastrointestinal symptoms. Thirteen days before his death he developed diplopia and was found to have a right sixth nerve palsy. A CT brain scan showed no abnormality. A lumber puncture showed clear CSF with 10 lymphocytes and a protein of 0.78 g/l. No pathogens were isolated and he was cryptococcal antigen negative. Cytology of the CSF was normal. A trial of high dose steroids made no difference to his deterioration. He became progressively weaker, developed right-sided posis and dysarthria and died a few days later.

 Necropsy examination demonstrated dissemi-

nated lymphoma, including large ulcerating deposits in the stomach. Postmortem examination of the brain showed the typical appearance of CPM with a large well defined focus of myelin loss in the base of the midpons and upper-pons (figure). Focal infiltrates of lipid containing macrophages were present in the area of myelinolysis but there was no inflammation otherwise associated with this lesion. Axons were partly preserved. There was some mild cerebral atrophy, mild diffuse meningeal pallor in the cerebrum and slight lymphomatous infiltration of the meninges.

To our knowledge there have been no previous reports of CPM in association with HIV. The only significant biochemical abnormality was the hypoalbuminaemia which developed in the month preceding his death. This may have been due to gastro-intestinal losses as well as to his underlying condition. His serum sodium remained normal throughout his stay in hospital, and he received no intravenous fluids. His albumin remained at 29 g/l until he died. CPM has been described in liver transplant recipients and in severe burn cases, as well as in association with alcoholism and malnutrition. It has been thought that the rapid correction, or over correction, of hyponatraemia was the crucial factor in causing CPM in most cases. This is obviously not the only cause. In a series of CPM in severely burnt patients, hyponatraemia was not a feature, whereas each patient who developed CPM had a prolonged episode of extreme hyperosmolality. Our patient did not have an episode of serum hyperosmolality, but had one month of hypoalbuminaemia. A low serum albumin may be a significant factor in the development of CPM. This could be a feature common to our patient and the groups of patients described above in which CPM had been reported. Another possibility is that CPM may be a primary effect of HIV, representing yet another neu-

ronopathological manifestation of this virus. The p24 HIV protein was detected immunocytochemically in rare perivascular macro-
A pilot study of N-methyl-D-aspartate (NMDA) antagonist in Parkinson's disease.

J L Montastruc, O Rascol, J M Senard and A Rascol

*J Neurol Neurosurg Psychiatry* 1992 55: 630-631
doi: 10.1136/jnnp.55.7.630-a

Updated information and services can be found at: http://jnnp.bmj.com/content/55/7/630.2.citation

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to: http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to: http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to: http://group.bmj.com/subscribe/