inferior olives the 6th, 8th, and 10th cranial nerves were affected. All levels of the spinal cord were involved with neuronal degeneration in the anterior horns; Clark’s column was similarly damaged. The crossed pyramidal tracts showed gross axonal degeneration. The spinal roots appeared normal.

The findings of a low grade encephalitis and the Pseudomonas sepsis unusual for the age led to the immunological investigation of a stored deep-frozen specimen of serum. The results were IgG < 2 IU, IgA < 2 IU, IgM 18 IU. The enterovirus isolated from the CSF at 16 months was found to be an Echo virus type 29.

Patients with immunoglobulin deficiency usually recover uneventfully from virus infections. However enteroviruses may occasionally cause persistent encephalitis in children with X-linked hypogammaglobulinaemia.1-3 Intact B-cell function seems to be essential especially for the termination of CNS echovirus infections.1-3 This suggests, that antibody-dependent, cell-mediated cytotoxicity which was demonstrated on target cells infected with herpes and mumps virus4 might also have a role in the elimination of Echo virus. In addition Echo virus is known to inhibit lymphocytic transformation.5 This effect upon cellular immunity and the high rate of spontaneous mutations among RNA viruses,6 are some other factors which acting together may conceivably cause an infection to become chronic. Wilfert1 and Medici7 suggest that hypogammaglobulinaemic patients with the HLA type B7 are predisposed to develop persistent encephalitis. Treatments with gammaglobulin,1-3 a specific antibody containing plasma1 and with intraperitoneally implanted thymus1 have not been successful. No treatment was attempted in our patient, because the warning signs and symptoms of an immunodeficiency were not present in early life, and thus, the diagnosis was not reached during life time. It is of particular interest to note that progressive spastic paraplegia was the only sign of the CNS infection.

This case supports the view that intact B-cell function is essential for the eradication of Echo virus infection of the central nervous system.

References

Transient unilateral mydriasis with basilar aneurysm

Sir: Third nerve palsy is a valuable localising sign of a posterior communicating aneurysm and is characteristically accompanied by dilatation of the pupil. Third nerve compression in basilar artery aneurysm is less common. We report a patient with transient unilateral pupillary dilatation without external ophthalmoplegia associated with sacular basilar aneurysm. This sign in basilar aneurysm, has not to our knowledge, previously been reported.

Following a generalised convulsion with no aura or focal features a 31-year-old decorator complained of severe headache and vomiting. Both plantar responses were extensor. After about 24 hours the abnormal signs had resolved, but his headache persisted in diminishing intensity for the subsequent week. Two weeks later, a few minutes after sexual intercourse, he had a further seizure lasting several minutes, following which he vomited and again complained of severe generalised headache and photophobia. About 16 hours later his left pupil became dilated, remained unreactive to light for about an hour and then returned to normal. No mydriatics had been instilled and there was no associated change in his level of consciousness or headache, no diplopia and no alteration in pulse rate or blood pressure. Examination revealed neck stiffness, positive Kerneg’s sign and mild bilateral papilloedema. A computed tomographic scan showed moderate dilatation of lateral and third ventricles and a small amount of blood in the occipital horns of both lateral ventricles. Bilateral carotid and vertebral angiography showed a large bilobed aneurysm at the bifurcation of the basilar artery projecting to the left. No other aneurysms were seen. At operation (HAC) through a right pterional approach, a bilobed aneurysm, pointing to the left, was identified at the basilar bifurcation. It was directly related to the left third nerve. There was minimal thrombus around the aneurysm but most of the blood clot was within the third ventricle into which the aneurysm had ruptured. A straight Heifetz clip was applied. After operation he returned to his pre-operative clinical state. Three weeks later a ventriculo-peritoneal shunt was inserted for persistent ventricular dilatation and further progress was uneventful.

The cause of the transient pupillary dilatation in this patient may have been
pressure on the third nerve by the basilar aneurysm. The oculomotor nerve is closely related to the bifurcation of the basilar artery, and such aneurysms may cause third nerve palsy. Isolated internal ophthalmoplegia with basilar aneurysm, however, has not been described. Oculomotor palsy characteristically occurs with aneurysms of the posterior communicating artery at the time of rupture or rapid enlargement. Pupillary dilatation without external ophthalmoplegia was reported in a case of Payne and Adamkiewicz in which the mydriasis was present for two weeks before surgery and persisted after operation. Pupillary dilatation may precede other signs in third nerve compression due to uncial herniation as the pupillomotor fibres are situated in an arc superficially on the superior surface of the nerve. Acute hydrocephalus was present in our patient, but there was no coincidental deterioration in his conscious level, neurological state, heart rate or blood pressure. However, oculomotor nerve compression at the tentorial hiatus may have been the cause of the transient mydriasis in the patient reported here, as ventricular dilatation is a known complication following subarachnoid haemorrhage and is caused by blockage of arachnoid villi by blood and breakdown products. Unilateral pupillary dilatation may occur with seizures, but in such cases the mydriasis is accompanied by conjugate deviation of the eyes and occurs during or immediately following the seizure. In our case it occurred 16 hours after the ictus. Transient unilateral pupillary dilatation associated with a clinical picture of subarachnoid haemorrhage therefore may be of some diagnostic value and has not been reported hitherto in association with basilar aneurysm. The present case serves to emphasise the importance of proceeding to vertebral angiography as many aneurysms in the posterior cerebral circulation can now be treated surgically with excellent results.

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References

Recurrent cerebral abscess in association with pulmonary arteriovenous fistulae

Sir: Brain abscess usually occurs as a complication of local or systemic disease and when an abscess is found some underlying cause must be sought. However, in 20-25% of cases no such predisposition is identified. The association of brain abscess and pulmonary arteriovenous fistula was first described in a postmortem study by Reading in 1932. Since then this complication has become well recognised and several reviewers have quoted an incidence of 1-5%, which is as high or higher than the incidence of cerebral abscess in cyanotic congenital heart disease. The characteristic findings of cyanosis, clubbing and an extra-cardiac murmur do not always accompany pulmonary arteriovenous fistula and diagnosis may be extremely difficult as in our patient.

A previously fit 49-year-old housewife was admitted to hospital in December 1977 with a three week history of occipital headache. For two days before admission she had become increasingly lethargic and had developed photophobia. On admission she was drowsy with a fever of 37.7°C. There was neck stiffness. She was mildly ataxic and there was nystagmus to the right. Power was normal but the tendon jerks were brisker on the left with a left extensor plantar response. No other abnormality was noted on general examination. Haemoglobin was 15.9 g/dl. Packed cell volume was 0.50 and red cell count was 5.91 x 10^12/dl. White cell count was 11.00 x 10^9/dl. Plain radiographs of the chest and skull were normal. Computed tomographic scan (CT) of the head did not show any abnormality but did not demonstrate the posterior fossa well. Two days later a vertebral angiogram showed an avascular mass in the right cerebellar hemisphere, and surgical exploration revealed this to be an abscess. Culture yielded a mixed growth of bacteroides, haemophilus and anaerobic streptococci. Antibiotic treatment was commenced with metronidazole, ceftriaxone and benzyl penicillin. The patient was recovering well until the 8th day after operation, when she complained of chest pain and was then noticed to be cyanosed. There was no clubbing of the fingers and auscultation of the chest and heart was normal. A chest radiograph revealed bilateral basal shadowing. Arterial blood gases were Po2 46 mmHg, PCO2 26 mmHg, pH 7.47. A diagnosis of pulmonary embolus was entertained but bilateral phlebo-mograms were normal. The patient improved after further specific treatment and the basal shadowing cleared, but she remained cyanosed and blood gas analysis revealed persistent hypoxaemia with Po2 varying from 50-60 mmHg. Pulmonary angiography revealed multiple pulmonary arteriovenous fistulae throughout both lung fields. The large number of fistulae made surgical resection impossible, and the patient was discharged and advised to take antibiotic prophylaxis before undergoing potentially bacteriemic procedures such as dentistry. She remained well until May 1980 when she was re-admitted to hospital with a two day history of severe headache and transient right hemiparesis, lasting two hours. By the time she was admitted the hemiparesis had recovered completely and there were no other signs apart from a fever of 38.2°C. Haemoglobin was 15.3 g/dl with normal packed cell volume and red cell count, white cell count was 14.000 x 10^9/dl. CT scan showed an area of low attenuation in the left parieto-occipital region with ring enhancement, the appearances being those of a cerebral abscess. Treatment was commenced with benzyl penicillin, flucloxacillin, ceftriaxone and metronidazole. After five days the right hemiparesis recurred and
Transient unilateral mydriasis with basilar aneurysm.

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