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 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 planter 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 × 10⁹/dl. White cell count was 11,000 × 10⁹/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, co-trimoxazole and benzyl penicillin. The patient was recovering well until the 8th day after operation, when she complained of chest pain and she 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 phlebograms were normal. The patient improved without 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 bacteraemic 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 × 10⁹/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, co-trimoxazole and metronidazole. After five days the right hemiparesis recurred and
CT scan showed that the lesion was larger. A left parietal burr hole was made and pus was aspirated from the hemisphere. Antibiotic therapy was continued for three months and the patient made a full recovery once again.

Neurological complications are recorded in about 30% of cases of pulmonary arteriovenous fistulae. Hypoxaemia and polycythaemia may predispose the patient to headache, convulsions and transient ischaemic attacks. The incidence of cerebrovascular accidents in one series of pulmonary arteriovenous fistulae was 6%. Embolism of thrombus formed in the fistula has been reported once. A cerebral arteriovenous malformation may occur in hereditary haemorrhagic telangiectasia, but the co-existence of pulmonary and cerebral arteriovenous malformation has not been reported. Air embolism from the fistula following a bout of haemoptysis has been reported in one case.

Cerebral abscess is an important complication. The mechanism of formation is probably similar to that in congenital cyanotic heart disease. The resistance of the brain to infection may be compromised by hypoxaemia and polycythaemia and by-pass of the pulmonary capillary bed by the fistula, may allow paradoxical septic embolism. The relative importance of these factors is disputed but the rarity of sepsis elsewhere when brain abscess complicates either pulmonary arteriovenous fistula or cyanotic congenital heart disease suggests that the brain is some way predisposed to infection. The commonest symptom of pulmonary arteriovenous fistula is exertional dyspnoea. However, 56% of one large series were asymptomatic. Cyanosis and clubbing are classical features but are not always present. A bruit may be heard overlying the fistula and is usually well localised. It may be continuous or systolic and is made louder by inspiration. Pulmonary arteriovenous fistulae are strongly associated with hereditary haemorrhagic telangiectasia and the characteristic skin lesions may suggest the possibility of underlying pulmonary arteriovenous fistula. However, in hereditary haemorrhagic telangiectasia pulmonary arteriovenous fistula and complicating brain abscess may develop before the skin lesions. Polycythaemia develops consequent on arterial hypoxaemia but may be masked by bleeding. Most pulmonary arteriovenous fistulae appear on the chest radiograph. They are usually well circumscribed round or lobular lesions lying near the pleura, more commonly in the lower zones. The feeding vessels may be seen connecting them with the hilum. Tomography may help discern these features, and pulmonary angiography will almost invariably demonstrate the fistula. In our patient the diagnosis of pulmonary arteriovenous fistulae was unusually difficult. She was asymptomatic and had no physical signs of pulmonary arteriovenous fistula except mild cyanosis, which was overlooked until attention was drawn to it by complications after surgery. She was only marginally polycythaemic and her chest radiograph was normal. The diagnosis was suggested by persistent hypoxaemia and confirmed by pulmonary angiography.

In previous reports of brain abscess complicating pulmonary arteriovenous fistula characteristic clinical and radiological features had led to the recognition of the fistula prior to the development of an abscess. In some cases cyanotic congenital heart disease had been suspected. Recurrent brain abscess in a patient with pulmonary arteriovenous fistula stresses the importance of the association, but this is rare. One previous report described recurrent brain abscess as a complication of pulmonary arteriovenous fistula and hereditary haemorrhagic telangiectasia. In that case telangiectasia and an abnormal chest radiograph suggested the diagnosis and the serious implication of the skin lesions was emphasised. This unusual case is reported as it clearly demonstrates the predisposition to brain abscess that pulmonary arteriovenous fistulae carry and illustrates how difficult detection of the fistulae can be. It is tempting to speculate whether a proportion of hitherto idiopathic cerebral abscesses might have underlying pathology.

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