Spontaneous extradural haematomas

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SYNOPSIS Spontaneous extradural haemorrhage may be due to neighbourhood infections, vascular malformations of the dura mater, and disorders of blood coagulation. Two cases are described here: in one, infection was present; in the other, there was a berry aneurysm of the middle meningeal artery with a small parietal dural angioma. Operation was successful in both patients.

Traumatic extradural haemorrhage occurs in 1.5% of head injuries (Jamieson and Yelland, 1968). In about 83% of cases a fracture line in the skull that is responsible for the bleeding can be demonstrated radiographically (Gallagher and Browder, 1968). In some patients the previous head injury is not apparent because trauma was insignificant, not recent, or because the patient is a young child, lives alone, or there are other circumstances debarring the taking of an adequate history. If no fracture is shown in these

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cases the bleeding may wrongly be considered to be spontaneous. Extradural haematoma should normally be considered to be traumatic even in the absence of known head injury or skull fracture, unless a cause for bleeding can be demonstrated. We exclude those cases of iatrogenic trauma which appear after ventricular tapping or drainage (Fukai and Hasegawa, 1967; Frera, 1969; Sengupta and Hankinson, 1972; and others) or after operations on tumours of the posterior fossa which have caused hydrocephalus (Fiskin and Kurze, 1964), and, in

FIG. 1 Case 2. (a) Lateral and (b) oblique views of right carotid angiogram showing the arteriovenous malformation and aneurysm of the middle-meningeal artery.

general, after any type of cerebral surgery that may produce a brain collapse. True causes of spontaneous extradural haemorrhage are illustrated by the following cases.

CASE 1

A 13 year old boy, without history of injury, was admitted because he had become stuporose 12 hours previously. He had unequal pupils ($R > L$) with bilateral extensor plantar reflexes and no motor deficit or meningeal signs. There was purulent discharge from the right ear. Blood examination showed marked leucocytosis with 'shift to the left',

FIG. 2 Case 2. Anteroposterior view with evidence of the extradural haematoma and the arteriovenous dural malformation.

and increased erythrocyte sedimentation rate. Straight radiographs of the skull were normal. Right carotid angiography demonstrated a temporal extracerebral space-occupying lesion which was avascular.

A large extradural haematoma, which was not infected, was evacuated by right temporal craniectomy. The postoperative course was very satisfactory with total recovery.

CASE 2

This 59 year old woman complained of headache of sudden onset 12 days before admission. There was no history of head injury. Conscious level deteriorated progressively. On examination she was drowsy, disorientated, and had a left hemiparesis. Radiographs of the skull were normal. A right carotid angiogram showed an extradural temporal haematoma and a true berry aneurysm of the middle meningeal artery with a small parietal dural angioma with irregularities in the lumen of the abnormal vessels, and extravascular diffusion of the contrast medium (Figs 1a and b, and 2).

The haematoma was evacuated by right posterior temporal craniectomy. The dura mater bled profusely. Abnormal vessels could be seen in a greater area than was expected from the angiogram. The dura mater was opened and the cerebral cortex looked normal. The postoperative course was uneventful, with full recovery.

DISCUSSION

Three mechanisms are recognized for spontaneous extradural haemorrhage. These are neighbourhood infections, vascular malformations of the dura mater, and disorders of blood coagulation (spontaneous or iatrogenic).

NEIGHBOURHOOD INFECTIONS Only six cases have been attributed to neighbourhood infections. Three cases had chronic otitis (Schneider and Hegarty, 1951; Novaes and Gorbitz, 1965; Clein, 1970), two had frontal sinusitis (Kelly and Smith, 1968; Rajput and Rozdilsky, 1971), and one had orbital cellulitis after a furuncle of the ala nasi (Schneider and Hegarty, 1951). The present case 1 is an example of this type.

Two mechanisms have been proposed to account for bleeding in the extradural space. (1) Arteritis may weaken the wall of meningeal vessels. This is supported by radiological, surgical, or necropsy evidence of involvement of contiguous bone structures (focal osteitis). Generally, the affected vessel is of small calibre and the clinical course is subacute, but in one patient there was necrosis at the foramen spinosum with abrupt rupture of the main trunk of the middle meningeal artery and acute onset of symptoms (Schneider and Hegarty, 1951). (2) Progressive detachment of the dura mater from the inner table of the skull may be caused by

accumulation in the extradural space of exudate and pus from the infected area, or of air through a bone defect from the middle ear, mastoid cells, and paranasal sinuses (Novaes and Gorbitz, 1965). This mechanism is well known in extradural haematomas associated with diffuse venous bleeding caused by traumatic pneumatocele.

It is important to be aware of this complication because some patients of this type follow a rapidly fatal course. Progressive stupor and focal neurological signs are often misinterpreted as being due to cortical thrombophlebitis, subdural or extradural empyema, or even brain abscess, since carotid angiography shows an avascular space-occupying lesion.

VASCULAR MALFORMATIONS OF DURA MATER
Vascular dural malformations are very rare. We exclude false aneurysmal dilatations of meningeal arteries associated with a fracture line, in which there is no question of the traumatic onset (Paillas *et al.*, 1964; Lepoire *et al.*, 1965; Pellet *et al.*, 1971; and others). This category includes only arteriovenous malformation of congenital origin. Our case 2 is a further example.

In a cooperative study (Sahs *et al.*, 1969), only seven dural arteriovenous malformations were found in 549 intracranial angiomas. There are few isolated reports of congenital arteriovenous malformations of the dura mater. According to Newton *et al.* (1968) and Newton and Cronqvist (1969), this type is found much more often when systematic selective external carotid angiograms are performed.

Some of these malformations cause little trouble to the patient and may pass unnoticed. The most common clinical manifestations are intracranial bruits (65%), headaches (30%), epileptic seizures (13%), hydrocephalus, and, less frequently, visual troubles, motor weakness, cardiac failure, etc. (Fernández Urdanibia *et al.*, 1974). They have little tendency to bleed and, when they do, the haemorrhage extends towards the subdural or subarachnoid space. Only one case has been reported of extradural haemorrhage produced by a dural arteriovenous malformation (Gallagher and Browder, 1968). Several authors have described the extravasation of contrast medium during angiography in extradural haemorrhage (Vaughan, 1959; Leman *et al.*, 1964; Helmer *et al.*, 1968). In our case 2

this leakage was considered to indicate danger of rebleeding and this, with the poor neurological status, caused us to perform an emergency operation without selective external carotid angiography or study of the vertebrobasilar vascularization which might have demonstrated a larger malformation.

DISORDERS OF COAGULATION Extradural haematoma due to a defect of blood clotting is a theoretical possibility but we have not found an example in a survey of the world literature. The incidence of intracranial haemorrhage in patients undergoing anticoagulant therapy is well known (2% of cases according to Kubicek and Praschl, 1968) but must be exceptional in the extradural space. In 124 cases of intracranial haemorrhage during administration of anticoagulants, Lizuka (1972) found 82% in the subdural space, 18% intracerebral, and none extradural. We have found only one case of extradural haematoma after extracorporeal cardiac surgery (Hoffman and Mustard, 1973). Although anticoagulants may play an important role, a decisive factor in localizing the haemorrhage could be the collapse of the brain produced by hypothermia with subsequent detachment of the dura mater.

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