Letters

has remained well almost a year after his first presentation (fig c).

Eleven per cent of extradural haematoma occur in the frontal region and there is experimental evidence to suggest that dural separation may occur at impact with subsequent formation of clots. This was probably the mechanism of formation of the first haematoma in the case described. It is the formation of the subdural collection for which there are a number of possible explanations. More than a third of patients with an extradural haematoma have an associated intradural haematoma. Delayed intracerebral haematoma are known to occur after evacuation of chronic extracerebral collections. There is probably no single aetiological factor but mechanisms such as focal ischaemia and rapid shift have been implicated. In this case, it is unlikely that these mechanisms could be used to explain the formation of a delayed subdural hygroma. The arguments against this would be: (a) no loss of consciousness at presentation, (b) the long duration between injury and presentation of the initial haematoma, (c) no evidence of associated clot or focal damage on the initial scan, (d) the brain remained slack at the end of the first operation with no discolouration of the dura, (e) the patient was neurologically normal immediately after operation for 48 hours.

Another possible explanation might be that the dural hitches may have caused cortical or cortical venous injury during insertion. We believe that this was not the case because the patient made a full recovery and was well for the first three days post operatively. The brain showed no signs of injury at the second operation when the dura was widely opened and the cortex inspected; the subdural fluid at the second operation was straw-coloured with no clots or blood seen. The mechanism of formation of this patient’s subdural hygroma is obviously a rare one, and probably resulted because of the long duration of contact between the dura and arachnoid, resulting in a tearing of the arachnoid and escape of CSF subdurally on insertion of the “Poppen” suture. A small tear could then have resulted in a “flap valve” mechanism leading to further accumulation of hygroma fluid ultimately causing pressure effects. Because the brain had remained depressed for 48 hours prior to the first operation, we believe that the insertion of the “Poppen” suture resulted in the formation of an ex vacuo subdural hygroma which needed surgical evacuation. When using the “Poppen” suture, we suggest that this possible complication be borne in mind.

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Fig (c) Follow up CI, showing complete evacuation of the clot.

References


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Traumatic intracranial aneurysms and fistula associated with epidural haematoma

Sir: Traumatic intracranial aneurysms and fistulas, whether due to blunt trauma, penetrating trauma or trauma at the time of operation, are rare and only 124 cases have been reported in the literature. However, a combination of a pseudoaneurysm of the middle meningeal artery, an aneurysm of the prerolandic branch of the middle cerebral artery and an arteriovenous fistula between a branch of the middle cerebral artery and a cortical draining vein associated with epidural haematoma in the same individual has not been reported. Recently, we had an opportunity to treat such a patient; the details of this are described and the pertinent literature reviewed.

A 59-year-old man was admitted to our unit having been knocked down by a minibus. He was unconscious for approximately five minutes. Examination on admission revealed multiple superficial bruises and abrasions over both knees and left elbow. He was fully conscious, orientated and cooperative. Only a mild degree of nominal dysphasia was evident. There were no localising neurological signs. Radiographs of the skull revealed a linear fracture. Next morning he was found to be well, apart from nominal dysphasia, and was discharged home. Two weeks later he was re-admitted with the complaints of difficulty in finding words, and not being able to express himself even though he knew what he wanted to say. On examination again a moderate degree of motor dysphasia was evident. There were no other abnormal neurological signs and there was no papilloedema. CT scan could not be performed owing to technical difficulties.
Next morning, while waiting for carotid angiography, he suddenly became comatose (Glasgow coma scale 8). Both pupils were mid-dilated, equal and nonreactive to light. Right hemiparesis was evident with extensor plantar response. An urgent biplane left carotid angiogram was performed. Anteroposterior films (fig) revealed a large avascular space-occupying lesion strongly suggestive of epidural haematoma on the left side with shift of the midline structures. Also, two aneurysms (a and b) were evident. One of these aneurysms, (a) was thought to originate from the left middle meningeal artery and the other, (b) from a branch of the middle cerebral artery. The rest of the vasculature of the left carotid artery was entirely normal; however, the right carotid and vertebral angiograms were not performed. Lateral films, in addition, showed the presence of a linear fracture. Postoperative review of the angiograms, however, revealed the presence of a vein (v) filling during early arterial phase which was not detected before the operation. Also, in view of the operative findings it was possible to confirm that the aneurysm (b) was indeed originating from a peripheral branch of the left middle cerebral artery.

The patient was immediately taken to the operating theatre. On removing approximately 150 ml of unorganised epidural blood clot, a globular swelling at the bifurcation of the middle meningeal artery was noted. It was approximately one centimetre in size and enclosed within densely adherent blood clot. On opening the dura, a cortical artery was found to be in communication with a cortical vein directly beneath a tear of the dura. The vein was arterialised and the site of the communication was surrounded by a small blood clot. When attempting to remove the blood clot, the communication gave way. Bleeding was controlled by coagulation of the bleeding point. Further exploration revealed an aneurysm about 4 mm in size, projecting laterally from a branch of the middle cerebral artery. The aneurysm was easily clipped at its neck.

The postoperative course was uneventful. Dysphasia started to improve and within 48 hours of the operation he could converse. No trace of dysphasia or hemiparesis could be detected four days later. He was discharged home ten days after the operation. Since then he has regularly reported for follow-up and remains well.

The first case of pseudoaneurysm was reported by Smith.6 While describing difficulties attending the surgical diagnosis of aneurysms and medical jurisprudence, he briefly noted the case of a boy who received a blow on the left temple. Subsequently, when a tumour developed, excision was attempted. The extirpated sac was found to be an aneurysmal sac originating from the middle meningeal artery. Since then 24 additional cases of pseudoaneurysm have been reported.2 3 5 Twenty of the 25 cases were associated, as in our case, with a fracture of the skull and thirteen showed neurologic deterioration from 3–30 days after receiving head trauma. Furthermore, of the 25 patients, 24 required surgical intervention for intracranial haematomas (16 epidural, six intracerebral and two subdural). One patient was operated upon for symptomatic pseudoaneurysm and fistula. A traumatic pseudoaneurysm is therefore prone to rupture and it carries a mortality rate of approximately 20%.1 However, delayed but sudden loss of consciousness and rapid development of localising neurological signs following head injury, as in our case, are pathognomonic of ruptured pseudoaneurysm. This should pave the way for early diagnosis and treatment.

Fistula between the middle meningeal artery and a dural sinus following head injury was first reported by Fincher.7 Since then 39 cases of fistula have been reported.1 5 Of the 39 cases, 26 were associated with intracranial haematomas and lacerations of the brain. The majority of the cases showed immediate or early post-traumatic neurologic deterioration, with at least 10 of the patients dying shortly after the injury. Neither gross cerebral damage nor contusion were evident in our case. The aetiological factor may be the damage of the adjacent cortical artery and vein caused by the depression of sharp edge of fractured bone.

Traumatic aneurysms of the peripheral cerebral arteries have been reported following blunt head trauma (62%), penetrating trauma (27%) and iatrogenic trauma (11%). A recent review revealed 60 cases.8 Associated fracture of the skull was observed in only 11 patients, subdural haematoma in 17, and a combined subdural and epidural haematoma in only one patient.2 Nearly half of the reported traumatic cerebral aneurysms bled, usually 15 days after injury and haemorrhage was fatal in approximately 50% of the cases.5 9 In our case there was no evidence of bleeding from the cerebral aneurysms.

The clinical presentation of the case is interesting and difficult to explain. Nominal and motor dysphasia might have been both due to compression of motor speech area by the slowly accumulating epidural blood clot, and the "steal phenomenon" due to arteriovenous fistula.1 However, the absence of additional progressive neurological signs until the day of operation, the lack of organisation of haematoma, the demonstration of arteriovenous fistula and the operative findings of arterial vein, would all
favour fistula as the cause of dysphasia rather than chronic epidural haematoma. Besides, the sudden progression of neurological signs 14 days after injury may be attributed to acute epidural haematoma due to ruptured pseudoaneurysm. This was the presentation in over half the reported cases of pseudoaneurysm, particularly in those in whom neurological deterioration occurred at an average of 11 days after the time of initial injury. 

Coincident existence of an arteriovenous malformation and mycotic aneurysm of the peripheral cerebral artery were considered as the differential diagnosis. However, in the absence of preoperative history of meningitis, cardiac disease and epilepsy, these possibilities were considered unlikely. Besides, both of these pathologies of this patient showed evidence of trauma to the adjacent bone, dura or cerebral tissue, thus implicating trauma as their sole cause.

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A case of Parkinsonism following striatal lacunar infarction

Sir: The concept of vascular Parkinsonism is a controversial one. Critchley first introduced the concept in 1929 when he proposed that Parkinsonism due to vascular disease could be distinguished from idiopathic Parkinson's disease on the basis of clinical and pathologic criteria. 

However, he did not present individual case data, either clinical or pathological, to demonstrate convincingly how to distinguish these two conditions or to exclude other recognised causes of Parkinsonism. Subsequently, other authors have questioned the existence of vascular Parkinsonism as a distinct entity, based on a lack of clinical, 

epidemiological 

and pathological 

evidence. Nevertheless, there are authorities who accept vascular Parkinsonism as a recognisable condition, generally associated with chronic hypertension and clinical evidence of multiple small lacunar infarctions. 

A diagnosis of a vascular aetiology of Parkinsonism in a particular case would be acceptable if the following criteria were met: (1) the Parkinsonian signs were acute in onset; (2) the signs showed spontaneous improvement; (3) other known causes of Parkinsonism were excluded; (4) pathological examination failed to show either neuronal loss in the substantia nigra or Lewy bodies, but did show vascular lesions affecting the corpus striatum. In the absence of histopathologic evidence, vascular Parkinsonism cannot be diagnosed with certainty, but it can be considered the probable diagnosis if the clinical criteria are met, and there is neuroradiologic evidence of striatal infarction. We recently encountered an example of probable vascular Parkinsonism, which supports belief in the existence of this condition.

This 37-year-old left-handed man with a 4-year history of untreated hypertension had sudden onset of weakness of the right arm and leg, slurred speech and right peripheral numbness. His speech quickly returned to normal and he regained most of his strength in two hours. He denied difficulty with comprehension or formulation of language. He had no history of diabetes mellitus, heart disease or cigarette smoking. He had never taken antipsychotic drugs. On examination his blood pressure was 180/100 mm Hg. He had diminished facial expression. Cranial nerves were intact. There was mild rigidity of the neck, more pronounced rigidity in the right arm and leg with cogwheeling in the right arm, and very slight rigidity of the left arm and leg. Rapid successive movements were diminished and slow on the right side and less so on the left side. Strength was normal. Deep tendon reflexes were brisk bilaterally, more so on the right. A Babinski response was present on the right side. Sensory examination was normal. He walked without swinging his right arm. There was postural instability with retropulsion. Cerebellar testing was normal. CT scan (fig) showed generalised mild to moderate atrophy, bilateral putaminal lucencies and a right cerebellar hemispheric lucency. A four-vessel cerebral angiogram was normal. Cerebrospinal fluid was normal. The level of homovanillic acid (HVA) in the cerebrospinal fluid was 41 ng/ml; the mean value for Parkinson's disease patients in our laboratory is 25·3 ± 12·3 ng/ml.

He improved gradually over the next 10 days in hospital. After discharge he slowly improved further without any anti-parkinsonian medication, with residual rigidity on his right side. Three months after discharge he noted sudden weakness of all extremities and slowing of his gait. Ten minutes later he noted numbness of his right foot and right hand. All these symptoms resolved in about one half hour. His blood pressure was 150/100 mm Hg. There was no change in his neurological state. A repeat CT scan showed no new lesion. By the next day he was less rigid.

Several features of this case make the diagnosis of vascular Parkinsonism possible.

Fig  CT scan showing bilateral putaminal lucencies (arrows) with a larger one on the left and moderate generalised atrophy.
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