Idiopathic Haemorrhage Cerebellar Infarction In an 11 Year Old Child.

Sir - Haemorrhagic strokes in childhood are very rare. Whilst a number of causes are recognised, many unique to the paediatric population (vide infra), up to 25% of cases are idiopathic. In any age group, oedema associated with cerebellar infarction, unlike in the supratentorial compartment, can lead to a sudden demise by brainstem compression. Emergency posterior fossa decompression by occipital craniectomy may thus provide a lifesaving measure in selected cases.

We report a case of idiopathic cerebellar haemorrhagic infarction in an 11 year old girl who was managed surgically, and comment generally on the value of neurosurgical intervention for cerebellar haemorrhagic infarction.

Case Report

An eleven year old girl sustained a severe occipital headache of sudden onset followed by collapse. Her past medical history was unremarkable with normal development and no history of substance abuse or bleeding disorder. Prior to sedation and intubation at the referring hospital, she exhibited a flexor response to painful stimuli, no eye opening and no verbal response. Her pupils were equal and reacting to light and there was marked neck stiffness. CT brain scan performed at the referring hospital 4 hours post ictus showed bihemispheric haemorrhagic infarction with concomitant obstructive hydrocephalus (Figure 1). Four vessel digital subtraction angiography was normal.

At operation, a right frontal external ventricular drain was inserted prior to posterior fossa decompression by occipital craniectomy. Diffuse cerebellar oedema was encountered with liquefaction and haemorrhage. These findings were confirmed by subsequent histological examination.

She was extubated on the third post operative day and external ventricular drainage was dis-

continued on day 5. Parenteral feeding was instituted for 3 weeks on account of persistent vomiting. At discharge, 7 weeks post operatively, she was alert, orientated but withdrawn. At 4 months post ictus, she had fully recovered.

The following investigations were performed post operatively: MRI brain and CSF analysis, both of which revealed post operative changes with no other abnormality; platelet count, INR and APTT ratio, fibrinogen level, antithrombin III, protein S, prothrombin C and factor Xa levels; echocardiography and metabolic screen, all of which were within normal limits.

Discussion

This case demonstrates a favourable outcome following aggressive management of a rare problem. Recognised causes of childhood haemorrhagic strokes include the following: vascular (eg. congenital heart disease, moya moya disease), metabolic (eg. homocysteinuria, MELAS syndrome, sulphite oxidase deficiency, urea cycle defects), haematological (eg. thrombophilia: protein C/protein S/antithrombin III deficiency, sickle cell disease).

Whilst most would recognise the value of surgically evacuating a cerebellar haematoma, the practice of decompressive craniectomy for infarction alone has not gained universal acceptance. We believe that an infarctive process within the posterior fossa can produce a fatal outcome by three mechanisms: firstly, by inclusion within the infarctive field of the vital brainstem structures, secondly, by oedema causing hydrocephalus and thirdly, by cerebellar oedema causing compression of a brainstem which is not itself involved in the infarctive process. Thus, for persistent coma following relief of hydrocephalus in a patient with a distal cerebellar haemorrhagic infarct and significant mass effect but no overt brainstem involvement on routine imaging, we propose that an occipital craniectomy and evacuation of haematoma should be expeditiously performed, particularly in the younger age group.

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References
