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Short reports

Endoscopic evacuation of an intracerebral and intraventricular haemorrhage

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SUMMARY An intracerebral and intraventricular haemorrhage associated with acute post-haemorrhagic ventricular dilatation was evacuated under endoscopic view in a 9 day old preterm infant. Ventricular dilatation was arrested, but a moderate left spastic hemiparesis remained. The physical and mental development of the baby was normal.

Intracerebral and intraventricular haemorrhage occurs in up to 43% of very low birthweight infants,[1] and their prognosis is poor because of acute brain damage and post-haemorrhagic hydrocephalus.[1–3] We were unable to find any previously published reports of endoscopic evacuation of an intracerebral and intraventricular haemorrhage in neonates.

Case report

A boy weighing 1410 g was delivered by caesarean section at 30 weeks' gestation. The Apgar score was 4 at one minute and (after intubation) 8 at three minutes. Respiratory support was continued owing to wet lung. A routine echoencephalogram performed five hours after birth was normal and the baby was neurologically unremarkable. His condition deteriorated 43 hours later. While he was on continuous positive airway pressure he had repeated spells of severe apnoea with cyanosis and bradycardia, apathy, and hypotonia as well as loss of prompt responses to stimulation which he had shown before. At this time brain ultrasonography showed an extensive haemorrhage in the right frontoparietal region with displacement of the midline structure and blood in the lateral ventricles. Subsequent ultrasound examinations showed that the extent of the haemorrhage increased until the seventh day and that a rapid dilatation of the ventricles took place. The computed tomography (CT) scan findings on the eighth day are shown in fig 1a. The neurological state was unchanged. There were no spontaneous movements and there were poor reactions to stimuli. Spontaneous breathing was infrequent and intermittent mandatory ventilation was therefore continued.

Because of the relatively stable neurological state and vital signs, we decided to evacuate the intracerebral blood. We anticipated the procedure might lead to a reduction in the intracranial pressure as well as arresting the ventricular dilatation. We assumed that the dilatation was at least partially caused by occlusion of the interventricular foramina

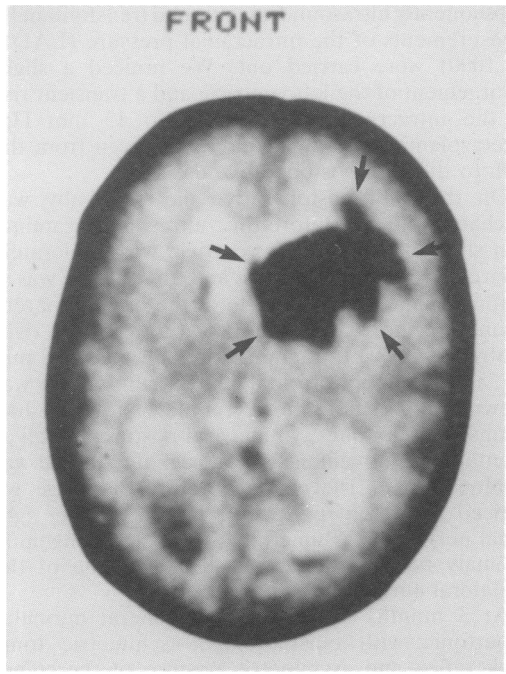

Fig 1a *Computed tomogram one day before surgery. Blood clot is situated in right anterior horn and periventricular white matter (indicated by arrows).*

by blood clot. On the ninth day, after general anaesthesia, a right frontal trepanation (1 cm) was performed. A neuroendoscope with a built-in neodymium YAG laser (Storz type 26162 B) with an external shaft of 6·0 mm in diameter was introduced under sonographic control. A large part of the parenchymal portion of the haemorrhage was removed using endoscopic channels for irrigation and suction. The ventricular system was then entered and inspected and blood clot was removed from the anterior horn and cella media of the right lateral ventricle as well as the right interventricular foramen.[4 5] Finally an external drain was placed into the right anterior horn which permitted continuous measurement of intracranial pressure and cerebrospinal fluid production.

Six hours after surgery the patient's neurological state had improved considerably. He had normal muscle tone, prompt responses to painful stimuli, symmetrical movements, and regular breathing; intermittent mandatory ventilation was therefore discontinued. Over the next six days intracranial pressure was no more than 8 mm Hg, and ventricular fluid secretion averaged 6 ml/day (both measured by the external drain). The computed tomogram taken on the seventh postoperative day (the drain having been removed a day before) is shown in fig 1b. Subsequently ultrasound imaging and transfontanelle measurements of the intracranial pressure (LADD M 1000) were carried out. We noticed a slight enlargement of the left ventricle and a transient rise of the intracranial pressure up to 15 mm Hg. Acetazolamide (50 mg/kg/day) was given from the 12th to the 40th postoperative day.

On the 46th postoperative day the baby was discharged home. Before this, ultrasound examination showed that the left ventricle was moderately enlarged (6 mm in width) and that there was a porencephalic cyst at the site of the original parenchymal haemorrhage communicating with the right ventricle (fig 2). Intracranial pressure was 6–8 mm Hg, and the occipitofrontal head circumference was growing along the 25th percentile. The baby had normal muscle tone, symmetrical posture as well as spontaneous movements, good sucking, and he was gaining weight. Eleven days before discharge we noticed a discrete peripheral paresis of the right facial nerve. We assumed that this was a congenital anomaly because there was also dysplasia of the ipsilateral auricle.

At 3 months of age he had general muscular hypertonia with opisthotonus, asymmetric tonic neck reflex and asymmetric posture of the spine; intensive physiotherapy was started. At 5 months he was following moving objects, grasping only with the right hand, and crawl activity of the left leg was

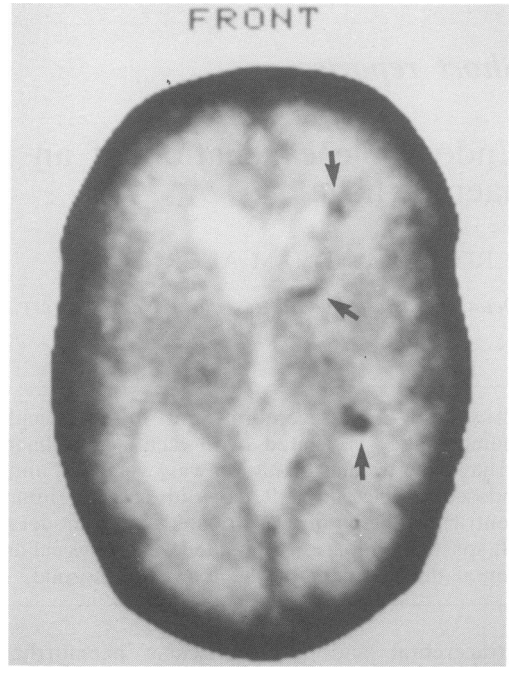

Fig 1b *Computed tomogram seven days after surgery. Remaining blood clot is indicated by arrows.*

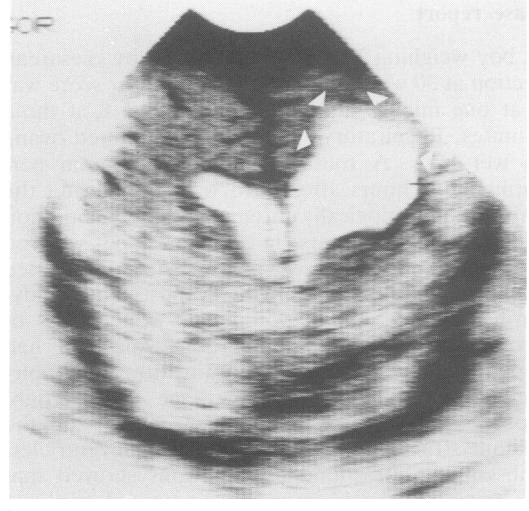

Fig 2 *Coronal ultrasound scan of the brain before discharge home. Note porencephalic cyst at site of original parenchymal haemorrhage on the right side (arrows). Left ventricle is moderately enlarged.*

weak. At 9 months the spastic hemiparesis on the left side became more evident. Head circumference continued to grow along the 25th percentile, and echoencephalography confirmed that the width of the left ventricle and the porencephalic cyst had not increased in size. At 1 year the child was alert, was able to call 'daddy', and attempted to stand. He could walk freely at 16 months with a slight limp in the left leg.

Discussion

Large intracerebral and intraventricular haemorrhage has been reported to be fatal in 76–100% of cases.[1 2] Virtually all surviving patients have moderate to severe neurological deficits,[1] and up to 58% of them develop hydrocephalus.[1] It is unclear how much of the neurological deficit after such a haemorrhage is due to immediate parenchymal damage and how much is due to hydrocephalus.[1 6] Shunt complications can make matters worse.

Directly evacuating the parenchymal haemorrhage and intraventricular blood clots theoretically reduced two pathogenetic mechanisms. Firstly, pressure reduction alleviated ischaemic damage to the perifocal parenchyma; the rapid neurological improvement of our patient six hours postoperatively might support this. Secondly, evacuation of the blood clots possibly helped to arrest developing hydrocephalus which might have been produced by temporary occlusion of the interventricular foramina. As the dilatation of the ventricles was arrested at a moderate stage, definitive shunting or lumbar punctures were not necessary.

It is impossible to determine whether evacuation of the haemorrhage influenced the outcome at 16 months. Potential benefit of the procedure may be confined to the rapid relief of abnormal neurological signs in the acute stage of intracerebral and intraventricular haemorrhage, or it may also reduce the incidence of progressive post-haemorrhagic obstructive hydrocephalus.

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Atrial natriuretic peptide and patent ductus arteriosus in preterm infants

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SUMMARY Preterm infants with symptomatic patent ductus arteriosus had considerably raised plasma concentrations of atrial natriuretic peptide. Surgical ligation of the patent ductus arteriosus was associated with an immediate fall in plasma atrial natriuretic peptide concentration. Thus left to right shunting and left atrial distension may cause atrial natriuretic peptide release in preterm infants with patent ductus arteriosus.

The existence of a natriuretic hormone released by atrial distension and volume expansion was postulated more than 30 years ago. Recently such a hormone, atrial natriuretic peptide, was discovered.[1] It is produced by mammalian atriae, stored in granules of atrial myocytes and released into the circulation when volume expands and the atrium distends.[1 2]

Preterm infants with symptomatic patent ductus arteriosus are a good group in which to study atrial natriuretic peptide release. In these infants arterial blood is shunted from the aortic root into the pulmonary circulation leading to an increase in pulmonary and left atrial blood volume and to a considerable rise in left atrial pressure.[3] Thus if atrial distension is a major stimulus for atrial