Posthaemorrhagic hydrocephalus in newborn term infants

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SUMMARY Intraventricular haemorrhage may occur de novo in previously well, term newborn infants. In a group of six neonates a high incidence of posthaemorrhagic hydrocephalus was found. Posthaemorrhagic hydrocephalus may be more common in full term newborn infants than previously recognised.

With the increased use of two dimensional real time ultrasound scanning periventricular haemorrhage has been recognised more often in the neonatal period of very low birthweight infants. The origin of the bleeding is the vascular subependymal germinal matrix, an anatomical structure that diminishes in size, with little present at term. In a proportion of infants, especially when there is a bleed of major degree (grade 3 or 4 on Papile's scale), secondary hydrocephalus develops, and a shunt is required.

Several cases of haemorrhage originating in the choroid plexus, caudate nucleus, and thalamus have been recognised in term babies. In these more mature babies periventricular haemorrhage has been noted in the absence of precipitating factors that are recognised in very low birthweight infants and presentation is often delayed until the infant has been discharged from the maternity hospital. Reports on such babies have found that progressive hydrocephalus developed in a minority of cases.

Over an 18 month period we noted a series of six term babies in whom diagnostic confusion initially existed. A high incidence of posthaemorrhagic hydrocephalus followed.

Case 1

A boy weighing 4340 g was born at 39 weeks' gestation after a normal pregnancy and delivery. Apgar scores were 9 and 10 at 1 and 3 minutes, respectively. Vitamin K (1 mg) was given intramuscularly after birth. He was discharged home with his mother at 4 days but was readmitted at 11 days with a 24 hour history of loose stools, vomiting, irritability, and poor colour. On examination he was grey and mottled with a full fontanelle, stiffness of the neck, and increased muscle tone.

It was not possible to obtain cerebral spinal fluid on admission, and the baby was treated for neonatal meningitis. Biochemical and haematological indices were within normal limits. Cerebral spinal fluid fluid subsequently obtained by lumbar puncture, contained a red blood cell count of 260×10⁶/l, white blood cell count 0.48×10⁹/l, protein concentration 1.3 g/l, glucose concentration 2.4 mmol/l, and sterile culture. Ultrasound scanning showed intraventricular haemorrhage with normal ventricular size. Subsequent ultrasound scans and computed tomograms confirmed the intraventricular haemorrhage and showed increasing ventricular size (Fig. 1 (a) and (b)).

At 5 weeks of age, over a 48 hour period, he developed symptoms of increasing intracranial pressure with an increase in circumference of his head and ventricular dilatation. A ventriculoperitoneal shunt was inserted. By 7 weeks of age symptoms had settled and he had no detectable neurological abnormality.

Case 2

A 3520 g girl was born at 42 weeks' gestation after normal pregnancy and delivery. Apgar scores were 9 and 9 at 1 and 5 minutes, respectively. Vitamin K (1 mg) was given intramuscularly after birth. During the first day she had an episode of cyanosis and vomited several times. She was discharged home with mother at 4 days of age but was readmitted two days later with poor feeding, irritability, and a brief left sided seizure. On examination she was having intermittent fits, the anterior fontanelle was tense,
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Fig. 1 Computed tomogram (a) and coronal ultrasound scan (b) for case 1.

Fig. 2 Computed tomogram for case 2.

Fig. 3 Computed tomogram for case 3.

circumference of her head was 36.5 cm, her posture opisthotonic, and the muscle tone increased in all limbs. Initial diagnoses considered included septicaemia and non-accidental injury. Intravenous antibiotics were given.

Investigations showed heavily blood stained cerebral spinal fluid with a red blood cell count of 200×10⁹/l, white blood cell count 4.8×10⁹/l, and sterile culture. Other biochemical and haematological indices were normal. Ultrasound scanning showed bilateral intraventricular haemorrhage with dilatation of the ventricles; the appearances were confirmed by computed tomogram (Fig. 2).

Serial ultrasound scanning showed an increase in ventricular size with the head growing along the 90th centile. At the age of 2 months the infant remained under review.

Case 3

A 4370 g boy was delivered at term to a 16 year old mother after normal pregnancy and delivery. Apgar scores were 9 and 9 at 1 and 5 minutes, respectively. Vitamin K (1 mg) was given intramuscularly after birth. The baby was readmitted from home at 8 days with fever and increasing head circumference (from 37.4 cm at birth to 38.5 cm on day 8). On examination the baby appeared well but was febrile with a tense fontanelle. Lumbar puncture showed dark brown fluid with a red blood cell count of 64×10⁹/l and white blood cell count of 4×10⁹/l with low cerebral spinal fluid sugar concentrations (0.5 mmol/l) and increased protein concentrations (12.3 g/l). A provisional diagnosis of meningitis was made and parenteral antibiotics given. No positive results were obtained from bacterial or viral cultures. Repeat lumbar puncture on day 12 showed xanthochromic fluid.

From day 14 the baby vomited intermittently despite feeding well. An ultrasound scan showed increasing ventricular size, and the baby was transferred to a surgical unit. At 19 days the occipitofrontal circumference was 40.5 cm and the fontanelle tense. Computed tomogram confirmed residual blood clot in the right lateral ventricle with enlargement of lateral, third, and fourth ventricles (Fig. 3). A ventriculoperitoneal shunt was inserted on day 28.
The diagnoses considered before computed tomography were meningitis, non-accidental injury, and aqueduct stenosis.

The Table and Figs. 4 and 5 give the clinical details of three other infants with neonatal intraventricular haemorrhages that progressed to hydrocephalus.

**Table Clinical details of cases 4, 5, and 6**

<table>
<thead>
<tr>
<th>Case no</th>
<th>Birthweight (g)</th>
<th>Gestation (weeks)</th>
<th>Apgar score at one and five mins</th>
<th>Age at presentation (days)</th>
<th>Clinical features</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>4</td>
<td>3630</td>
<td>41</td>
<td>7, 9</td>
<td>6</td>
<td>Seizure, shock, tense fontanelle, spasticity</td>
<td>Vasoperitoneal shunt at 21 days</td>
</tr>
<tr>
<td>5</td>
<td>2540</td>
<td>36</td>
<td>7, 9</td>
<td>9</td>
<td>Vomiting, opishtotonus</td>
<td>Vasoperitoneal shunt at 28 days</td>
</tr>
<tr>
<td>6</td>
<td>3380</td>
<td>40</td>
<td>8, 10</td>
<td>2</td>
<td>Seizures, irritability, crying</td>
<td>Vasoperitoneal shunt at 28 days</td>
</tr>
</tbody>
</table>

**Discussion**

Reports of intraventricular haemorrhage in term babies link the development of haemorrhage with preceding perinatal conditions. In the infants that we studied intraventricular haemorrhage of classic appearance by ultrasonography and computed tomography developed in the absence of evident perinatal or postnatal trauma, asphyxia, or other precipitating factors. Five of the six infants were male, the average age at presentation was 7 days, and five of six were readmitted after discharge from the maternity hospital. Initial investigation by real-time ultrasound scanning, followed in three infants by computed tomography, delineated the extent of the haemorrhage. No evidence of vascular malformation was seen, though angiography would be required to exclude completely this possibility. No infant bled further, and the origin of the intraventricular haemorrhage remained unclear.

The site of bleeding in intraventricular haemorrhage in term infants is often unknown. It has been reported in association with haemorrhage from the germinal matrix, choroid plexus, caudate nucleus, and thalamus.

In two of our infants (case 4 and 5) attempts to control accumulation of cerebral spinal fluid by daily lumbar puncture were unsuccessful. All babies showed evidence of rapidly increasing ventricular size on serial ultrasound scanning. In addition, five had features of raised intracranial pressure, and in these babies a drainage procedure was performed. One infant (case 2) at 2 months of age had a head circumference growing along the 90th percentile and has remained under observation.

Follow up of term neonates with intraventricular haemorrhage has indicated that the Papile grading system, which has predictive value for outcome in preterm babies, is of less value in term babies. Grade III haemorrhage was present in four of five babies (cases 2, 3, 4, and 5) and Grade IV in one (case 6). No infant died, and all made satisfactory progress once the hydrocephalus had been con-
trolled. The follow up period, however, was less than one year in all infants.

Previous reports of intraventricular haemorrhage in term infants have shown a variability in clinical picture and outcome. They include the ‘primary’ intraventricular haemorrhages occurring in infants with no recognisable preceding asphyxial or traumatic insult and also those after asphyxia at birth, postnatal dehydrating illness, and non-accidental injury. From such a heterogeneous group it is difficult to predict subsequent outcome or incidence of posthaemorrhagic hydrocephalus. Scher’s summary of reported studies included 10 cases similar to those in our series. Of these, four required ventriculoperitoneal shunt insertion, one had arrested hydrocephalus, and one died. In two of four cases of thalamic haemorrhage with intraventricular haemorrhage, ventriculoperitoneal shunt insertion was required.

Intraventricular haemorrhage in term infants appears aetiologically and prognostically different to that seen in very low birthweight infants who sustain haemorrhages of equivalent size. The presence of increasing hydrocephalus requires early diagnosis, close monitoring, and prompt decompression. Early treatment may be an important factor for the neurodevelopmental outcome of this group of infants.

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References


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