CASE REPORT

Repeated antenatal intracranial haemorrhage: magnetic resonance imaging in a fetus with alloimmune thrombocytopenia

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Magnetic resonance imaging was used to show repeated antenatal intracranial haemorrhage in a fetus with alloimmune thrombocytopenia.

CASE REPORT

A healthy 23 year old primigravida was referred by her midwife at 34 weeks gestational age for ultrasonography because the fetus appeared to be small on clinical examination. The ultrasound showed a normal sized fetus. However, there was unilateral irregular ventriculomegaly, dysplasia of the inferior vermis of the cerebellum, enlarged cisterna magna, and a lesion suggestive of a porencephalic cyst. Subsequent fetal magnetic resonance imaging (MRI) showed considerable enlargement of cisterna magna with reduction in the size of the cerebellum. There was dilatation of the left lateral ventricle with almost certain communication with the subarachnoid space (fig 1), the appearance being that of schizencephaly rather than a porencephalic cyst. On the right side there was an intracerebral lesion with mixed signal on both T1 (fig 1) and T2 (fig 2) weighted sequences. There was also hypoplasia of the cerebellum. In summary, there were several different lesions suggestive of separate different times of events. The lesion on the right was consistent with a recent haemorrhage. The lesion on the left and the hypoplasia of the inferior vermis were suggestive of injuries early in pregnancy.

The diagnosis of neonatal alloimmune thrombocytopenia was confirmed by the presence of HPA 1a antibodies in maternal serum.

DISCUSSION

Neonatal alloimmune thrombocytopenia is caused by transplacental passage of maternal platelet specific antibodies leading to destruction of fetal platelets. Published estimations of incidence range from 1.5 per 1000 neonates to 1 per 5000 neonates. Most intracranial haemorrhages occur in the third trimester, but early cases have been reported, possibly recurrent. The most common site of intracranial haemorrhage is beneath the cerebral cortex, with possible expansion to a subarachnoid bleed and dissection through the brain reaching the ventricles, resulting in the appearance of schizencephaly.

The MRI appearance in our patient was consistent with several separate events. Diagnosis was not made until 36 weeks gestation, making further antenatal intervention difficult. The postnatal course was uncomplicated, with recovery of the platelet count after one single HPA 1a neg platelet transfusion. Postnatal MRI confirmed the antenatal findings.
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