

Characteristics of Six Newborn Infants with Postnatal Findings of Severe Intracranial Haemorrhage

JPL Wong,¹FAMS, MRCOG (Lond), M Med (O&G) (S'pore), WT Seow,²FAMS, FRACS (Neurosurgery), GSH Yeo,³FAMS, FRCOG (Lond)

Abstract

Objective: The objective of this study was to study the characteristics of newborn infants with postnatal findings of severe neonatal intracranial haemorrhage. **Methods:** All the records of babies who underwent surgery from 1997 to 2002 for intracranial haemorrhage were reviewed. These were correlated with their antenatal records to see if fetal intracranial haemorrhage had been detected at the 20 weeks' screening scan or any other incidental scan e.g. growth scan. The perinatal records were also reviewed to see if there was associated birth trauma such as instrumentation or obstetric manoeuvres at delivery. **Results:** Six cases of severe intracranial haemorrhage were diagnosed postnatally. Of these, only 1 case was detected antenatally on ultrasound scan. None of the cases were due to birth trauma. Three babies were found to have clotting factor deficiency. One of them subsequently developed cerebral palsy. One baby was diagnosed to have alloimmune thrombocytopenia. One case underwent an emergency Caesarean section for non-reassuring fetal status. Extensive intracranial haemorrhage, attributed to hypoxia, was found. The baby died. **Conclusions:** Our study suggests that neonatal intracranial haemorrhages are not exclusively due to birth trauma. The study also shows that fetal intracranial haemorrhage may not be detected antenatally by the routine practice. The causes in our study included clotting deficiency, alloimmune thrombocytopenia and hypoxia.

Ann Acad Med Singapore 2004;33:789-92

Key words: Antenatal, Birth trauma, Fetal, Neonatal, Ultrasound

Introduction

A postnatal finding of intracranial haemorrhage carries potential medico-legal implications for the obstetrician. However, one cannot exclusively attribute this to birth trauma.

Many cases of neonatal intracranial haemorrhages, especially small ones, are managed conservatively with no surgical intervention. These cases are not included in our study. We reviewed all the records of babies who underwent surgery in our hospital from 1997 to 2002 for intracranial haemorrhage. There were 5 cases. We came across 1 case who was not operated on as the prognosis was dismal.

The babies' records were correlated with their mothers' antenatal records to see if fetal intracranial haemorrhage had been detected during the pregnancy. The antenatal records were also reviewed to see if there was associated birth trauma.

There were a total of 94,681 births in our hospital from

1997 to 2002. There were 5 cases of severe intracranial haemorrhage which warranted surgical intervention, giving an incidence of 1:18,936. Only 1 case was detected antenatally on ultrasound scan. None of the cases were due to birth trauma.

Three babies were found to have a clotting factor deficiency. One of them subsequently developed cerebral palsy. One baby was diagnosed to have alloimmune thrombocytopenia. One case underwent an emergency Caesarean section for non-reassuring fetal status. Extensive intracranial haemorrhage, attributed to hypoxia, was found. The baby died.

Case 1

This was the only case where a fetal intracranial haemorrhage was antenatally diagnosed. The mother was a 32-year-old Chinese lady. This was her second pregnancy. Her first pregnancy was uneventful. She booked early, and had a dating scan done. A screening scan at 18.7 weeks'

¹ Department of General Obstetrics & Gynaecology

² Department of Paediatric Neurosurgery

³ Department of Maternal Fetal Medicine

KK Women's and Children's Hospital, Singapore

Address for Reprints: Dr Judy Wong Pui Ling, Department of General Obstetrics and Gynaecology, KK Women's and Children's Hospital, 100 Bt Timah Road, Singapore 229899.

gestation was normal. The head circumference (HC) was 167 mm, between the 50th to 97th percentile. A growth scan at 34 weeks' gestation showed asymmetry of both hemispheres of the brain, dilated horns, as well as a 2.6-cm possible subarachnoid haemorrhage between the left cerebral hemisphere and the skull. There was also a suspicion of a blood clot in the left occipital region. The HC was above the 97th percentile.

The mother noted reduced fetal movement the same day the growth scan was carried out. A cardiotocograph (CTG) showed a non-reassuring fetal status. An emergency Caesarean section was performed. As the baby's big head presented difficulty with delivery, the forceps blades were applied. At birth, the baby was found to be pale, with Apgar scores of 2 and 5 at 1 and 5 minutes, respectively.

The baby's haemoglobin level was found to be 5.3 g/dL. Both the prothrombin time (PT) and partial thromboplastin time (PTT) were prolonged. There was factor IX deficiency. The baby's factor IX level was 38%. Fresh frozen plasma, cryoprecipitate and factor IX replacement were given.

Postnatal CT scan of the head showed large bilateral subdural haematomas extending from the frontal to the occipital lobe. There was no fracture. The haematomas were drained surgically. Postoperatively, the baby developed seizures. When reviewed at 14 months of age, the child had quadriplegic cerebral palsy and needed special home placement.

Case 2

Fetal intracranial haemorrhage was not detected antenatally in this case. The mother was a 31-year-old Chinese lady in her third pregnancy. Her first 2 pregnancies were uneventful. She booked early, and had a dating scan done. Mid-trimester screening scan at 20 weeks was normal. The fetal HC was 176 mm, between the 50th and 97th percentile. A growth scan at 32 weeks' gestation was also normal. The fetal HC was 317 mm, at the 97th percentile.

The baby was delivered uneventfully at full term via normal vaginal delivery. No instrumentation was required. The Apgar scores were 8 and 9 at 1 and 5 minutes, respectively.

At birth a large left parietal cephalohaematoma was found. The baby's platelet count was found to be 70,000. Postnatal CT scan of the head showed 2 left acute extradural haematomas over the left tempo-parietal region. There was no fracture. The haematomas were evacuated. The cause was found to be alloimmune thrombocytopenia. Anti-double stranded DNA, anti-nuclear antibody and anti-cardiolipin antibody were negative. The baby required platelet transfusion and intravenous gamma globulin whilst in hospital. After discharge from hospital, the child was regularly followed up. The platelet count was 252,000

without any further treatment. At the time of writing, the child was 2 years old and was well.

Case 3

Fetal intracranial haemorrhage was not detected antenatally in this case. The mother was a 23-year-old Malay lady in her second pregnancy. Her first child, delivered by Caesarean section for non-reassuring fetal status, was well.

She had a dating scan at her booking visit of 8 weeks' amenorrhoea during her second pregnancy. Both her screening scan at 20 weeks as well as her growth scan at 31 weeks did not reveal any abnormality. The fetal HC remained at the 50th percentile on both occasions.

The pregnancy proceeded uneventfully and she underwent an elective Caesarean section at term for previous Caesarean section. The baby was found to have a large head at delivery. Apgar scores were 9 at both 1 and 5 minutes. Postnatal CT scan of the head showed a large acute subdural haematoma occupying the posterior half of the left cranium, with extensive midline shift. There was no fracture. The haematoma was evacuated. Intraoperatively acute and chronic subdural haemorrhage was found. The cause was unknown. The child was later found to have cerebral palsy.

Cases 4 and 5

These were dichorionic diamniotic twins. The mother was a 35-year-old Chinese lady. This was her second pregnancy of spontaneous conception. Her first pregnancy was uneventful. She booked at 7 weeks' amenorrhoea and had a dating scan done, confirming dichorionic diamniotic twins. The mother declined amniocentesis.

She had gestational impaired glucose tolerance diagnosed on a 75-g oral glucose tolerance test at 29 weeks' gestation. Mid-trimester screening scan at 20 weeks as well as growth scans at 25 and 29 weeks were normal.

Both twins were delivered vaginally following spontaneous labour at 37 completed weeks of gestation. The delivery went smoothly. No instrumentation was necessary. Apgar scores for both twins were 9 at 1 and 5 minutes.

At birth, 1 of the twins was found to have widened sutures. CT scan of the head showed a moderately sized right posterior fossa haemorrhage. There was no fracture. The second twin was noted to be pale. CT scan of the head showed 2 subdural haematomas both infra-tentorially in the midline as well as supra-tentorially to the right up to the falx. There was no fracture.

The twins underwent craniotomy and evacuation of the clots. The haemoglobin level for 1 twin was 11.5 g/dL. The platelet count was 100,000. PT was 17.7. PTT was 43.2.

Bleeding time was 2 minutes (0.85 to 1.65) after transfusion with platelets, fresh frozen plasma and cryoprecipitate. The maternal platelet count was 200,000.

The twins remained asymptomatic until 5 months of age, when 1 twin presented with a sudden onset of a cheek haematoma 5 cm in diameter. This resolved spontaneously with platelet and cryoprecipitate transfusion.

The parents sought a second opinion in another hospital, where both twins were subsequently diagnosed to have factor XIII deficiency.

Case 6

Fetal intracranial haemorrhage was not detected antenatally in this case. The mother was a 22-year-old Malay lady in her third pregnancy. Her first 2 pregnancies were uneventful. She was not booked and did not have any antenatal care at all.

She presented to us at 36 weeks' amenorrhoea with abdominal cramps associated with absent fetal movement for 3 days. CTG registered late decelerations. The cervix was 2.5 cm dilated, with moderately meconium-stained liquor on amniotomy.

An emergency Caesarean section was carried out. The baby weighed 2780 g. Unfortunately, the Apgar score was 1 at both 1 and 5 minutes. There was pallor and a large head with bulging fontanelles. CT scan of the head showed extensive intracranial haemorrhage with midline shift and transtentorial herniation of the right thalamus. There was no fracture. There was a large extradural haemorrhage over the right frontal and temporal lobes. Blood was present within the ventricular systems. Intraparenchymal haemorrhages were present within the left occipital lobe superiorly. Generalised cerebral oedema was present. Surgery was not possible in view of the bleak prognosis. The baby succumbed on day 6 of life.

Discussion

A postnatal finding of intracranial haemorrhage, especially with associated mortality or neurological impairment, carries potential medico-legal implications for the obstetrician.

Predisposing maternal factors¹ include alloimmune and idiopathic thrombocytopenia, von Willebrand's disease, specific medications (warfarin) or illicit drug (cocaine) abuse, seizures, severe abdominal trauma inflicting subsequent fetal injury, amniocentesis, cholestasis of pregnancy and febrile disease. Fetal conditions predisposing to fetal intracranial haemorrhage¹ include congenital factor X and factor V deficiencies, haemorrhage into various congenital tumours, twin-twin transfusion, demise of a co-twin or feto-maternal haemorrhage.

Three out of our 6 cases were found to have a clotting

factor deficiency as the underlying cause. Alloimmune thrombocytopenia was found to be a cause in 1 case.

Vergani et al² described that the anatomic location of the haemorrhage is an important prognosticator: parenchymal and subdural haemorrhages are associated with a poor prognosis in nearly 90% of cases and intraventricular haemorrhages have a poor prognosis in 45% of cases.

In our study, the 2 cases who were later found to have cerebral palsy had sustained subdural haemorrhages. Sudden changes in cerebral blood pressure can lead to rupture of the germinal matrix capillary or capillary-venous junction. Perinatal asphyxia with its attendant hypoxia can induce fluctuations in blood pressure, resulting in intracranial haemorrhage.³ This is depicted by case 6, where hypoxic damage inflicted extensive intracranial haemorrhage such that surgery was not deemed possible.

It is a common belief that fetal subdural haematoma is inexorably linked to obstetric intervention and is hence related to birth trauma.^{4,5-7} However, neonatal intracranial haemorrhage is often not due to birth trauma. In most cases, perinatal hypoxia and even clotting factor disorders are added factors.^{4,8-10} In our study, there was no associated birth trauma in any of the cases.

Subdural haematoma in term infants may occur as a sequela of an uncomplicated delivery.⁸ Demir et al⁴ reported a case where even in the absence of medical or obstetric factors potentially associated with fetal subdural haematoma, the complication can occur and be severe enough to be associated with fetal death.

Since documentation of an intracranial haemorrhage with ultrasonography at a remote time from labour and delivery excludes the obstetrician from culpability, this should be looked for during routine scanning. In particular, fetuses at high risk of developing in utero hypoxia (i.e., intrauterine growth restriction, pre-eclampsia and premature rupture of membranes) should be monitored and sonographically evaluated to detect early signs of intracranial haemorrhage.¹¹ The presence or absence of such a lesion may be taken into consideration when the obstetrician is deciding on the time and mode of delivery.

Not all obstetricians carry out growth scans on expectant mothers. Yet, despite our 5 cases with both mid-trimester and growth scans carried out during the pregnancy, antenatal diagnosis was possible only in 1 case (Case 1). This means that the condition may not be detected antenatally by routine practice.

Conclusion

Out of a total of 94,681 births in our hospital from 1997 to 2002, there were 5 cases of severe intracranial haemorrhage which warranted surgical intervention. All 5 cases were due to causes other than birth trauma. There was

1 case which was not operated on as the prognosis was dismal. This case was also not due to birth trauma.

This suggests that neonatal intracranial haemorrhages are not exclusively due to birth trauma. Our study also shows that fetal intracranial haemorrhage may not be detected antenatally by routine practice.

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