Severe subdural hemorrhage due to minimal prenatal trauma

Case report

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The authors report a case of minimal prenatal trauma producing a large subdural hematoma in the fetus, which was diagnosed in utero by MR imaging. The occurrence of such a complication is extremely rare in the absence of significant maternal trauma. Prenatally diagnosed intracranial hemorrhages, particularly those that are subdural in origin, have a poor prognosis in most cases. After birth, brain compression required a complex neurosurgical intervention because simple hematoma evacuation was not possible. The clinical and neurological outcome at 6 months was excellent, as confirmed by the neuroimaging findings. (DOI: 10.3171/2009.7.PEDS08223)

Key Words • subdural hemorrhage • prenatal trauma • fetal trauma • intracranial hemorrhage

B lunt abdominal trauma during pregnancy can cause fetal brain injury and intracranial hemorrhage. Until now, the possible effects of maternal trauma on surviving fetuses has been explored mostly as a consequence of motor vehicle accidents. Often emergency cesarean sections are needed during the 3rd trimester after major traffic accidents and are associated with significant rates of neonatal morbidity and mortality. Brain pathology, including skull fracture, intracranial hemorrhage, and hypoxic-ischemic encephalopathy, in neonates after major intruterine trauma during the 3rd trimester has been reviewed in the recent literature. Minimal maternal trauma may be undervalued and can produce a so-called spontaneous intrauterine fetal intracranial hemorrhage. The consequences of even minimal injury to the fetal brain can be actually unpredictable.

Case Report

History and Examination. This newborn boy was delivered via cesarean section at 37 weeks’ gestation to a 34-year-old woman. This was her second pregnancy; the first pregnancy was uneventful. At 30 weeks’ gestation, an ultrasonography examination was performed because of a minimal maternal trauma (abdominal hit from her 17-month-old daughter) that caused a fetal intracranial extracerebral hemorrhage. The routine 22-week-gestation morphological ultrasound did not reveal any abnormalities. Therefore, a prenatal MR imaging evaluation was carried out 10 days after the trauma and showed a large right SDH compressing the ipsilateral ventricle and causing a midline shift; the left lateral ventricle was slightly enlarged (12.5 mm; Fig. 1). On control echo scanning performed at 34 weeks, the ventriculomegaly had increased (9 × 17 mm). Based on these findings, we decided to perform the delivery by cesarean section as soon as fetal maturation allowed. At birth, the boy’s body weight was 2980 g and he was 52 cm long with a head circumference of 35.4 cm (> 95th percentile). The Apgar scores were 8 and 9 at 1 and 5 minutes, respectively. The Moro reflex was present, and the anterior fontanel was moderately tense. During the first postnatal days the baby was moderately reactive, and oral feeding was introduced. Right-sided ptosis and mild left hemiparesis were present. Shortly after birth, a coagulative impairment was detected (prothrombin time 24.8 seconds, prothrombin activity 24%, activated partial
thromboplastin time 77.5 seconds, fibrinogen 263 mg/dl, antithrombin 45%). Findings from antithrombotic factor screening were as follows: anticoagulation protein S 38%, anticoagulation protein C 24%, activated protein C resistance ratio 1.08 (range 0.76–5.00). Anticardiolipin antibodies were absent, results of homocysteine screening were negative (2.5 μmol/L, normal range 5–15 μmol/L), and a prothrombin G20210A mutation was not found. The abnormal coagulation profile was deemed to be due to consumption of clotting factors by the expanding hematoma; therefore, no procoagulant therapy was undertaken except for the administration of vitamin K. Findings from the toxoplasma, rubeola, cytomegalovirus, and herpes (TORCH) studies and blood and urine cultures were negative. A postnatal cerebral MR imaging study confirmed the large right hemispheric SDH (maximum thickness 15 mm) that caused mass effect and a 4.4-mm midline shift. Right lateral ventricle compression and left ventricle enlargement were also evident as well as the presence of a circumscribed right parietal SAH and 2 small contusions on the anterior portion of the left frontal lobe (Fig. 2). Vascular malformations were excluded by MR angiography.

Fig. 1. Coronal T2-weighted MR images obtained in the fetus during the 31st week of gestation showing a large intracranial isointense SDH displacing the right hemisphere. The right lateral ventricle is displaced contralaterally and the left one is mildly enlarged.

Fig. 2. Neonatal (1st week after birth) brain T1-weighted axial (A and B), coronal (C), and sagittal (D) MR images, and 3D angio-MR image (E) confirming the presence of the right SDH (thickness 15 mm) causing mass effect with subsequent 4.4-mm midline shift and compression of the right lateral ventricle. Note the parietal SAH (white arrows) and the frontal contusions (black arrows).
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**Fig. 3.** Postoperative T2-weighted axial MR images obtained 1 month after surgery, demonstrating the resolution of the hemispheric subdural collection and the physiological evolution of the frontal contusion.

**Operation.** After improvement in the clotting tests, the baby underwent a neurosurgical procedure. A traditional evacuation of the hematoma through bur holes was planned. However, during surgery, a solid hematoma was found so that a right frontoparietal craniotomy was required to completely evacuate the hemorrhagic collection. The collection consisted of a scarce fluid component and a large blood clot attached to thick parietal and visceral membranes. Intraoperatively, 20 ml/kg hydroxyethyl starch and 10 ml/kg 20% human albumin were infused over 240 minutes. The baby was referred to the pediatric intensive care unit for postoperative intensive treatment and further hemodynamic support (heart rate 125 bpm, blood pressure 58/30 mm Hg, pH 7.27, HCO₃ 19 mEq/L, base excess -4 mEq/L, and lactate 3 mmol/L on admission). He was given saline solution to treat hyperglycemia (222 mg/dl), and he required a transfusion of packed red blood cells (15 ml/kg); saline solution to treat hyperglycemia (222 mg/dl), and he required a transfusion of packed red blood cells (15 ml/kg); subsequently, the hematocrit remained stable. Hemostasis assessment was within normal limits; the D-dimer level peaked on postoperative Day 3 (840 ng/ml), and antithrombin supplementation was given. Mechanical ventilation was needed until 48 hours postoperatively. On emergence from sedation, he showed a good respiratory activity, and he was extubated. Clinical examination showed a marked opisthotonus and irritability.

**Postoperative Course.** On the 3rd postoperative day, the infant suffered lateralized seizures starting from the left hemisoma; these responded to intravenous phenobarbital. A 2-day dexamethasone course was given. Electroencephalography showed spikes over the right temporal area and a global asymmetrical tracing with better organization in the left hemisphere. The baby was discharged to the pediatric neurosurgery department on the 5th postoperative day in good clinical condition. The postoperative MR imaging examination demonstrated evacuation of the right hemisoma and resorption of the left clots (Fig. 3). Current neurological assessment (8 months after surgery) shows the almost complete regression of the left motor deficit and no developmental delays to date.

**Discussion**

An SDH detected postnatally in a newborn is usually related to trauma at the time of vaginal delivery. A neonatal SDH is venous in origin and in most cases is associated with trauma. Etiopathogenesis of neonatal SDH is explained by shearing of bridging veins or other venous structures caused by trauma. With improvement in obstetric methods, the overall incidence of this problem has declined. Otherwise, SDH in full-term infants may occur as a sequela of an uncomplicated delivery. Demir et al. reported a case in which fetal death occurred even in the absence of medical or obstetric factors usually associated with fetal SDH.

However, with advances in prenatal ultrasonography, it has been recognized that SDH may occur in utero before the onset of labor. Even if detected antenatally, fetal neonatal intracranial hemorrhages can be associated with an increased mortality rate or neurological impairment. Vergani et al. reported that the anatomical location of the hemorrhage is an important prognostic factor. Parenchymal and subdural hemorrhages are associated with a poor prognosis in nearly 90% of cases, while intraventricular hemorrhage has a poor prognosis in 45% of cases. Fetal conditions predisposing to antenatal intracranial hemorrhage include congenital factor V and X deficiencies, hemorrhage into various congenital tumors, twin-twin transfusion, demise of a twin, or fetal-maternal hemorrhage. Among maternal factors, excluding hematological conditions (idiopathic thrombocytopenia, Willebrand disease, and anticoagulation drugs), abuse, and severe abdominal trauma appear to cause subsequent fetal injury not infrequently.

As expected, SDH and brain injury have been described following prenatal blunt trauma, mostly in the setting of major trauma. Maternal trauma, including falls, assaults, and motor vehicle accidents, represents the best-documented cause of SDH in the fetus before the onset of labor. Several fatal cases have been described in the past decade. Prognosis of the fetus affected by SDH is dependent on the severity of the hemorrhage and on the underlying cause; among infants who sustained intrapartum SDH, < 50% were live-born. Interestingly, surgical evacuation in the neonatal period has been performed in very few cases, whereas subdural tapping has been reported with some success. It is very uncommon that minor trauma, as in our case, can cause severe fetal brain damage. In fact, while traumatic injury—either due to direct blunt trauma or the use of safety belts or air bags in a motor vehicle accident—has an enormous potential for fetal injury and demise, minimal trauma as the origin of a large intrapartum SDH has not been described before. We hypothesized a cause-and-effect mechanism because the previous routine ultrasonography evaluation, as well as other maternal and neonatal hematological and coagulative investigations, revealed normal findings.

According to a literature review of all intrapartum SDHs with prenatal diagnoses (22 cases reported to date), the cases do not seem to be strictly related to a traumatic cause, as reported for those with perinatal onset. Only 2 cases with antecedent maternal trauma have been reported, and in 1 of them surgical drainage was performed after birth. The case presented here seems to suggest a traumatic injury at the base of the subdural collection.
Actually, the SAH could have been caused by the direct impact of the sister’s body against the fetus’ head while the contralateral frontal contusions could have resulted from a contrecoup trauma. The subdural collection, as usual, would have arisen from the bleeding of 1 or more small bridging veins, resulting from the inertial trauma due to the acceleration impressed on the fetus’ head. Such a mechanism would have been favored by a relatively large subdural space due to the immature brain tissue. The subdural collection, therefore, would have had an acute origin as demonstrated by the anomalous findings at surgery, showing a solid hematoma rather than the typical fluid chronic subdural collection.

Although prenatal intracranial hemorrhages as a whole have a poor outcome (40% of fetuses dying either in utero or within the 1st month after birth), surviving infants with SDH may exhibit better neurodevelopmental performance. The patient sample is inadequate to provide prognostic figures; however, previous experience and the present case demonstrate that surgical treatment is indicated in patients who present with increased intracranial pressure and clinical deterioration. Above all, surgery can be life-saving when there are signs and symptoms of brainstem dysfunction.

Our experience suggests that early diagnosis and appropriate treatment can improve survival and minimize brain damage. Optimal management and outcome of these young infants require close interdisciplinary cooperation at a tertiary referral institute.

Disclaimer

The authors do not report any conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

References


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