Contribution of surgical evacuation of a large subgaleal hematoma to the resolution of severe conjugated hyperbilirubinemia in a neonate

Case report

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Subgaleal hematomas (SGHs) in neonates may result from trauma to the scalp sustained during delivery. In the majority of cases, these lesions will resolve on their own without serious or long-term consequences. The authors report on a case of SGH resulting in hypotension, anemia, coagulopathy, and eventually direct hyperbilirubinemia in a neonate. After several weeks of medical management failed to resolve the hyperbilirubinemia, surgical evacuation of the clot was undertaken and yielded favorable results. The direct bilirubinemia in this case was believed to be the result of an overload of iron to the hepatocytes. It is the authors’ contention that the evacuation of the hematoma resulted in a reduction in the amount of iron being presented to the liver for metabolism and significantly contributed to this patient’s recovery.

KEY WORDS • subgaleal hematoma • birth injury • liver • cranium • treatment • pediatric neurosurgery

SUBGALEAL hematomas are commonly encountered in the neonate after scalp trauma sustained during the birthing process. Unlike cephalohematoma, SGH is not contained by the suture lines of the skull. Thus, the large potential space between the galea aponeurotica and the peri-cranium, extending from the orbital ridges to the nape of the neck, can accommodate the equivalent of an entire blood volume in some newborns. Although SGH may occur after any method of delivery, several studies indicate a strong association with vacuum extraction compared with all other methods. In fact, the first use of the term “subgaleal hematoma” in the literature appeared in an article by Malström, the Swedish physician who introduced the metal-cup vacuum extractor in 1954.

In a majority of cases SGH will resolve on its own without serious or long-term consequences. However, given the potential redistribution of such a significant portion of the neonate’s intravascular volume to the aponeurotic space, SGH can cause hypovolemic shock, anemia, and coagulopathy, although it is unclear if coagulopathy is a contributor to or the result of the massive blood loss. These exsanguinating complications can prove fatal in as many as 25% of patients with SGH. Hyperbilirubinemia is another complication that can occur during the dissolution of an established blood clot, and in one reported case series it was noted in just over half of the patients with SGH. In the present study we describe a case of SGH resulting in hypotension, anemia, coagulopathy, and eventually elevated direct hyperbilirubinemia. After several weeks of medical management failed to resolve the hyperbilirubinemia, surgical evacuation of the clot was undertaken and yielded favorable results.
Case Report

History and Examination. This female infant was born at 37 weeks’ gestation to a 30-year-old woman (gravida 1) via vaginal delivery at a referring community hospital. Delivery assistance was initially attempted with vacuum extraction but became complicated by failure to progress, multiple late and variable decelerations, and possible placental abruption. An emergency cesarean section was performed in the delivery suite. The birth weight was recorded as 1960 g (low for the gestational age), and the Apgar scores were 1, 5, and 5 at 1, 5, and 10 minutes, respectively. The patient was initially resuscitated with endotracheal tube placement and positive-pressure ventilation, but 2 hours after birth the child was noted to be in hypovolemic shock with acidemia (blood pH 6.97, pCO₂ 68, pO₂ 27). The patient received multiple doses of intravenous epinephrine and NaHCO₃ for immediate cardiac support. Over the next 48 hours she was also treated with 330 ml of PRBC, 110 ml of fresh frozen plasma, 60 ml of platelets, and 5 to 20 μg/kg/min of dopamine to maintain an adequate mean arterial blood pressure and prevent further bleeding. During the first 48 hours after her birth, episodic oxygen desaturation was noted; this raised concerns over possible seizure activity and promptly intravenous phenobarbital treatment.

On DOL 3 the patient was transferred to The Children’s Hospital in Birmingham, Alabama. On arrival, she was noted to have severe jaundice affecting her entire body and significant cephalic bruising and swelling extending to the nuchal ridge; edema was observed in her face, neck, and anterior chest (Fig. 1). Her head circumference was 37 cm, which was increased from the 32-cm measurement recorded at birth (Fig. 2), giving an estimated intravascular blood loss of 190 ml. Laboratory results obtained at admission revealed the following: a hematocrit of 34%; platelet count of 134 × 10³/μl; prothrombin time of 33 seconds (international normalized ratio 2.9); activated partial thromboplastin time of 76 seconds; aspartate transaminase of 2833 U/L; alanine aminotransferase of 398 U/L; and a total bilirubin level of 6.1 mg/dl (Fig. 3). Additional PRBC and platelet transfusions as well as two doses of factor VII were administered. A head CT obtained on DOL 3 confirmed the presence of an SGH (Fig. 4). Because these lesions typically follow a benign course, surgical intervention was initially deemed more risky than beneficial because of the patient’s demonstrated coagulopathy and was thus deferred.

Treatment and Posttreatment Course. Over the course of the next 16 days an upward trend was noted in the patient’s total bilirubin level, rising as high as 55.5 mg/dl on DOL 18 (Fig. 3). This effect was almost entirely attributable to an increase in the conjugated bilirubin level, which also peaked on DOL 18 at 49.6 mg/dl. An abdominal ultrasonography study performed on DOL 17 revealed diffuse hepatic enlargement with no evidence of gallstones. A head CT scan obtained on DOL 23 demonstrated an enlarging hematoma with a small new parenchymal contusion in the right temporoparietal white matter without a significant mass effect. Elective evacuation of the SGH was performed on DOL 26 because of the failure of medical management, and 200 ml of hematoma was removed. A Jackson–Pratt drain was left in place for 24 hours with an additional yield of 70 ml serosanguinous fluid. On the day after surgery, the patient’s con-

Fig. 1. Photograph of the patient obtained just before surgery on DOL 26, showing an enlarged head circumference, periorbital bruising, and considerable jaundice.

Fig. 2. Line graph representing the progression of the patient’s head circumference (in cm) over time. Asterisk indicates time of surgery.

Fig. 3. Line graph demonstrating progression of total bilirubin and conjugated bilirubin (mg/dl) levels over the first 36 DOLs. Asterisk indicates time of surgery.
jugated bilirubin level dropped by 9.2 mg/dl (Fig. 3), with a more modest decrease each day thereafter. The child’s head circumference returned to 33 cm (Fig. 2), and there was improvement in the degree of jaundice noted on physical examination over the following weeks.

Discussion

Subgaleal hematoma is a commonly encountered lesion in neonates. Typically SGHs have a small volume, present little threat to the child’s health, and can be managed conservatively. In the rare case of a neonate with an enlarging SGH, the first approach to treatment is appropriate resuscitation and management of intravascular volume and blood pressure. Beyond this, there are no well-defined treatment parameters. Pressure bandaging of the affected area has been recommended, but this is cumbersome in newborns and may cause further damage in the presence of cerebral edema. Amar et al. described successful surgical evacuation in a case of SGH in which extracranial cerebral compression was present. However, given the risk of introducing infection into an otherwise sterile hematoma and the usually benign course the small SGH takes, regular use of surgical evacuation should be avoided when medical options exist.

In our patient, extreme conjugated hyperbilirubinemia was present soon after development of a significant SGH. Attempts at medical management included administration of ursodiol (30 mg orally, three times daily) for several weeks. In addition phenobarbital (hourly 10-mg oral dose) was given for its antiseizure effects and propensity to increase liver function. Despite this therapy, the child’s elevated total bilirubin and conjugated bilirubin levels did not show improvement. The patient in the present case received the equivalent of two or more blood volumes of PRBC, which is well below the seven exchange transfusions used in a similar case. However, after more than 3 weeks of medical treatment, the hematoma continued to enlarge. Given our concern over SGH calcification and future skull deformity, we decided to evacuate the blood. To the best of our knowledge this is the first report of extreme conjugated hyperbilirubinemia associated with an SGH, as well as resolution of the hyperbilirubinemia after surgical intervention.

One question that remains is why did conjugated hyperbilirubinemia develop in this patient? Intuitively, one would expect the dissolving clot to produce unconjugated hyperbilirubinemia that would overwhelm the liver, at least initially. In this case, unconjugated bilirubin was never elevated to the level of conjugated bilirubin, suggesting that the liver had no difficulty processing the immense bilirubin load. Abdominal ultrasonography performed on DOL 17 demonstrated diffuse hepatic enlargement and some gallbladder thickening, but no evidence of gallstones and no clear evidence of biliary sludging. It is possible that there was some ischemic injury to the liver during the complicated delivery, an idea supported by the transaminits present at admission. It is also probable that the liver was functioning as the rate-limiting step in clearance of the bilirubin, and once part of the burden was removed (by surgical evacuation), the liver output was able to catch up with the input.

The direct bilirubinemia in this case was believed to be the result of iron overload to the hepatocytes. The fact that the direct bilirubin decreased prior to surgical intervention may reflect the positive effects of iron chelation therapy, which had been started preoperatively. We in no way support the routine evacuation of SGHs, and certainly not during the acute phase. In the present case, the decision to intercede surgically was considered for many days prior to the procedure. We believe that the evacuation of the hematoma resulted in a smaller amount of iron being presented to the liver for metabolism and contributed significantly to this infant’s recovery.

References


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