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Case Report

Spontaneous Fetal Skull Fracture in an Apparently Uncomplicated Vaginal Delivery

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Abstract

Fetal skull fracture is a rare occurrence in the absence of instrumentation at the time of vaginal delivery. While most commonly associated with the use of forceps, the etiology of spontaneous skull fractures remains elusive. We report a case of a 3355-g female infant born at 4 weeks gestation by uncomplicated vaginal delivery (Apgars 8/8) after an unremarkable labor course. At two hours of life, the infant was found to be listless with a left parietal hematoma consistent with a subgaleal hemorrhage and imaging studies showed a small linear occipital fracture. The clinical course was complicated by hypotension and multi-organ failure, with the infant expiring on day of life 4. While most linear skull fractures are clinically insignificant, devastating complications including neonatal death can occur. Diagnosis of a fetal skull fracture requires a high level of suspicion, as it can occur even in uncomplicated vaginal deliveries.

Keywords: Neonatal death; Newborn; Spontaneous skull fractures; Subgaleal hemorrhage; Vaginal delivery

Case Presentation

The patient is a 21 year old primigravida who presented to the Labor and Delivery Unit in active labor at 41 weeks of gestation she received regular prenatal care with the Certified Nurse Midwives and had an uncomplicated antenatal course. Her past medical, surgical, obstetrical and gynecological histories were noncontributory. At the time of presentation, membranes were intact and the fetal heart tracing was reassuring with a baseline of 140 beats per minute (bpm), moderate variability and accelerations. Her initial vaginal exam revealed cervical dilation of 6 cm with 90% effacement, cephalic presentation with the fetal vertex at -1 station. Upon admission, the patient requested epidural anesthesia and pitocin augmentation was later initiated. Spontaneous rupture of membranes occurred 4 hours after admission and the patient remained comfortable. An intrapartum temperature of 100.9^{O} F was noted and 1000mg of Acetaminophen was administered with resolution of the fever. During this time, there were no other signs or symptoms of chorioamnionitis. Intermittent early decelerations were noted. The patient was fully dilated within 2 hours of membrane rupture with pitocin infusion at 7 milliunits per minute during the first stage of labor. The fetal heart tracing was reassuring with a baseline heart rate of 150 bpm and moderate variability. The fetal vertex was now at +1 station in left occiput anterior position. The patient then began pushing, with the second stage of labor lasting 3 hours and 14 minutes. The fetal heart tracing remained reassuring during this time with a baseline of 140 bpm and moderate variability. In the last 30 minutes before delivery, variable decelerations were noted with contractions in the absence of tachysystole; however, spontaneous recovery was noted with a baseline of 130 bpm between contractions. A viable female child with Apgars of 8/8 was delivered. Thick meconium was noted at time of delivery; the infant was suctioned and noted to have a vigorous cry.

At two hours of life, the infant was noted to be listless and the pediatricians were notified. Physical exam revealed a hypotonic, pale infant with diminished peripheral pulses and a weak cry. The infant was tachypneic and tachycardic. A baseline oxygen saturation of 90% was noted on room air, which improved to 100% on supplemental oxygen. A full anterior fontanelle was appreciated, as well as a "boggy" left parietal hematoma. CT scan revealed extensive bilateral extracranial fluid collections consistent with subgaleal hemorrhage and a small undisplaced, nondepressed occipital fracture. The infant was transferred to the NICU for further management. In the first 24 hours, the infant was persistently hypotensive and in respiratory distress requiring multiple pressors and intubation. She then developed acute renal failure, disseminated intravascular coagulopathy, and seizure activity. The infant was started on peritoneal dialysis and received multiple transfusions. Despite all resuscitation efforts, the neonate expired on day of life 4.

Discussion

Subgaleal hemorrhage (SGH) is defined as bleeding between the galea aponeurosa of the scalp and the periosteum, It is caused by rupture of the emissary veins, which are connections between the dural sinuses and the scalp veins. The galea aponeurosa is a fibrous layer of tissue that covers the entire cranial vault from the nape of the neck to the orbital ridges and laterally to the ears. This potential space is continuous with the superficial fascia of the neck and creates a large potential space for blood collection [1]. While the incidence of SGH is low at only 0.8/ 1000 live births, this rises precipitously with instrumentation during delivery. In vacuum- assisted deliveries, for example, this rate can be as high as 6.4/1000 [2]. Instrumentation can cause SGH by different mechanisms, including skull fractures and soft tissue injuries leading to shearing of the bridging emissary veins. The single most significant risk factor for SGH, however, appears to be the use of vacuum devices during delivery. In fact, some studies show that up to 89% of SGH cases had a vacuum device applied [1].

SGH can be clinically unapparent immediately after birth- most

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cases present hours to days post-partum. Clinically, SGH presents as a fluctuant hemorrhagic mass that crosses suture lines, extending towards the nape of the neck and towards the ears laterally and may cause lateral displacement of the ears. Presenting signs include respiratory distress, lethargy, tachycardia, hypotension, hypotonia, and metabolic acidosis [3,4]. Because of the large potential space in the subaponeurotic compartment, hypovolemic shock and death may ensue, with mortality estimates ranging from 2.7- 22.8% [2]. For this reason, early volume support and transfusions are of critical importance in optimizing survival. Indeed, in a retrospective study of 34 neonates with SGH, Kilani et al. reported a mortality rate of 11.8%. Those neonates who died were more likely to have significant volume loss, coagulopathy, and shock requiring transfusion. Interestingly, these infants also had a larger head circumference (mean 40 cm, p= 0.038) and advanced gestational age (mean 39 weeks, p= 0.049) than those with SGH who survived [5]. Our neonate's head circumference was 41cm.

Skull fractures alone are also a rare occurrence in vaginal deliveries and its risk increases significantly with forceps or vacuum assisted deliveries. Skull fracture constitutes only 2.9% of all neonatal head and neck injuries. Previously reported risk factors for skull fractures and other birth injuries include male sex, macrosomia, prolonged labor, malpresentation, vacuum- assisted delivery, forceps delivery, and primiparity [6]. When they do occur, most skull fractures are linear, non-displaced fractures. These often require no treatment and often heal spontaneously without intervention. Depressed fractures, on the other hand, are also a complication of traumatic delivery or instrumentation, and are more likely to be associated with intracranial pathology [2]. Dupuis et al. described 68 cases of depressed skull fracture, of which 18 (26%) were the result of spontaneous (non-instrumented) deliveries. Dupuis postulates that these fractures may be secondary to pressure on the fetal skull during delivery from various maternal anatomical landmarks, including the fifth lumbar vertebrae, sacral promontory, symphysis pubis, ischial spines, an asymmetric or contracted pelvis, or uterine fibroid [7]. In the absence of cephalopelvic disproportion, extrinsic trauma, instrumentation, or other risk factors, the skull fracture may be due to intrinsic compression of the fetal head- either against bony pelvic landmarks, or because of excessive uterine pressure during contractions [8].

Coagulopathy can also play an important role in the severity of SGH, most commonly hemophilia A [9]. Another possibility is a congenital deficiency in bone formation; although no evidence of such a disorder was identified on X ray with the fetus presented in this case report. Because of the severity of hemorrhage in this case, there was high suspicion for a hematologic disorder, although no family history was reported and the initial coagulation work up was essentially normal. Unfortunately for this neonate, due to the extent of the hemorrhage, many of the complications often associated with massive hemorrhage ensued. These included jaundice, coagulopathy, cardiovascular collapse, and ultimately, multi-organ failure [1].

The presence of massive SGH from a small linear fracture resulting in a fatal neonatal outcome is exceedingly rare after a seemingly uncomplicated and non-instrumental vaginal delivery. Although rare, this is troubling for obstetricians as there are no clinically useful predictors for the occurrence of spontaneous intrapartum fetal skull fracture. It is important to point out that from a legal perspective, many fetal fractures and injuries may have happened in utero and hence, unrelated to the medical staff's assistance at delivery. It is prudent, therefore, to closely monitor not only babies born via assisted deliveries, but also those who underwent uncomplicated vaginal deliveries, as skull fracture can occur without risk factors or known cause. For this reason, a high level of suspicion must be maintained in all infants who appear to be lethargic, hypotonic, or in respiratory distress. This is true regardless of the method of delivery, as the absence of instrumentation does not preclude fetal trauma and skull fracture.

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