Journal of Neonatal Care
Volume 1 | Issue 1

Case Report Open Access

Hypovolemic Shock Following Subgaleal Hemorrhage with Associated Hypoxic Ischemic Encephalopathy in a Term Infant

Bharti D*, Whittlesey A, Smith V and Hollinger S

Department of Pediatrics, Quillen College of Medicine, Johnson City, East Tennessee State University, Tennessee

***Corresponding author:** Bharti D, Professor of Pediatrics, Quillen College of Medicine, Johnson City, Department of Pediatrics East Tennessee State University, Tennessee 37614, Fax: 423-439-8066, Tel: 423-439-6222, E-mail: bharti@etsu.edu

Citation: Bharti D, Whittlesey A, Smith V, Hollinger S (2018) Hypovolemic Shock Following Subgaleal Hemorrhage with Associated Hypoxic Ischemic Encephalopathy in a Term Infant. J Neonat Care 1(1): 102

Abstract

We are reporting an infant who was born by emergency cesarean section following failed vaginal delivery. This infant was noted to be in hypovolemic shock soon after birth. He developed large subgaleal hemorrhage within 6 hours after birth with associated diffuse intravascular coagulopathy and hypoxic ischemic encephalopathy. He required multiple transfusions of packed red blood cell, platelets transfusion, fresh frozen plasma transfusions and a cryoprecipitate. The infant was on body cooling therapy for hypoxic ischemic encephalopathy for three days. His neurological exam at the time of discharge was unremarkable. At the time of discharge infant is enrolled in early intervention program with a multi-disciplinary follow-up scheduled.

Keywords: Subgaleal Hemorrhage, Hypoxic Ischemic Encephalopathy

List of abbreviations: SGH: subgaleal hemorrhage; HIE: hypoxic ischemic encephalopathy; PRBC: packed red blood cells; DIC: disseminated intravascular coagulopathy; EEG: electro encephalogram; Hct: hematocrit, NICU: neonatal intensive care unit; MRI: magnetic resonant imaging; NPO: nil per os or nothing by mouth)

Introduction

Subgaleal hemorrhage (SGH) is a rare condition in the neonatal period with a potentially lethal outcome. The origin of the hemorrhage is from rupture of the emissary veins that connect dural sinuses with the scalp veins. The blood tends to accumulate between epicranial aponeurosis of the scalp and periosteum. This space is very large and can potentially accommodate the total blood volume of the infant. This space extends from orbital edges in the front, back to the nuchal ridge, and laterally to the temporal facial. Following a large amount of blood loss in this potential space, the infant may develop severe blood volume loss with associated hypovolemic shock. SGH is most commonly seen after vacuum assisted delivery or forceps assisted delivery. It is more frequent in primgravida delivery, shoulder dystocia and precipitous vaginal delivery. The most common findings of SGH are rapid increase in head circumference, bruises and ecchymosis over the scalp, respiratory distress, tachycardia, poor skin perfusion, pallor and jaundice. SGH may progress very rapidly leading to hypovolemic shock, respiratory distress, prolonged apnea, perinatal asphyxia, seizures and sudden death. Early diagnosis and quick management of hypovolemia and blood loss is a key for survival and good outcome of these infants. Acute blood loss and ensuing hypovolemia may have multi-system involvement including respiratory failure, disseminated intravascular coagulopathy, hypoxic ischemic encephalopathy, seizure disorder, cardiac dysfunction, electrolyte imbalance, and renal failure.

Case presentation

A full-term, male infant was born by urgent cesarean section because of failed progression of labor. He had Apgar scores of 1, 1, and 4 at 1, 5 and 10 minutes respectively. The mother is G1P0, her blood type is A positive, and she was GBS screen positive. She received 2 doses of penicillin G prior to delivery. She had unremarkable pregnancy and normal prenatal studies. She denied history of smoking, drug use, or alcohol use.

Mom was in second stage of labor for over two hours. Infant's head was impacted in the pelvis and he required a vaginal push for the delivery of the head at cesarean section. There was evidence of fetal distress with type two tracing. Cesarean section was performed under epidural anesthesia. Infant was noted to have acute pallor and respiratory distress soon after birth. He was dusky, hypotonic, and hypoactive with significant molding of the head. Infant was started on positive pressure ventilation via face mask, and he required chest compressions for thirty seconds. He was intubated and connected to a respirator at the level one nursery.

Infant was noted to have decreased breath sounds on the right side. Chest x-ray showed tension pneumothorax on the right side. A thoracentesis was performed initially and about 30 ml of free air was suctioned out at referral hospital. Follow up chest x-ray on transfer to NICU showed re-expansion of the lungs. Infant had recurrence of right pneumothorax requiring two additional thoracentesis in first 24 hours of life.

He weighed 2925 g, length was 49.5 cm, and head circumference was 34.2 cm. Infant was given vitamin K immediately following delivery. Initial vital signs were: Heart rate 157 per minute, respiratory rate 75 per minute, axillary temperature 95.2° F, arterial blood pressure 60/41(50th percentile for systolic and diastolic BP). Infant had significant molding and a misshapen head, and boggy swelling that was noted all over the head with no bruising of the skin. The pupils were dilated and nonreactive. The findings of dilated, fixed pupils and associated significant hypotonia were consistent with severe hypoxic ischemic encephalopathy. The infant had substernal retractions, tachypnea, with equal breath sounds bilaterally (after initial thoracentesis on the right side). Umbilical artery and venous catheters were placed. Infant was given two infusions of normal saline 30 ml each. Infant received 45 ml of unmatched O negative packed red blood cells. Hematocrit was 40.7% prior to departure from the nursery at referral hospital to the NICU. Infant was transported to level three neonatal ICU without any complications. He was extubated and weaned to bubble CPAP.

Soon after admission to NICU, the infant was noted to have worsening skin pallor with tachycardia; he received 60 ml of matched PRBC. The infant had profound metabolic acidosis with lactate level of 20.3 moll/l, pH 6.9, CO2 24, PaO2 85, unmeasurable HCO3 and base deficit. A follow up venous hematocrit two hours after PRBC transfusion was 22.8%. The infant received an additional transfusion of 73 ml of matched PRBC. Repeat hematocrit continued to be low at 21.9%; 45 ml PRBC transfusion was given with an additional transfusion of 45 ml given prior to next lab value. Follow up hematocrit was 20.1%, 45 ml PRBC transfusion was given at this time. At approximately 27 hours of life hematocrit was 27.9%, the patient received another 45ml PRBC transfusion. No further PRBC transfusions were needed. Graph1 depicts the change in hematocrit for our index patient during the hospital stay. Additional resuscitation included 140 ml fresh frozen plasma given in four transfusions; 90 ml platelets were given in 3 transfusions and the infant received 30 ml of cryoprecipitate.

In the first 24 hours of life, the infant received 160 ml/kg of blood products which included 100 ml/kg of packed red blood cells. Initial coagulation profile study revealed: Fibrinogen 35 mg/dl; platelets 130,000/ul; INR 3.1; PTT >200. Follow-up coagulation studies prior to discharge were in normal range. In first 24 hours the infant had eight cm increase in head circumference with downwards rotation of the ears and bruises were noted under the eyes bilaterally. However, by day four of life the infant was found to have a decrease in head circumference to the baseline. The infant had a low but stable platelet count of 58,000/ul prior to discharge. The highest bilirubin recorded during hospital stay was 12.9 mg/dl.

The infant was extubated on arrival to NICU, he required bubble CPAP for two days. After that time he remained stable on room air. He had few bradycardias with brief desaturation during rewarming from body cooling. There was a gradual improvement in tone in the first 24 hours and by 72 hours of age, the infant had normal neurological examination. The infant was kept NPO while undergoing 72 hours of body cooling. Following rewarming, he was started on oral feedings. The infant was on full feedings by day five of life. Infant was initially found to be hypotensive and he was on dopamine at five microgram/kg/minute for three days. Report of head ultrasound on day 1 (figure-1) showed a large scalp hematoma with normal ventricles. There was no evidence of germinal matrix hemorrhage, parenchymal hemorrhage, or intraventricular hemorrhage. The MRI of the head without contrast showed large subgaleal hemorrhage, two punctate foci of diffusion restriction in the right frontal and left frontal region. These foci were thought to be consistent with ischemia. Figure 2 and 3 shows sagittal and coronal MRI views with large SDH indicated by arrows.

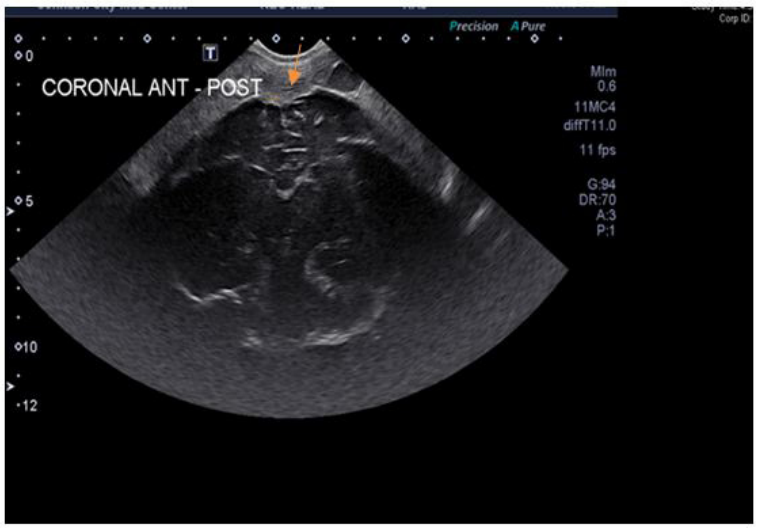


Figure 1: Coronal sonographic view with normal ventricles and blood noted anterior to the ventricles indicated with arrow.

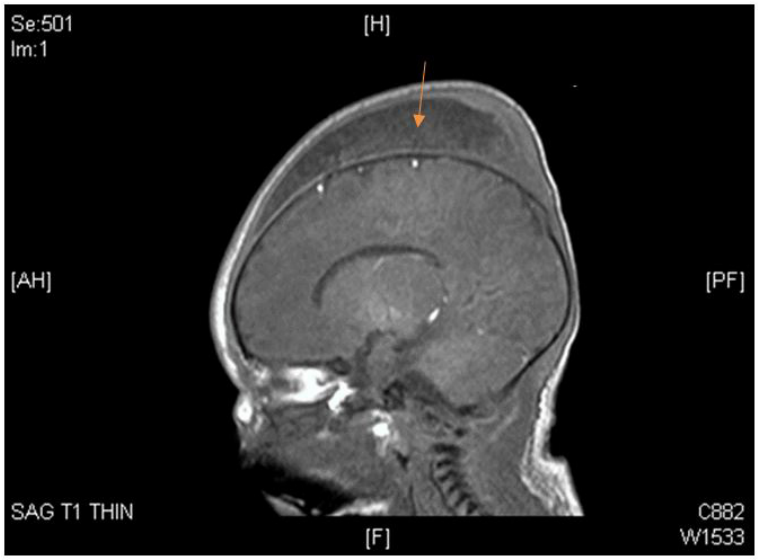


Figure 2: Sagittal MRI scan with large SGH indicated by arrow.

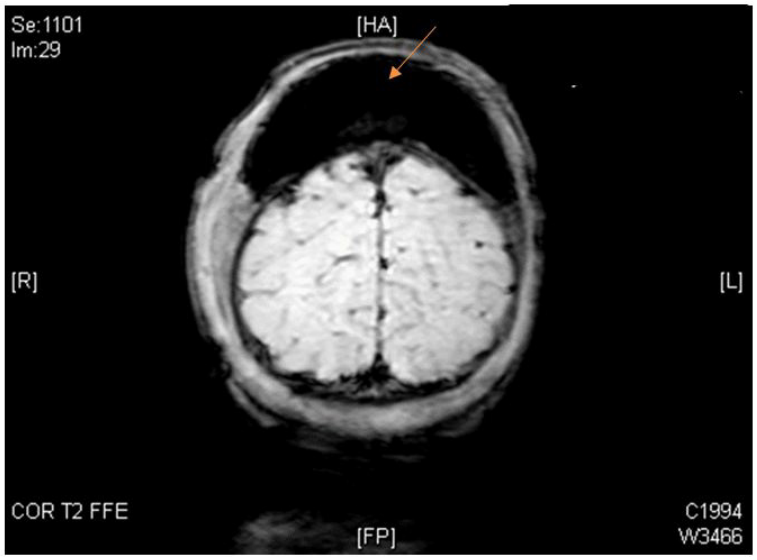


Figure 3: Coronal MRI scan with large SGH indicated by arrow

At the time of discharge the infant had normal tone and activity. He was nursing on maternal breast milk. He was discharged to primary care physician office. A follow-up examination in the high risk clinic, at 1 year of age showed normal neurological examination and appropriate acquisition of milestones.

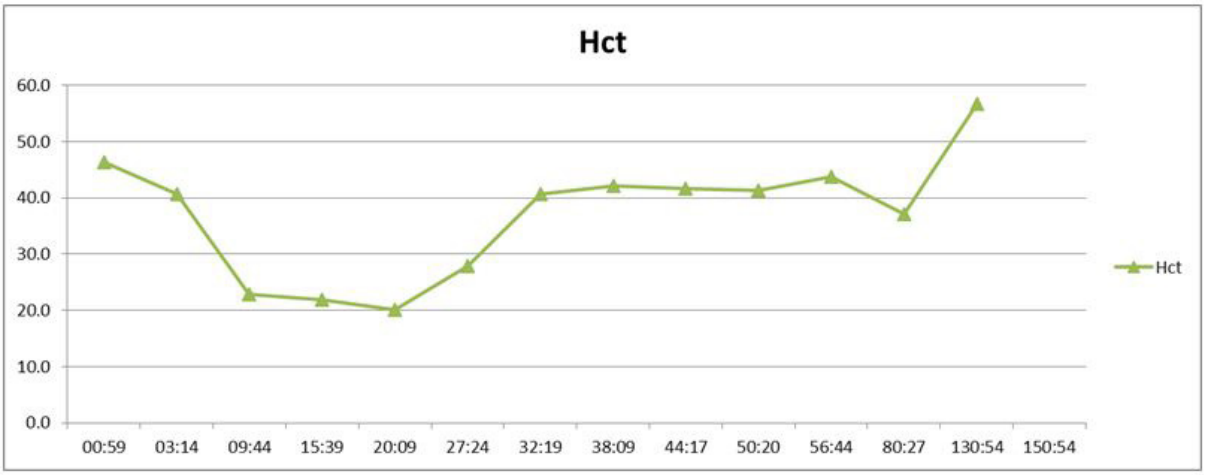
Discussion

Subgaleal hemorrhage is a rare occurrence in the neonatal period. The incidence of subgaleal hemorrhages is approximately 1.5 per 10,000 births [1]. A fatal outcome can be seen in over 25% of the infants admitted to NICU with subgaleal hemorrhage. Even though typically it is the result of instrumentation, spontaneous subdural hemorrhage has been reported [2]. SGH is commonly seen after vacuum extraction or with the use of forceps for vaginal delivery. Incidence is higher in large for gestational age infants with associated shoulder dystocia. Vacuum use is reported in approximately 49% of all subgaleal hemorrhage [3]. Risk factors associated with SGH after vacuum- assisted delivery are Nulliparous mother, sequential use of vacuum and forceps, Apgar score less than 8 at 5 min following vacuum assisted delivery, cup edge close to anterior fontanel, cup centered more than 1 cm lateral to sagittal suture [4]. SGH risk with vacuum extraction is associated with multiple pop-offs, applications more than 10 minutes, increased number of pulls, incorrect manipulation of the vacuum, jerking, rocking, or rotational pulls. The steady smooth pulls that mimic natural labor are recommended.

Galea aponeurotica is a thick fibrous tissue that covers the entire scalp. It has close attachment to the skin and subcutaneous tissues. There are several large emissary veins under this aponeurosis. With the application of forceps or vacuum, there is an intense amount of force that can tear these emissary veins. The subgaleal space potentially can accommodate the total blood volume of the infant. The subgaleal area is not limited by sutures. There are no barriers to prevent hemorrhage. A massive hemorrhage can quickly occur.

Acute blood loss can be associated with severe hypovolemic shock that can be potentially fatal unless managed aggressively and appropriately. In majority of infants the diagnosis can be made in the first few hours of life. The classical presentation is a soft boggy fluctuating mass all over the scalp that is not limited by sutures. The ears can be displaced inferiorly and posteriorly. Blood loss can be associated with extreme pallor, hypotension, tachycardia, hypo tonicity and potentially a respiratory compromise. Discoloration of the scalp is a late sign. The bruising and discoloration of the skin may develop and spread to neck, superior orbital ridge, upper eyelids, lower eyelids and ears can be displaced downwards and backwards bilaterally. The severity of the blood loss can be measured by frequent hematocrit measurement, frequent measurement of head circumference, frequent blood pressure assessment along with monitoring of the platelet count and coagulation studies. A swift diagnosis, careful monitoring and prompt treatment is the key for the good outcome for these infants. The diagnosis is made by quick review of the maternal history, birth history and physical examination of the infant. Majority of the infants presenting with subgaleal hemorrhage have had vacuum assisted delivery or forceps application. Our patient's head was impacted deep in the pelvis and significant pressure had to be exerted from the vaginal canal to deliver the head through cesarean incision.

Infants presenting with subgaleal hemorrhage need frequent physical examination on hourly basis, continuous monitoring of the vital signs, monitoring of the blood pressure and assessment of urinary output. Initial hypovolemic shock is managed by normal saline infusion and transfusion of unmatched O- negative packed red blood cells preferably through umbilical vein.



Graph 1: This graft depicts the change in hematocrit for our patient during the hospital stay. X-axis is hours after birth and Y–axis is Hct %.

Complications associated with SGH include anemia, hyperbilirubinemia, hypovolemic shock, renal failure, disseminated intravascular coagulopathy, hypoxic ischemic encephalopathy with resultant neurologic deficit. Bacterial infection in an SGH is extremely rare. There are some rare reported cases of SGA getting infected and they were not associated with generalized sepsis. Respiratory support with ventilator or noninvasive ventilation may be required in infants with severe hypoxic ischemic encephalopathy until infant has spontaneous breathing and correction of respiratory and metabolic acidosis.

Hypovolemic shock is defined as compromised intravascular blood volume leading to in adequate tissue perfusion and manifesting clinically as pallor with associated hypoactivity. Initially, blood pressure could be maintained in normal range with a blood loss of up to 25% of blood volume, however, with a blood loss of 40% or more there is persistence of poor perfusion of tissues, hypotension and associated low urine output. Degree of hypovolemia may not be easy to be gauged immediately after birth because hypovolemia and asphyxia both lead to vasoconstriction, pallor, tachycardia and acidosis. Key factor in the management includes adequate perfusion of brain, heart, kidneys and bowel. Shock is managed by infusion of crystalloids and colloids. Our index patient was given two infusions of normal saline immediately followed by multiple infusions of packed red blood cells. He received 100 ml/kg of packed red blood cells in the first 24 hour and a total of 160 ml/kg of blood products in the first 24 hours. Because of the urgency of the situation, the first transfusion was given as unmatched packed red blood cells but subsequent transfusions were appropriately matched blood transfusions. Plauche and associates reported that a 1 cm increase in head circumference could be associated with a blood loss of 40-260 mL [2].

Infants presenting with shock may require echocardiogram to evaluate the cardiac function. Echocardiogram may also be needed in a hypoxic infant to rule out pulmonary artery hypertension. Dopamine infusion at 3 to 10 mcg per kg per minute may be indicated to improve cerebral, mesenteric and renal blood flow. For severely hypotensive infants, addition of intravenous hydrocortisone may be helpful after the initial hypovolemia has been corrected. The infants with hypotension not responsive to volume infusion and dopamine, benefit with hydrocortisone with rapid normalization of the cardiovascular status and sustained decreases in volume and pressor requirement [5-6].

Disseminated intravascular coagulopathy is managed by multiple transfusions of packed red blood cell, platelets, fresh frozen plasma, and cryoprecipitate. Frequent monitoring of coagulation profile is important until DIC is resolved. Our patient required

four infusions of fresh frozen plasma, one transfusion of cryoprecipitate and two platelet transfusions during hospital stay. Effective and aggressive management of DIC is important to avoid the risk for intracranial hemorrhage.

Sarnat classified hypoxic encephalopathy in 3 stages. Stage I is associated with mild alteration in level of consciousness with normal tone and posture, normal reflexes and mydriasis. Stage II is associated with lethargy, hypotonia, decorticate posturing, variability of reflexes and miosis. Stage III is associated with stupor, flaccidity, absent deep tendon reflexes, unequal pupils with poor light reflex. Our patient had poorly responsive, dilated fixed pupils, complete flaccidity on admission. Deep tender reflexes were not elicited. Initial clinical picture was consistent with stage III hypoxic encephalopathy. We noted significant improvement in infant's breathing, tone and activity over the first 24 hours of life.

The benefit of induced hypothermia with body cooling in infants with hypoxic-ischemic encephalopathy has been proven in high-quality randomized controlled trials [7]. Incidence of death and disability at 18-22 months of age is lower with this therapy. The current evidence does not support cooling of infants with mild hypoxic-ischemic encephalopathy (HIE) or those born before 35 weeks. Body cooling is indicated in infants with evidence of intrapartum hypoxia that includes Apgar score 5 or less at 10 minutes, need for mechanical ventilation and/or ongoing resuscitation at 10 minutes, metabolic or mixed acidosis defined as arterial cord gas, or any blood gas within the first hour of life showing pH of 7 or less, or base deficit of ≥12 mmol/l. Infants are not eligible for body cooling if birth weight is less than 2000 g, gestational age less than 36 weeks, inability to initiate cooling by 6 hours of age, life-threatening abnormalities of the cardiovascular or respiratory systems such as complex congenital heart disease of the newborn, major congenital malformations, suspected neuromuscular disorders, or presence of known lethal chromosomal anomaly, or when death appears inevitable. Targeted rectal temperature during body cooling is 33.5 to 34.5 degree Celsius. The total period of cooling is 72 hours. Upon completion of therapy, the infants are gradually re-warmed over at least 4 h. Hypothermia increased survival with normal neurological function at 18 months and in survivors reduced the rates of severe disability, and cerebral palsy [8-9]. An infant with moderately severe HIE presents with lethargy, hypoactivity, constricted pupils while severe HIE is manifested by dilated and unresponsive pupils, decerebrate posture and apnea. Our patient had unresponsive dilated pupils but he was having spontaneous irregular shallow breathing on admission to NICU.

Conclusions

Early assessment of subgaleal hemorrhage with prompt volume infusion and blood replacement is indicated for good outcome and to avoid possible sequelae of post hemorrhagic shock that can lead to death or disability. Careful attention needs to paid for resuscitation and respiratory support, maintenance of perfusion and blood pressure, management of DIC, early introduction of body cooling in asphyxiated infants and careful monitoring of neurological function. EEG and neuroimaging during or prior do discharge is indicated. These infants require multidisciplinary follow-up after discharge.

References

1. Chadwick LM, Pemberton PJ, Kurinczuk JJ (1996) Neonatal subgaleal hematoma: associated risk factors, complications and outcome. J Paediatr Child Health 32:228-32.

2. Plauché WC (1980) Subgaleal hematoma. A complication of instrumental delivery. JAMA 3; 244:1597-8

3. Schierholz E, Walker SR (2010) Subgaleal hematoma. A complication of instrumental delivery.

4. Patient Safety authority. Advisory Library. 2009

5. Ng Pak C, Lee Cheuk H, Bnur FL, Chan IH, Lee AW, et al. (2006) A Double-Blind, Randomized, Controlled Study of a "Stress Dose" of Hydrocortisone for Rescue Treatment of Refractory Hypotension in Preterm Infants Refractory Hypotension in Preterm Infants. Pediatrics 117: 367-75.

6. Istvan Seri, Rosemarie Tan, Jaquelyn Evans (2001) Cardiovascular Effects of Hydrocortisone in Preterm Infants With Pressor-Resistant Hypotension. Pediatrics 107: 1070-4.

7. Mosalli R (2012) Whole Body Cooling for Infants with Hypoxic-Ischemic Encephalopathy. J Clin Neonatol 1: 101–6.

8. Perlman JM, Davis P, Wyllie J, Kattwinkel J (2010) Therapeutic hypothermia following intrapartum hypoxia-ischemia. An advisory statement from the Neonatal Task Force of the International Liaison Committee on Resuscitation. Resuscitation 81: 1459-61.

9. Azzopardi DV, Strohm B, Edwards AD, Dyet L, Halliday HL, et al. (2009) Moderate hypothermia to treat perinatal asphyxial encephalopathy. N Engl J Med 361: 1349-58.