Announcements
Faculty, Staff and Residents

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Lee selects West Tennessee hospital executive to be new health commissioner (1/18/19)

Gov. Elect Bill Lee has selected Dr. Lisa Piercey to serve as the next commissioner of the Department of Health.

Gov.-Elect Bill Lee has selected a West Tennessee hospital executive to lead the Tennessee Department of Health in his administration, according to a Thursday press release. Dr. Lisa Piercey, of Gibson County, was appointed to serve as the next commissioner of the Department of Health.

Piercey has a clinical background in pediatrics and specializes in child abuse pediatrics. She also serves as the medical director for the Madison County Child Advocacy Center and a faculty member at the Vanderbilt University School of Medicine.

She will succeed Dr. John Dreyzehner as the next health commissioner, and in her new role, she will be responsible for overseeing the Ballad Health hospital merger, ensuring it abides by the Terms of Certification agreed upon as part of the Certificate of Public Advantage.

Congratulations to Dr. Evan Los! He has been appointed to be communications director for the diabetes in Youth Special Interest Group of the Americans Diabetes Association.

As most of you are aware, Dr. David Wood recently announced that he was stepping down as Chair of Pediatrics pending the hiring of a replacement. As this process is taking longer than anticipated, we have agreed to transition the Chair to interim leadership for the near future. Dr. Wood’s contributions to the Department and to the health of the children of the region are many. Please join me in thanking him for his vision and service to the College and Department. As of January 1, 2019, Dr. Dawn Tuell will begin serving as Chair of Pediatrics (Interim). Please join me in welcoming Dr. Tuell to her new position in the Department of Pediatrics.

As transitions occur in the pediatric service lines of Ballad and East Tennessee State University, Dr. Darshan Shah has decided to relinquish his role as Division Director of Neonatology and focus his efforts on patient care, research, and education within the Department of Pediatrics.

Dr. Shawn Hollinger has agreed to begin serving as the Medical Director of Neonatology, Northeast Tennessee Regional Perinatal Center, and the Division Director for Neonatology. Drs. Hollinger and Shah partner with Dr. Bharti to provide high quality care to the most critically ill newborns in the region. Their efforts over the years have resulted in amazing outcomes and many lives saved.

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NEONATOLOGY

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JOURNAL PUBLICATIONS:


ORAL PRESENTATIONS:

Pictured Above:


ABSTRACTS AND/OR POSTER PRESENTATIONS:

Hajianpour, MJ, Expanding the phenotype of MED13L-associated mental retardation and distinctive facial features with or without cardiac defects; MRFACD. “2018 American Society of Human Genetics Annual Conference”

February 1st, National Go Red Day, The Department of Pediatrics wore red in order to pursue a common goal of raising awareness of cardiovascular disease.
Hypovolemic Shock Following Subgaleal Hemorrhage with Associated Hypoxic Ischemic Encephalopathy in a Term Infant

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

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

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Introduction (Continued)
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 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 thoracenteses 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 subclinical 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 mmol/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. To continue reading, please access the link below.