



Necrotizing Enterocolitis Following Repair of Congenital Diaphragmatic Hernia – Case Report

Naga Venkatesh K*, Karthikeyan Kadirvel, Harikrishna Devadas

Department of Pediatrics, Mahatma Gandhi Medical College, India

Abstract

Necrotizing Enterocolitis (NEC), seen primarily in preterm neonates, is a severe condition associated with significant mortality and morbidity. A 26-year-old primigravida, without any prior comorbidity, had her baby diagnosed antenatal with left sided Congenital Diaphragmatic Hernia (CDH) containing the left lobe of liver, stomach and intestine. Antenatal ultrasound showed an observed lung to head ratio of 44.5% and quantitative lung index of 0.5. The rest of the ultrasound examination was normal. The case series of post surgery NEC by Shanbhogue et al. showed the leading cause being neurosurgery.

Keywords: Necrotizing enterocolitis; Congenital diaphragmatic hernia; Abdominal compartment syndrome

Introduction

Necrotizing Enterocolitis (NEC), seen primarily in preterm neonates, is a severe condition associated with significant mortality and morbidity [1]. The etiology of NEC is not entirely understood, and various mechanisms are postulated in the development of NEC. In a susceptible host, a combination of factors like ischemia to intestines, impaired or immature gut barrier, infectious agents and cytokines and growth factors contribute to the NEC development [2,3]. However, other factors like increased abdominal pressure, intestinal abnormalities, perinatal stress, heart defects, which could affect mesenteric blood flow, can also cause NEC [4,5].

NEC following abdominal surgeries has been reported and is more commonly associated with post gastroschisis. Even though it is rare, postoperative NEC is a severe condition with overall survival rates between 33% and 69% and should be considered in the differential diagnosis in case of post-surgical degradation [6]. These cases are such situation need to be reported to help in early diagnosis and appropriate management.

NEC following congenital diaphragmatic hernia repair only few cases are reported in the literature. Here, we report on our experience concerning an unusual NEC with intestinal perforations of the small bowel following the surgical repair of a left congenital diaphragmatic hernia.

Case Presentation

A 26-year-old primigravida, without any prior comorbidity, had her baby diagnosed antenatally with left sided Congenital Diaphragmatic Hernia (CDH) containing the left lobe of liver, stomach and intestine. Antenatal ultrasound showed an observed lung to head ratio of 44.5% and quantitative lung index of 0.5. The rest of the ultrasound examination was normal.

She delivered a male neonate at 37+1 weeks of gestation by spontaneous vaginal delivery with weak cry at birth with birth weight 2,400 grams and developed cyanosis immediately after birth with APGAR was 6/10 at one minute of life. The child was intubated in delivery room and shifted to intensive care and he was managed with ventilator. Initial medical examination revealed a scaphoid abdomen and isolated left asymmetry of the pulmonary auscultation. A nasogastric tube was inserted for decompression, and left CDH was confirmed by chest X-ray.

The child maintained saturation (pre ductal and post ductal SPO₂ >95%) and hemodynamic stability on initial ventilator settings (FiO₂ to 40%, ventilatory rate- 40/min, Peak pressure -12, PEEP-6). Preductal arterial blood gas showed: pH-7.4, pO₂-189, pCO₂-27, HCO₃-17. He maintained a 2 ml/kg/h urine output, capillary refilling time of 2 sec, and normal volume pulses. ECHO revealed normal pulmonary pressures with no structural or vascular defects.

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*Correspondence:

Naga Venkatesh K, Department Of Pediatrics, Mahatma Gandhi Medical College, 54-3-23/7, Sarva Residency F-3, AVA Road , Rajahmundry, Andhra Pradesh, 533101, India, Tel: 08147430335; E-mail: venkatesh.kuntamukkala@gmail.com

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Figure 1: Abdominal muscle closer was not attempted, and skin closure was done.

The initial stability of the child allowed us to perform the repair of CDH after 48 h of life. The repair was performed by laparotomy using a sub-costal approach. There was a large left posterolateral CDH containing the small intestine, colon, stomach, spleen and left lobe of the liver in the left intrathoracic position. It was reported as a C defect according to the Congenital Diaphragmatic Hernia Study Group classification [7]. After examination and reduction of the CDH contents, the defect was primarily repaired. On attempted closure of abdominal muscles, there was undue tension and difficulty in ventilation. Hence only skin closure was done, leaving a ventral hernia. Intravesical pressure measured with the Foley catheter was 12 mmHg.

Postoperatively the child was maintained on conventional ventilation (setting) and sedated with midazolam (8 mcg/kg/min) and fentanyl (2 mcg/kg/min). On the POD day 1, the child maintained normal saturation, hemodynamic stability and had a 2.4 ml/kg/h urine output. On the POD day 2, the child had decreased perfusion manifested by tachycardia, prolonged CFT, low blood pressure and dobutamine was started at 15 mcg/kg/min. An echocardiogram done postoperatively showed no pulmonary hypertension or ventricular dysfunction. The perfusion improved, and there was no further worsening of ventilator requirements.

Bowel sounds were present in the Postoperative Day (POD) 3, and small amounts of enteral feeding were started. However, the patient had feed intolerance manifested by non-bilious vomiting, and hence feeds were stopped, and parental nutrition was started. The sepsis screen was normal. Antibiotics were upgraded after taking blood culture, which was sterile. The abdominal radiograph showed dilated bowel loops. The patient was extubated on POD5 since the child remained stable with inotropic support. However, on POD6, we noticed feculent staining of the abdominal wound; the patient was taken for laparotomy with the suspicion of perforation.

Exploration was done through the same incision site. Examination showed two perforations (2.5 cm and 0.5 cm) with an intervening gangrenous bowel at the distal ileum 25 cm proximal to the ileocolic junction. Ileum distal to perforation showed serosal tears and edema. The colon and rest of the bowel were normal, and there were no other significant findings. Double barrel ileostomy was fashioned at the right iliac region. Abdominal muscle closer was not attempted, and skin closure was done (Figure 1).

The postoperative course following the second surgery was uneventful, stoma functioned on POD3, and feeds were started and slowly increased. The baby was extubated on POD4. Full feeds were

established on POD-7. There was mild wound dehiscence which required secondary suturing under local anesthesia. Weight gain was adequate, and the child was discharged at 32 day of life.

Discussion

NEC following abdominal surgery is rarely seen in full term neonates. Postoperative NEC is seldom encountered but can be associated with serious complications. Any operation on a neonate can predispose to the development of postoperative NEC and this diagnosis should be considered. The case series of post surgery NEC by Shanbhogue et al. [6], showed the leading cause being neurosurgery. A high incidence of NEC following repair of gastroschisis has been reported previously. Oldham et al. described 54 infants with gastroschisis, ten of whom developed 21 episodes of NEC.

NEC following primary repair of CDH is a rare event, in the case series by Shanbhogue et al. 5/33 patients had diagnosis of CDH [6]. Other than that only two case reports been reported about development of NEC following primary repair in CDH.

In this case, the perioperative description of the intestines during the CDH repair does not show any sign of abdominal reintegration seems to be responsible for the secondary development of NEC. Post-surgery intravesical pressure measured with Foley Catheter showed normal range and there is no evidence of mismatch perfusion and hypoxia during NICU stay of baby. Another hypothesis might be the pulmonary hypertension, leading to perfusion deficiencies and thus diffuse ischemic phenomena on the entire intestines that would explain the NEC development affecting the small and large bowels but in this case there is no evidence of pulmonary hypertension as echocardiography done post surgery showed normal pulmonary pressure. Raised Intra-Abdominal Pressure (IAP) and Abdominal Compartment Syndrome (ACS) can be involved in the cause of NEC. It causes a decrease in organ blood flow in splanchnic organs leading to intestinal ischemia. But we had made a ventral hernia, and the measured intravesical pressure IVP was also normal. The child maintained adequate urine output throughout the admission, making the possibility of ACS is less [7].

Also, in the case of CDH, selective intestinal ischemia may occur during periods of hypoxia. But in our child, there was no episode of hypoxia during the surgery or perioperative period. The echocardiograms done at 48 h and post-surgery showed no pulmonary hypertension or other structural heart defects and hence cannot be the cause. On POD2, although there was a mild tachycardia and cool extremities, the blood pressure and peripheral perfusion was maintained throughout the time.

It seems that this case of NEC following CDH repair is likely due to a combination of many factors, and we couldn't find an apparent cause. Postoperative NEC is a severe condition and can be encountered after any surgery in a newborn irrespective of the term. The survival rates are reported between 33% to 69%. Hence, NEC's diagnosis should be considered in neonates following surgery, notably when they have abdominal symptoms.

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