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NECROTIZING ENTEROCOLITIS AND TWIN-TO-TWIN TRANSFUSION SYNDROME: A RARE CASE REPORT

Pham Ngoc Minh[1], Nguyen Hien[1], Bui Thanh Son[2], Pham Ngoc Thach[1*]

[1]*Children's Hospital 2 - 14 Ly Tu Trong, Saigon Ward, HCM City*
[2]*University of Medicine and Pharmacy, HCM City -217 Hong Bang, Cho Lon Ward, HCM City*

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ABSTRACT

Overview: Necrotizing enterocolitis (NEC) is a critical gastrointestinal emergency in preterm infants. Twin-to-Twin Transfusion Syndrome (TTTS) represents a significant risk factor, particularly for the donor twin due to chronic intestinal hypoperfusion.

Case presentation: A male infant, born at 31 weeks 4 days, birth weight 900g, was the donor twin in a TTTS pregnancy. The patient presented with signs of bowel obstruction, abdominal distension, and bilious gastric residuals. Preoperative diagnosis suggested duodenal atresia. Intraoperative findings revealed a segment of focal necrotic bowel causing obstruction, 65cm from the Treitz ligament. Resection and end-to-end anastomosis were performed. Histopathology confirmed NEC with the presence of ganglion cells.

Discussion: This case illustrates the ischemic mechanism in donor twins leading to intestinal mucosal injury and necrosis, presenting as bowel obstruction rather than classic peritonitis.

Conclusion: A high index of suspicion for NEC is required in preterm donor twins of TTTS, even with atypical clinical and radiological presentations.

Keywords: Necrotizing enterocolitis; Twin-to-twin transfusion syndrome; Donor twin, preterm; Neonatal intestinal obstruction.

1. INTRODUCTION

Necrotizing enterocolitis (NEC) is a common condition in premature infants, characterized by intestinal mucosal ischemia and inflammation leading to necrosis, typically presenting after the initiation of oral feeding. It is considered a prevalent gastrointestinal emergency in the Neonatal Intensive Care Unit (NICU) and represents one of the leading causes of mortality in low-birth-weight preterm infants. The exact pathogenesis of NEC remains incompletely understood. Current studies hypothesize that NEC has a multifactorial etiology involving formula feeding, intestinal ischemia, gut dysbiosis, and an immature immune system. Twin-to-Twin Transfusion Syndrome (TTTS) occurs in approximately 10 – 15% of monochorionic twin pregnancies. TTTS results from an unbalanced blood flow across vascular anastomoses within the shared placenta. If this unbalanced flow is significant, it causes one twin to "donate" blood volume to the other. The donor twin experiences hypodynamic and hypovolemic states, leading to decreased renal perfusion, activation of the renin-angiotensin-aldosterone system, subsequent oliguria, decreased intestinal perfusion and heart failure. We report a case of NEC presenting as intestinal obstruction in a premature donor twin of a TTTS pregnancy to emphasize the role of ischemic mechanisms in this pathology.

2. CASE PRESENTATION

The patient was a male infant, the second of a twin pair, born via cesarean section at 31 weeks and 4 days due to fetal distress, with a birth weight of 900 grams (the first twin weighed 1500 grams). Prenatal history noted TTTS without intervention. The patient was the donor twin, presenting with oligohydramnios, chronic anemia and intrauterine growth restriction (IUGR). After birth, the infant had tachypnea and mild abdominal distension requiring respiratory support. An initial unenhanced abdominal X-ray revealed a massively dilated bowel loop in the subhepatic region, leading the maternity hospital to suspect partial duodenal obstruction and transfer the patient to Children's Hospital 2. At 2 days of age, the infant developed progressive abdominal distension, bilious nasogastric residuals and had not passed meconium. Upon clinical examination, the infant was alert, moving extremities and not irritable. The abdomen was moderately distended in the epigastric region, soft on palpation, without rebound tenderness or erythema of the abdominal wall. A rectal enema yielded no meconium. No other associated congenital anomalies were noted. Hematological indices showed a decreased white blood cell count of 4.69 x10^3/µL, hemoglobin of 14.8 g/dL and a platelet count of 173 x10^3/µL. Other biochemical tests were within normal limits. An abdominal X-ray demonstrated massive dilation of the stomach and duodenum, a hazy abdomen, an absence of gas in the lower gastrointestinal tract and no evidence of pneumoperitoneum or pneumatosis intestinalis. An abdominal ultrasound revealed markedly dilated bowel loops, a small amount of ascites and normal anatomical positioning of the mesenteric vessels. An upper gastrointestinal (UGI) contrast study was indicated, showing delayed contrast transit, retention in the stomach, dilated bowel loops, and an absence of gas in the pelvic region. The patient was indicated for an exploratory laparotomy. Intraoperatively, the clear yellow ascites was noted. Exploration of the entire small

*Corresponding author
Email: dr.thachpham@yahoo.fr **Phone:** (+84) 945275112 **Https:**//doi.org/10.52163/yhc.v67iE1.5287

intestine revealed a segment of small bowel that was massively dilated, with a purplish-black, focally necrotic wall, and aperistaltic, measuring approximately 15 cm in length. The proximal margin of the lesion was 65 cm from the ligament of Treitz and the distal margin was 55 cm from the ileocecal valve. Examination showed a normal stomach and duodenum; normal anatomical positions of the ligament of Treitz and the cecum; and a microcolon. We resected the necrotic bowel segment and flushed the distal bowel with physiological saline, confirming good patency. An end-to-end with single-layer interrupted anastomosis was performed. The resected necrotic bowel segment was sent for histopathological examination.

The patient was reintroduced to oral feeding on postoperative day 5 with gastric residuals becoming clear and spontaneous passage of thick green stools. On postoperative day 10, the infant was extubated and transitioned to Nasal Continuous Positive Airway Pressure (NCPAP). Due to pneumonia and IUGR, the patient required prolonged treatment in the NICU.



Figure 1. Upper gastrointestinal contrast study showing delayed contrast transit, gastric retention, dilated bowel loops and an absence of gas in the pelvic region.



Figure 2. The small bowel segment had inflamed, necrotic, aperistaltic and dilated four times the size of the normal distal bowel.

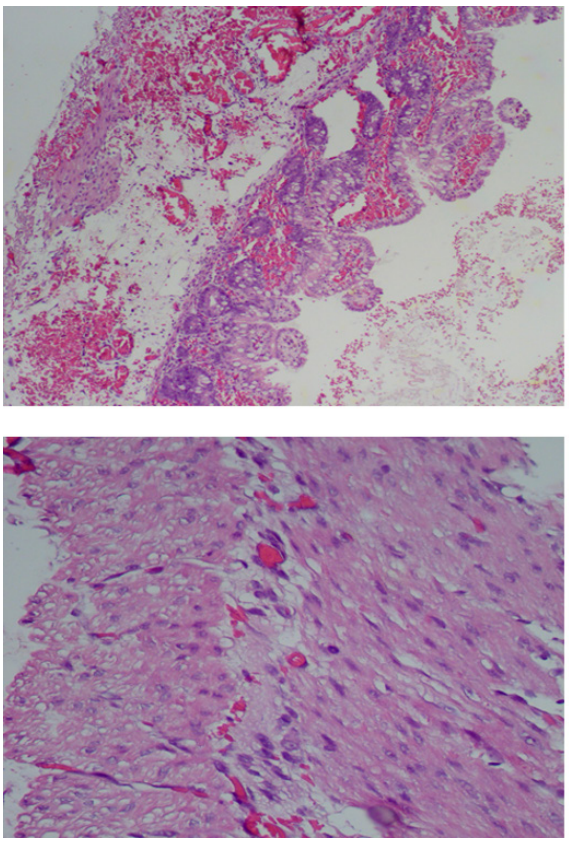


Figure 3. Extensive hemorrhagic and necrotic intestinal tissue with dense inflammatory cell infiltration and the presence of mature ganglion cells between the two muscle layers.

3. DISCUSSION

Our patient illustrates the complex and profound relationship between TTTS and the onset of NEC, while highlighting the diagnostic challenges when the clinical presentation mimics mechanical bowel obstruction rather than classic peritonitis. Regarding pathogenesis, this case aligns with modern literature confirming TTTS as a severe, independent risk factor for NEC. A 2024 study by Rebai et al. on an identical twin model demonstrated that monochorionic twins with TTTS complications have a significantly higher incidence of NEC (70%) compared to those without TTTS (46%), particularly when treated with amnioreduction. The primary mechanism is identified as prolonged intestinal hypoperfusion. In the donor twin, hemodynamic imbalance leads to chronic hypovolemia, activating the renin-angiotensin-aldosterone system and inducing peripheral vasoconstriction to prioritize blood flow to the brain and heart. Consequently, intestinal mucosal injury occurs in utero, predisposing the infant to fulminant necrosis upon postnatal exposure to bacteria and enteral feeding. This explains why our patient, a donor twin weighing only 900 grams compared to the 1500-gram recipient, developed severe disease as early as day 2 of life. The most challenging aspect of this case was the atypical clinical presentation, easily mimicking congenital obstructive pathologies. The patient presented predominantly with high bowel obstruction: bilious emesis, localized epigastric distension, and an unenhanced abdominal X-ray showing massive gastroduodenal dilation with a hazy abdomen and absent distal gas, suggesting partial duodenal obstruction or intestinal atresia. The absence of bloody stools in this case contrasts with the 2024 report by Tran et al., where the patient presented with overt bloody

diarrhea. We hypothesize that this discrepancy stems from the histopathological nature of the lesion: a 15-cm focal necrosis of the small bowel causing functional ileus and luminal obstruction, thereby preventing the transit of digestive fluids and blood to the rectum. Furthermore, the low WBC count (4.69 x10^3/µL) in this patient is a crucial laboratory finding reflecting severe sepsis and bone marrow suppression commonly seen in NEC, further aiding in differentiating it from simple mechanical obstruction. Regarding management strategy, the decision to perform an exploratory laparotomy based on clinical and radiological signs of mechanical obstruction was accurate and timely. Although conservative medical management is typically the initial choice for uncomplicated NEC, the presence of obstruction caused by focal bowel necrosis mandates surgical intervention to eradicate the source of infection and restore gastrointestinal continuity. Performing bowel resection with primary end-to-end anastomosis, rather than an enterostomy, was an optimal choice in this context. This decision was based on intraoperative findings of a clearly demarcated lesion, well-perfused residual bowel, and stable hemodynamics, sparing the infant the severe complications associated with a stoma and a major second-stage reversal surgery. Histopathological results documenting the presence of mature ganglion cells ruled out Hirschsprung's disease, confirming the lesion's nature as aseptic ischemic necrosis. From this case, we recommend that clinicians maintain a high index of suspicion for NEC in premature donor twins of TTTS presenting with signs of bowel obstruction, even in the absence of classic signs such as bloody stools or pneumoperitoneum.

4. CONCLUSION

In the current report, alongside the widespread advancement of assisted reproductive technologies, particularly in vitro fertilization, the rate of multiple pregnancies is rising, leading to a concurrent increase in specific complications such as TTTS. This clinical case report documents an occurrence of NEC in a neonate with a history of TTTS, thereby highlighting the increased risk of NEC in this patient population. The robust association between TTTS and NEC is considered the result of a compounding effect of multiple risk factors, including prematurity, chronic intestinal hypoperfusion in the donor twin, and postnatal infection, which ultimately lead to severe complications such as bowel necrosis, perforation, peritonitis, and death. Based on these clinical findings, we strongly advocate for early recognition, vigilant monitoring, and proactive management of premature donor twins from TTTS pregnancies, alongside a readiness for prompt surgical intervention in severe NEC cases. Furthermore, additional in-depth studies are imperative to elucidate the precise pathophysiological mechanisms, thereby facilitating the development of optimal preventive strategies and treatment protocols for this high-risk cohort.

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