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Ileal Atresia in a Preterm Neonate Complicated by Necrotizing Enterocolitis: A Case Report

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ABSTRACT

Background: Ileal atresia is a congenital intestinal obstruction that frequently affects premature neonates and is associated with high morbidity, particularly when complicated by necrotizing enterocolitis (NEC) and sepsis. Optimal outcomes depend not only on surgical intervention but also on comprehensive perioperative management by a multidisciplinary team, particularly neonatologists.
Case Presentation: We report a male preterm neonate born at 32 weeks and 5 days of gestation with progressive abdominal distension, bilious gastric residue, and failure to pass meconium. Initial respiratory distress improved with non-invasive ventilation. However, gastrointestinal deterioration occurred on day four of life, with radiographic findings consistent with NEC stage III and pneumoperitoneum. Emergency laparotomy revealed ileal atresia, and staged surgical management was performed. Discussion: The pediatric or neonatal team played a crucial role in preoperative stabilization, including respiratory support, fluid and electrolyte correction, broad-spectrum antibiotics, and nutritional optimization. Postoperatively, close monitoring of respiratory status, infection control, gradual advancement of enteral nutrition, and growth assessment enabled recovery without major complications. Full oral feeding was achieved, and the patient was discharged in stable condition on day 27 of life. Conclusion: This case highlights the pivotal role of pediatricians or neonatologists in perioperative management of ileal atresia in premature neonates, emphasizing that meticulous supportive care significantly contributes to favorable surgical outcomes.

1. INTRODUCTION

Ileal atresia is a congenital anomaly of the small intestine characterized by discontinuity of the ileal lumen, resulting in intestinal obstruction during the neonatal period. The primary pathophysiological mechanism of this condition is explained by the classic vascular theory proposed by Louw and Barnard, which suggests that intrauterine vascular disruption of branches of the superior mesenteric artery leads to ischemia, necrosis, and subsequent resorption of the affected intestinal segment during gestation (Louw & Barnard, 1955; Grosfeld et al., 2006; Osuchukwu & Rentea, 2023).

In preterm neonates, ileal atresia often presents a diagnostic challenge because its early clinical manifestations may mimic necrotizing enterocolitis (NEC), an inflammatory intestinal condition commonly affecting premature infants due to immaturity of the intestinal vasculature and immune system. Overlapping features such as abdominal distension, bilious gastric residuals, feeding intolerance, and non-specific radiographic findings in the early phase frequently result in ileal atresia being misdiagnosed as NEC, thereby increasing the risk of delayed diagnosis and definitive surgical management.

Delayed diagnosis of ileal atresia in preterm neonates carries serious clinical consequences, including an increased risk of sepsis, intestinal perforation, fluid and electrolyte imbalance, and higher mortality, as well as long-term morbidity such as short bowel syndrome and impaired growth. These risks are further amplified in low birth weight infants, in whom physiological reserves are limited and systemic inflammatory responses may progress rapidly

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(Spitz, 2006; Stoll et al., 2015; Oh, 2023). The reported incidence of small bowel atresia is approximately 1 in 1,000–5,000 live births, and it remains a major cause of neonatal intestinal obstruction with poorer outcomes observed in the preterm population.

Clinically, ileal atresia is typically characterized by failure to pass meconium, progressive abdominal distension, bilious vomiting, and greenish gastric residuals. Initial radiologic evaluation, such as an abdominal babygram, is commonly used as a screening modality; however, findings may be non-specific, particularly in preterm neonates with suspected necrotizing enterocolitis (NEC). Therefore, optimal management requires a multidisciplinary approach encompassing neonatal stabilization, gastric decompression, correction of metabolic disturbances, and timely surgical intervention. Staged management strategies, such as the Santulli procedure, have been reported to reduce the risk of complications in high-risk patients with systemic instability (Grosfeld et al., 2006; Osuchukwu & Rentea, 2023; Oh, 2023).

Although the association between ileal atresia, prematurity, and NEC is well recognized, case reports that specifically highlight diagnostic challenges and the crucial role of the neonatologist in perioperative management of NEC associated with ileal atresia remain limited. This gap in the literature is clinically significant, as active involvement of neonatologists in early clinical decision-making can influence diagnostic timing, patient stabilization, and postoperative outcomes.

Therefore, this case report aims not only to describe an instance of ileal atresia in a preterm neonate complicated by NEC but also to emphasize important clinical lessons regarding diagnostic vigilance, multidisciplinary coordination, and perioperative management implications to improve outcomes in this high-risk neonatal population.

2. METHOD

This study is a case report prepared in accordance with the CARE Guidelines, describing the clinical course, diagnostic process, and perioperative management of a preterm neonate with ileal atresia complicated by necrotizing enterocolitis (NEC). Data were obtained retrospectively from the patient's medical records. Ethical approval for the publication of this case was granted by the Health Research Ethics Committee of RSUD Prof. Dr. Margono Soekarjo, and written informed consent was obtained from the patient's parents.

Case Report

A male neonate was delivered spontaneously at RSUD Prof. Dr. Margono Soekarjo on June 29, 2025, at 7:45 PM, born to a 24-year-old primigravida mother (G1P0A0) at a gestational age of 32 weeks and 5 days. The delivery was classified as spontaneous preterm labor with premature contractions and an oblique fetal lie. The maternal history was notable for fetal distress, preeclampsia with differential diagnosis of nephrotic syndrome, gross hematuria suspected to be secondary to medical bleeding related to antiplatelet therapy (aspirin, resolved), morbid obesity, asymptomatic bacteriuria, and hyponatremia.

At birth, the neonate appeared premature with weak crying and hypotonia, prompting initial resuscitative measures. Following stabilization, spontaneous breathing was present with a heart rate >100 beats per minute, no chest retractions or grunting, a respiratory rate of 52 breaths per minute, oxygen saturation of 98%, but persistent cyanosis. Supplemental oxygen was administered via nasal cannula. The infant received intramuscular vitamin K and gentamicin ophthalmic ointment. Birth weight was 2,000 g, body length 45 cm, head circumference 29 cm, chest circumference 28 cm, abdominal circumference 28 cm, and mid-upper arm circumference 9 cm. APGAR scores were 7, 8, and 9 at 1, 5, and 10 minutes, respectively, with a Downes score of 2. The initial clinical impression was low birth weight (LBW), preterm infant, appropriate for gestational age, with mild respiratory distress.

The neonate was transported from the delivery room to the Melati Ward with nasal cannula oxygen support and placed in a pre-warmed incubator. Upon arrival at the High Care Unit (HCU) at 9:40 PM, the infant appeared lethargic with unstable vital signs, mild chest retractions, and cyanosis that improved with oxygen therapy. Given the indication of prematurity, continuous

positive airway pressure (CPAP) was initiated with FiO_2 30% and PEEP 5 cmH_2O, along with placement of an orogastric tube (OGT). Subsequent evaluation revealed a heart rate of 133 beats per minute, respiratory rate of 54 breaths per minute, body temperature of 35.2°C, and oxygen saturation of 100%. Physical examination showed mild retractions without grunting, good air entry with bilateral vesicular breath sounds, normal heart sounds, a non-distended abdomen with normal bowel sounds, and normal external genitalia and anus. The infant had not yet passed meconium or voided urine, exhibited hypersalivation, and the OGT drained cloudy gastric residuals. Management included CPAP (FiO_2 30%, PEEP 5 cmH_2O), intravenous 10% dextrose, intravenous ampicillin–sulbactam 100 mg every 12 hours, intravenous gentamicin 10 mg every 36 hours, and laboratory investigations including complete blood count, random blood glucose, C-reactive protein, blood culture, and babygram imaging.

On day 0 of life (June 29, 2025), the neonate had not passed meconium, continued to exhibit hypersalivation, and the OGT continued to drain cloudy gastric contents, indicating suboptimal gastrointestinal function. On day 1 of life, respiratory distress improved with CPAP adjusted to FiO_2 25% and PEEP 5 cmH_2O. The OGT continued to drain cloudy mucus, and the infant began to pass meconium. Abdominal babygram imaging did not demonstrate features of ileus or pneumoperitoneum (Figure 1). Chest radiography supported findings consistent with grade I hyaline membrane disease (Figure 2).

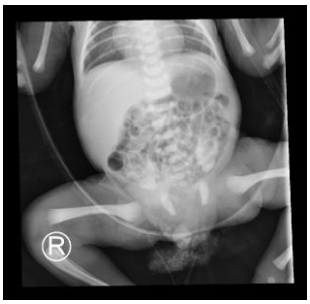

Figure 1. Babygram Abdomen (30/06/2025)

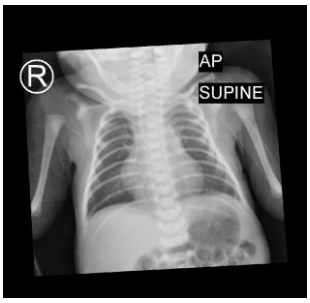

Figure 2. Babygram Thorax (30/06/2025)

On day 2 of life, respiratory distress further improved with CPAP adjusted to an FiO_2 of 21% and PEEP of 5 cmH_2O. The infant subsequently developed recurrent gastrointestinal symptoms, including progressive abdominal distension and yellow–green gastric residuals from the orogastric tube (OGT), accompanied by clinical jaundice. Abdominal circumference increased from 30 cm to 32 cm. Laboratory evaluation revealed unconjugated hyperbilirubinemia (total bilirubin 9.81 mg/dL; indirect bilirubin 9.10 mg/dL). Based on these findings, impaired gastrointestinal absorption was suspected. A repeat abdominal babygram and gastric decompression were planned to further evaluate abdominal distension. A rectal tube was inserted, after which the infant was transferred to the surgical neonatal intensive care unit (NICU). Parenteral nutrition was initiated, consisting of amino acids (AA2) 40 mL/24 hours and lipid emulsion (SL1) 10 mL/24 hours.

On day 3 of life, respiratory status remained stable with low-flow oxygen at FiO_2 21% (1 L/min). However, gastrointestinal dysfunction persisted, characterized by ongoing abdominal distension (abdominal circumference 32 cm), increased bowel peristalsis, and yellowish gastric

residuals. The clinical pattern began to suggest impaired gastrointestinal emptying. Based on the clinical findings, functional ileus was considered, with Hirschsprung disease included in the differential diagnosis, and a colon-in-loop study was planned.

On day 4 of life, the clinical condition deteriorated, marked by absence of bowel movements, persistent and significant abdominal distension (32–35 cm), and continued yellow gastric residuals. Respiratory status remained stable without distress on low-flow oxygen (FiO_2 21%, 1 L/min). The planned colon-in-loop study was subsequently canceled. Repeat abdominal babygram imaging demonstrated progression to stage III necrotizing enterocolitis (NEC) with evidence of pneumoperitoneum (Figure 3). Antibiotic therapy was reassessed and escalated to a combination of ceftazidime (60 mg every 12 hours), gentamicin (10 mg every 36 hours), and metronidazole (30 mg every 12 hours). The patient required urgent surgical intervention. Preoperative laboratory evaluation revealed signs of coagulopathy, including prolonged prothrombin time (PT) and activated partial thromboplastin time (aPTT), necessitating transfusion of fresh frozen plasma (FFP) at a dose of 40 mL.

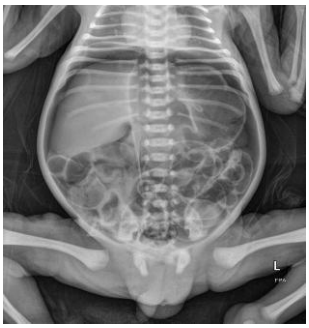

Figure 3. Babygram Abdomen (03/07/2025)

On day 5 of life, the infant underwent exploratory laparotomy with subsequent colostomy formation. Intraoperative exploration revealed ileal atresia with marked proximal bowel dilatation and distal microcolon. Resection of the atretic segment was performed, followed by end-to-side ileoileal anastomosis using the Santulli procedure. After thorough peritoneal irrigation, an abdominal drain and nasogastric tube (NGT) were placed, and the surgical wound was closed.

Postoperatively, abdominal distension persisted (abdominal circumference 31 cm), and bowel movements had not yet occurred. The patient was kept nil per os until the stoma became productive, with surgical dressing changes scheduled every three days. The postoperative condition was characterized by marked lethargy, absent crying, unstable respiratory rate, and mild chest retractions; cyanosis resolved with supplemental oxygen. The Downes score was 2. The patient was intubated with a 3.5-mm endotracheal tube positioned at 8.5 cm at the lips and placed on mechanical ventilation in IV A/CMV-PC mode (RR 30 breaths/min, FiO_2 40%, PIP 15 cmH_2O, PEEP 6 cmH_2O, inspiratory time 1.0 s, flow trigger 0.5). Postoperative laboratory evaluation revealed hyperglycemia (464 mg/dL) and hyponatremia (131 mmol/L). Supportive postoperative therapy included ranitidine 2 mg every 12 hours, analgesia with fentanyl infusion (1 mg, 0.5 mL/hour), calcium gluconate 2 mL every 12 hours, and correction of hyponatremia with 3% NaCl (20 mL at 1.6 mL/hour).

On day 6 of life (postoperative day [POD] 1), early improvement in gastrointestinal status was observed, with a reduction in abdominal distension (abdominal circumference 28 cm). The stoma appeared viable without signs of infection but was not yet productive. The abdominal drain yielded 5–7 mL of yellow-brown fluid, bowel sounds were increased, and the patient had not yet passed stool. No gastric residuals were detected, suggesting gradual recovery of gastric emptying. The infant remained clinically jaundiced (Kramer grade 3), with transcutaneous bilirubin of 15.7 mg/dL and total serum bilirubin of 13.4 mg/dL, prompting initiation of 36 hours of phototherapy. Respiratory distress was controlled with mechanical ventilation in IV A/CMV-PC mode (RR 40 breaths/min, FiO_2 30%, PIP 15 cmH_2O, PEEP 6 cmH_2O, inspiratory time 1.0 s, flow trigger 0.5).

Between days 7 and 9 of life, progressive improvement in gastrointestinal function was noted. On day 7 (POD 2), mild abdominal distension persisted (abdominal circumference 30 cm), with a non-productive stoma and 10 mL of drain output. On day 8 (POD 3), mild distension further decreased (abdominal circumference 29 cm), and the stoma began producing approximately 8 mL of yellow-brown stool, indicating restoration of intestinal continuity and successful Santulli procedure, although minimal purulent discharge was observed around the surgical incision. Blood cultures remained sterile, and repeat blood cultures along with wound discharge cultures were planned. Respiratory status remained stable, allowing initiation of ventilator weaning to IV SIMV-PC mode (RR 35 breaths/min, FiO_2 30%, PIP 15 cmH_2O, PEEP 6 cmH_2O, inspiratory time 0.4 s).

On day 9 of life (POD 4), mild abdominal distension persisted (abdominal circumference 29 cm), the stoma remained productive, and 5 mL of green gastric residual was noted. Endotracheal tube obstruction occurred, resulting in oxygen desaturation (SpO_2 80%), which was managed with suctioning followed by extubation and planned endotracheal tube culture. Respiratory support was transitioned to noninvasive positive pressure ventilation (NIPPV) in A/CMV-PC mode (RR 35 breaths/min, FiO_2 40%, PIP 15 cmH_2O, PEEP 6 cmH_2O, inspiratory time 0.4 s, flow trigger 0.5). The patient also received nebulized adrenaline every 6 hours and intravenous dexamethasone (0.2 mg/kg body weight) for associated bradycardia (heart rate 118–124 beats/min). Overall, the POD 2–4 period demonstrated gradual recovery of intestinal motility, evidenced by the onset of stoma output, although close monitoring of gastric residuals and abdominal distension remained necessary as indicators of gastrointestinal stabilization.

During postoperative days (POD) 5–10 (days of life 10–15), the infant maintained stable respiration on mechanical ventilation in NIPPV SIMV-PC mode (RR 35 breaths/min, FiO_2 40%, PIP 15 cmH_2O, PEEP 6 cmH_2O, inspiratory time 0.4 s), with no clinical signs of sepsis. Respiratory distress was well controlled, allowing gradual weaning to CPAP (FiO_2 30%, PEEP 5 cmH_2O), followed by low-flow oxygen at FiO_2 21% (1 L/min). Gastrointestinal function showed gradual improvement, with a stable abdominal circumference of 28–30 cm and absence of abdominal distension. Previously minimal greenish gastric residuals (approximately 1 mL) progressively resolved in parallel with improving intestinal motility, while stoma output remained active, with stool color transitioning from dark green to brown. Enteral feeding tolerance improved from trophic feeding at 10 mL/kg/day (10 × 2 mL) to 10 × 15 mL per day without vomiting, abdominal distension, or aspiration. The infant demonstrated a good sucking reflex, active movements, and a strong cry. On day 14 of life, the orogastric tube was removed, oral feeding was initiated, and breastfeeding training commenced. Culture results revealed a multibacterial nosocomial infection (*Klebsiella pneumoniae, Acinetobacter baumannii,* and *Escherichia coli*); therefore, antibiotic therapy was adjusted by replacing gentamicin with amikacin (21.5 mg once daily), while continuing metronidazole (15 mg every 12 hours) and ceftazidime (60 mg every 12 hours).

During POD 11–15 (days of life 16–20), the infant remained clinically stable, breathing spontaneously on room air. Gastrointestinal status remained stable, with an abdominal circumference of 29–30 cm and ongoing stoma output showing improved absorptive function, as evidenced by stool color changing from dark green to brown and subsequently yellowish. On day 16 of life, the infant demonstrated good tolerance to breastfeeding practice, and enteral volumes were gradually advanced to 10 × 30 mL per day without gastrointestinal complaints. Parenteral nutrition (AA2 and SL1) was discontinued. The most recent laboratory evaluation (July 17, 2025) showed hematological and inflammatory parameters within normal limits. Given stable spontaneous breathing, resolution of infection, and adequate nutritional tolerance, the patient was deemed suitable for transfer to the High Care Unit (HCU) for further monitoring.

On day 21 of life (POD 16), the patient was transferred to the HCU in stable condition, breathing room air without chest retractions or tachypnea. Abdominal circumference measured 30 cm without distension. Repeat babygram imaging demonstrated abdominal meteorism (Figure 4), neonatal pneumonia, and grade I–II hyaline membrane disease (Figure 5). Nutrition continued via the orogastric tube with good tolerance, as indicated by the absence of significant gastric residuals, vomiting, or aspiration. The infant exhibited adequate sucking reflex and coordinated swallowing, and intravenous access was converted to an IV plug.

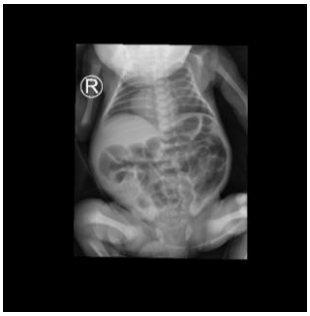

Figure 4. Babygram Abdomen (20/07/2025)

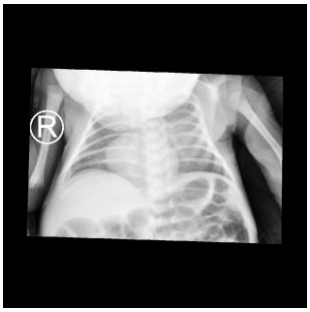

Figure 5. Babygram Thorax (20/07/2025)

On day 22 of life (POD 17), the patient remained clinically stable, with an increase in abdominal circumference to 32 cm, which was still within tolerable limits, and minimal clear gastric residuals. Antibiotic therapy was reassessed, leading to discontinuation of amikacin and metronidazole, while meropenem was continued at 60 mg every 8 hours as day 5 of therapy. Between days 23 and 25 of life (POD 18–20), the clinical condition remained stable, with no abdominal distension, minimal gastric residuals, and well-controlled respiration without the need for increased ventilatory support. Enteral nutrition via the orogastric tube was well tolerated, and oral feeding ability continued to improve with coordinated suck–swallow reflexes. Laboratory evaluations revealed no significant abnormalities; therefore, therapy was continued without modification.

On day 26 of life, the patient remained stable with an abdominal circumference of 32.5 cm, without signs of abdominal distension or gastrointestinal intolerance. Nutritional tolerance remained good, and the infant demonstrated optimal activity levels, prompting initiation of discharge planning. By day 27 of life, the patient showed marked clinical improvement and was able to breastfeed directly with effective suck–swallow coordination. Body weight increased from a birth weight of 2,000 g to a discharge weight of 2,135 g, indicating adequate nutritional intake. Physical examination demonstrated stable vital signs (temperature 36.5°C; heart rate 151 beats/min; respiratory rate 57 breaths/min; SpO_2 98% on room air), active movement, strong crying, unlabored respiration without retractions or grunting, vesicular breath sounds, and a non-distended abdomen with good bowel sounds and regular stoma output.

Based on respiratory and hemodynamic stability, adequate nutritional tolerance, and effective oral feeding, the patient was deemed fit for discharge at 27 days of life. All antibiotic therapies had been discontinued in accordance with clinical improvement. The parents received comprehensive education regarding stoma care, nutritional management, and recognition of warning signs. Definitive surgical management with stoma closure (Santulli takedown) was planned for October 2025.

The patient was subsequently followed up at the pediatric surgery outpatient clinic on October 14, 2025, for planned Santulli closure and was admitted to the Seruni ward without significant complaints, with regular bowel movements. Physical examination revealed a body weight of 6 kg, body length of 52 cm, and stable vital signs (heart rate 130 beats/min, respiratory rate 26 breaths/min, temperature 36.5°C, SpO_2 98% on room air). The stoma showed minimal output, with adequate per-anal defecation. Laboratory investigations demonstrated hemoglobin

10.8 g/dL, leukocyte count 9,600/µL, platelet count 585,000/µL, normal hemostatic function, sodium 151 mmol/L, and potassium 5.18 mmol/L. The patient received cefotaxime 300 mg every 12 hours and metronidazole 20 mg every 8 hours as preoperative therapy. Repeat preoperative laboratory evaluation showed normalization of serum sodium to 137 mmol/L. Preoperative chest radiography demonstrated a normal cardiac silhouette and clear lung fields (Figure 6).

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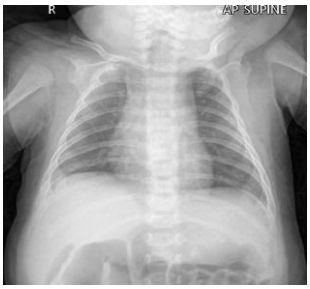

Figure 6. Babygram Thorax (14/10/2025)

On October 15, 2025, the patient underwent Santulli stoma closure without intraoperative complications. In the immediate postoperative period (POD 0), the patient was crying appropriately and received intravenous fluid therapy with normal saline, along with antibiotic therapy consisting of cefotaxime (400 mg every 12 hours) and metronidazole (20 mg every 8 hours), as well as analgesia with paracetamol (60 mg every 8 hours).

On October 16, 2025 (POD 1), the patient appeared comfortable with minimal pain, demonstrated good tolerance to breastfeeding, and had normal urination and bowel movements. Vital signs were stable (heart rate 106 beats/min, respiratory rate 21 breaths/min, temperature 36.6°C, and oxygen saturation 99% on room air). The surgical wound was covered with a dry dressing and showed no signs of infection. The patient was deemed fit for discharge and was prescribed oral cefadroxil (2 × ½ teaspoon daily) and oral paracetamol (3 × ½ teaspoon daily) upon discharge.

3. RESULT AND DISCUSSION

The ileum originates from the midgut, which develops from the endoderm, while the mesoderm gives rise to the muscular layers and mesentery, and the ectoderm—via migration of neural crest cells—forms the enteric nervous system. The midgut receives its primary vascular supply from the superior mesenteric artery (Wilson & Bordoni, 2025). In contrast to duodenal atresia, which results from failure of recanalization, ileal and jejunoileal atresia are primarily caused by intrauterine vascular disruption. This process leads to intestinal ischemia, segmental necrosis, and subsequent formation of luminal discontinuity. This vascular theory is supported by experimental studies demonstrating that mesenteric vessel occlusion can produce atretic lesions resembling those observed in human clinical conditions (Choi et al., 2022; Louw & Barnard, 1955; Grosfeld et al., 2006; Osuchukwu & Rentea, 2025; Oh et al., 2023).

In jejunoileal atresia, associated congenital anomalies are relatively uncommon (<10%) because the insult occurs during the later stages of fetal development and is typically localized and vascular in nature. Compared with duodenal atresia, jejunoileal atresia is less frequently associated with chromosomal abnormalities such as trisomy 21. The most commonly reported associated conditions include cystic fibrosis, intestinal malrotation, and gastroschisis (approximately 10%). A survey conducted by the Korean Association of Pediatric Surgeons (KAPS) in 2010 reported associated anomalies including congenital heart disease (8.6%), other gastrointestinal anomalies (7.5%), and genitourinary anomalies (4.8%) (Schmedding et al., 2021; Oh, 2023).

The most widely used classification system for jejunoileal atresia is the Grosfeld classification, which categorizes the condition into types I–IV based on intestinal continuity, mesenteric defects, and the length of remaining bowel. This classification has important

prognostic and therapeutic implications, as it is directly associated with the risk of short bowel syndrome and postoperative outcomes (Spitz, 2006; Grosfeld et al., 2006; Oh et al., 2023).

Table 1. Clasification of Atresia Jejunoileal

Type	Description
I	An intraluminal membrane with intact serosal continuity and no mesenteric defect
II	Serosal discontinuity a fibrous cord connecting the proximal and distal bowel segments
IIIa	Serosal discontinuity associated with a mesenteric defect
IIIb	Serosal discontinuity with a large mesenteric defect, producing an apple-peel
IV	Atresia multipel

(Grosfeld et al., 2006)

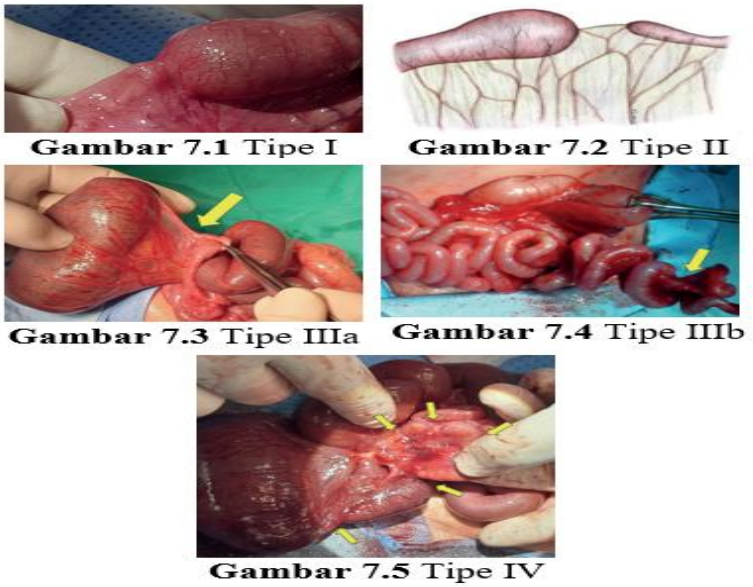

Figure 7. Atresia Type
(Oh et al., 2023; Shalkow, 2023)

The choice of surgical management depends on the type of atresia. Type I–II atresia is generally managed with limited bowel resection followed by primary anastomosis. In contrast, type III–IV atresia often requires staged management strategies, such as enterostomy or the Santulli procedure, particularly in preterm or hemodynamically unstable neonates. These approaches aim to preserve bowel length and reduce postoperative complications (Grosfeld et al., 2006; Osuchukwu & Rentea, 2025; Oh et al., 2023).

Risk factors for ileal atresia include both environmental and genetic influences that contribute through intrauterine vascular disruption. Maternal exposure to vasoactive substances during pregnancy, such as cigarette smoke, cocaine, and pseudoephedrine, may induce vasoconstriction and fetal mesenteric ischemia. In addition, maternal infections and alcohol consumption have been associated with an increased risk of structural intestinal abnormalities. From a genetic perspective, although most cases are sporadic, multiple intestinal atresias may be inherited in an autosomal recessive pattern, particularly in association with mutations in the TTC7A gene (Fair et al., 2022; Huang et al., 2025; Oh, 2023).

Prematurity is a major risk factor for the development of necrotizing enterocolitis (NEC), primarily due to immaturity of the intestinal mucosal barrier, underdeveloped regulation of mesenteric perfusion, and an exaggerated inflammatory response to early bacterial colonization. These factors render the premature neonatal intestine highly susceptible to segmental hypoxia–ischemia, a pathophysiological mechanism that closely parallels the intrauterine vascular accident theory underlying jejunoileal and ileal atresia (Louw & Barnard, 1955; Neu & Walker, 2011; Oh, 2023).

Necrotizing enterocolitis (NEC) may represent an initial manifestation of, or a condition that obscures, underlying ileal atresia, particularly in preterm neonates. Congenital intestinal obstruction leads to stasis of intestinal contents, progressive bowel distension, and bacterial translocation, which subsequently accelerates the development of severe NEC and may progress to intestinal perforation. Therefore, early-onset NEC accompanied by pneumoperitoneum should raise strong suspicion for an underlying congenital obstructive anomaly (Spitz, 2006; Grosfeld et al., 2006; Oh, 2023).

This correlation was evident in the present case, in which a preterm infant born at a gestational age of 32 weeks and 5 days initially showed respiratory improvement but subsequently developed progressive abdominal distension, bilious gastric residuals, and impaired gastrointestinal emptying. Clinical deterioration on day 4 of life, with findings consistent with stage III NEC and pneumoperitoneum, prompted surgical exploration, which revealed ileal atresia as the underlying etiology. These findings support the notion that NEC in this patient was likely secondary to congenital obstruction and intestinal ischemia rather than primary NEC, as has been described in recent literature (Louw & Barnard, 1955; Stoll et al., 2015; Osuchukwu & Rentea, 2025).

Establishing a diagnosis of ileal atresia in neonates is often challenging due to non-specific early clinical manifestations and radiologic findings. Prenatal ultrasonography may demonstrate small bowel dilatation and polyhydramnios. Postnatal radiologic evaluation typically reveals dilated bowel loops and air–fluid levels with absence of distal gas; in ileal atresia, proximal bowel distension is often more pronounced and may mimic conditions such as meconium ileus or total colonic aganglionosis. As obstruction progresses, increased intraluminal pressure and impaired perfusion may lead to ischemia and perforation, resulting in radiologic findings such as peritoneal calcifications or pneumoperitoneum secondary to meconium peritonitis (Durán et al., 2022; Choi et al., 2022; Oh, 2023).

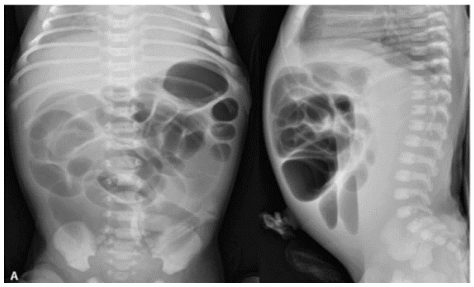

Figure 8. Small Bowel Dilatation in Ileal Atresia with Absence of Rectal Air Distribution (Choi et al., 2022)

Clinical deterioration in this case, manifested by pneumoperitoneum, stage III necrotizing enterocolitis (NEC), or signs of peritonitis, constitutes an absolute indication for emergency laparotomy, as these findings signify intestinal perforation and failure of conservative management. Surgical exploration often serves as the definitive diagnostic step, revealing ileal atresia as the underlying etiology (Oh, 2023; Osuchukwu & Rentea, 2025).

Initial management of ileal atresia emphasizes rapid stabilization through gastric decompression, fluid and electrolyte resuscitation, and administration of broad-spectrum antibiotics when NEC, perforation, or sepsis is suspected. This approach aims to reduce the risk of aspiration, improve mesenteric perfusion, and control systemic infection prior to surgical intervention. Definitive surgery should be performed promptly once stabilization is achieved, as delays are associated with an increased risk of intestinal necrosis, sepsis, and mortality (Oh, 2023; Osuchukwu & Rentea, 2025).

The most commonly employed surgical technique involves resection of the dilated proximal bowel segment and the atretic segment, followed by primary end-to-end anastomosis, with or without proximal tapering enteroplasty (Osuchukwu & Rentea, 2023). In emergency settings with clinical deterioration, such as sepsis or a hypercatabolic state, enterostomy using the Santulli procedure is a preferred operative option (Rachman et al., 2024). This approach allows

effective decompression of the proximal bowel while maintaining enteral flow to the distal intestine, thereby reducing the risk of anastomotic leakage compared with primary end-to-end anastomosis. Such staged management facilitates sepsis control, preservation of bowel length, and reduction in the risk of short bowel syndrome (Schattenkerk et al., 2022).

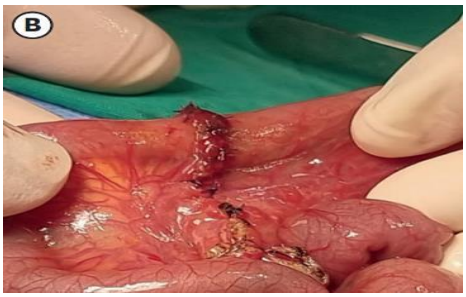

Figure 9. End-to-end anastomosis (Oh, 2023)

Postoperative management of ileal atresia focuses on respiratory and hemodynamic stabilization, infection control, and gradual nutritional support. In the early postoperative phase, patients typically require ventilatory support according to clinical needs. Broad-spectrum antibiotics are administered to prevent or treat sepsis, while total parenteral nutrition (TPN) serves as the primary source of nutrition. Transition to enteral nutrition is performed gradually, as enteral feeding plays a critical role in intestinal mucosal maturation and gastrointestinal adaptation and reduces the risk of parenteral nutrition–associated liver disease (PNALD). A delayed enteral feeding strategy (>48 hours postoperatively) remains the safest and most commonly applied approach in neonates following intestinal atresia surgery (Jarkman & Salö, 2019; Lu et al., 2023; Oh, 2023).

The success of postoperative management is assessed through clinical parameters, including a productive stoma, adequate stool output, stable abdominal circumference without progressive distension, good tolerance of enteral nutrition until full enteral feeding is achieved, and appropriate weight gain and linear growth. Studies have shown that the average time to achieve full enteral feeding in neonates with jejunoileal atresia is approximately 2–3 weeks postoperatively. This timeline is influenced by the type of atresia, the length of the remaining bowel, and comorbid conditions such as prematurity and sepsis (Jarkman & Salö, 2019; Peterson & Burjonrappa, 2021).

In the present case, all of these success parameters were achieved gradually, as evidenced by productive stoma output, stable abdominal circumference without distension, and good tolerance of enteral nutrition culminating in full oral feeding. The patient remained clinically stable without signs of sepsis, maintained hemodynamic stability, and demonstrated adequate weight gain. Based on comprehensive clinical evaluation, the patient was deemed fit for discharge at 27 days of life (POD 21), with a plan for elective Santulli stoma closure.

Santulli stoma closure may be considered once the patient's general condition is stable. In this case, stoma closure was performed two months after the initial surgery, at a time when clinical stability, nutritional status, and growth parameters had reached optimal targets. These favorable short-term outcomes are consistent with existing literature indicating that staged management approaches in premature infants with ileal atresia provide safer and more controlled clinical results, particularly in reducing the risk of complications and short bowel syndrome (Peterson & Burjonrappa, 2021; Oh, 2023; Rachman et al., 2024)

4. CONCLUSION

This case report demonstrates that ileal atresia in preterm neonates may follow a complex and progressive clinical course and can initially masquerade as necrotizing enterocolitis (NEC). Such a presentation necessitates a high index of clinical suspicion and comprehensive perioperative management to prevent further deterioration. Early recognition of clinical worsening, optimization of preoperative stabilization, and timely decision-making regarding the

need for surgical exploration are critical in preventing progression to sepsis and life-threatening complications. In this patient, postoperative management—particularly ventilatory support, antibiotic therapy guided by clinical and microbiological evaluation, and a stepwise nutritional strategy—resulted in favorable clinical outcomes. These outcomes were evidenced by stabilization of the general condition, gradual recovery of gastrointestinal function, adequate nutritional tolerance, and appropriate growth. A multidisciplinary approach with close coordination among pediatricians, pediatric surgeons, and neonatal intensive care teams played a pivotal role in achieving safe and stable clinical outcomes in this case.

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