

Case Report

Delayed presentation of pure duodenal atresia in a preterm infant: a rare case report

Edamakanti Swetha Soni^{1*}, Aditya Pratap Singh², Mayur Shyam Soni³,
Shital Narendra Bhaisare¹

¹Department of Pediatric Surgery, AIIMS Nagpur, Maharashtra, India

²Bhagwan Mahavir Hospital Sumerpur Pali, Maharashtra, India

³KIIMS-Kingsway Hospital, Nagpur, Maharashtra, India

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*Correspondence:

Dr. Swetha Edamakanti Soni,

E-mail: Swethareddysoni22@gmail.com

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ABSTRACT

Duodenal atresia is a rare congenital anomaly that usually presents in the early neonatal period with non-bilious vomiting. We report a preterm male neonate born at 33 weeks of gestation who presented at 17 days of life with persistent non-bilious vomiting since day two. Imaging showed the classical 'double bubble' sign, and contrast study confirmed complete duodenal obstruction. Exploratory laparotomy revealed type III duodenal atresia, for which a duodeno-duodenostomy was performed. The postoperative recovery was uneventful, and the infant was thriving at one-month follow-up. This report emphasizes that even pure type III duodenal atresia can rarely present late, highlighting the importance of high suspicion in preterm neonates with persistent vomiting.

Keywords: Pure duodenal atresia, Preterm infant, Delayed presentation

INTRODUCTION

Duodenal atresia is a rare congenital obstruction of the duodenum, occurring in approximately 1 in 6,000 to 10,000 live births.¹ It results from failure of recanalization of the duodenum during embryonic development. Infants typically present with early-onset, non-bilious vomiting and may demonstrate the classical "double bubble" sign on radiographs. Associations with prematurity and congenital anomalies, particularly Down syndrome and annular pancreas, have been described.^{2,3} Early surgical intervention, usually via duodeno-duodenostomy, is the standard of care and is associated with excellent prognosis.⁴

CASE REPORT

A 17-day-old preterm male neonate, born at 33 weeks of gestation by vaginal delivery with a birth weight of 2 kg,

was admitted with complaints of repeated non-bilious vomiting since the second day of life. The infant had passed a small amount of meconium within 24 hours of birth and was discharged on breastfeeding. Despite being on oral antiemetics, vomiting persisted. At the time of presentation, the infant weighed 1.6 kg and appeared dehydrated.

An abdominal ultrasound performed outside was reported as normal. Repeat ultrasound at our center showed a dilated stomach and first part of the duodenum with suspicion of annular pancreas. Erect abdominal radiograph revealed the classical double bubble sign (Figure 1).

The contrast study confirmed complete obstruction, as the dye failed to pass beyond the first part of the duodenum (Figure 2) even after 24 hours. The baby had no associated anomalies or syndromic features.



Figure 1: Erect abdomen showing the double bubble sign.



Figure 2: Contrast study revealing complete obstruction at the 2nd part of the duodenum.

The infant was stabilized with intravenous fluids, and exploratory laparotomy was performed. Intraoperatively, a type III duodenal atresia (Figure 3) was identified.

A duodeno-duodenostomy (Figure 4) was performed.

The postoperative course was uneventful; RT feeds started on day 2, gradually transitioned to breastfeeding, and were tolerated well. The infant passed stools normally and was discharged on the 10th postoperative day. At one-month follow-up, the baby was thriving well and symptom-free.

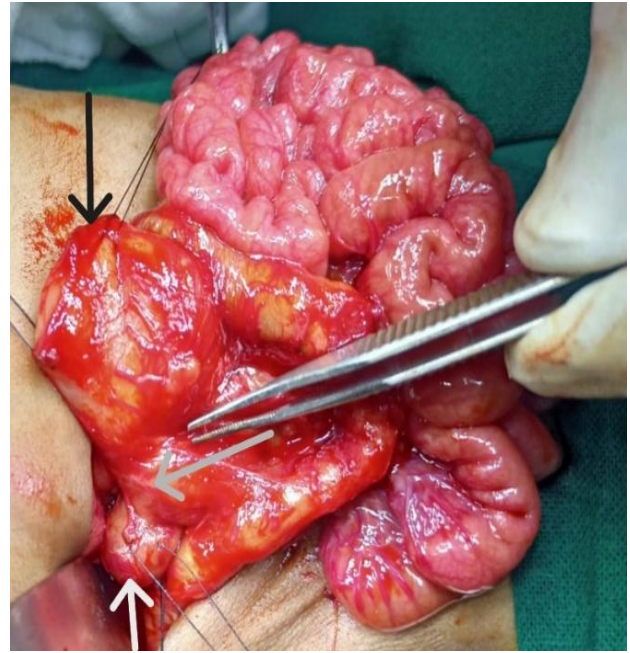


Figure 3: Intra-operative picture revealing pure duodenal atresia type 3 black arrow-proximal dilated duodenum with blind end. Grey arrow-pancreas. White arrow-distal duodenum.



Figure 4: Duodenoduodenostomy.

DISCUSSION

Duodenal atresia is a congenital anomaly resulting from failure of recanalization of the duodenal lumen between the 8th and 10th week of gestation, leading to complete luminal obstruction.¹ It represents the most common site of congenital intestinal obstruction, with an incidence of approximately 1 in 6,000-10,000 live births. Nearly 50% of cases are associated with other anomalies, particularly trisomy 21, congenital heart disease, and annular pancreas.^{1,5}

Clinically, duodenal atresia usually presents within the first 24-48 hours of life with recurrent, non-bilious

vomiting when the obstruction lies proximal to the ampulla of Vater. Antenatally, polyhydramnios and the “double bubble” sign on ultrasound may raise suspicion. Postnatally, plain abdominal radiographs showing the classical double bubble appearance are diagnostic, while contrast studies confirm the site and severity of obstruction.^{2,5} Choudhry et al reported that over half of patients with duodenal atresia have associated anomalies—most commonly Trisomy 21 and congenital heart disease—and that prenatal diagnosis aids early management without negatively influencing outcomes. In our patient, although vomiting started on the second day of life, the diagnosis was delayed until day 17.⁶ This relatively late recognition can be attributed to the baby’s preterm status, subtle initial symptoms, and absence of associated anomalies such as Down syndrome, which often prompt earlier and more aggressive evaluation.

Most cases of delayed or atypical presentation described in the literature are associated with fenestrated duodenal webs or diaphragms rather than complete atresia. Mousavi et al reported a series of patients with delayed presentation, with ages at operation ranging from 1 to 72 months, all of which were related to duodenal membranes.² Tiwari et al reported a series of infants with delayed presentation of duodenal atresia caused by fenestrated duodenal webs, emphasizing that partial intrinsic obstruction can mimic complete atresia and contribute to diagnostic delay.³ Tecos et al documented an extreme case of an 11-year-old girl with longstanding symptoms who was ultimately diagnosed with duodenal obstruction caused by a membrane.⁴ Sega et al reported a case of delayed presentation of duodenal atresia in a neonate with Trisomy 21, emphasizing that even complete atresia with a hole may present later than expected when early symptoms are mild or intermittent.⁷ Eltayeb and Ibrahim noted that incomplete duodenal obstructions, such as fenestrated webs, can present well beyond the neonatal period because of partial luminal patency.⁸ Al Ghannam reported that duodenal webs with partial patency may present late and mimic functional gastrointestinal disorders, underscoring importance of considering structural causes in infants with persistent non-bilious vomiting.⁹ These reports highlight that partial obstruction can mask symptoms and significantly delay diagnosis. By contrast our case represents a rare instance of pure type III duodenal atresia with diagnosis established relatively late in neonatal period.

Surgical intervention remains the mainstay of treatment. Duodeno-duodenostomy is the gold standard for intrinsic duodenal obstruction and has been shown to provide excellent long-term outcomes.^{1,5} In our case, duodeno-duodenostomy resulted in early initiation of feeds, satisfactory weight gain, and uneventful recovery. Prognosis is generally favourable, particularly in infants without major associated anomalies.^{1,5} Patrycja and Przemysław emphasized that early diagnosis and timely surgical correction of duodenal atresia are crucial for achieving favorable long-term growth and

neurodevelopmental outcomes, particularly in the absence of major associated anomalies.¹⁰

This case emphasizes the importance of maintaining a high index of suspicion for duodenal obstruction in preterm neonates with persistent vomiting, even in the absence of syndromic features or congenital anomalies. Furthermore, while delayed presentations are well documented in the context of webs and membranes, this report underscores that even pure duodenal atresia can occasionally present later than expected, making clinical vigilance essential.

CONCLUSION

Delayed presentation of pure Type III duodenal atresia is extremely uncommon. This case underscores the importance of maintaining a high index of suspicion for duodenal obstruction in preterm neonates with persistent non-bilious vomiting, even in the absence of syndromic features. Prompt imaging and surgical correction can result in favourable outcomes.

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