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DIAGNOSTIC PROCESS IN A LATE DIAGNOSIS OF LADD'S SYNDROME IN A 4-YEAR-OLD GIRL WITH EDWARDS SYNDROME: A CASE REPORT

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ABSTRACT

Congenital anomalies affect 2–5% of newborns, with gastrointestinal malformations accounting for 5–6% of cases. Intestinal malrotation is a developmental disorder caused by incomplete rotation and fixation of the midgut between the 5th and 10th week of gestation. It is usually diagnosed in the neonatal period, often prenatally or within the first year of life, with more than 90% of cases identified before the age of 1. Late presentation in older children is therefore uncommon and diagnostically challenging.

This case report describes an unusual late diagnosis of malrotation with Ladd's bands in a 4-year-old girl. The aim is to emphasize that congenital gastrointestinal anomalies should remain part of the differential diagnosis of vomiting in children of any age.

We report the case of a 4-year-old girl with Edwards syndrome presented with five days of coffee ground vomiting after each attempt to drink. The patient has not passed stool for one week.

Ultrasound was inconclusive, and X-ray suggested right-sided diaphragmatic hernia with obstruction. A laparotomy was performed to resolve the intestinal obstruction and repair the diaphragmatic hernia. During the intraoperative bowel inspection, intestinal malrotation and associated Ladd's bands, causing duodenal obstruction, were identified. The Ladd's bands were released, and Ladd's syndrome was diagnosed.

This case highlights the rare late presentation of Ladd's syndrome in a 4-year-old child. It underlines the need to consider intestinal malrotation in the differential diagnosis of acute vomiting and bowel obstruction at any pediatric age, even when symptoms are nonspecific.

KEYWORDS

Intestinal Malrotation; Ladd's Syndrome; Edwards Syndrome; Pediatric Intestinal Obstruction, Congenital Gastrointestinal Anomalies; Vomiting; Delayed Diagnosis

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Introduction

Congenital anomalies are defined as structural, functional, biochemical, or molecular abnormalities that arise during intrauterine life and may be detected prenatally, at birth, or later in childhood. They remain an important cause of neonatal and infant morbidity and mortality worldwide and affect approximately 2–5% of all live-born infants. Their etiology is multifactorial and may include chromosomal abnormalities, single-gene disorders, environmental exposure, maternal illness, infections during pregnancy, and unknown developmental disturbances. Many congenital abnormalities involve more than one organ system and frequently coexist as part of recognized genetic syndromes [1].

Gastrointestinal congenital anomalies account for approximately 5–6% of all congenital defects and may affect any part of the digestive tract, from the esophagus to the anorectal region [3]. These anomalies include esophageal atresia, duodenal atresia, intestinal malrotation, omphalocele, diaphragmatic defects, Hirschsprung disease, and anorectal malformations. Early diagnosis is essential because delayed recognition may lead to bowel ischemia, perforation, sepsis, nutritional compromise, and significant long-term morbidity.

One of the important congenital abnormalities of the gastrointestinal tract is intestinal malrotation. Intestinal malrotation is a developmental disorder resulting from incomplete or abnormal rotation and fixation of the primitive midgut during embryogenesis[3,4,10]. Between the 5th and 10th week of gestation, the developing midgut normally herniates into the umbilical cord and undergoes a 270-degree counterclockwise rotation around the superior mesenteric artery (SMA). After returning to the abdominal cavity, the bowel becomes fixed in its normal anatomical position, with a broad mesenteric base that protects against volvulus.

Failure of this normal developmental process results in abnormal bowel positioning, incomplete fixation, and a narrow mesenteric attachment to the posterior abdominal wall. This narrow mesenteric pedicle significantly increases the risk of midgut volvulus, which may rapidly lead to bowel ischemia and life-threatening necrosis. In addition, abnormal peritoneal fibrous attachments known as Ladd's bands may form[10,15]. These bands extend from the abnormally positioned cecum across the duodenum to the retroperitoneum and may cause extrinsic compression of the duodenum, resulting in partial or complete obstruction.

The clinical presentation of intestinal malrotation depends on the age of the patient and the degree of obstruction [7]. In neonates, the most common symptom is bilious vomiting caused by acute duodenal obstruction or volvulus [9]. This presentation is often dramatic and leads to early diagnosis. However, in older children, symptoms may be much less specific and may include recurrent vomiting, feeding intolerance, abdominal pain, chronic constipation, poor weight gain, intermittent abdominal distension, gastroesophageal reflux–like symptoms, or failure to thrive[4,5,10]. Because of this nonspecific presentation, diagnosis may be delayed for months or even years.

Although more than 75–90% of patients are diagnosed during infancy, especially within the first year of life, delayed diagnosis in older children is well described in the literature [5,6,10]. Therefore, late presentation should not be considered exceptionally rare but rather an important diagnostic challenge. Many cases are discovered incidentally during surgery performed for another reason or during imaging performed for unrelated symptoms.

Prenatal diagnosis may occasionally be suspected on fetal ultrasonography, particularly when associated anomalies such as diaphragmatic hernia, omphalocele, bowel dilatation, polyhydramnios, or abnormal bowel position are present [7]. However, isolated malrotation is often difficult to identify before birth because specific ultrasonographic findings may be absent.

Patients with chromosomal disorders frequently present with multiple congenital anomalies involving the gastrointestinal tract. Edwards syndrome, also known as trisomy 18, is one of the most severe chromosomal disorders associated with multisystem developmental abnormalities. It occurs due to the presence of an extra copy of chromosome 18 and is characterized by severe developmental delay, cardiac defects, craniofacial abnormalities, limb deformities, feeding difficulties, respiratory problems, and gastrointestinal malformations [15].

Common gastrointestinal abnormalities associated with trisomy 18 include omphalocele, esophageal atresia, diaphragmatic defects, intestinal fixation disorders, and intestinal malrotation [15]. These patients often require complex medical and surgical management because several anomalies may coexist and mask one another clinically. In addition, decisions regarding surgical treatment may be influenced by the child's general condition, prognosis, associated cardiac disease, and quality-of-life considerations.

We present a case of a 4-year-old girl with genetically confirmed Edwards syndrome in whom intestinal malrotation with obstructing Ladd's bands and associated bilateral diaphragmatic defects was diagnosed during urgent surgery performed for intestinal obstruction. The educational value of this case lies not only in the delayed diagnosis beyond infancy but also in the diagnostic complexity caused by overlapping congenital abnormalities and the importance of maintaining suspicion for malrotation in older children with persistent vomiting.

Methodology

A 4-year-old girl with genetically confirmed Edwards syndrome (trisomy 18) was admitted to the emergency department because of persistent coffee-ground vomiting for five days. Vomiting occurred after every attempt at oral feeding and progressively worsened, resulting in poor tolerance of both liquids and solid food. One episode of fever reaching 38.5°C was reported during the night before admission. According to the caregiver, the patient had not passed stool for seven days before presentation, and progressive abdominal distension had become increasingly visible.

The child was under home hospice care because of severe psychomotor developmental delay and multiple congenital abnormalities associated with trisomy 18. Her past medical history included feeding difficulties since infancy and general poor nutritional status related to her underlying syndrome. She was not receiving regular medications at the time of admission and had no known drug allergies. There was no relevant family history of congenital gastrointestinal disorders, and no significant information regarding parental medical conditions was available.

On admission, the patient appeared clinically weakened and dehydrated. Physical examination revealed visible abdominal distension, especially in the upper abdomen, with signs suggestive of high intestinal obstruction. The abdomen was tense but without clear signs of peritoneal irritation. Repeated vomiting and prolonged absence of stool passage raised concern for significant gastrointestinal obstruction requiring urgent investigation.

Initial management focused on stabilization of the patient's general condition. Oral feeding was immediately discontinued. Intravenous fluid therapy was started to correct dehydration and electrolyte imbalance. Gastric decompression was initiated, and the patient underwent laboratory evaluation including complete blood count, inflammatory markers, electrolyte assessment, and basic biochemical analysis. Preoperative optimization was particularly important because of the patient's poor baseline condition and underlying syndromic disease.

Abdominal ultrasonography was performed as the first imaging study. The examination was significantly limited because of excessive bowel gas, which reduced visualization of deeper abdominal structures. Nevertheless, the ultrasound demonstrated a single markedly dilated bowel loop located in the right mid-abdomen measuring approximately 3.5 cm in diameter. The bowel wall thickness was preserved, and visible peristalsis was still present, suggesting mechanical rather than paralytic obstruction. A trace amount of free fluid was noted in the right lower abdomen.

Additionally, the urinary bladder was displaced to the right side and showed a relatively large volume of retained urine, approximately 55 mL before voiding and 40 mL after voiding. The bladder wall was smooth, but a significant amount of urine sediment was present. Although these findings were not considered the primary cause of symptoms, they reflected the altered intra-abdominal anatomy and the degree of abdominal distension.

Because the ultrasound findings were inconclusive and the clinical suspicion of bowel obstruction remained high, an upright abdominal and chest X-ray was performed. The radiograph revealed multiple dilated

bowel loops measuring up to 40 mm in diameter with several air–fluid levels visible in the upper abdomen, mid-abdomen, and notably within the right hemithorax. Only a small amount of gas was visible in the pelvic region. These findings initially raised suspicion of intestinal obstruction associated with a right-sided diaphragmatic hernia.

The radiological differential diagnosis included congenital diaphragmatic hernia, diaphragmatic eventration, bowel obstruction secondary to malrotation, and possible bowel incarceration. Because of persistent vomiting, prolonged constipation, abdominal distension, and worsening clinical status, urgent surgical intervention was considered necessary.

A contrast study of the upper gastrointestinal tract and computed tomography were not performed. Although such investigations may provide valuable preoperative information regarding the position of the duodenojejunal junction and the relationship between the superior mesenteric artery and vein (SMA/SMV), they were omitted because of the patient's deteriorating clinical condition, repeated vomiting, poor tolerance of oral intake, and the need for prompt operative management. Delaying surgery for additional imaging was considered unsafe.

After surgical qualification, exploratory laparotomy was performed through a transverse upper abdominal incision.

Results

Upon opening the abdominal cavity, a right-sided diaphragmatic defect was identified with herniation of bowel loops and greater omentum into the thoracic cavity. The herniated structures were carefully reduced back into the abdominal cavity without evidence of bowel ischemia or perforation.

Further intraoperative inspection unexpectedly revealed an additional defect involving the left hemidiaphragm. This left-sided defect was smaller but also required repair. Both diaphragmatic defects were repaired during the same procedure. These findings clarified the discrepancy between preoperative radiological suspicion of a primarily right-sided diaphragmatic hernia and the operative discovery of bilateral diaphragmatic abnormalities.

The diaphragmatic defects were interpreted as congenital defects rather than simple eventration. Their coexistence with trisomy 18 was consistent with the known association between Edwards syndrome and thoracoabdominal developmental abnormalities.

Further exploration of the abdominal cavity revealed abnormal bowel fixation consistent with intestinal malrotation. The duodenojejunal junction was abnormally positioned and failed to reach its normal location to the left of the spine at the level of the ligament of Treitz. The bowel mesentery had a narrow base, increasing the potential risk of volvulus.

Most importantly, obstructing Ladd's bands were identified crossing the duodenum and causing significant extrinsic compression of the proximal small bowel. This resulted in high intestinal obstruction and explained the patient's prolonged vomiting and inability to tolerate oral intake. No midgut volvulus was observed intraoperatively, and bowel perfusion remained preserved.

Division of the Ladd's bands was performed carefully, relieving the duodenal compression and restoring bowel passage. The release of obstruction confirmed the diagnosis of Ladd's syndrome. The bowel was repositioned appropriately to reduce the future risk of volvulus. No intraoperative complications occurred, and bowel viability remained satisfactory throughout the procedure.

The postoperative course was uncomplicated. The patient remained under close observation with continued intravenous hydration, gradual decompression withdrawal, and careful nutritional monitoring. Enteral feeding was reintroduced progressively after restoration of bowel function. Vomiting resolved completely, abdominal distension decreased significantly, and normal bowel movements returned.

The patient tolerated oral feeding without recurrence of symptoms. She survived the procedure and was discharged in stable general condition. Short-term surgical follow-up confirmed sustained clinical improvement, absence of recurrent vomiting, and satisfactory gastrointestinal function.

Discussion

This case illustrates the diagnostic complexity of intestinal malrotation presenting beyond infancy in a child with multiple congenital anomalies. Although intestinal malrotation is classically associated with neonatal bilious vomiting and acute presentation, delayed diagnosis in older children is well documented and should be recognized as an important clinical problem rather than an exceptional rarity [5,6,10].

In neonates, malrotation is often diagnosed rapidly because bilious vomiting strongly suggests proximal bowel obstruction and prompts urgent imaging [9]. In contrast, older children frequently present with chronic, intermittent, or nonspecific symptoms that mimic many other gastrointestinal disorders [2]. Recurrent vomiting, feeding intolerance, constipation, abdominal pain, poor appetite, and failure to thrive may be attributed to reflux disease, functional constipation, infection, or nutritional problems, especially in children with complex neurological or genetic conditions [3,4,6,9].

Our patient presented with coffee-ground vomiting rather than classic bilious emesis. This atypical presentation, combined with severe constipation and abdominal distension, suggested upper gastrointestinal obstruction but did not immediately indicate malrotation as the primary diagnosis. Furthermore, the coexistence of diaphragmatic defects complicated the radiological interpretation and shifted the initial suspicion toward diaphragmatic hernia [14].

The upright chest and abdominal radiograph suggested a right-sided diaphragmatic hernia because bowel loops with air–fluid levels were visualized within the right hemithorax. However, surgery demonstrated bilateral diaphragmatic defects in addition to intestinal malrotation. This finding highlights the limitations of plain radiography in complex congenital anatomy and emphasizes that final diagnosis may only become evident intraoperatively [13,14].

The primary mechanism of obstruction in our patient was not bowel incarceration within the diaphragmatic defect alone, but extrinsic duodenal compression caused by Ladd's bands. This distinction is clinically important because the treatment must address not only reduction of herniated bowel and diaphragmatic repair but also definitive correction of malrotation to prevent recurrence and future volvulus [10,15].

The diagnosis of malrotation is usually established radiologically using an upper gastrointestinal contrast study, which remains the gold standard for identifying abnormal position of the duodenojejunal junction. Additional imaging may include ultrasonography demonstrating abnormal SMA/SMV relationship or computed tomography in selected stable patients [10,15]. In our case, contrast study and CT were not performed because the patient's worsening clinical condition required urgent operative management. This decision reflects real-life surgical practice, where immediate intervention may be safer than delayed imaging in a child with progressive obstruction.

The association between Edwards syndrome and gastrointestinal malformations played a major role in this case. Trisomy 18 is characterized by multiple congenital abnormalities affecting nearly every organ system. Gastrointestinal defects such as omphalocele, diaphragmatic defects, intestinal fixation abnormalities, and feeding disorders are common [16]. Because these patients frequently have chronic feeding difficulties and poor nutritional tolerance, new symptoms of obstruction may initially be underestimated or attributed to baseline disease.

In addition, surgical decision-making in children with trisomy 18 may be complex because prognosis is influenced by severe cardiac defects, respiratory problems, neurological impairment, and overall life expectancy [16]. Historically, aggressive surgical treatment was sometimes avoided because of poor survival expectations. However, modern management increasingly supports individualized decision-making focused on symptom relief, quality of life, and family-centered care. In our patient, despite hospice care status, surgical intervention was clearly indicated because the obstruction was progressive, potentially life-threatening, and surgically correctable.

Delayed presentation of malrotation beyond infancy has been described in numerous pediatric studies. Some reports describe diagnosis in school-age children and even adolescents presenting with chronic abdominal pain, recurrent vomiting, malnutrition, or incidental findings during surgery [5,6,10]. Therefore, describing late presentation as extremely rare may be misleading. The educational importance lies more in recognizing diagnostic clues and maintaining clinical suspicion than in the patient's age alone.

Another important aspect is that intestinal malrotation is frequently discovered incidentally during operations performed for other abdominal pathology [13]. In our patient, the initial operative indication was bowel obstruction associated with suspected diaphragmatic hernia. Definitive diagnosis of Ladd's syndrome

was made only after complete abdominal exploration. This reinforces the importance of systematic intraoperative assessment, particularly in syndromic children with known congenital abnormalities.

The standard treatment for symptomatic malrotation remains the Ladd procedure [15,17]. This procedure includes division of Ladd's bands, widening of the mesenteric base, placement of the small bowel predominantly on the right and the colon on the left, and appendectomy in many centers because of the abnormal location of the appendix. In our patient, division of the obstructing bands and correction of bowel positioning successfully relieved the obstruction. No volvulus was present, which likely contributed to the favorable postoperative outcome.

Postoperative recovery in such patients requires careful monitoring because feeding intolerance may persist temporarily and nutritional rehabilitation may be difficult, especially in children with underlying syndromic disease. In our patient, gradual reintroduction of enteral feeding was successful, and symptoms resolved without recurrence during short-term follow-up.

This case supports the recommendation that intestinal malrotation should remain in the differential diagnosis of persistent vomiting and bowel obstruction at any pediatric age. This is particularly important in patients with known chromosomal syndromes and multiple congenital anomalies, where overlapping abnormalities may delay diagnosis and obscure the true mechanism of obstruction [4,5].

Conclusions

The educational value of this case lies not simply in the age of presentation but in the complex diagnostic process involving coexisting bilateral diaphragmatic defects, Edwards syndrome, and intestinal malrotation with obstructing Ladd's bands [14,16].

Although the majority of patients with intestinal malrotation are diagnosed during infancy, delayed presentation in older children is well documented and should not be underestimated [5,6]. Persistent vomiting, feeding intolerance, abdominal distension, constipation, and signs of upper gastrointestinal obstruction should prompt consideration of malrotation even beyond the neonatal period [4,10].

In children with trisomy 18 and multiple congenital anomalies, maintaining a high index of suspicion is particularly important because associated thoracoabdominal abnormalities may mask the underlying diagnosis and delay definitive treatment [16]. Imaging may suggest one dominant pathology, while the true mechanism of obstruction may only become apparent during surgery.

This case also demonstrates that urgent operative management may be necessary even without complete preoperative radiological characterization when the patient's clinical condition is deteriorating. Prompt surgical intervention allowed both correction of diaphragmatic defects and definitive treatment of Ladd's syndrome [15,17].

Early recognition, appropriate surgical management, and careful postoperative follow-up remain essential for achieving favorable outcomes in children with intestinal malrotation, especially in patients with complex syndromic conditions such as Edwards syndrome [4,5,10,16].

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