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CASE REPORT

Congenital duodenal stenosis: a rare cause of failure to thrive

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Abstract

Introduction: Duodenal stenosis is a congenital condition involving partial obstruction of the duodenal lumen. It often presents later with vague symptoms, delaying diagnosis. **Case Report:** We describe the case of a 19-month-old girl with recurrent vomiting since the neonatal period, initially resolving with no signs of obstruction. At 16 months, she showed faltering growth, initially attributed to colitis, with temporary symptom improvement. Three months later, vomiting recurred. The combination of chronic vomiting, growth delay and symptom recurrence raised the suspicion of a structural issue. Imaging confirmed duodenal stenosis, and she underwent a corrective duodenoduodenostomy. **Discussion:** This case highlights incomplete intestinal obstruction with non-specific symptoms delaying diagnosis. Clinicians should maintain a high index of suspicion for structural anomalies in children with persistent vomiting and failure to thrive.

Keywords: Duodenal atresia. Duodenal stenosis. Failure to thrive.

Estenose duodenal congênita: uma causa rara de falha no crescimento

Resumo

Introdução: A estenose duodenal é uma anomalia congénita que causa obstrução parcial do duodeno, frequentemente com sintomas inespecíficos e diagnóstico tardio. **Relato de caso:** Menina de 19 meses com vômitos recorrentes desde o período neonatal, inicialmente atribuídos à colite. Aos 16 meses, apresentou atraso no crescimento. Após melhora temporária, os sintomas retornaram. A combinação de vômitos crônicos, atraso ponderal e recorrência levou à suspeita de estenose duodenal, confirmada por imagem. Foi submetida a duodenoduodenostomia corretiva. **Discussão:** Este caso ilustra uma obstrução intestinal incompleta com apresentação clínica inespecífica. É essencial manter alta suspeição diagnóstica em crianças com vômitos persistentes e atraso no desenvolvimento.

Palavras-chave: bstrução duodenal. Atraso de crescimento. Diagnóstico tardio.

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Keypoints

What is known
- Duodenal stenosis is a congenital anomaly causing intestinal obstruction and often presents with non-specific symptoms.
- Delayed diagnosis of duodenal stenosis can lead to chronic complications such as failure to thrive.
- Imaging studies, including ultrasonography, are key to identifying partial intestinal obstructions.

What is added
- This case highlights a rare case of duodenal stenosis presenting as faltering growth with mild recurrent vomiting in early childhood.
- It demonstrates the role of flow ultrasonography in confirming the diagnosis.
- It emphasizes the importance of early surgical intervention for better growth outcomes.

Introduction

Congenital intestinal obstruction due to atresia, stenosis or webbing is a rare congenital anomaly with a prevalence of approximately one in 5,000 to 10,000 births and a higher incidence in males compared to females[1]. Duodenal atresia is a complete obstruction and more common than duodenal stenosis. It may be diagnosed antenatally or present very early during the neonatal period. By contrast, duodenal stenosis is a partial obstruction and often has a delayed and varied presentation, making diagnosis and early intervention more challenging[1].

These conditions arise from disruptions to gastrointestinal development, including failure of recanalization during weeks eight to ten, defects in the embryonic foregut or vascular disruptions leading to ischemic necrosis of intestinal segments[2]. This may be recognized antenatally on ultrasound with the pathognomonic finding being a 'double bubble sign' for duodenal atresia[1]. The clinical manifestations of duodenal stenosis include recurrent vomiting, abdominal distension, failure to thrive and electrolyte abnormalities[3].

A plain abdominal radiograph is the first step to evaluate and it typically shows a dilated stomach with distal bowel gas[3]. Further imaging, such as upper gastrointestinal contrast studies, may be required to confirm the diagnosis in cases where the obstruction is not clear on plain abdominal radiographs[4]. A diagnosis of congenital duodenal stenosis may be delayed until adolescence, especially in cases of incomplete intestinal obstruction with non-specific symptoms[3]. We present this rare, delayed case to emphasize the need for healthcare professionals to consider duodenal stenosis as a potential diagnosis in children experiencing recurrent vomiting and failure to thrive.

Case report

We report the case of duodenal stenosis in a 19-month-old girl who presented with intermittent episodes of recurrent vomiting and failure to thrive since her neonatal period. She was born at 37 weeks of gestation via emergency lower segment caesarean section due to cervical dystocia, with a birth weight of 2.49 kg and was discharged postnatally with no complications.

Her parents reported recurrent episodes of vomiting since the neonatal period, occurring 2 – 3 times daily, post-feeding. Her vomitus was yellowish fluid with occasional milk curds or food particles. There was no history of bilious vomiting. Despite seeking medical attention at community clinics multiple times, her parents were reassured and discharged home. At 16 months of age, she was admitted to the hospital for infective colitis with subacute intestinal obstruction and acute kidney injury.

During this admission, she presented with fever and vomiting for four days. Clinical examination revealed mild dehydration with no signs of abdominal distension or bilious aspirate. An abdominal radiograph during this admission showed dilated proximal bowel with thickened bowel wall and paucity of distal bowel gas, as shown in Figure 1. An abdominal ultrasound scan showed prominent hepatic and splenic flexure with thickened bowel wall. She was treated as for infective colitis and was started on intravenous antibiotics. During this admission, she was noted to have faltering growth, falling from the 10th percentile at birth to the 5th percentile subsequently. At 19 months of age, her weight was 7.5 kg (below the 5th percentile) and her height was 76 cm (below the 5th percentile). She was discharged fourteen days later with complete resolution of symptoms. Her faltering growth was attributed to poor nutrition.

Subsequently, she developed constipation with Bristol Type 1 – 2 stools from the age of 17 months. Although she had a normal bowel movement frequency of five times per week, she would cry and strain upon defecating. This temporarily improved to Bristol stool types 2 – 3 after switching formula milk brands. However, it subsequently worsened again for two weeks prior to the most recent presentation at the

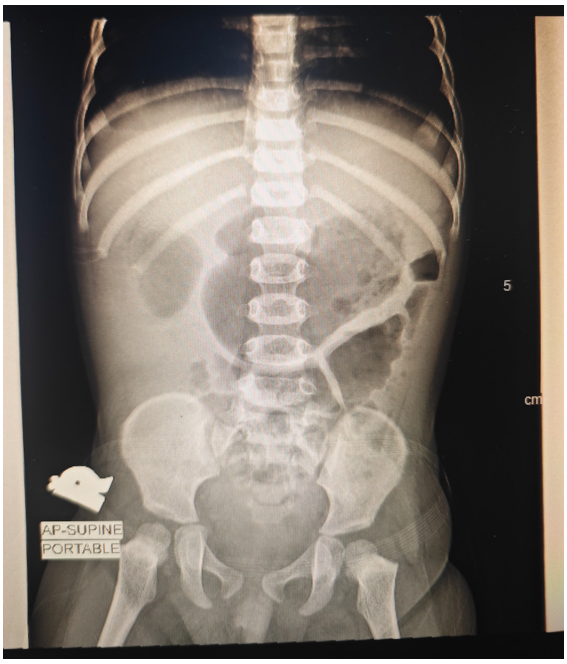

Figure 1. Abdominal radiograph at 16 months of age.

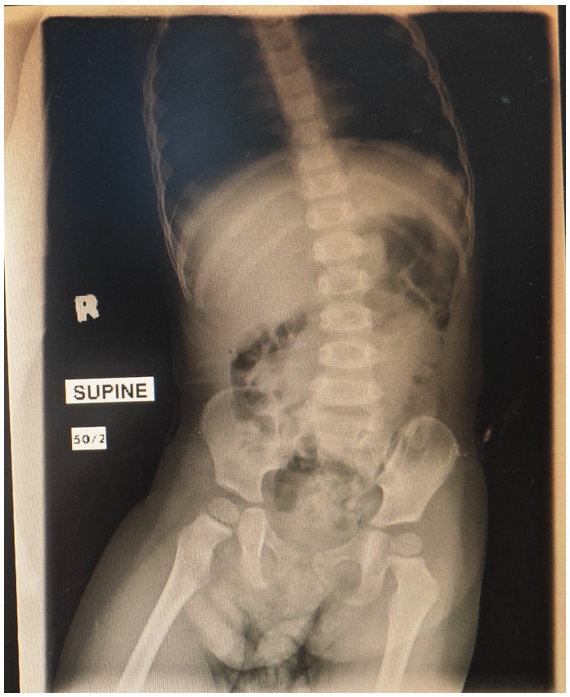

Figure 2. Abdominal radiograph at 19 months of age.

hospital, with Bristol Type 1 – 2 stools and one episode of blood-streaked stool.

Her dietary history indicated that she tolerated a solid diet three times per day with one snack in between. She was not a picky eater. Normally, she ate rice or biscuits for breakfast, rice with chicken or vegetables, or sometimes noodles, for lunch and had similar meals for dinner. She took 210 ml of normal infant formula milk three times a day. She did not have iron deficiency anemia and there were no other indications of nutritional deficiencies.

Otherwise, the child was active prior to this illness, and there was no abdominal pain. She did not experience a drastic weight loss, diarrhea, steatorrhea, pain during urination, foul-smelling urine, recurrent illness, yellowish discoloration, heart failure symptoms, head trauma, inconsolable crying, cough or any infectious symptoms.

There was no significant past surgical history or food and drug allergies. Immunizations were up to date for her age. Her development was normal, and there was no significant family history of bowel disease or malignancy. There were no family members who had passed away at a young age.

Examination upon admission showed a small build with poor muscle bulk. There were no dysmorphic features, and she had good perfusion with no pallor or jaundice. The abdomen was slightly distended with a palpable fecal mass in the left iliac fossa upon palpation. There was no hepatosplenomegaly. Bowel sounds were heard. Otherwise, other systemic examinations revealed unremarkable findings.

Initial laboratory investigations revealed thrombocytosis and leukocytosis with normal hemoglobin levels. She also exhibited metabolic alkalosis with a high serum lactate level. A midstream urine sample showed positive leukocytes with the presence of ketones. However, the urine culture was negative for any bacterial growth. An abdominal radiograph showed a hugely dilated stomach and duodenum up to the descending part with minimal gas presence in the distal bowel, suggestive of partial obstruction, as shown in figure 2.

An abdominal ultrasound with water injection into the nasogastric tube showed progressive dilatation of the stomach and proximal duodenum as water moved through, with no further movement of water seen passing through D3 of the duodenum, suggesting a duodenal stenosis. Pediatric endoscopy is not available in our setting. She initially required intravenous fluid maintenance and correction for moderate dehydration. A nasogastric tube was inserted and bilious content was drained. She was consequently referred to the pediatric surgical team in a tertiary referral center and was transferred there for surgical intervention. She underwent a duodenoduodenostomy and was discharged well after nutritional and post-operative rehabilitation. She has

not had any vomiting since, and her weight is now on the 10th percentile with evidence of catch-up growth.

Discussion

The non-specific presentation of congenital duodenal stenosis poses significant diagnostic challenges, often resulting in delayed management and complications, as reported by Gisella et al.[3], Velmishi et al.[5] and Eyad et al.[6]. In partial congenital duodenal obstruction, diagnosis may be delayed until adolescence[5]. This delayed diagnosis in children might be explained by the fact that they can tolerate small feedings due to the incomplete blockage of the duodenal lumen. Delayed diagnosis may lead to frequent vomiting and aspiration pneumonia. It also results in inadequate nutrient absorption, causing poor weight gain and malnutrition[5]. This is seen in this patient who experienced recurrent episodes of vomiting and failure to thrive.

Duodenal atresia and stenosis are similar abnormalities, differing only in the degree of obstruction of the duodenal lumen. Duodenal atresia involves complete obstruction while duodenal stenosis involves incomplete obstruction. These conditions are often associated with Down Syndrome and other congenital malformations[1]. About one third of patients with duodenal atresia or stenosis have Down Syndrome[1]. Our patient had no risk factors, as she was born at term and did not have Down Syndrome or congenital heart defects.

Initial management of intestinal obstruction includes stomach decompression, fluid resuscitation and correction of electrolyte imbalances, primarily metabolic alkalosis[2]. Definitive management is surgical correction[2].

The challenges faced by this patient are primarily due to her non-specific symptoms. This patient presented with recurrent vomiting, failure to thrive and constipation. Recurrent vomiting is a symptom of various illnesses including pyloric stenosis, intestinal atresia or stenosis, inflammatory bowel disease, intussusception, meningitis, encephalitis, urinary tract infection, gastroesophageal reflux, congenital heart disease and many more. It is unfortunate for this patient as her mother had a history of seeking treatment multiple times for her recurrent vomiting.

This case highlights the diagnostic challenges posed by atypical presentations of congenital duodenal obstruction, particularly in the absence of classic neonatal symptoms or associated risk factors. Clinicians must maintain a high index of suspicion for such rare congenital anomalies when faced with recurrent vomiting, failure to thrive and chronic gastrointestinal symptoms. Early recognition and prompt surgical intervention are crucial to prevent prolonged morbidity and ensure optimal outcomes for affected patients. Enhanced awareness and timely diagnosis can significantly reduce delays in care, improving the quality of life for children with this condition.

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Author contributions

A. Mohammad Khuzaini: Idea behind the study and design. N. Afifah Aziz and A. Mohammad Khuzaini: Data collection. N. Afifah Aziz, K. Alhuda Kamilen, and A. Mohammad: Interpretation of results; Draft manuscript preparation. All the authors reviewed and approved the final manuscript.

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Conflicts of interest

None.

Ethical considerations

Protection of humans and animals. The authors declare that no experiments involving humans or animals were conducted for this research.

Confidentiality, informed consent, and ethical approval. The authors have followed their institution's confidentiality protocols, obtained informed consent from patients and received approval from the Ethics Committee. The SAGER guidelines were followed according to the nature of the study.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing of this manuscript.

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