

Case Report

Delayed Diagnosis of Congenital Duodenal Stenosis in a 16-Year-Old Girl

Virtut Velmishi ¹, Dritan Alushani,² Ermira Dervishi ¹, Saimir Heta ², Spiro Sila,² and Paskal Cullufi ¹

¹Service of Pediatric Gastroenterology “Mother Teresa” University Hospital Center, Tirana, Albania

²Service of pediatric Surgery “Mother Teresa” University Hospital Center, Tirana, Albania

Correspondence should be addressed to Virtut Velmishi; tutimodh@yahoo.com

Received 22 February 2024; Revised 21 April 2024; Accepted 22 April 2024; Published 4 May 2024

Academic Editor: Simon Ching Shun Kao

Copyright © 2024 Virtut Velmishi et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Background. Duodenal atresia or stenosis are different degrees of the same abnormality. They usually occur at the level of the ampulla of Vater and are thought to be an embryologic defect during the development of the foregut, leading to abnormal recanalization. Complete duodenal atresia is usually symptomatic in the early neonatal period, while partial obstruction (web, stenosis) may have a late presentation and a more challenging diagnosis such as in our case. **Case Presentation.** The patient, a 16-year-old girl, presented with abdominal pain, recurrent vomiting, and growth failure. An upper GI study with barium showed an image compatible with gastropnoxis. Further diagnostic procedures confirmed a rare finding such as congenital duodenal stenosis. She underwent surgical intervention, and the recovery period was uneventful. **Conclusion.** Gastropnoxis is not diagnostic for a particular disease. This rare radiological finding in children may obscure uncommon diagnosis, such as congenital duodenal stenosis, which can present a diagnostic challenge beyond early childhood.

1. Background

Congenital duodenal obstruction caused by duodenal atresia or duodenal stenosis is a rare congenital anomaly. The prevalence of duodenal obstruction (atresia, web, stenosis) is 1 : 6000 [1–3]. Nowadays, approximately half of all cases are detected antenatally on fetal ultrasonography with the characteristic presence of a “double bubble” sign in the upper abdomen representing dilated fluid-filled stomach and proximal duodenum. Suspicion of congenital duodenal obstruction demands an accurate investigation of associated anomalies, such as cardiac and aneuploidy, commonly occurring in up to 84% of infants, depending on the reported population [4]. Congenital duodenal obstruction can occur in association with an annular pancreas, resulting in a partial or complete duodenal obstruction. A significant number of affected infants also have Down syndrome. The definitive management of congenital duodenal stenosis is surgical, aiming to restore gastrointestinal continuity while avoiding

damage to adjacent structures, principally the biliary and pancreatic ducts. Surgeons use several different procedures, techniques, and approaches, and none have proven benefit over any other [5, 6]. Most patients with atresia or duodenal stenosis are diagnosed postnatally or at a young age. This case shows a delayed diagnosis of duodenal stenosis in a 16-year-old girl. This manuscript was prepared following the CARE guidelines (<https://www.care-statement.org>).

2. Case Report

A 16-year-old girl was sent to the gastroenterology service because of abdominal pain, recurrent vomiting, and malnutrition. Gastrointestinal symptoms began when she was a little girl. Her parents were divorced and her appointments at healthcare centers were not regular. According to her sister she was never hospitalized but was treated for her gastrointestinal symptoms by her family doctor without significant improvement. No family diseases were reported.

The CBC showed mild anemia, while biochemical examinations were within the normal range. Her objective assessment showed low staturponderal growth. Her weight was 40 kg (z score = -2.30 ; percentile 1.072%), and her height was 155 cm (z score: -1.17 ; percentile 12%). She was pale, but no facial dysmorphism was noticed. Heart sounds were normal. Her arterial pressure was in the normal range according to her age. Lungs were clear during auscultation. The abdomen was soft without enlargement of the liver or spleen. Her extremities were thin, but the skin was intact. An upper GI study with barium showed images compatible with gastropnoptosis (Figure 1). During an upper digestive endoscopy, we noticed food residues in the stomach (Figure 2) and a narrow duodenal bulb, making it difficult to pass the endoscope through the duodenum. Initially, we thought of bulbar stenosis due to bulb ulceration. In this context, an antral biopsy was taken. The result was negative for *helicobacter pylori*, confirmed by a negative breath test. We treated her with a high dose of omeprazole for four weeks, but clinical signs were the same. In the context of growth failure, we excluded celiac disease and inflammatory bowel diseases (IBD). Serology for celiac disease and fecal calprotectin were normal. An abdominal CT did not reveal an annular pancreas or other abnormalities compatible with IBD despite the view of an enlarged stomach. A second upper digestive endoscopy confirmed a post-bulbar stenosis approximately 4-5 mm. After many consultations, the decision was made that the definitive treatment would be surgery. During the intervention, it was noticed that the stomach was enlarged (Figure 3) which ended in a small and narrowed duodenum (Figure 4). Finally, it was clear that the cause of recurrent vomiting and abdominal pain was congenital stenosis of the duodenum, which age of the patient. The surgeons proceeded with an intervention similar to Billroth type 1 because of the enlarged stomach (Figure 5). After the surgical procedure, the girl became stable and did not present dysmotility symptoms. Gradually, she was tolerating the liquid enteral nutrition and later solid foods. Initially, we used a transanastomotic tube to nourish her. She was discharged two weeks after the intervention. We performed another upper GI study with barium three months later, which showed a normal regular form and stomach position (Figure 6).

3. Discussion

Gastropnoptosis is a radiological finding. There are some mechanisms suggested to cause gastropnoptosis and GI symptoms. The downward displacement of the stomach is thought to be caused by the relaxation or stretching of the muscle or by a decrease in the muscle tone [7]. In the literature, gastropnoptosis seems to be associated with delayed gastric emptying [8, 9]. In our case, delayed gastric time was caused by congenital duodenal stenosis, which is very rare, making surgery the best option for recovery. Initially, because of patient's age, we thought of postbulbar stenosis due to a chronic bulbar ulcer. The misdiagnosis of postbulbar duodenal stenosis was made based on failure to examine the duodenum on gastroscopy and the interpretation of upper



FIGURE 1: Barium swallow shows gastropnoptosis: stomach is under the level of iliac crests.

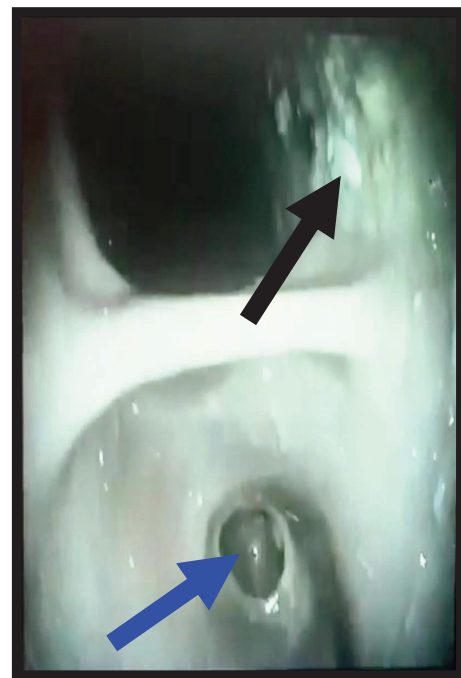


FIGURE 2: Upper digestive endoscopy shows a narrow duodenal bulb (blue arrow) and some food residues into the stomach (black arrow).

gastrointestinal barium examination showing a large stomach and obstruction beyond duodenal bulb. It is unexpected to discover a congenital abnormality of the duodenum in a 16-year-old. Despite gastrointestinal symptoms, her intellect was perfect. Our patient had no other diseases, including trisomy 21, congenital cardiac disease, and prematurity [10] associated with congenital duodenal obstruction. It has been reported that diagnosis of congenital duodenal obstruction (CDO) may be delayed even up to adolescence [11–13]. The delayed diagnosis in children may be explained because they can tolerate small feedings due to



FIGURE 3: Enlarged stomach during the intervention.



FIGURE 5: The anastomosis similar to Billroth type 1.

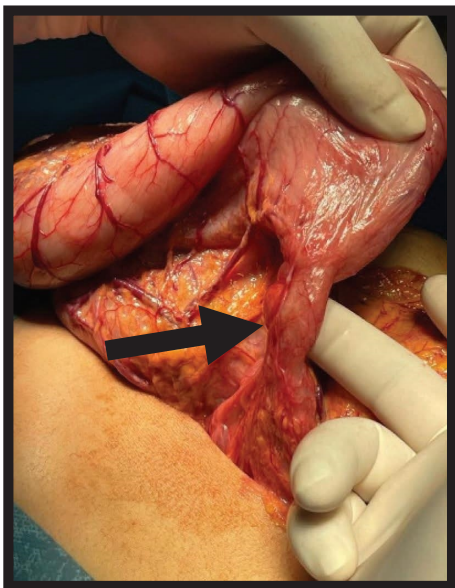


FIGURE 4: Congenital duodenal stenosis during the intervention.

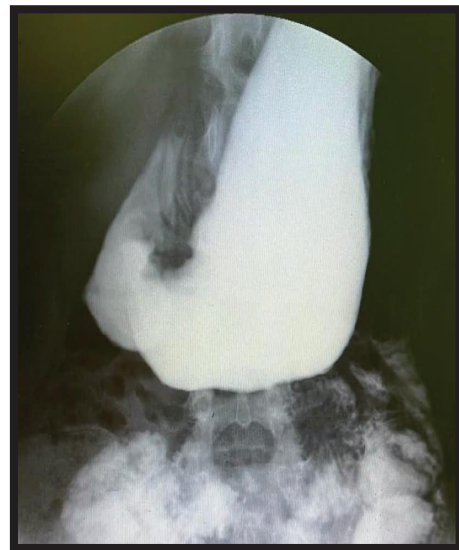


FIGURE 6: The stomach is in a normal position 3 months after the intervention.

the incomplete nature of the obstruction. In some cases, duodenal stenosis goes unrecognized for long periods, leading to recurrent episodes of vomiting, failure to thrive, and aspiration pneumonia. In our case, the most frequent symptom was recurrent vomiting. Following surgery, infants frequently have a period of upper gastrointestinal dysmotility secondary to chronic in-utero obstruction of proximal duodenum and gastric dilatation. Several strategies are used to provide nutrition while normal gastrointestinal function returns. This includes intravenous (parenteral) feeding and enteral feeding into the bowel distal to the level of obstruction through a trans-anastomotic tube. No approach is shown to be superior to any other [14–16]. After taking into consideration the patient's age and enlarged stomach, our

surgeons preferred a procedure similar to Billroth type 1. Postoperatively, the patient was started on enteral nutrition with a transanastomotic tube (TAT) combined with parenteral nutrition. The recovery period was uneventful, and the patient was discharged home two weeks after the intervention.

4. Conclusion

Gastroptosis is not diagnostic for a particular disease. This rare radiological finding in children may obscure uncommon diagnosis, such as congenital duodenal stenosis, which can present a diagnostic challenge beyond early childhood.

Abbreviations

CDO: Congenital duodenal obstruction
 TAT: Transanastomose tube
 CT: Computed tomography
 CD: Celiac disease
 IBD: Inflammatory bowel disease
 GI: Gastrointestinal.

Data Availability

All information is available in the clinical files archive of the University Hospital “Mother Teresa”, Tirana, Albania.

Ethical Approval

This case report was conducted in accordance with the fundamental principles of the Declaration of Helsinki. An approval by ethics committee of the University Hospital “Mother Teresa” was taken.

Consent

Written informed consent was obtained from the patient parents for publication of this case report and any accompanying images. A copy of the written consent form is available for review by the Editor-in-Chief of this journal.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Authors' Contributions

VV drafted the manuscript. DA provided images and approved the manuscript. All co-authors read and approved the manuscript.

References

- [1] M. K. Jangid, R. Ahmad, and S. Pandre, “Delayed presentation of a congenital duodenal web managed successfully with incision of web,” *BMJ Case Reports*, vol. 13, no. 12, p. e237307, 2020.
- [2] S. A. Mousavi, H. Karami, and H. Saneian, “Congenital duodenal obstruction with delayed presentation: seven years of experience,” *Archives of Medical Science*, vol. 5, pp. 1023–1027, 2016.
- [3] P. V. Bailey, T. F. Tracy, R. H. Connors, D. P. Mooney, J. E. Lewis, and T. R. Weber, “Congenital duodenal obstruction: a 32-year review,” *Journal of Pediatric Surgery*, vol. 28, no. 1, pp. 92–95, 1993.
- [4] D. Takahashi, T. Hiroma, S. Takamizawa, and T. Nakamura, “Population-based study of esophageal and small intestinal atresia/stenosis,” *Pediatrics International*, vol. 56, no. 6, pp. 838–844, 2014.
- [5] B. Parmentier, M. Peycelon, C.-O. Muller, A. El Ghoneimi, and A. Bonnard, “Laparoscopic management of congenital duodenal atresia or stenosis: a single-center early experience,” *Journal of Pediatric Surgery*, vol. 50, no. 11, pp. 1833–1836, 2015.
- [6] D. C. Van der Zee, “Laparoscopic repair of duodenal atresia: revisited,” *World Journal of Surgery*, vol. 35, no. 8, pp. 1781–1784, 2011.
- [7] A. J. M. Van Welie, W. M. Klein, and J. M. ThDraaisma, “The clinical or radiographic diagnosis of gastropothesis: still relevant?” *Gastro- Open Journal*, vol. 2, no. 1, pp. 14–19, 2017.
- [8] E. Christianakis, K. Bouchra, A. Koliatou, N. Paschalidis, and D. Filippou, “Gastroparesis associated with gastropothesis presenting as a lower abdominal bulking mass in a child: a case report,” *Cases Journal*, vol. 2, no. 1, 2009.
- [9] M. Kusano, F. Moki, H. Hosaka et al., “Gastropothesis is associated with less dyspepsia, rather than a cause of dyspepsia, in Japanese persons,” *Internal Medicine*, vol. 50, no. 7, pp. 667–671, 2011.
- [10] S. Bairdain, D. C. Yu, C. Lien et al., “A modern cohort of duodenal obstruction patients: predictors of delayed transition to full enteral nutrition,” *Journal of Nutrition and Metabolism*, vol. 2014, pp. 1–6, 2014.
- [11] A. Savino, V. Rollo, and F. Chiarelli, “Congenital duodenal stenosis and annular pancreas: a delayed diagnosis in an adolescent patient with Down syndrome,” *European Journal of Pediatrics*, vol. 166, no. 4, pp. 379–380, 2006.
- [12] P. Klein, R. Anetsberger, R. Stangl, and H. P. Hummer, “Kongenitale Duodenalstenose im Erwachsenenalter,” *Langenbecks Archiv fur Chirurgie*, vol. 379, no. 1, pp. 54–57, 1994.
- [13] R. AlGhannam and Y. A. Yousef, “Delayed presentation of a duodenal web,” *Journal of Pediatric Surgery Case Reports*, vol. 3, no. 12, pp. 530–533, 2015.
- [14] N. J. Hall, M. Drewett, R. A. Wheeler, D. M. Griffiths, L. J. Kitteringham, and D. M. Burge, “Trans-anastomotic tubes reduce the need for central venous access and parenteral nutrition in infants with congenital duodenal obstruction,” *Pediatric Surgery International*, vol. 27, no. 8, pp. 851–855, 2011.
- [15] M. Bishay, B. Lakshminarayanan, A. Arnaud et al., “The role of parenteral nutrition following surgery for duodenal atresia or stenosis,” *Pediatric Surgery International*, vol. 29, no. 2, pp. 191–195, 2012.
- [16] G. S. Bethell, A. M. Long, M. Knight, and N. J. Hall, “Congenital duodenal obstruction in the UK: a population-based study,” *Archives of Disease in Childhood- Fetal and Neonatal Edition*, vol. 105, no. 2, pp. 178–183, 2019.