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Congenital Duodenal Obstruction: Diagnostic Challenges and Surgical Outcomes in Libya Case Series

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Abstract

Background: Congenital duodenal obstruction [CDO] is a rare cause of upper intestinal obstruction in children. While most cases are diagnosed during the neonatal period, partial obstruction may remain undetected until later infancy or childhood, leading to delayed diagnosis and chronic gastrointestinal symptoms.

Case Presentation: We report three pediatric cases of congenital duodenal obstruction managed at Misrata Medical Center, Libya. The first case involved a 19-month-old female with chronic non-bilious vomiting, failure to thrive, and recurrent respiratory infections secondary to a congenital duodenal web associated with patent ductus arteriosus. The second case was a 6-month-old infant with Down syndrome and annular pancreas presenting with persistent vomiting and severe malnutrition. The third case involved a neonate with a congenital duodenal web presenting shortly after birth with vomiting and melena. All patients underwent successful surgical correction with excellent postoperative recovery.

Conclusion: Congenital duodenal obstruction [CDO] may present beyond the neonatal period with nonspecific symptoms, resulting in delayed diagnosis. Persistent vomiting and growth failure should prompt early radiological evaluation. Early recognition and timely surgical intervention are essential to improve nutritional and clinical outcomes.

I) INTRODUCTION:

The duodenum is the most common site of intestinal obstruction, accounting for nearly half of all cases, while the ileum is the least affected. [1, 2]

Congenital duodenal obstruction [CDO] represents a spectrum of intrinsic and extrinsic anomalies involving the duodenum, ranging from complete atresia to partial stenosis or web formation. [4,6]
Intrinsic lesions include duodenal stenosis and mucosal webs, whereas annular pancreas represents an extrinsic cause of obstruction. [4, 6]
Its incidence is estimated at approximately 1 in 5,000–10,000 live births. [3] The majority of cases are detected in the neonatal period due to classical bilious vomiting and the "double bubble" sign on imaging. [3, 5]
However, when the obstruction is incomplete, the condition may remain unrecognized for months or even years.

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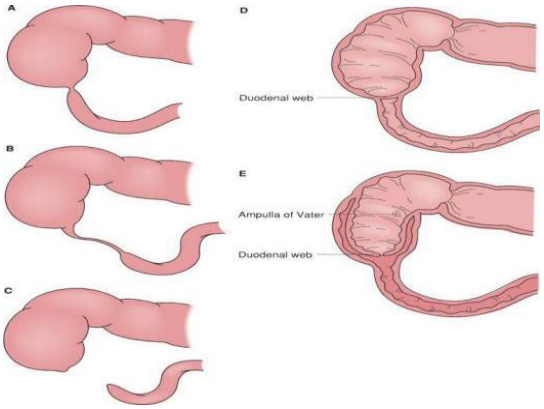
Nonetheless, several factors continue to affect the overall outcome, including premature birth, a high rate of associated anomalies, and the need for repeat surgeries. [6.7]
The overall long-term outcome is also affected by the high prevalence of Down syndrome. [7]
Delayed diagnosis can lead to chronic vomiting, malnutrition, and growth failure. [6]
Awareness of atypical and delayed presentations of CDO is therefore essential for pediatricians and pediatric surgeons to facilitate early diagnosis and timely surgical management.

Figure 1

Early recognition of atypical presentations is essential to avoid diagnostic delay and prevent nutritional and metabolic complications. In this report, we present three cases of congenital duodenal obstruction with variable clinical presentations, highlighting the diagnostic challenges and surgical outcomes in a tertiary center in Libya.

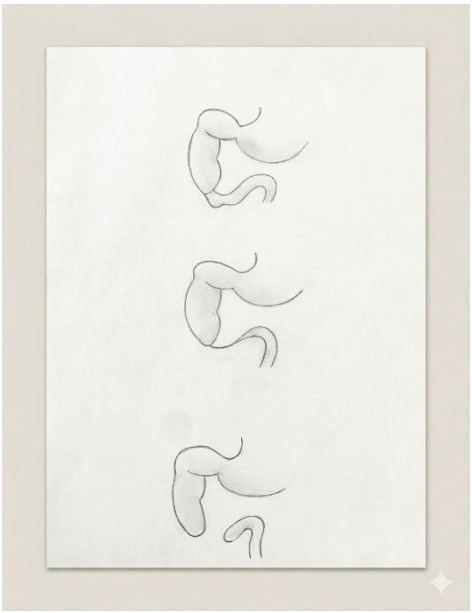
Figure 2 schematic illustration demonstrating the morphology classification of duodenal atresia

II) Case Presentation



A) Case 1

A 19-month-old female from Ubari, Libya , born at term via normal vaginal delivary was referred to our department, who presented with a 10-month history of recurrent, projectile non-bilious vomiting, progressive postprandial fullness, early satiety, and failure to thrive, associated with intermittent abdominal distention for one month, and chronic constipation. She had a known large patent ductus



arteriosus [PDA] [Figure 6] and recurrent hospital admissions for respiratory infections; she also had a history of multiple blood transfusions.

On examination, the patient appeared underweight and mildly dehydrated, with visible epigastric distension and active visible gastric peristalsis, and no organomegaly.

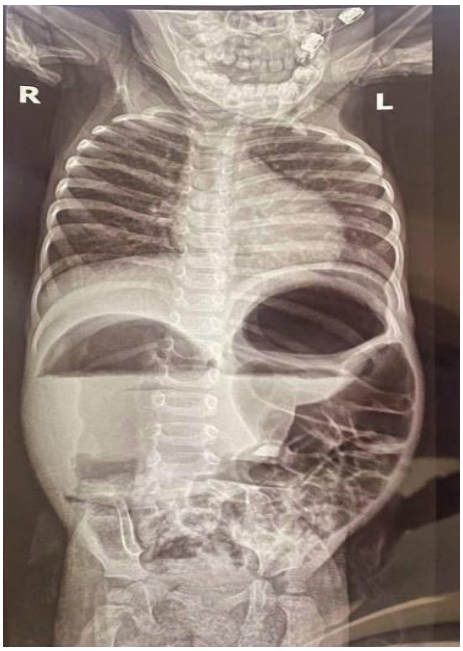
Laboratory tests revealed metabolic alkalosis secondary to recurrent vomiting.

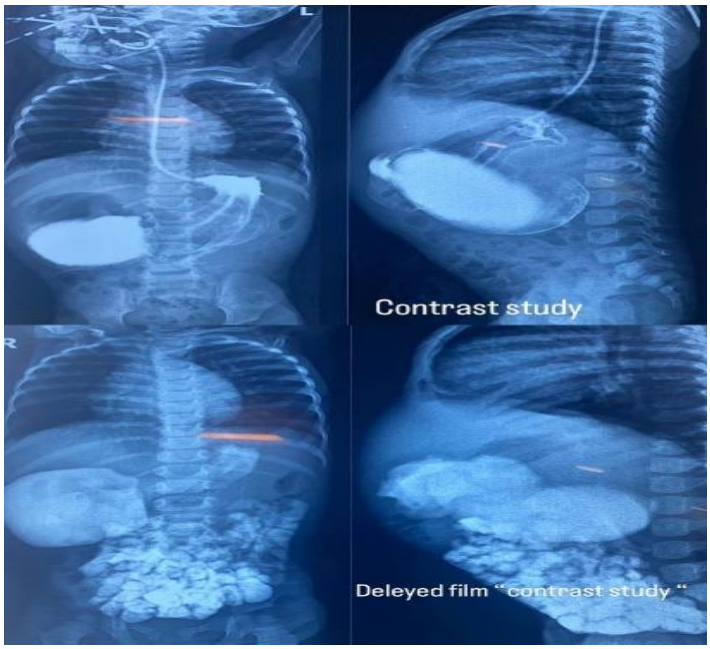
An abdominal X-ray showed a distended stomach and a prominent gas in the proximal duodenum with distal bowel gas, suggesting partial obstruction. ['Double bubble–like appearance ' but distal gas present]. [Figure 2]

Figure 3: X-ray chest and abdomen, huge dilated stomach and 1st part of duodenum with normal gas distribution in small bowel, sign of air fluid level in duodenum.

An upper gastrointestinal contrast study[Figure 3] demonstrated marked hold-up of contrast at the second portion of the duodenum with proximal dilatation, consistent with a duodenal web or stenosis.

Figure 4: Upper gastrointestinal contrast 'double bubble sign





CT scan abdomen confirmed [Figure 5,6] severe diffuse dilation of the stomach, dilation of the 1st and 2nd part of the duodenum, a severe stenosis at the site of the third part of the duodenum with significant gastric and proximal duodenal dilatation.

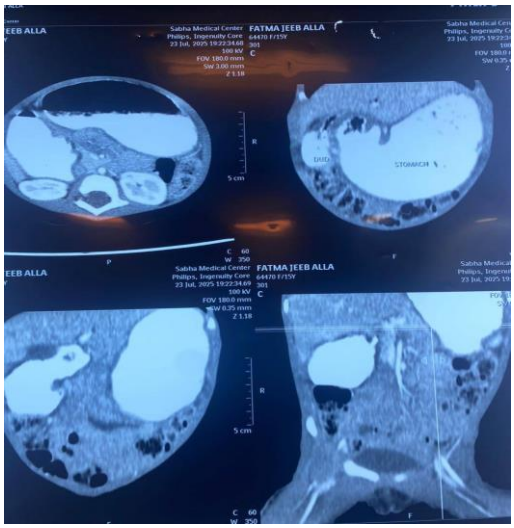


Figure 3 CT abdomen

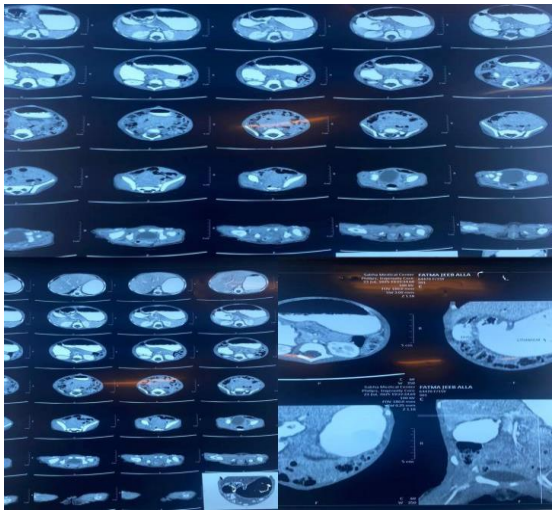


Figure 4 CT abdomen

Echocardiography revealed a large PDA without other structural defects, bidirectional shunt LVF. [Figure 6]

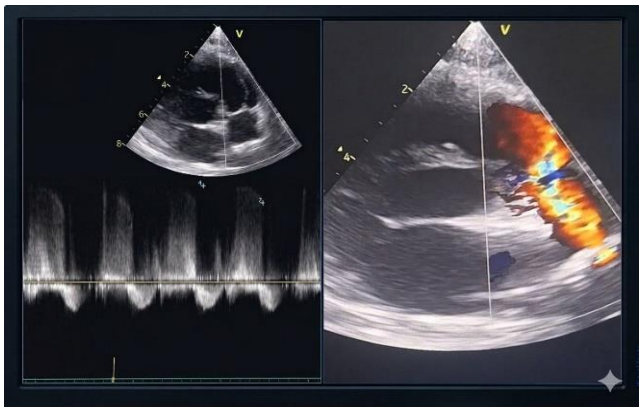


Figure 5: Echocardiogram with Doppler of PDA

A right upper transverse incision was made to perform an exploratory laparotomy. The dilated proximal duodenum was mobilized.

A transverse incision was created in the second part of the duodenum to improve exposure. A longitudinal incision of the 2nd part of the duodenum was then performed.

The duodenal web was visualized and confirmed as the cause of obstruction. Careful inspection showed its proximity to the sphincter of Oddi.[Figure 7]
A Trans-anastomotic nasogastric tube was inserted across the pylorus and advanced into the distal duodenum, approximately 15–20 cm distal to the anastomotic site, for continuity. The repair was tension-free and well perfused. Partial excision of the duodenal web was performed, followed by duodenplasty and restoration of the luminal.

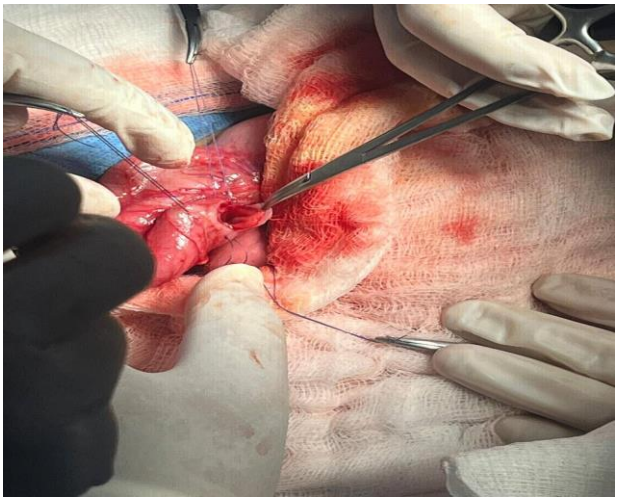


Figure 6: Intraoperative incision of the duodenum with diaphragm

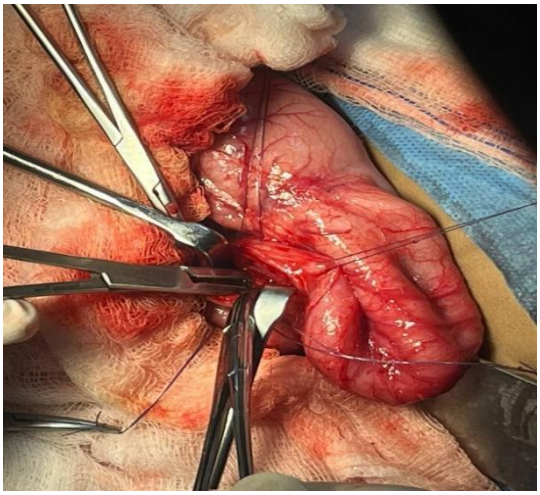


Figure 9 picture showing different between the proximal huge dilated and distal stenotic of the duodenum

The patient recovered smoothly following surgery. Nasogastric feeding was started on postoperative day 3 and advanced gradually. The nasogastric tube was removed on day 7.
She tolerated oral feeds well and was discharged in good condition.
Follow-up at six months showed significant weight gain and complete resolution of vomiting; a regular echocardiogram confirmed the small PDA and improved general cardiac condition. Surgical correction resulted in remarkable nutritional recovery and overall clinical improvement, with resolution of chronic vomiting, progressive weight gain, and subsequent improvement in cardiac status related to severe malnutrition and prolonged feeding intolerance.

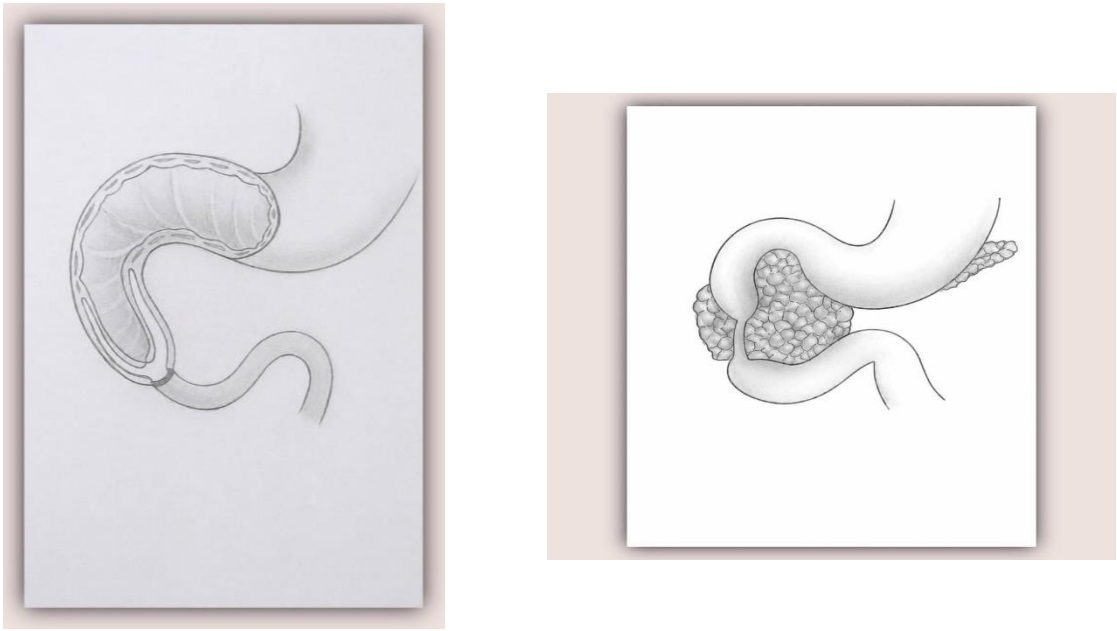


Figure 10 duodenal web

B) Case 2

A 6-month-old female Libyan infant, born at term (birth weight 3 kg), a known case of Down syndrome associated with congenital heart disease [CHD], presented with a history of persistent vomiting and failure to thrive since the neonatal period. The vomiting, initially non-bilious, later become brownish in color, accompanied by melena. Melena an uncommon presentation of congenital duodenal obstruction, was also noted. , although initial abdominal ultrasonography was reported as normal and the condition was misdiagnosed as esophageal dilatation elsewhere; persistent growth failure prompted further investigation.

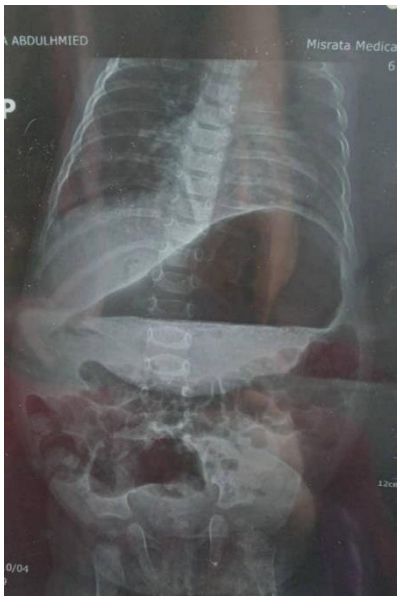


Figure 11: chest and abdomen, a huge dilated stomach, a single air fluid level of the stomach, and present intestinal gases.

A contrast upper gastrointestinal (UGI) study confirmed a high-grade duodenal obstruction. Following the contrast study, the patient experienced acute clinical deterioration, including severe dehydration and abdominal distension, requiring stabilization with nasogastric decompression, fluid resuscitation, and blood transfusion. [Figure 11A,11B]

Upon exploratory laparotomy, an annular pancreas was identified as the cause of extrinsic duodenal compression. A duodenduodenostomy bypass procedure was successfully performed.

Figure 12 congentail duodeneal obstruction 2ry annular pencreas

The postoperative period was marked by complete resolution of vomiting and rapid nutritional catch-up. At the 12-month follow-up, the patient was thriving and developmentally appropriate for her condition, a small congenital cardiac septal defect, which later underwent spontaneous closure.

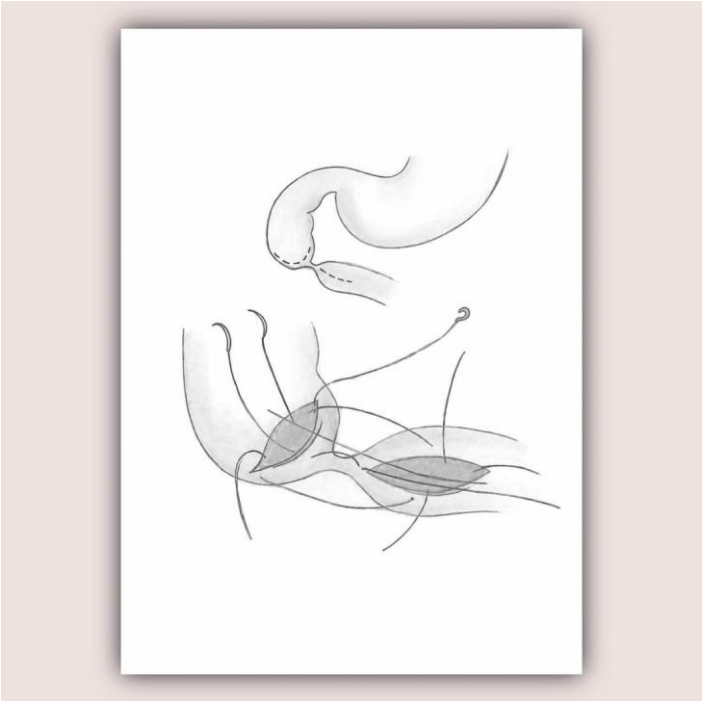


Figure 13duodenduodenostomy ;Diamond anastomosis

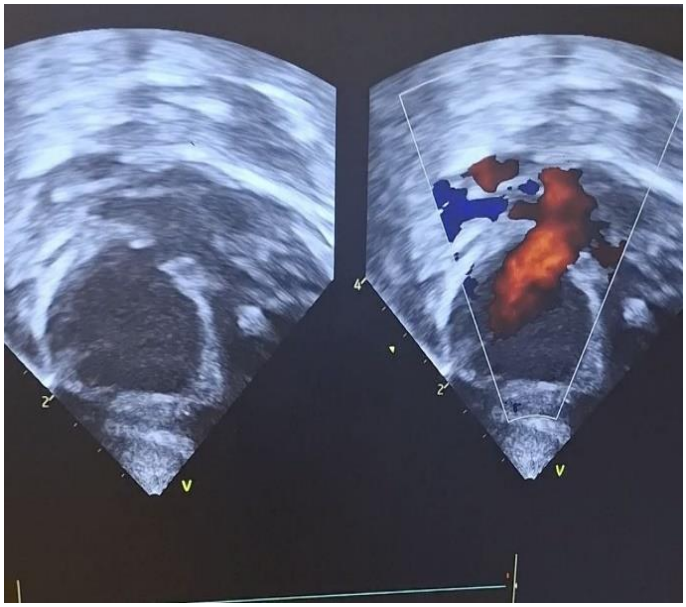


Figure 7: Echocardiogram with Doppler ASD II

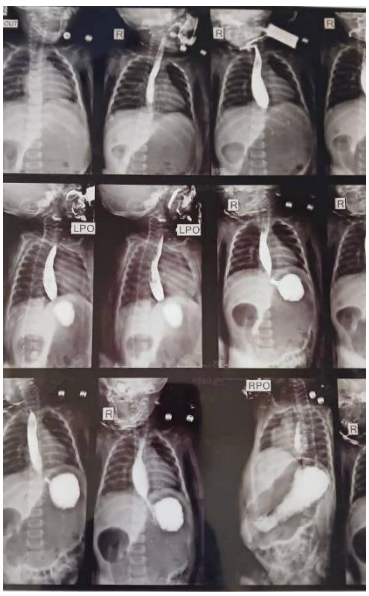


Figure 15A upper GIT contrast, esophagus dilation, huge stomach dilation, and 1st part of the duodenum 'double bubble ' with stenotic 2nd part of the duodenum.

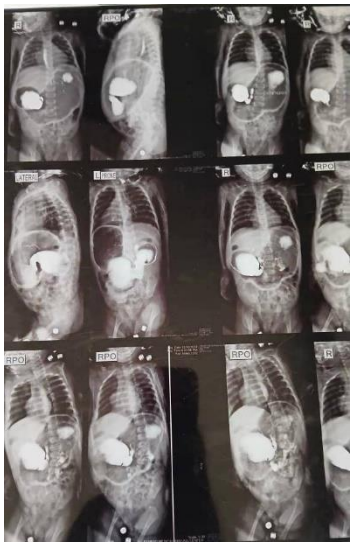


Figure 15B upper GIT contrast, esophagus dilation, huge stomach dilation, and 1st part of duodenum 'double bubble ' with stenotic 2nd part of duodenum

C) Case 3

A 2-day-old male neonate presented with persistent vomiting beginning on the first day of life. On the second postnatal day, the patient developed melena, an uncommon presentation of congenital duodenal obstruction, was also noted. , raising concern for significant upper gastrointestinal obstruction and gastrointestinal stasis. Initial evaluations performed at another healthcare facility were inconclusive, prompting referral to Misrata Medical Center for further management.

The patient was born at term through normal vaginal delivery with no reported antenatal complications. Upon admission, the infant was hemodynamically stable but continued to exhibit recurrent vomiting and poor feeding tolerance. Physical examination showed no obvious external congenital anomalies.

Routine laboratory investigations were within acceptable limits for surgical intervention.

On day 5 of age, an exploratory laparotomy was undertaken. Intraoperative findings demonstrated a congenital duodenal web causing high-grade proximal intestinal obstruction. Surgical correction was successfully performed with restoration of normal intestinal continuity.

Figure 8 complete duodenal web

Postoperative management included intravenous antibiotics for 7 days, nasogastric feeding, intravenous fluid support, and gradual initiation of enteral feeding. The patient showed marked clinical improvement following surgery, with complete resolution of vomiting and significant improvement in feeding tolerance.

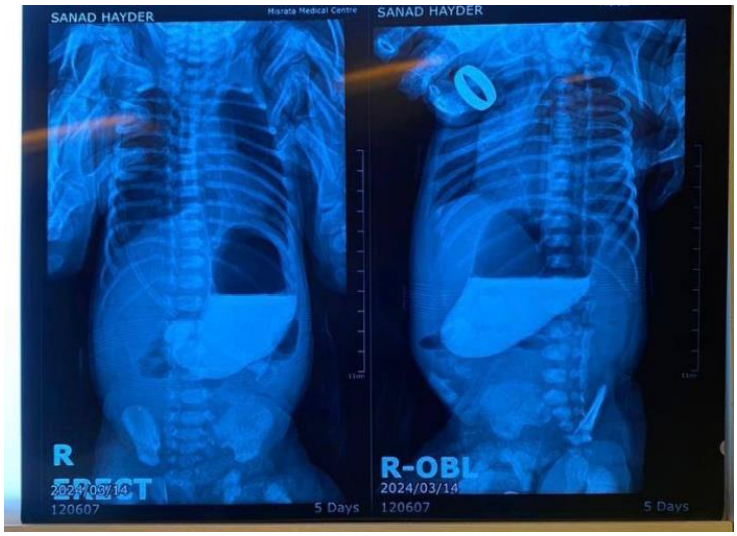


Figure 9 X-ray with contrast, huge dilatation of the stomach, mild dilation of 1st part of the duodenum

At discharge , at 10 days of age, the infant weighed 3.8 kg, and the patient was discharged in good general condition.

This case highlights the importance of early recognition of congenital duodenal obstruction in neonates presenting with persistent vomiting shortly after birth.

FEATURE	CASE 1	CASE 2	CASE3
AGE AT PRESENTATION	19 months	6 months	2 days
PRIMARY SYMPTOM	Projectile non- bilious vomiting, FTT	Persistent vomiting, Melena	Early vomiting, Melena
ASSOCIATED ANOMALIES	P DA**, Recurrent URTIs***	Down Syndrome, Cardiac Septal Defect	None identified
INITIAL MISDIAGNOSIS	Failure to Thrive (Non-specific)	Esophageal dilatation	Inconclusive (Initial facility)
DIAGNOSTIC TOOL	UGI Contrast, CT Abdomen	UGI Contrast Study	X-ray study
INTRAOPERATIVE FINDING	Duodenal Web	Annular Pancreas	Duodenal Web (Type I)
SURGICAL PROCEDURE	Deudenplasty	Deudendeudostomy	Web Excision & Repair
FOLLOW-UP OUTCOME	Thriving, significant weight gain	Resolved vomiting, catch-up growth	Normal feeding, stable weight

Table 1: Clinical Characteristics, Diagnostic Findings, and Management of the Case Series
**FTT: Failure to Thrive **PDA: Patent Ductus Arteriosus ***URTI: Upper Respiratory Tract Infection*

III) Discussion

This case exemplifies the challenging clinical scenario of CRPS development following carpal tunnel release and highlights several important considerations relevant to contemporary orthopaedic practice. The pathogenesis of CRPS remains a subject of ongoing investigation, with current understanding implicating aberrant inflammatory responses, sympathetic nervous system dysfunction, central sensitization, and psychological factors in a complex, multifactorial process. [14,15] Surgical trauma, even in the context of technically successful procedures, may trigger this cascade in susceptible individuals through mechanisms that remain incompletely elucidated.

Congenital duodenal obstruction should remain an important differential diagnosis in infants and children presenting with persistent vomiting, feeding intolerance, growth failure, or unexplained upper gastrointestinal symptoms, even beyond the neonatal period.

Congenital duodenal obstruction is an interesting anomaly, both embryologically and clinically. During the fourth to sixth weeks of intrauterine life, the duodenal lumen is occluded by rapidly proliferating epithelium. The duodenal lumen is then re-established by the twelfth week. Intrinsic duodenal obstruction is thought to result from a failure of recanalization or interruption in duodenal growth [9]. This differentiates congenital duodenal atresia from atresia elsewhere in the bowel, which results from a vascular accident in utero [10].

Duodenal atresia is a congenital intestinal obstruction that can cause bilious or non-bilious vomiting within the first 24 to 38 hours of neonatal life, typically following the first oral feeding [20]

Duodenal atresia occurs in 1 in 5000 to 10,000 live births. It is often associated with other anomalies, including trisomy 21/Down syndrome and cardiac malformations. Approximately 30% to 40% of children with duodenal atresia have Down syndrome. There is a 3% prevalence of congenital duodenal atresia among patients with trisomy 21/Down syndrome. There is no difference in prevalence between the genders. There is an association with VACTERL, annular pancreas, and other bowel atresias, including jejunal atresia, ileal atresia, and rectal atresia.[20]

DO most commonly presents as an isolated anomaly; however, it has also been associated with various other congenital anomalies. Cardiac anomalies and trisomy 21 are the most frequently documented, which both account for almost one-third of patients with DO according to previous studies. The incidence of cardiac anomalies without the presence of trisomy 21 remains unknown, as well as the potential risk factors for cardiac anomalies.

Information about the potential associated cardiac anomalies prior to surgery is essential in patients with DO, as they may have serious (anesthesiological) consequences. It is Important to have full knowledge of the associated cardiac anomalies, since severe cardiac Anomalies can have a major influence on the outcome and care of patients with DO. [23]

A persistent foramen ovale was the most frequent diagnosed cardiac anomaly in patients with DO with an occurrence of 35%, followed by atrial septal defect and ventricular septal defects, which both occurred in 33% of the patients with DO. DO due to complete atresia (type 3) was the most frequent cause of DO and occurred in 54% of the patients. Trisomy 21 was seen in 28% of the patients with DO. In patients with both DO and trisomy 21, the risk of having a cardiac anomaly of 16%, whereas the pooled percentage of cardiac anomalies without presence of trisomy 21 in these patients was 15%. The most common cardiac anomalies in combination with trisomy 21 were ventricular septal defects and persistent ductus arteriosus with an occur-rence of nine percent.[24]

Congenital duodenal obstruction should remain an important differential diagnosis in infants and children presenting with persistent vomiting, feeding intolerance, growth failure, or unexplained upper gastrointestinal symptoms, even beyond the neonatal period.

Aspect	Finding	Clinical message
Prevalence of CHD in DO	~30% of patients	Cardiac anomalies are common, not rare.
Trisomy 21 Association	Present in ~28–31% of DO; OR ~6.5 for CHD	Strongly consider comprehensive cardiac evaluation in DO patients with Down syndrome.
CHD without Trisomy 21	~1/3 of DO patients have CHD regardless of karyotype	Cardiac screening should not be limited to patients with Trisomy 21.
Prognostic Impact	Associated cardiac defects worsen survival (p = 0.004)	Early identification and multidisciplinary management are essential to improve outcomes.

Table 2: Clinical associations and prognostic impact of cardiac anomalies in duodenal obstruction.
**CHD: Congenital heart disease ** OR: Odds ratio ***DO: duodenal obstruction*

The duodenum is temporarily obliterated in the fifth to twelfth weeks of gestation due to epithelial cell proliferation. When the epithelial cells degenerate, vacuolation occurs, and the duodenum is recanalized. If the vacuolation process were to be defective, congenital atresia of the duodenal lumen would occur. [21]

Down syndrome is associated with several congenital anomalies, with cardiovascular and gastrointestinal anomalies being most significant. Duodenal obstruction represents the most common structural anomaly of the gastrointestinal tract in patients with Down syndrome.[25]

The variability in clinical presentation depends on whether the obstruction is complete or partial. Complete obstruction usually presents during the neonatal period with classical bilious vomiting and the radiological “double-bubble” sign,[3] whereas partial obstruction may present later with intermittent vomiting, feeding intolerance, recurrent chest infections, malnutrition, or failure to thrive.[22]

Delayed presentation of congenital duodenal stenosis beyond infancy is uncommon. Most cases of congenital duodenal obstruction are diagnosed in neonates, but partial webs or stenosis may remain undetected due to incomplete blockage.

Recent literature (Chen et al., *J Pediatr Surg Case Rep*, 2023; Kaza et al., *Front Pediatr*, 2022) describes similar cases diagnosed between 12–24 months of age, often misinterpreted as gastro esophageal reflux or feeding intolerance.[14][15]

Melena, although uncommon in congenital duodenal obstruction, initially shifted clinical suspicion toward gastrointestinal bleeding disorders, thereby contributing to delayed diagnosis.

In our case, the coexistence of PDA and recurrent respiratory infections further obscured the diagnosis. Upper GI contrast studies remain the gold standard for confirming obstruction, while CT adds value in defining the anatomical site and severity.

The presence of melena in our patients initially diverted attention toward gastrointestinal bleeding disorders rather than congenital obstruction, further contributing to delayed diagnosis.

Delayed diagnosis in our patients was partly attributed to the nonspecific and intermittent nature of symptoms, which initially mimicked gastro esophageal reflux disease (GERD). Recurrent vomiting and feeding intolerance were therefore managed conservatively before congenital duodenal obstruction was ultimately identified.

Definitive treatment is surgical, with duodenoduodenostomy being the procedure of choice, providing excellent long-term results and minimal postoperative complications.[12]

This case emphasizes the importance of considering congenital causes of chronic vomiting and growth failure in infants and toddlers, even when symptoms appear mild or intermittent.

The treatment of congenital duodenal obstruction is standardised. Duodeno-duodenostomy remains the preferred treatment. Kimura et al. in 1977 described a diamond-shaped duodeno-duodenostomy [12]. None of our patients underwent a diamond-shaped anastomosis, but several reports support this type of anastomosis, as these patients began oral feeding earlier compared to side-to-side duodeno-duodenostomy {8,12}. For those with a duodenal web, we continue to perform duodenal web excision and duodenoplasty.[13]

The spectrum of congenital duodenal obstruction (CDO) includes atresia, stenosis, and annular pancreas. [22] While complete atresia typically presents within hours of birth with the "double-bubble" sign, partial obstructions—like those seen in our 19-month-old and 6-month-old patients—allow for the passage of air and some liquid nutrition, often delaying diagnosis for months or even years Our series illustrates three distinct points on this spectrum. In Case 1, the delay until 19 months led to significant malnutrition and metabolic alkalosis, highlighting that "partial" does not mean "benign.

Case 2 emphasizes the known association between Trisomy 21 and CDO; up to 40% of these patients have associated cardiac anomalies, which were present in both our first and second cases (PDA and VSD).[22]

Diagnostic pitfalls remain a concern. As seen in Case 2, initial ultrasonography may be reported as normal if the duodenum is decompressed at the time of the study. Therefore, an Upper GI contrast study remains the definitive gold standard for visualizing the site and nature of the obstruction.

In resource-limited settings, delayed diagnosis of congenital duodenal obstruction remains a significant challenge due to overlap with common pediatric gastrointestinal disorders and limited access to advanced imaging modalities. Increased clinical awareness is therefore essential to improve early detection and surgical outcomes.

The incidence of congenital duodenal obstruction-related malformations varies [6]. Down syndrome and congenital heart diseases remain the most common malformations associated with congenital duodenal obstruction. The incidence of Down syndrome in our series was 33.3%, which is more than that reported (Ann Saudi Med was 28.6) [11]

Bailey and others reported a 14% reoperation rate in a cohort of 138 infants and children with congenital duodenal obstruction attributed to technical factors, initially unrecognized stenosis, and other unrelated pathologies. In our small series of three patients [7], no reoperations were required for these indications, corresponding to a reoperation rate of 0%.

Our series highlight that Congenital duodenal obstruction should remain an important differential diagnosis in infants and children presenting with persistent vomiting, feeding intolerance, growth failure, or unexplained upper gastrointestinal symptoms, even beyond the neonatal period. early recognition and timely surgical intervention are essential to prevent sever nutritional and metabolic complication.

IV) Conclusions:

This study demonstrates that congenital duodenal obstruction can manifest at variable ages, often presenting beyond infancy when the degree of luminal compromise is incomplete. Clinical presentation is directly dictated by the anatomical nature of the obstruction; while a complete diaphragm without a central opening lead to immediate neonatal symptoms, an incomplete stenosis or a fenestrated web may present with non-specific gastrointestinal symptoms that mimic more common pediatric disorders.

Maintaining a high index of clinical suspicion for persistent vomiting and feeding intolerance is essential to prevent diagnostic delays. Timely radiologic evaluation and precise intraoperative identification of the obstructive pathology are paramount. Ultimately, surgical correction remains the

definitive curative treatment, providing excellent long-term clinical and nutritional outcomes with a highly favorable prognosis.

V) Clinical Pearls:

A) Duodenal atresia is the most common cause of upper gastrointestinal obstruction
B) Duodenal atresia can be diagnosed antenatally.
C) Persistent bilious vomiting and poor weight gain in infants are not always due to reflux.
D) Congenital duodenal stenosis should be considered in prolonged unexplained vomiting.
E) Early imaging can prevent delayed diagnosis and complications.
F) Surgical repair ensures full recovery and normal growth potential.
G) Multidisciplinary collaboration optimizes outcomes in complex pediatric cases.
H) We advocate for routine preoperative echocardiography in all DO patients.

VI) Limitations:

This case series is limited by the small sample size and retrospective nature of data collection. However, it highlights the diagnostic challenges of congenital duodenal obstruction in resource-limited settings.

VII) PATIENT CONSENT AND ETHICAL APPROVAL:

Informed written consent was obtained from the patient's parents for publication of this case report and any accompanying images. Consent was obtained from the patient's guardian. All ethical guidelines for case report publication have been followed, and patient confidentiality has been maintained throughout.

VIII) Conflict of Interest:

The authors declare no conflict of interest.

IX) FUNDING:

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X) ACKNOWLEDGMENTS:

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