

Research Article

Incomplete duodenal obstruction: a rare and late presentation

Almoutaz A. Eltayeb*, Ibrahim A. Ibrahim

Assiut University, Egypt

Received: 27 July 2015

Revised: 30 July 2015

Accepted: 19 August 2015

***Correspondence:**

Dr. Almoutaz A. Eltayeb,

E-mail: almoutazeltayeb@hotmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Incomplete duodenal obstruction accounts for only 2% of all duodenal anomalies. Because of its rarity those patients can be easily misdiagnosed and delayed in their presentation. The aim of this study is to assess these rare conditions with its delayed presentation and their outcome.

Methods: Nineteen cases presented with incomplete duodenal obstruction during the period from January 2010 to January 2015. Cases with complete duodenal obstruction were excluded from the study. None of those nineteen cases were diagnosed antenatal. The assessment parameters were; their provisional preoperative diagnosis, definite post-operative diagnosis, complications related to miss or delayed diagnosis and their final outcome.

Results: All nineteen cases except six presented with persistent or recurrent attacks of vomiting since birth. Their provisional clinical diagnosis was malrotation in 9 cases, duodenal stenosis in 6 cases and atresia in 4. Their final diagnosis was; duodenal stenosis or web in fifteen cases, preduodenal portal vein in two, superior mesenteric artery syndrome in one case and duodenal hematoma in one case. The cause of misdiagnosis was related to lack of experience that leads to unsuccessful exploration or delayed surgical intervention. The post-operative complications occurred in 26.3% of cases. No mortality encountered in this study.

Conclusions: Rare causes of incomplete duodenal obstruction should be looked for routinely to avoid delayed presentation, misdiagnosis and unsuccessful exploration.

Keywords: Incomplete duodenal obstruction, Mucosal web, Duodenal stenosis, Predudodenal portal vein

INTRODUCTION

Congenital duodenal anomalies are rare lesions that originate in the early embryologic development of the foregut with an incidence of 1 to 20000 to 40000.¹ Failure of recanalization of the duodenal lumen during the eighth to tenth week of gestation, results in duodenal atresia. Incomplete recanalization can lead to duodenal stenosis or web and accounts for only 2% of all duodenal anomalies.² Because of its rarity and some of them may be asymptomatic, difficult to diagnose and may be presented later during childhood life. These conditions can be easily misdiagnosed initially with pyloric stenosis, infantile gastro esophageal reflux, metabolic

abnormalities or intracranial pathology due to the recurrent and progressive nature of the vomiting. The aim of this study is to assess these rare conditions of incomplete duodenal obstruction with its misdiagnosis, delayed presentation and outcome.

METHODS

This prospective study was conducted on nineteen cases (12 males and 7 females) presented to Assiut University Children Hospital from January 2010 to January 2015 with persistent vomiting with or without nutritional derangement. Cases with complete duodenal obstruction were excluded from the study. All cases were subjected to routine laboratory investigations, plain X-ray erect to

the abdomen and upper contrast study. The assessment parameters for all cases were; their mode of presentation, provisional preoperative diagnosis, final post-operative diagnosis, frequency of misdiagnosis, any complications related to this misdiagnosis and the outcome of these cases. All patient's parents were consented before any surgical procedure done to their babies or child. Approval of ethical committee of Assiut University faculty of medicine was obtained for conducting this study. All cases were prepared for surgical exploration by preoperative correction of anaemia, hypoproteinaemia and electrolyte disturbance when present.

RESULTS

Antenatal diagnosis was not estimated in any case. Thirteen cases presented with persistent or recurrent attacks of vomiting since birth, while only six cases developed persistent vomiting later on. Their provisional clinical diagnosis was malrotation in 9 cases, duodenal stenosis in 6 cases and atresia in 4. The results of these 19 cases were as follow:

First, duodenal stenosis or mucosal web was present in fifteen cases, where all cases presented by persistent bilious vomiting since birth except four cases had delayed onset of vomiting. The provisional diagnosis of these 15 cases was; malrotation in 7 cases, duodenal stenosis in 5 cases and atresia in 3. On surgical exploration and after mobilization of the duodenum; the site of obstruction was evident by proximal duodenal dilatation and distal collapse after injection of saline through the nasogastric tube. The level of obstruction was determined in the 2nd part of the duodenum in 4 cases and in the 3rd part in 11 cases. A longitudinal incision was made in the proximal duodenum (duodenotomy), where congenital duodenal stenosis was found in nine cases (Figure 1-3) and a mucosal web in six cases (Figure 4). After localization of the ampulla of vater, excision of the mucosal web with over sewn of the resection line, then closure of the duodenum (duodenoplasty) was done (Figure 5 & 6). For cases with duodenal stenosis; side to side duodenoduodenostomy was amenable. Closure of the abdominal wall in layers after insertion of tubal drain was done.



Figure 1: Congenital duodenal stenosis in the third part of duodenum.



Figure 2: Congenital duodenal stenosis in the third part of duodenum.



Figure 3: showing dilated stomach, first and second part of the duodenum with collapsed third part of duodenum in 9 years old boy.

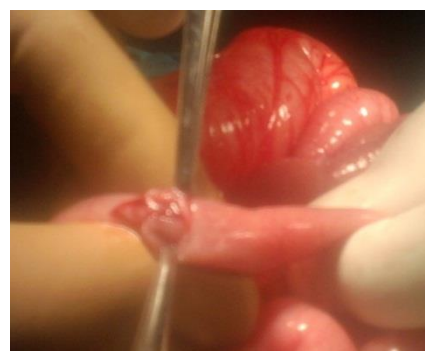


Figure 4: Mucosal web causing duodenal obstruction.

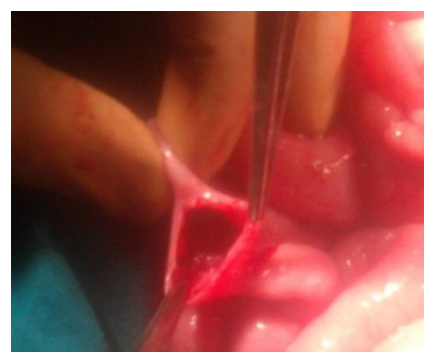


Figure 5: After localization of the ampulla of vater, excision of the mucosal web with over sewn of the resection line.

Table 1: Shows demographic data, onset of vomiting, associated anomalies, nutritional and fluid loss and the post operative complications in the 19 cases.

No.	Age at diagnosis	Weight	Start of persistent vomiting	Ass. anomalies	Nutritional & fluid loss	Post operative complications
1	15months	8250 gm	Delayed onset		Anemia, dehydration & low Alb	Wound infection
2	3 days	2800 gm	Since birth			
3	5 days	3100 gm	Since birth			
4	4 days	2900 gm	Since birth	PDA		
5	16 months	7400 gm	Delayed onset		Anemia, dehydration & low Alb	Wound infection
6	6 days	2450 gm	Since birth	Patent foramen ovale		
7	10 days	2300 gm	Since birth	Down syndrome		
8	11 days	2200 gm	Since birth			
9	14 days	2900 gm	Since birth	Malrotation		
10	12 days	3100 gm	Since birth			
11	22 months	8.800 gm	Delayed onset		Anemia, dehydration & low Alb	Wound infection
12	40 days	3200 gm	Since birth	Malrotation	Anemia& dehydration	
13	2 months	3150 gm	Since birth		Anemia& dehydration	
14	5 months	3650 gm	Since birth		Anemia & dehydration	
15	9 years	12600 gm	Delayed onset		Anemia, dehydration & low Alb	Adhesive intestinal obstruction
16 (PDPV)	3 days	2340 gm	Since birth			
17 (PDPV)	2 months	4200 gm	Since birth	Situs inversus& malrotation	Dehydration	
18 (SMA)	10 years	18400 gm	Delayed onset	Midgut volvulus	Anemia, dehydration & low Alb	Minor anastomotic leak
19 (duodenal hematoma)	6 years	20500 gm	Delayed onset		Anemia	

Alb: albumin, PDPV: preduodenal portal vein and SMA: superior mesenteric artery syndrome.

There were no intra-operative complications apart from minor bleeding from duodenal wall in two cases, stopped spontaneously by compression. The mean operative time was 54 ± 6.1 minutes. All cases started oral feeding on the 3rd to the 4th post-operative day and discharged after removal of the abdominal drain on the 7th to 10th days except three cases developed minor wound infection that responded to conservative measures and discharged on the 12th to 15th post-operative day (Table 1). An attack of adhesive intestinal obstruction occurred in a 9 year old boy with duodenal stenosis (case 15) one month after his

duodeno-duonostomy. He was admitted again to our hospital complaining of recurrent attacks of vomiting (up to seven times per day), abdominal pain and distention. His clinical diagnosis was post-operative adhesive intestinal obstruction that confirmed by surgical exploration, where dense obstructing adhesions over the small intestinal loops was found; necessitating adhesolysis. After that he made good progress until he was discharged on the 10th post-operative day then was followed up for nine months where he gained weight and

did not show any recurrent attacks of intestinal obstruction.



Figure 6: Closure of the duodenum (duodenoplasty) was done.

Two cases out of these fifteen had previous exploration in another hospital; they were misdiagnosed as malrotation. Because both had persistent post-operative symptoms and their post-operative upper contrast study revealed persistent duodenal obstruction. The decision of re-exploration was made, where duodenal stenosis at the 3rd part of the duodenum was detected and side to side duodenoduodenostomy was done with smooth post-operative course.

Second, one full term male (2.340 gm) and 2 months old male (4.230 gm) cases 16 & 17 presented with dehydration, recurrent persistent non bilious vomiting and mild abdominal distension. Plain X-ray to the abdomen revealed a double bubble sign and the upper contrast study showed dilated stomach along with the first part of duodenum (Figure 7-9). Also situs inversus was noted in one of them (Figure 10). Their provisional diagnosis was duodenal atresia in one and malrotation in the other. On surgical exploration both stomach and first part of the duodenum were dilated and the cause of duodenal obstruction found to be a preduodenal portal vein (PDPV) that was abnormally crossing in front of the first part of the duodenum besides the kinked, non-rotated duodenum in the other infant (Figure 11 & 12). After duodenal freeing and mobilization, duodenotomy was done in the dilated duodenum to exclude any luminal cause of obstruction. Loose over bridging side to side duodeno-duodenostomy in front of the portal vein was done. Both cases started oral feeding on the 3rd post-operative day and discharged on the 8th and 9th post-operative day respectively without complications during the follow up period.

Third, a 10 years old female (cases 18) presented with sever weight loss, chronic recurrent abdominal pain, and frequent attacks of bilious vomiting. On clinical examination she was malnourished (weight 15 kg), dehydrated, her hemoglobin count was 7.9 gm and serum albumin 2.2 gm. The upper contrast study showed hugely dilated stomach and duodenum down to the 2nd part with

delayed gastric emptying. Her provisional diagnosis was malrotation of the bowel. On surgical exploration she was found to have Superior Mesenteric Artery syndrome (SMA) obstructing the third part of the duodenum with malrotation and volvulus of the small bowel. After Ladd's procedure a duodenojejunostomy was done. On the 9th post-operative day she developed minor small intestinal leak that stopped spontaneously after 5 days and she was discharged on the 15th post-operative days with smooth follow up period.



Figure 7: A preoperative plain X-ray to the abdomen revealed a double bubble sign.

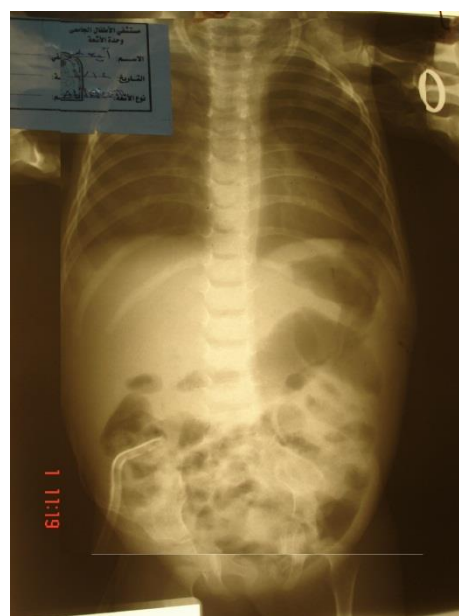


Figure 8: A post operative plain X ray film of the same patient.



Figure 9: Upper contrast study showing dilated stomach and first part of duodenum.

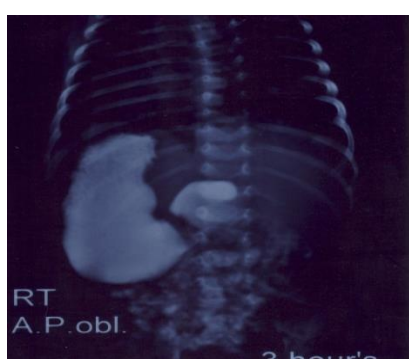


Figure 10: Upper contrast study showing situs inversus, dilated stomach, first part of duodenum with partial duodenal obstruction.



Figure 11: PDPV as a cause of duodenal obstruction.

Fourth, a 6 years old boy (case 19) came to our unit with recent history of recurrent bilious vomiting and dehydration. His laboratory tests were within normal apart from anaemia (HB 8.7 gm). His upper contrast study was suggestive of partial duodenal obstruction. The provisional diagnosis was duodenal stenosis. On surgical exploration there was an unexpected duodenal hematoma at the fourth part of the duodenum and duodenojejunal junction. Side to side duodeno-jejunal anastomosis was achieved. His parents deny any history of abdominal trauma preoperatively but his mother delivered post operatively a history of abdominal trauma since 2 weeks before admission. Both preoperative and post-operative

coagulation parameters were normal. He did not show any post-operative complications during the follow up period.



Figure 12: PDPV & non-rotation as a cause of duodenal obstruction.

Patients in this series were followed up for a period ranged from 6 to 9 months with normal growth curve and without any further complications.

DISCUSSION

Congenital causes of duodenal obstructions may be intrinsic as atresia, stenosis and web or extrinsic as malrotation, Ladd's bands, annular pancreas, duplications and rarely, a preduodenal portal vein. Duodenal atresia can be classified into 3 types (I, II, and III) and could be diagnosed antenatally in up to 50% of cases, by the classic ultrasound signs of polyhydramnios, dilatation of the stomach and proximal duodenum visible on the third trimester. Also "double bubble" sign with gaseous distension of the stomach and proximal duodenum and total absence of intestinal gas distally are diagnostic radiographic signs.³⁻⁵ However if small bowel gas is observed distal to the double bubble, the differential diagnosis should include duodenal stenosis, duodenal web and intestinal malrotation with midgut volvulus; thus congenital duodenal stenosis may be difficult to diagnose in comparison to duodenal atresia.

Recently upper fiber optic flexible endoscopy has been used for evaluation of duodenal obstruction as it allows direct observation of intraluminal causes as; duodenal stenosis, atresia and membrane.^{5,6} In our study three cases were diagnosed by upper endoscopy preoperatively (Figure 13).

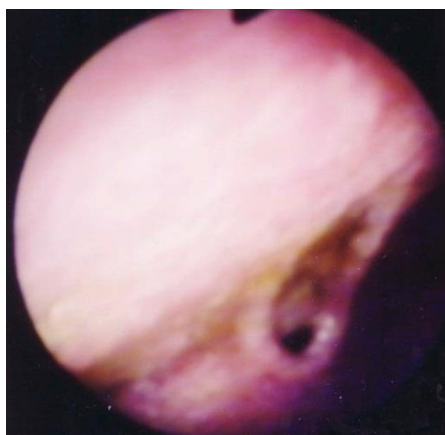


Figure 13: Preoperative endoscopic view of the 9 years old boy with duodenal stenosis showing narrowing of the duodenal lumen.

More than 50% of affected patients have other associated congenital anomalies including variants of gastrointestinal anomalies, renal anomalies, cardiac, central nervous system lesions and rarely, biliary tract anomalies.

Down syndrome is recognized as a possible predisposing factor for gut anomalies and congenital heart disease as part of the VATER syndrome.⁷ The presence of these congenital anomalies may help in the early suspicion or diagnosis of congenital duodenal stenosis. In this study we encountered associated congenital anomalies in seven cases (36.8%) one of them had down syndrome. Another cause of delayed diagnosis may be that those infants and children were passing stool and able to tolerate small feedings due to incomplete nature of obstruction. Since its first description⁸ less than 100 patients of PDPV has been reported in the literature.⁹ PDPV usually associated with other anomalies. Yi et al.¹⁰ reviewed the largest series of PDPV cases yet, with multiple associated anomalies reported including; intestinal malrotation in 64%, situs inversus in 26%, duodenal and pancreatic anomalies in 26% & 22% of them. Prenatal diagnosis or preoperative identification of PDPV as a cause of duodenal obstruction is rarely made even in the cases of duodenal obstruction caused only by the PDPV. 50% of cases of PDPV discovered incidentally during surgery.^{11,12} In this series the diagnosis was not finalized either prenatally or preoperatively and PDPV was incidentally found during operation. This incidental finding should alert the surgeon to be familiar to deal with it to prevent unnecessary trauma during operation with serious outcomes.

Duodenal hematoma is rare, generally occurring after abdominal trauma or endoscopic biopsy or in association with peptic ulcer disease. Duodenal hematoma usually resolves spontaneously within 7-10 days; thus, conservative treatment as first line therapy is recommended instead of surgery. Surgery or percutaneous drainage should be reserved for patients

with persistent obstruction or expanding hematomas.^{13,14} This agrees with our findings where persistent vomiting led to surgical exploration with the incidental finding of duodenal hematoma in the 4th part.

Superior Mesenteric Artery syndrome (SMA) is considered a rare extrinsic cause of obstruction of the third part of the duodenum caused by reduction of the angle between SMA and the aorta.¹⁵ In this study we had a 10 years old female with SMA, malrotation and volvulus. She was suffering from dramatic nutritional loss that lead to significant post-operative morbidity in the form of minor anastomotic leak that stopped spontaneously and the patient survived.

The choice of surgical procedure is largely based on the preference of the surgeon. Duodenotomy with incision or excision of the diaphragm, duodenoduodenostomy, duodenojejunostomy, or duodenoplasty can all be considered as different methods of operative management.⁷

In this study duodenoplasty was performed for cases with duodenal web where the bowel just proximal and distal to the obstruction was joined after excision of the duodenal membrane. Then the duodenum was closed transversely in Heineke-Mikulicz fashion. For duodenal stenosis, PDPV, SMA and duodenal hematoma; side to side duodenoduodenostomy or duodenojejunostomy were performed as we expect that it is the best operative procedure as it is the most direct, physiologic repair and has the least potential for later complications.

In recent years, apart from the above-mentioned open procedures, laparoscopic duodeno-duodenostomy and image-guided balloon dilatation have been tried. Laparoscopic duodeno-duodenostomy has been done in neonates without intra or postoperative complications with early start of oral feeding and rapid recovery leading to short hospital stay.¹⁶⁻¹⁸

The post operative complications rate in previous reports ranged from 12 to 15% of patients with associated mortality rate of 6%.^{5,19-21} In this study the incidence of post operative complications were 26.3%, but we did not lose any patient.

Delayed diagnosis and presentation in this study was evident in 9 cases; 7 of duodenal stenosis or web and in cases 17 & 18. This delayed diagnosis and presentation resulted in nutritional derangement with concomitant anemia in 42.1%, hypoalbuminemia in 26.3%, dehydration and electrolyte disturbance in 47.3%.

Misdiagnosis along the whole series varied from malrotation to duodenal atresia. The cause of misdiagnosis may be related to lack of experience of this rare anomaly by the pediatricians or junior surgeons that lead to unsuccessful exploration or delayed definitive surgical intervention. This was evident in the two cases

that had previous exploration for misdiagnosis of malrotation.

Both mis and delayed diagnosis in this series resulted in prolonged unsuccessful medical treatment, severe nutritional derangement and improper or delayed surgical intervention.

CONCLUSION

Rare causes of incomplete duodenal obstruction could be easily misdiagnosed or present after the neonatal period due to incomplete nature of obstruction and leads to severe nutritional derangements, unsuccessful or delayed surgical intervention.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the ethics committee of Assiut University faculty of medicine

REFERENCES

- Redel CA, Jeff ZR. Anatomy and anomalies of the stomach and duodenum. In: Feldman ME, eds. Sleisenger & Fordtran's Gastrointestinal and Liver Disease. 2nd ed. Philadelphia: W. B. Saunders Co.; 1997.
- Ladd AP, Madura JA. Congenital duodenal anomalies in the adult. Arch Surg. 2001;136(5):576-84.
- Bailey PV, Tracy TF Jr, Connors RH, Mooney DP, Lewis JE, Weber TR. Congenital duodenal obstruction: a 32-year review. J Pediatr Surg. 1993;28:92-5.
- Gray SW, Skandalakis JH. The embryological basis for treatment of congenital defects. In: Gray SW, Skandalakis JH, eds. Embryology for Surgeons. 1st ed. Philadelphia, PA: Saunders; 1972: 177-217.
- Gourevitch A. Duodenal atresia in the newborn. Ann Roy Coll Surg Engl. 1974;148:141.
- De Backer A, Bové T, Vandenplas Y, Peeters S, Deconinck P. Contribution of endoscopy to early diagnosis of hypertrophic pyloric stenosis. J Pediatr Gastroenterol Nutr. 1994;18:78-81.
- Escobar MA, Ladd AP, Grosfeld JL, West KW, Rescorla FJ, Scherer LR 3rd, et al. Duodenal atresia and stenosis: Long-term follow-up over 30 years. J Pediatr Surg. 2004;39(6):867-71.
- Knight HO. An anomalous portal vein with its surgical dangers. Ann Surg. 1921;74:697-9.
- Baglaj M, Gerus S. Preduodenal portal vein, malrotation and high jejunal atresia: a case report. J Pediatr Surg. 2012;47:E27-30.
- Yi SQ, Tanaka S, Tanaka A, Shimokawa T, Ru F, Nakatani T. An extremely rare inversion of the preduodenal portal vein and common bile duct associated with multiple malformations Report of an adult cadaver case with a brief review of the literature. Anat Embryol. 2004;208:87-96.
- Mordehai J, Cohen Z, Kurzbart E, Mares AJ. Preduodenal portal vein causing duodenal obstruction associated with situs inversus, intestinal malrotation and polysplenia: a case report. J Pediatr Surg. 2002;37:E5.
- Choi SO, Taegu PWH. Preduodenal portal vein: a cause of prenatally diagnosed duodenal obstruction. J Pediatr Surg. 1995;30:1521-2.
- Abbas MA, Collins JM, Olden KW. Spontaneous intramural small bowel hematoma: imaging findings and outcome. AJR Am J Roentgenol. 2002;179:1389-94.
- Borsaru AD, Nandurkar D. Intramural duodenal haematoma presenting as a complication after endoscopic biopsy. Australas Radiol. 2007;51:378-80.
- Le Moigne F, Lamboley JL, Vitry T, Stoltz A, Galoo E, Salamand P, et al. Superior mesenteric artery syndrome: a rare etiology of upper intestinal obstruction in adults. Gastroenterol Clin Biol. 2010;34:403-6.
- Kay S, Yoder S, Rothenberg S. Laproscopic duodenoduodenostomy in the neonate. J Pediatr Surg. 2009;44:906-8.
- Van Rijn RR, Van Lienden KP, Fortuna TL, D'Alessandro LC, Connolly B, Chait PG. Membranous duodenal stenosis: initial experience with balloon dilatation in four children. Eur J Radiol. 2006;59:29-32.
- Splide TL, St Peter SD, Keckler SJ, Holcomb GW 3rd, Snyder CL, Ostlie DJ. Open vs. laparoscopic repair of congenital duodenal obstructions: a concurrent series. J Pediatr Surg. 2008;43:1002-5.
- Grosfeld JL, Rescorla FJ. Duodenal atresia and stenosis: Reassessment of treatment and outcome based on antenatal diagnosis, pathologic variance and long-term follow-up. World J Surg. 1993;17(3):301-9.
- Ein SH, Kim PC, Miller HA. The late nonfunctioning duodenal atresia repair--a second look. J Pediatr Surg. 2000;35(5):690-1.
- Okada PJ, Hicks B. Neonatal surgical emergencies. Clin Pediatr Emerg Med. 2002;3(1):3-13.

Cite this article as: Eltayeb AA, Ibrahim IA. Incomplete duodenal obstruction: a rare and late presentation. Int Surg J 2015;2:566-72.