Distal Duodenal Obstruction: a Surgical Enigma

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Abstract The postbulbar segment also known as the distal duodenum is a separate clinical entity in terms of the intrinsic pathologies of this region and its varied anatomical relations. The common bile duct and the pancreatic duct open through the major papilla, which marks the beginning of this segment. Distal duodenal obstruction can be defined as a clinicoradiological entity that is characterized by features of gastric outlet obstruction with recurrent bilious vomiting and a radiological evidence of postbulbar obstruction. A Medline search for distal duodenal obstruction revealed 1409 entries, mostly in the form of case reports. In the last 10 years, 310 cases are reported. Clinical features like abdominal pain, nausea, and vomiting are non-specific and quite similar to gastric outlet obstruction. Clinical findings are also non-specific and do not aid in diagnosis. Laboratory findings also do not add much. Contrast-enhanced CT scan and MR enterography are diagnostic modalities of choice. Distal duodenal obstruction is a clinical entity that has a wide list of differential diagnosis which requires due consideration in terms of its management and follow-up. Most of these lesions require adequate workup with the help of a multidisciplinary team consisting

of radiologists, gastroenterologists, and pathologists to adequately diagnose and stage the disease before a treatment plan is formulated. A thorough knowledge is a must regarding the treatment options available for each of the conditions so that the management can be personalized leading to better results.

Keywords Duodenal obstruction · SMA · Annular pancreas · Postbulbar stricture · Postbulbar stenosis

Introduction

The duodenum or as the name suggests the 12 finger width conduit is a highly guarded segment of the upper gastrointestinal tract that connects the gastric pylorus to the proximal jejunum. It comprises of four different segments that are divided on the basis of their anatomical positions. The postbulbar segment also known as the distal duodenum is a separate clinical entity in terms of the intrinsic pathologies of this region and its varied anatomical relations. It is present in the anterior right pararenal space and is fixed between the posterior peritoneum and the fascia Gerota. The common bile duct and the pancreatic duct open through the major papilla, which marks the beginning of this segment. The segment then courses just anterolateral to the inferior vena cava, posterior to the transverse colon, intimately in relation to the head of pancreas [1].

Distal duodenal obstruction can be defined as a clinicoradiological entity that is characterized by features of gastric outlet obstruction with recurrent bilious vomiting and a radiological evidence of postbulbar obstruction. This entity is well documented in neonates and infants where it occurs due to congenital anomalies at the second and third part of duodenum that represents the embryological foregut and midgut junction.

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A Medline search for human case reports (“Duodenal obstruction”) OR (“duodenal web”) OR (“duodenal stenosis”) OR (“post-bulbar stenosis”) OR (“post-bulbar ulcer”) NOT (urethra) NOT (gastric) reveals 1409 entries out of which 310 are reported in the last 10 years. Though a large number of entities are known to present as distal duodenal obstruction, the physical findings are mostly non-specific and frequently lead to delay in diagnosis. This further can lead to fatal complications due to [2]

- Electrolyte abnormalities
- Gastric perforation
- Gastric pneumatosis and portal venous gas
- Obstructing duodenal bezoar

Also included within this same subset are a group of diseases that present with similar complaints but without any luminal obstruction which are termed as duodenal dysmotility disorder. Diabetes mellitus, scleroderma, chronic idiopathic intestinal pseudo-obstruction, and collagen vascular diseases are commonly implicated underlying conditions in this disorder [3]. However, these are not amenable to surgery and hence out of the scope of this review.

We discuss the clinical features and various diagnostic modalities that are used for assessment of distal duodenal obstruction followed by a discussion over the common etiologies leading to this condition.

Clinical Features

The clinical features of distal duodenal obstruction (DDO) are similar to patients presenting with gastric outlet obstruction. The term gastric outlet obstruction (GOO) is actually a misnomer, because most of these cases are not due to isolated gastric pathology and frequently involve the duodenum as well.

The severity of symptoms depends on the nature of obstruction. Benign cases generally have a long-standing history (years), whereas malignant etiologies have a much more rapid course (months) though this is not the rule [4].

Common clinical features of DDO include

- Epigastric/periumbilical pain
- Nausea/bilious vomiting
- Early satiety
- Weight loss
- Abdominal distention (rarely)

We find a similar clinical presentation in cases of GOO as well, though the presence of bilious vomiting in DDO is more commonly noted. In a series of 49 patients, epigastric pain was noted in 94%, vomiting in 92%, and weight loss in 63% of

cases presenting with GOO, more than 70% of which were of malignant etiology. In another set of patients with benign GOO, early satiety and bloating were the most common features [5, 6].

As mentioned previously, physical examination (abdominal distension, succussion splash, and high-pitched bowel sounds) is non-specific and rarely aids in diagnosis. Sensitivity of succussion splash in documented cases of GOO is reported to be only 48% [7]. Further, it is difficult to differentiate between GOO and DDO based on physical examination alone, though the severity of obstruction can be gauged by general condition of the patient which can help in further deciding the course of management of these patients.

Based on the abovementioned clinical features and physical examination, a diagnosis of GOO/DDO may be suspected which is confirmed by radiologic and endoscopic evaluation. Radiologic investigations generally precede endoscopic evaluation.

Laboratory Evaluation

Laboratory findings are frequently normal or can be non-specifically abnormal. Hyponatremia with or without hypokalemia is the most common electrolyte abnormality that is noted due to bile loss. Anemia may be noted due to chronic blood loss or malabsorption. Specific lab tests like serum amylase and lipase may be elevated in chronic pancreatitis, and serum tumor markers may be elevated in cases of pancreatic malignancy leading to DDO.

Radiological Tests

Radiological tests are generally useful to clinch the diagnosis. Plain radiographs that are normally carried out in emergency setting are generally done to rule out a more sinister pathology like perforation, but it can also reveal dilated gastric bubble with dilated proximal duodenum with paucity of air in the rest of the bowel loops. It can further sometimes suggest the underlying pathogenesis, like a calcified mass in RUQ that has been reported in 25% of cases of Bouveret syndrome [8] or calcifications suggestive of chronic pancreatitis.

Contrast studies have long been used especially in cases of partial obstruction and have been able to delineate the mucosal pathologies, but in the current era of high-performance CT scan and MRI, their usefulness is highly debatable [9].

Multidetector CT has become the imaging modality of choice for duodenal and periduodenal abnormalities. The ability to create isotropic helical data that allows multiplanar reconstructions has an accurate delineation of the anatomy of this region. However, CT remains limited in the ability to differentiate the duodenal wall layers and hence has a low

sensitivity for picking up intramural pathologies [7]. The use of iodinated IV contrast is further beneficial in characterization and staging of these conditions and is routinely administered in this subset of patients.

There is a definite role of MRI/MR enterography in the assessment of duodenal lesions as it offers excellent soft tissue contrast resolution without any ionizing radiations. Rapid breath-hold acquisitions are now being used to reduce artifacts and provide heavily weighted images for assessment of the mucosal enhancement [10].

Endoscopic Evaluation

Upper GI endoscopy is often required to establish the diagnosis; further, it can also permit therapeutic procedures. Routine endoscopic biopsies have been known to have poor sensitivities, especially when the lesion is extramural; however, with addition of endoscopic US, diagnostic capability of this procedure has increased. With this, we are now able to assess the duodenal wall and assess the extent of disease with a high spatial resolution and perform guided biopsies from the same [9] (Table 1).

Discussion

1. Congenital disorders

Duodenal diverticula, duodenal web, malrotation of gut, and annular pancreas are the congenital disorders that can present in adulthood as distal duodenal obstruction.

a. Duodenal diverticula

Duodenal diverticula have been classified as true or pseudo-diverticula depending on where it contains all the layers of the duodenal wall or not. They can be present as an intraluminal lesion as an extramural compression. They are more common in females and are most often asymptomatic. Psathakis in 1994 documented that around 5% of patients harbor duodenal diverticula, 90% of which are asymptomatic [11]. Eggert et al. found that most of these diverticula are present in the periampullary region and they can be a cause of recurrent cholangitis or pancreatitis [12]. Rarely, they have been reported as the cause of obstruction especially when perforation and abscess formation complicate it. The diagnosis is mostly done by CT or incidentally during ERCP. Surgical excision is generally curative, though most techniques require an experienced surgeon.

b. Intraduodenal diverticula

Table 1 Classification of conditions presenting as distal duodenal obstruction

• Congenital
Duodenal duplications/diverticula
Duodenal web
Annular pancreas
Malrotation of gut
• Acquired
Intraluminal
Hematoma
Duodenal bezoars
Luminal
Benign
Crohn's disease
Duodenal tuberculosis
Postbulbar peptic ulcers
Disseminated histoplasmosis
Neoplastic
Mucosal
Duodenal polyps
Duodenal adenocarcinoma
Duodenal lymphoma
Vascular hemangiomas
Intramural
GIST
Lipomas
Hemangioblastomas
Brunner cell hamartoma
Extraluminal
SMA syndrome
Chronic midgut volvulus
Pancreatic pathologies
Pancreatic pseudocysts
Chronic pancreatitis
Benign cystic and solid tumors of pancreas
Periampullary carcinoma
Pancreatic adenocarcinomas
Pancreatic neuroendocrine tumors
Lesions of hepatobiliary system
Hepatic masses
Gallbladder pathologies
Cholelithiasis
Colonic lesions
Retroperitoneal process
Fluid collections
Lymphoma
Primary retroperitoneal tumors

Huang et al. in 1998 reported a case of intraluminal duodenal diverticula that presented itself as obstructive chronic pancreatitis

[13]. Economides et al. further presented a review of this entity in adults in 1977 [14]. It is a rare congenital condition that does not have any gender predilection and presents at the third or fifth decade of life with complaints of dyspepsia and vomiting depending on the completeness of obstruction. It is thought to arise from failure of recanalization that might occur during embryonic period. The intraduodenal diverticulum lies at the medial end of the duodenal wall and is mostly diagnosed on UGI endoscopy or during in CT as an air fluid density in the distal duodenum. Finnies et al. in 1994 have described successful treatment of this entity by endoscopic excision [15].

c. Duodenal webs

There have been reports of congenital duodenal webs presenting itself in adulthood, though most of these are partial obstructions [16].

d. Duodenal duplications

Duodenal duplications are rare, and 12% of all the GI duplication anomalies are known to occur in this region. It most often arises from the medial wall from the second or third part of the duodenum. They normally do not communicate with the duodenal lumen and are often asymptomatic. However, carcinoma can arise from the duplication cyst, which may present itself as duodenal obstruction [17]. Most of these lesions require triple-contrast CT for diagnosis, and surgical excision with proximal gastric bypass is the only possible treatment option.

e. Malrotation of gut

Due to differential growth of duodenal wall, the duodenum takes a c-shaped turn so that the fourth part of duodenum comes to lie left of the midline. This occurs during the sixth week of intrauterine period. When this fails, the duodenum comes to lie on the right of the midline and the colon overrides it [18]. Malrotation can easily be diagnosed on CT by studying the anatomic relationship of the duodenum with the SMA and the aorta. Patients with malrotation can present with acute or chronic obstruction due to the congenital bands, which eventually require surgical excision.

f. Annular pancreas

Annular pancreas has a varied presentation in adulthood [19]. More than 50% of cases are asymptomatic, mostly detected incidentally when investigated for some other diseases, most of the remaining present with features of pancreatitis (Fig. 1). Rarely, these patients present with complaints of nausea or bilious vomiting that are suggestive of an obstructive lesion in adulthood. Sandrasegaran et al. in 2009 have

Fig. 1 Congenital lesions affecting duodenum. CT scan showing annular pancreas engulfing the duodenum causing obstruction at that segment

reported a multitude of presentations of annular pancreas in adults [20]. They have classified annular pancreas into complete and incomplete variety. The etiopathogenesis of this disease lies in the malrotation or non-rotation of the ventral pancreatic buds leading to a circumferential bridge of pancreatic tissue at the second and third part of duodenum. MRCP commonly shows the encircling pancreatic duct that is the characteristic lesion of this anomaly. Treatment involves duodenoduodenostomy or Billroth II gastrectomy.

2. Duodenal lesions

These lesions tend to present as either intraluminal obstruction or as stricture/stenosis. It can occur due to myriad of causes from benign to malignant. The most common etiologies that present as intraluminal obstruction are bezoars and duodenal hematomas, whereas postbulbar peptic ulcer disease, duodenal tuberculosis, and Crohn's disease may present as luminal pathology like a stricture or stenosis.

a. Duodenal bezoars

Bezoars are partially digested or undigested foreign material in the GI tract that is known to cause obstruction that may be partial or complete in nature. Duodenal bezoars have been an uncommon cause of mechanical obstruction but nonetheless have been frequently reported in the literature by the virtue of its rare presentation [21]. Development of bezoars is generally related to some predisposing condition that either causes duodenal dysmotility or narrows the lumen for the bezoar formation to initiate. A variety of materials have been implicated from hair, food to date seed, a list that can never be too exhaustive [22]. Diagnosis is easy by endoscopic visualization, which can be therapeutic as well, though in few cases, surgical removal is warranted.

b. Duodenal hematoma

These most commonly result after blunt abdominal trauma, due to its fixed position in the retroperitoneum. It occurs due to mechanical compression forces that cause the submucosal plexus of vessels to burst when the duodenum compress with the stiff vertebra behind its [23]. CT is the preferred modality of diagnosing duodenal hematoma, which also helps in delineating other injuries if present. The treatment is mostly non-operative with nasogastric decompression till the hematoma resolves although complete transection of the second part or associated pancreatic injuries warrant an immediate surgical intervention [24].

c. Duodenal tuberculosis

The incidence of gastroduodenal tuberculosis is very low in the present literature, and even rare is the incidence of primary duodenal tuberculosis [25]. The overall incidence is quoted to be around 0.5% of all GOO cases [26]. Most of these cases are initially treated as intractable peptic ulcers or early malignant thickening of the duodenal wall. But lack of response to anti-*Helicobacter pylori* treatment and negative repeated biopsies for evidence of malignancy in an endemic region should raise a suspicion of this otherwise well-documented entity (Fig. 2a, b). The treatment consists of a multidrug regimen for abdominal tuberculosis with good results, and in intractable cases, bypass procedures can be planned without the need of radical dissections [27].

d. Crohn's disease

Crohn's disease is known to affect any part of the GI system though its cause is yet unknown. Isolated duodenal affection is rare (Fig. 2c) but is seen in 5–20% of patients with colonic and small bowel involvement [28]. Clinical manifestation is dictated by the degree of stenosis or stricture that develops, though a degree of reversible spasms may co-exist. These patients mostly present with partial obstruction except those due to adhesions that present with acute intestinal obstruction [29]. The initial management consists of hydration, nasogastric decompression, and parenteral nutrition with successful response within 24–48 h. Parenteral glucocorticoids or infliximab can be given to those that are not responding to the conservative management [30]. Surgery is considered only as the final resort, but it may be indicated early in cases of small bowel ischemia.

e. Postbulbar peptic ulcer disease

Duodenal ulcers are known to affect adults of all ages and are mostly benign. They account 10% of all ulcerations in this

region; most of them located just above the ampulla of Vater [31]. Clinically, most of these patients present with epigastric pain, occurring after few hours of taking the meal. Duodenal obstruction can result from spasm and subsequent edema that develops in the acute phase and from strictures in the chronic (Fig. 2d). Most of these ulcers are caused due to *H. pylori* infection, the treatment of which is by antibiotics and proton pump inhibitors (PPI); surgery is rarely indicated. On the contrary, the presence of multiple ulcers or an underlying mass is suggestive of a much sinister pathology. Multiple ulcers can indicate CMV infection, lymphoma, or gastrinoma all of which can mimic peptic ulcers. On CT, ulcerations can be seen as duodenal wall thickening with periduodenal fat stranding. Surgery is only recommended when etiological diagnosis cannot be made or when conservative management with PPI fails [32].

f. Neoplastic lesions of the duodenum

Duodenal polyps are the most common benign neoplasms in this region. They are usually less than 2 cm and are usually asymptomatic [33]. Though rarely, they can attain sizes that can obstruct the duodenal lumen. They can occur as solitary lesions or as part of diffuse polyposis syndrome. Adenomatous polyps are an important subtype as they have the propensity for malignant transformation. These polyps are composed mainly of dysplastic epithelium and can be classified as tubular, villous, or tubulovillous. Villous polyps are considered premalignant, and they have a characteristic soap-bubble appearance on CT or contrast studies [34]. Hemangiomatic polyps (can be a part of the Osler-Weber-Rendu disease) have also been reported though they are primarily mucosal lesions and rarely lead to duodenal obstruction.

Adenocarcinoma accounts for about 30% of small bowel cancers. They usually present in the sixth or seventh decade and are slightly more prevalent in female population [34]. Crohn's disease, HNPCC, and FAP are known risk factors. The incidence is highest in the duodenum and decreases progressively along the length of the small bowel. Patients with advanced adenocarcinoma frequently present with DDO and with other features like epigastric pain and weight loss. On CT, they appear as polypoidal masses or ulcerative lesions distal to the second part of duodenum (Fig. 3). Treatment is mainly surgical either for palliation or cure [35].

Neuroendocrine tumors are another set of tumor that has a characteristic morphological appearance with the ability to produce biologically active amine precursors. The most common of these are gastrinoma that is associated with excess gastrin production. Though the majority is localized in the pancreas, around 15% are located in the duodenum that forms the part of the so-called gastrinoma triangle [36].

Most of the intramural lesions arise from the submucosa. From a wide variety of tumor that may originate from this

Fig. 2 Benign lesions of duodenal wall. **a** Benign duodenal lesion, probably tubercular. **b** Duodenal wall thickening, tubercular in view of necrotic lymph nodes. **c** Benign duodenal lesion; biopsy come to be Crohn's disease. **d** Benign duodenal stricture

region, gastrointestinal stromal tumor (GIST) constitutes 90% of them. They are mostly asymptomatic but can grow into

Fig. 3 Malignant lesions of duodenum. CT scan showing irregular duodenal thickening with infiltration into surrounding structures, s/o adenocarcinoma of duodenum

large proportions presenting with DDO. They may be endoduodenal, exoduodenal, or having a dual extension. As these lesions grow, they tend to outgrow their vasculature that leads to hypoxic necrosis in the central part, which can be characteristically noted, on the imaging modalities. The presence of multiple such lesions may be indicative of a metastatic process or lymphoma, as GISTs are rarely multifocal. Ten percent of these masses may harbor a malignant potential, and hence, excision is warranted even if asymptomatic at the time of diagnosis. Depending on the size and extent, the treatment can be through endoscopic approach or through a surgical laparotomy [34, 37].

Other rare etiologies include duodenal leiomyomas, duodenal lipomas, lymphangioma, duodenal ectopic pancreatic rests, and Brunner gland hamartomas that can present as DDO and require surgical treatment either for cure or palliation [38].

3. Extraduodenal compression

a. SMA syndrome

It is a rare cause of proximal intestinal obstruction and has been referred as variety of eponyms including Wilkie syndrome, cast syndrome, arteriomesentric duodenal obstruction, and chronic duodenal ileus [39].

This syndrome is characterized by compression of the third part of duodenum due to abnormally acute angle between the SMA and the aorta with the loss of intervening fat pad (Fig. 4).

Fig. 4 Extrinsic compression. CT scan showing compression of third part of duodenum due to SMA syndrome

The normal angle of 30°–60° is reduced to 6° with the reduction in aortomesenteric distance from 10 to 28 to 2 mm [40].

It is commonly associated with debilitating illnesses like AIDS [41] and other conditions with extreme weight loss (following bariatric surgery [42], paraplegia [43], prolonged bed rest, and drug abuse [44]). Rarely, it may be due to a congenitally shortened ligament of Treitz. This genetic predilection is further suggested by a couple of case reports showing the presence of similar condition in identical twin [45] and in utero diagnosis in another [46]. There are several surgical options for the treatment of superior mesenteric artery syndrome if conservative nutritional therapy fails to resolve the condition. These include Strong's procedure, gastrojejunostomy, and duodenojejunostomy with or without division or resection of the fourth part of the duodenum [47].

b. Pancreatic pathologies

Acute pancreatitis, chronic pancreatitis [48], pancreatic pseudocysts [49], and pancreatic neoplasms are the possible pancreatic pathologies that can cause duodenal obstruction.

Groove pancreatitis [50], a form of segmental pancreatitis that occurs within the pancreaticoduodenal groove, can lead to acute fluid collection and inflammation, which can cause

duodenal/bile duct stenosis in 50% of such cases. The presence of pancreatic enzymes not only can cause duodenal scarring but can also lead to intramural hemorrhage due to injury to duodenal vasculature. CT images show peripancreatic fat stranding and periduodenal edema, showing an increased duodenal sweep. Most of these patients are managed conservatively though some cases may require endoscopic drainage of acute fluid collections. Surgery is generally withheld for later stage, when a gastrojejunostomy with or without choledochenterostomy is planned.

Pancreatic acute fluid collections get consolidated to pseudocysts within 4–6 weeks which may then present as masses compressing the adjacent structures which in this case is the duodenum; the management is generally some kind of drainage procedure whether percutaneous, endoscopic, or surgical.

Pancreatic neoplasms that can cause duodenal obstruction can be divided into benign cystic and solid pancreatic lesions and malignant pancreatic adenocarcinomas, periampullary masses, and pancreatic neuroendocrine tumors. Lesions with an infiltrative pattern are known to involve the duodenum early and can present as duodenal obstruction early in the disease natural history. Triple-contrast CT is the investigation of choice that is used to identify the various etiologies, with periampullary masses normally showing up as hypoattenuating lesions or neuroendocrine masses which light up as hypervascular and hyperintense images. Treatment of most of these lesions consists of multiple modalities, including surgery, chemotherapy, and radiotherapy [51]. Prognosis greatly varies within the various subtypes, and hence, characterization of the lesion is of utmost importance.

c. Lesions of hepatobiliary system

Gallbladder pathologies, especially GB adenocarcinoma, commonly presents as DDO with its ability to rapidly infiltrate the surrounding structures [52]. Other benign pathologies like the mucocele of GB [53], GB hypokinesia [54], and the Bouveret syndrome that has also been described (in which gallstone burrows into the duodenum forming a cholecystoduodenal fistula) are implicated into causing features of intestinal obstruction. The preferred mode of treatment is surgical though endoscopic palliation is warranted in few cases.

d. Retroperitoneal process

The specific retroperitoneal position of duodenum predisposes it to compression, displacement, and obstruction due to a myriad of retroperitoneal disorders. The list includes inflammatory or infective etiologies, cystic or solid masses having a genitourinary origin, hemorrhage, or primary retroperitoneal tumors [54].

Lymphoma, which is a common retroperitoneal tumor, originating from the retroperitoneal lymphatic tissues can cause duodenal lumen to get compressed, but strictures

are rare. Primary retroperitoneal tumors like liposarcoma (40%), leiomyosarcoma (30%), and malignant fibrous histiocytoma (15%) are known to reach up to large proportions with the ability to cause DDO [55]. The treatment is mostly surgical excision of tumor, and radiotherapy is required in few selected cases.

Conclusion

Distal duodenal obstruction is a clinical entity that has a wide list of differential diagnosis which requires due consideration in terms of its management and follow-up. Lack of specific physical signs precludes differentiation based on clinical characteristics. Most of these lesions require adequate workup with the help of a multidisciplinary team consisting of radiologists, gastroenterologists, and pathologists to adequately diagnose and stage the disease before a treatment plan is formulated. A thorough knowledge is a must regarding the treatment options available for each of the abovementioned conditions so that the management can be personalized leading to better results.

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Author 2 (PG)—Literature review and drafting of manuscript.

Author 3 (RK)—Mentor author, critical revision of data, and final approval of manuscript.

Author 4 (DD)—Literature review, patient data acquisition (radiographic images), and drafting of manuscript.

Compliance with Ethical Standards

Conflict of Interest The authors declare that they have no conflict of interest.

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