E-ISSN: 2708-1508
P-ISSN: 2708-1494
IJCRS 2024; 6(1): 18-20
www.casereportsofsurgery.com
Received: 16-01-2024
Accepted: 19-02-2024

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Obstruction secondary to preduodenal portal vein and a jejunal web

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DOI: https://doi.org/10.22271/27081494.2024.v6.i1a.93

Abstract
Preduodenal portal vein (PDPV) is a rare congenital anomaly and accounts for a small portion of duodenal obstructions. A PDPV can be associated with malrotation, duodenal webs, and annular pancreas. Our case is of a 1-month-old female born at 38 weeks and 6 days with Sickle Cell Disease who presented for intermittent vomiting with associated weight loss and failure to thrive secondary to feeding intolerance. Ultrasound (US) demonstrated a pyloric channel measuring 17 mm and an anterior muscle wall thickness of 4mm, concerning for pyloric stenosis; however, gastric contents were able to pass into a dilated duodenal bulb. An upper gastrointestinal series (UGI) showed malrotation. The patient was taken to the operating room (OR) for laparoscopy with Ladd's procedure and a preduodenal portal vein was identified. A laparoscopic duodenoduodenostomy was performed to bypass the preduodenal portal vein. The patient represented 2 months postoperatively for failure to thrive secondary to recurrent feeding difficulty. UGI was repeated and demonstrated distention of the proximal duodenum with hyperperistalsis and minimal emptying suggestive of a parietal obstruction in the duodenum. Esophagogastroduodenoscopy (EGD) was performed, and a scope was not able to pass through the duodenoduodenostomy for presumed stricture. The patient was taken to the OR for exploratory laparotomy with intraoperative EGD and a jejunal web was identified distal to the duodenoduodenostomy anastomosis. A side-to-side jejunojejunostomy was performed to bypass the web. In our review of literature, the jejunum is an uncommon site of intestinal webs and rarely associated with PDPV. Our case is one of a few identified cases of preduodenal portal vein associated with jejunal atresia.

Keywords: Obstruction secondary, preduodenal portal vein, jejunal web

Introduction
Preduodenal portal vein (PDPV) was first described by Knight in 1921 [3]. PDPV is a rare anomaly presenting an abnormal developmental vascular course, where an anteriorly placed portal vein results from the embryonic mal development of the portal venous system [1]. PDPV can be asymptomatic but various clinical presentations and co-existing conditions can be present, the most common being duodenal obstruction [7]. Symptomatic duodenal obstruction occurs in approximately 50% of patients with this anomaly. PDPV is frequently seen with coexisting anomalies such as duodenal web, duodenal atresia, malrotation, and annular pancreas [8]. Our review of literature has not found a case of PDPV associated with jejunal webs. Here we present a case of Preduodenal portal vein associated with jejunal web.

Case Presentation
A one-month-old female born full-term with Sickle Cell Disease was referred to the Pediatric Surgery service after presenting to the emergency department with nonbilious non-bloody emesis since birth and was admitted for failure to thrive as she had an associated 15.7% weight loss secondary to feeding intolerance. When she was born, she had spent several days in the NICU due to feeding difficulties which resolved shortly after birth. Two abdominal ultrasounds for pyloric stenosis were equivocal as the anterior muscle wall was thickened, but gastric contents were able to pass through the pyloric channel. Initial US demonstrated a pyloric channel measuring 17 mm and an anterior muscle wall thickness of 4mm, concerning for pyloric stenosis; however, gastric contents were able to pass into a dilated duodenal bulb. An UGI series showed malrotation without volvulus. The patient was having bowel movements and was having intermittent episodes of emesis. Given clinical findings and imaging results, the patient was taken for a diagnostic laparoscopy. On the index operation, the patient was found to have intestinal malrotation without volvulus

and a preduodenal portal vein was found to be traversing the second portion of the duodenum with evidence of obstruction as there was dilation of the duodenum proximal to the preduodenal portal vein. A Ladd's procedure was completed along with a duodenal-duodenostomy in a side-to-side fashion to bypass the preduodenal portal vein. Post-operatively, the patient was NPO with a nasogastric tube to low intermittent suction with TPN for several days. An UGI series showed no leak or obstruction and the patient was started on breast milk. Her post-operative course was complicated by difficulty with adequate weight gain. On post-operative day nine the patient developed intermittent episodes of non-bilious non-bloody emesis which prompted a KUB that was negative for obstruction. The emesis was thought to be attributed to formula and a transition to only breast milk resulted in improvement in emesis and adequate weight gain. The patient was able to be discharged after several days of adequate weight gain.

The patient was admitted to the hospital at the age of three months for failure to thrive secondary to feeding intolerance again. The patient was having a progressive increase in episodes of emesis and we were consulted for concern of bowel obstruction. An UGI series was repeated and demonstrated distention of the proximal duodenum with hyperperistalsis and minimal emptying suggestive of a parietal obstruction in the duodenum. Esophagogastroduodenoscopy (EGD) was performed, and an endoscope was not able to pass through the duodenoduodenostomy for presumed stricture. The patient was taken to the OR for exploratory laparotomy and multiple adhesions were found to be possibly causing an obstruction at the site of the duodenoduodenostomy. These adhesions were lysed and an intra-operative EGD was then performed, and the scope was able to be passed through the anastomosis, but we were unable to visualize distally, although the distal small bowel was insufflating with air. We then decided to widen the anastomosis, which was opened, and when a stapler was attempted to be passed distally, we encountered an obstruction and a jejunal web in the proximal jejunum was identified distal to the duodenal-duodenostomy anastomosis. A side-to-side jejunojejunostomy was performed to bypass the web. A red rubber was then passed through the jejunojejunostomy, and no further obstructions were found. Post-operatively there were no complications.

In outpatient follow-up with Pediatric Surgery two weeks after discharge, the patient continued to be doing well as she was eating without excessive emesis and gaining adequate weight.

Discussion

Preduodenal portal vein (PDPV) is a rare anomaly presenting an abnormal developmental vascular course, where an anteriorly placed portal vein results from the embryonic mal development of the portal venous system [1]. PDPV was first described by Knight in 1921 [3]. Since the first report concerning this congenital disorder in 1921 up until 2012, less than 100 cases had been described in the literature [1, 5]. More recently, Yi *et al.* reviewed a large series of PDPV cases and found 323 reported cases of PDPV with multiple associated anomalies including intestinal malrotation (in 64%), situs inversus (in 26%), duodenal and pancreatic anomalies (in 26% and 22%, respectively) [2]. The true incidence of PDPV cannot be precisely estimated, as some patients do not produce clinical symptoms [5].

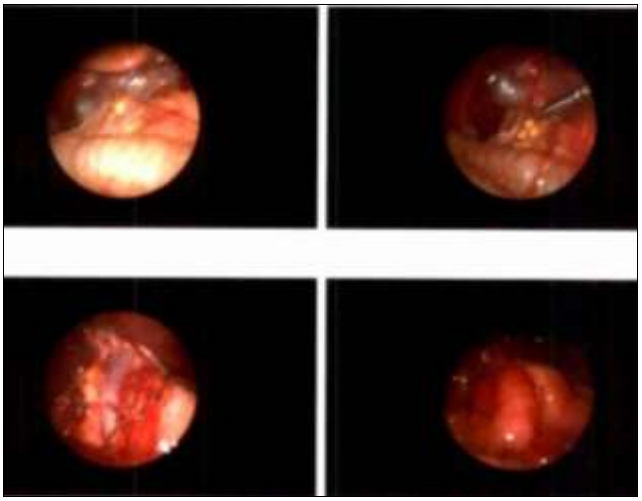
PDPV can be explained as an embryonic anomaly resulting from a persistent caudal anastomosis between the vitelline veins [1]. Normally, the two parallel vitelline veins are joined by three interconnecting veins; the cephalad branch within the liver, the middle branch posterior to duodenum, and the caudal branch anterior to duodenum. Later in development, the caudal and the cephalad anastomosis, with the caudal part of the right vitelline vein and the cephalad part of the left vitelline vein, disappear, leaving an S-shaped portal vein passing behind the duodenum [4]. A variation in this process, in which the middle and the cephalad anastomosis disappear together with the left vitelline vein, accounts for the PDPV [4].

Being an early embryonic event, PDPV is very rarely an isolated defect [5]. In reviews of literature, Kim *et al*. and Srivastava *et al*. observed that PDPV is typically associated with multiple congenital anomalies, including heterotaxia or polysplenia syndrome, situs inversus, cardiac defects, malrotation, biliary or duodenal atresia, malrotation, duodenal webs, and annular pancreas [1, 2]. A majority of patients with PDPV are asymptomatic but various clinical presentations and co-existing conditions can be present with the most common being duodenal obstruction [7]. Clinically, symptomatic duodenal obstruction occurs in approximately 50% of patients with this anomaly, caused by itself or coexisting anomalies such as duodenal web, duodenal atresia, malrotation, and annular pancreas [8]. In the remaining 50% of asymptomatic patients, PDPV is typically an incidental finding during surgery [1].

In our case, a PDPV was associated with malrotation and a jejunal web. Individually, these anomalies have low incidence rates, but do have an association. As previously stated, a PDPV is not always the cause of symptomatic duodenal obstruction and in our case was not the only contributing factor to obstructive symptoms seen in this patient. Having three congenital anomalies along with intermittent obstructive symptoms made the clinical course difficult to navigate. It seemed that the PDPV was the cause of obstruction as the patient had improvement in symptoms immediately post-operatively and due to this transient palliation in symptoms, it was likely that the PDPV was partially contributing to obstructive symptoms. When the patient was admitted a second time for failure to thrive there was low suspicion for a third anomaly as a cause for obstruction had been previously identified and more common causes of obstruction were pursued.

The jejunum is the location of 8% of intestinal webs [6]. Identifying a jejunal web would have required a high index of suspicion, but passing something such as a red rubber catheter can help identify distal obstructions intra-operatively. However, doing so would have required an exploratory laparotomy and our index case was done via laparoscopy. Another consideration is intra-operative endoscopy if laparoscopy is performed in these situations.

Because of the true nature of vascular anomaly and its associated abnormalities, PDPV remains almost exclusively a pediatric surgical condition [5]. It is of utmost importance for the surgeon to recognize this congenital anomaly. PDPV, when unrecognized, is at risk to injury later in life during surgery in the vicinity such as during cholecystectomy, gastrectomy, portoenterostomy, and pancreatectomy [2].



Case Pictures

Conclusions

PDPV is a rare anomaly presenting an abnormal developmental vascular course. When any congenital anomaly is found, other associated anomalies should be ruled out. These anomalies can be difficult to identify if the surgeon does not already have a high index of suspicion. Some anomalies can only be found by exploratory surgery and these associations can be vast. If found in the operating room, imaging modalities to identify associated anomalies are limited. Since the PDPV is rarely a source of obstruction, intestinal webs should be taken into consideration as a cause. More research should go into identifying PDPV as a potential cause of intestinal obstruction.

Conflict of Interest

Not available

Financial Support

Not available

Reference

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How to Cite This Article
Shin C, Cobb W, Bernshteyn A. Obstruction secondary to preduodenal portal vein and a jejunal web. International Journal of Case Reports in Surgery. 2024;6(1):18-20