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A Rare Cause of Congenital Duodenal Obstruction: Preduodenal Portal Vein

Nadir Görülen bir Konjenital Duodenal Obstrüksiyon Nedeni: Preduodenal Portal Ven

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Abstract

PREDUODENAL portal vein (PDPV) is a rare cause of duodenal obstruction. While half of PDPV cases present with obstructive findings in the neonatal period, the other half are diagnosed incidentally at advanced ages. In half of the obstructed PDPV cases, the cause of obstruction is another congenital anomaly. In our case, the aberrant ventral extension of the pancreas with PDPV caused incomplete duodenal obstruction by compressing the duodenum externally.

Keywords: Preduodenal portal vein, Congenital duodenal obstruction, Duodenoduodenostomy, Malrotation.

Özet

PREDUODENAL portal ven (PDPV), duodenal tıkanıklığın nadir bir nedenidir. Yenidoğan döneminde PDPV olgularının yarısı obstrüktif bulgularla ortaya çıkarken, diğer yarısı ileri yaşlarda rastlantısal olarak teşhis edilmektedir. Tıkanmış PDPV vakalarının yarısında, tıkanıklığın nedeni başka bir konjenital anomalidir. Bizim olgumuzda PDPV'ye eşlik eden pankreasın anormal ventral uzantısı duodenuma dıştan bası yaparak inkomplet tıkanıklığa neden olmuştu.

Anahtar Kelimeler: Preduodenal portal ven, Konjenital duodenum obstrüksiyonu, Duodenoduodenostomi, Malrotasyon.

Introduction

PREDUODENAL portal vein (PDPV) is a very rare anomaly causing duodenal obstruction in the neonatal period and was first described by Knight in 1921 [1-3]. Pathology occurs as a result of the persistence of the vitelline vein that passes in front of the duodenum and obliteration of the branch that passes behind the duodenum [1]. Duodenal incomplete obstruction findings are present in the diagnosis. The definitive diagnosis is usually made intra-operatively [4]. There is usually a congenital anomaly that causes the main obstruction [5-7].

Case Report

A 17-day-old female patient, 2650 gr on term, was admitted to the emergency department with non-bilious vomiting since birth. The patient had electrolyte imbalance secondary to vomiting, metabolic alkalosis, and decreased turgor and tone. On abdominal X-ray, the stomach was enlarged and there were gas shadows only at the left side. Ultrasonography also showed that the first part of the duodenum was enlarged and the transition to the second part was quite slow. After correcting the metabolic alkalosis, the patient was operated with a prediagnosis of duodenal incomplete obstruction. The cecum was mobile, but there were no Ladd bands compressing the duodenum. On the first and second parts of the duodenum, there was a PDPV suppressing the duodenal passage and aberrant ventral extension of the pancreas ([Figure-1](#)). There was no annular pancreas, but it was observed that the distal duodenum was slightly narrowed due to compression of the portal vein and anterior pancreatic tissue. After the catheter air inflated the first part of the duodenum significantly, it was observed that the air slowly passed distally. The patient underwent duodenoduodenostomy anterior to the portal vein ([Figure 2](#)). Postoperatively, she was fed parenterally with total parenteral nutrition. On the 7th day, the nasogastric catheter was removed and oral feeding began. The patient was discharged on the 12th postoperative day without any problem.

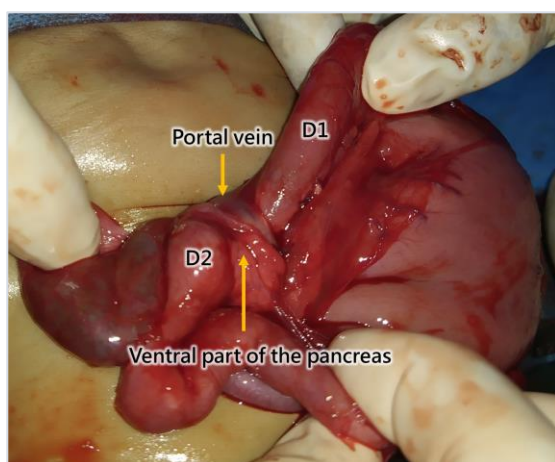


Figure 1: PDPV and the aberrant ventral extension of the pancreas compresses between the first (D1) and second parts of the duodenum (D2).

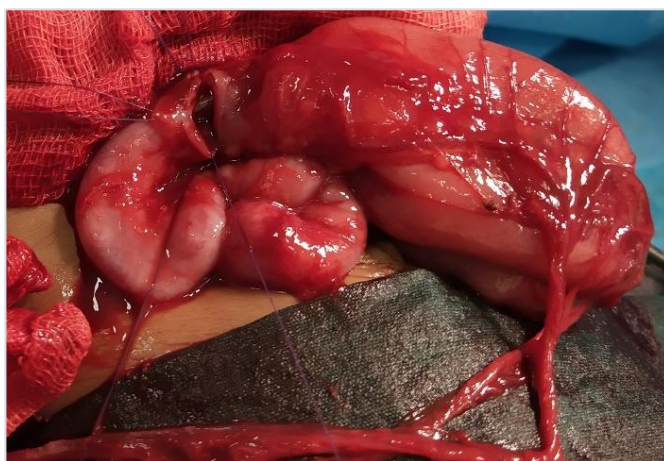


Figure 2: Duodenoduodenostomy anastomosis.

Discussion

Congenital duodenal obstructions are responsible for 50% of intestinal obstructions in the neonatal period with an incidence of 1:2.500-10.000 [8-10]. The causes of obstruction are extrinsic (Ladd bands, annular pancreas, preduodenal portal vein) or intrinsic (atresia, stenosis, web) causes [10-12]. PDPV is responsible for 4% of duodenal obstructions [3]. PDPV presents with obstruction during neonatal and infant period, while half is detected incidentally in advanced ages without any symptoms [13]. In half of PDPV cases presenting with obstruction, there is an additional extrinsic or intrinsic anomaly that causes obstruction [14,15]. In our case, the extension of the ventral part of the pancreas with PDPV was compressing the duodenum. PDPV is often accompanied by cardiovascular, gastrointestinal, and urinary system abnormalities [5-7,16]. Malrotation is associated with %31-54 of the cases [9,14]. In our case, the cecum was mobile but there were no Ladd bands that pressed on the duodenum. The diagnosis of preop is difficult, because PDPV does not have a specific marker. In the antenatal period, a history of polyhydramnios, biliary or non-biliary vomiting after birth, and double bubble appearance on direct radiography suggest a duodenal obstruction. The diagnosis of PDPV is usually made intraoperatively [4]. Treatment of obstructive PDPV, as we do, is duodenoduodenostomy [17]. In cases where this is not

possible, gastroduodenostomy or gastrojejunostomy are among the methods. In the literature, duodenoduodenostomy was not performed in cases where portal vein did not obstruct duodenum [4,18]. Obstruction findings were not observed in the follow-up of these patients [4,18]. PDPV should be considered in cases of incomplete duodenal obstruction to avoid portal vein injury, especially during Ladd band excision. Likewise, the possibility of encountering an asymptomatic PDPV should not be ignored in patients who are scheduled for operation for cholecystectomy, biliary atresia, and liver transplantation [19,20].

Declaration of interest: The authors declare no conflict of interest and alone is responsible for the content and writing of the paper.

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