Case Report

Recurrent Non-bilious Vomiting in a Child: A Case Report of an Uncommon Diagnosis

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Abstract

Annular pancreas (AP) is a congenital anomaly and a recognised cause of duodenal obstruction which can affect all age groups. It may manifest early in the neonatal period, but it may also have varied and often delayed symptoms. We report the case of AP in an 18-month-old girl with a long history of recurrent post-prandial non-bilious vomiting treated for a long time with the clinical diagnosis of gastroesophageal reflux. Upper gastrointestinal study was suggestive of partial duodenal obstruction and computed tomography revealed a complete ring of pancreatic parenchyma surrounding the second part of the duodenum. Diamond-shaped duodenoduodenostomy was achieved successfully and the post-operative period was uneventful. Although rare, AP must be kept in mind of any paediatric surgeon while confronted to symptoms of partial duodenal obstruction.

Keywords: Annular pancreas, case report, child, duodenal obstruction, duodenoduodenostomy, recurrent vomiting

Introduction

Congenital duodenal obstruction is one of the most common anomalies in newborns and infants.[1] It results from embryologic defects during foregut development leading to inadequate canalisation or malrotation. Different aspects of obstruction can be encountered (atresia, stenosis, web and Ladd's bands). Furthermore, pancreas anomalies, especially annular pancreas (AP), may also cause duodenal obstruction.[2]

AP is a rarely reported congenital anomaly and a recognised cause of duodenal obstruction in all age groups. It may manifest early in the neonatal period, but it may also have varied and often delayed symptoms, depending on the degree of obstruction, making diagnosis more challenging.[3]

The purpose of this article is to present a case of AP with delayed diagnosis in an 18-month-old girl and to shed light on this unusual aetiology of duodenal obstruction in children.

Case Report

A full-term 18-month-old girl was admitted to our paediatric emergency unit with acute convulsive seizures and poor general condition. She had a history of recurrent post-prandial non-bilious vomiting that got worse a few days before with failure to thrive. The neonatal period was unremarkable. She had been treated for her chronic vomiting with the clinical diagnosis of gastroesophageal reflux. On physical examination, the child was afebrile, hypotonic, dehydrated and malnourished. The abdomen was distended but soft and non-tender with no signs of peritonitis. The hernial orifices were free and the anus was normal. Biologically, the white blood cell count was 10,760 cells/mL. Haemoglobin was 12.2 g/dL. Urea was elevated at 0.6 g/L (normal: 0.1–0.3 g/L) and creatinine was normal. Albumin was 31 g/L (normal: 30–42 g/L). The blood ionogram showed hyponatraemia (124 mEq/L) and hypokalaemia (2.6 mEq/L). The erect abdominal radiography showed a very dilated stomach [Figure 1]. Ultrasound confirmed the significant gastric distension without clearly visible obstacle. Upper gastrointestinal tract

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contrast study demonstrated grossly dilated stomach and duodenum with delayed passage of contrast distally suggestive of partial duodenal obstruction [Figure 2]. Pre-operative gastroduodenoscopy noted bilious reflux which goes up to the oesophagus. The first portion of the duodenum appeared dilated and the second distorted. Beyond that, the duodenal lumen was not visible. Abdominal computed tomography (CT) revealed pancreatic parenchyma surrounding the second part of the duodenum suggesting AP [Figure 3]. After fluid resuscitation and correction of renal function and electrolyte disorders, a right upper abdominal transverse laparotomy was performed. On exploration, the stomach and the first portion of the duodenum were obviously dilated. The typical aspect of AP was noted and a conventional diamond-shaped duodenoduodenostomy was achieved [Figures 4 and 5]. The post-operative course was uneventful. Oral nutrition was gradually reintroduced on a post-operative day 3 with good tolerance. During 26 months of follow-up period, the patient was doing well and vomiting has totally disappeared.

Discussion

Congenital duodenal obstruction is a relatively frequent anomaly which can be complete or partial, intrinsic or extrinsic. Extrinsic obstruction may be related to several causes where intestinal malrotation and AP are the most common.[4] AP is a partial to complete ring of pancreatic tissue, which may include a major pancreatic duct, encircling the second portion of the duodenum.[5] This malformation was firstly named AP by Ecker in 1862.[6]

The true incidence of AP is difficult to determine accurately. Historically, it has been considered a rare anomaly. Early autopsy series reported an estimated incidence of 3 in 20,000,[6] but with the advent of sophisticated diagnostic modalities (CT, magnetic resonance imaging [MRI] and endoscopy), AP has been documented with increasing incidence (approximately 1 in 1000).[7]

The pathogenesis of AP is controversial and several theories regarding the embryological basis for the development of AP were reported.[5] The most commonly accepted is that of Lecco which suggests that the tip of the ventral pancreatic bud adheres abnormally to the duodenum and this abnormal adhesion leads to improper rotation around the second portion of the duodenum during the 4th–9th week of development resulting in duodenal encirclement[5] [Figure 6]. AP in children is frequently associated with other congenital anomalies. The most common anomalies are trisomy 21 (Down syndrome), cardiac anomalies, intestinal atresia and malrotation.[5]



Figure 1: Chest and abdominal X-ray showed marked gaseous distension of the stomach (S) and proximal duodenum. Bowel gas is seen beyond the proximal duodenum

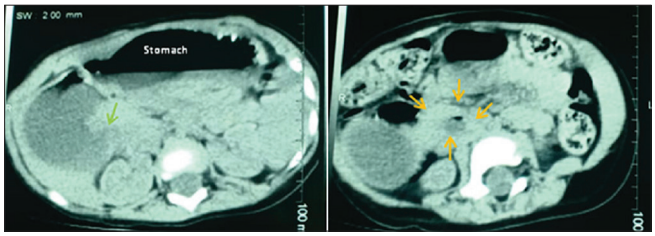


Figure 3: Axial non-contrast computed tomography of the abdomen showed a ring of pancreatic tissue that completely encircles the second duodenum (yellow arrows) leading to the diagnosis of annular pancreas with gastric outlet obstruction (green arrow)

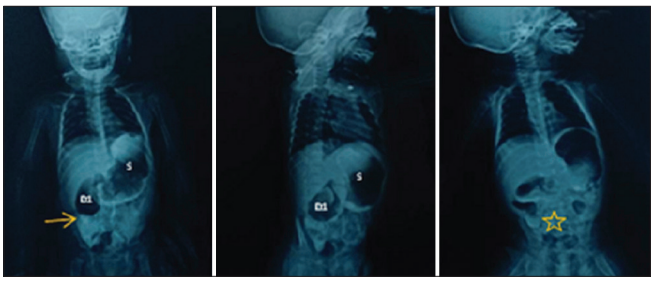


Figure 2: Double-contrast barium radiography showed stenosis of the second duodenum (D2) (yellow arrow) associated with proximal dilation of the duodenal bulb (D1) and stomach (S), classically known as the 'double bubble' sign. The barium contrast passes beyond the duodenal bulb (star)



Figure 4: An intraoperative view showing the annular pancreas as a ring surrounding the second part of the duodenum



Figure 5: Intraoperative view showing the final appearance of duodenoduodenostomy

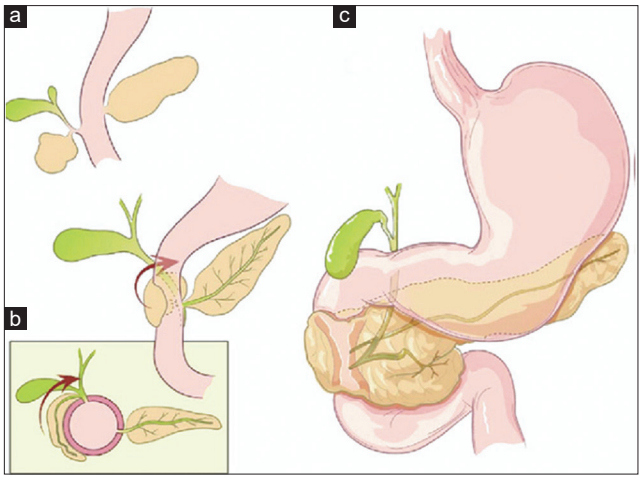


Figure 6: Embryologic development of annular pancreas according to Lecco. (a) Dorsal and ventral buds of the gut tube at about 4 weeks of development. (b) Tethering of the ventral bud tip to the duodenum and rotation leads to a ring of pancreatic tissue encircling the duodenum. (c) Annular pancreas (Zyromski NJ, Sandoval JA, Pitt HA, Ladd AP, Fogel EL, Mattar WE, *et al*. Annular pancreas: Dramatic differences between children and adults. J Am Coll Surg 2008;206:1019-25.)

Although it is congenital origin, AP can be symptomatic at any age and has been reported in both children and adult. Typically, children with AP presented with signs of gastrointestinal obstruction.[8] The pancreatic tissue encircling the duodenum may be responsible for an extrinsic compression leading to partial obstruction, but an underlying intrinsic duodenal stenosis is actually the main cause of obstructive symptoms.[3,9] Pre-ampullary obstruction resulting in non-bilious vomiting seems to be more common in children with AP than in those with other aetiologies of duodenal obstruction.[8] These findings have an important clinical implication and explain the diagnostic delay in our patient. The diagnosis of AP in a child with non-bilious vomiting and an incomplete tolerated obstruction is more likely to be missed.[8]

AP remains a diagnostic challenge for radiologists.[10] Plain radiography is usually sufficient to diagnose complete congenital duodenal obstruction and contrast study is generally not essential except when there is a suspicion of acute midgut volvulus.[3,10] However, the diagnosis of incomplete duodenal obstruction in a child with non-bilious vomiting by simple radiography is more likely to be missed since the double bubble or double stomach image is not constant.[10] CT and MRI may be useful but are unable to make the diagnosis in all cases. Therefore, the gold standard for diagnosing AP is by thorough surgical exploration.[10] In our patient, a contrast study suggested duodenal obstruction and the CT was able to identify the AP.

The AP is treated surgically by bypassing the obstacle to avoid damage to the pancreatic ducts. The aim of surgery is to remove the obstruction and maintain the continuity of the digestive tract. This is best achieved by duodenoduodenostomy which is more physiological and causes fewer complications.[8] However, when the duodenum cannot be mobilised adequately, duodenojejunostomy or gastrojejunostomy is recommended.[3]

Conclusion

Although rare in children, AP should be considered in the differential diagnosis of recurrent non-bilious vomiting. Diagnosis can be suggested by contrast study and abdominal CT and is only confirmed by surgical exploration. The surgical procedure is based on bypassing the duodenal obstruction and duodenoduodenostomy is the best treatment option.

Consent of patient

Written informed consent was obtained from the child's parents to publish this case report and all associated images. A copy of this written consent is provided if requested by the journal/editor.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient's parents have given their consent for patient's images and other clinical information to be reported in the journal. The parents understand that patient's names and initials will not be published and due efforts will be made to conceal the identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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