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Duodenal Duplication a Rare Case Report

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Abstract: Bilious vomiting is a relevant sign in children that requires immediate evaluation and diagnosis. A duplication of the intestinal tract is a possible cause of obstruction if located distally to the major duodenal papilla of Vater and most of them involve the jejunum, stomach, or colon. Duodenal duplications are very rare and can have an endoscopic or surgical treatment after diagnosis. This case report presents a 1year and 6 months old child that presented because of vomiting, abdominal distension and tenderness. Diagnosis was made after exploratory laparotomy that showed a Duodenal Duplication (DD) at the level of the third portion causing compression of the intestinal lumen that required surgical resolution.

Keywords: Duodenal Duplication (DD), Kidneys, Ureters, and Bladder (KUB), Computed Tomography (CT scan), Magnetic Resonance Imaging (MRI), and Endoscopic Ultrasound (EU).

INTRODUCTION

Duplications of the alimentary tract are rare developmental abnormalities. They can be observed anywhere along the alimentary tract, and they are located most often in the ileum and least often in the duodenum. Duodenal duplication constitutes 5%-7% of all gastrointestinal duplications [1]. The clinical features of duodenal duplication cysts vary from asymptomatic cases to nonspecific symptoms such as abdominal pain, abdominal distention, and vomiting. Some patients develop symptoms of gastric outlet obstruction or small bowel obstruction. Ulceration or perforation due to the presence of an ectopic mucosa may cause duodenal bleeding or peritonitis, respectively [2]. We present a case of a one year and six months old child that presented because of vomiting, abdominal distension and tenderness. Diagnosis was made after exploratory laparotomy that showed a Duodenal Duplication (DD) at the level of the third portion causing compression of the intestinal lumen that required surgical resolution.

CASE REPORT

A one year and six months old boy previously healthy, to first degree consanguineous parents not fully vaccinated, presented for vomiting, abdominal distension and bloating with obstipation of one day duration associated with oliguria since 8 hours and undocumented fever. At Emergency department baby developed three episodes of non-bloody, non-bilious vomiting with exacerbation of the abdominal pain. Later on he developed two episodes of coffee ground vomiting. Vital signs upon admission were within

normal range. On physical examination the child was conscious, cooperative, oriented, irritable, ill looking, anicteric sclera, well injected conjunctiva, normal ENT examination, no lymph node enlargement, good bilateral air entry on lung auscultation, regular S1 and S2 with no heart murmurs, abdominal examination revealed positive bowel sounds, distended abdomen, with tenderness to deep and superficial palpation all over abdomen. Abdomen cannot be assessed for any organomegaly or palpable masses. No signs of dehydration, Capillary Refill < 2 sec. On per rectum examination blood on the fingertip was observed but no apparent anal fissures or bulging masses. As for his neonatal history, he was born by C-section to a G7P5A2L2D3 mother, with ICN admission for 3 days for suspected sepsis. Obstetric history: G1 and G2 born at term but passed away soon after birth for unknown reason to parents, G3 born at 32 weeks of gestation and passed away for also unknown cause, G4 and G5 were both miscarriages at 28 weeks of gestation, and G6 and G7 fortunately healthy newborns. Nutritional history was on breast feeding which was stopped three days prior to presentation and solid food (started at 5 months of age). Parents stated that their child ingested three kilograms of banana one day prior to presentation. Laboratory data upon admission were as following: Hemoglobin 10.9 g/dl with WBC count of 24,000 (76% Neutrophils and 19% Lymphocytes), CRP: 69, Prothrombin time: 15.4, PTT: 41.1, INR: 1.23 and electrolytes were normal with urine analysis negative. KUB was done and showed dilatation with fecal retention in the large bowels (Figure 1).



Fig 1: KUB with dilated and fecal impaction of the large bowel

Then Ultrasound of abdomen showed bowel wall edema and diffuse thickening. At this point the differential diagnosis was: Diffuse Enterocolitis, Intestinal obstruction or Intussusception, Late presentation of Hirschprung's disease, Fecaloma and Meckel's diverticulum. The child was admitted for further investigations and management. Amikacin stat dose was given then Ceftriaxone with Metronidazole were started. Next day Prothrombin time, PTT were

repeated showing: PT: 17.1, PTT: 43.8, INR: 1.41. Enema with gastro grafin was done showing no intestinal obstruction or Intussusception (Figure 2 and 3).

Nasogastric and rectal tube were inserted, KUB repeated after 3 hours showing decrease in bowel distension (Figure 4).

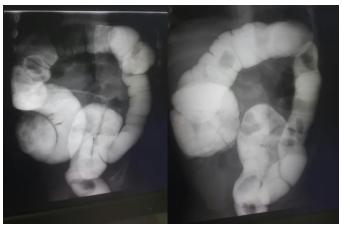


Fig- 2 and 3: Normal gastrografin enema with no signs of intestinal obstruction



Fig 4: Decrease of bowel distension

Patient clinically was still looking ill with diffuse abdominal tenderness on physical examination and elevated INR. 15 mg/kg of Fresh Frozen Plasma and 2 mg of vitamin K were given. Three days in the hospital, the baby sometimes showed clinical improvement with decrease in abdominal distension,

sometimes deterioration with re-distension of the abdomen and vomiting. Final decision was to undergo exploratory laparotomy that showed Duodenal Duplication with unilateral Duodenal Volvulus and gangrene (Figure 5).



Fig 5: Duodenal Duplication with Duodenal Volvulus

Unilateral Duodenal Volvulus repair with necrotic part resection were done. Patient post operation was maintained on Ceftriaxone and Metronidazole. Repeated KUB showed improvement in bowel distension (Figure 6).



Fig 6: Postop Normal KUB

Patient clinically showed rapid improvement, two days later baby passed stools. Oral feeding was started and well tolerated. Follow up laboratory tests showed Hemoglobin of 9.1 g/dl with Platelet count of 512 \times 10, WBC count of 12 300 (Neutrophils 39%, 36% of Lymphocytes), INR: 1.11, CRP: 22 mg/l and Electrolytes were normal. Patient was discharged home Day four post operation.

DISCUSSION:

Duplications of the alimentary tract are rare developmental abnormalities. They can be observed anywhere along the alimentary tract, and they are located most often in the ileum and least often in the duodenum. Duodenal Duplication constitutes 5-7% of all gastrointestinal duplications. It can be cystic or tubular, communicating or non-communicating, but the most common type is cystic and non-communicating. These are generally located at the medial border of the first and second parts of the duodenum and extend to

the anterior or posterior side. Duplication of the third portion is even less frequent [1]. Duodenal Duplication observed in our case was cystic and located in the third part of the duodenum, but it was of the communicating type. The abnormality is usually diagnosed in infancy and childhood. However, many patients can remain asymptomatic until adulthood, and about one third of patients present after 20 years of age.

The clinical features of Duodenal Duplication cysts vary from asymptomatic cases to nonspecific symptoms such as abdominal pain, abdominal distention, and vomiting. Some patients develop symptoms of gastric outlet obstruction or small bowel obstruction. Ulceration or perforation due to the presence of an ectopic mucosa may cause duodenal bleeding or peritonitis, respectively. Duodenal Duplication cysts may also cause recurrent episodes of acute pancreatitis because of the direct pressure applied against the pancreatic duct. Stone formation was

reported in some cases due to stasis inside the cyst. Jaundice and intussusceptions have also been reported. Hemorrhagic ascites is a very rare complication of duplication cysts [2]. The preoperative diagnosis of intestinal duplications is rarely accurate. The differential diagnosis encompasses all cystic lesions in this region, which include: Choledochoceles, Pancreatic Pseudo cysts, Pancreas Cystic Tumors, Mesenteric Cysts, and Duodenal Diverticulum. In barium studies, in non-communicating cysts, the first and the second parts of the duodenum can be seen as compressed and displaced by a mass, whereas, in the communicating type, the cyst itself can be observed as being filled with barium. Duodenal Duplication is differentiated from other cystic lesions by the "gut signature" of its wall observed by abdominal or endoscopic ultrasound. Gut signature refers to the layered pattern of the wall, with the hyper echoic inner layer representing the sub mucosa and the hypo echoic outer layer representing the smooth muscle. Peristalsis of the cyst wall noted upon real-time ultrasound is strongly suggestive of a duplication cyst. Ultrasound is an operator-dependent method and unfortunately it was not helpful in the diagnosis of our case. Computed Tomography (CT) is valuable in identifying the type, location and the size of the duplication cyst. Although Magnetic Resonance Imaging (MRI) and Gastroduodenoscopy are the other modalities that can be used for diagnosis, CT images are usually sufficient. Endoscopic Ultrasound (EU) examination is very important for the differential diagnosis, especially to differentiate it with the Choledochocele and to distinguish between solid and cystic lesions. EU shows duplication cysts as anechoic, homogenous lesions with regular margins arising from the sub mucosal layer or extrinsic to gut wall; although, a hypo echoic pattern can also be seen with a duplication cyst. Duplication cysts may contain thick mucinous material, septations, fluid levels, debris and sometimes, detached ciliary tufts which could be diagnostic [3]. In spite of the diagnostic workup performed before the operation, accurate diagnosis of Duodenal Duplication is by histological examination. According to the analysis made by Merrot et al, two types of intra- or juxta Duodenal Duplications occur: 1) - A common wall formed by two separate mucosa with their own muscularis mucosa and a layer of intervening connective tissue; and 2) - A common wall that comprises two mucosal layers with two smooth muscle layers, but that also contains biliary and pancreatic ducts (1). Surgical management is the most common measurement for Duodenal Duplication and is made in accordance with the relationship between the duplication and the Pancreatico-biliary system. A total excision is an optimal approach. However, if the lesion occurs closed to the Pancreatico-biliary tract, performing the total excision is difficult and risky. However, if it is not possible, partial resection or internal derivation must be carried out. In partial resection, the entire cyst wall is removed wherever possible, while the area of maximum adherence to the

duodenum is preserved (1, 2, and 4). Recently, more and more cases which treated by endoscopic fenestration, have been reported and this method was thought to, it has been reported that the Duodenal Duplication has the possibility to become malignant. In some cases, Carcinoid Tumor was found in the wall of the Duplication Cyst. If only perform drainage endoscopically to relieve symptoms without excising the lesion, the duplication cyst may turn malignant [4]. Therefore, we treated our patient with subtotal excision and internal derivation, which cured the patient. Although duodenal cysts are considered a rare entity, we believe that further research is needed to design a specific classification system and management criteria for these lesions. Such diagnostic and therapeutic criteria would spare many patients the squeals of laparotomy by taking advantage of the recent advances in minimally invasive and endoscopic techniques. This would only be possible through accumulation of more cases. Therefore, we encourage surgeons to report any lesion of this type with enough details about location and appropriate treatment options.

CONCLUSION:

In conclusion, Duodenal Duplication should be considered in the differential diagnosis of a patient who presents with vomiting, abdominal symptoms, especially when cystic structures neighboring the duodenum are demonstrated radiologically. Ideal treatment is total excision but, if not possible, subtotal excision and/or internal derivation should be performed. When treated properly, these lesions usually have favorable outcomes.

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