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Published in:
International Journal of Surgery Case Reports

DOI:
10.1016/j.ijscr.2015.06.013

Publication date:
2015

Document version
Final published version

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Citation for published version (APA):

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Acute pancreatitis secondary to duodeno-duodenal intussusception caused by a duodenal membrane, in a patient with intestinal malrotation

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1. Introduction

Duodeno-duodenal intussusception, which involves the distal invagination of a segment of the duodenum, is a rare condition owing to the retroperitoneal fixation of the duodenum, which is sometime absent in cases of intestinal malrotation. The vast majority (80–90%) of cases of intestinal intussusception are secondary to pathological masses in the intestine, such as Brunner's gland hamartomas, lipomas and adenomas [1,2]. The presence of a duodenal membrane may cause retention of food resembling a pathological mass, which may in turn facilitate intussusception.

We describe the interesting case of a patient with intestinal malrotation who developed acute pancreatitis as a result of a duodenal intussusception involving the head of pancreas which was caused by a duodenal membrane. The most common symptoms of duodenal intussusception are intermittent abdominal pain and vomiting. Other symptoms include gastro-oesophageal reflux, weight loss, fatigue, anaemia due to iron deficiency; pancreatitis due to obstruction of the outlet duct has also been described [3–6]. Owing to the intermittent nature of the symptoms delay in diagnosis is common [2].

2. Presentation of case

A 19-year old male was admitted with acute onset abdominal pain. Symptoms had started three days previously and consisted of intermittent peri-umbilical pain, nausea and vomiting.

The initial physical examination indicated a healthy young man with mild epigastric tenderness. In infancy he had undergone surgery for a congenital heart malformation involving closure of atrial and ventricular septal defects. During childhood he had been treated with proton-pump inhibitors for symptoms of gastro-oesophageal reflux, and he had a history of intermittent post-prandial abdominal pain, but had never undergone endoscopic examination, or any abdominal surgery. Presented with a normal height and weight for his age with a body mass index of 25.

Routine blood analyses revealed elevated levels of S-amylose (408 U/l; ref. value 10–65 U/l) and bilirubin (41 μmol/l; ref. value 5–25 μmol/l). Subsequently the patient developed fever, (core body temperature 38.6°C); acute cholecystitis was suspected so he was treated with intravenous antibiotics. Subsequent abdominal ultrasonography was inconclusive, although there were signs consistent with a tumour in the head of the pancreas. A CT-scan revealed a duodenal-duodenal intussusception involving the head of the pancreas and the common bile duct (Figs. 1 and 2).

These finding were confirmed at laparotomy and non-rotation of the duodenum was also noted (Fig. 3). The intussusception was released, and a duodenotomy revealed a duodenal membrane with a narrow central opening (Fig. 4). The top of the intussusception was at the level of the membrane. The membrane was excised and the duodenotomy closed transversely. Post-operative recovery was uneventful. S-bilirubin and S-amylose levels were within the normal range at discharge.

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3. Discussion

To our knowledge this is the first case report of pancreatitis resulting from duodenal invagination caused by a duodenal membrane. High peristaltic pressure proximal to the stenosis may have contributed to the development of the invagination, which may also have been facilitated by the mobile, non-rotated duodenum, which had a vertical straight course without retroperitoneal fixation.

Cases in which an intraluminal polyloid mass or tumour in the duodenum resulted in duodeno-jejunal intussusception have been reported [4,5]. In a few cases the intussusception involved the head of the pancreas, resulting in acute pancreatitis [5]. These patients were treated by surgical reduction of the intussusception and duodenal resection or polypectomy.

Membranous duodenal stenosis is often treated by duodenotomy and excision of the membrane followed by a duodeno-jejunal anastomosis, with good results [7,8]. In our case the patient had persistent symptoms of gastro-oesophageal reflux, which should have triggered an upper GI endoscopy; had this revealed the intestinal malformation the patient could have been treated in a timely fashion and which would have avoided this serious complication. The intussusception was not detected by ultrasonography but was revealed by a CT-scan. A previous study found that 27.1% of patients with intestinal malrotation had a history of congenital cardiovascular defects [9]. This suggests that all children with intermittent upper gastrointestinal symptoms such as vomiting, abdominal pain and gastro-oesophageal reflux, particularly those with a history of congenital heart malformation, should undergo upper GI endoscopy and imaging of the GI-tract to exclude GI anomalies.

4. Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

References