ISR Journal of Medical Case Reports (ISRJMCR)
Homepage: https://isrpublisher.com/isrjmcr/
Volume 1, Issue 1, May, 2025 ISSN: XXXX-XXXX (Online)

Radio-Clinical Aspect of Occlusive Syndrome Associated with Pancreatitis Secondary to Duodenal Haematoma: A Case Report

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Article History

Received: 13-05-2025

Accepted: 20-05-2025

Published: 23-05-2025

Summary: Case Report

This case report describes a 13-year-old adolescent, diabetic for 3 years, admitted for severe abdominal pain associated with bilious vomiting and cessation of bowel movements. Clinical and paraclinical evaluation revealed a bulbar ulcer complicated by a duodenal haematoma leading to a partial occlusive syndrome and acute reactive pancreatitis. Management included digestive rest, parenteral nutrition and eradication of Helicobacter pylori by injection. The favourable clinical and radiological outcome highlights an effective therapeutic approach and raises questions about the management of rare gastrointestinal complications often secondary to upper gastrointestinal endoscopy in children.

Keywords: Duodenal Hematoma, Bulbar Ulcer, Pediatric Pancreatitis, Diabetes Mellitus, Helicobacter Pylori Eradication.

INTRODUCTION

Duodenal haematoma is a rare entity, most often observed in children and adolescents, due to the fragility of the duodenal wall and its vascularisation. Trauma, however minor, is often the most common cause, although a proportion of cases are secondary to coagulation disorders, endoscopic procedures or, more rarely, pancreatitis. Accumulation of blood in the duodenal wall causes thickening of the wall and compression of the intestinal lumen, resulting in occlusion. Abdominal CT is the examination of choice to confirm the diagnosis, assess the extent of the haematoma and look for any associated complications.

We report here the case of a 13-year-old adolescent with type 1 diabetes who presented with a complex occlusive presentation in the context of a duodenal haematoma. Investigation revealed the presence of a bulbar ulcer with Helicobacter pylori, suggesting a multifactorial interaction between the ulceration, pancreatic inflammation and the formation of the haematoma. This observation illustrates the diagnostic and therapeutic complexity of these atypical presentations.

OBSERVATION

The patient was 13 years old, known to have been diabetic for 3 years and on insulin therapy, with no notable digestive history. He was consulted for repeated vomiting

associated with intense epigastralgia with no initial transit problems, which led to an oeso-gastroduodenal fibroscopy revealing a bulbar ulcer with biopsy confirming the presence of Helicobacter pylori. The evolution was marked 24 hours later by the appearance of bilious vomiting, exacerbation of gastric pain and the onset of an occlusive syndrome with cessation of matter and gas.

On physical examination, the patient was conscious, asthenic but respiratory and haemodynamically stable, with a capillary blood glucose of 3.6 g/l, UC of 3+ and GU of 2+; epigastric, periumbilical and left hypochondrium tenderness.

A. Additional tests:

1. Biology:

- Alkaline reserve at 9 meq/L
- Lipasemia: 2,505 IU/L, i.e. 41x the N

2. Imaging :

- RX ASP: No subdiaphragmatic air crescents or hydroaerobic levels
- Abdominal ultrasound: Head of pancreas enlarged, echogenic.
- Abdominal CT scan: Parietal haematoma of the 3rd portion of the duodenum extending into the proximal jejunum reducing the digestive lumen, without upstream gastric distension, associated with oedematous swelling of the head of the pancreas of reactive appearance.

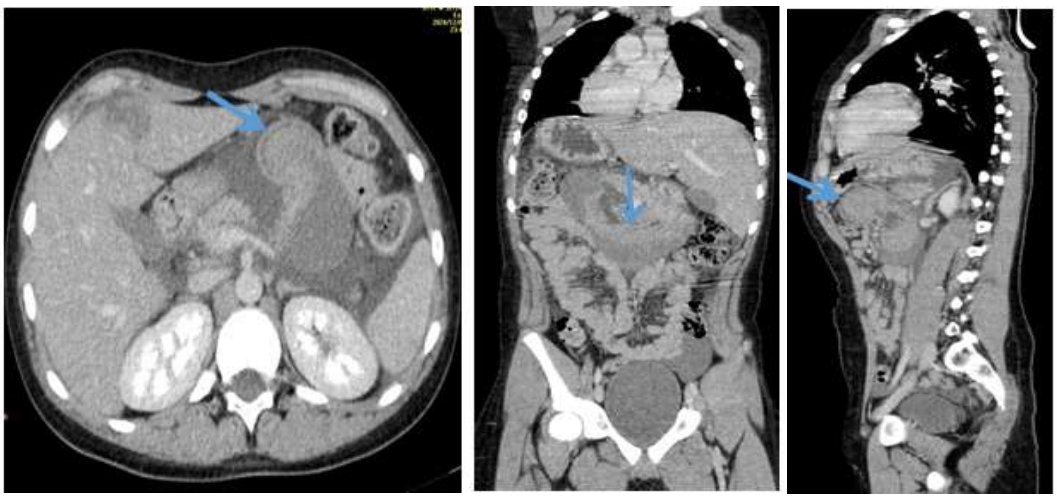

Figure 1: Thoracic-abdominal-pelvic CT scan in axial, coronal and sagittal sections

Enlargement of the 3rd duodenum and the proximal jejunal portion with a spontaneously hyperdense heterogeneous parietal haematoma which is not enhanced after injection of PDC.

3. Fibroscopy: Petechial, erythematous and diffuse nodular gastritis, bulbar ulcer, longitudinal striae in the oesophagus.

4. Biopsy: Presence of Helicobacter pylori

B. Diagnosis:

- Partial occlusive syndrome secondary to complicated duodenal haematoma
- Acute reactive pancreatitis
- Erythematous bulbar ulcer caused by Helicobacter pylori

C. Therapeutic management:

- Complete digestive rest: to allow resolution of the haematoma and recovery of the pancreas
- Parenteral nutrition: compensating for nutritional requirements
- Insulin therapy in SAP: optimal glycaemic control
- HP eradication treatment by injection (Amoxicillin + Metronidazole)
- Double-dose PPIs: reduce gastric acidity and promote ulcer healing

D. Clinical course :

- On the seventh day, there was clinical improvement with a return to stool output and liquid feeding without vomiting or pain.
- Abdominal ultrasound shows persistence of the duodenal haematoma with partial repermeabilisation of the digestive lumen at this level.
- Standardised lipasemia
- Transition to oral treatment to complete HP eradication

DISCUSSION

The association between bulbar ulcer, duodenal haematoma and acute pancreatitis is a rare entity, particularly in children. In this case, the underlying pathophysiological mechanisms are complex and merit detailed analysis to better understand their interdependence.

A. Anatomical proximity and spread of inflammation

The duodenal bulb is in direct contact with the head of the pancreas via vascular and lymphatic structures. Severe inflammation of the ulcer can:

- Spread an inflammatory reaction to adjacent pancreatic tissue, leading to secondary pancreatitis.
- Impair local lymphatic or venous drainage, which may aggravate pancreatic oedema
- Haematoma in the 3rd portion of the duodenum may have compressed the main pancreatic duct (Dictus Wirsungi), causing enzyme stasis and intra-pancreatic activation of digestive enzymes, triggering acute pancreatitis.

B. Radiographic features: Imaging explained the mechanism of occlusion

Ultrasound:

- Ultrasound usually reveals a uniform echogenic mass along the duodenal convexity
- A small intramural haematoma may take the form of a thickening of the intestinal wall
- Obstruction of the duodenal lumen

Scanner:

- An isolated haematoma will classically have a high heterogeneous attenuation of 50-60 HU with narrowing of the duodenal lumen.
- Small haematomas appear as a thickened duodenal wall
- This generally leads to a narrowing or obstruction of its lumen
- The duodenum is dilated near the affected segment
- A non-contrasting collection of fluid may be seen in both the perforation and the

C. The role of digestive alterations in diabetes

In this patient, diabetes would probably have exacerbated the situation:

- Diabetic microangiopathy and oxidative stress have compromised the regenerative capacity of the duodenal mucosa, facilitating the development and progression of ulcers.
- Diabetic gastroparesis, often associated with gastric stasis and reflux of acid contents into the duodenum, could also have played an aggravating role.

D. Influence of Helicobacter pylori

Biopsy-confirmed H pylori infection probably contributed to ulceration by:

- An increase in gastric acid secretion and a decrease in the production of protective bicarbonates in the duodenum.
- An exacerbation of local inflammation, making neighbouring tissues, including the pancreas, more vulnerable.

E. Rare spontaneous resolution of pancreatitis

This case is unusual in that the acute pancreatitis resolved spontaneously with conservative treatment (digestive rest and parenteral nutrition). This phenomenon may be explained by:

- The absence of pancreatic necrosis or collection, suggesting moderate oedematous pancreatitis.
- Progressive lifting of the obstruction of the pancreatic duct due to partial regression of the duodenal haematoma

CONCLUSION

This case illustrates a rare but potentially severe complication in adolescents: duodenal haematoma, in a multifactorial context combining bulbar ulcer, Helicobacter pylori infection and acute pancreatitis, all in a setting of type 1 diabetes mellitus. The onset of occlusive syndrome 24 hours after diagnostic fibroscopy raises the question of endoscopic trauma to a mucosa previously weakened by ulceration and vascular terrain. This highlights the need for particular vigilance during endoscopic procedures in patients with fragility factors, and reminds us of the importance of rapid imaging diagnosis in the event of post-endoscopic clinical deterioration. Treatment is usually conservative, but requires close clinical and radiological monitoring.

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