Received: 7 July 2023 | Accepted: 14 December 2023

DOI: 10.1002/jpr3.12034

CASE REPORT

Pancreatology

Duodenal obstruction: A rare complication of severe acute pancreatitis in children

Olivier Leclercq[1] | Laurie Lecomte[2] | Xavier Stephenne[3] | Isabelle Scheers[3]

[1]Department of Pediatrics, Cliniques Universitaires Saint-Luc, Brussels, Belgium

[2]Department of Pediatrics, Hopital de Jolimont, La Louvière, Belgium

[3]Department of Pediatrics, Pediatric Gastroenterology and Hepatology Unit, Cliniques Universitaires Saint-Luc and Laboratoire PEDI, IREC, Université Catholique de Louvain, Brussels, Belgium

Correspondence

Olivier Leclercq, Department of Pediatrics, Cliniques Universitaires Saint-Luc, Ave Hippocrate 10, 1200 Brussels, Belgium.
Email: olivier.leclercq@student.uclouvain.be

Isabelle Scheers, Department of Pediatrics, Pediatric Gastroenterology and Hepatology Unit, Cliniques Universitaires Saint-Luc and Laboratoire PEDI, IREC, Université Catholique de Louvain, Brussels, Belgium.
Email: Isabelle.scheers@saintluc.uclouvain.be

Funding information

Belgian Foundation Against Cancer, FNRS (fondation nationale pour la recherche scientifique), Salus Sanguinis and Fondation Saint-Luc pour le Cancer

Abstract

Duodenal obstruction (DO) is an uncommon complication of pancreatitis. It has been described in groove and severe acute and chronic pancreatitis in adults but, to the best of our knowledge, it has not yet been reported in pediatric acute pancreatitis. Current guidelines comment on management of several early and late-onset complications, but DO is not mentioned. We describe two patients with acute necrotizing pancreatitis who presented with several complications including walled-off necrosis and DO. In adults, DO is generally managed with adapted nutrition but may require surgical bypass, such as gastroenterostomy. Our patients were managed conservatively and fully recovered 2 months after DO diagnosis. DO may require lengthy hospitalizations and markedly restrict patients' quality of life; however, prolonged conservative treatment was effective in our patients and should be considered even in severe pediatric cases.

KEYWORDS

acute pancreatitis, complication, conservative treatment, duodenal obstruction

1 | INTRODUCTION

Knowledge of acute pancreatitis (AP) in children has grown in the past decades. In 2017, pediatric-specific management recommendations for AP were released,[1,2] which have helped to harmonize practice on the disease around the world. These guidelines are based on literature review and expert opinion, and identify gaps in the available data. Among these gaps, recommendations for the management of rare but severe complications are limited.

2 | CASE REPORT 1

A 4-year-old boy without medical history other than obesity (BMI:27, +5.1 SD) presented with severe periumbilical pain and vomiting after receiving ibuprofen for an Influenza B infection.

On initial evaluation, his parameters were as follows: temperature 38.1°C, heart rate 122 bpm, blood pressure 110/70 mmHg, and SpO2 90% on room air. Physical examination revealed a peritoneal abdomen, tachypnea, delayed capillary refill, and increasing lethargy. His laboratory results on admission showed

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a white blood cell count (WBC) of 33,000/μL (normal values for lab (NV): 5000–19,000), C-reactive protein (CRP) of 150 mg/L (NV: <5), lactic acidosis (pH 7.11, pCO2 37 mmHg, lactate 6.0 mmol/L), elevated serum lipase of 1432 U/L (NV: 0–160) and acute kidney injury (AKI) with a serum creatinine of 2.9 mg/dL (NV: 0.3–0.6) and urea nitrogen of 70 mg/dL (NV: 8–20).

The patient received fluid resuscitation (30 mL/kg of crystalloids, followed by 1.5x maintenance fluids during the first 24 h), ventilatory support and was transferred to the pediatric intensive care unit (PICU). Pain was managed by nalbuphine and paracetamol. Necrotizing AP was confirmed on an abdominal CT scan. Pancreatitis related necrosis progressed and collected in the retroperitoneum. Multisystem organ failure (MOF; involving lungs and heart) persisted for 14 days. Necrosectomy was performed percutaneously at Week 3. Control ultrasounds showed resolution of the collections and the drains were removed 4 days later. Walled-off necrosis (WON) recurred within 2 days requiring endoscopic placement of transgastric pigtail drains, Liquid culture was positive for fluconazole-resistant Candida Glabrata, and the patient was subsequently treated with a 14-day course of caspofungin.

Fourteen weeks after initial admission, the patient, who was still hospitalized, presented with increased abdominal pain and bilious vomiting. Serum lipases were normal at the time. Stenosis of the second duodenum was evidenced on the upper gastrointestinal series (UGS) (Figure 1) and confirmed by endoscopy. Conservative treatment was attempted after medico-surgical discussions. A nasojejunal (NJ) tube (8 Fr) was placed and feeding was permitted using this route. The patient was discharged at Week 18 with discontinuous NJ feeding to facilitate daily activities and obtain a better quality of life. The stenosis progressively resolved (Figure 2) and oral feeding was permitted first with liquids and then with solids. The patient was weaned from NJ tube feeding at Week 23 and showed no symptom recurrence at 6-month follow-up.

3 | CASE REPORT 2

A 10-year-old boy with a medical history of obesity (BMI:25.9, +2.1 SD) was admitted to the emergency department for severe acute periumbilical pain, vomiting, and jaundice.

His admission vitals were as follows: temperature 35.5°C, heart rate 170 bpm, blood pressure 83/52 mmHg, and SpO2 92% on room air. Physical examination revealed a peritoneal abdomen and delayed capillary refill. His laboratory results on admission showed a WBC of 19,200/μL, CRP of 130 mg/L, lactic acidosis (pH 7.20, lactate 4.4 mmol/L), serum creatinine 0.75 mg/dL, gamma-glutamyl transferase 57 IU/L (NV: <25), aspartate aminotransferase 82 IU/L (NV: <50), alanine aminotransferase 98 IU/L (NV: <45), total/direct bilirubin 3.1/2.7 mg/dL. Abdominal CT scan confirmed the presence of multiple stones in the distal common bile duct, as well as necrotic pancreatitis (>40% necrosis).

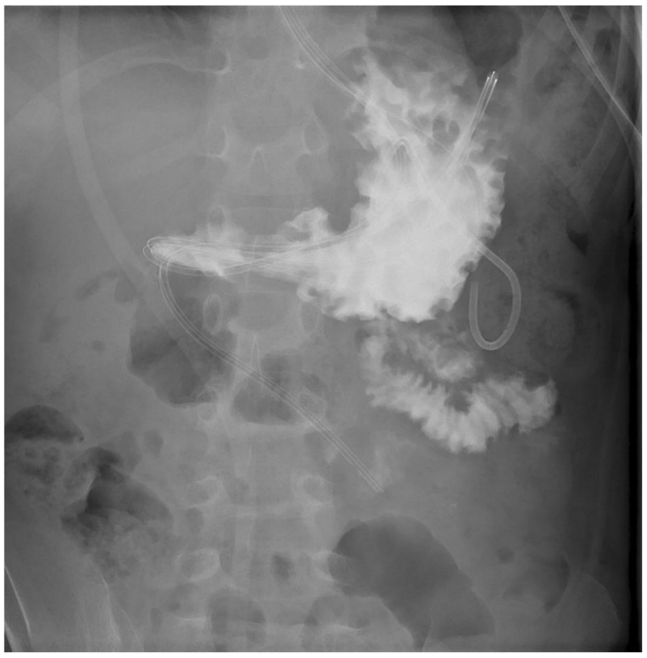

FIGURE 1 Gastrointestinal series (Patient 1): Prolonged stenosis of the second duodenum at the time of diagnosis.

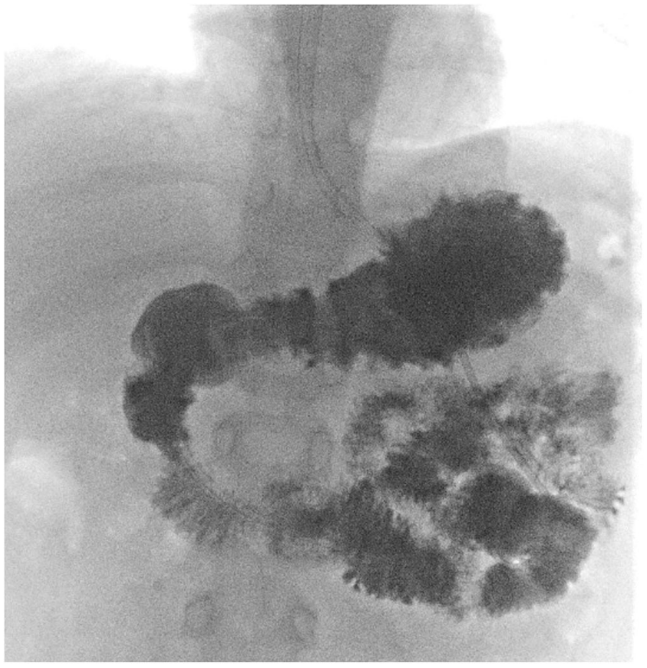

FIGURE 2 Gastrointestinal series (Patient 1): After 7 weeks of conservative treatment showing a partial resolution of the stenosis.

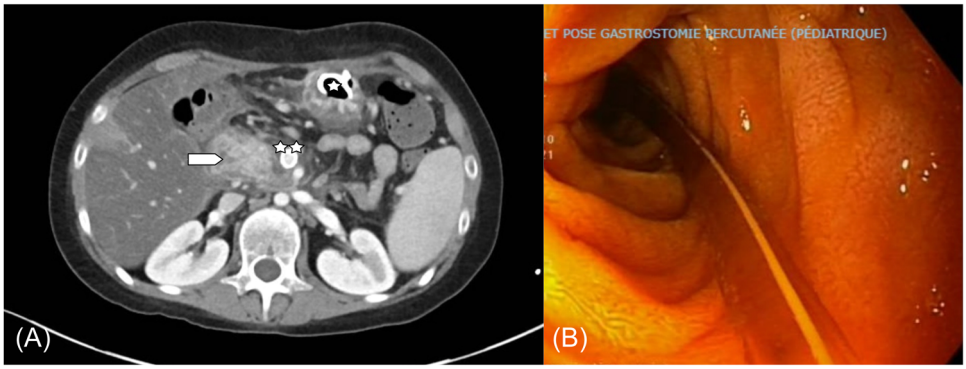

FIGURE 3 (A) MRI T2 sequence (Patient 2) showing important edema of D2 resulting in the narrowing of the bowel lumen by >90% (arrow) as evidenced on subsequent endoscopy (data not shown). The patient has a cystogastrostomy pigtail stent in place (*) and a stent was inserted in the thrombosed portal vein (**stent extending in the superior mesenteric vein). (B) Follow-up endoscopy showing progressive regression of the obstruction and edema of the duodenum.

The patient received fluid resuscitation (20 mL/kg of crystalloids, followed by 1.4x maintenance fluids during the first 24 h), ventilatory support and was transferred to the PICU. MOF (involving lungs, heart, and kidney) persisted for 10 days. The patient developed a portal vein thrombosis treated by implanting a portal vein stent. Furthermore, multiple collections were drained by cystogastrostomy 3 weeks after admission. A lumen-apposing stent (LAS) was placed and necrosectomy was performed at Weeks 4 and 5. Necrotic collections progressively resolved, the LAS was removed and replaced by two pigtail drains at Week 8. Cholecystectomy was performed at Week 12 to prevent recurrence and a gastrostomy was placed as the patient did not accept oral or NG-tube feeding. As there was a high suspicion for a disconnected pancreatic body/tail, the patient was discharged at Week 14 with the pigtail drains in place. At Week 17, the boy was readmitted with bilious vomiting and increasing abdominal pain. Lipase remained within normal ranges, but CRP raised to 240 mg/L without demonstrated infection. Signs of pancreatitis and major gastric and duodenal wall edema were observed on the control CT scan (Figure 3). The two pigtail stents draining the previous necrotic collection had spontaneously migrated to the digestive system. Conservative treatment was attempted, endoscopy showed near-occlusion of the second part of the duodenum, and a 10 Fr NJ tube was placed past the stenosis. The patient was discharged at Week 18 with continuous feeding. Oral feeding was progressively allowed from Week 20, first with liquids and then with solids. NJ tube feeding was weaned off at Week 23. There was no symptom recurrence, and the patient is doing well after 15 months.

4 | DISCUSSION

Although the incidence of AP has been estimated to be approximately 1/10,000 children in recent studies,[1,3] severe evolutions are fortunately rare in children. Of the numerous recognized risk factors for AP,[4,5] both patients presented with severe obesity which has been demonstrated to increase the risk of severe AP by 1.6-fold.[6] Patients with severe AP must be followed for late-onset complications including thrombosis, biliary strictures, pseudocysts (8%–41%),[1] and WON. Furthermore, severe pancreatic damage may lead to endocrine and/or exocrine pancreatic insufficiency.[7]

The severity of both cases and multiple complications including WON followed by duodenal obstruction (DO), made medical management difficult. WON and pancreatic pseudocysts are commonly described complications of severe AP[1,3,5] whereas, to the best of our knowledge, DO has not been reported in children.

DO is a well-defined complication of WON in adult acute and chronic pancreatitis. The pathogenesis of DO involves severe and sustained pancreatic inflammation extending to adjacent digestive structures (pylorus, duodenum, jejunum). It may also be due to extrinsic compression by a pseudocyst or WON, or by swelling of the mesentery.[8]

Other potential causes of DO such as congenital conditions (annular pancreas, congenital duodenal web, duodenal duplication), mesenteric artery syndrome (MAS), malignancies, or groove pancreatitis (GP) must be ruled out. In our two patients, initial imaging and endoscopic procedures did not evidence DO, a congenital etiology was therefore unlikely. Malignancies are very rare in children and occur in a different context. Imaging did not evidence MAS. Finally, GP can be differentiated from AP-DO as it is a form of chronic pancreatitis centered in the groove. It is not associated with peripancreatic fluid effusion and inflammation as seen in our patients.[9]

Imaging is essential for diagnosing DO and differentiating it from other intrinsic or extrinsic causes. The technique of choice greatly depends on the suspected underlying cause. Although irradiating, CT-scan remains the favored technique for imaging gastric

outlet obstruction in adult patients with severe pancreatitis. It is also the method of choice in imaging complications of severe AP in children.[1] Ultrasound may reveal gastric distension and a thickened gastric or duodenal wall and help in deciphering the differential diagnosis. Evaluation may be limited in obese children and air or other organ interposition may hamper the examination. Barium UGS may show the site of obstruction. MRI/MRCP is the technique of choice for assessing congenital anomalies of the pancreas but is usually not readily available.

The absence of similar pediatric cases rendered management unclear. In adults, patients are managed conservatively with parenteral nutrition to ensure bowel rest and with endoscopy to drain pseudocysts or WON. If the bowel obstruction fails to resolve within 3–4 weeks, endoscopic or surgical gastroenterostomy is recommended, as fibrosis causes fixed obstructions.[8,10,11]

5 | CONCLUSION

DO can occur as a late complication of severe AP in children. Our cases show that conservative treatment with enteral nutrition seems to be an appropriate first-line management for this complication. Furthermore, resolution of symptoms can take up to 6–9 weeks. Endoscopic or surgical gastroenterostomy should therefore only be discussed in patients with fixed (fibrotic) obstruction or failure of conservative treatment.

ACKNOWLEDGMENTS

Isabelle Scheers is a recipient of Research Grants from the Belgian Foundation Against Cancer, FNRS (fondation nationale pour la recherche scientifique), Salus Sanguinis and Fondation Saint-Luc pour le Cancer.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

ETHICS STATEMENT

Informed consent was obtained from both families on behalf of the patient to share case details and imaging.

ORCID

Olivier Leclercq https://orcid.org/0009-0001-5793-4387

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How to cite this article: Leclercq O, Lecomte L, Stephenne X, Scheers I. Duodenal obstruction: a rare complication of severe acute pancreatitis in children. *JPGN Rep*. 2024;5:86-89. doi:10.1002/jpr3.12034