

GASTRIC OUTLET OBSTRUCTION SECONDARY TO ANNULAR PANCREAS WITH SIGNS OF COMPLICATED ACUTE PANCREATITIS AND DUODENAL PERFORATION

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Abstract

We present the case of a male patient with a history of compensated advanced chronic liver disease secondary to hepatitis C virus infection and clinical signs of portal hypertension, who required three hospital admissions due to gastric outlet obstruction caused by duodenal stenosis secondary to acute pancreatitis in the setting of an annular pancreas. The patient had remained asymptomatic until the first episode of acute pancreatitis. Initial management was conservative, including symptomatic treatment and total parenteral nutrition until resolution of the inflammatory process.

He later presented with recurrent vomiting and abdominal pain. Imaging studies revealed multiple intra-abdominal fluid collections and duodenal perforation as complications of the previous episode. Given the severity and unfavorable clinical course, surgical management was indicated once the acute

episode subsided. Portal hypertension significantly influenced therapeutic decision-making, and the surgical approach was adapted to select the safest technique for this high-risk patient.

Keywords: annular pancreas, gastric outlet obstruction, collections.

Introduction

An annular pancreas is a rare congenital anomaly in which pancreatic tissue partially or completely surrounds the duodenum, causing varying degrees of obstruction¹². It is caused by a defect in the rotation and fusion of the pancreatic buds during embryogenesis³. Under normal conditions, the ventral bud migrates around the duodenum to fuse with the dorsal bud; when this process fails, a ring of pancreatic tissue forms around the duodenum³.

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CLINICAL CASE

The exact prevalence is not well established, as many patients remain asymptomatic and the diagnosis may be incidental. Population studies and clinical series suggest that it is probably an underdiagnosed entity in adults [4]. It may be associated with other congenital anomalies and most commonly affects the second portion of the duodenum^{3,4}.

The clinical presentation varies according to age. In the pediatric population, neonatal duodenal obstruction predominates, while in adults the presentation is usually more heterogeneous and often delayed¹. Although many patients remain asymptomatic, the most characteristic clinical manifestation is obstruction of the gastric outlet tract secondary to duodenal compression^{1,5}. Other common symptoms include abdominal pain, nausea, vomiting, and postprandial fullness^{1,5}. Inflammatory processes, especially acute pancreatitis, can act as triggers or aggravators of obstructive symptoms².

Complications include persistent duodenal stenosis, recurrent pancreatitis, duodenal perforation, upper gastrointestinal bleeding, obstructive jaundice, retroperitoneal collections, and intra-abdominal infections^{2,6}.

The diagnosis is based on imaging tests such as contrast-enhanced computed tomography, magnetic resonance imaging, and magnetic resonance cholangiopancreatography, which allow for evaluation of the pancreatic anatomy and associated complications⁴. Upper gastrointestinal endoscopy may reveal extrinsic duodenal compression⁴.

Treatment depends on clinical complexity. In patients with mild symptoms, conservative management may be chosen; however, in cases of significant or persistent obstruction, surgical treatment is the preferred option, with duodenal bypass techniques being the most commonly used^{1,6}. Cephalic duodenopancreatectomy is reserved for selected cases⁶ (Figure 1 and Table 1).

Clinical case

A 64-year-old man with a history of hepatitis C virus-induced liver cirrhosis with sustained viral response and clinically significant portal hypertension.

He came to the emergency room with repeated dark vomiting, with an initial suspicion of upper gastrointestinal bleeding. In the face of uncontrollable vomiting, a nasogastric tube was placed, with a discharge of up to 3000 cc of retained content. An abdominal CT scan was performed, showing obstruction of the gastric outlet tract secondary to paraduodenal pancreatitis in the context of an annular

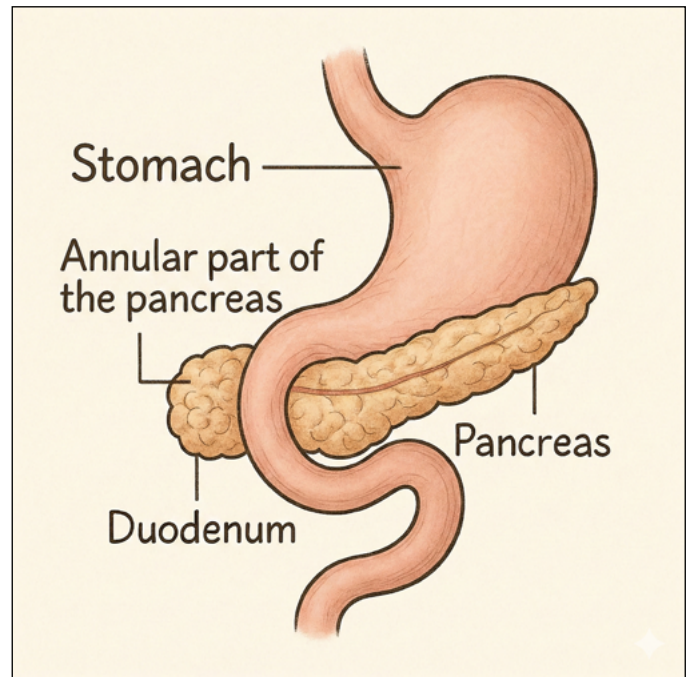


Figure 1. Anatomy of the annular pancreas.

TIPE I	The annular duct empties directly into the main pancreatic duct.
TIPE II	The Wirsung duct surrounds the duodenum but drains into the major papilla.
TIPE III	The annular duct drains into the dorsal surface of the common bile duct.
TIPE IV	The annular duct drains into the common bile duct without connection to the Wirsung duct.
TIPE V	The annular duct drains into the Santorini duct from the ventral side.
TIPE VI	The annular duct drains into the Santorini duct with aberrant fusion.

Table 1. Anatomical variants of the annular pancreas.

pancreas (Figure 2). He progressed favorably with conservative management and was discharged tolerating a pureed diet.

One week later, he returned to the hospital with vomiting and abdominal pain. He was admitted for duodenal stenosis secondary to annular pancreas with retention stomach, complicated by duodenal perforation, intra-abdominal collections, retroperitoneum, vena cava compression, and pleural effusion (Figure 3).

During this admission, he required parenteral nutrition and presented with febrile episodes initially treated with empirical antibiotic therapy with piperacillin-tazobactam. Given the slow progress, percutaneous drainage of collections was performed by interventional radiology, ruling out endoscopic drainage due to location and lack of encapsulation. Purulent material was obtained with growth of *Enterobacter*

cloacae and *Candida* spp., and treatment with cefepime and fluconazole was initiated.



Figure 2. Axial CT scan of the abdomen - Anterior pancreatic tissue surrounding the duodenum consistent with an annular pancreas. There is an alteration of the periduodenal fat as well as inflammatory changes in the duodenal wall related to paraduodenal pancreatitis. Signs of chronic liver disease with portal hypertension (collaterals and splenomegaly).



Figure 3. Coronal CT scan of the abdomen - Fluid collections are identified at the retropancreatic level and in the anterior and posterior right pararenal spaces, accompanied by retroneumoperitoneum bubbles. Obstruction of gastric emptying, with a change in caliber in the second portion of the duodenum. These findings suggest the presence of a pancreatic inflammatory process with possible associated duodenal microperforation. Compression of the inferior vena cava without thrombosis. NG tube ending in the antropyloric region.

He also presented with perihepatic ascites controlled with diuretics and right pleural effusion causing respiratory failure, requiring thoracentesis. Isolated fever spikes persisted, and catheter-associated infection was ruled out. Radiological examinations reveal persistent retroperitoneal, pararenal, and pelvic collections, which are treated with different antibiotic regimens, with progressive improvement in the clinical picture (Figure 4).

Despite the improvement, persistent oral intolerance necessitated hospitalization. The case was presented to the surgical committee, which decided to perform a Roux-en-Y duodenal bypass, ruling out cephalic duodenopancreatectomy due to the risk associated with portal hypertension.

The postoperative period was favorable. The only incident was rapid atrial fibrillation assessed by cardiology, and it was decided not to anticoagulate in the acute phase. Abdominal progress was favorable, with the initiation of oral tolerance and a decrease in collections and pleural effusion observed in imaging controls. He was discharged asymptomatic.



Figure 4. Axial CT scan of the abdomen - Chronic liver disease with signs of portal hypertension (splenomegaly, umbilical vein recanalization, ascites). Retroperitoneal and pararenal collections with air bubbles.

Discussion

Annular pancreas is a rare condition in adults. Although its clinical presentation may be associated with inflammatory processes such as acute pancreatitis, the most characteristic manifestation continues to be obstruction of the gastric outlet tract secondary to duodenal compression, although this form of presentation is less common than in the pediatric population¹⁵. In adults, symptoms are often more insidious and nonspecific, which can delay diagnosis³.

In our case, duodenal obstruction was the main feature of the clinical presentation, occurring in the context of an episode of acute pancreatitis and its complications. The coexistence of both processes illustrates the pathophysiological complexity of annular pancreas in adults, in whom inflammatory phenomena can aggravate preexisting duodenal stenosis and promote the onset of clinically significant obstructive symptoms².

The therapeutic approach depends on the severity of the symptoms and the presence of complications. In mild cases, conservative management with nutritional support and treatment of associated complications may be chosen. However, in the event of persistent obstruction or serious complications, surgical treatment is the option of choice^{3,6}. The most commonly used techniques include duodenal bypass via Roux-en-Y anastomosis, while cephalic duodenopancreatectomy is reserved for selected situations⁶.

In this patient, portal hypertension secondary to liver cirrhosis influenced both the clinical course and therapeutic decisions, increasing surgical risk and favoring complications such as ascites and pleural effusion.

It is worth noting the rare combination in the same adult patient of gastric outlet obstruction and acute pancreatitis secondary to annular pancreas, as well as the added complexity resulting from advanced chronic liver disease. This case highlights the importance of comprehensive clinical and radiological evaluation and a multidisciplinary approach.

Recent studies, including a meta-analysis published in 2024, reinforce that, although many adults remain asymptomatic, duodenal obstruction continues to be the most representative clinical manifestation of annular pancreas, while pancreatitis and other inflammatory processes often act as triggers or aggravating factors of the clinical picture⁴.

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