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REVIEW ARTICLE

Gastrointestinal complications in acute and chronic pancreatitis

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Abstract

Pancreatitis is one of the important medical conditions. Gastrointestinal (GI) complications of pancreatitis are important and lead to significant morbidity and mortality. Diagnosis of these complications is difficult and may require a strong clinical suspicion coupled with various imaging features. This review provides an extensive update of the whole spectrum of GI complication of pancreatitis, both acute and chronic, from inflammation, ischemia, and necrosis to obstruction, perforation, and GI fistulae. The focus is on the clinical and imaging features of this less commonly described aspect of pancreatitis.

Introduction

Acute pancreatitis is defined as a clinical disorder characterized by severe abdominal pain, which is epigastric in location and may or may not radiate to the back, along with raised serum amylase and lipase levels (to more than three times normal).[1] Imaging is not necessary to establish the diagnosis if these findings are present, especially in the first week of illness.

In contrast, chronic pancreatitis is a progressive fibroinflammatory disorder, which is a result of irreversible structural changes with loss of lobular architecture of the pancreas, leading to dilatation of the pancreatic duct, with or without the presence of dystrophic calcification. Chronic pancreatitis eventually leads to exocrine and endocrine deficiency with recurrent episodes of abdominal pain.[2–4]

Acute pancreatitis can either be interstitial or necrotizing. The International Symposium on Acute Pancreatitis, held in Atlanta in 1992, attempted to establish a multispeciality consensus-based classification system for acute pancreatitis. The rationale behind this classification system was to delineate a standard set of terminology that could be used to manage the disease, as well as help in conducting research. However, this classification had its limitations, which included inconsistencies in the definition of the severity of pancreatitis and ambiguous terminology for fluid collections. So, a revised Atlanta Classification was introduced in 2012, which divided acute pancreatitis into early (<1 week) and late (>1 week) phases in relation to the onset of the disease.[1]

The severity of the disease in the early phase is clinically determined by the systemic inflammatory response syndrome and organ failure. Imaging has a limited role in the diagnosis and the severity of the disease in the early phase as morphological changes do not correspond with the clinical findings and do not help determine the subsequent course of the disease. However, imaging may be needed in the early phase (i) to confirm the diagnosis if the clinical symptoms are atypical and/or the serum amylase/lipase levels are not raised up to three times normal, (ii) when malignancy is suspected as an underlying cause, or (iii) to confirm necrosis when the patient is not improving clinically.[1]

The late phase is characterized by ongoing inflammation, organ failure, and complications. Imaging is required in this phase in addition to clinical symptoms (i) to determine the current disease status and complications; (ii) in diagnosing absence

or presence of necrosis and its complications; (iii) to determine whether or not active intervention is required, be it surgical, endoscopic, or radiological; and (iv) to determine treatment response.[1]

Complications

A wide variety of complications are associated with both acute and chronic pancreatitis. These complications may either be local or remote, acute or delayed, and abdominal or systemic. The negative prognostic factors associated with these complications include increasing age, gallstone disease, organ failure on admission, and pancreatic necrosis. Among these factors, pancreatic necrosis is the most common complication and important indicator for the severity of the disease.[5]

Acute pancreatitis. The complications associated with acute pancreatitis can be classified into vascular and nonvascular complications.[6]

- The vascular complications can be either arterial (pseudoaneurysm or active contrast leak) or venous (acute thrombosis of splenic vein, superior mesenteric vein, and portal vein, or collateral formation).
- The nonvascular complications include collections (which are classified according to the revised Atlanta Classification into acute peripancreatic fluid collections, pseudocysts, acute necrotic collections, and walled-off necrosis depending on duration and nature of the collections[1]), bowel complications, and pancreatic fistulas.

Of these, bowel complications have not been described properly in the literature, and due importance has not been given to them due to the priority given to the correction of organ failure. Because to this, these complications can often be missed early on in the disease and can lead to significant morbidity later on. In this article, we will be describing the intestinal complications of acute and chronic pancreatitis separately to provide a complete overview of this cause of morbidity.

Gastrointestinal (GI) complications of acute pancreatitis can be broadly divided into complications caused by action of pancreatic enzymes and complications caused by pseudocyst formation (Table 1).

Table 1 Gastrointestinal complications in acute pancreatitis and associated key imaging features

Due to enzymatic action	Associated with pseudocyst formation
Inflammation	Rupture and fistula
Circumferential mural thickening	Extensive air in a pancreatic collection
Mural enhancement and stratification	Direct communication of a pancreatic cyst and a part of GI tract
	Significant change in size and internal characteristics of a fluid collection
Ischemia and necrosis	Obstruction
Circumferential mural thickening	Dilatation of bowel loops
Mural enhancement and stratification	Pancreatic pseudocyst at the transition point
Marked mural thinning	
Lack of enhancement	
Intramural air	
Obstruction	
Dilatation of bowel loops	
Transition point	
Mural thickening at transition point	
"Colon cut-off" sign	
"Apple core" appearance (pseudocarcinoma sign)	
Perforation	
Discontinuity of the bowel wall	
Contrast leak on contrast enhanced CT pneumoperitoneum or localized air collection around the splenic flexure	
Fistula	
Extensive focal air collection	
Direct extension of a peripancreatic inflammatory changes into a part of GI tract	
Contrast seen entering into peripancreatic inflammatory process on CT performed with intraluminal contrast or as CT fistulogram	
Paralytic ileus	
Diffuse dilatation without any transition point	

CT, computed tomography.

Intestinal complications due to enzymatic action. The bowel complications associated with acute pancreatitis are predominantly due to the pancreatic enzymes released in the mesentery, which can track along different paths to involve different segments of the intestine. The transverse colon and the splenic flexure of the colon are the most common sites of involvement (i) as they are closely related to the pancreas, and severe inflammation of the body and tail may cause extrinsic impression; (ii) retroperitoneal extravasation of enzymes may cause pericolitis and/or pericolic fibrosis; and (iii) thrombosis of the mesenteric vessels can occur due to enzymatic action or due to the background hypercoagulable state in systemic inflammatory response syndrome. In addition, the splenic flexure is the watershed area of the colon, so any compromise in the blood supply secondary to systemic hypotension or mesenteric vascular thrombosis leads to its ischemia and necrosis.[7] The small intestine can also be involved through these mechanisms; however, it is less common and occurs predominantly through retroperitoneal inflammation.

The various bowel complications are not discrete entities but are instead a spectrum that starts with inflammation and ends with perforation or obstruction.

Inflammation. As the enzymes track through the mesentery, they involve the adjacent bowel walls and cause an inflammatory

reaction. This is seen on computed tomography (CT) as circumferential mural thickening with mural stratification.[7] This may progress further to ischemia and necrosis.

Ischemia and necrosis. When the mesenteric vessels are involved by the inflammatory process, blood supply to the involved bowel segment is impaired, leading to ischemia and finally necrosis (Fig. 1). Imaging features of ischemia are indistinguishable from inflammation, showing mural thickening with stratification. The stage of necrosis may be characterized by thinning of bowel wall, with or without pneumatosis.

Obstruction. As described previously, splenic flexure is the most common site for stricturing and obstruction. This is because of the intimate anatomical relationship between the pancreatic tail and the splenic flexure. The pancreatic tail lies within the phrenoleinal and phrenocolic ligaments, which facilitates the direct extension of the pancreatic enzymes from the pancreatic tail to the splenic flexure.[7] These enzymes, particularly lipase, cause fat necrosis, and the released fatty acids form complexes with calcium, leading to calcium deposits.[8] Finally, fibrosis develops with progressive narrowing of the bowel lumen, resulting in proximal dilatation of the ascending and transverse colon, the "colon cut-off sign". Imaging may also show an "apple core" appearance, which may present as a pseudocarcinoma, so a history of acute pancreatitis few months prior must be taken into consideration before evaluating for malignancy.[7]

The treatment for colonic obstruction has classically been resection and primary anastomosis.[9] However, nonoperative treatment in the form of colonic stenting may be performed. Colonic stenting is primarily used in malignant diseases, however, its use in pancreatitis-related disease is not yet fully explored and remains controversial.[10,11] Technical advances in stenting have allowed the possible stenting of the splenic flexure region; however, placing uncovered stents in a benign condition will lead to difficulty in their subsequent removal. Self-expanding stents may be used as a temporary measure until inflammation and obstruction improve; however, experience with other benign diseases suggests that surgery is eventually required due to primary or secondary failure of the stent.[8]

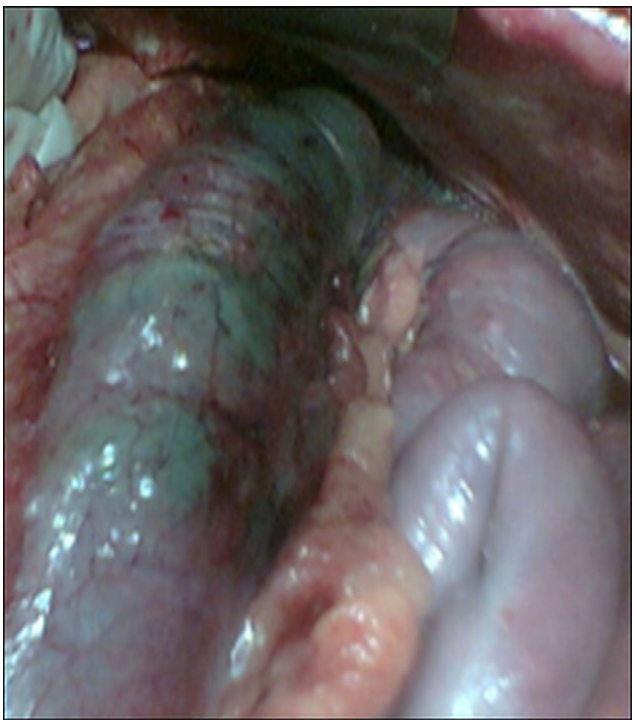

Figure 1 Intraoperative image of a patient with colonic necrosis.

Perforation. The most common site of perforation is the splenic flexure due to the reasons enumerated above. In a retrospective study by Nakanishi *et al.*, it was found that the median interval from the onset of acute pancreatitis to colonic perforation was 13 days, ranging from 6 to 47 days.[12] All the colonic perforations were confirmed by laparotomy, and resection was performed in all with no mortality.

Imaging features of a colonic perforation may be (i) direct visualization of the discontinuity of the bowel wall or contrast leak on contrast-enhanced (CECT) or (ii) indirect sign such as pneumoperitoneum or localized air collection around the splenic flexure. The most significant risk factor for colonic perforation, as described by Nakanishi *et al.*, seemed to be the presence of at least two collections in different locations (corresponding to Balthazar grade E). This was supported by previous studies which showed that perforation was more likely with a Balthazar grade greater than D.[13–15] This is important as Balthazar classification is based on noncontrast CT, which enables us to determine the risk of colonic perforation in patients with deranged renal function, as well as to prevent contrast-induced nephropathy due to repeated contrast studies.

Gastroduodenal (Fig. 2) and caecal perforations have also been reported in the literature, which may be caused by direct contact by the fluid collections with the intestinal segments.[15–18]

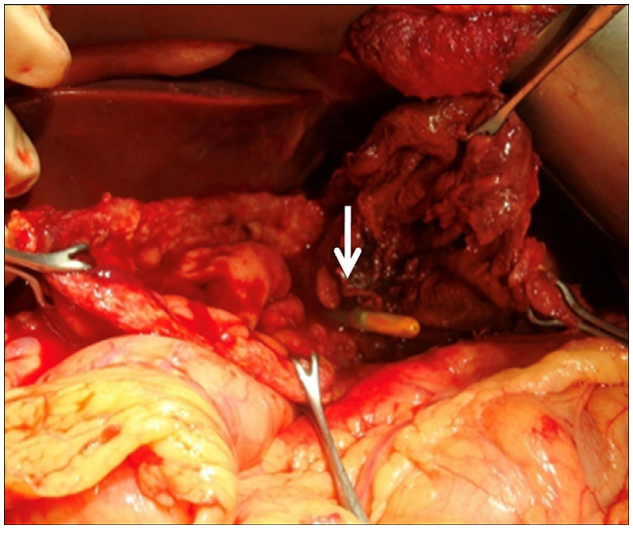

Figure 2 Intraoperative image of a patient with gastric perforation. Ryle's tube is seen protruding out of the perforation (arrow).

GI fistulas. These are a well-known complication of severe acute pancreatitis; however, it has been reported in limited literature. They are generally associated with increased morbidity due to intractable complications like hemorrhage and secondary bacterial infection, leading to increased duration of hospital stay.[19] GI fistulas can form either due to direct enzymatic action of the pancreatic juice on the adjacent bowel wall or due to intestinal necrosis secondary to vascular thrombosis, or they may even arise due to iatrogenic factors like radiological or endoscopic interventions.[20,21]

Clinical features of acute pancreatitis with GI fistula may not differ from those without it. However, occurence of diarrhea, GI bleed, and prolonged sepsis should lead to a suspicion of GI fistula. In a patient with percutaneous catheter drain, increased drain output or frank feculent output also points toward a fistula. Diagnosis is usually made on fistulography (fluoroscopy or CT), endoscopy (Fig. 3), or as an intraoperative finding (Fig. 4).[22] Imaging diagnosis of GI fistula is suggested when there is extensive focal air in a collection, a direct extension of peripancreatic inflammatory changes into a part of GI tract, or contrast is seen entering into peripancreatic inflammatory process on CT performed with intraluminal contrast or as a CT fistulogram.

Jiang at al evaluated 928 patients with acute pancreatitis and found GI fistula in 119 (12.8%, 160 fistula) patients.[23] Incidence was 38.3% in patients with infected pancreatic necrosis (n = 119). Colonic fistulae were the most common form of GI fistulae (60.5%). This was followed by duodenal fistulae (26.6%). Majority of the fistulae (84.9%) were reported after 4 weeks. All duodenal fistulae could be managed successfully by nonsurgical management, while surgical management (in the form of ileostomy or colostomy) was required for 61.1% of the colonic fistulae. The other patients with colonic fistulae were successfully treated by percutaneous drainage or continuous negative pressure irrigation. While the mortality of patients with GI fistulae (as an entire group) did not differ significantly from those without GI fistulae, those with colonic fistulae had a significantly higher mortality.

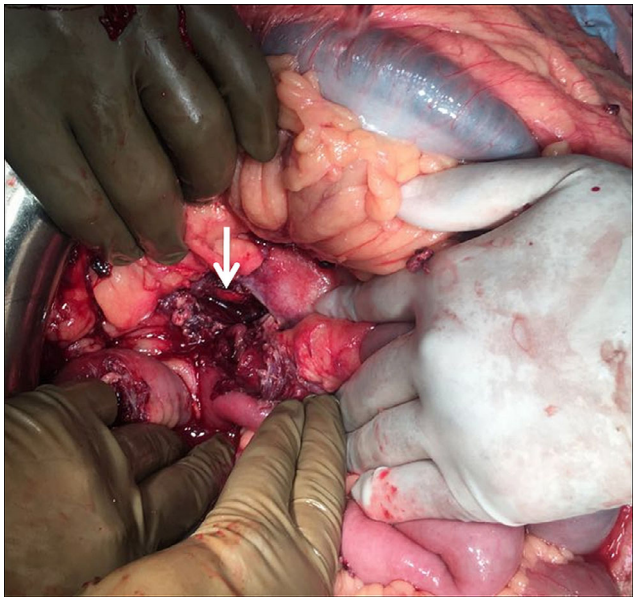

Figure 4 Intraoperative image of a patient with cystoenteric fistula (arrow) with proximal jejunum.

Hua *et al.* studied 344 patients with severe acute pancreatitis and found GI fistulae in 52 patients (15.12%).[24] In this study, only infected necrosis (intra- or extra-pancreatic) and modified CT severity index were found to be independent risk factors for the formation of GI fistulae. Early enteral nutrtion was confirmed to confer protection against this complication. It was also found that 85% of the patients with severe acute pancreatitis developed fistulae after a period of 4–8 weeks, suggesting it to be a delayed complication due to long-term effects of the inflammatory process. Importantly, in this study, fistula resolution was reported in 42 (80.7%)

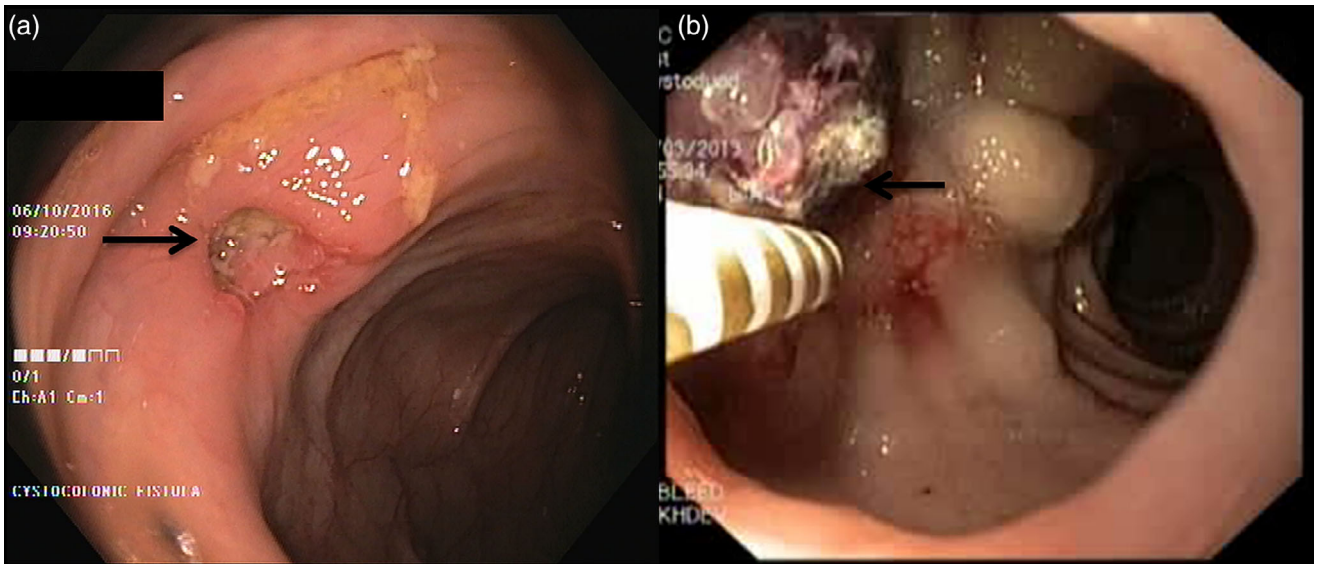

Figure 3 Endoscopic images showing cystocolonic (a, arrow) and cystogastric fistula (b, arrow).

patients after percutaneous drainage or control of infection. Ten patients underwent ileostomy or colostomy.

Management of a GI fistula is based on the principle that upper GI fistulae usually close spontaneously over time, while colonic fistulae usually need active intervention. Upper GI fistulae settle with aggressive control of infection and percutaneous drainage of pancreatic collection. Regarding management of colonic fistula, several recent studies suggest that conservative managment with percutaneous drainage may be sufficient.[21] In a systemic review of colonic complications of acute pancreatitis by Mohamed *et al.*, nonoperative management was suggested for patients with colonic fistulae and stable clinical course. Surgical approach should be immediately adopted if there is GI hemorrhage in the setting of GI fistulae.[21] For patients with stable clinical course who fail to resolve with percutaneous catheter drainage and those with contraindications to surgery, endoscopic treatment with over-the-scope clips has been reported.[25–28]

Paralytic ileus. Colonic paralytic ileus is a relatively common, but less severe, complication of acute pancreatitis. The mechanism is not clearly understood, and it may be attributed to a viscerally mediated reflex within the superior mesenteric plexus secondary to retroperitoneal inflammation or colonic ischemia.[7] On radiographs, there is dilatation of small and large bowel loops, without any transition point. CT scan confirms the lack of mechanical causes and transition point, as well as more dreaded complications like colonic necrosis and fistula (Fig. 5).

GI complications associated with pseudocyst formation or walled-off necrosis

Spontaneous rupture and fistulization. Spontaneous rupture of a psuedocyst can occur either into the peritoneal cavity or the surrounding hollow viscera. The rupture of the pseudocyst occurs once the intracystic pressure reaches a critical level and the weakest point of pseudocyst gives way. Alternatively, the pseudocyst may cause pressure on an adjacent vessel supplying a segment of the bowel, leading to ischemia and necrosis of the bowel wall.[29] Spontaneous rupture can occur into the stomach, duodenum, or colon.

Once the pseudocyst ruptures into the upper GI tract, there is resolution of pressure symptoms. However, patients may sometimes experience vomiting, diarhhea, or melena.[29] On the other hand, colonic fistulization of the pseudocyst is a serious complication as it has the potential for severe haemorhhage and sepsis.[30]

Obstruction. Any part of the GI tract may be obstructed by a large pseudocyst with symptoms depending on the size and location of the pseudocyst.[31] Duodenum is the most common site for obstruction due to direct contact of the second and third part of the duodenum with the head of the pancreas. Ectopic pancreatic resting in the antrum of the stomach may also become enlarged and compromise the lumen, resulting in gastric outlet obstruction.[32]

GI complications associated with chronic pancreatitis. Duodenal obstruction is the most common GI complication associated with chronic pancreatitis, and its incidence in hospitalized patients is approximatedly 1%.[33] Duodenal obstruction may be transient or fixed.[2]

Transient obstruction is seen in the setting of acute pancreatitis flare, resulting from swelling in the pancreatic head and the adjacent duodenal wall.[2] Fixed obstruction occurs due to compression by a pseudocyst or fibrotic changes occuring in the head of the pancreas in chronic pancreatitis.

Groove pancreatitis is another entity of chronic pancreatitis that involves the head of the pancreas near the pancreaticoduodenal groove and results in duodenal obstruction.[34] The diagnosis is usually made on CECT, but it can often be difficult to distinguish it from a malignancy.[35] CECT features include sheet-like soft tissue in the pancreaticoduodenal groove showing delayed enhancement due to the presence of fibrosis. The medial

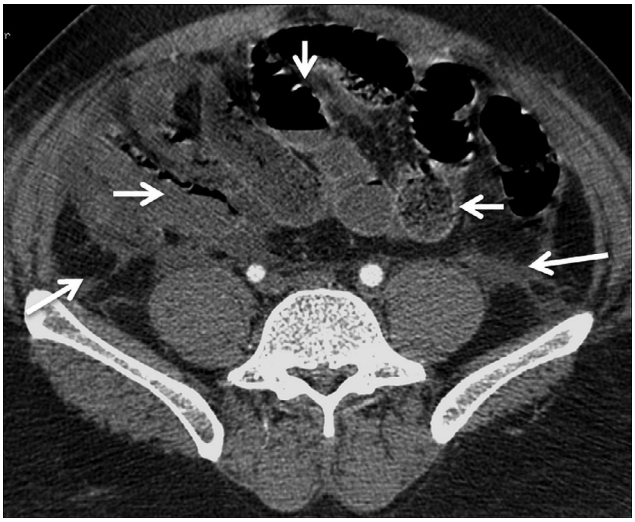

Figure 5 Axial CT image shows dilatation of multiple small bowel loops with air fluid levels (short arrows). Also note the thickening of fascia bilaterally (arrows).

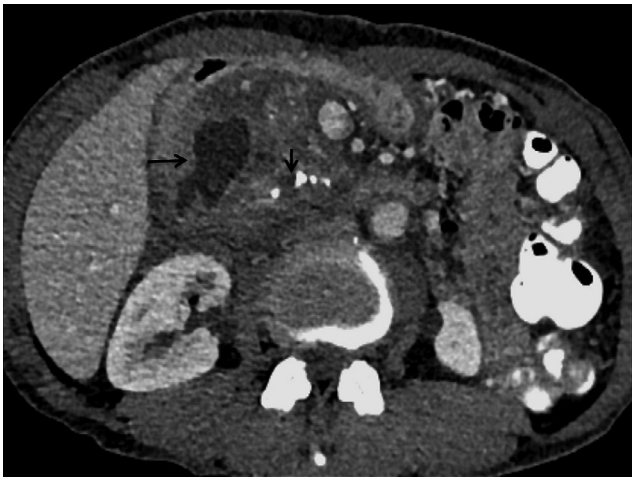

Figure 6 Axial CT shows features of groove pancreatitis in the form of cystic lesion in the medial wall of the second part of the duodenum (arrow) and pancreatic calcifications (short arrow).

duodenal wall may be thickened with the presence of small cysts either in the duodenal wall or the groove itself (Fig. 6).[36,37]

Patients with obstruction because of pseudocysts can be managed by draining the collection endoscopically. Endoscopic duodenal stenting is generally not advised for benign conditions because of the risk of delayed bowel perforation, so obstructions due to fibrosis are generally managed surgically.[2]

Conclusion

Pancreatitis, whether acute or chronic, is a common cause of morbidity and is associated with varied complications, some more life-threatening than others. GI complications, although less common, are still a significant cause for increased morbidity and hospital stay in the clinical course of a patient. Recognizing these complications early and preventing them goes a long way in improving the quality of life of a patient.

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