

Descending colon stenosis as a complication of acute pancreatitis

Zwężenie zstępnicy jako powikłanie ostrego zapalenia trzustki

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Słowa kluczowe: niedrożność jelita grubego, ostre zapalenie trzustki, włóknienie trzustki.

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Abstract

The most common intestinal complications of acute pancreatitis include paralytic ileus, colonic necrosis and intestinal fistula. Large bowel obstruction has rarely been reported. We present the case of a 53-year old male patient with large bowel obstruction, which developed 2 months after his hospitalization for acute pancreatitis. As the initial conservative management was unsuccessful, he underwent laparotomy. On exploration we found a stenosizing tumour located in the splenic flexure and extensive pancreatic necrosis. Hartmann's procedure and left pancreatectomy were performed. Postoperative pathology showed chronic fibrosing inflammation of the pancreatic tail involving the splenic flexure. The postoperative period was complicated by a low-output pancreatic fistula, which healed within a month, and ureteral stones. In the second stage the patient underwent large bowel continuity restoration. We suggest in a patient with signs of bowel obstruction and recent history of acute pancreatitis that a full work-up with CT scan is invaluable before surgical treatment. This helps to choose the best treatment option and the way of surgical access, when necessary.

Streszczenie

Najczęstsze jelitowe powikłania ostrego zapalenia trzustki obejmują niedrożność porażenną, martwicę okrężnicy oraz przetokę jelitową. Rzadko opisywano niedrożność jelita grubego. W niniejszej pracy zaprezentowano przypadek 53-letniego mężczyzny z niedrożnością jelita grubego, która wystąpiła 2 mies. po hospitalizacji z powodu ostrego zapalenia trzustki. W tomografii komputerowej obserwowano masywne poszerzenie jelit do wysokości zagłębia śledzionowego oraz cechy niedokrwienia ogona trzustki. Podczas laparotomii stwierdzono guz zamkający światło zagłębia wątrobowego okrężnicy oraz rozległą martwicę trzustki. Wykonano operację Hartmanna oraz lewostronną pankreatektomię. Na podstawie badania pooperacyjnego rozpoznano przewlekłe zwłókniające zapalenie ogona trzustki obejmujące zagłębie śledzionowe okrężnicy. Przebieg pooperacyjny był powikłany przetoką trzustkową, która zamknęła się w ciągu miesiąca, oraz kamicą moczowodową. W drugim etapie odtworzone ciągłość okrężnicy. Autorzy proponują, aby każdemu pacjentowi z niedrożnością jelita grubego po przebytym ostrym zapaleniu trzustki przeprowadzić przed operacją pełną diagnostykę, obejmującą tomografię komputerową jamy brzusznej. Pozwala to na wybór najlepszej metody leczenia oraz dostępu chirurgicznego, jeśli konieczna jest operacja.

Introduction

Severe necrotizing acute pancreatitis may affect many organs, either by continuity of the inflammatory process or by its systemic consequences. Cases of colonic involvement are rare, but often fatal, with mortality rate > 50% [1]. The most common intestinal complications of acute pancreatitis are paralytic ileus (with cut-off sign), colonic necrosis, intestinal fistula

and stenosis [2]. Large bowel obstruction has infrequently been reported. The predominant site of colonic involvement is the transverse colon or splenic flexure [3]. This location of colonic obstruction is due to the contiguity of the pancreatic tail with the splenic flexure, but also to the anatomy of peritoneal reflexions [4]. The pathomechanism of bowel “invasion” is either acute obstruction due to an inflammatory mass, such as a large pseudocyst, or

pericolic fibrosis. In the latter case the colonic tumour may mimic colon cancer [2]. Those rare complications may develop at different times after acute pancreatitis. They have been described as early as a few weeks after onset of acute pancreatitis and as late as 10 years after the disease [3].



Fig. 1. A computed tomography scan at admission (massively distended transverse colon is visible)

Ryc. 1. Obraz tomografii komputerowej przy przyjęciu (widoczna masywnie rozdęta poprzecznica)

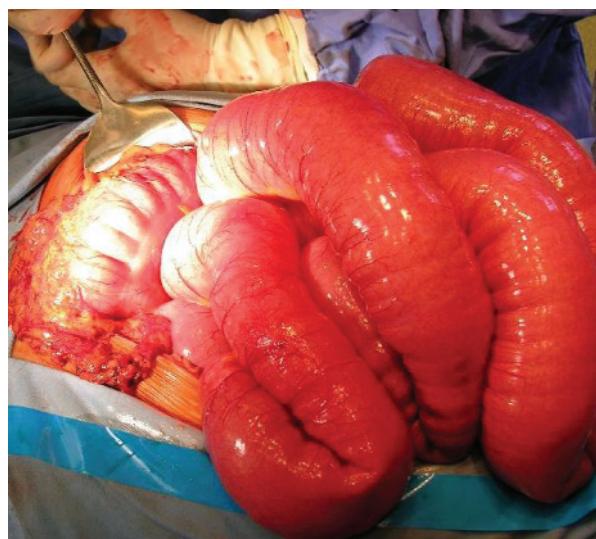


Fig. 2. View of the intestines during laparotomy. Distended transverse colon and small bowel loops are visible

Ryc. 2. Widok jelit podczas laparotomii. Widoczne są rozdęte pętle jelita cienkiego oraz poprzecznica

Case report

We present the case of a 53-year old male patient with large bowel obstruction complicating acute pancreatitis. The patient was admitted with one week history of progressing flatulence, constipation, colicky abdominal pains and eventually vomiting. Two months previously, the patient had been discharged from the Department of Gastroenterology after four weeks' hospitalization for alcohol-induced necrotizing acute pancreatitis treated successfully by conservative means. Surgical history included classical cholecystectomy a few years before.

On admission, the patient was severely dehydrated, but not febrile. The abdomen was grossly distended and tender, without rebound tenderness. The bowel sounds were high. Laboratory indices of inflammatory process were moderately elevated. Abdominal X-ray showed massively distended small bowel loops along with the right colon. The ultrasound was unreliable because of a huge amount of intestinal gases. On computed tomography scan (Figure 1), we observed bowel distension down to the level of the descending colon. No free fluid in the abdominal cavity could be seen. The pancreas was enlarged and its tail showed much poorer contrast enhancement than the head.

As the initial conservative management had been unsuccessful, it was decided to perform laparotomy. Because of grossly distended small bowel loops (Figure 2), manual decompression of intestinal fluid and gases through a naso-gastric tube was necessary, before thorough inspection of the peritoneal cavity could be performed. The bowel was distended down to the origin of the descending colon with no distension below. There was peripancreatic inflammatory infiltrate involving the transverse mesocolon, the posterior wall of the stomach and the left colon. After dissection of fibrous adhesions, we found in the splenic flexure an intramural tumour 3 cm in diameter, causing nearly complete bowel obstruction (Figure 3). Behind it, there was an abscess of 80 ml of white pus with a narrow bore fistula into the bowel lumen. On opening of the omental sac, there was extensive pancreatic necrosis involving the tail and partially also the body of the pancreas, white in colour (Figure 4). Left pancreatectomy with necrosectomy of surrounding tissues was performed. Hartmann's procedure was also performed with resection of the bowel tumour together with the left half of the transverse colon. A stoma was created on the remaining transverse colon. Drains were inserted into the omental cavity, rectovesical recess and subhepatic space, the naso-jejunal tube was introduced and the abdomen was closed.

Postoperative histopathological examination showed chronic fibrosing inflammation of the pancreatic tail involving the splenic flexure.

After the operation, there was prolonged drainage from the pancreatic bed, which declared as a pancreatic fistula. The patient obtained nutritional treatment via parenteral and enteral routes and antibiotics. Additionally the postoperative period was complicated by right-sided ureteral stones.

After four weeks of treatment the signs of inflammation subsided, the patient's general condition and nutritional status improved and the patient was discharged on oral feeding with a low-output pancreatic fistula. The consecutive treatment in the out-patient clinic resulted in spontaneous closure of the fistula in three weeks. Imaging showed no residual fluid collections in the abdominal cavity. The patient underwent restoration of intestinal continuity as a second stage operation, without any complications.

Discussion

Although large bowel obstruction is a relatively common condition, it is rarely due to acute or chronic pancreatitis. In a patient who has recently undergone acute pancreatitis, it is necessary to consider it as a possible cause of bowel obstruction. Among possible aetiologies there are: colorectal tumour (usually cancer), adhesions (the described patient underwent cholecystectomy), ileus due to recurrent acute pancreatitis, and, rarely, local complication of pancreatitis. A bile stone has also been described as a cause of both acute pancreatitis and bowel obstruction [5].

It is not always easy to diagnose large bowel obstruction, especially in a critically ill patient with severe acute pancreatitis complicated with multi-organ failure [6]. As the initial evaluation, it may be useful to perform a contrast enema [3]. Infrequently it might show large bowel stenosis of the splenic flexure, sometimes mimicking a carcinoma [6].

As this patient had no signs of bowel necrosis such as rebound tenderness, fever and acidosis, we initiated a conservative treatment after initial evaluation. The patient's general condition improved with rehydration, but his abdominal symptoms did not settle down. Therefore it was decided to operate. Although laparoscopic intervention has been described for some complications of pancreatitis, as in a case of pseudocyst [7], in this specific case, because of massive intestinal distension and possible pancreatic necrosis, we chose laparotomy. As

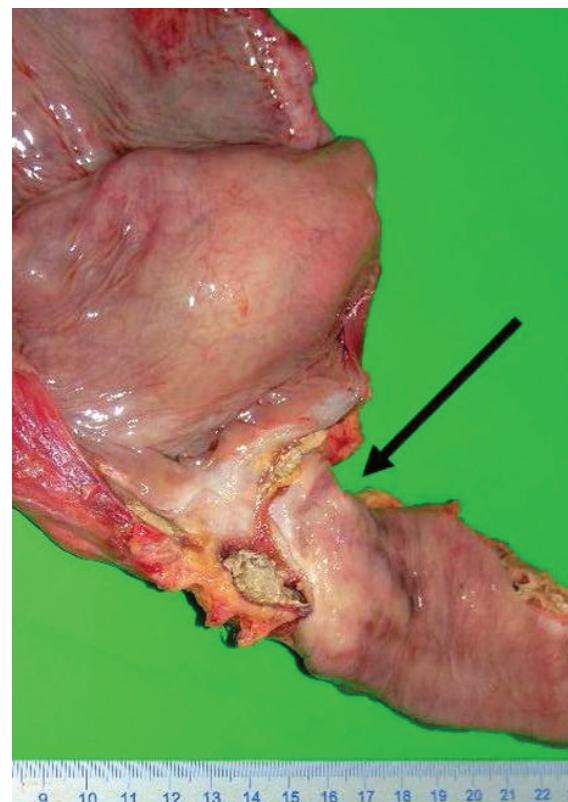


Fig. 3. View of specimen of splenic flexure of the colon. The tumour is visualized (arrow) with grossly distended bowel above and narrow below the place of obstruction

Ryc. 3. Widok preparatu zagięcia śledzionowego okrężnicy. Uwidoczniono guz (strzałka) ze znacznie poszerzonym jelitem grubym powyżej i wąskim poniżej miejsca niedrożności



Fig. 4. View of pancreas during laparotomy. Necrosis of pancreatic tail (arrow) is indicated by its whitish colour

Ryc. 4. Widok trzustki podczas laparotomii. Martwicę ogona trzustki (strzałka) sugeruje biatawy kolor

described in the case presentation, we found a tumour of the left colonic flexure with a fistula to a retroperitoneal abscess and necrosis of the pancreatic tail. The close anatomical relationship between the tail of the pancreas and the left colonic flexure may explain the involvement of the large bowel in some cases of complicated pancreatitis. A few papers published have been described reporting spread of peripancreatic exudate, fibrosis or pancreatic pseudocyst as a cause of significant large bowel stenosis [1, 4, 6].

An important issue remains the choice of surgical treatment. It may range from ileostomy without bowel resection [1] to subtotal colectomy [3]. It has been proposed that the preferred primary management is defunctioning colostomy [8]. Because we found an abscess next to the tumour and there was high suspicion of neoplastic aetiology of the process, we decided to perform a bowel resection and left pancreatectomy. It must be remembered that pancreatic carcinoma might also have a similar clinical presentation as that described in our patient [6]. We decided to delay the anastomosis of the bowel for three reasons: large bowel obstruction with the presence of huge bowel distension, pancreatic necrosis, and pericolic abscess formation.

We were considering leaving the abdominal cavity open after the operation. This procedure is deemed justified in the presence of extensive pancreatic necrosis and pericolic abscess [9]. However, the surgical incision was median and not transverse, the latter being better to control pancreatic necrosis. The infection was not spread in the abdominal cavity. Eventually we decided to close the abdomen after inserting four drains. In seven weeks, we obtained healing of the pancreatic fistula which developed postoperatively. As a second stage the restoration of large bowel continuity was performed, without any complications.

In our view, it is important to remember that acute pancreatitis may in some cases involve the large bowel. We propose that in patients with signs of bowel obstruction and recent history of acute pancreatitis a full work-up with CT scan should be performed before any surgical treatment. This helps to choose the best treatment option and the route of surgical access, when necessary.

References

1. Chung NS, Kim YS, Park CH, et al. A case of colon obstruction developed during the recovery period of acute pancreatitis. Korean J Gastroenterol 2005; 45: 206-9.
2. Negro P, D'Amore L, Saputelli A, et al. Colonic lesions in pancreatitis. Ann Ital Chir 1995; 66: 223-31.
3. Pascual M, Pera M, Martinez I, et al. Intestinal occlusion due to pancreatitis mimicking stenosing neoplasm of the splenic angle of the colon. Gastroenterol Hepatol 2005; 28: 326-8.
4. Büyükerber S, Mahmutyazıcıoğlu K, Ertas E, et al. Ileus secondary to pancreatic pseudocyst. Clin Imaging 1998; 22: 42-4.
5. Fenchel RF, Krige JE, Bornman PC. Bouveret's syndrome complicated by acute pancreatitis. Dig Surg 1999; 16: 525-7.
6. Gardner A, Gardner G, Feller E. Severe colonic complications of pancreatic disease. J Clin Gastroenterol 2003; 37: 258-62.
7. Davila-Cervantes A, Gomez F, Chan C, et al. Laparoscopic drainage of pancreatic pseudocysts. Surg Endosc 2004; 18: 1420-6.
8. Hudson DA, de Beer JD. Acute large bowel obstruction complicating acute pancreatitis. South Med J 1988; 81: 804-5.
9. Sherck J, Seiver A, Shatney C, et al. Covering the "open abdomen": a better technique. Am Surg 1998; 64: 854-7.