CASE REPORT

Recurrent pancreatitis after unremarkable colonoscopy, temporalised by CT imaging: an unusual case

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SUMMARY

Acute pancreatitis is a common surgical presentation, frequently caused by gallstones and alcohol. Here we present an unusual case of a recurrent episode of pancreatitis after an unremarkable colonoscopy, in a patient with several pre-existing risk factors for pancreatitis. Before and after abdominal CT scans clearly demonstrate the acute inflammatory process affecting the pancreas and temporalise its development. Early resuscitation and appropriate involvement of high dependency care is advocated by all current guidelines to improve patient outcome. We consider possible aetiology and how early diagnosis and recognition of possible high-risk situations can expedite its investigation and management, helping to provide the best possible care.

BACKGROUND

Pancreatitis is characterised by inflammation of the pancreatic tissue. Its pathogenesis remains unclear and varies depending on aetiology, although raised intracellular calcium causing cellular necrosis due to increased protease activity is thought to play an important role.¹ The incidence of acute pancreatitis in the UK is 150–450 per million population.² Although it commonly follows a benign course, responding well to conservative management, there are one in four cases where this progresses to severe disease.³ These patients often demonstrate signs of haemodynamic instability progressing to organ failure, and early high dependency input is important. Guidelines for treatment revolve around a few main principles: first, haemodynamic assessment and resuscitation; followed by a risk assessment, typically using the Glasgow or Ranson scoring criteria; and early involvement of high dependency care if appropriate.⁴ A healthy suspicion of acute pancreatitis supported by prompt action is essential.

Part of recognising pancreatitis is being aware of possible triggers, the two most common being gallstones and alcohol. Their prevalence depends on the cohort of patients studied, however, figures vary from 40 to 70% for gallstones and 25–35% for alcohol.⁴ After these, there are many other causes that have been reported such as: trauma (including iatrogenic), drug induced, abnormal anatomy, hypothermia, infections, autoimmune; and those labelled ‘idiopathic’, where no cause has yet been demonstrated.⁵

We present a case of acute pancreatitis occurring in a 69-year-old woman, shortly after colonoscopy.

Interestingly, she underwent an abdominal CT scan before her colonoscopy, demonstrating a normal pancreas. After developing abdominal pain, a subsequent CT demonstrated changes representing acute pancreatitis, demarcating the window in which her pancreatitis developed.

CASE PRESENTATION

This patient had a complicated surgical history: she had undergone appendectomy for appendicitis when young, and cholecystectomy for cholelithiasis a few years prior to this admission. She suffered from Crohn’s disease, which, although under control with mesalazine, caused a hospital admission in January 2011, with small bowel obstruction secondary to a terminal ileum stricture. She required an emergency laparotomy at the time, and right hemicolectomy was undertaken with formation of an end ileostomy and mucus fistula. This ileostomy was complicated by prolapse requiring refashioning in April 2001. Finally, in June 2013, she opted to have her stoma reversed, which was achieved by end-to-end ileocolic anastomosis. Unfortunately, she awoke from this operation with acute pancreatitis; this required high dependence management and, despite full investigation, no cause was found. An autoimmune pancreatitis screen was negative. From these surgical interventions she went on to develop a symptomatic incisional hernia. Throughout all of this, her Crohn’s disease remained under control, with no acute flare-ups and no suggestion of recurrence in the neo-ileum or anastomosis.

She was presented to the general surgical team as part of a work up for difficult eating with abdominal pain and bloating, felt to be a result of her large

Figure 1 Initial abdominal CT as preoperative work up: the pancreas appears normal.

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hernia. A CT abdomen followed by colonoscopy was organised with planned clinic follow-up to consider operative intervention. She took one sachet of Picolax for bowel preparation the day before. Her CT abdomen scan with contrast was unremarkable (figure 1), aside from a large incisional hernia (figure 2). She went on to have an unremarkable colonoscopy; of note, four biopsies from the colon and terminal ileum were taken. Over the following days, she developed severe epigastric pain radiating through to her back, and associated with nausea and vomiting.

On screening for risk factors for intra-abdominal pathology: the patient was an ex-smoker of 1 year, and although she occasionally still smoked, she had not smoked in the weeks preceding this admission; she denied alcohol consumption; she did not take non-steroidal anti-inflammatory drugs on prescription or over the counter; and described no family history of intra-abdominal pathology. There had been no change in her normal pattern of activity or symptoms preceding this presentation.

INVESTIGATIONS

Bloods on admission demonstrated a raised white cell count ($18.3 \times 10^9/L$) and raised C reactive protein (15.6 mg/L). Other bloods were unremarkable: urea 2.4 mmol/L, creatinine 60 mmol/L, albumin 29 g/L, bilirubin 5 $\mu\text{mol/L}$, alkaline phosphatase 125 iu/L, alkaline transaminase 13 iu/L, calcium 2.01 mmol/L and amylase of 91. We do not routinely perform γ -GT and do not have the means to perform lipase, and so have no admitting values for these. CT of the abdomen with contrast demonstrated an acutely inflamed pancreas (figure 3), which was noted to be normal on the comparison CT 5 days earlier (figure 1). Both demonstrate a large incisional hernia of the anterior abdominal wall (figures 2 and 4).

DIFFERENTIAL DIAGNOSIS

The patient described this pain as similar to her previous episode of pancreatitis, so suspicion for this diagnosis was high. Our other primary concern, given the recent colonoscopy, was of perforation and less concerning differential diagnoses included gastritis or symptoms originating from her large abdominal hernia.

TREATMENT

As recommended by international guidelines,⁴ our initial approach involved haemodynamic assessment though the ABCDE approach with fluid resuscitation. Given the concerning

differential diagnoses, a CT was undertaken rapidly, demonstrating pancreatitis as the reason for the patient's symptoms. Her Glasgow Pancreatitis score was 3, scoring points for age, neutrophilia and hypoalbuminaemia, and so discussions were had with the high dependency team. It was agreed that, given her haemodynamic stability, careful management on the ward was appropriate. A catheter was inserted for fluid balance and her oral intake reduced to sips of water.

OUTCOME AND FOLLOW-UP

The patient responded well to this approach, remaining haemodynamically stable with good urine output for the subsequent 24 h. Her symptoms resolved and on the second day her catheter was removed and oral intake increased. We involved the gastroenterology team for advice on her Mesalazine therapy, which was continued. She was then discharged home with both gastroenterology follow-up for continued investigation into her (now recurrent) pancreatitis and Crohn's disease, and general surgical follow-up for ongoing consideration of surgical intervention for her incisional hernia.

DISCUSSION

Given the temporalisation allowed by these two CT scans, we feel the colonoscopy may have played a role in this patient's development of acute pancreatitis. Pancreatitis secondary to colonoscopy is very rare, although it has been reported in the literature. This is the first case of recurrent pancreatitis as a result of colonoscopy that we have been able to find. Two earlier reports describe trauma from passage of the endoscope as probable factors in its development.^{6 7} Specifically, both these cases describe procedural difficulties, particularly around the splenic flexure. In a more recent case, this difficulty was not noted, however, subsequent CT imaging demonstrated haemorrhage around the tail of the pancreas; so given its close proximity to the splenic flexure, it was again felt the trauma of insufflation on endoscopic passage was the cause.⁸

Before colonoscopy there are several choices for bowel preparation, including sodium picosulfate/magnesium citrate (CitraFleet; Picolax) and Moviprep. Side effects of Picolax are quite common, including nausea, vomiting, abdominal pain, anal pain and bloating.⁹ As well as those side effects described, the diarrhoea induced by Picolax causes fluid loss, which without adequate fluid intake can cause dehydration. There is one case report suggesting dehydration as a cause for

Figure 2 Initial abdominal CT as preoperative work up: large incisional hernia through the anterior abdominal wall on the right side is clearly visible.

Figure 3 Second CT to investigate acute abdominal pain: the pancreas is acutely inflamed. No evidence of haemorrhage or other aetiology.

Figure 4 Second CT to investigate acute abdominal pain: again, the large incisional hernia is evident.

pancreatitis,¹⁰ however, this case was after vigorous exercise where trauma has been implicated,¹¹ and we were unable to find any reports associating pancreatitis with Picolax directly, so feel this is unlikely a sole aetiology. It should be noted that our patient herself reported not feeling comfortable taking two sachets of Picolax as prescribed, as she did not think she would be able to tolerate it, and so only took one sachet in preparation for her colonoscopy.

Given the innocuous nature of this patient's colonoscopy, we wonder whether other aetiological factors may have played a role, and made her particularly susceptible to pancreatitis.

Although there was no history of alcohol use and the patient had previously undergone cholecystectomy (with no evidence of ductal gallstones on imaging), there were other factors in her case that have associations with pancreatitis. First, she described a previous episode of pancreatitis. Patients who suffer an episode of pancreatitis are more susceptible to further attacks.¹² This can be as a consequence of persisting aetiological factors; however, in addition, once irritated, the pancreas is more susceptible to other causes of aggravation and inflammation. In this case, we were unable to find a persisting aetiology; however, an underlying susceptibility to inflammation may have been present.

Pancreatitis in its acute and chronic forms is associated with inflammatory bowel disease.¹³ This may be as a consequence of use of medication, for example, mesalazine, which we will discuss shortly, however, there are multiple other suggested interactions. These include a predisposition to various types of biliary calculi, anatomical abnormalities and both an abnormal immunological and inflammatory environment.¹³ Although there were no gallstones demonstrated in this case, these other factors may well have played a role in increasing the patient's susceptibility to acute inflammation.

Over 100 drugs have been associated with acute pancreatitis, accounting for 0.3–1.4% of cases.¹⁴ Specific to this case, mesalazine has been implicated in numerous research articles,¹⁵ and although pancreatitis typically occurs when first starting treatment, cases presenting after a significant duration of therapy, have been recorded.¹⁶ The incidence of drug-induced pancreatitis is noted to be higher in patients with inflammatory bowel disease, most likely due to their medication profile, such as mesalazine use, in this case.

Outside of the causes described above, we have been unable to find any other associations. The patient had been tested previously for autoimmune pancreatitis, with negative results. Lipid

profile was normal. No anatomical abnormalities were demonstrated on imaging. There was no evidence of concurrent infection, hypoglycaemia or other trauma. She described consuming adequate water to remain hydrated, as she was concerned that without this, the hernia would become symptomatic, and she was not prescribed diuretics, making dehydration unlikely. She was not taking azathioprine, 6-mercaptopurine or any medication other than mesalazine for her Crohn's disease.

To summarise, in this case, there are multiple possible contributors to the patient's development of pancreatitis: namely a previous episode of pancreatitis, Crohn's disease and on-going therapy with mesalazine. These issues, however, were all stable at the time of presentation, and so unlikely to trigger an acute episode of pancreatitis. CT images isolate her colonoscopy with the development of acute pancreatitis, and so we believe that the impact of both this procedure and the bowel preparation, in this patient with an already fragile pancreas, may have had a significant role in its aetiology.

Learning points

- ▶ Pancreatitis should be considered in the differential diagnosis for acute abdominal pain in patients having undergone colonoscopy.
- ▶ When planning colonoscopy for patients who have predisposing factors such as a previous episode of pancreatitis, other risk factors should be considered and controlled for as much as possible.
- ▶ The risks and benefits of bowel preparation should be considered carefully in these patients.
- ▶ The endoscopic team should be aware of risk factors for pancreatitis in patients who they are investigating.

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