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Case Report

Disappearing portal venous gas in acute pancreatitis and small bowel ischemia

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ABSTRACT

We report an usual case of hepatic portal venous gas (HPVG) in the setting of acute pancreatitis and small bowel ischemia. Interestingly, the HPVG disappeared within 2 hours of the original computed tomography scan, despite the patient having small bowel ischemia. The patient had a complicated clinical course, dying 62 days postadmission. This case highlights that HPVG in setting of acute pancreatitis and small bowel ischemia has a very high morbidity and mortality, requiring early detection and aggressive surgical management.

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Introduction

Acute pancreatitis affects an estimated 20,000 people annually in the UK [1], and it accounts for the most gastrointestinal related hospital admissions in the United States [2]. A study conducted in Wales (1999–2010) observed that the incidence of acute pancreatitis is increasing annually by approximately 2.7% each year [3]. Rates are also increasing internationally. In the "Japan National Survey," incidence increased from 12.1 to 15.4 per 100,000 between 1987 and 1998 [4]; meanwhile, in the Netherlands, the incidence has increased from 13.2 to 14.7 per 100,000 over the time period 2000–2005 [2]. Reported overall mortality rates vary from 4% to 10%, increasing substantially to 15% to 30% for severe cases [1].

The most common etiology for acute pancreatitis is largely due to biliary pathology (40%) and alcohol misuse (22%). Less common causes include trauma, secondary to iatrogenic procedures (post-ERCP), hypercholesterolemia, drug/medications (corticosteroids, azathioprine), or idiopathic causes [5].

Complications of acute pancreatitis can be either local or systemic events. The Revised Atlanta Classification aims to standardize the reporting of severity of acute pancreatitis to ensure consensus and standardized treatment protocols [6]. Alternatively, acute pancreatitis can also be subdivided into interstitial edematous pancreatitis and necrotizing pancreatitis [6]. Whereas the overwhelming majority of interstitial pancreatitis settles within a couple of weeks, necrotizing pancreatitis can have a more turbulent course. Approximately,

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5%—10% develop necrosis of pancreatic parenchyma [6]. The clinical course of necrotizing pancreatitis is very variable and is associated with the life-threatening complications. Portosplenomesenteric venous thrombosis is reported as high as 50% in patients with necrotizing pancreatitis, but usually has a benign course [7]. Alternatively, hepatoportal venous gas (HPVG) is a rarer complication and more ominous finding [8]. It usually indicates concurrent mesenteric ischemia and is associated with substantial complications [8].

Case report

A 64-year-old gentleman was referred to our hospital from another institution with a diagnosis of acute pancreatitis (Imrie/Glassgow Score = 6). Underlying biliary pathology was the suspected causative factor. Initial computed tomography (CT) scan of the abdomen demonstrated extensive edematous changes involving the entire pancreas with a significant volume of HPVG (Fig. 1). Concern regarding one segment of small bowel was raised. The patient proceeded to have a CT mesenteric angiogram within 2 hours of the original CT scan which showed a poorly enhancing segment of small bowel that was concerning for venous thrombosis (Fig. 2). Interestingly, there was a significant reduction in the volume of HPVG (Fig. 3). Despite this, the patient's clinical condition disimproved and the decision to proceed for emergency laparotomy immediately was decided. At laparotomy, resection of a nonviable ischemic segment of small bowel (60 cm) was performed. Omental saponification was also evident (Fig. 4).

The patient had a prolonged intensive care admission (45 days) for the management of multiorgan dysfunction including acute kidney injury and adult respiratory distress syndrome before being discharged to the ward. The patient was readmitted to the intensive care unit 72 hours later due to overwhelming sepsis. Repeat imaging confirmed large peripancreatic necrotic collections (Fig. 5), and several interventional radiology drainages were performed. Despite escalation in both antimicrobials, antifungal and inotropic support the

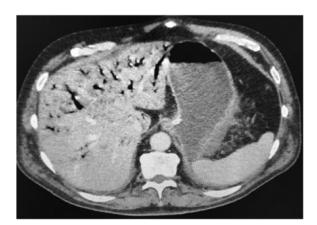


Fig. 1 — CT abdomen (cross-sectional) showing there is a branching linear gas pattern within the hepatic parenchyma extending to the periphery more pronounced in the right liver lobe consistent with extensive portal venous gas. CT, computed tomography.

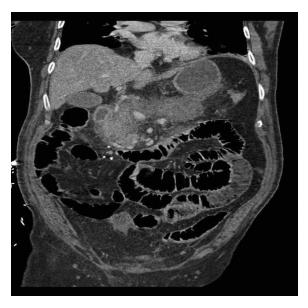


Fig. 2 – CT abdomen showing poorly enhancing segments of small bowel which is concerning for venous thrombosis. CT, computed tomography.

patient's clinical condition deteriorated and he died on the 62nd day of admission.

Discussion

HPVG is a worrisome radiological finding [7]. It typically indicates an underlying life-threatening gastrointestinal pathology such as ischemia and/or inflammation, which frequently requires emergency surgical intervention [9]. Historically, the presence of HPVG has been associated with mortality rates >75% [10], but in contemporary times, mortality is 30%—40% [9]. Traditionally, the presence of HPVG mandated explorative laparotomy, but with improvements in radiological



Fig. 3 – CT Abdomen (cross-sectional) showing small residual focus of portal venous gas in hepatic segment IVb on CT acquired at 2-hour interval (significant reduction). CT, computed tomography.



Fig. 4 – Intraoperative picture showing omental saponification noted at time of laparotomy.

investigations (helical multislice CT), HPVG is now attributed to other benign causes that can be managed conservatively [8].

The underlying pathogenesis of HPVG in pancreatitis is not fully understood. There are several mechanisms speculated regarding its etiology. It is hypothesized that pancreatic enzymes could directly damage the intestinal wall or alternatively the presence of intestinal ischemia allows air to translocate from the bowel lumen into the portal venous system [8,10]. Alternatively, in the case of sepsis, gas producing bacteria such as Escherichia coli may enter into the intestinal wall through mucosal defects and become the source of the HPVG [8,10].

There are some sparse reports of management of HPVG without surgical intervention and some rare incidents of

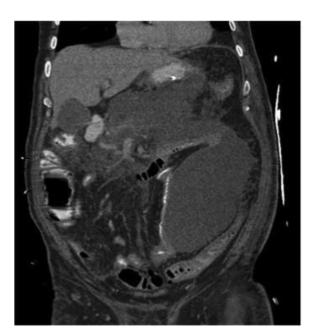


Fig. 5 — Repeated CT scan (coronal section) showing large peripancreatic and left flank collection. CT, computed tomography.

disappearing HPVG. Niki et al. describe a case of disappearing HPVG in a 64-year-old woman with severe lower abdominal pain. The pain resolved quickly, and no underlying pathology was evident on radiological imaging. Repeat imaging showed complete resolution of HPVG within 18 hours of the initial CT scan. She was later diagnosed with terminal ileitis at colonoscopy [11].

Martin-Smith et al. reported a case of extensive pancreatic necrosis with HPVG, splenic vein thrombosis, and pneumatosis coli of the cecum and ascending colon. The patient was managed conservatively. Repeat CT scan 5 days later showed progression of pancreatic necrosis but with complete resolution of the HPVG. By day 12, the patient was asymptomatic, diet was reintroduced, and biochemical markers were all improving [12]. However, Park et al. [8] reported two cases of acute pancreatitis with intestinal ischemia and HPVG that had very poor outcomes.

Our case is quite unusual as the HPVG rapidly disappeared (within 2 hours) despite the patient having small bowel ischemia. However, we still advocate that exploratory laparotomy was necessary, especially when imaging raised the possibility of nonviable bowel and when there was a significant deterioration in patient's clinical status. A recent study from Korea observed a lower mortality rate (40%) for patients undergoing prompt emergency laparotomy, compared with those who did not undergo surgery (100% mortality) when imaging predicted HPVG was unlikely to resolve with conservative management [13]. Key indicators for prompt surgery included the presence of intraperitoneal free air or HPVG with nonviable bowel on CT imaging [13].

The presence of HPVG in acute pancreatitis is a poor prognostic sign, requiring early detection combined with aggressive (surgical) management, especially if associated with mesenteric ischemia. It indicates a complicated disease course and is associated with high morbidity and mortality.

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