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Gastric Pneumatosis in Acute Pancreatitis: A Diagnostic Dilemma

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Gastric pneumatosis is the presence of air within the stomach wall, which may be associated with a spectrum of clinical entities ranging from benign gastric emphysema to ischaemic gastropathy to fatal emphysematous gastritis. Ischaemic gastropathy is rare due to the rich collateral blood supply of the stomach. In this case report, we present a unique case of ischaemic gastropathy secondary to acute necrotizing pancreatitis with splenic vein thrombosis. Our patient showed a remarkable recovery with timely management. We highlight the role played by imaging in early diagnosis and differentiation of benign gastric emphysema and gastric ischaemia from the more lethal emphysematous gastritis, thus guiding appropriate management.

Case Report

A 68-year-old woman presented with severe epigastric pain radiating to the back and vomiting for one day. Physical examination was notable for epigastric tenderness with no palpable mass. The patient was afebrile and preliminary lab investigations revealed leukocytosis (16800cells/mm^3; normal range 4000-11000 cells/mm^3) with neutrophilia (9472cells/mm^3; normal range 2000-7000 cells/mm^3), elevated serum amylase level (4230U/L; normal range 22-80 U/L) and lipase level (10690 U/L; normal range <67U/L). Serum lactate levels were normal (1.7 mmol/L; normal range < 2 mmol/L).

Conservative management, including fluid resuscitation by administration of IV fluids, IV analgesics for the pain, and parenteral feed for bowel rest, was done.

However, the patient continued to have severe abdominal pain and vomiting. Due to a lack of symptomatic improvement despite conservative management, abdominal ultrasonography was done on day 5 of admission. It revealed an edematous and bulky pancreas with a poorly circumscribed peripancreatic fluid collection.

Esophagogastroduodenoscopy (EGD) done on the same day for nasojejunal tube placement was unremarkable.

Contrast-enhanced CT abdomen performed within an hour after EGD demonstrated a bulky pancreas with non-enhancing areas within and an ill-defined heterogenous peripancreatic collection, suggestive of acute necrotizing pancreatitis with acute necrotic collection. Long-segment splenic vein thrombosis was seen **(Figure 1)**.

Gastric fundus and body showed non-enhancing mucosa with extensive intramural air, extending into the immediate perigastric soft tissue, and the portal venules. Several small venous collaterals were seen within the gastric wall **(Figure 2,3)**. These findings suggested the diagnosis of gastric ischemia.

Mucosa in gastric antrum showed normal enhancement. The coeliac trunk and its branches were patent and of normal caliber.

Given the discrepancy between findings on the EGD and CECT scan, a repeat endoscopy was performed on day 7 of admission, which revealed multiple gastric mucosal ulcerations and sloughed-off mucosa along the greater curvature, thus confirming the diagnosis of ischaemic gastropathy **(Figure 4)**. Patient was promptly started on heparin infusion.

After a multidisciplinary team discussion, a laparoscopy was done on day 10 of admission to assess the severity of gastric ischaemia, the presence and extent

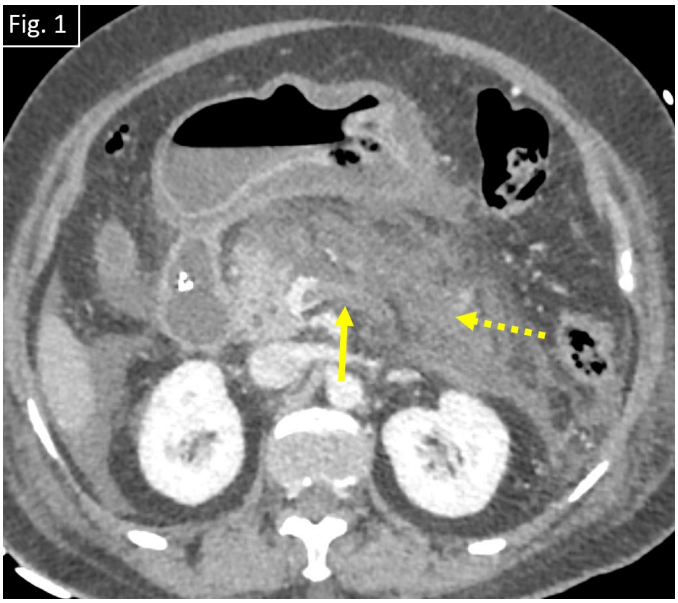

Figure 1: Representative venous phase axial CECT image on day 5 of illness showing non-enhancing necrotic pancreatic parenchyma (dotted arrow) suggestive of acute necrotizing pancreatitis with thrombosis of splenic vein (solid arrow).

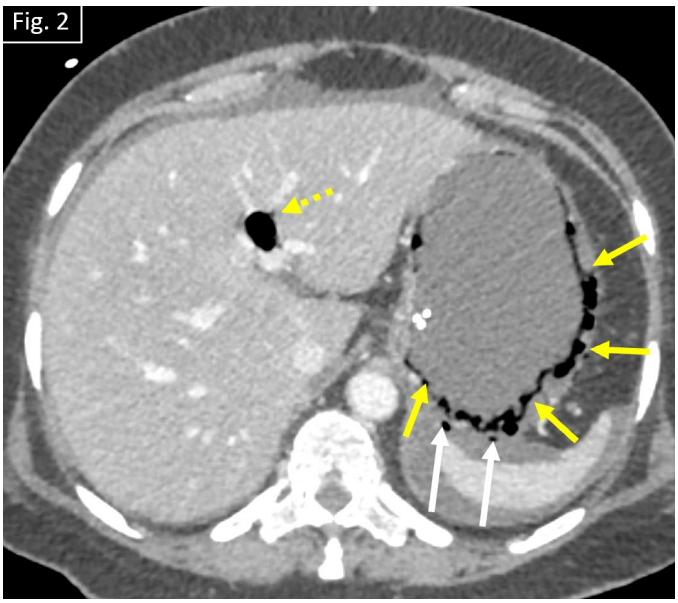

Figure 2: Representative venous phase axial CECT image showing gastric mural air (solid yellow arrows), air in small gastric veins (white arrows) and portal venous air (dotted yellow arrow).

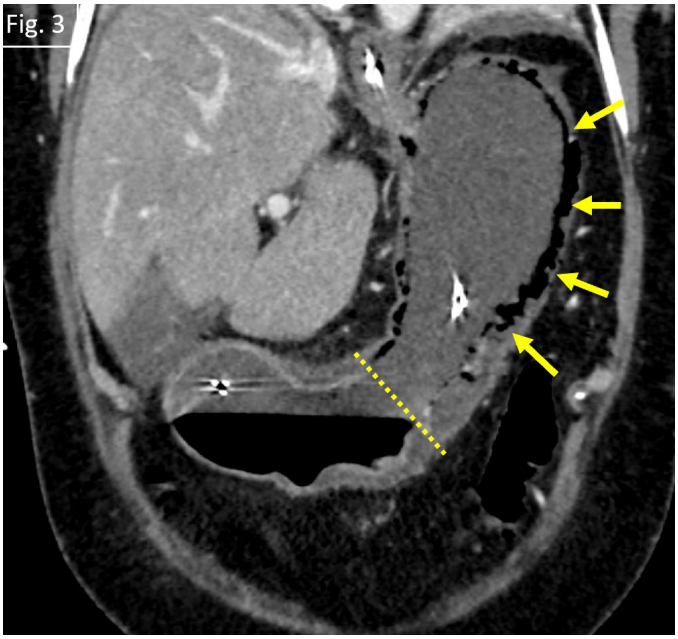

Figure 3: Representative venous phase oblique coronal CECT image showing gastric mural air (solid arrows) with lack of overlying mucosal enhancement to the right side of dotted line. The distal stomach wall shows preserved mucosal enhancement (to the left side of dotted line).

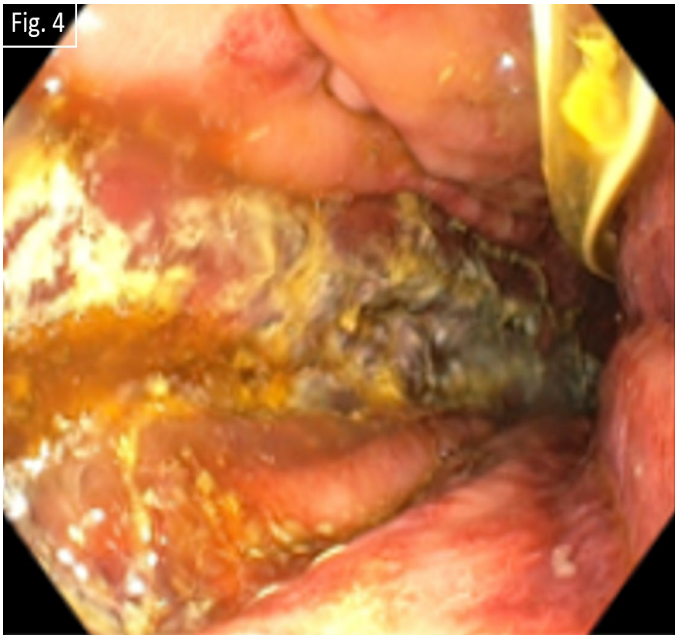

Figure 4: Upper GI endoscopy image on day 7 showing erythematous gastric mucosa with ulcerations and few areas of sloughed off friable mucosa, suggestive of ischaemic gastropathy.

of gastric necrosis, and perform partial gastrectomy if indicated. However, it revealed a normal exterior stomach surface with normal indocyanine-green (ICG) enhancement and delayed ICG washout in the posterior wall of the stomach, suggesting a viable gastric wall. Lavage of the gangrenous fluid in the lesser sac was performed and a drainage tube was placed.

Following anticoagulation and supportive therapy, a repeat CT on day 15 of admission showed complete resolution of the gastric mural air and portal venous air with good restoration of gastric mucosal enhancement **(Figure 5)**. The peripancreatic necrotic collection showed temporal evolution with a partly defined wall.

The patient was discharged 3 weeks from illness onset in stable condition. She was asymptomatic at 1-month and 3-month follow-up visits. Abdominal ultrasound with colour doppler showed resolution of pancreatic necrotic collections and chronic thrombosis of the retropancreatic splenic vein.

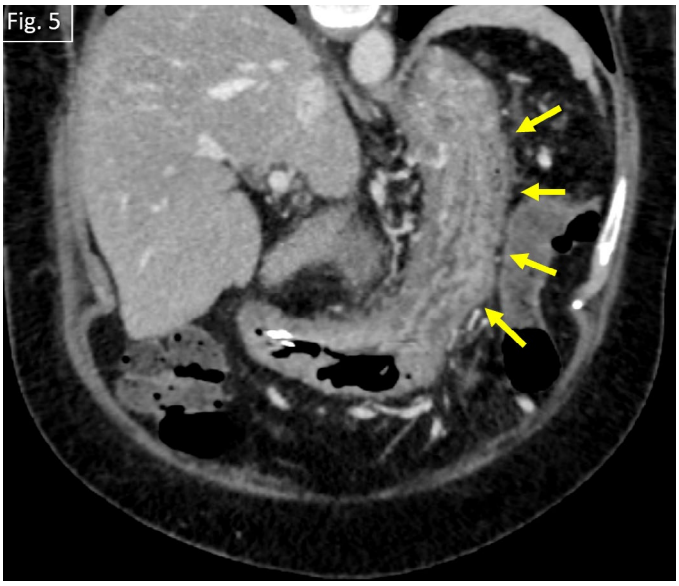

Figure 5: Representative venous phase oblique coronal CECT image on day 15, post conservative management, showing completely resolved gastric mural air with restored gastric mucosal enhancement.

Discussion

The presence of air in the gastrointestinal wall can be seen anywhere from the esophagus to the rectum, with the stomach being the least common site due to its rich collateral blood supply, accounting for less than 9% of all reported cases.[1] The presence of air in the stomach wall is termed Gastric pneumatosis (GP) with multiple factors playing an interactive role in its formation, including alteration in mucosal integrity, intraluminal pressure and gaseous distension, and bacterial flora.

GP can be seen with a spectrum of clinical entities ranging from benign gastric emphysema and ischaemic gastropathy to emphysematous gastritis, which have widely varied management algorithms and prognostic implications. Radiologist plays a crucial role in differentiating between these distinct entities showing overlapping imaging findings.

Benign gastric emphysema is caused by a disruption in the gastric mucosal integrity due to increased intragastric pressure, leading to the entry of air into the wall. Patients present with mild or no symptoms. Imaging features include linear distribution of submucosal gastric air locules and portal venous air. It is seen without wall thickening or perforation, which helps differentiate it from other causes of gastric intramural air. It is a self-limiting condition requiring conservative management and has an excellent prognosis.[2]

Ischaemic gastropathy is a rare entity due to the rich gastric vascular supply by the celiac artery and its branches. It occurs due to decreased gastric blood flow, either by vascular insufficiency or following reperfusion injury. Its clinical presentation is that of an acute abdomen, unlike benign gastric emphysema. Imaging findings are characterized by linear distribution of intramural air locules, and absence of overt stomach wall thickening, with or without air in the gastric venules and portal vein, like benign gastric emphysema.[1] However, reduced or absent wall enhancement points towards the ischaemic pathology.

In our case, the definite lack of gastric mucosal enhancement and the presence of splenic vein thrombosis helped in clinching the diagnosis.

Management includes anticoagulation, intermittent nasogastric suction with acid suppression[1],

and broad-spectrum antibiotics. Surgery is recommended when there is no response to conservative management or in case of gastric perforation.[3]

Emphysematous gastritis is a fulminant condition caused by gas-forming organisms in immune-compromised patients. It is associated with systemic toxicity and a high mortality rate.[4]

Both emphysematous gastritis and ischaemic gastropathy can present with intramural air locules, perigastric fat stranding, and portal venous gas on imaging. However, the presence of irregularly thickened gastric mucosal folds with a mottled pattern of intramural air is seen in emphysematous gastritis, which helps differentiate it from ischaemic gastropathy, where the mucosal folds of normal thickness are associated with a linear pattern of intramural air locules.

It needs prompt aggressive management with IV broad-spectrum antibiotics and supportive therapy. Barring perforation, surgery should be postponed until the sepsis is better controlled.[5]

Although we come across many patients with acute pancreatitis complicated by extensive splenoportal venous thrombosis in our tertiary care referral center, this is the first case presenting with gastric ischemia. We found splenic vein thrombosis to be the only contributing factor for gastric ischemia in this case. There was no documented systemic hemodynamic compromise or arterial insufficiency at any point during the illness.

Interestingly, the EGD performed one hour before the CT scan revealed normal gastric mucosa while repeat endoscopy two days after the CT confirmed gastric mucosal necrosis. This suggests that CT might be able to identify early signs of gastric ischaemia before manifesting on endoscopy. The timely CT diagnosis leading to prompt initiation of anticoagulation and the presence of gastric mural collaterals could have contributed to the resolution of ischemia, which was well documented in the follow-up CT scan.

Our literature search found that very few cases of gastric ischaemia complicating acute pancreatitis have been previously reported.[6,7] Most of them were associated with reduced perfusion secondary to arterial thrombosis.[7] Only one documented case report of gastric ischaemia in the background of necrotizing pancreatitis and splenic vein thrombosis was noted, where total gastrectomy was done as heparin infusion was not curative.[6]

Conclusion

It is crucial to differentiate benign gastric emphysema from more worrisome gastric ischemia and emphysematous gastritis, both of which benefit from early diagnosis and prompt treatment. Knowledge of the clinical presentation and combination of CT scan and EGD are essential for an accurate diagnosis.

Abbreviations:
GP - Gastric pneumatosis
NJ - Nasojejunal
EGD – Esophagogastroduodenoscopy
CECT - Contrast enhanced computed tomography

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