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Case Report

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# *C. perfringens* Blood Stream Infection due to Nontransmural Ischemia of the Esophagus, Stomach, and Left Colon: Case Report

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**Keywords**
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**Abstract**
We report the case of a 74-year-old female with abdominal pain, tarry stools, and tachycardia. Previous history included diabetes mellitus with micro- and macroangiopathy. Imaging revealed portal gas, left sided colitis, and emphysematous gastritis, besides severe atherosclerosis with subtotal celiac trunk occlusion and moderate stenosis of the inferior mesenteric artery. Upper endoscopy revealed findings consistent with focal necrotizing gastritis at the greater curvature and acute esophageal necrosis. Blood cultures immediately grew *Clostridium perfringens*. The patient was treated with broad spectrum antibiotics and was discharged after 21 days in the hospital. This case demonstrates the rare coincident occurrence of nontransmural ischemia of the left colon, the esophagus, and the stomach as a result of low-flow circulatory compromise, which then precipitated *C. perfringens* associated emphysematous gastritis and blood stream infection.



## Introduction

Mesenteric ischemia is a dreaded acute condition because of its dramatic presentation, its typical occurrence in elderly, often multimorbid patients, and its generally believed poor prognosis. It may manifest as transmural or nontransmural (mucosal) necrosis due to either occlusion of a large arterial or venous vessel or due to low-flow circulatory compromise, especially in patients with congestive heart failure, chronic kidney disease, or diabetes. Ischemic colitis is well recognized as usually due to low-flow circulatory compromise. As such, it carries a much better prognosis than occlusive mesenteric ischemia of the small bowel for instance due to an embolism to the superior mesenteric artery.

*Clostridium perfringens* is a rod-shaped, Gram-positive, anaerobic, spore-forming pathogenic bacterium. It is a well-known cause of a self-limited food poisoning [1, 2]. Sepsis due to *C. perfringens* is uncommon and is not to be confused with emphysematous myonecrosis. It carries a mortality rate exceeding 50% [3–5]. The source of *C. perfringens* sepsis most commonly is the hepatobiliopancreatic system usually in conjunction with diabetes, malignancy, or severe immunocompromise making it an important pathogen for gastroenterologists and GI surgeons [3, 6, 7]. Occasionally, emphysematous gastritis has

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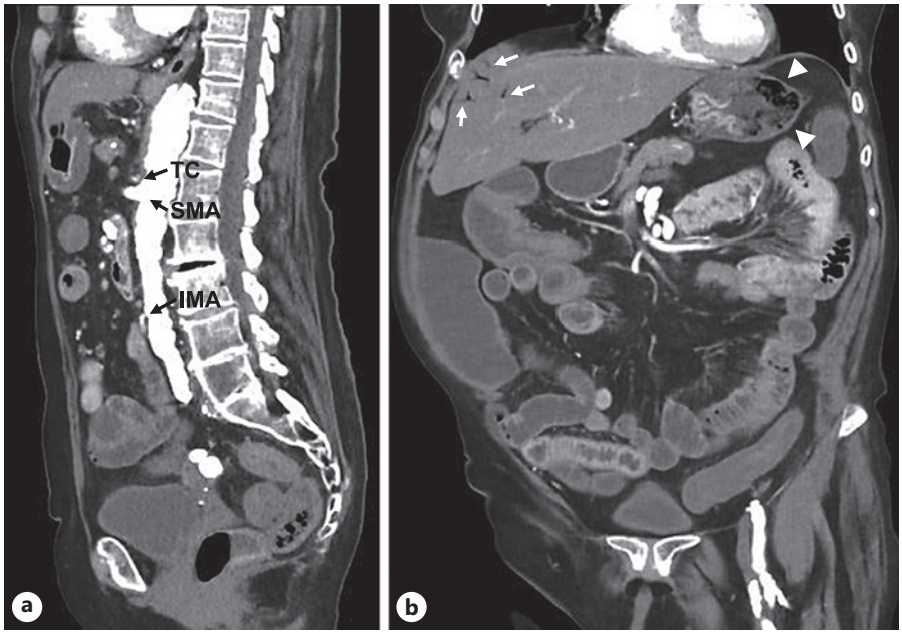


**Fig. 1.** CT findings: arterial phase. **a** Sagittal reformation of the contrast-filled aorta – note subtotal stenosis of celiac trunk (TC), prominent superior mesenteric artery (SMA), and stenosed inferior mesenteric artery (IMA). **b** Coronal reformation – note the intrahepatic portal gas, indicated by arrows. The source appeared to be intramural gas formation in the stomach, indicated by arrowheads. The inner lining of the lesser curvature is well contrasted, while the greater curvature lacked contrast uptake. Also note the thickened sigmoid colon in the left lower quadrant showing little contrast uptake.

been observed in the context of immunosuppression, however most often with fatal outcome [8–11]. We here report a case of successful medical treatment of *C. perfringens* blood stream infection due to nontransmural ischemia of the esophagus, stomach, and left colon.

## Case Report

A 74-year-old female presented with slurred speech and dizziness to the stroke unit. Cerebral MRI showed general leukoencephalopathy but no signs of ischemia or hemorrhage. The patient also reported abdominal pain, reduced appetite, and vomiting, as well as tarry stools for 2 days, and was therefore transferred to the medical emergency room. Previous history included long-standing type 2 diabetes with micro- and macroangiopathy, chronic kidney disease, and occlusive peripheral arterial disease with previous balloon dilatation of the right femoral artery. There was no history of tobacco or alcohol use. Vital signs were HR 112/min, RR 136/58 mm Hg, $SpO_2$ 92% on room air, respiratory rate 14/min, and temperature 35.8 °C. The abdomen was tender, absent bowel sounds were noted, and rectal examination revealed tarry stool. Laboratory values at presentation showed hemoglobin 7.2 mmol/L, leukocyte count 8.7 Gpt/L, CRP 372 mg/L, procalcitonin 129 ng/mL (ref. <0.064 ng/mL), creatinine 338 μmol/L, and urea 28.6 mmol/L. CK was elevated at 937 U/L (ref. <170 U/L). Bilirubin, glucose, lactate, and pH were normal. The chest X-ray performed thereafter revealed pulmonary congestion and aortic sclerosis. Volume resuscitation was initiated. Blood cultures were drawn, and piperacillin/tazobactam was immediately started. Bed-side ultrasonography depicted small amounts of interenteric free fluid, wall thickening of the left colon, a prominent common bile duct (8 mm), and widespread portal gas in the liver. The contrast-enhanced CT scan confirmed portal gas and showed a severely atherosclerotic aorta, as well as subtotal celiac trunk occlusion with arterial collaterals in the epigastrium. The superior mesenteric artery was patent, and the inferior mesenteric artery showed moderate stenosis. The gastric wall at the lesser curvature appeared well perfused; however, the gastric wall at the greater curvature was not and presented signs of intramural gas. The left colon appeared poorly perfused (Fig. 1). Explorative laparotomy was performed because of suspected colonic gangrene. It revealed signs of blood in the cecum but was otherwise normal. Of note, the stomach as well as the left colon appeared macroscopically normal. Upper endoscopy was performed in search of the source of GI bleeding; it showed signs of necrotic esophageal mucosa from the gastroesophageal junction up to 25 cm. In the stomach at the greater curvature, a large, map-like superficial mucosal necrosis was visible (Fig. 2).

Blood culture immediately grew Gram-positive rods, later to be identified as *C. perfringens*. In addition, *Morganella morganii* was detected later. Antibiotic therapy was switched to meropenem and clindamycin. Therapeutic unfractionated heparin was started and later switched to low molecular weight heparin. Clopidogrel, which the patient was receiving for her peripheral arterial disease, was continued. Of note, the patient required unexpectedly low amounts of insulin in the ICU given the underlying diabetes. Also of note, the patient did not require pressor support or ventilation or dialysis/CVVHD. Recurrent tarry stools required transfusion of six units packed red blood cells. The patient was kept nil per os for 3 days and diet was advanced very carefully thereafter. After 6 days in the ICU, the patient recovered uneventfully from her sepsis. GFR remained diminished at 54 mL/min/1.73 $m^2$ and anemia persisted (Hb 5.4 mmol/L). The patient was discharged home after 21 days in the hospital.

## Discussion

Mesenteric ischemia may result from acute embolization, atherosclerotic plaque rupture, or venous thrombosis, or it may be due to low-flow ischemia. The paradigmatic manifestation of low-flow ischemia in the splanchnic bed is ischemic colitis, which is due to an acute, self-limited, apparently segmental decrease in perfusion rather than a specific vascular lesion or embolic event [12, 13]. Another much less common manifestation of low-flow ischemia is acute esophageal necrosis (AEN; "black esophagus"), most

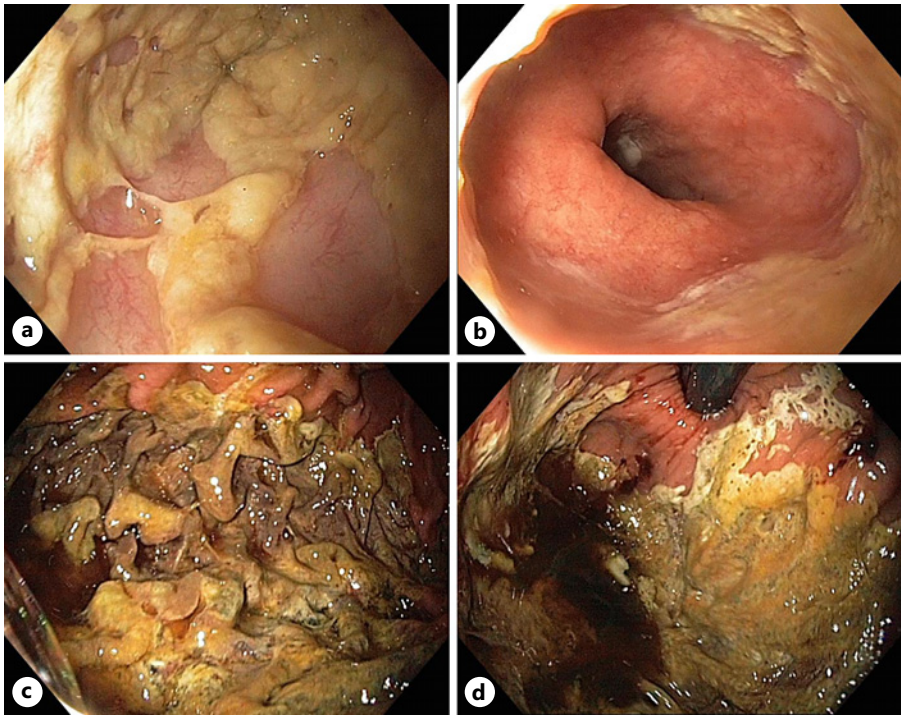


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**Fig. 2.** Endoscopic findings. **a** Confluent fibrinous plaques on ulcerated esophageal mucosa. **b** Abrupt transition at the gastroesophageal junction. **c**, **d** Map-like superficial gastric necrosis in the antegrade view (**c**) and in the inverse view (**d**).

often due to general hemodynamic compromise [14]. Low-flow ischemia of the stomach is extremely rare, but has been described when mesenteric macroangiopathy coexists [15–19].

In this case, the CT findings were highly suggestive of nontransmural ischemia of the left colon. In addition, it showed emphysematous gastritis. Upper GI endoscopy showed AEN. The fact that nontransmural necrosis occurred in three different arterial beds underscores low-flow circulatory compromise as the common cause. It also indicates that the manifestation is not limited to the colon but may occur elsewhere. Severe atherosclerosis and subtotal occlusion of the celiac trunk with large collaterals at the pancreaticoduodenal arcade may have been an underlying factor. Single vessel disease as a reason for chronic mesenteric ischemia as an indication for revascularization remains controversial [20]. Because the patient did not have symptoms of chronic mesenteric ischemia, we opted against revascularization in the acute epidose.

Upper GI endoscopy showed a large, map-like area of superficially necrotic appearing mucosa at the greater curvature. While endoscopic findings of ischemic colitis and AEN are well described, the endoscopic appearance of nontransmural ischemia in the stomach is not well characterized. This case adds another example to the endoscopic findings of ischemic injury to the stomach. The rare complication of a *C. perfringens* infection also makes this case remarkable. The patient presented with the clinical picture of *C. perfringens* blood stream infection, which is typically accompanied by hypoglycemia and hemolysis, the latter likely being present in our patient given only mild tarry stools but the requirement of 6 units of packed red bloods cells. This highlights the notion that mucosal necrosis may have been the entry site for sometimes commensal *C. perfringens* [12], which otherwise usually originates from the biliopancreatic system [3]. Another entry site appeared unlikely. On the one hand, food poisoning pathophysiologically would be "intestinal infection," a toxin-mediated process presenting as high-volume watery diarrhea, for which the patient had no recent history [21]. On the other hand, clinical examination of the patient did not reveal any wounds or ulcers of extremities or skin.

Therapeutic approach was based on the principles for low-flow ischemia: (a) inhibition of the plasmatic and corpuscular coagulation, (b) antimicrobial therapy to treat blood stream infection and to counteract further translocation, and (c) repair of low-flow circulatory compromise to the degree possible.

Taken together, this case reminds us that nontransmural ischemia often results from low-flow circulatory compromise and that it can manifest in other segments of the GI tract apart from the colon. It adds to the endoscopic description of nontransmural necrosis in the stomach, which may have been the entry site of *C. perfringens* with its characteristic presentation and usually poor prognosis. It also reminds us of the usually nonoperative or nonresective treatment.

## Statement of Ethics

Written informed consent was obtained from the patient for publication of the details of their medical case and any

accompanying images. Medical Ethics Committee approval was not required for this case report in accordance with local practice.

## Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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## Author Contributions

J.R. and G.L. wrote the manuscript. K.R., I.K., M.W., A.P., and C.S. provided surgical details. F.S. and M.-A.W. provided radiological information. D.M. and B.B. provided medical insight and details. All authors read and approved the final version of the manuscript.

## Data Availability Statement

Data can be made available upon request in anonymized form. CARE checklist: https://www.equator-network.org/reporting-guidelines/care/.

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