


Gastric ischemia complicating acute pancreatitis

Paul Chanzy and Francois Philippart *

Medical and Surgical Intensive Care Department, Groupe Hospitalier Paris Saint Joseph, Paris, France

*Correspondence address. Medical and Surgical Intensive Care Department, Groupe Hospitalier Paris Saint Joseph, 185 rue R. Losserand, Paris 75014, France.

Tel: +33144123085; E-mail: fphilippart@ghpsj.fr

A 50-year-old patient with excessive alcohol use and no other clinical history presented to the emergency department with epigastric pain and hematemesis. The initial biological tests showed were leucocytes: 17 500/mm³, neutrophils: 90.9%, CRP: 7 mg/l, platelets: 88 G/l, lipase: 708 UI/l, total bilirubin: 43 μmol/l, arterial blood lactate: 16 mmol/l, bicarbonate: 17 mmol/l,

CPK: 7818 UI/l (peak: 18 155 UI/l) and LDH: 2089 UI/l. Upper digestive tract endoscopy showed a hypoperfused aspect of the mucosa from the esogastric junction to the third segment of the duodenum. A CT-scan performed at the time of admission showed edematous-interstitial pancreatitis and thrombosis of the splenic vein.

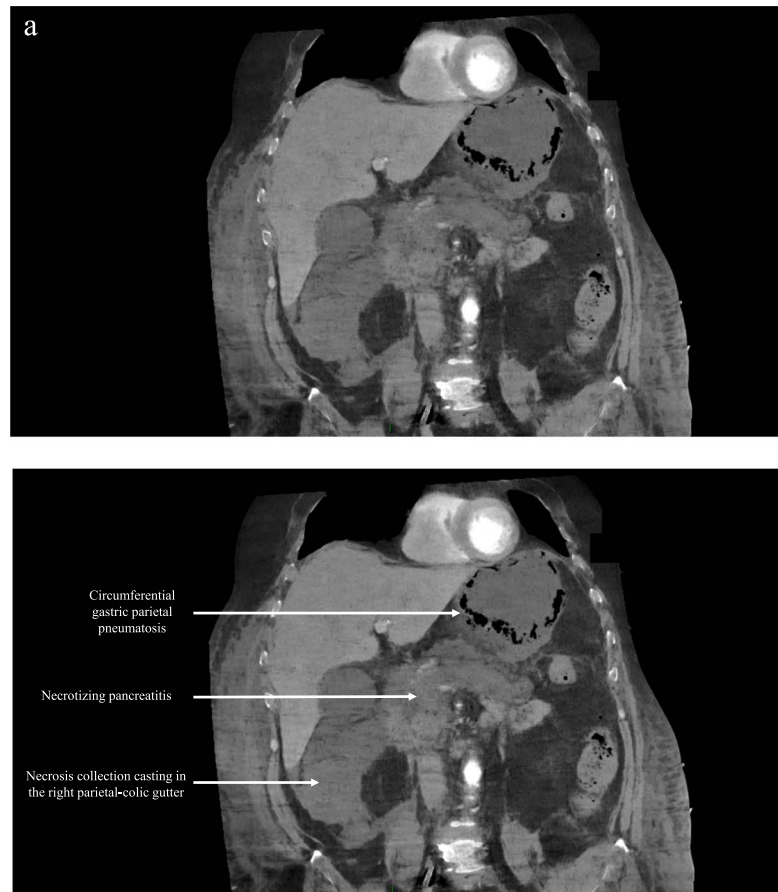


Figure 1. (a) Frontal view: gastric pneumatosis (arrow n°1) associated with absence of enhancement of the gastric wall by the contrast product at portal time. Pancreatic necrosis (arrow n°2) complicated by necrosis flows (arrow n°3). (b) Day 1: arterial time: (a) splenic and gastric ischemia, (b) celiac trunk without gastric vascularization and (c) hepatic artery permeability. (c) Day 6: coronal view. Arterial time. Renal and digestive arterial vascularization. Of note: reduction of blood contrast favored by inflammatory shock.

Received: August 3, 2022. Revised: April 29, 2023. Accepted: May 23, 2023

© The Author(s) 2023. Published by Oxford University Press. All rights reserved. For Permissions, please email: journals.permissions@oup.com

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<https://creativecommons.org/licenses/by-nc/4.0/>), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited.

For commercial re-use, please contact journals.permissions@oup.com

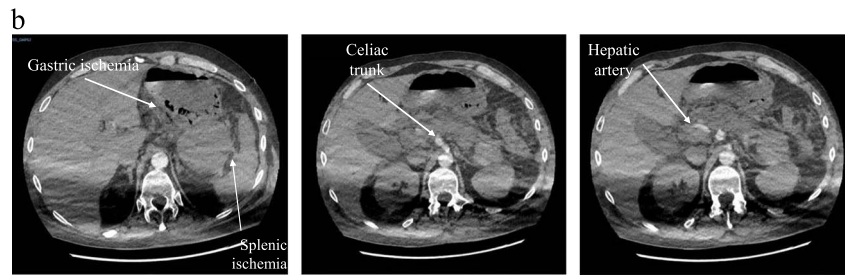


Figure 1. Continued.

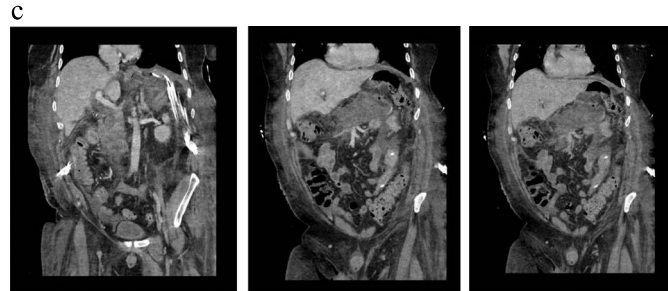


Figure 1. Continued.

The context of probable inhalation led to the introduction of empirical antibiotic therapy with piperacillin and tazobactam. Respiratory tract aspirates showed the presence of *Enterobacter cloacae* and *Klebsiella oxytoca*, leading to modification of the antibiotics to Cefepime. The initial inflammatory and septic shock required the introduction of norepinephrine (peak: 3.5 $\mu\text{g}/\text{kg}/\text{min}$) concomitantly with vascular expansion by crystalloids (3500 ml on Day 1). Three dialyses were performed in the first 3 days. Curative anticoagulation was initiated.

Secondary hemodynamic deterioration at D6 led to a new CT-scan (Figure 1), which revealed necrotizing pancreatitis of the entire organ, with a collection due to necrosis casting in the right paracolic gutter, associated with a diffuse parietal enhancement defect of the stomach and circumferential gastric parietal pneumatosis subsequent to contiguous vascular thrombosis. Biological analysis at that time showed leucocytes: 26 600/mm³, neutrophils: 89.6%, arterial blood lactate: 2.1 mmol/l and bicarbonate: 17 mmol/l. Of note, the administration of anticoagulation therapy with heparin during this period was not curative. The patient underwent a total gastrectomy without the restoration of digestive continuity. A splenectomy and cholecystectomy were performed at the same time due to ischemia. The clinical evolution was initially unfavorable, requiring a second surgery due to perforation of the esophagus by the gastric tube on D6 and cardiac arrest and septic shock (*E. cloacae* and *Enterococcus faecium*

bacteremia) on D43. The subsequent course was favorable, allowing transfer for rehabilitation. The restoration of digestive tract continuity (esojejunal anastomosis and bile-duodenum anastomosis) was carried out after 7 months, followed by the closure of the lateral esophagostomia 1 month later.

Very few cases of gastric ischemia complicating acute pancreatitis have been previously reported [1–5].

REFERENCES

1. Yu HM, Myung E, Kim SH, Myung DS, Cho SB, Lee WS et al. Gastric and splenic infarctions in acute pancreatitis. *J Dig Dis mai* 2016;**17**: 340–3.
2. Ishikawa T, Itoh A, Kawashima H, Ohno E, Itoh Y, Nakamura Y et al. Peripancreatic vascular involvements of autoimmune pancreatitis. *J Gastroenterol Hepatol* 2012;**27**:1790–5.
3. Scholefield JH, Goodman AJ, Morgan WP. Abdominal wall and gastric infarction in acute pancreatitis. *Pancreas* 1988;**3**:494–6.
4. Kumaran C, Chung AF, Ooi LPJ, Chow PH, Wong WK. Coeliac artery trunk thrombosis in acute pancreatitis causing total gastric necrosis. *ANZ J Surg* 2006;**76**:273–4.
5. Rieger A, Bachmann J, Schulte-Frohlinde E, Burzin M, Nährig J, Friess H et al. Total gastric necrosis subsequent to acute pancreatitis. *Pancreas* 2012;**41**:325–7.