

Case Report

Fatal *Clostridium perfringens* sepsis due to emphysematous gastritis and literature review



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ARTICLE INFO

Article history:

Received 26 January 2016

Received in revised form

25 March 2016

Accepted 25 March 2016

Available online 30 March 2016

Handling Editor: Andrew Bruce Onderdonk

Keywords:

Emphysematous gastritis

Clostridium perfringens

Sepsis

CT-scan

ABSTRACT

A 76-year-old female patient was admitted to the Level I Emergency Department of University of Szeged with severe abdominal pain and vomiting. The clinical assessment with laboratory tests and radiological investigations confirmed severe sepsis associated with intravascular hemolysis and multiorgan failure and acute pancreatitis. On the abdominal CT, besides of other abnormalities, the presence of gas bubbles in the stomach, small intestines and liver were seen. The gastric alterations pointed to emphysematous gastritis. Despite of the medical treatment, the patient's condition quickly deteriorated and eight hours after admission the patient died. The autopsy evaluation revealed systemic infection of abdominal origin caused by gas-producing Gram-positive bacteria, and the post-mortem microbiological cultures confirmed the presence of *Clostridium perfringens* in many abdominal organs. Emphysematous gastritis seemed to be the primary infectious focus.

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1. Case report

A 76-year-old woman was admitted to the Emergency Department (ED) with abdominal pain and vomiting. Her previous medical history included obesity (Body Mass Index: 36.86 kg/m²), hypertension, generalized atherosclerosis and cholecystectomy. There were no clinical data about that the patient suffered from manifest diabetes mellitus, however, the elevated serum glucose level (8 mmol/l) might have been the part of metabolic syndrome. On admission the patient's vital signs were stable, with mildly decreased body temperature (35.2 °C), but the abdominal examination was difficult to perform due to the severe diffuse pain on palpation. Rectal digital examination showed no abnormalities, but the inserted nasogastric tube drained substantial amount of coffee ground like gastric juice. Initially arterial blood gas (ABG) analysis referred to compensated metabolic acidosis. Oxygen and volume therapy were started and the patient was transferred to the

Resuscitation Room of the ED for further monitoring, including invasive blood pressure monitoring (IABP). Blood samples for laboratory and microbiological investigations were sent and abdominal ultrasound (US) then computer tomography (CT) were requested.

The results of laboratory investigations referred to septicemia (elevated White Blood Cell count ($2,361 \times 10^7$ cell/mL), sensitive CRP (68,60 mg/L), procalcitonin (6.92 ng/ml)) with multiorgan failure, acute pancreatitis (elevated level of alpha-amylase (1165 U/L) and lipase (2766 U/L)), renal failure (blood urea nitrogen (12,11 mmol/L), creatinine (158 µmol/L) and intravascular hemolysis (hemoglobin (152 g/L)), deterioration of liver function with elevated liver enzymes (aspartate-aminotransferase (476 U/L), alanine-aminotransferase (363 U/L), gamma glutamil-transferase (210 U/L), alkaline phosphatase (150 U/L)).

CT scans of the lung showed atelectasis, dominated in the right lower lobes. CT of the abdomen showed gas in the stomach wall (Fig. 1). This pathological phenomenon pointed to emphysematous gastritis, which is a rare, but potentially fatal disease characterized by the invasion of the stomach wall by mostly gas-producing bacteria. In the small intestines - especially in the duodenum wall - and in both lobes of the liver air were detected (Fig. 2). Wall of the colon

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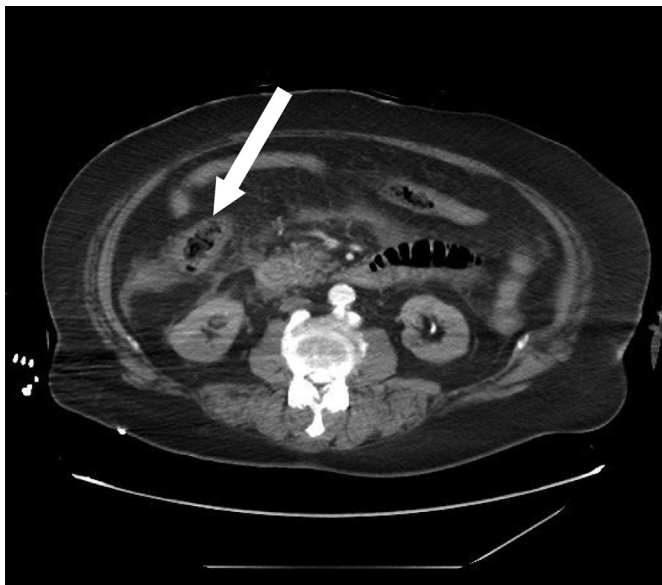


Fig. 1. CT scan of the stomach. Gas within the stomach wall can be seen.

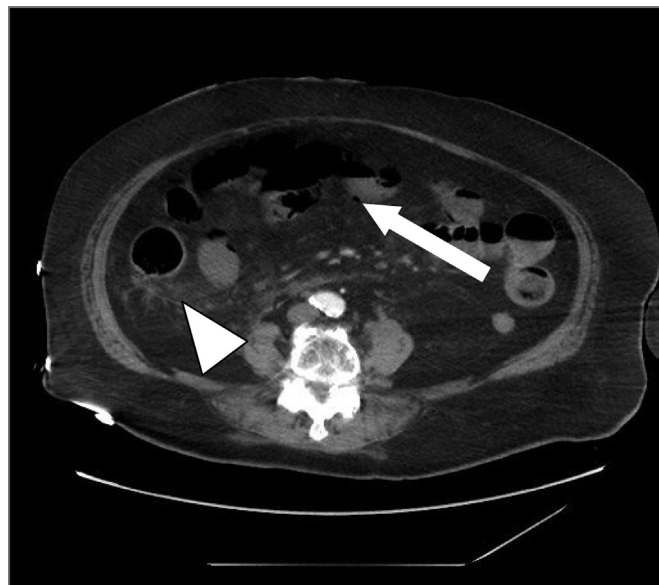


Fig. 3. CT scan of the large intestines shows thickened wall with gas (arrow) and pericolic adipose tissue infiltration (arrowhead).

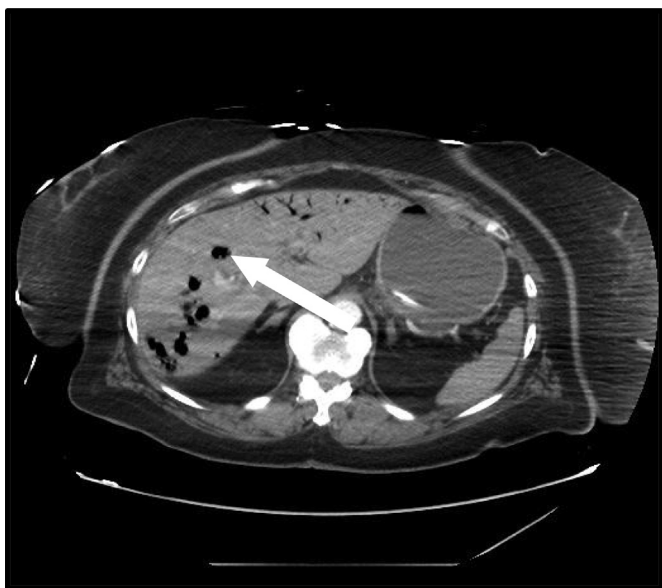


Fig. 2. CT scan of the liver with large amount of air.

edema, map-like purplish red (in the web version) discoloration and remarkable crepitation of the skin and the gastric mucosa with cobblestone appearance (Figs. 4 and 5). The internal examination showed air predominantly in the subcutaneous and abdominal adipose tissue, gastric and duodenal walls, liver, and virtually in all organs except the brain. There were no signs of trauma or perforation of the gastrointestinal tract. Histopathology demonstrated necrotizing infection of the skin, subcutaneous tissues, liver, small and large intestines, and stomach with the abundance of entrapped gas, surrounded by numerous Gram-positive bacteria with almost total absence of acute inflammatory cell infiltrates (Fig. 6). Post-mortem microbiological culture of intestinal and subcutaneous tissue of the chest revealed the simultaneous presence of Gram-positive thick rods, which was identified as *Clostridium*



Fig. 4. Autopsy findings of the stomach. The mucosa is brownish with cobblestone appearance.

was moderately thickened and in the pericolic adipose tissue of hepatic flexure inflammatory infiltration could be observed (Fig. 3). Despite the complex intensive care supportive therapy given at the ED – including monitoring, oxygen therapy, aggressive fluid replacement and broad spectrum antibiotics (imipenem/cilastatin) – the patient's condition has rapidly deteriorated therefore she was transferred to the Intensive Care Unit (ICU). After the ICU admission the patient's condition became critical. Due to respiratory failure the patient was intubated, then ventilated and noradrenaline was started for circulatory support. The combined therapy showed no effect though and approximately eight hours after the primary ED admission the patient died. In order to be able to substantiate the underlying pathology, autopsy was requested and performed within 48 h after the patient's death. The autopsy revealed signs of advanced autolysis and putrefaction, with severe, generalized

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