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Fulminant Emphysematous Hepatic Abscesses Due to Clostridium perfringens With Massive Intravascular Hemolysis: A Case Report

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Abstract

Clostridium perfringens is a gram-positive, anaerobic, gas-forming bacterium that can cause fulminant sepsis. Its alpha-toxin may induce massive intravascular hemolysis, and gas-forming hepatic infection with this organism often progresses to death within hours.

An 80-year-old man presented to the emergency department with acute epigastric pain radiating to the back, tachycardia, hypertension, and fever. Triple-phase computed tomography demonstrated two gas-forming hepatic abscesses in segments 7 and 8 with portal venous gas, while the gallbladder appeared normal. He developed a severe high anion gap metabolic acidosis, hyperkalemia, and indirect hyperbilirubinemia with a low haptoglobin and a rapid hemoglobin drop, consistent with massive intravascular hemolysis. Percutaneous drainage could not be performed because of rapid hemodynamic deterioration. He suffered a cardiac arrest with transient return of spontaneous circulation, followed by a second, fatal arrest two hours and 11 minutes after presentation. Blood cultures drawn at presentation grew *C. perfringens, Enterococcus faecalis, and Serratia marcescens*, and post-mortem examination with culture of the gallbladder wall identified the gallbladder as the primary source of infection.

This case illustrates that gas-forming hepatic abscesses combined with unexplained hemolysis should immediately raise suspicion of *C. perfringens* sepsis and that a radiologically normal gallbladder does not exclude it as the source. Given the extremely compressed clinical course, early recognition and emergent source control are critical.

Categories: Emergency Medicine, Infectious Disease, Gastroenterology
Keywords: acalculous cholecystitis, clostridial sepsis, clostridium perfringens, gas-forming liver abscess, intravascular hemolysis, metabolic acidosis, portal venous gas, post-mortem microbiology, septic shock

Introduction

Clostridium perfringens is a gram-positive, spore-forming, anaerobic bacillus that forms part of the normal gastrointestinal flora. *Clostridium* species are the second most common cause of anaerobic bacteremia, accounting for 0.5-2% of all bacteremias, and the population incidence of *C. perfringens* bacteremia has been estimated at 1.8-4.8 per 100,000 person-years [1]. Cancer and immunosuppression are the comorbidities most consistently associated with *C. perfringens* infection [1]. In a review of 40 published cases of *C. perfringens* septicemia with massive intravascular hemolysis, diabetes (30%), hematological disorders (22.5%), and pancreatic or gastric cancer (12.5%) were common underlying conditions, and the focus of infection was most often hepatobiliary (45%) [2].

The virulence of *C. perfringens* is largely mediated by its alpha-toxin, a phospholipase C (lecithinase) that hydrolyzes phospholipids in red blood cell membranes; the resulting loss of membrane integrity leads to rapid lysis of red blood cells within the circulation, which can cause massive intravascular hemolysis [3,4]. The reported mortality of *C. perfringens* septicemia is at least 60% [4], and in a recent series, the 30-day mortality of *C. perfringens* bacteremia approached 30%, rising to 43% in patients with cancer [1]. Once massive intravascular hemolysis develops, the course is particularly fulminant: 80% of reviewed patients died, with a median interval between admission and death of only eight hours [2], and death within hours of presentation despite maximal resuscitative efforts has been described [4,5]. Reported complications include acute kidney injury requiring hemodialysis and multiorgan failure requiring intensive organ support [6,7]. Diagnosis is challenging, as blood cultures may take many hours to become positive and the source of infection is not always apparent on initial imaging, so that the diagnosis must often be suspected on clinical grounds alone.

We report a case of fulminant *C. perfringens* sepsis with gas-forming hepatic abscesses and massive intravascular hemolysis in an 80-year-old man, who died two hours and 11 minutes after presentation to the emergency department. This case is reported for three reasons. First, it illustrates the extremely narrow therapeutic window of this entity, which left no time for source control. Second, post-mortem examination

identified the gallbladder as the primary source of infection, although the gallbladder appeared normal on initial computed tomography (CT). Third, it highlights the bedside clues, gas-forming hepatic lesions combined with unexplained intravascular hemolysis, that should prompt early suspicion of *C. perfringens* sepsis and emergent source control before microbiological confirmation is available.

Case Presentation

History and physical examination

An 80-year-old man presented to the emergency department with acute epigastric pain radiating to the back. His medical history included lumbar laminectomy (L4-L5), multiple denervation procedures, and robot-assisted laparoscopic prostatectomy. He had no relevant home medication other than low-dose aspirin (Asaflow). He described severe epigastric pain of an unclear character, radiating to the back, occurring at rest and progressively worsening; nausea was the only associated symptom.

On examination, the patient was tachycardic (100/min), hypertensive (163/91 mmHg), and febrile (38°C) and appeared acutely unwell and diaphoretic. Abdominal examination revealed a rigid, diffusely tender abdomen.

Differential diagnosis

Given the patient's age and the acute onset of epigastric pain radiating to the back, life-threatening causes were prioritized, principally aortic dissection and septic shock of intra-abdominal origin. Other diagnoses considered included gastric perforation, pancreatitis, cholecystitis, acute coronary syndrome, bowel perforation, and bowel infarction.

Investigations

A bedside Rapid Ultrasound for Shock and Hypotension (RUSH) examination was performed shortly after presentation, alongside the initial blood work-up. It demonstrated hyperdynamic cardiac function, no intra-abdominal free fluid, and a normal abdominal aortic diameter.

Initial blood work showed an elevated leukocyte count of 24.2×10^{9}/L with a C-reactive protein of 144 mg/L. A point-of-care venous blood gas on admission showed a deep high anion gap metabolic acidosis (pH 7.11, base excess −16.4 mmol/L, lactate 13.7 mmol/L) with hyperkalemia (K+ 6.9 mmol/L). A metabolic panel showed hyperbilirubinemia of 6 mg/dL, predominantly indirect, and a haptoglobin of 0.37 g/L, consistent with intravascular hemolysis (Table *1*).

Parameter (unit)	Result	Reference range
Leukocytes (×10⁹/L)	24.2	4.2-10.3
Hemoglobin (g/dL)	12	13.3-17.1
C-reactive protein (mg/L)	144	<3.0
Total bilirubin, predominantly indirect (mg/dL)	6	0.3-1.2
Haptoglobin (g/L)	0.37	0.40-2.80
pH (venous)	7.11	7.32-7.43
Base excess, venous (mmol/L)	−16.4	−2.0 to +3.0
Lactate, venous (mmol/L)	13.7	0.50-2.20
Potassium, venous (mmol/L)	6.9	3.5-5.1

TABLE 1: Initial laboratory results on presentation

Reference ranges are those of the Antwerp University Hospital (UZA) laboratory for adult men.

Rotational thromboelastometry (ROTEM) is summarized in Table *2*. Clotting times were severely prolonged across all channels, with minimal clot amplitude on both EXTEM (A5, A10, A20, and MCF each 5 mm) and FIBTEM (A5, A10, A20, and MCF each 1 mm). Conventional coagulation testing (activated partial thromboplastin time and prothrombin time) was attempted but could not be performed, likely owing to interference from the severe hemolysis with optical clot-detection methods and/or the near-absent clot formation reflected in the ROTEM results. Together, these findings indicate a severe coagulopathy with

near-absent clot formation and a markedly reduced fibrinogen contribution to clot firmness (FIBTEM MCF 1 mm); plasma fibrinogen levels were not available.

Assay	CT (sec)	A5 (mm)	A10 (mm)	A20 (mm)	MCF (mm)
EXTEM	338	5	5	5	5
INTEM	4778	-	-	-	-
FIBTEM	4525	1	1	1	1
HEPTEM	4567	-	-	-	-

TABLE 2: Rotational thromboelastometry (ROTEM) results on presentation

EXTEM: extrinsically activated assay; INTEM: intrinsically activated assay; FIBTEM: extrinsically activated assay with platelet inhibition (fibrinogen contribution); HEPTEM: intrinsically activated assay with heparinase; CT: clotting time; A5, A10, A20: clot amplitude 5, 10, and 20 minutes after CT; MCF: maximum clot firmness; -: not measurable

An acute triple-phase CT scan demonstrated two sharply demarcated, air-filled nodular zones in liver segments 7 and 8, measuring 6.7 cm and 5 cm, respectively, most consistent with emphysematous (gas-forming) hepatic abscesses (Figure *1*). Secondary hepatic portal venous gas was also observed. There were no signs of aortic dissection or rupture and no signs of bowel ischemia. The gallbladder was explicitly assessed and appeared normal, without wall thickening, pericholecystic fluid, or other signs of cholecystitis. Imaging did not identify a definitive source or origin for the abscesses.

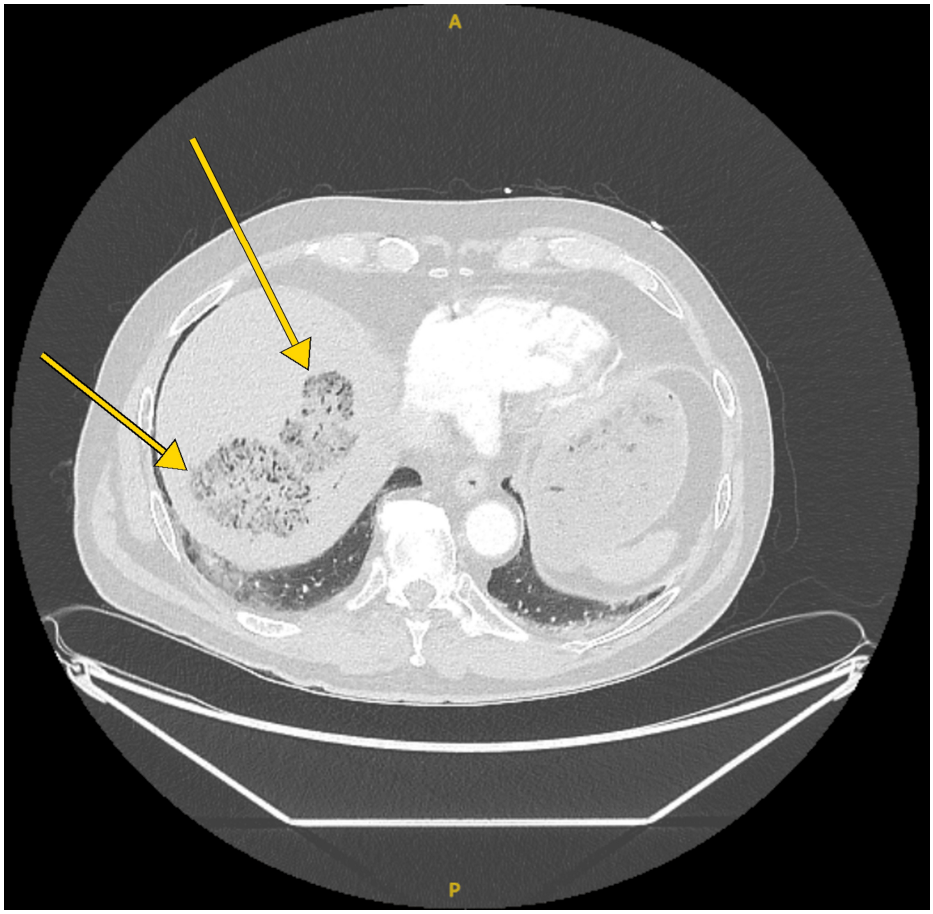


FIGURE 1: Computed tomography showing two sharply demarcated, air-filled nodular zones in liver segments 7 and 8, consistent with emphysematous hepatic abscesses

Blood cultures (aerobic and anaerobic bottles) were obtained by venipuncture at presentation, before antibiotic therapy was started.

Management

Following initial diagnostic work-up, the patient was transferred to the resuscitation room in view of progressive signs of shock. In line with current guidelines, initial treatment consisted of sodium bicarbonate, a balanced crystalloid solution, calcium gluconate, and an insulin-glucose infusion, with piritramide titrated to pain. Percutaneous drainage of the hepatic abscesses was considered but could not be pursued, owing to the patient's increasing hemodynamic instability and rapid clinical deterioration.

A repeat venous blood gas approximately one hour after initial presentation showed worsening acidosis (pH 7.05, base excess −19.4 mmol/L, bicarbonate 9.8 mmol/L) with persistent hyperlactatemia (13.7 mmol/L) and a drop in hemoglobin from 12 to 8 g/dL despite partial improvement in potassium (6.4 mmol/L), raising concern for ongoing hemolysis and/or blood loss. In view of this deterioration, sodium bicarbonate was re-administered together with further balanced crystalloid and broad-spectrum antibiotic therapy (piperacillin-tazobactam and amikacin), started empirically in keeping with standard first-line coverage for undifferentiated intra-abdominal sepsis; the causative organism's gangrene-forming, clostridial nature only became apparent retrospectively, once blood and post-mortem cultures returned. Increasing discomfort and agitation, attributed to hypoxia presumed secondary to diffuse alveolar hemorrhage (no other bleeding focus was identified), prompted a decision to intubate; before this could be performed, the patient suffered a cardiac arrest with an initial rhythm of pulseless electrical activity (PEA), accompanied by trismus. Cardiopulmonary resuscitation (CPR) was initiated per Advanced Life Support (ALS) protocol with adrenaline 1 mg, and return of spontaneous circulation (ROSC) was achieved after two cycles.

During the resuscitation, intubation was performed using the Suction-Assisted Laryngoscopy and Airway Decontamination (SALAD) technique because of a large volume of blood in the oral cavity; more than 500 cc of blood was aspirated via the tracheal tube. A repeat blood gas obtained during this period showed pH 6.78, lactate 19.1 mmol/L, and base excess −20.3 mmol/L. The patient subsequently lost pulses again; CPR was restarted per ALS protocol, with an initial rhythm of asystole. Following multidisciplinary discussion, resuscitation was discontinued in view of persistent asystole.

Outcome

The patient died two hours and 11 minutes after presentation. The blood cultures drawn at presentation flagged positive after 9.6 hours of incubation. *Serratia marcescens* and *Enterococcus faecalis* were isolated from the aerobic bottle, whereas the anaerobic bottle grew *C. perfringens* alongside the same *E. faecalis* and *S. marcescens*. Susceptibility testing was performed on the two aerobic isolates; no antibiogram was reported for *C. perfringens*, and full results are provided in Table *3*.

Antibiotic	*Enterococcus faecalis* (MIC µg/mL)	*Serratia marcescens* (MIC µg/mL)	High-dose regimen
Amoxicillin	S 1	-	-
Amoxicillin/clavulanate	-	R	-
Temocillin	-	I	2000 mg 3×/day IV
Piperacillin/tazobactam	-	S	-
Cefuroxime (parenteral)	-	R	-
Ceftriaxone	-	R	-
Ceftazidime	-	R	-
Cefepime	-	S	-
Aztreonam	-	S	-
Meropenem	-	S	-
Vancomycin	S 2	-	-
Gentamicin	S ≤32	-	-
Amikacin	-	S	-
Ciprofloxacin	-	S	-
Co-trimoxazole	-	I	240/1200 mg 2×/day PO or IV
Linezolid	S 2	-	-
Tigecycline	S ≤0.06	-	-

TABLE 3: Susceptibility results for the aerobic blood isolates Enterococcus faecalis and Serratia marcescens (blood culture drawn at presentation, 08:48 hours)

MIC: minimum inhibitory concentration; S: sensitive; I: sensitive at high dosing; R: resistant; IV: intravenous; PO: oral

Post-mortem examination

Post-mortem examination showed emphysematous transformation of the liver parenchyma, and *C. perfringens* was identified in the gallbladder on biopsy. Post-mortem cultures, provided in full in Table *4*, showed rich growth of *E. faecalis* and *S. marcescens* and a positive anaerobic culture for *C. perfringens* from the gallbladder wall specimen; cultures from the two liver specimens showed only poor growth of the aerobic co-flora, with no anaerobe recovered, despite abundant gram-positive rods seen on direct microscopy of the pseudocyst swab. Together, these ante- and post-mortem findings support a diagnosis of fulminant clostridial hepatobiliary sepsis and bacteremia, with massive intravascular hemolysis, as the cause of death.

Specimen	Gram stain	Aerobic culture	Anaerobic culture
Gallbladder wall (post-mortem biopsy)	Many gram-negative rods; many gram-positive rods; few gram-positive cocci	Rich growth of *E. faecalis*; rich growth of *S. marcescens*; poor growth of mixed flora	Positive for *C. perfringens*
Liver, swab from pseudocyst	Many gram-positive rods; moderate gram-negative rods	Poor growth of *E. faecalis*; poor growth of *S. marcescens*	No strict anaerobes cultured
Liver, subcapsular toward cyst	No organisms seen	Poor growth of *E. faecalis*; poor growth of *S. marcescens*	No strict anaerobes cultured

TABLE 4: Specimens obtained at post-mortem examination, with gram stain and aerobic/anaerobic culture results

Discussion

The combination of gas-forming hepatic lesions and unexplained intravascular hemolysis should immediately suggest *C. perfringens* sepsis. In our patient, these warning signs, together with a severe lactic acidosis and a profound coagulopathy on ROTEM, were already present at the initial work-up, hours before microbiological confirmation. Other gas-forming organisms can produce emphysematous hepatic abscesses, but massive intravascular hemolysis is characteristic of *C. perfringens* and reflects alpha-toxin-mediated destruction of red blood cell membranes [3,4]. A discrepancy between the red blood cell count and the hemoglobin concentration on automated blood counts has been proposed as an additional early laboratory clue [4]. The course in our patient was among the most fulminant reported: comparable to a case of sudden death 2.5 hours after admission [5] and considerably faster than a case in which death occurred 21 hours after presentation despite abscess drainage and antibiotic therapy [8].

Post-mortem examination identified the gallbladder as the primary source of infection, although it appeared normal on initial CT. A similar discordance has been reported in a patient with *C. perfringens* septicemia and hemolysis, in whom initial ultrasound and CT showed no gallstones or cholecystitis and emphysematous cholecystitis with multiple hepatic abscesses was identified only later [9]. Acalculous cholecystitis has likewise been described as the source of a *C. perfringens* hepatic abscess with bacteremia and hemolysis [6]. The structural signs of cholecystitis, such as wall thickening, pericholecystic fluid, or intramural gas, require time to develop and may be absent in a fulminant infection. A radiologically unremarkable gallbladder therefore does not exclude it as the source, and the gallbladder should be explicitly assessed and reported on CT even when initial imaging appears reassuring.

Source control appears to be the decisive determinant of survival. In reported survivors, percutaneous drainage of the hepatic abscesses, in two cases combined with drainage of the gallbladder, was performed alongside antibiotic therapy [6,7,9]. For clostridial myonecrosis, current guidelines recommend definitive therapy with penicillin and clindamycin [10], and clindamycin is favored in *C. perfringens* infection for its antitoxin activity [6,7]. Our patient received piperacillin-tazobactam and amikacin, which provide broad empirical coverage for intra-abdominal sepsis, including anaerobes, but lack this antitoxin effect. The rapidity of hemodynamic collapse precluded drainage, and whether earlier addition of clindamycin would have altered the course remains speculative. Nonetheless, gas-containing hepatic lesions on imaging, particularly in combination with hemolysis, may warrant empirical anti-clostridial and antitoxin therapy and a low threshold for emergent percutaneous or surgical drainage, even before microbiological confirmation is available.

No ROTEM-guided hemostatic intervention was administered. Given the fulminant course, with early signs of hemolysis and shock, it may be reasonable to start hemostatic treatment empirically, for example, by activating a massive transfusion protocol with early administration of plasma and fibrinogen, rather than delaying treatment until ROTEM results are available. This mirrors the management of trauma-induced coagulopathy, in which current guidelines recommend initiating coagulation support immediately upon hospital admission, with empirical plasma- or fibrinogen-based strategies in expected massive hemorrhage and subsequent goal-directed therapy guided by viscoelastic testing [11]. Whether this would have changed the outcome in this specific patient remains speculative, however, given the dominant role of the uncontrolled underlying source in the clinical course.

Finally, this case illustrates the limitations of microbiological confirmation. The blood cultures turned positive only 9.6 hours after collection, long after the patient's death, and anaerobic cultures of both post-mortem liver specimens were negative despite abundant gram-positive rods on direct microscopy of the

pseudocyst swab, possibly owing to post-mortem sampling and preceding antibiotic exposure. Management decisions in suspected *C. perfringens* sepsis should therefore be driven by the clinical and radiological picture, and a negative anaerobic culture should not be used to exclude the diagnosis.

Conclusions

This case highlights the fulminant course of *C. perfringens* sepsis with gas-forming hepatic abscesses and massive intravascular hemolysis, in which death may occur within hours of presentation. Several take-home messages emerge. First, the combination of gas-forming hepatic lesions and unexplained intravascular hemolysis should immediately raise suspicion of *C. perfringens* infection. Second, a radiologically normal gallbladder does not exclude it as the source of infection. Third, because clinical deterioration outpaces microbiological confirmation, management decisions must be driven by the clinical and radiological picture, including empirical anti-clostridial and antitoxin therapy and a low threshold for emergent source control. Fourth, early empirical hemostatic resuscitation may be considered in the presence of severe coagulopathy and hemolysis. Finally, negative anaerobic cultures, particularly when obtained post-mortem or after antibiotic exposure, do not rule out the diagnosis.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ritchie Mulleman, Annelies Truyen, Koen Monsieurs

Acquisition, analysis, or interpretation of data: Ritchie Mulleman, Annelies Truyen

Drafting of the manuscript: Ritchie Mulleman, Annelies Truyen

Critical review of the manuscript for important intellectual content: Koen Monsieurs

Supervision: Koen Monsieurs

Disclosures

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Ritchie Mulleman and Annelies Truyen contributed equally to the work and should be considered co-first authors.

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