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# Massive intravascular haemolysis due to *Clostridium perfringens*

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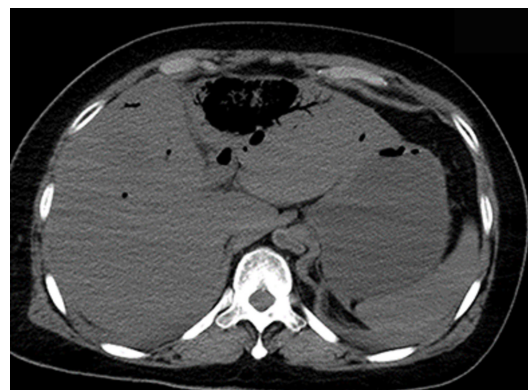
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## DESCRIPTION

A 68-year-old woman came to our hospital because of a disturbance in consciousness the previous day and fever 3 days prior. She had no abdominal pain, nausea, vomiting or diarrhoea prior to admission. She had diabetes and haemoglobin A1c of 7.0%. Vital signs at the time of her visit were as follows: body temperature 40.5°C, blood pressure 137/63 mm Hg, heart rate 120 bpm and a respiratory rate of 50/min. She had jaundice. Chest examination revealed pan-inspiratory crackles and she had blood sputum. There was no knocking pain around the liver, nor Murphy's sign. The blood test revealed a white cell count of 3100/ $\mu$ L (normal range 4000–9000/ $\mu$ L). Her haemoglobin was 6.9 g/dL (normal range 13.5–17.6 g/dL). Her mean corpuscular volume (MCV) could not be measured because of significant haemolysis; however, there was no erythrocyte fragmentation in her peripheral blood smear. The platelet count was  $16 \times 10^9$ /L (normal range  $150 \times 10^9$ – $400 \times 10^9$ /L). Aspartate aminotransferase was 1905 IU/L (normal range 7–97 IU/L), alanine aminotransferase was 690 IU/L (normal range 6–43 IU/L), total bilirubin was 8.1 mg/dL (normal range 0.3–1.2 mg/dL), blood urea nitrogen was 27.0 mg/dL (normal range 10–20 mg/dL) and creatinine was 2.0 mg/dL (normal range 0.6–1.0 mg/dL); that is, liver and kidney dysfunctions were also observed. The blood glucose level was 278 mg/dL. The prothrombin time–international normalised ratio was 2.82 (normal range 0.85–1.15), activated partial thromboplastin time was 54.6 s (normal range 24.3–36.0 s), fibrinogen was 130.1 mg/dL (normal range 150–400 mg/dL) and D-dimer was 195.4  $\mu$ g/mL (normal range <1.0  $\mu$ g/mL). In a urine test, her urine had macroscopic haematuria. These data and a bleeding tendency showed disseminated intravascular coagulation.

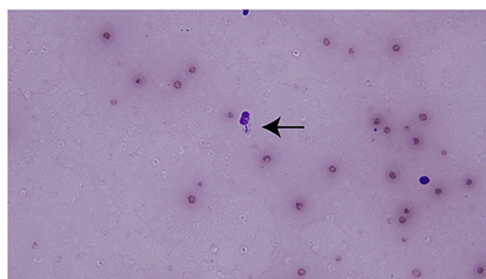
We suspected sepsis of unknown focus and attempted to administer vancomycin and



**Figure 2** Gas-forming liver abscess in postmortem CT.

piperacillin/tazobactam as empiric therapy. However, 30 min after admission, her systolic blood pressure fell to 40 mm Hg and heart rate to 30 bpm. Her monitor ECG initially showed tall peaked T waves, but then P waves disappeared and QRS widened. Based on the severe course of sepsis and haemolysis, we suspected *Clostridium perfringens* bacteraemia. Giemsa staining of the blood smear showed *Bacillus* phagocytosed by leucocyte (figure 1). We clinically diagnosed this as massive intravascular haemolysis due to *C. perfringens*. Although we arrived at a diagnosis, she rapidly worsened, and at 1 hour after the visit, cardiac arrest occurred. Cardiopulmonary resuscitation was performed, but we were unable to save her. We suspected hyperkalaemia because of characteristic electrocardiographic changes. Gas-forming liver abscess was observed in postmortem CT (figure 2). *C. perfringens* were cultured in blood culture after 4 hours.

Massive intravascular haemolysis occurs in 7–15% of cases as a complication of *C. perfringens* bacteraemia.<sup>1</sup> Gas-forming liver abscess is sometimes found.<sup>2</sup> Symptom progression is furious, the mortality rate is 70–90% and the mean death time is 9.7 hours.<sup>3</sup> Early diagnosis is necessary for life-saving treatment. Severe haemolysis, especially the inability to obtain a MCV measurement and blood smear are clues for early diagnosis. Penicillin G mass



**Figure 1** *Bacillus* phagocytosed by leucocyte in Giemsa staining of blood smear (Giemsa staining, original magnification  $\times 400$ ).



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## Learning points

- ▶ *Clostridium perfringens* bacteraemia should be part of the differential if patients have severe sepsis with haemolysis.
- ▶ Blood smear is a clue for early diagnosis of *C. perfringens* bacteraemia.

administration and administration of clindamycin are useful for treatment and require source control at an early stage.<sup>3</sup>

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