increasing the risk of heatstroke.9 The explanation proposed here does not preclude these possibilities. Indeed, infection is a major cause of both pyrexia and decreased arousal due to cytokine production. Thus, a number of these factors could act together to bring about a fatal outcome.


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**Nosocomial empyema caused by Clostridium difficile**

A J H Simpson, S S Das, S Tabaqchali

**Abstract**

Pleural infection with *Clostridium difficile* is extremely rare. A case of nosocomial empyema following chest drain insertion in a 46 year old man is described. The potential of *C difficile* to cause extra- intestinal infections should be recognised and its isolation from other sites should not be ignored.


**Keywords:** Clostridium difficile, empyema, nosocomial infection.

**Case report**

A 46 year old man was transferred from another hospital to St Bartholomew's Hospital, London, with a history of incessant atrial tachycardia secondary to alcoholic cardiomyopathy. Following admission, his tachycardia was treated and stabilised with digoxin 0-25 mg per day and verapamil 80 mg three times daily. However, an admission chest x ray revealed bilateral pleural effusions, larger on the right. There was increased shadowing and a cavitating mass in the left upper zone, with erosion of the first and second ribs anteriorly, suggestive of malignancy. There was no previous history of tuberculosis. The patient became pyrexial (38°C) 24 hours after admission, and after collection of blood cultures, was started on intravenous cefuroxime 750 mg three times daily and oral erythromycin 500 mg four times daily for a suspected chest infection. He had not previously received any antibiotics. No sputum samples were produced. A diagnostic tap of the right pleural effusion showed pus cells, but no bacteria, and was sterile on culture. A Ziehl–Neelsen (ZN) stain for acid/alcohol fast bacilli was negative. The protein content was 37 g/l and the glucose concentration 6 mmol/l. Cytology showed abundant polymorphs and reactive mesothelial cells, but no malignant cells. A rapid micro-agglutination test (RMAT) titre for *Legionella pneumophila* was less than 1:8.

The patient had a low grade fever over the following week and enlarging pleural effusions. His peripheral white cell count rose from 14-8 x 10⁹/l to 19-6 x 10⁹/l. A pleural biopsy was performed, followed by right chest drain insertion and drainage of 1200 ml blood stained fluid. Routine culture on blood, chocolate and cystine lactose electrolyte deficient (CLED) agar in an aerobic atmosphere with 5% CO₂ at 37°C, and on blood agar anaerobically (80% nitrogen, 10% hydrogen and 10% CO₂) at 37°C, yielded no growth after 48 hours. ZN staining was again negative and all cultures for acid fast bacilli were negative at eight weeks. Tuberculin skin testing (1:1000) was negative. Erythromycin was stopped and intravenous metronidazole added (500 mg three times daily), in case of aspiration post-cardioversion (attempted before admission to this hospital). Three days later, drainage from the chest drain ceased; a further pleural tap and biopsy was performed, producing thickened blood stained fluid. Cytology showed fibrinous material and many white cells consistent with an empyema. Direct Gram staining showed scanty large Gram positive rods and many neutrophils; anaerobic culture, as described above, revealed a pure growth of *Clostridium difficile*. Plates incubated aerobically showed no growth. No other specimens processed during this period showed evidence of Gram positive rods on staining, nor was *C difficile* isolated from any other specimen, suggesting that laboratory contamination was extremely unlikely. The isolate was positive for toxin A using the Premier *C difficile* Toxin A EIA kit (Meridian Diagnostics...
Gastroenteritis caused by *Aeromonas trota* in a child

J Reina, A Lopez

Abstract
A case of acute diarrhoea caused by *Aeromonas trota* (formerly HG 13 group) in a Spanish child is reported. The strain was isolated in the faeces using the CIN agar (cefsulodin-irgasan-novobiocin) culture media. The strain was initially identified as *A sobria* by the commercial GNI card and API 20E biochemical systems. The strain was, however, Voges-Proskauer and sucrose negative, so complementary tests of cellobiose fermentation and gluconate oxidation were performed. These tests, together with the strain susceptibility to ampicillin (MIC 1 μg/ml) and carbenicillin (MIC <16 μg/ml) led to the final iden-
Nosocomial empyema caused by Clostridium difficile.

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