Tetanus is a neuromuscular disease in which *Clostridium tetani* exotoxin (tetanospsasmin) produces muscle spasms, incapacitating its host. *C. tetani* can be found globally, usually recovered from soil, faeces and house dust, more commonly in third-world countries. The incidence of *C. tetani* in the USA has been reported to be 233 cases from 2001 to 2008 (average of 0.10 individuals per 1 million persons), with a 13.2 % case-fatality rate among cases with known outcomes and 31.3 % among persons aged ≥65 years (CDCP, 2011a). Since tetanus first became a reportable disease, the use of tetanus antitoxin for wound management in addition to improved child birth practices and increased vaccinations has led to improvements in disease outcomes and to decreases in the number of tetanus cases. Approximately 50 % of tetanus cases are secondary to infected wounds, traumatic and surgical, and abscesses, and may even be related to intravenous drug use (Brook, 2008). *C. tetani* is a Gram-positive, anaerobic, rod-shaped bacterium, which can develop a terminal spore. The spores are very resistant to heat and the usual antiseptics (CDCP, 2011b). The usual port of entry is through a break in the skin, and risk factors for acquiring tetanus are related to clinical conditions that include this, especially surgical site wounds. Although rare, the gastrointestinal tract can provide a possible endogenous source of *C. tetani* (Mori *et al.*, 2012).

### Case report

To our knowledge, this is the first case in the literature of *C. tetani* bacteraemia. We present the case of an 87-year-old African American female with a past medical history significant for gastrointestinal obstruction in 1965, asthma, hypertension, diabetes mellitus and chronic obstructive pulmonary disease (COPD), who was recently discharged from the hospital where she was admitted initially for shortness of breath. Her symptoms were attributed to COPD exacerbation, and she was managed with steroids and levavquin along with nebulizer treatments. During her hospital stay, two anaerobic bottles from two blood culture sets drawn on admission had a 48 h growth of Gram-variable rods and the patient was started on cefepime. Her repeat cultures drawn 2 days later were negative. The patient subsequently improved and was discharged home on oral vantin for an additional 5 days. After her discharge, her appetite got worse and she started feeling progressively weaker. She came back to the hospital complaining of generalized weakness and diarrhoea for 3 days. The diarrhoea was described as loose and watery. She denied any blood in the stools. She also denied any nausea, vomiting, fever or chills. On physical examination, the patient was afebrile but was tachycardic at 112 beats per minute. Other findings were remarkable for a large non-reducible ventral hernia measuring around 7 × 4 cm, painful to palpation, along with surrounding erythema. The remainder of her physical exam was unremarkable and in particular, her skin was intact with no evidence of any wounds or skin breakdowns. Laboratory tests revealed a white blood cell count of 6.8 × 10^3 μL^-1 (72 % neutrophils, 11 % lymphocytes and 15 % monocytes), a haemoglobin of 10.5 g dl^-1 and a platelet count of 214 × 10^3 μL^-1. Liver enzymes were within normal range. The patient’s stool was positive for *Clostridium difficile* toxins and she was started on oral metronidazole. Blood cultures from her previous admission revealed the presence of *C. tetani*, which was identified on the fourth day, along with *Clostridium clostridioforme* and *Bacteroides fragilis*, using the RapID ANA II system developed by Remel. The patient did not have any sign or symptoms consistent with tetanus. A computed tomography (CT) scan of the abdomen and pelvis revealed diverticulosis of the colon, with possible mild inflammation of the sigmoid segment along with a large non-obstructing ventral hernia. No abscess was detected on imaging. The patient underwent a colonoscopy that showed moderately severe colitis, diverticulosis, internal haemorrhoids, and a large hernia extending to the ascending colon with narrowing of the lumen that made it hard to pass the endoscope. The endoscope was inside the hernia sac, which was proximal to the ileo-caecal valve and caecum. A booster

### Abbreviation

Td vaccine, *tetanus* and diphtheria vaccine.
A careful and extensive review of the literature did not reveal any reported case of *C. tetani* bacteraemia. Three anaerobes were isolated in the blood cultures and we strongly suspect that the patient’s ventral hernia might be the source of those anaerobes. Surgical correction of the hernia was offered to the patient but she refused any surgical intervention. From reviewing the literature, there have been multiple cases of clinical tetanus that were reported after abdominal surgery. In one of the cases, a 75-year-old man developed tetanus 24 h after a resection for a gangrenous perforated small intestine (Furui *et al.*, 1999). Tetanus was thought to be caused by a spillage of the intestinal contents harbouring *C. tetani*; however, this was not identified by culture. The diagnosis of tetanus was made only when opisthotonus in this patient became evident and normal tetanus treatment proved to be successful. In another case (Mori *et al.*, 2012), an 84-year-old woman developed tetanus 3 days after the resection of a gangrenous small intestine caused by obturator hernia incarceration. The diagnosis of tetanus was made clinically after the appearance of generalized spastic contractions with opisthotonus. *C. tetani* organisms residing in the gastrointestinal tract were presumed to have been endogenously inoculated into the strangulated intestine, where it produced tetanospasmin, causing tetanus. In this case report, the authors concluded that physicians should be aware of the fact that, although extremely rare, *C. tetani* residing in the gastrointestinal tract is a possible endogenous source of tetanus infection. Our patient’s vaccination status was up-to-date. She was given the Td vaccine to prevent any complication from a possible toxin release in the future. We do not know if the patient was at risk to develop signs and symptoms of tetanus if left untreated. Given that the patient was asymptomatic, we elected not to give any tetanus antitoxin. The lack of signs and symptoms of tetanus in this patient was due either to the fact that the patient’s blood contained tetanus toxin antibody that neutralized any tetanus toxin present, since the patient’s vaccination was up-to-date, or to the fact that the infecting bacterium lacked the ability to produce tetanus toxin. The management in this case remains unclear, given that there is no literature to guide the therapy. Close follow-up of this patient is definitely warranted and monitoring her for any sign of clinical tetanus is critical.

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In loving memory of our beloved Dr George Perez.

References


