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Please read this section first

The HPCSA and the Med Tech Society have confirmed that this clinical case study, plus your routine review of your EQA reports from Thistle QA, should be documented as a "Journal Club" activity. This means that you must record those attending for CEU purposes. Thistle will not issue a certificate to cover these activities, nor send out "correct" answers to the CEU questions at the end of this case study.
The Thistle QA CEU No is: **MT-2014/004**.
Each attendee should claim THREE CEU points for completing this Quality Control Journal Club exercise, and retain a copy of the relevant Thistle QA Participation Certificate as proof of registration on a Thistle QA EQA.

MICROBIOLOGY LEGEND

CYCLE 35 ORGANISM 4

Clostridium sordellii

Clostridium sordellii was first isolated in 1922 by the Argentinean microbiologist Alfredo Sordelli who named it Bacillus oedematis sporogenes on the basis of its morphology and the marked tissue edema characteristic of infection. In 1927, the organism was renamed Bacillus sordellii. Two years later, it was shown to be identical to Clostridium oedematoides, and the name *C. sordellii* was adopted. Similarities in morphology and biochemical profile suggested that *C. sordellii* was simply a virulent strain of Clostridium bifermentans; however, urease production by *C. sordellii* clearly distinguished the 2 species. In the late 1970s, *C. sordellii* antitoxin was found to neutralize the cytotoxic effect of stool specimens collected from patients with Clostridium difficile–associated pseudomembranous colitis. It was later shown that virulent isolates from both species produced a common cytotoxin.

Clostridium sordellii is a rare anaerobic, gram-positive, spore-forming rod with peritrichous flagella that is capable of causing pneumonia, endocarditis, arthritis, peritonitis, and myonecrosis. Colonies appear translucent to opaque with small zones of β hemolysis on sheep or rabbit blood agar. The organism is commonly found in the soil and in the intestines of animals, including 0.5% of all humans.

C. sordellii bacteremia and sepsis rarely occur. Most cases of sepsis from *C. sordellii* occur in patients with underlying conditions. Severe toxic shock syndrome among previously healthy persons has been described in a small number of *C. sordellii* cases, most often associated with gynecologic infections in women and infection of the umbilical stump in newborns. It has also been described in post-partum females, medically induced abortions, injection drug users and trauma cases. So far, all documented post-partum females who contracted *C. sordellii* septicaemia have died, and all but one woman who contracted the bacterium post-abortion have died.

The source of *C. sordellii* in obstetric/gynecological-associated infections is unknown, although 2 reports have documented prolonged vaginal carriage of *C. sordellii* in 0.5%–10% of healthy women suggesting that some women may be natural *C. sordellii* carriers. There are several clinical features which are unique to *C. sordellii*: marked leukocytosis (Leukemoid reaction), refractory

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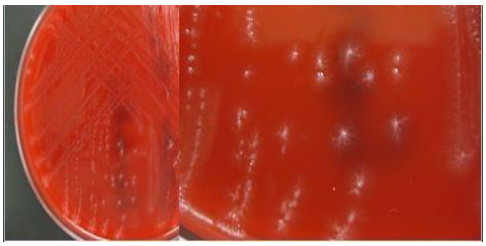
hypotension, severe tachycardia, haemo - concentration, persistent apyrexia and profound capillary leak. In terms of management there is no hard and fast rule as with most bacterial pathogens but past data reveals C. sordellii susceptibility to beta-lactams, clindamycin, tetracycline and chloramphenicol but resistant to aminoglycosides and sulphonamides.



Epidemiology

The rate of vaginal colonization with a variety of Clostridium species during the period after childbirth or abortion is reportedly as high as 29%. Alternatively, fecal contamination of the vagina during vaginal delivery could provide a source of organisms that could infect vaginal tears or the episiotomy site or ascend to the uterus through the open cervix. However, no C. sordellii–related deaths have been reported following instrumented abortion or routine dilatation and curettage, both of which require opening of the cervix, perhaps because fecal contamination of the vagina in these settings is less likely.

Several studies of fatal C. sordellii soft-tissue infection in injection drug users in Europe and North America have been published. In 1999, an outbreak of necrotizing soft-tissue infection occurred in California stemming from "skin popping" of black tar heroin. C. sordellii was identified in 6 of 9 wound specimens, and 4 of 9 patients died.

C. sordellii infections were by no means limited to gynecological procedures and illicit drug use. In fact, 19 (42%) of 45 cases of C. sordellii infection occurred after nongynecological surgical procedures or penetrating, crush, or traumatic injuries to the soft tissues in previously healthy men, women, and children . Of these 19 patients, 10 (53%) died.

Clinical Features

Early clinical symptoms of infection include nausea, dizziness, lethargy, and mild tenderness or rash at sites of infection. Most patients (73%) were afebrile. Within hours after presentation to the hospital, patients have developed hypotension and tachycardia. Laboratory tests have demonstrated elevated hematocrit, increased WBC and platelet counts, and decreased serum calcium and protein levels. As infections progressed, 6 distinctive clinical features developed that, in total, are unique to C. sordellii infection: a marked leukocytosis termed "leukemoid reaction," refractory hypotension, severe tachycardia, profound capillary leak syndrome, hemo-concentration, and a persistent absence of fever. Specifically, the leukemoid reaction has been defined as a WBC count >50×10^9/L resulting from an acute condition, such as infection. With C. sordellii infection, WBC counts routinely increased acutely (within 48–72 h) to 100×10^9/L with 1 reported case as high as 200×10^9/L. Differential cell counts revealed increased percentage of

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mature and immature neutrophils (i.e. band cells, metamyelocytes, and myelocytes) and an increase in the absolute numbers of both lymphocytes and monocytes.

Overall, the mortality rate for patients with *C. sordellii* infection was nearly 69% and most patients died of hypotension and multiorgan failure within days to hours after the initial presentation. Postmortem findings included soft-tissue necrosis at the site of infection and visceral edema. On microscopic examination, infected tissues displayed acute inflammatory changes and localized thrombosis of blood vessels. Often, the margin between healthy and necrotic tissue contained heavy neutrophil degeneration. In all cases but 1, *C. sordellii* was identified at the site of infection, and in 9 of 45 cases, *C. sordellii* was found in the blood.

Pathogenesis

Pathogenic strains of *C. sordellii* produce up to 7 identified exotoxins. Of these, lethal toxin (LT) and hemorrhagic toxin (HT) are regarded as the major virulence factors. Other exotoxins include an oxygen-labile hemolysin, neuraminidase, DNase, collagenase, and lysolecithinase.

Treatment

There is little, if any, information regarding appropriate treatment for *C. sordellii* infection. In fact, the time between onset of symptoms and death is often so short that little time exists to initiate empirical antimicrobial therapy. Indeed, anaerobic cultures of blood and wound aspirate specimens are time consuming, and many hospital laboratories do not routinely perform antimicrobial susceptibility testing on anaerobes. Antibiotic susceptibility data from older studies suggest that *C. sordellii*, like most clostridia, are susceptible to β-lactams, clindamycin, tetracycline, and chloramphenicol but resistant to aminoglycosides and sulfonamides. Potentially, antibiotics that suppress toxin synthesis (e.g. clindamycin) could be useful adjuncts to therapy, because these agents have been proven to be effective in necrotizing infections due to other toxin-producing, gram-positive organisms.

Intensive care measures, including administration of intravenous fluids, are required for patients with tachycardia and hypotension. Emergency surgery to remove necrotic tissues is important for diagnosis, source control, and reducing the buildup of toxins. Other reported treatments included the administration of steroids, morphine, atropine, and/or vasopressors, all of which have proven to be ineffective once infection was established.

References

1. http://en.wikipedia.org/wiki/Clostridium_sordellii
2. http://cid.oxfordjournals.org/content/43/11/1436.

Questions

1. Discuss the morphological characteristics of *C. sordellii*.
2. Discuss the role of *C. sordellii* in disease.
3. Discuss the clinical features of *C. sordellii*.