Fulminant Leukemoid Reaction Due to Postpartum *Clostridium Sordellii* Infection

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ABSTRACT

Clostridium sordellii is gram positive anaerobic spore forming rod and it has been demonstrated to cause gas gangrene, refractory shock, leukemoid reaction, and pleuroperitoneal effusion due to capillary leak. We report here a case of postpartum female who presented with leukemoid reaction, ascites, pleural effusion, and shock without fever 7 days after normal vaginal home delivery.

Key words: *Clostridium sordellii*, Leukemoid reaction, Toxic shock syndrome, Postpartum sepsis

INTRODUCTION

In most cases of *Clostridium sordellii* infections occur after trauma, childbirth, and routine gynecological procedures or intravenous drug abuse.[1] *C. sordellii* is an uncommon member of the clostridium species. The organism first described in 1922 by Sordellii who isolated the bacteria from a patient with postoperative gas gangrene. It produces several exotoxins that contribute to the progressive edema and refractory shock.[2] These infections have a high mortality rates of upto 70%.

CASE REPORT

A 22 year old female presented in the emergency department with complaints of fatigue, malaise, dizziness, shortness of breath, and abdominal distension for the last 5 days. She had a full-term normal vaginal home delivery 10 days back and the baby expired 6 days after the birth. There was no history of fever, abdominal pain, or post partum hemorrhage.

On examination pulse was 110/min, blood pressure was 80/60 mmHg. Pallor, icterus, cyanosis, clubbing, lymphadenopathy were absent. Pitting edema was present in both lower limbs.

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Respiratory system revealed bilateral pleural effusion, evident by absent breath sounds, and stony dullness on the bilateral lower axillary area. Abdominal examination revealed ascites with no hepatosplenomegaly. CNS and CVS were normal. On gynecological examination *Labia majora* were edematous and vaginal mucosa was congested and friable. There was no evidence of bleeding or foul smelling lochia. On bimanual examination, uterus was tender, bilateral fornices were nonpalpable and nontender.

On laboratory examination Hb 11.6 gm/dL, hematocrit 35%, and total leukocyte count of 64,030/m^3 with polymorph 83%, lymphocyte 4%, and promyelocyte 13%, ESR 58 mm, general blood picture was normocytic normochromic. CRP was >150 mg/L. Renal and Liver Function Test and blood sugar were normal. HIV and HbsAg were nonreactive. On ultrasound there was fluid collection in peritoneal cavity and mild hepatomegaly. On ascitic fluid examinations, total cell count were 3200/mm^3 with 60% RBC, 37% lymphocyte, and 3% polymorphs. On Gram staining gram positive rods were present and culture was positive for clostridia; differentiation of clostridium species could not be done due to unavailability of laboratory facility for different clostridia subspecies. Ascitic fluid AFB staining was negative, pleural fluid was exudative in nature and culture was sterile for gram staining and AFB. Blood culture was negative. Urine culture showed *E. coli*.

Patient was shifted in intensive care unit and administered fluid vasopressors, fresh frozen plasma, and intravenous

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antibiotic (piperacillin/tazobactam, gentamicin, and clindamycin). Patient gradually improved and her vitals became stable. Her TLC fall to 6200/mm^3 gradually over 3 weeks. Patient improved clinically and was discharged.

DISCUSSION

In the world's literatures from 1927 to 2006 only 45 cases of *C. sordellii* infection have been reported, 69% of patients usually died within 2 to 6 days of developing the infection. *C. sordellii* is confounded by early, nonspecific signs and symptoms and by the absence of fever. Diagnosis is often delayed because there is no rapid diagnostic test for the pathogen.[3]

Patients with a *C. sordellii* characteristically develop a profound systemic capillary leak, refractory hypotension, and a marked leukemoid reaction (LR) along with fatal tachycardia.

C. sordellii have been isolated in vaginal secretions of 5–10% of a nonpregnant women. During labor or abortion open cervix permits the passage of vaginal pathogens that appears to be the critical event that leads to infection of the endometrium. Infection occurs almost exclusively in association with infection of the uterus or the perineum after either infected episiotomy or postpartum endometritis.[4]

Clinical presentation includes sudden onset influenza such as prodrome, progressive refractory hypotension, hypothermia, or absence of fever and absence of purulent discharge. Distinctive pathological finding includes significant local edema with pleural and peritoneal effusion, localized tissue necrosis, and thrombosis of nearby vessels. *C. sordellii* is associated with a profound exotoxin-mediated systemic response characterized by anasarca, refractory hypotension, and marked leukocytosis.

Diagnosis of *C. sordellii* toxic shock syndrome (TTS) should be suspected when previously healthy women with recent "clean" obstetric wound present with rapidly spreading edema with cardiovascular decompensation with progressive refractory shock. Definitive diagnosis requires isolation of *C. sordellii* from infected tissue as blood culture is usually negative. Aldape *et al.* have described 45 cases, 8 (18%) were associated with normal childbirth, 5 (11%) were associated with medically induced abortion, and 2 (0.4%) were associated with spontaneous abortion. The case-fatality rate was 100%. Ten (22%) of the *C. sordellii* infections occurred in injection drug users. Other cases of *C. sordellii* infection (in 19 patients [43%]) occurred after trauma or surgery. Overall, the mortality rate was 69%. Eighty-five percent of all patients with fatal cases died within 2–6 days of initial infection, and nearly 80% of fatal cases developed LR.

C. sordellii neuraminidase stimulates proliferation of promyelocytic HL-60 cells. It also modifies vascular cell adhesion molecule 1, which orchestrates the release of mature and immature granulocytes from bone marrow stromal cells. Thus, neuraminidase likely plays an important role in the characteristic LR in *C. sordellii* infection.[5]

Our case is comparable to cases described by Mc Gregor[6] *et al.* Rorbye,[7] and Bitti[8] *et al.* have reported a case of postpartum death due to *C. sordellii* TTS. Fischer[9] *et al.* and Cohen[10] *et al.* have reported a fatal case after abortion with mifepristone and misoprostol.

CONCLUSION

New diagnostic approaches are needed to define the true burden of *C. sordellii* in gynecologic infections as they pose a difficult clinical challenge. Limited data are available regarding optimum therapy for *C. sordellii*. Improved treatments are needed to reduce the morbidity and mortality of these infections as these infections are usually fatal.

Strong suspicion should be made if peripartum female presents with toxic shock syndrome with refractory shock, edema, ascites, pleural effusion without fever or pain.

Early recognition of *C. sordellii* TSS may be life saving as this infection has higher mortality and fatal out come with delayed treatment.

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