Clinical Case Reports

Open Access

CASE REPORT

Life-threatening necrotizing myometritis, due to Group A streptococcus – still a life-threatening condition

Sidsel Boie[1], Jan Krog[2], Sofus Tørring[3] & Isil Pinar Bor[1]

[1]Department of Obstetrics and Gynecology, Regional Hospital Randers, Skovlyvej 1, 8900 Randers, Denmark
[2]Department of Anesthesia, Regional Hospital of Randers, Skovlyvej 1, 8900 Randers, Denmark
[3]Department of Radiology, Regional Hospital of Randers, Skovlyvej 1, 8900 Randers, Denmark

Correspondence
Sidsel Boie, Department of Obstetrics and Gynecology, Regional Hospital Randers, Skovlyvej 1, 8900 Randers, Denmark.
Tel: 78421069; Fax: 78424340;
E-mail: sidselmoellerandersen@gmail.com

Funding Information
No funding information provided.

Received: 10 October 2014; Accepted: 19 December 2014

Clinical Case Reports **2015; 3(5): 291–293**

doi: 10.1002/ccr3.217

Key Clinical Message

Puerperal infection with Group A streptococcus (GAS) can present with few symptoms and rapidly progress to a life-threatening condition. Often, the infection can be treated with antibiotics. Delay in diagnosis increases risk of sepsis, multiorgan failure, and death. GAS infection is a differential diagnose for all postpartum women with unexplained symptoms.

Keywords

Group A streptococcus, myometritis, postpartum infection, puerperal sepsis.

Introduction

Puerperal sepsis is the second most common cause of maternal death [1]. Worldwide postpartum infection is estimated to occur in 6% of all deliveries [2] and cause 5–10% of all maternal deaths [3]. Group A streptococcal (GAS) can cause severe puerperal sepsis [4]. It is a serious, rapidly progressive, and life-threatening condition. Despite preventive efforts, antibiotic and early treatment intervention, severe complications can occur primary caused by the pyogenic exotoxin A, which is responsible for fever, toxic shock syndrome, and multiple tissue injury [3].

Case

A 34-year-old woman, gravida 4, para 3, was presented to the Obstetric Department for labor induction, because of suspected macrosomic fetus at the 39th gestational week. Her medical history was unremarkable; apart from periodic depression, whiplash and overweight (BMI 34). Her current and previous pregnancies were uneventful. She entered the active phase of labor 6 h after amniotomy exhibiting clear amnion fluid. Oxytocin was used to augment the labor for 4 h. She gave spontaneous birth to a male infant weighting 4230 g with Apgar score of 7 and 10 at 1 and 5 min, respectively. Both the labor and immediate postpartum period were uneventful. The patient was discharged 4 h after delivery.

Approximately 24 h postpartum, the patient was readmitted to the hospital complaining of pain and chills. Physical examination revealed hypotension (96/72 mmHg), tachycardia (112), fever, oliguria, generalized abdominal tenderness, especially in both lower quadrants with guarding, peritoneal signs, and an enlarged uterus that reached the umbilicus. Abdominal and transvaginal ultrasound examination disclosed an empty uterus without any intra-abdominal fluid. After culture samples were collected, intravenous antibiotic therapy with metronidazole and cefuroxime was initiated. Laboratory exams revealed elevated CRP and creatinine, normal white blood cell counts, hemoglobin, and platelets. An abdominal CT-scan with intravenous contrast demonstrated an enlarged uterus and ascites (36 h postpartum, 8 h after readmission) (Fig. 1A). Due to circulatory instability during the CT scan, the patient was transferred to the intensive care unit (ICU) for supportive care.

The tentative diagnosis was severe puerperal endometritis. After circulatory stabilization, the patient was taken to the operation theater (38 h postpartum, 12 h after

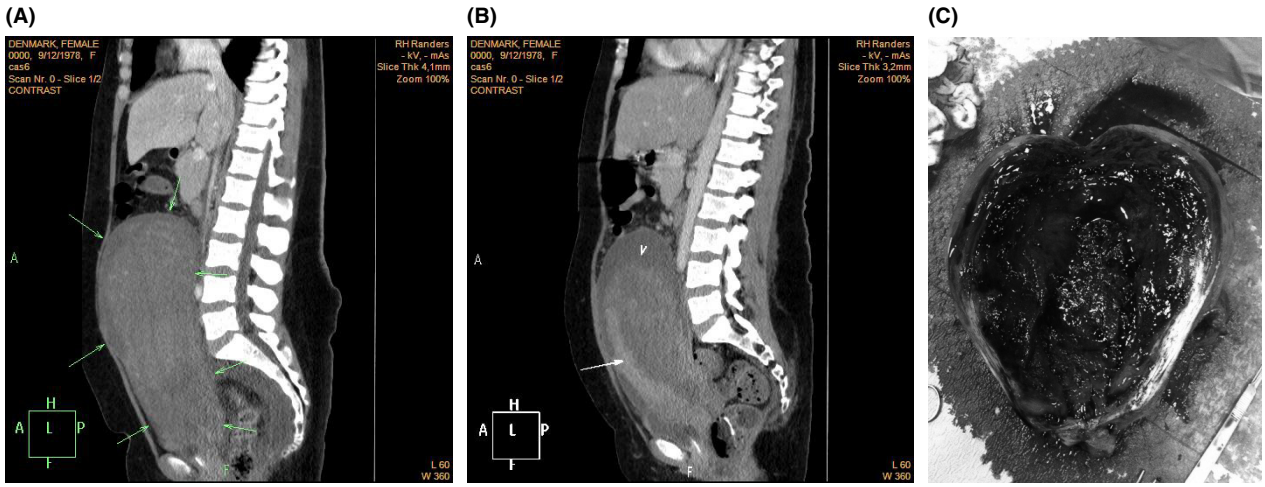

Figure 1. (A) Arrows grossly enlarged uterus with slight inhomogeneous contrast enhancement. (B) Arrowhead – large areas with nonenhancing tissue. Arrow – tissue with contrast enhancement. (C) Uterus after hysterectomy.

readmission) for manual exploration of the uterus due to progressive elevation of CRP and leucocytosis. This procedure did not reveal any retained placental tissue nor any signs of uncontrolled hemorrhage. The antibiotic therapy was changed to intravenous broad-spectrum meropenem and metronidazole. During ICU readmission the patient developed fulminate circulatory shock, despite fluid resuscitation and heavy vasopressor therapy. Cardiac arrest occurred, but one cycle of basic cardiopulmonary resuscitation combined with 1 mg of intravenous adrenaline, brought the patient back to sinus rhythm. A skilled cardiologist performed transthoracic echocardiography (TTE), revealing both right and left ventricular heart failure with a lowered ejection fraction, estimated less than 20%.

The patient was transferred to the highly specialized thoracic intensive care unit (TICU) (38 h postpartum and 15 h after readmission), Here, she was connected to invasive cardiopulmonary support (CPS) for 3 days. The cardiac failure reversed and the kidney function slowly improved.

Both urine sample and culture from vagina on the date of readmission revealed Group A streptococcus. However, all blood cultures were negative. Due to the patient's allergy toward penicillin, meropenem and metronidazole were continued.

One week later, the patient returned to the regional ICU in a stable condition, intubated, and sedated. After arrival, she became circulatory unstable again, her temperature increased to above 42°C, and she needed active cooling, induced by continuous venous hemodiafiltration (CVVHDF). A new CT scan raised suspicion of necrosis in the uterus, which in turn was suspected to be the result of a thrombosis in the right uterine artery (Fig. 1B). An explorative laparotomy and a total abdominal hysterectomy were performed. The uterus was necrotic and enlarged (21 × 16 × 13 cm). A thromboembolus in the right uterine artery was found (Fig. 1C). Microscopic examination revealed "extensive acute necrotizing inflammation with reactive changes in the endometrium. Several septic thromboemboli were present in uterine artery."

Despite intensive care management and adequate resuscitative efforts, the need of respiratory assistance and dialysis continued the days following hysterectomy. Clinical examination revealed altered consciousness, paralysis of all four extremities with absent of tendon reflexes, but preserved eye movements and oculus reflex. MRI-scan, EEG (electroencephalography), and lumbar puncture were performed, but no obvious explanation for the condition was found by these procedures. The patient fulfilled the clinical criteria of severe Critical Illness Polyneuropathy (CIP) [5].

She was transferred to a highly specialized department for neurorehabilitation and is still patient at the center (4 months postpartum). Her condition is slowly improving.

Discussion

As sterile techniques and antibiotic treatment became standard, the morbidity and mortality from streptococcal puerperal infections declined. Since 1980, only small epidemics were reported [6]. During the past decade, a more virulent and aggressive group of GAS has been seen and has now posed a global public health problem.

The prevalence of peripartum GAS infections is 0.18% of total births and 1.4% of all women with puerperal fever [7]. Several bacteria can cause puerperal sepsis; GAS, Group B streptococcus (GBS), staphylococci, myco-

plasma, Chlamydia, clostridium difficile, coliform bacteria, and bacteria associated with polymicrobial vaginosis.

Postpartum women have a 20-fold increase in incidence of GAS and GBS compare to nonpregnant women [8]. Pregnancy is a highly immunomodulated state that permits implantation and development of the immunologically distinct fetus, thereby resulting in a vulnerable genital tract, more accessible for infection. Puerperal infection occurs when bacteria colonize in the genital tract and invade the endometrium. After delivery, the genital tract remains susceptible to invasion for several days. The translocation from vagina can be caused by maternal colonization or nosocomial exposure at parturition. Bacteria can also enter through cesarean section incision or result from a distal infection (i.e., pharyngitis) traveling through maternal bloodstream. The diagnosis of GAS as cause of puerperal sepsis was, in this case, demonstrated by the isolation of GAS from both urine sample and culture from vagina.

A lack of symptoms in the early stage of the infection is common. Later, minor somatic complaints can quickly progress to septic shock, as effects of the exotoxin A manifestation. Fever, abdominal pain, and heavily vaginal bleeding are common symptoms of the GAS infection. Unexplained systemic symptoms in the early postpartum period should result in detailed anamnesis and physical examination. Delay in diagnosis increases the risk of profound sepsis.

Rapidly initiated treatment with antibiotic immediate after collecting cultures is crucial and usually produces a cure. However, the bacteria do not always respond to antibiotics. GAS and exotoxin A produce rapid skin and soft tissue necrosis, fever, septic shock, and multiple organ failure with mortality rate up to 50% [3]. GAS produced toxins allow the necrosis of tissue and the local clotting of blood vessels produces a life-threatening situation, where GAS are protected against the action of antibiotics and the toxin continues destruction producing widespread organ failure.

This patient presented with a severe GAS infection and streptococcal toxic shock syndrome in the postpartum period. The presence of the above indicates that GAS is sequestered in an endometrial site large enough both to be untreatable by antibiotics and to release enough toxin into the vasculary system to produce septic shock. As soon as GAS is suspected and the patient fulfills the sepsis criteria, admission to the ICU should be considered. Focus on multiorgan dysfunction, including early TTE, is essential. Illustrated in this case, fast admission to the TICU gives the opportunity of CPS and is crucial and lifesaving. But as also illustrated, early hysterectomy or other aggressive surgery can be necessary to stop the organ damage.

Conclusion

Our case report illustrates that puerperal infection with GAS can present with few unspecific symptoms and rapidly progress to a life-threatening condition. Often the infection can be treated with antibiotics. Delay in diagnosis increases the risk of sepsis, multiorgan failure, and death. Therefore, we suggest that GAS infection should be on the list of differential diagnosis for all postpartum women with unexplained systemic symptoms.

Conflict of Interest

None declared.

References

1. Drife, J. O. 2004. Why mothers die 2000–2002, the sixth report of the Confidential Enquiries into Maternal Deaths in the United Kingdom, RCOGP.
2. Chuang, I. 2002. Population-Based surveillance for postpartum Invasive group A streptococcus infections 1995–2000. Clin. Infect. Dis. 35:665–670.
3. Mason, K. 2011. Postpartum Group A streptococcus sepsis and maternal immunology. Am. J. Reprod. Immunol. 67:91–100.
4. Aronoff, D. 2008. Postpartum Invasive Group A streptococcal disease in modern era. Infect. Dis. Obstet. Gynecol. 2008:796892 Epub.
5. Lacomis, D. 2001. Neuromuscular disorders in critically III patients: review and update. J. Clin. Neuromuscul. Dis. 12:197–218.
6. Al-ajmi, J. 2012. Group A streptococcus toxic shock syndrome: an outbreak report and review of the literature. J. Infect. Public Health 5:388–393.
7. Lurie, S. 2008. Group A streptococcus causing a life-threatening postpartum necrotizing myometritis. A case report. J. Obstet. Gynaecol. Res. 34:645–648.
8. Deutscher, M. 2011. Incidence and severity of invasive Streptococcus pneumoniae, Group A streptococcus and group B streptococcus Infections among pregnant and postpartum women. Clin. Infect. Dis. 53:114–123.

