Case Report

Severe postpartum sepsis secondary to a deep vaginal hematoma: clinical and therapeutic aspects

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Abstract

Introduction. Puerperal sepsis is still a life-threatening obstetric complication worldwide, even in settings with advanced medical resources. Clinical manifestations may be subtle and atypical during pregnancy and the puerperium, leading to delays in recognition and treatment. Multidrug-resistant organisms such as Stenotrophomonas maltophilia further complicate management, often requiring aggressive multidisciplinary approaches.

Case presentation. We present a case of vaginal hematoma and peripartum infection with Stenotrophomonas maltophilia accompanied by multiple organ dysfunction syndrome.

Conclusions. The presence of pelvic contusion leads to sepsis caused by multiple infections in the peripartum period and can significantly increase the incidence of diseases associated with bleeding, infection, surgery, and blood product transfusions. The clinical condition of the patient may be worsened in the presence of pre-existing conditions prior to pregnancy.

Keywords: Perinatal infection, pelvic bruising, systemic inflammatory response syndrome, septic shock, multiple organ dysfunction syndrome

Introduction

Microorganisms induce inflammation as a tissue response to multiple infectious agents. Affected cells release precursor mediators (such as histamine) and generate inflammatory substances, including eicosanoids (e.g., prostaglandins, thromboxane, and leukotrienes) and cytokines such as interleukin (IL)-1 and tumor necrosis factor (TNF)-α. These mediators start a non-specific inflammatory response.

Microbial invasion is recognized by the immune system via endotoxins (lipopolysaccharides - LPS) and exotoxins from Gram-negative bacteria, in our case, Stenotrophomonas maltophilia, which can be considered a "newly emerging pathogen of concern", as well as peptidoglycans (PG), lipoteichoic acids (LTA), and immunomodulatory toxins from Gram-positive bacteria [1]. Systemic activity of these inflammatory mediators can provoke an excessive and often harmful response. SIRS is a widespread activation of the endothelium, leading to increased production of vascular mediators and vascular imbalance. Inflammatory cytokines such as IL-1 and TNF-α activate the endothelium, which, in turn, produces additional inflammatory cytokines and elevated levels of nitric oxide (NO – via inducible NO synthase), prostaglandins (via inducible cyclooxygenase-2 enzymes), and endothelin-1 [2].

The first effect of SIRS is pulmonary vasoconstriction, leading to pulmonary hypertension [3], mediated by thromboxane A2 [4]. This first hypertensive phase is followed by systemic hypotension due to decreased

DOI: 10.15386/mpr-2903

Manuscript received: 06.06.2025
Received in revised form: 31.08.2025
Accepted: 19.09.2025

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arterial pressure and reduced left ventricular preload, associated with venous dilation in high-capacity vessels, which impairs venous return.

The progression of these processes affecting the cardiovascular system results in shock. Shock occurs when cardiac and vascular function is severely impaired, and hypotension cannot be corrected by the physicians with intravenous fluid administration, needing the use of inotropic and/or vasoconstrictive agents [5]. Shock reflects profound dysfunction of the heart and vasculature associated with SIRS and is a key part of MODS.

What is still unknown about the topic addressed in this case?

At present, the pathogenic mechanisms underlying the uncontrolled systemic inflammatory response triggered by multiple infections in the perinatal period—particularly those caused by Stenotrophomonas maltophilia—remain inadequately defined. If inflammation of the primary site is not held and becomes generalized, it may lead to the development of systemic inflammatory response syndrome (SIRS). The spread of infection from the primary site to the bloodstream can activate systemic endothelial responses, potentially progressing to sepsis, severe sepsis, and septic shock. The transition of sepsis into shock can result in multiple organ dysfunction syndrome (MODS) and, death.

Research hypothesis

We present here a life-threatening condition: septic shock with MODS resulting from infections resistant to multiple antibiotics, bought during pregnancy, labor, and the postpartum perinatal period.

Novel contribution to existing scientific literature

This study elucidates the pathogenetic correlation between severe sepsis and MODS triggered by multiple infections of the genital and urinary tract in perinatal women. It presents a case of a refractory vaginal hematoma that progressively developed within two hours postpartum and was complicated by puerperal sepsis and MODS, following an otherwise uncomplicated and non-traumatic vaginal delivery.

Methods - Case details

A clinical case was analyzed by our team involving a postpartum woman treated in the obstetric and intensive care units of the Perinatology Centre at the "Gheorghe Paladi" Municipal Clinical Hospital in Chișinău, Republic of Moldova, during the period June 2021 – July 2021.

The endothelial cells triggered the pathological effects through the secretion of prostacyclin (PGI_2) and nitric oxide (NO), leading to a hyperdynamic shock syndrome characterized by increased heart rate and cardiac output. These changes represented compensatory mechanisms aimed at preserving tissue perfusion [6].

In sepsis, the systemic inflammatory response impairs the cardiovascular compensatory mechanisms by reducing left ventricular preload, diminishing cardiac contractility through the action of myocardial depressant factors such as nitric oxide (NO), tumor necrosis factor-alpha (TNF-α), and interleukin-1 (IL-1). It also decreases myocardial responsiveness to β-adrenergic stimulation and leads to myocardial edema, which further reduces ventricular compliance. Other alterations in the microvascular bed further contributed to compromised tissue perfusion. The progressive deterioration of the microcirculation, resulting in failure, may be the final common pathway of SIRS-induced injury, contributing to or directly leading to the development of multiple organ dysfunction syndrome (MODS).

Case presentation

Chronological timeline of clinical events

- 41–42 weeks' gestation – 25-year-old primigravida admitted for painful, irregular contractions persisting for two weeks.
- + 8h monitoring – Transferred to the obstetric ward (onset of labor).
- Medical history: acute respiratory infection at 24 weeks with fever, anosmia, and ageusia (no COVID-19 test performed); urinary tract infections treated with urinary antiseptics; history of allergic reactions to unknown medication.
- Labor: epidural at 6 cm dilation; after 10h – vaginal birth of male infant (3800 g, Apgar 7/8). Umbilical cord loosely around the neck, meconial foul-smelling fluid. Minor tears sutured.
- +2h postpartum – Shock symptoms (T 38.2°C, BP 80/60 mmHg, leukocytosis 28.5 × 10^9/L). Treated with antibiotics and IV fluids.
- +15 min – Condition worsened; large vaginal hematoma (~600 ml) drained; total blood loss ~1500 ml. Transfusions and resuscitation needed.
- Postoperative – Oligo-anuria, acute kidney injury → continuous hemodiafiltration (CVVHDF).
- Day 8 postpartum – Ultrasound: bilateral pleural effusion, normal uterus, small free fluid.
- Day 15 postpartum – Severe sepsis, AKI, respiratory failure → resumption of hemodiafiltration, CytoSorb, supportive therapy.
- Day 18 postpartum – Diagnostic laparoscopy: 1500 ml serous peritoneal fluid, polyserositis → abdominal drainage.
- Day 20 postpartum – Bilateral basal pneumonia with effusion; right pleural puncture (900 ml), complicated by pneumothorax → chest drainage.
- Day 21 postpartum – Upper GI bleeding (erosive esophagitis). Stenotrophomonas maltophilia confirmed in cultures.
- Day 28 postpartum – Clinical deterioration → total hysterectomy with bilateral salpingectomy + drainage

of organized parametrial hematoma. Diagnosis: sepsis, multiple organ failure.

• Days 32–33 postpartum – Clinical and imaging improvement, drain removal, pneumonia in remission.

• Day 38 postpartum – Discharged in satisfactory condition.

A 25-year-old primigravida at 41–42 weeks' gestation was admitted by the on-duty obstetrician to the emergency department of the "Gheorghe Paladi" Municipal Clinical Hospital, Chișinău, Republic of Moldova, for irregular painful contractions persisting over two weeks. The pregnancy was planned by the pregnant, but antenatal follow-up was irregular. At 24 weeks, the patient developed an acute respiratory infection with fever, anosmia and ageusia, managed symptomatically without confirmatory COVID-19 testing. She also reported recurrent urinary tract symptoms treated with urinary antiseptics and had a history of drug allergies of unknown origin.

After eight hours of monitoring, labor was proved by the attending obstetrician on duty, and epidural anesthesia was administered by the anesthesiologist at 6 cm cervical dilation. Ten hours later, she delivered vaginally a healthy male infant weighing 3800 g, Apgar 7/8. The amniotic fluid was meconial and foul-smelling, and the umbilical cord was seen by the midwife to be loosely encircling the neonate's neck. The obstetrician sutured minor perineal and vaginal tears.

Two hours postpartum, the patient suddenly developed suprapubic pain, chills, weakness, fever (38.2°C), hypotension (80/60 mmHg), and leukocytosis (28.5 × 10⁹/L). Despite empiric antibiotic therapy and fluid resuscitation, her condition deteriorated rapidly. Examination under anesthesia revealed a large vaginal hematoma displacing the uterus; approximately 600 ml of coagulated blood was drained by obstetricians, with an estimated blood loss of 1500 ml. Hemostatic packing was performed by anesthesiologists, and the patient received transfusion of red blood cells, plasma, and crystalloids.

In the immediate postoperative course, she developed oliguria–anuria and acute kidney injury. Despite diuretic challenge, anuria persisted for 24 hours, and continuous hemodiafiltration (CVVHDF) was started by the anesthesiology team.

Over the following days, the patient showed septic deterioration, with bilateral pleural effusion, metabolic acidosis, and progressive respiratory failure. On postpartum day fifteen, a multidisciplinary team recommended intensified therapy including broad-spectrum antibiotics, CytoSorb hemadsorption, correction of anemia with iron and erythropoietin, and supportive care.

On day eighteen, diagnostic laparoscopy revealed 1500 ml of serous peritoneal fluid consistent with pelvic peritonitis and polyserositis; drainage was performed by an experienced team of obstetricians. On day twenty, pleural effusion required puncture and drainage, complicated by pneumothorax needing chest drainage. On day twenty-one, she developed upper gastrointestinal bleeding due to erosive esophagitis. Microbiological cultures from drains, urine, and vaginal secretions grew Stenotrophomonas maltophilia, a multidrug-resistant pathogen.

By day twenty-eight, despite maximal supportive measures, the patient remained critically ill with worsening sepsis, purulent endometritis, and progressive peritonitis. Laboratory values showed leukocytosis, elevated procalcitonin (18 ng/mL) and lactate (7.2 mmol/L). A total hysterectomy with bilateral salpingectomy and drainage of an organized parametrial hematoma was performed by an experienced obstetrician. Postoperatively, the patient's course remained complicated by multi-organ failure (renal, respiratory, hepatic, and cardiac).

Clinical evolution and treatment

By postpartum day thirty-two, imaging studies revealed signs of clinical improvement. Right-sided pneumonia was in remission, and the left lung appeared clear. Fluid output from the anterior and posterior drains was minimal, and the posterior drain was removed by the thoracic surgeon. On postpartum day thirty-three, the surgeon also removed the anterior pleural drain. The patient's clinical status showed gradual improvement.

The patient was later transferred to the obstetrics department for ongoing treatment. On postpartum day thirty-eight, in a satisfactory condition, she was discharged home in satisfactory condition by the attending obstetrician, with proper medical recommendations.

Pharmacological treatment administered:

- Concentration Erythrocyte: forty-four units.
- Fresh frozen plasma: six units
- Albumin (200 mL): five units
- Antibiotics: Vancomycin 300 mg, Imipenem 1000 mg, Colistin 1 million IU, Lincomycin 600 mg, Fluconazole 400 mg, Levofloxacin 500 mg, Amikacin 500 mg, Linezolid 1200 mg, Doxycycline 100 mg, Meropenem 500 mg, Ciprofloxacin 400 mg, Metronidazole 500 mg, Ceftriaxone 2 g
- Anticoagulant: Enoxaparin (Clexane) 0.2 mL
- Hemostatic: Etamsylate 500 mg
- Diuretics and metabolic support: Furosemide, Mannitol
- Gastro- and hepatoprotective agents: Pantoprazole, Heptral, Ademetionine, Hepametionine
- Probiotics and enzymes: Linex, Panzymed
- Antianemic therapy: Venofer 100 mg, Erythropoietin 0.3 mL
- Vitamins: B1, B6, B12, C, and folic acid 5 mg

Discussion

Vaginal hematomas, most commonly vulvovaginal and sometimes involving branches of the uterine and internal pudendal arteries, can develop rapidly, causing significant pain and maternal hemodynamic impairment, thus requiring prompt recognition and management to prevent adverse outcomes [8]. However, the hemorrhage can also be of venous origin since the veins of the perineum are valveless and have free anastomoses with large intrapelvic venous plexuses [9,10]. Most hematomas will present within 24 hours of delivery. Perineal pain and pressure are common presenting symptoms along with a palpable tender mass [11].

Management of puerperal hematomas is controversial. Conservative management, surgical intervention, and selective arterial embolization are the three main methods for managing puerperal hematomas. Conservative management consists of pressure packing, ice packs, analgesia, and close surveillance. Surgical intervention is indicated for large or acutely expanding puerperal hematomas or when conservative management fails, involving clot evacuation, bleeding control—despite the frequent inability to identify the bleeding vessel due to venous and multifocal origin—and layered closure, with vaginal packing or balloon tamponade used as adjuncts for 12–24 hours, while transcatheter arterial embolization serves as an effective alternative when suturing and packing are insufficient or arterial bleeding is present [7,8]. In our patients, the rapid expansion of concealed vaginal hematoma resulted in profound hemodynamic instability, needing immediate surgical drainage and transfusion. The clinical condition may deteriorate in the presence of pre-existing pathologies, as in our patient, where severe sepsis was most likely triggered by a polymicrobial infection, leading to a complex pathophysiologic cascade involving inflammation and coagulation, progressing from a localized infection to systemic inflammatory response syndrome (SIRS), characterized by fever or hypothermia, tachycardia, tachypnea, leukocytosis or leukopenia, altered mental status, and hyperglycemia in the absence of diabetes mellitus.

The acute phase response, which usually begins within hours or days of the onset of inflammation and/or infection, includes changes in the concentration of plasma proteins (e.g., acute phase proteins), skeletal muscle catabolism (muscles weakness), elevated erythrocytes sedimentation rate, and leukocytosis. These responses are generated by activated macrophages and other immune cells through the release of pro-inflammatory cytokines, particularly IL-1, IL-6, and TNF-α. These cytokines affect the thermoregulatory center in the hypothalamus to produce fever, the most obvious sign of the acute phase response. IL-1 and other cytokines induce an increase in the number and immaturity of circulating neutrophils by stimulating their production in the bone marrow. Other manifestations of acute phase response include anorexia, somnolence, and malaise because of the actions of IL-1 and TNF -α on the central nervous system. The metabolic changes, including skeletal muscle catabolism, release amino acids that are taken up by hepatocytes and immune cells for acute phase protein synthesis and tissue repair.

In severe bacterial infections (sepsis), the massive presence of microorganisms in the bloodstream triggers an uncontrolled inflammatory response with excessive production and release of cytokines (particularly IL-1 and TNF-α), leading to a condition recognized by clinicians as the systemic inflammatory response syndrome (SIRS). These cytokines cause generalized vasodilation, increased vascular permeability, intravascular fluid loss, myocardial depression, and circulatory shock.

Severe sepsis manifests clinically through arterial hypotension, hypoxemia, oliguria, metabolic acidosis, edema, thrombocytopenia, myocardial depression, and circulatory shock. The pathogenesis of sepsis involves a complex process of cellular activation resulting in the release of proinflammatory mediators such as cytokines; recruitment of neutrophils and monocytes; involvement of neuroendocrine reflexes; and activation of complement, coagulation, and fibrinolytic systems. Initiation of the response begins with the activation of the innate immune systems by pattern-recognition receptors (e.g., Toll-like receptors [TLR]) that interact with specific molecules present on microorganisms. Binding of Toll-like receptors (TLRs) to microbial epitopes stimulates the transcription and release of proinflammatory mediators such as tumor necrosis factor-alpha (TNF-α), interleukin-1 (IL-1), and interleukin-6 (IL-6), as well as anti-inflammatory cytokines like interleukin-10 (IL-10). Two of these mediators, TNF-α and IL-1, take part in leukocyte adhesion, local inflammation, neutrophil activation, suppression of erythropoiesis, generation of fever, tachycardia, lactic acidosis, ventilation-perfusion abnormalities, and other signs of sepsis. Although activated neutrophils kill microorganisms, they also injure the endothelium by releasing mediators that increase vascular permeability. In addition, activated endothelial cells release nitric oxide, a potent vasodilator that acts as a key mediator of septic shock.

Another important aspect of sepsis is the alteration of the PR coagulation- anticoagulation balance with an increase in PR coagulation factors and a decrease in anticoagulation factors. Lipopolysaccharide on the surface of microorganism stimulates endothelial cells lining blood vessels to increase their production of tissue factors, thus activating coagulation. Thrombin then converts fibrinogen to fibrin, leading to the formation of microvascular thrombi that further amplifies tissue injury. In addition, sepsis lowers levels of protein C, protein S, antithrombin III, and antithrombin III, and tissue factor pathway inhibitor, substances that modulate and

inhibit coagulation. Lipopolysaccharide and TNF-α also decrease the synthesis of thrombomodulin and endothelial protein C receptors, impairing activation of protein C, and they increase the synthesis of plasminogen activator-1, impairing fibrinolysis.

Sepsis and septic shock typically manifest with hypotension and warm, flushed skin. Septic shock often presents a decrease in systemic vascular resistance. There is hypovolemia due to arterial and venous dilation, plus leakage of plasma into interstitial spaces. Abrupt changes in cognition or behavior are due to reduced cerebral blood flow and may be early indications of septic shock. Regardless of the underlining cause, fever and leukocytosis are present. An elevated serum lactate or metabolic acidosis shows anaerobic metabolism due to tissue hypoxia or cellular dysfunction and altered cellular metabolism. Tissue hypoxia produces continued production and activation of inflammatory mediators, resulting in further increases in vascular permeability, impaired vascular regulation, and altered hemostasis.

Septic shock usually may lead to MODS (Multiple Organs Dysfunction Syndrome). MODS is the presence of altered organ function in an acutely ill patient, in whom physiological homeostasis cannot be kept by the body without medical intervention.

MODS pathogenetically can be explained by the release of proinflammatory mediators (TNF- α, eicosanoids, proteases, platelet-activated factors, and oxidant generating enzymes). The imbalance between pro-and anti-inflammatory mediators with exceed levels of proinflammatory mediators or reduced levels of anti-inflammatory factors such as (soluble TNF receptor, IL-1 receptor antagonist and transforming growth factor).

Secretion of proinflammatory mediators increases the expression of adhesion molecules and increases the margination of blood cells such as platelets, which further reduces blood flow. At the level of the liver, plasma-derived mediators are synthesized as well as complement factors, acute-phase proteins, and factor XII (Hageman factor activation). All of them increase the inflammatory cascade triggering cardiac and pulmonary injury accompanied by hepato-enteric syndrome.

The later clinical course in this case was dominated by severe sepsis and multiple organ dysfunction. Stenotrophomonas maltophilia, isolated from vaginal, urinary, and drain cultures, played a decisive role in the patient's deterioration. This opportunistic, multidrug-resistant Gram-negative bacillus is increasingly shown in nosocomial settings, particularly in immunocompromised hosts or patients exposed to prolonged broad-spectrum antibiotics. Its inherent resistance to multiple antibiotic classes, including most beta-lactams and carbapenems, significantly restricts therapeutic options, often leaving trimethoprim-sulfamethoxazole, fluoroquinolones, or tetracyclines as the mainstay of treatment [1]. In our case, the presence of S. maltophilia complicated antimicrobial management and contributed to persistent systemic inflammation, progressive organ dysfunction, and the eventual need for radical surgical intervention (hysterectomy).

In the end, when we draw our conclusions on this case, there were seven lessons we learned, and six conclusions reached.

- Irregular pregnancy follow-up may lead to delayed identification of risk factors (history of infections, potential COVID-19, recurrent urinary tract infections). Lesson learned: to minimize complications during childbirth and postnatally, it is necessary to promote prenatal education among pregnant women and encourage regular visits to detect risk factors and improve the mother's condition before birth.

- Postpartum hemorrhage with a vaginal hematoma was the critical moment. Rapid recognition of the condition and surgery to remove approximately 600 ml of clots and a total blood loss of 1,500 ml saved the patient's life. Lesson learned: vigilance in the first hours after delivery is essential; occult bleeding (hematoma) must be actively considered when shock is disproportionate to visible blood loss.

- The patient suffered septic shock and acute kidney injury requiring dialysis. Treatment was complicated by multidrug-resistant Stenotrophomonas maltophilia, needing the use of broad-spectrum antibiotics such as meropenem, levofloxacin, and trimethoprim-sulfamethoxazole. Lesson learned: it is essential to move quickly to a multidisciplinary approach (intensive care physicians, nephrologists, infectious disease physicians, surgeons). In our case, the treatment proved effective through the successive combination of antibiotics: Vancomycin 300 mg, Imipenem 1000 mg, Colistin 1 million IU, Lincomycin 600 mg, Fluconazole 400 mg, Levofloxacin 500 mg, Amikacin 500 mg, Linezolid 1200 mg, Doxycycline 100 mg, Meropenem 500 mg, Ciprofloxacin 400 mg, Metronidazole 500 mg, and Ceftriaxone 2 g.

- Despite intensive conservative treatment, it was necessary to perform a hysterectomy with salpingectomy on day twenty-eight to control the sepsis. Lesson learned: delay in performing a life-saving hysterectomy in cases of septic puerperal complications leads to increased morbidity. Considering early surgery may reduce multiple organ involvement.

- Massive blood transfusions (44 units of red blood cells, 6 units of fresh frozen plasma) and four renal replacement therapy were needed. The use of CytoSorb hemadsorption is an example of advanced care for sepsis. Lesson learned: modern techniques in intensive care units (CVVHDF, CytoSorb) can contribute to survival in critical obstetric care situations when traditional treatments fail.

- Pneumonia, pleural effusion, gastrointestinal

bleeding, and hepatic and cardiac dysfunction are all signs of a series of systemic reactions caused by sepsis. Lesson learned: sepsis in obstetrics is a multi-organ disease that requires initiative-taking monitoring beyond the reproductive system (lungs, kidneys, gastrointestinal tract, liver, and heart).

- This case highlights the importance of multidisciplinary collaboration (obstetricians, anesthetists, intensive care physicians, nephrologists, surgeons, microbiologists). Lesson learned: outcomes improve when critical obstetric cases are treated in a Level III perinatal center that provides access to advanced technology and specialists.

Conclusions

1. Vigilant postpartum monitoring is essential, as concealed vaginal hematomas can precipitate both hemorrhagic and septic complications even in the absence of over external bleeding.

2. Rapid escalation of care is needed when conservative management fails; prompt surgical exploration can be lifesaving.

3. Multidisciplinary management (obstetrics, anesthesiology, intensive care, nephrology, thoracic surgery) ensures prompt recognition and intervention in complex sepsis scenarios.

4. Microbiological identification of multidrug-resistant pathogens such as *Stenotrophomonas maltophilia* should guide tailored antibiotic therapy and underscore the importance of hospital infection surveillance.

5. Systemic complications (SIRS, MODS) follow a predictable inflammatory and coagulation cascade; early recognition of hypotension, metabolic acidosis, or rising lactate levels should trigger aggressive resuscitative and supportive interventions.

6. Surgical source control (in this case, drainage of hematoma and hysterectomy) is still a cornerstone of sepsis management alongside advanced supportive therapies.

References

1. Said MS, Tirthani E, Lesho E. Stenotrophomonas Maltophilia. 2023 Jun 12. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 Jan–. PMID: 34283489.
2. Wort SJ, Evans TW. The role of the endothelium in modulating vascular control in sepsis and related conditions. Br Med Bull. 1999; 55:30-48.
3. Lavoie JP, Madigan JE, Cullor JS, Powell WE. Hemodynamic, pathological, hematological and behavioral changes during endotoxin infusion in equine neonates. Equine Vet J. 1990; 22:23-29.
4. Morris DD. Endotoxemia in horses. A review of cellular and humoral mediators involved in its pathogenesis. J Vet Intern Med. 1991; 5:167-181.
5. Purvis D, Kirby R. Systemic inflammatory response syndrome: septic shock. Vet Clin North Am Small Anim Pract. 1994; 24:1225-1247.
6. Liepert D, Rosenthal M, Thomas S. Shock: A comparative physiologic approach to mechanisms and therapy. Semin Anesth Perioper Med Pain. 1999; 18:1–14.
7. Cantwell R, Clutton-Brock T, Cooper G, Dawson A, Drife J, Garrod D, et al. Saving Mothers' Lives: Reviewing maternal deaths to make motherhood safer: 2006-2008. The Eighth Report of the Confidential Enquiries into Maternal Deaths in the United Kingdom. BJOG. 2011; 118:71-76.
8. Dellinger RP, Levy MM, Carlet JM, Bion J, Parker MM, Jaeschke R, et al. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2008. Crit Care Med. 2008; 36:296-327.
9. Lewis G (Ed). The Confidential Enquiry into Maternal and Child Health (CEMACH). Saving Mothers' Lives: Reviewing Maternal Deaths to Make Motherhood Safer 2003–2005. The Seventh Report on Confidential Enquiries into Maternal Deaths in the United Kingdom. London: CEMACH; 2007;76-85.
10. Stefonek KR, Maerz LL, Nielsen MP, Besser RE, Cieslak PR. Group A streptococcal puerperal sepsis preceded by positive surveillance cultures. Obstet Gynecol. 2001; 98:846-848.
11. Barnham MR, Weightman NC. Bacteraemic Streptococcus pyogenes infection in the peri-partum period: now a rare disease and prior carriage by the patient may be important. J Infect. 2001; 43:173-176.