

Septic pelvic thrombophlebitis following cesarean delivery: a case report

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Summary

Despite rare prevalence of septic pelvic thrombophlebitis (SPT), high mortality and morbidity in untreated or delayed diagnosed cases are expected. Thus, early diagnosis and therapeutic management of disease is very vital. The present report describes a febrile case with strong suspicion to SPT that successfully responded to anticoagulant therapeutics. The patient was initially diagnosed with severe sepsis due to cesarean section and managed by antibiotic therapy. Because of persisting fever and no response to antibiotics, and with a strong suspicion of SPT, patient was treated with anticoagulant medication. Two days after beginning anticoagulant therapy, the nature of fever significantly changed to low grade and spiking. Four days later, she was completely afebrile. Therefore, SPT may imitate some common postoperative complications and thus should be considered as a serious post-cesarean event.

Key words: Septic pelvic; Thrombophlebitis; Cesarean delivery.

Introduction

The nature of septic pelvic thrombophlebitis (SPT) has widely evolved within the last decades. Most cases reported earlier suffering from this phenomenon had been obstetrical, leading with high prevalence of abortions [1]. SPT was initially described by von Recklinhausen with thrombosis of one or both ovarian veins that finally required surgical excision [2]. The overall incidence of SPT has been discovered to be 0.05 percent or one per 9,000 in vaginal deliveries and one per 800 for cesarean sections [3]. Despite its rare prevalence, high mortality and morbidity in untreated or delayed diagnosed cases are expected. In the early 20th century, the mortality of 50% was reported even in those undergoing surgical treatment, while within the last two decades, the mortality due to SPT declined to less than 4.4 percent as well as with survival rate more than 90% after the ligation of inferior vena cava and ovarian veins [4, 5]. Thus, early diagnosis and therapeutic management of disease is very vital. The present report describes a febrile case with strong suspicion of SPT that successfully responded to anticoagulant therapeutics.

Case Report

A 13-year-old pregnant girl with gestational age 41 weeks referred to Mahdiah Hospital in Tehran in September 2015 with the complaints of vaginal bleeding, labor pain, and vaginal leak. Regarding her hemodynamic status on admission, the systolic/diastolic blood pressure was 120/70 mmHg with pulse rate of 110 /minute, respiratory rate of 18 /min, and oral temperature of

37.2°C. In initial sonography, the placenta was positioned posteriorly. No abnormal finding was detected in vaginal examination. Clotting time and bleeding time tests were normal. The patient underwent FHR monitoring, but because of repeated late deceleration due to abruption, the patient was transferred to operation room. Preoperatively, she was premedicated with cefalotin sodium. In preoperative assessment, the WBC count was 18,000, serum hemoglobin level was 10.9 g/dl, and platelet count was also 185,000 with a normal liver function test. The fetal heart rate was shown to be 120/minute. Cesarean delivery led to born a cephalic male neonate with an Apgar score of 5.7. Amniotic fluid (AF) was completely bloody and putrid with about 30 ml clot posterior to placenta. In arterial blood gas analysis, a mixed acidotic disorder was revealed with a pH of 7.04, PCO₂ of 56.5, and HCO₃ of 14.5. The curtains with meconium color were clinging completely into the uterine cavity that was completely isolated. Inside the uterus was completely irrigated with three liters of normal saline and was evacuated. Fascia was sutured by nylon and the skin was then sutured separately. During procedure, pulse rate was about 130 /minute. She was transferred to ICU after controlling vital signs. Before this, the patient was medicated with gentamycin followed by ampicillin, clindamycin, and gentamycin in ICU. After transferring to ICU, she suffered tachycardia and tachypnea without fever. The uterine was global with normal VB and suitable urine out. As soon as possible was requested, she was out of bed without abnormality on urination. Because of presenting tachycardia in ICU, CBC, and TSH tests were scheduled again indicating WBC count of 5,500, hemoglobin level of 10.4, and platelet count of 173,000. She was hydrated. Cardiovascular consulting was also considered showing a left ventricular ejection fraction of 50% with a mild mitral valve regurgitation and tricuspid valve regurgitation. Because of partial stability, the patient was transferred to the ward. About 12 hours after operation, enoxaparin was ordered daily. The neonate was also transferred to NICU; however he died a day after NICU admission. About 36 hours after cesarean delivery, fever (39.5°C) with tachycardia (PR =

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120), and tachypnea (RR = 30) appeared in patient that was readmitted to ICU with suspicion of post-cesarean sepsis. Further laboratory tests including blood and urine cultures were requested for ruling out end organ damage that were all negative. The patient was medicated by vancomycin and meropenem. In chest X-ray, atelectasis was reported and thus deep breathing exercise was begun. Abdominal and pelvic cavities were assessed by sonography indicating free air in uterine myometrium suggesting anaerobic infections. In CBC reassessment, WBC count raised to 10,900 and hemoglobin level also decreased to 7.7. Because of primary diagnosis of severe sepsis, a unit of packed cell was prescribed to correct anemia. Following infection disorders consultation and raised clostridium diagnosis, clindamycin was added to previous orders and massive hydration via CV line was also considered. About four days after operation and because of continuing fever in spite of antibiotic therapy and with suspicion to collection and dehiscence of incision point, abdominal and pelvic CT were requested. Simultaneously, echocardiography was performed to rule out pulmonary thromboembolic event. In CT scan, sub-segmental baseline atelectasis was revealed. Also, an abscess with a diameter of 26×65 mm was revealed in posterior portion of uterine posterior to rectum. In reassessment by abdominal and pelvic sonography, in the left portion of incision, a wedge shape defect with the diameter of 18×23 mm with air focuses was observed. In the posterior cul-de-sac, a fluid collection with the diameter of 20×58 mm was also found. Because of the lack of response to antibiotic therapy and also persisting fever, the patient underwent laparotomy with the suspicion of abscess. After opening the abdomen, serous turbid secretions were originating out of the abdomen that were analyzed. The uterine incision site was normal. The uterine cavity was normal without necrosis. Abdomen was explored with a normal condition. The abdomen and uterine were washed with normal saline and suctioned. Drainage was placed at posterior cul-de-sac and fascia was sutured by loop nylon. During the first hours of the second operation, the patient was afebrile. However, she suffered gradually from fever and tachycardia and the new condition was consulted by infection disease expert and thus the antibiotic therapy was changed to vancomycin (1,250 mg/bd), imipenem (500 mg/bd), and gentamycin (160 mg/stat). Because of persisting fever and no response to antibiotics, and with a strong suspicion of SPT, patient was treated with anticoagulant medication. Two days after beginning anticoagulant therapy, the nature of fever significantly changed to low grade and spiking. Four days later, she was completely afebrile. Seven days after stability of body temperature and hemodynamic parameters, she was discharged without any order of antibiotics or anticoagulants. About ten days after discharge, vital signs remained stable with normal incision site.

Discussion

SPT is a rare complication of normal vaginal delivery that is also rarely reported following cesarean section. It is commonly manifested by fever, abdominal pain, and even abdominal mass [6]. The pathognomonic feature of this phenomenon is spiking and persistence of fever despite antimicrobial therapy that frequently appears within a week after delivery. Various predisposing factors have been introduced such as venous stasis after childbirth, increased circulation of clotting factors during pregnancy, and vascular damage due to iatrogenic trauma [7]. Based on patho-

physiological assessments, and dextrotorsion of the enlarging uterus during pregnancy may lead to compression of the right ovarian vein, leading to thrombosis of the vein. If left untreated, ovarian vein thrombophlebitis may extend into the renal veins or the inferior vena cava and can result in a pulmonary embolism, a very life-threatening event [8]. The diagnosis is most commonly made with contrast-enhanced CT, but CT and MRI are equally sensitive for detecting this disorder, and both are more sensitive than ultrasound. Treatment with antibiotics alone may lead to a spiking fever, which is often refractory to antibiotic therapy [9]. The main protocol for treating SPT is based on medical approaches including administration of intravenous anticoagulants such as heparin along with antibiotic therapy that should be considered until the patient is afebrile or clinically well for 48 hours [9, 10]. Conversion to oral anticoagulation is not usually recommended unless there are pulmonary emboli or extensions of clot into the iliac veins or the inferior vena cava. Surgical treatment is reserved for patients who do not respond to intravenous anticoagulation or whose condition deteriorates despite heparin therapy [6].

The present report has some important points. First, some common manifestations following cesarean delivery such as severe sepsis persistent to antibiotic therapy may be in differential diagnosis of SPT that may mislead the physicians to diagnose SPT and thus may lead to patient death. Thus, in the presence of any signs suspected to be SPT, this event should be considered for further assessment. Second, as occurred in the present report, the pregnancy product may be lost due to occurrence of SPT. In final, monitoring of hemodynamic parameters during management of the patient suffered from SPT should be considered until achieving stability.

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