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Maternal group B streptococcal pneumonia in postpartum period

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Abstract

Group B streptococcus is a postpartum cause of maternal bacteremia and endometritis. We report a case of a 44-year-old woman with a negative prenatal screening who presented an acute respiratory distress syndrome in a context of postpartum group B streptococcal pneumonia and sepsis.

Keywords

pneumonia; group B streptococcus; postpartum; bacteremia

Abbreviations

GBS: Group B Streptococcus**;** WG**:** Weeks' Gestation; CRP: C-Reactive Protein

Introduction

Group B Streptococcus (GBS) is a commensal bacterium of the intestinal and female genital tracts. Asymptomatic vaginal colonization occurs in 5-35 per cent of pregnant women [1]. Before systematic maternal prenatal screening and administration of antibiotic prophylaxis, GBS was a major cause of neonatal mortality and morbidity resulting from sepsis, pneumonia and meningitis. In postpartum period, GBS can also be the cause of maternal bacteremia and endometritis [2,3]. Rare cases of maternal meningitis [1,4], tricuspid endocarditis [5], epidural [6] or cerebellar abscess [7] due to GBS are reported. To our knowledge, no case of postpartum maternal group B streptococcal pneumonia with a negative prenatal screening was ever published previously in the literature. We report our clinical experience based on one case.

Case Presentation

A 44-year-old woman with gestational diabetes treated by diet, gravid 4 para 0, had a vacuum delivery at 38 + 1 weeks' gestation (WG) for prolonged second stage of labor. Labor was induced by misoprostol for 3-days ruptured membranes. At admission, patient showed no sign of infection and C-reactive protein (CRP) was at 1.4 mg/l.

Pregnancy was characterized by a preterm labor leading to a fetal pulmonary maturation at 28 WG. An anovaginal swab was done to research GBS and was positive. Culture of urine was negative. At 35 +2 WG, another anovaginal swab was reiterated and was negative. So, the patient did not receive any antibiotics during labor.

About 20 hours after delivery, the patient reported an episode of chills without fever. Three days postpartum she developed pyrexia of 39°C with chills as well as abdominal pain. White blood cell count was elevated to 20.400/mm^3 and CRP was at 119 mg/l. A diagnosis of endometritis was made and intravenous amoxicillin-clavulanate was started. A few hours later the patient complained of dyspnea and oxygen saturation dropped to 67%. The blood pressure was low (65/45 mm Hg) with a heart rate of 120 bpm. We noted the persistence of pyrexia (39.4°C). The patient was confused; she did not know where she was. She was hypoxemic with a PaO_2 of 56.4 mm Hg. So, she was transferred to intensive care unit and required mechanical ventilation by intubation for 4 days. An urgent computed tomographic pulmonary angiogram excluded a pulmonary embolism but highlighted bilateral pleural effusions, multiple ground-glass opacities and sub pleural and intraparenchymal nodules mainly in the right lower and middle lung lobes (Figure 1). Amikacin was also begun. An echocardiography was performed and no sign of endocarditis was highlighted. Indeed, the four cardiac valves functioned normally. No valvular vegetation was found. Systolic and diastolic functions were conserved with an ejection fraction of 60%. Cultures of vaginal smear, urine and blood produced GBS. A diagnosis of acute respiratory distress syndrome in a context of postpartum streptococcal pneumonia and sepsis was made. Nine days postpartum, a follow-up chest X-ray showed marked improvement. The patient was discharged with amoxicillin. Her baby had received prophylactic penicillin and developed no sign of disease.

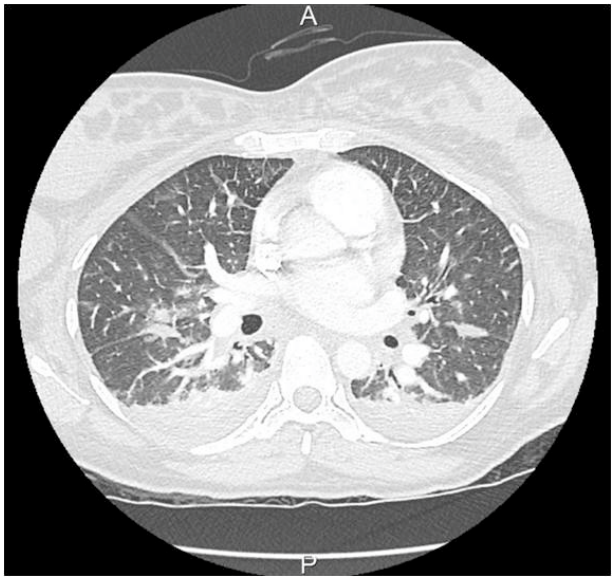

Figure 1: CT pulmonary angiogram with bilateral pleural effusions, ground-glass opacities and intraparenchymal nodules in the right lung.

Discussion

Streptococcus pneumonia and group A Streptococcus are commonly responsible for causing postpartum pneumonia whereas GBS is a major postpartum maternal cause of bacteremia without focus, chorioamnionitis and endometritis [2,3]. Relation between GBS carriage and pneumonia probably results from intrapartum GBS bacteremia and septic emboli. Indeed, GBS is a common pathogen in puerperal sepsis. Moreover, few cases reported the occurrence of postpartum septic pulmonary emboli but as complication of endocarditis [8]. Presence of a vaginal trauma with episiotomy or, like our case, vaginal lacerations, could explain the passage of GBS into the bloodstream. Another explanation for the bacteremia may be early postpartum endometritis due to prolonged rupture of membranes. In a series described by Faro, one-third of patients with endometritis presented a simultaneous GBS bacteremia [9]. Disseminated pneumonia outbreaks across both lungs suggest a probable pulmonary septic embolism.

A conversion from positive to negative GBS test between the second and the third trimester is possible. Indeed, women may acquire or lose colonization in the last trimester [3]. Other plausible hypotheses to explain the third trimester negative test are a sample identification error or a poor quality of the smear.

Our patient did not receive intrapartum antibiotic prophylaxis. Indeed, the vaginal-rectal GBS screening was negative at 35 + 2 WG with an absence of bacteriuria during the second trimester. In agreement with the Committee of the American College of Obstetrician and Gynecologists, administration of intrapartum antibiotic prophylaxis is not recommended in case of rupture of membranes for 18 hours or more with a culture negative for GBS at 35-37 WG [10].

An impressive feature was the rapid deterioration of the general state in our previously healthy patient. Except an episode of chills without fever within the first 24 hours postpartum, the patient developed a high temperature and shacking chills three days after delivery. The evolution was fast: In few hours: The patient presented disturbances of hemodynamic parameters with a drop of the blood pressure, a tachycardia and an increase of the respiratory rate. A deterioration of the consciousness was also noted. These observations are highlighted in different published cases of GBS bacteremia [1,4]. Twenty hours after delivery the occurrence of shivering was surely the first sign of bacteremia. Indeed, the fact that the onset of postpartum GBS bacteremia often occurs within the first day after delivery is described [1].

In non-pregnant adults, GBS is a well-known cause of pneumonia mainly in patients with altered immune function [11]. In our case, only gestational diabetes and postpartum status were potential risk factors to develop GBS pneumonia. Except in patients with allergy, penicillin is the drug of choice for GBS pneumonia.

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