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[Case report]

Maternal septic shock due to *Acinetobacter lwoffii* infection : a case report

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Abstract

The incidence of *Acinetobacter* infections has increased in recent years. *Acinetobacter* infections are resistant to most antibiotics and can be found in hospitalized patients. Pregnancies complicated by severe sepsis or septic shock are associated with a higher rate of preterm labor and delivery, fetal infection, and operative delivery. This case report describes septic shock due to *Acinetobacter lwoffii* infection in the 31st week of gestation. A 47-year-old woman, with a gestation of 31 weeks and one day, presented with a fever, and signs of bacterial infection on laboratory tests. Although the patient was started on tazobactam/piperacillin, she went into septic shock, and was transferred to our hospital. Cesarean section was performed at a gestation of 31 weeks and 4 days because of severe maternal pneumonia and non-reassuring fetal status. *A. lwoffii* was detected in blood cultures collected at the previous hospital, and susceptibility to piperacillin and meropenem to *A. lwoffii* was confirmed. The pneumonia responded to antibiotic treatment and there were no findings of infection in the neonate. Maternal sepsis is an infrequent but important complication, causing significant maternal and fetal morbidity and fetal and neonatal mortality ; therefore, early antibiotic therapy is required to improve the clinical outcome.

Key words : *Acinetobacter lwoffii*, septic shock, chorioamnionitis, case report

Introduction

Acinetobacter species are a group of bacterial microorganisms, gram-negative coccobacillus[1,2]. *Acinetobacter* infections have attracted increasing attention in recent years because they are resistant to most antibiotics and can be found in both hospitalized patients and the community[1]. Among those, *A. baumannii* mainly infects patients with impaired host defense, such as those who are in intensive care units, therefore *A. baumannii* infection is associated with increased mortality[3-5]. Furthermore, sepsis is the leading cause of direct maternal and fetal morbidity and mortality[6]. However, there are a few reports of outcomes of *Acinetobacter* infection during pregnancy and puerperium[1,7]. Moreover, there are no reports of septic shock due to *A. lwoffii* infection during pregnancy. In this report, we present a case of septic shock due to *A. lwoffii* infection in the 31st week of gestation.

Case report

A 47-year-old, gravida 2, para 1 (cesarean section due to placenta previa) woman had undergone a prenatal checkup at a previous hospital. Her past medical history was unremarkable. She was diagnosed with gestational diabetes at 12 weeks of gestation and was started on insulin self-injection. Her blood hemoglobin A1c (HbA1c) level was 5.9%. Glycemic control was appropriate after starting insulin, with blood HbA1c ranging from 5.6% at 19 weeks of gestation to 5.5% at 25 weeks of gestation. At 28 weeks of gestation, she was admitted to the

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hospital with a diagnosis of threatened premature labor due to a shortened cervix (length : 17 mm) and was started on intravenous magnesium preparations. At 31 weeks and one day of gestation, she developed a fever of 38.5°C. Laboratory findings showed an elevated white blood cell (WBC) count of 14,700 /μL and a C-reactive protein (CRP) level of 8.9 mg/dL. She was started on tazobactam/piperacillin (TAZ/PIPC) 13.5 g/day. Her percutaneous oxygen saturation (SpO_2) dropped to 90%, and oxygen administration was started (mask 5 L/min). Two days later, laboratory tests showed a WBC count of 11,400 /μL, CRP level of 14.2 mg/dL, and procalcitonin level of 16 ng/mL. A urinary tract infection was suspected because she had grossly cloudy urine and a transabdominal ultrasound showed a dilated right renal pelvis. Her antibiotic dosage was elevated to TAZ/PIPC 18 g/day. At 31 weeks and 4 days of gestation, her body temperature increased to 40°C, her systolic blood pressure had dropped to the 70-mmHg range, and laboratory tests showed a WBC count of 18,200 /μL, CRP level of 28.8 mg/dL, and thrombocytopenia (platelet count : 14×10^4/μL). Two sets of blood cultures were performed, and gram-negative rods were detected in both. The urine culture did not detect any organisms. She was diagnosed with septic shock and associated disseminated intravascular coagulation, and transferred to our hospital. Rapid antigen tests at the previous hospital for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), Group A *Streptococcus*, and influenza virus were negative.

Chest radiography and contrast-enhanced computed tomography (CT) imaging on arrival at our hospital revealed infiltrating and frosted shadows in the lower and upper lobes of the right lung (Figure 1), and a pleural effusion, leading to the diagnosis of severe bacterial pneumonia. There was no evidence of urinary tract infection, such as dilated renal pelvis on CT or loin pain. The fetal heart rate baseline on cardiotocogram (CTG) was around 100/min, with frequent bradycardia of 80-90 bpm appearing about once a minute (Figure 2). With a diagnosis of non-reassuring fetal status (NRFS), an emergency cesarean section was performed on the same day. The newborn was male with a birthweight of 1,788 g, and 1-minute and 5-minute Apgar scores of 5 and 7, respectively. The umbilical arterial pH was 7.247 with a base excess of −5.20. The newborn was given an artificial surfactant with a diagnosis of respiratory distress syndrome. Culture specimens from blood, sputum, nasal mucus, skin, ear discharge, and anus were collected on the day of birth, but no causative organisms were detected in any of them.

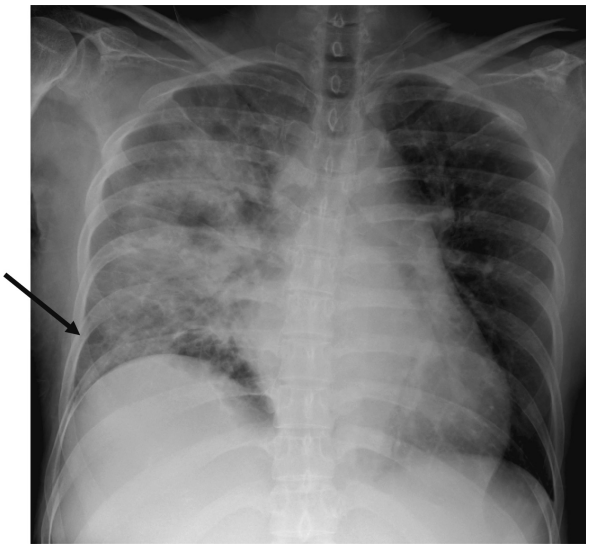

Fig. 1-1. Imaging on arrival at our hospital (Chest radiography)
→ : infiltrating and frosted shadow

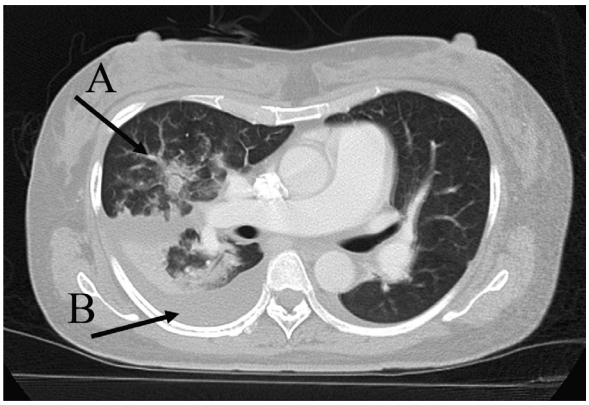

Fig. 1-2. Imaging on arrival at our hospital (contrast-enhanced computed tomography)
A → : infiltrating and frosted shadow, B → : pleural effusion

The patient was intubated and admitted to the intensive care unit (ICU) postoperatively. Postoperative antibiotics were meropenem (MEPM) 3 g/day and azithromycin (AZM) 500 mg/day. On the first postoperative day (POD 1), laboratory findings showed a WBC count of 19,000 /μL and a CRP level of 30.1 mg/dL. On the POD 3, *A. lwoffii* was detected in two sets of blood cultures collected at the previous hospital. The antibiotic susceptibility findings of the blood culture isolates are shown in Table 1. As the results confirmed that the isolate was sensitive to MEPM, the patient was continued on MEPM, and AZM was discontinued. Laboratory

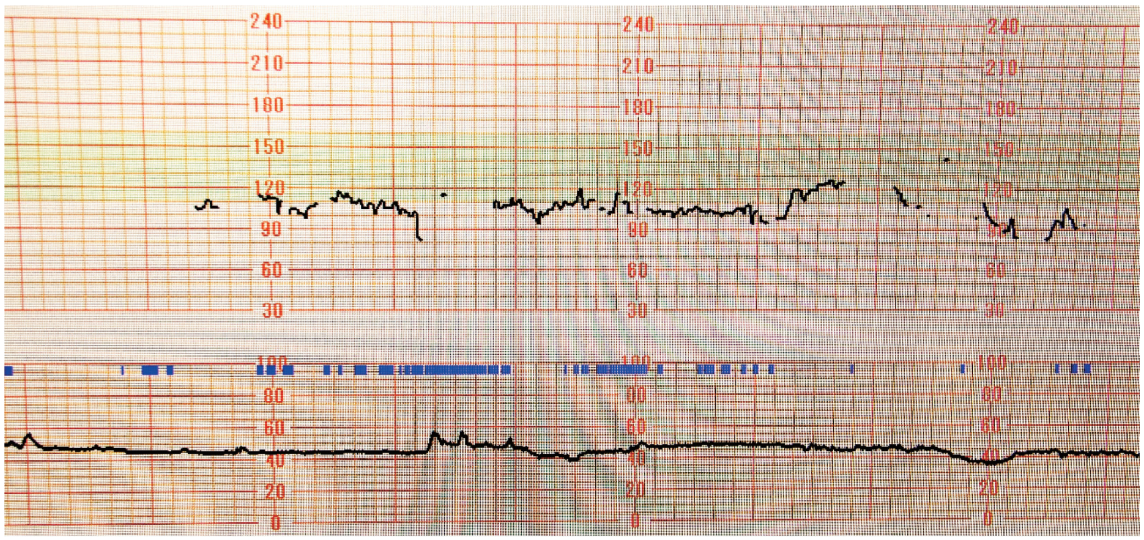

Fig. 2. Fetal cardiotocogram (CTG) on arrival at out hospital

Table 1. Antibiotic susceptibility of the *Acinetobacter lwoffii* isolate grown in blood culture

Antibiotics	MIC (μg/ml)	Interpretation	Antibiotics	MIC (μg/ml)	Interpretation
ABPC	≦ 8	NA	MEPM	≦ 1	S
PIPC	≦ 8	S	AZT	8	NA
CEZ	>16	NA	CPZ/SBT	≦ 16	S
CTM	>16	NA	ABPC/SBT	≦ 8	S
CTX	>2	N/R	GM	≦ 2	S
CAZ	≦ 4	S	TOB	≦ 2	S
CTRX	>2	N/R	AMK	≦ 8	S
CFPM	16	I	MINO	≦ 2	S
CZOP	≦ 4	S	CPFX	≦ 0.25	S
CMZ	16	NA	LVFX	≦ 0.5	S
CCL	≦ 8	NA	CL	≦ 2	S
CFPN	>1	NA	FOM	>16	NA
FMOX	32	NA	ST	≦ 40	S
IPM	≦ 1	S			

(Abbreviations)
ABPC, ampicillin ; AMK, amikacin ; AZT, azidothymidine ; CAZ, ceftazidime ; CCL, cefaclor ; CEZ, cefazolin ; CFPN, cefcapene ; CL, colistin ; CPZ, chlorpromazine ; CTM, ceftizoxime ; CTRX, ceftriaxone ; CTX, ceftriaxone ; FMOX, flomoxef ; FOM, fosfomycin ; GM, gentamicin ; I : intermediate, IPM, imipenem ; LVFX, levofloxacin ; MIC, minimal inhibitory concentration ; MEPM, meropenem ; MINO, minocycline ; NA : not available, N/R : not reported ; PIPC, piperacillin ; S : susceptible ; SBT, sulbactam ; ST, streptomycin ; TOB, tobramycin.

findings showed an improvement in the inflammatory response over time, and the chest radiography showed improvement in the pneumonia. Oxygen administration was discontinued on POD 8. Laboratory findings showed a decrease in the WBC count to 5,600 /μL and CRP level to 0.62 mg/dL, and MEPM administration was discontinued on POD 10. She was discharged on POD 13. Blood cultures taken on POD 5, and vaginal cultures taken on POD 10 did not detect any pathogenic bacteria. Histopathology of the placenta revealed neutrophil infiltration of the chorioallantois membrane, and the patient was diagnosed with chorioamnionitis (CAM). On the other hand, no Gram-negative coccus was detected in the placental tissue, indicating placental infection with *A. lwoffii*. The patient's clinical course is summarized in Figure 3.

Informed consent was obtained from the patient for the publication of her case details.

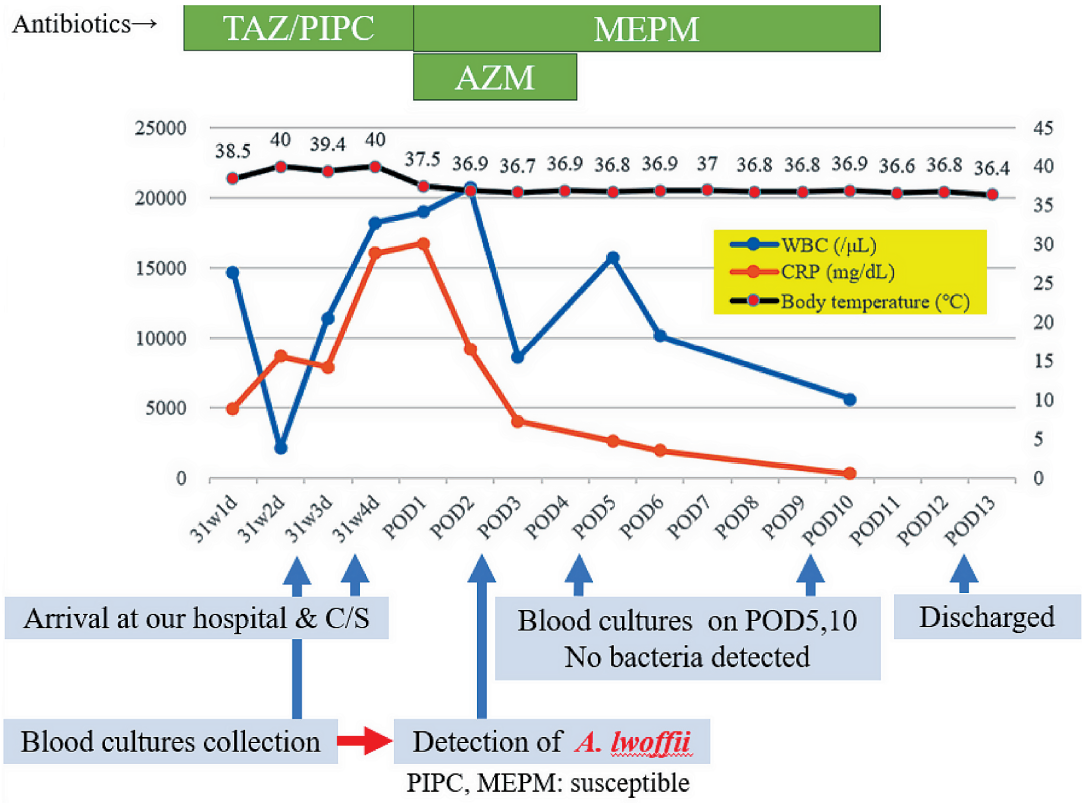

Fig. 3. Summary of the patient's clinical course

(Abbreviations)

A. lwoffii, *Acinetobacter lwoffii* ; AZM, azithromycin ; C/S, cesarean section ; CRP, C-reactive protein ; MEPM, meropenem ; PIPC, piperacillin ; POD, postoperative day ; TAZ, tazobactam ; WBC, white blood cell.

Discussion

More than 30 different *Acinetobacter* species have been identified[1]. Among them, *A. baumannii* tends to be resistant to multiple antibiotics[3]. This antibiotic resistance has led to an increasing number of reports of associated morbidity and mortality worldwide[1]. Most *Acinetobacter* infections occur in individuals with some form of impaired immunity.

There are a limited number of reports on the clinical outcome of *Acinetobacter* infection in pregnancy. Aivazova *et al.*[7] reported a case of a vaginal *A. baumannii* infection during pregnancy and puerperium. The patient had CAM and contractions ; therefore, a cesarean section was performed. In that case, *A. baumannii* was only detected from cervical swabs and not from amniotic fluid or blood. Therefore, the authors concluded that *Acinetobacter* may have been a colonizer rather than the true cause of infection. He *et al.*[1] also determined perinatal and pregnancy outcomes of *A. baumannii* infection. They reviewed a total of 40 bacterial cultures positive for *A. baumannii* over a five-year period, consisting of 33 maternal cultures, 3 neonatal cultures, and 4 autopsy cultures. Lochia was the most common sample source (16/40, 40%), followed by wound (13/40, 32.5%), and others (breast milk, lung aspiration catheter tip, throat, sputum, tracheal aspiration, and blood from a central line). Adverse pregnancy outcomes, including CAM, spontaneous abortion, and preterm labor were seen in all pregnancies with positive *A. baumannii* cultures (including non-sterile sites) around the time of labor. The authors concluded that *A. baumannii* could lead to premature contractions and be associated with CAM during pregnancy.

A. lwoffii is a commensal organism of the human skin, oropharynx, perineum, and urinary tract[8]. Compared to *A. baumannii* infection, there have been few reports of *A. lwoffii* infection[9]. Ku *et al.*[8] conducted a retrospective study of the clinical features of *A. lwoffii* bacteremia. They reviewed 18 cases of confirmed *A. lwoffii* bacteremia in patients whose underlying conditions included cancer (11/18 patients), systemic lupus erythematosus (1/18 patients), chronic obstructive pulmonary disease (2/18 patients) and other diseases (4/18 patients)[9]. They showed that indwelling catheter-related *A. lwoffii*

bacteremia in immunocompromised individuals is associated with a low risk of mortality if the catheter is removed and appropriate antimicrobial therapy is administered[8]. In contrast, multidrug-resistant *A. lwoffii* is emerging as a pathogen in neonatal sepsis. Mittal *et al.*[10] evaluated the clinical characteristics and antibiotic susceptibility profile of *A. lwoffii* in cases of neonatal sepsis. They concluded that the incidence of multidrug-resistant *A. lwoffii* infection is increasing, particularly in premature and very low birth-weight neonates, and that it is important to use antibiotics judiciously and timeously[10].

Although septic shock is rare in pregnancy, occurring in 0.002-0.01% of all deliveries, it leads to a high rate of preterm delivery and perinatal death[11]. The maternal mortality rate among pregnant women with septic shock is 20-28%, and 3.6% of pregnant women diagnosed with sepsis before delivery experience an intrauterine fetal death[11,12]. Acute pyelonephritis is the most common cause of septic shock in pregnant women, and *Escherichia coli* is the most common causative organism[13]. Several risk factors for maternal septic shock have been identified, including vaginal discharge, history of pelvic infection, history of Group B *Streptococcus* infection, multiple pregnancy, assisted reproduction, amniocentesis, cervical cerclage, prolonged spontaneous rupture of membranes, cesarean section, vaginal trauma, wound hematoma, and retained products of conception as obstetric factors ; and obesity, impaired glucose tolerance or diabetes, impaired immunity, immunosuppressant medication, maternal age over 35 years, Group A *Streptococcus* infection in close contacts and family members, and medical conditions (malaria, hepatitis, HIV infection, sickle cell disease) as non-obstetric factors[14]. As the fetal consequences of maternal sepsis are related primarily to vascular changes and poor fetal perfusion, CTG, doppler flowmetry, and umbilical artery doppler assessment are useful for assessing fetal well-being[15]. However, in the presence of CAM and NRFS, delivery should be expedited as a source control measure, regardless of the gestational age[16]. Pregnancy represents an immunocompromised state created in order not to reject the growing fetus, which predisposes the mother to infection[17]. Additionally, respiratory infections in pregnancy are more likely to be severe because the diaphragm is elevated by the pregnant uterus[18].

In our case, the patient had no underlying medical conditions that made her susceptible to infection and no immunosuppressive drug use. Nevertheless, in addition to changes in the immune system and physiology associated with pregnancy, pregnancy at an advanced maternal age, and the patient's susceptibility to infection due to gestational diabetes may have led to severe pneumonia caused by nosocomial infection. Although it is unlikely that the *A. lwoffii* isolate was multidrug-resistant, based on the antibiotic susceptibility findings of the blood culture isolates, these factors may have contributed to the pneumonia. As sputum cultures were not collected and bacteria were detected only from blood cultures, the pneumonia may have been secondary to a bloodstream infection acquired by a nonrespiratory portal of entry. As no bacteria were detected in the urine culture, it is unlikely that the route of entry was the urinary tract. Furthermore, none of the cultures of samples collected from the neonate were positive for *A. lwoffii* or any other pathogens. Therefore, the delivery may have occurred before the infection spread from the amniotic fluid to the fetus. Because the patient was already suffering from NRFS due to shock at the time of transport to our hospital, this suggests that prompt delivery may have prevented severe infection of the fetus. Furthermore, the early initiation of appropriate antibiotics may also have contributed to preventing infection of the fetus.

In conclusion, this case shows that pregnant women without underlying medical conditions may develop nosocomial infections due to reduced immunity caused by pregnancy or gestational diabetes, and that this can lead to septic shock, CAM, and NRFS. Therefore, to improve the clinical outcome, antibiotic therapy should be initiated without delay and delivery should be expedited.

Disclosure of conflict of interest

We have no conflict of interest with regard to our case report.

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