ONLINE FIRST

CASE REPORT

PPROM and Chorioamnionitis following E. Coli UTI - A Case Report

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ABSTRACT

A female patient, 38 years old, G2+P0+A1, at 20 weeks plus six days of gestation, was admitted to emergency with a complaint of watery vaginal discharge for the last 72 hours. There was a hind-water rupture of membranes, and the AmniSure test was positive. She had an OPD visit about a month back, and during that visit, her urine was sent for culture and sensitivity report, which showed E. coli, but she had not taken treatment as she did not come back. Her emergency room baseline labs were in the normal range, and high vaginal swab (HVS) and urine culture and sensitivity results returned negative later. She was asymptomatic for chorioamnionitis. A prophylactic antibiotic was started. Two days later, she developed a fever. She was counseled about septicemia and the risk of maternal death, but she refused. A few hours later, she expelled the fetus and went into hypotension. ERPC (Evacuation of retained products of conception) was done, and one unit of packed red cells was transfused. She was shifted to the ICU. Broad-spectrum IV antibiotics were started. All inflammatory markers were high during her illness. Chest X-ray showed bilateral pleural effusion. She stayed in the ICU for three days and was then discharged.

The objective is to discuss a case of septicemia due to preterm premature rupture of membranes (PPROM) and chorioamnionitis, probably following a urinary tract infection (UTI) caused by E. coli, and its management.

KEYWORDS: PPROM, UTI, maternal sepsis, chorioamnionitis, E.coli

INTRODUCTION

Preterm premature rupture of membranes (PPROM) is premature rupture of membranes before 37 weeks of gestation and can complicate about one-third of preterm births.[1] It can be responsible for serious maternal complications like chorioamnionitis leading to maternal sepsis and death and neonatal complications leading to stillbirth. E. coli has been commonly believed to cause PPROM by ascending infection from the lower genital tract[2]. However, E. coli has never been reported to cause chorioamnionitis through hematogenous spread.

CASE DESCRIPTION

A female patient, age 38 years, was G2+P0+A1 at 20 weeks and six (6) days of gestational period. This was her second marriage. Her first marriage lasted for six years, but she had no pregnancy from that marriage. She was admitted to the emergency with a complaint of watery vaginal discharge for the last 72 hours. On per speculum examination, the forewater was intact; there was a hind-water rupture of membranes, and the AmniSure test was positive.

She had come to OPD on 17th July 2024 for a routine antenatal visit. Her urine was sent for culture and sensitivity (C/S). The urine culture report showed E. coli, but she did not return; hence, the infection remained untreated. She was prescribed Cyclogest pessary, 400 mg, as well, but she did not take it. Then, she came back to the emergency room on 26th August. She was kept under observation for signs and symptoms of chorioamnionitis and prophylactically started the antibiotic Cefazoline 500 mg intravenously three times a day. Laboratory tests were performed, which included a urine culture, high vaginal swab, complete blood count (CBC), C-reactive protein (CRP) level, mid-stream urine analysis, random blood sugar levels, screening for viral infections, rapid plasma reagin test, and HIV test.

An obstetric ultrasound revealed a single, viable intrauterine fetus with an estimated weight of 453 grams, an anterior placenta, and a deepest vertical pocket of amniotic fluid of 6.8 cm. A previous ultrasound on 29-06-2024 showed a nuchal translucency of 1.9 mm, and the internal os was closed and competent.

She was counseled about the fetal outcome because, as per the protocol of the Kingdom of Saudi Arabia, when the pregnancy is less than 24 weeks and the estimated fetal weight is less than 500 grams, it will be considered an abortion. The fetus will not be resuscitated if it is born alive. She was counseled that conservative management and feto-maternal surveillance would be continued for any sign of chorioamnionitis or fetal demise.

The plan of management included monitoring pulse and temperature, the colour of the liquor, uterine tenderness every four hours, CBC and CRP twice weekly, urine culture and vaginal swab weekly, pregnancy ultrasound once a week, and fetal heart monitoring by Doppler every 12 hours. Her baseline labs were in the normal range, and she was asymptomatic for chorioamnionitis.

On 27th August, she started having meconium on the pad. On 28th August 2024, she had a spike in fever to 37.8 ºC at 8:00 am. She was explained and counseled about the significance of fever and the plan for the termination of pregnancy. However, she firmly refused, although she was explained about septicemia and the risk of maternal death. She wanted to wait for her husband, but she did not know when he would arrive. She was explained about the rapid spread of the infection, but she did not accept it. She refused to sign a discharge against medical advice (DAMA) form, as she made her own decision. After four (4) hours, she expelled the fetus spontaneously and went into hypotension immediately. There was no bleeding, but her blood

pressure dropped. She was transferred to the operating theatre for an ERPC (Evacuation of retained products of conception). Two units of PRCs (Packed Red Cells) were cross-matched, and one unit was transfused.

She was then shifted to the ICU, and a massive workup was started. The infection control consultant started broad-spectrum IV antibiotics. She was under the care of a multidisciplinary team, including an ICU, a nephrologist, and an infection control consultant. Her white blood cell count increased from normal levels at admission to 23.22 × 10^9/L on 28th August, 38.84 × 10^9/L on 29th August, 36.39 × 10^9/L on 30th August'2024. CRP increased from 31 mg/L to 103 mg/L on August 28, 2024, and then to 263 and 273 mg/L. After treatment, the concentration decreased to 99 mg/L. Serum Uric acid increased from its normal value on 27th August to 471 µmol/L. Procalcitonin levels increased gradually from 16.32 ng/mL to 33.39 ng/mL and then exceeded 50 ng/mL. Haemoglobin levels upon admission were 8.8 g/dL, which increased to 11.2 on August 29th. Following the ERPC, levels fell to 9.6 g/dL and then increased to 9.7 g/dL after one transfusion. ANA/ANF was positive. ESR was 26 mm in 1st hour. NT-proBNP was 714 pg/mL (normal value = 0-125 pg/mL). INR was 1.3 on 28th August. The level of High-sensitive Troponin 1 was 0.012 ng/mL. Chest X-ray showed bilateral pleural effusion and hazy lung fields. Post-natal ultrasound also showed bilateral pleural effusion. Past medical history was unremarkable. Her HVS and urine C/S results came back negative. There was no history of surgeries or known allergies. She stayed in the ICU for three days. Then, after 24 hours of observation, she was sent home.

DISCUSSION

This particular patient did not take the UTI treatment. If left untreated, UTI can lead to preterm labor.[3,4] A study even showed a strong association between pyuria (without signs and symptoms of UTI) and PPROM before 28 weeks of gestation.[5] E. coli was found in the patient's urine C/S a month ago. The most common organisms causing UTI during pregnancy are E.coli.[6] E. coli are also frequently associated pathogens in PPROM.[7] The PPROM in this patient could be the result of an untreated UTI caused by E. coli, although her recent urine C/S and HVS results came negative.

Chorioamnionitis is a serious and most common complication following PPROM.[1] This occurs due to ascending infection due to vaginal pathogens after the amniotic barrier is decreased. Chorioamnionitis is usually diagnosed through a clinical examination of the patient, along with a histological examination of the placenta. Histological chorioamnionitis is often asymptomatic, and clinical signs, such as fever and maternal or fetal tachycardia, are lacking. In 50% of these cases, amniotic fluid cultures were found to be negative for microorganisms.[8] In this particular patient, HVS was found to be negative, but clinical signs and symptoms of chorioamnionitis were present. Also, her WBC count, CRP, serum uric acid, Procalcitonin, NT-proBNP levels, and INR remained high during her illness.

The longer the time between the rupture of membranes and the onset of labor, the greater the risk of ascending infections and chorioamnionitis.[9] This particular patient came to the ER after 72 hours of PPROM. Hence, she was at high risk of developing chorioamnionitis. The primary objective of antibiotic prophylaxis in patients with PPROM is to prevent chorioamnionitis.[7] This timely and proper administration of antibiotics is very important.[10,11] Although at the earliest evidence of chorioamnionitis, termination of pregnancy is advised irrespective of the gestational age,[12] this particular patient was desperate to keep the baby due to her poor obstetrical history.

CONCLUSION

PPROM and chorioamnionitis can occur following an untreated UTI, and chorioamnionitis can also occur without evidence of bacterial contamination, such as a negative HVS. Patients with UTI during pregnancy should be rigorously informed about the consequences if treatment and follow-up are not done appropriately. PPROM should be managed vigilantly by maintaining strict observation and administering antibiotic prophylaxis.

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AUTHOR CONTRIBUTION

Tullah FA: Concept, drafting, final approval, agreement to accountability
Tullah SN: Design, drafting, final approval, agreement to accountability
Bhutta F: Concept, critical revision, final approval, agreement to accountability
Sabir S: Design, critical revision, final approval, agreement to accountability
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