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A Case of Hematogenous Spread of *E. coli* causing Clinical Chorioamnionitis

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Abstract

This case report details a case of chorioamnionitis via hematogenous spread of bacteria following a perforation of the small bowel. Chorioamnionitis is typically thought of as an ascending infection so this case serves as a reminder that intraamniotic infection can arise from multiple sources including bacteremia. The signs and symptoms may be the same as a typical intraamniotic infection, however, may require different treatment due to the different route and pathogens associated with hematogenous spread.

Keywords

Chorioamnionitis, Bacteremia, Bowel perforation, Intraamniotic infection, Hematogenous spread

Introduction

A 27 year old G1P0 at 25w4d presented from the office where she was incidentally noted to have cervical dilation of 2 cm with funneling of membranes to the external os. Her medical history was significant for polycystic kidney disease requiring multiple abdominal surgeries complicated by previous bowel obstruction and resection secondary to adhesive disease. All initial labs were normal, including a urinalysis, urine drug screen, comprehensive metabolic panel, and complete blood count, with a white blood count of 8.1.

The patient was admitted and given antenatal corticosteroids, Group B strep prophylaxis, and magnesium sulfate. The family declined an amniocentesis to rule out infection. A growth scan revealed normal growth, anatomy, and amniotic fluid index. Two days later, she experienced preterm premature rupture of membranes, and latency antibiotics were initiated. Ten days later, she began to complain of back pain and contractions, was noted to have a urinary tract infection with a white blood cell count of 9.7, and was begun on cephalexin.

Thirty hours after the onset of her back pain and contractions, she was contracting every five minutes, was noted to have copious foul smelling green vaginal discharge, and was febrile to 38.4 degrees and tachycardic, meeting criteria for clinical chorioamnionitis [1]. She had a leukocytosis with left shift, of 21.2, and was now dilated to 4 cm with a bulging bag. Ampicillin and unasyn were started, in place of gentamicin given the patient's renal disease, and magnesium started for neuro protection. Decision was made to proceed with vaginal delivery with oxytocin augmentation as needed.

Two hours later, the patient had a temperature of 40.7 degrees, shaking chills, increased vaginal discharge, fetal monitoring indicating minimal variability, tachycardia, and late decelerations, and no change in her vaginal exam. The decision was made to proceed with cesarean section for delivery.

Upon gentle blunt opening of the peritoneum, greenish bowel fluid was noted to be present. The fetus and placenta were delivered. An approximately one centimeter opening was noted in a section of small bowel that was attached to the uterine fundus and posterior wall by dense adhesions. Trauma surgery was called in to evaluate and repair, and performed the repair after extensive adhesiolysis. The area was visually inspected for other enterotomies but due to the difficulty of the adhesions, the trauma surgeon elected not to free up the entire small bowel. He noted that the enterotomy appeared recent and not related to surgical procedure. The fascia was closed, and the skin and subcutaneous tissue left to close via secondary intention.

Preliminary blood culture results were positive for multi-drug resistant *E. coli*, sensitive to Imipenem. The patient's antibiotic coverage was changed to Imipenem, and she was discharged home with two weeks of intravenous antibiotic therapy via home health.

Final cultures demonstrated growth of multi-drug resistant E. coli in placental, maternal blood, and fetal blood cultures. A 252g placenta was noted to have acute chorioamnionitis and funisitis.

The etiology of chorioamnionitis in this case is a hematogenous spread of E. coli to the placenta and fetus following small bowel perforation. This likely occurred when the patient's urinary tract infection caused preterm contractions, causing the enterotomy due to the dense adhesive disease. As opposed to the typical ascending infection pathogens, the bacteria cultured from the placenta, maternal blood, and fetal blood all demonstrated bowel pathogen, demonstrating an unusual source of chorioamnionitis [2]. Direct spread from the enterotomy to the uterus was not possible as the uterine serosa was intact and not communicating with the intestine. The presentation is consistent with the clinical picture of rapid deterioration and acute sepsis faster than would be seen with intraamniotic infection secondary to ascending infection.

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The neonate was discharged three months later from the NICU, and is free of morbidities at age 1 year.

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