

# A case report and review of chorioamnionitis in the setting of preterm premature rupture of membranes

D A Crosby<sup>1\*</sup>, O O'Sullivan<sup>1</sup>, M O'Connell<sup>1</sup>

## Abstract

Chorioamnionitis is an acute inflammation of the amnion and chorion of the placenta, typically due to ascending polymicrobial bacterial infection in the setting of membrane rupture. Overall, 1–4% of all births in the US are complicated by chorioamnionitis<sup>1</sup>. Chorioamnionitis is present in 40 percent of women with preterm premature rupture of membranes (PPROM) admitted with contractions and 75 percent of those who labour after admission for PPRM<sup>2</sup>. This case highlights the acute and virulent nature of E. Coli chorioamnionitis.

## Case History

We present a 40 year old woman, P0+4 (4 1<sup>st</sup> trimester miscarriages), who attended our emergency department at 31+4 weeks gestation with a history of passing clear fluid vaginally for two hours. She was afebrile and haemodynamically stable, had no abdominal pain and her white cell count (WCC) was  $11.0 \times 10^9/L$ . Her antenatal course was uneventful. A sterile speculum examination confirmed preterm premature rupture of membranes (PPROM), corticosteroids were administered and she was commenced on clarithromycin orally. This was in accordance with local hospital guidelines. An ultrasound scan showed normal growth, normal umbilical artery dopplers and oligohydramnios.

Two days later, she reported reduced fetal movements. A cardiotocograph (CTG) showed two unprovoked decelerations. She remained afebrile, pain free and was haemodynamically stable. She was transferred to the labor ward, had a forewater artificial rupture of membranes (ARM) and was commenced on oxytocin.

Two hours later, the CTG showed a fetal tachycardia of 160bpm, reduced variability and variable decelerations. On examination, she was 3cm dilated, had clear liquor draining and had three contractions in ten minutes. Her WCC was  $24.5 \times 10^9$ . At this point, there was high clinical suspicion for chorioamnionitis and the patient was transferred to theatre for an emergency Cesarean Section (LSCS). The LSCS was performed under spinal anaesthesia. Intravenous co-amoxiclav was administered prior to the procedure. A live female infant was delivered in good condition with

clear liquor. The placenta was delivered by controlled cord traction and was complete. Full blood count, blood cultures, placental swab, and high vaginal swab (HVS) were taken.

Post-operatively, her blood pressure was 80/30 mmHg and her pulse was 133bpm. In view of the hypotension and recent delivery, the decision was made to explore her abdominal cavity and we excluded haemorrhage as a cause. Following repeat surgery, her temperature was 38°C and her WCC was  $2.0 \times 10^9/L$ . Oxygen saturation was 97% on room air. Her mean arterial pressure was 62mmHg and her lactate was 5mmol/L. She was commenced on intravenous cefotaxime, gentamycin and metronidazole and transferred to the intensive care unit (ICU) with suspected sepsis. Non-invasive ventilation and noradrenaline were commenced to maintain mean arterial pressure above 65mmHg. A central venous catheter (CVC) was inserted and 500 millilitres of colloid was administered. A chest x-ray showed bilateral consolidation. On review in ICU six hours later, noradrenaline was continued. However, the urine output decreased to between 10–20mls/hour and a fluid challenge of 500mL colloid was given, which improved urine output.

A further six hours later, she was self-ventilating on 40% oxygen. She was alert and awake, and was continued on low dose noradrenaline maintaining MAP >65. Her WCC was  $29 \times 10^9/L$ . Tazocin and meropenem were used as alternatives to cefotaxime and metronidazole and gentamycin was continued. Urine output was >30ml/hour. Noradrenaline was discontinued four hours later.

The following day, she was awake, orientated and required no inotropic support. Repeat CXR showed bibasal crepitations. She was afebrile on iv tazocin, meropenem and gentamycin. Her WCC was improving at  $24 \times 10^9/L$ . She was transferred to high dependency unit, was seen by physiotherapy, continued on iv antibiotics. Vitals were monitored hourly and one hourly urine output calculated.

Subsequently, placental swab, blood cultures and high vaginal swab taken at the time of delivery showed a heavy growth of E.Coli. Her white cell count was normal at  $12.2 \times 10^9/L$  on day 5 post operatively. Intravenous antibiotics were continued until discharge on day 12. She was seen in the outpatient clinic at six weeks postpartum and was well. This work conforms to the values laid down in the Declaration of Helsinki (1964). The protocol of this study has been approved by the relevant ethical committee related to our institution in which it was performed. All subjects gave full informed consent to participate in this study.

\*Corresponding author  
Email: crosbyd@tcd.ie

<sup>1</sup> Department of Obstetrics and Gynaecology, Coombe Women and Infant's University Hospital, Dublin 8, Ireland

## Discussion

Risk factors for chorioamnionitis include obesity, diabetes, anemia, PPRM and the presence of genital tract pathogens<sup>3</sup>. It can be diagnosed clinically, pathologically or subclinically. Clinical diagnosis is based on the presence of maternal fever (>38°C) and two of the following: maternal leucocytosis or leucopenia, maternal tachycardia (>100 bpm), fetal tachycardia (>160 bpm), uterine tenderness, and foul-smelling amniotic fluid<sup>4</sup>. Histological diagnosis is based on the identification of polymorphonuclear leucocytes on pathologic examination of the placenta and fetal membranes<sup>5</sup>. It is important to note that sepsis may present with hypothermia (core temperature <36°C) (6) or leucopenia (WCC <4 x 10<sup>9</sup>/L) as in this case.

When chorioamnionitis is suspected in the absence of typical clinical signs, biochemical, serum or amniotic fluid tests may be used to diagnose subclinical chorioamnionitis. Amniocentesis for amniotic fluid culture is the best method for diagnosis of subclinical chorioamnionitis in preterm gestations. Other tests including Gram stain, glucose concentration, white blood cell concentration, leucocyte esterase level, and measurement of cytokines (eg, IL-6), ceramide lactoside, or short-chain organic acids suggestive of infection, can be obtained more rapidly<sup>7</sup>.

The frequency of maternal temperature, pulse and fetal heart rate auscultation should be between every 4 and 8 hours<sup>8</sup>. Blood cultures should be obtained prior to antibiotic administration. Antibiotics should be administered within one hour of recognition of severe sepsis. Serum lactate should be measured within six hours of the suspicion of severe sepsis in order to guide management. Serum lactate ≥4 mmol/l is indicative of tissue hypoperfusion. Vasopressors should be used for hypotension not responding to initial fluid resuscitation to maintain mean arterial pressure >65mmHg<sup>3</sup>. The patient should be transferred to intensive care unit (ICU) if cardiovascular hypotension or raised serum lactate persist despite fluid resuscitation, pulmonary oedema, significantly decreased conscious level, multi-organ failure or uncorrected acidosis<sup>3</sup>.

The most common organisms identified in pregnant women dying from sepsis are Lancefield group A beta-haemolytic *Streptococcus* and *E.Coli*<sup>9</sup>. Maternal complications of chorioamnionitis include endometritis, bacteraemia, haemorrhage and death<sup>10</sup>.

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