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Chorioamnionitis and Sepsis in Pregnant Woman with Vaginal Prolapse of Intact Amniotic Membranes; Maternal and Fetal Outcome: A Case Report

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Abstract

Aim: To describe chorioamnionitis and sepsis caused by uncommon germs.

Case report: A healthy 28-year old woman, 23 weeks pregnant,admitted to our Hospital for risk of preterm delivery due to cervical incontinence with vaginal prolapse of intact amniotic membranes.

Results: The amniotic fluid was positive for *Citrobacter freundii*. Blood cultures were positive for *Citrobacter braaki* and *Morganella morganii*.

Conclusion: These findings indicate that ascension from the lower genital tract could be the primary pathway for intra-amniotic infection. It could be useful to collect a sample of the amniotic fluid and/or blood culture, and administer timely a broad spectrum antibiotic therapy to prevent sepsis.

Keywords: Chorioamnionitis; Prolapse of intact membranes; Sepsis; Pregnancy; Preterm birth

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Introduction

Chorioamnionitis is an acute inflammatory reaction of the membranes, placenta and the amniotic fluid which can lead to preterm birth and it's usually related to poor maternal and foetal outcomes [1-4]. It is often caused by a microbial invasion of the amniotic membrane before or during labour especially when rupture occurs, but it can unfrequently happen with intact membranes [5]. Chorioamnionitis is due to inflammatory response of the immune system following the microbial colonization of the amniotic sac. The more severe the inflammatory response syndrome, the more unfavourable the outcomes [6]. It has been proven that this intra amniotic infection is commonly linked to ascending pathogens for the lower genitourinary tract to the inferior pole of the membranes [7,8]. Incidence is higher in preterm delivery rather than term labour, given that it occurs in 0.5% and 10% of preterm pregnancies, and between approximately 0.5% and 2% of term pregnancies. The detection of chorioamnionitis is found in 40% to 70% of spontaneous preterm labours and p-PROMs [3,9].

Definition of chorioamnionitis itself, is made by clinical, microbiological or histopathological findings, it is defined as inflammatory syndrome characterized by fever (T > 38.0°C) in addition on one of the following criteria: leucocytosis (>15000 wbc), maternal or foetal tachycardia, uterine tenderness orfoul odour of the amniotic fluid [10]. Clinical evidence along with typical symptoms are generally what lead to the diagnosis. In this report, we had not only clinical and microbiological evidence of fluid/membranes contamination, but also confirmed histopathological diagnosis.

Case Report

On June 8th 2019 a healthy 28-year old woman, 23 weeks pregnant, has been admitted to our Hospital for risk of preterm delivery due to cervical incontinence with vaginal prolapse of intact amniotic membranes. Patient's anamnesis was positive for thrombophilia due to both MTHFR homozygous and factor V Leiden's heterozygous mutations. She had history of a previous abortion in the first trimester and underwent surgical uterine curettage in 2016. She denied the assumption of any drug but multivitamins ad folic acid and referred no other symptoms or

issues related to the ongoing pregnancy. Prenatal screening tests and morphologic examination were normal.

She presented to the emergency department where the clinical obstetric examination revealed a dilatation of the cervix of 2-3 cm with intact amniotic membranes that were prolapsed in the vagina through the external cervical orifice.

Lab assessment at admission showed increased PCR up to 10500 µg/l so the patient was managed ab initio with bed rest, empiric antibiotic therapy, betamethasone for foetal lung maturity, together with tocolytic medications, Nifedipine 20 mg three times a day. She stayed in the hospital for about eight weeks. On June the 12th the patient developed fever (37.2°C). Vaginal and Rectal microbiological specimens were negative for common pathogens. PCR values started decreasing at the 3rd day of antibiotic treatment.

Ultrasound scanning was performed repeatedly during the hospitalization period, at least three times a week. During the 28th week it revealed a normal foetal growth according to gestational age, with an estimated foetal weight of 1046 g (BPD 68.4 mm, AC 229 mm, FL 52 mm), normally inserted anterior placenta, normal amniotic fluid index (128 mm) and normal maternal-foetal Doppler velocimetry (UA PI 0.88, MCA PI 1.98, CPR 2.25). The flow in the foetal ductus venosus was also normal with positive A wave. Cardiotocography monitoring was also performed daily and no abnormalities were detected.

Inflammatory indexes and routine lab analysis were normal until July 11th (27w + 6 d g.a.): PCR 64100 µg/l, WBC 8.7 × 10^{-3}/ml, platelets 184 × 10^{-3}/ml, haemoglobin 10.1 g/dl. Autoimmunity standard screening panel was negative for anti-ANA antibodies (ab), anti DNA ab, anti b2GPI ab, anti ENA ab, anti cardiolipin ab. On July the 12th lab data showed: PCR 65100 µg/l, WBC 16.86 × 10^{-3}/ml, neutrophils 15.31 × 10^{-3}/ml, lymphocyte 0.86 × 10^{-3}/ml, platelets 187 × 10^{-3}/ml, haemoglobin 9.1 g/dl. This worsening of biochemical and clinical signs of inflammation, together with the modification of the obstetrical conditions, led to the indication for an immediate delivery, given an increased cervical dilatation of 3-4 cm, amniotic sac intact but still protruding in the vagina. On the same day the patient started Magnesium sulphate infusion protocol for foetal neuroprotection.

On July 12th at 28 w g.a., an emergency Caesarean section via spinal anaesthesia was performed. Foetal presentation was cephalic, the extraction was not complicated, a live female infant, 1020 g with an APGAR of 8/9 was delivered. Intraoperative amniorrhexis showed foul smelling amniotic fluid, of which 15 cc were collected to analyse. Placenta was intact with normal central cord insertion. Both placenta and the membranes showed macroscopical signs of inflammation as augmented thickness and were also sent for microbiological, cultural and pathological research.

Histopathological examination of the placenta and membranes indicated severe acute chorioamnionitis and intervillitis with villous edema, granulocyte infiltration plus the presence of villous fibrosis and acute congestion of placental vessels. The umbilical cord vessels showed discordance in their diameters. Cultural samples of placental tissue and amniotic fluid were positive for *Citrobacter freundii*.

One day after the surgery a routine TA US scanning of the pelvis was performed; the uterus was normally involving, endometrial pattern was thin and no endopelvic free fluid/blood was detectable, neither in the Douglas cavity nor in the surgical uterine scar.

On the 3rd day post Caesarean section, the patient started to develop headache and fever; on the 4th day it peaked up to 39.4°C and PCR value increased up to 100 µg/l while routine blood test were normal. Chest X-ray was also negative. We therefore decided to perform haemocultural samples that were positive for *Citrobacter braaki* and *Morganella morganii*.

These clinical findings along with lab results of bacteraemia were highly suggestive of a generalized sepsis. *Morganella morganii* is a common gastro-intestinal pathogen which is known to be responsible for infections in immunocompromised or hospitalized patients, such as those who maintain urinary catheterization for long periods [11,12]. Adult respiratory distress syndrome (ARDS), meningitis, brain abscess and sepsis are some serious complications that can occur in these individuals, but M. morganii is an otherwise uncommon pathogen for healthy people [13,14]. It is very rarely linked to obstetrics and gynaecological infections whereas its presence and colonization of the normal vaginal flora is not yet known [15,16]. *Citrobacter* species are gram-negative bacilli that are as well involved in nosocomial infections; they are estimated to account for 0.8% of all Gram-negative infections, and to represent a percentage between 3 and 6% of the isolates of Enterobacteriaceae in hospital settings [17]. They are not known to be part of the normal vaginal tract flora and have been exceptionally linked to intra amniotic infection [18].

Immediate infectious disease consultation was requested, and antibiotic treatment with Ertapenem 1 g IV daily was subsequently administered for 14 days. The patient became apyretic after 5 days, and her general conditions started to progress. PCR values lowered significatively starting from the 7th day after the beginning of antibiotic treatment and she was dismissed on July 29th, 17th day after surgery.

Discussion

In the majority of women with intra-amniotic infection, amniotic fluid culture is generally positive for typical vaginal commensal species. It has been proven that protruding, though intact, membranes through external cervical orifice represent a risk factor for microbial invasion of amniotic fluid due to the direct contact with the vaginal environment and the absence of usual protective mucous barrier [19,20]. Free transit of germs through amnion cells accounts for an inflammatory response of the whole amniotic cavity. It has been also clarified that intra amniotic inflammation is the foreboding event in the pathogenesis of premature cervical ripening and it is similarly found in up to 80% of patients with functional cervical insufficiency [21]. This condition is widely known to be a risk factor for premature rupture

of the foetal membranes, preterm delivery and adverse foetal outcomes [19,21] In this report, on the other hand, we found two types of usually non-commensal vaginal bacteria. Cultural samples of placental tissue and amniotic fluid were positive for *Citrobacter freundii* and repeated haemocultural samples were positive for *Citrobacter braaki* and *Morganella morganii*. These clinical evidences along with laboratory results and bacteraemia were highly suggestive of a generalized sepsis that occurred immediately after the Caesarean section. Besides, this could indicate that contamination from gastro-intestinal bacteria and subsequent ascension from the lower genital tract could be the primary pathway for intra-amniotic infection. Amniotic fluid culture is found to be positive in up to the 50% of the patients, and it correlates with amniotic inflammation, but *Citrobacter braaki* and *Morganella morganii* aren't described as pathogens responsible for chorioamnionitis [22,23].

For this reason we suggest administration of a broad spectrum prophylactic antibiotic therapy as soon as signs/symptoms of infection are detected and in all of those conditions that present high risk of amniotic fluid contamination such as amniotic membranes prolapse. We also believe it is convenient to perform amniotic fluid and blood culture examination in the clinical management of risk of preterm birth whenever cervical incontinence or possible infectious causes are involved. Cultural samples are mandatory in order to search for possible pathogens, even unusual, especially in cases of persistent increase of inflammatory indexes, since they correlate to poorer outcomes. Further studies are needed to investigate the pathway of vaginal ascending infection and its link to sepsis; it would be indeed advisable to address this issue to outline a standard protocol of prevention and to establish inflammatory indexes' cut off in order to avoid unfavourable maternal/foetal outcome.

Conclusion

Timely medication with broad spectrum antibiotic therapy appears equally important as providing an effective clinical intervention by accurate timing of C-section, because maternal sepsis can occur suddenly, even in the absence of severe symptoms before surgery, and inflammatory conditions are much more like to worsen after invasive procedures that implies handling of an infected placenta and the inevitable contamination, despite the utmost attention, of blood flow by infected amniotic fluid when amniorrhexis is performed.

Conflict of Interests

No potential conflict of interest was reported by the authors.

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