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Case Report

Puerperal Infections Complicated by COVID-19 Co-Infection: Two Independent Case Reports with Need for Intensive Care

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ARTICLE INFO

Article history:
Received: 25 May, 2021
Accepted: 9 June, 2021
Published: 18 June, 2021

Keywords:
COVID-19
SARS-CoV-2
puerperium
co-infection

ABSTRACT

Introduction: The current SARS-CoV-2 pandemic affects all medical fields. In obstetrics, the focus is on the effects on pregnancy and fetuses. Here we present two cases of parturient women that demonstrate the high risk of postpartum SARS-CoV-2 co-infections.

Materials and Methods: Patients` data were obtained from their medical records after confirmation of written informed consent. Literature review was conducted across PubMed.

Case Report: i) A 33-year-old Caucasian Gravida II, Para I was transferred to our tertiary hospital at 23+1 weeks of pregnancy with previous preterm premature rupture of membranes (PPROM) at 17+5 weeks of gestation. During antenatal corticosteroid therapy for fetal lung maturation with 24+0 weeks, intrauterine fetal death was diagnosed in the course of amniotic infection. Due to progressive hemodynamic instability and confirmed SARS-CoV-2 co-infection, our patient had to be treated with catecholamines for 24 hours and stayed at the intensive care unit (ICU) for 72 hours. An acute myocardial injury occurred during septic shock. Immediate intensive care prevented permanent damage. ii) A 27-year-old Caucasian Gravida III, Para III was admitted to our tertiary hospital, 11 days after her third caesarean section. After circulatory collapse at home, the patient arrived with signs of hemorrhagic shock. Emergency curettage was performed, and because of cardiopulmonary worsening pulmonary embolism was suspected. Computed tomography revealed typical signs of COVID-19 pneumonia. Our patient was treated by nasal oxygen in the ICU for 24 hours. Similar to the first patient, acute myocardial injury occurred without any lasting harm.

Conclusion: Our two cases show rapid and worse clinical courses in parturient women with SARS-CoV-2 co-infection. Rapid diagnosis and availability of intensive care were crucial for the prevention of long-term harm. In peri- and postpartum situations of acute clinical worsening, exclusion of SARS-CoV-2 co-infection is an important diagnostic step.

Introduction

Current SARS-CoV-2 pandemic is a major challenge for most countries in the world and affects all parts of their populations. Accordingly, a global effort to control the spread of the virus is needed to meet this challenge. Although many issues have not yet been adequately addressed, currently, the largest vaccination campaign in the history of mankind is most promising [1, 2]. Still in discussion is the risk of SARS-CoV-2 infection in pregnant women and their newborns. In the beginning, observational data from case series and cohort studies indicated no increased risk for pregnant women compared to the general population [3-8]. More recent studies suggested that pregnant women, especially with additional risk factors including obesity, coagulopathy, maternal age over 35 years, and other comorbidities have a higher risk for severe COVID-19 infection compared to non-pregnant women at the same age and may have an elevated risk of adverse pregnancy outcomes such as preterm delivery [9-12]. With regard to mother-to-child transmission studies, rare cases of vertical virus transmission to the fetus

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http://dx.doi.org/10.31487/j.CROGR.2021.01.03

with consecutive intrauterine fetal death, without any other plausible cause have been described [13, 14]. On the other hand, no case of vertical transmission was detected in a recent cohort of women with COVID-19 from the U.S [15]. The two case reports presented here demonstrate critical situations of postpartum infections complicated by COVID-19 co-infection that required intensive care.

Materials and Methods

Patients signed informed consent for the use of their anonymized data for scientific publication. Data were extracted from the patients' medical records. PubMed was used for literature review with the following keywords: SARS-CoV-2, COVID-19, co-infection, puerperium, and pregnancy.

Case Report

Case 1

A 33-year-old Caucasian Gravida II, Para I was hospitalized in a peripheral hospital with preterm premature rupture of membranes (PPROM) in 17+5 weeks of gestation. The pregnancy was complicated by a retrochorial hematoma prior to PPROM. Her past medical history was unremarkable up to this point, and no known risk factors were present. She was treated with antibiotics from 17+5 to 18+5 weeks of gestation (intravenous Ampicillin 2g 1-1-1 for 48 hours and afterwards peroral amoxicillin 1g 1-0-1 for 5 days as well as a single dose of peroral azithromycin 1g). She was transferred to our tertiary hospital at 23+1 weeks of pregnancy to evaluate the induction of lung maturation. The performed ultrasound scan showed oligohydramnios without any fetal abnormalities. After extensive interdisciplinary discussion and informed consent, the mother decided to wait until 24+0 weeks of pregnancy to start the induction of lung maturity with two doses of ventrogluteal betamethasone 12mg at an interval of 24 hours.

On the day of the second betamethasone injection, the patient started being symptomatic with a sore throat, shivering and fever up to 38.3 degree Celsius. In the performed ultrasound scan intrauterine fetal death (IUFD) was diagnosed. Laboratory diagnostics revealed slightly increased leucocytes (12G/l) and C-reactive protein (15mg/l). SARS-CoV-2 rapid antigen testing from nasopharyngeal swabs was performed and positive, verified by PCR analysis. Intravenous antibiotic therapy with 2.2g amoxicillin/clavulanic acid and induction of abortion with 200mcg misoprostol vaginally were started. Within the following two hours, the patient presented with septic shock. Due to suspicion of amnion infection, additional intravenous antibiotic therapy with 300mg clindamycin and 300mg gentamicin were administered.

Furthermore, "sectio parva abdominalis" was indicated, due to progressive hemodynamic instability. On the way to the surgical suite, a dead fetus was extracted, and the placenta followed completely after administration of 5 IE oxytocin as short infusion. Due to progressive hemodynamic instability, the patient had to be treated in the ICU with 12mcg intravenous noradrenaline per minute. As arterial hypotonia and tachycardia persisted, noradrenaline dose was increased to 18mcg per minute. Laboratory diagnostics showed severe hypokalemia, increased troponin (560ng/l) and BNP (885ng/l) as well as anemia (90g/l) and thrombocytopenia (73 G/l). Infection parameters showed following values: leucocytes 17.6 G/l, CRP 359mg/l, procalcitonin 97.15mcg/l, interleukin-6 77400ng/l, ferritin 203mcg/l and lymphocytes 0.3G/l.

Our patient was Jehova's Witness and declined any transfusion of blood products. It was kind of a balancing act finding the best treatment in regard to coagulation with thrombocytes of 73 G/l, INR of 1.8 and multiple factors of hypercoagulability (sepsis, puerperium, SARS-CoV-2 infection). After observing stable haemoglobin values at 90g/l, our patient was treated with 40mg enoxaparin subcutaneously twice a day. Oxygenation was at all times within normal limits. Echocardiography was normal, except for borderline left ventricular ejection fraction. This is why the increase of troponin was interpreted as acute myocardial injury in the context of the septic shock. After gradual reduction of catecholamines, our patient could be transferred to our obstetric ward. Another 10 days later, she could be discharged from the hospital in good general health, with normal echocardiography and laboratory values within normal limits. Histologic analysis of placenta revealed beginning chorioamnionitis with *Escherichia coli*, which was confirmed by placental culture. Bacterial blood culture was negative and SARS-CoV-2 RNA could not be detected in placental tissue.

Case 2

A 27-year-old Gravida III, Para III was admitted to our tertiary hospital by the Swiss Air Rescue 11 days after her third caesarean section, which was carried out in 25+3 weeks of pregnancy because of premature placental abruption. Due to recurrent vaginal antepartum hemorrhage, our patient got two red cell concentrates prior to cesarean section and was discharged with a haemoglobin value of 72g/l 6 days after surgery. Aside from the cesarean sections, our patient`s medical history was free of relevant illnesses.

After circulatory collapse at home, the puerpera arrived with signs of hemorrhagic shock with tachycardia of 102 beats per minute in sinus rhythm, coldish extremities and mean arterial pressure of 64mmHg. Oxygen saturation on room air was 100 percent. As acute vaginal bleeding was apparent and ultrasound examination revealed intrauterine coagula, postpartum curettage was indicated. During curettage, sulproston infusion was started to improve uterine tone and a catheter blocked with 15ml was inserted in the uterine cavity. In total two red cell concentrates were transfused; 1.5g tranexamic acid and 2g fibrinogen were given intravenously. Estimated intraoperative blood loss was 1100ml and the lowest intraoperative haemoglobin measured was 64g/l.

After initial observation on the general obstetrical ward, our patient showed cardiopulmonary worsening with positive shock index and acute hypoxemia with oxygen saturation of 75% on room air. As pulmonary embolism was suspected, computed tomography was performed with typical signs of COVID-19 pneumonia with ground-glass opacity and interstitial lung edema. SARS-CoV-2 PCR was positive from nasopharyngeal swabs. Laboratory diagnostics showed increased troponin (580ng/l) and BNP (680ng/l). As echocardiography was normal, troponin values were interpreted as acute myocardial injury in the context of the hemorrhagic shock. Infect parameters showed following values: leucocytes 14.3 G/l, CRP 178mg/l, procalcitonin 8.65mcg/l, interleukin-6 26ng/l, ferritin 658mcg/l and lymphocytes

0.3G/l. Our patient was transferred to the ICU for further monitoring and nasal oxygen therapy. After 24 hours of stabilization, she could be treated on general ward and after another 6 days she could be discharged in good general health. The patient`s newborn was tested negative for SARS-CoV-2 by nasopharyngeal swabs.

Discussion and Conclusion

Here we discuss two cases that presented to our hospital within a short time with relevant complications due to a co-infection with SARS-CoV-2 in puerperium. In our first case, we consider amnion infection to be the cause of IUFD, as placental bacteriologic culture was positive for *Escherichia coli* and SARS-CoV-2 PCR was negative in placental tissue. Additional investigations are necessary to confirm direct association between intrauterine SARS-CoV-2 infection and IUFD, as shown for many viral infections like rubella, measles, mumps, chickenpox, parvovirus B19, enterovirus and others [16]. The second case revealed acute SARS-CoV-2 pneumonia that got clinically relevant just after postpartum hemorrhage.

Both patients suffered from postpartum complications with rapid deterioration to circulatory shock and the need for treatment in the ICU. We also observed cardiac involvement with spontaneous recovery after accurate treatment. A recent systematic review and meta-analysis of 2567 pregnancies with SARS-CoV-2 infection in pregnancy revealed similar maternal morbidity to that of women of reproductive age [17]. In literature review, we identified one report of the Public Health Agency of Sweden showing an increased risk of requiring intensive care for 13/53 postpartum women with SARS-CoV-2 infection [18]. As shown in our cases, clinical course of SARS-CoV-2 infections seems to be more rapid and worse, especially in situations of peri- and postpartum complications. Because of our experience, we recommend SARS-CoV-2 screening for all pregnant and postpartum patients with a recent SARS-CoV-2 exposure and/or unexplained rapid worsening clinical course, even for patients without classical symptoms of SARS-CoV-2. This allows for more intensive monitoring for complications and rapid implementation of treatment options, if necessary.

Disclosure

None of the authors have financial interests in the manuscript.

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