

Placental Pathology in a COVID-19 Positive Patient with Abruptio Placentae- A Case Report

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ABSTRACT

The effects of Severe Acute Respiratory Distress Syndrome-Associated Coronavirus-2 (SARS-CoV-2) on the placental tissue are still being explored. Whether these placental changes result in adverse foeto-maternal outcome is an aspect that needs to be understood. This is a report of 32-year-old pregnant woman who presented with Antepartum Haemorrhage (APH) and decreased foetal movements. She was also diagnosed to be positive for the SARS-CoV-2. The patient had abruptio placentae unrelated to pre-eclampsia. On histopathological examination, the umbilical cord showed funisitis with increased perivillous fibrin deposition on section from foetal and maternal surface of the placenta. Though the foetus was stillborn, with timely management the maternal outcome was not compromised.

Keywords: Abruptio, Coronavirus disease-2019, Histopathology, Placenta, Pregnancy outcomes

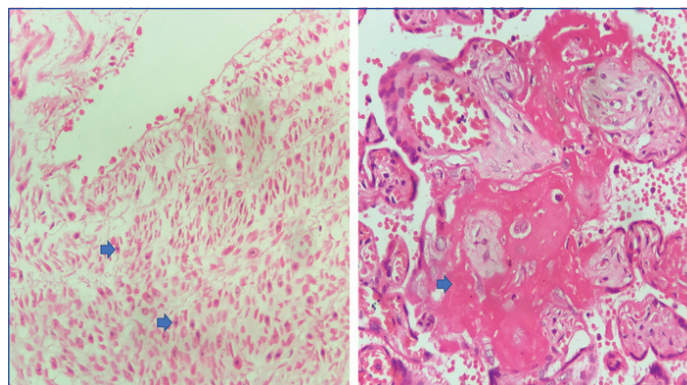
CASE REPORT

A 32-year-old secundigravida presented at gestation of 33 weeks and three days at Obstetric Emergency with complaints of abdominal pain since one day, decreased foetal movements and mild bleeding per vaginum. She had a history of previous caesarean section. The patient had no respiratory symptoms but she belonged to a locality with a high case load of Coronavirus Disease 2019 (COVID-19) and therefore, underwent a nasopharyngeal swab-Polymerase Chain Reaction (PCR) test for the SARS-CoV-2. The PCR turned out to be positive. She had normal blood pressure as per her antenatal records of this pregnancy and no complaints of headache, blurring of vision or epigastric pain which would otherwise point towards pre-eclampsia or hypertensive disorder of pregnancy. There was no precipitating factor for Antepartum Haemorrhage (APH) in the patient.

On examination, the patient was conscious and well-oriented but had significant pallor. She had tachycardia and was normotensive. The cardiovascular and respiratory system examinations were normal. On abdominal examination, the uterus was tense and tender and the fundal height corresponded to 36 weeks of gestation. Foetal heart sound was not heard on clinical examination and it was later confirmed to be an intrauterine foetal demise on obstetrics ultrasonography. The complete blood count revealed a haemoglobin of 6.7 g/dL, total leucocyte count of 12,400/cumm with 90% neutrophils and platelet count of 120,000/cumm. The liver function tests showed mildly elevated liver enzymes with Alanine Transaminase (ALT) of 113 IU/L and Aspartate Amino Transferase (AST) of 109 of IU/L. The prothrombin time and activated partial thromboplastin time were normal.

On per-speculum examination bleeding was present. On per-vaginum examination, the patient was found to be in latent labour. The diagnosis of non toxæmic abruptio placentae was made. Artificial rupture of membranes was noticed and blood mixed liquor was drained followed by torrential haemorrhage for which patient underwent an emergency caesarean section. She delivered a fresh stillborn male foetus weighing of 2.1 kg and no gross congenital anomalies. Per-operatively there was couvelaire uterus and the placental weight was 260 grams with 750 cc of retroplacental clots and 50 cc of intraplacental clots. The cord insertion and morphology was normal. The placenta was sent for histopathological examination. The patient was transfused multiple blood products intraoperatively and postoperatively. On histopathological examination, the umbilical cord showed funisitis with increased perivillous fibrin deposition on

section from foetal and maternal surface of the placenta [Table/ Fig-1]. An autopsy of the dead foetus could not be performed as the patient declined for the same. The patient was managed supportively and was discharged in a stable condition after seven days of hospital stay.



[Table/Fig-1]: Histopathological examination (100x magnification; H&E stain) showing presence of inflammatory cells in the section from umbilical cord (on left side) with increased perivillous fibrin deposition in section from foetal and maternal surface of the placenta (on right side).

DISCUSSION

The recent and ongoing pandemic of coronavirus disease 2019 (COVID-19) is caused by SARS-CoV-2 [1]. It is characterised by respiratory symptoms causing pneumonia [2]. As per the Royal College of Obstetricians and Gynaecologists recommendations, COVID-19 positive pregnant women are not considered to be at greater risk of becoming seriously unwell than other healthy adults [3]. Most of these pregnant women experiences only mild or moderate flu like symptoms. Other symptoms which they can develop includes cough, fever, shortness of breath, headache, anosmia and aguesia [3]. However, adverse perinatal outcomes including increased risks of miscarriage, pre-eclampsia, prematurity and stillbirth have been reported with COVID-19 [4]. Histopathological examination of placental tissue can provide significant information regarding the etiopathogenesis of the adverse perinatal outcomes associated with COVID-19.

With SARS-CoV-2 being a new infection, little is known about its effect on the placenta and subsequently on the foetus. In the initial studies available, evidence for vertical transmission of COVID-19

couldn't be conclusively demonstrated in small cohorts of patients [5]. However, the presence of IgM antibodies to SARS-CoV-2 in the neonate has been found, suggesting that vertical transmission is possible, although uncommon [6]. Vivanti AJ et al., have recently published a case report in which they have proven a transplacental transmission, and the newborn then went on to develop neurological symptoms attributable to COVID-19 [7]. Baergen RN and Heller DS, in a series of 20 pregnancies with COVID-19 demonstrated that 10 cases showed some evidence of foetal vascular malperfusion or foetal vascular thrombosis [8].

In COVID-19, the symptoms of hypercoagulability, are seen. This, in its most fulminant form can leads to gangrene, disseminated intravascular coagulopathy and multiorgan dysfunction [9]. Baergen RN and Heller DS in their series of 20 patients showed that fibrin deposition, villitis and funisitis were present in the placental histopathology of COVID-19 pregnancies. They attributed this to foetal vascular malperfusion. The funisitis and chorioamnionitis seen in their series were thought to be due to ascending infection [8]. The inflammation of the villi and cord may also result from a non specific inflammatory response to the virus. Present patient too had the evidence of foetal vascular malperfusion on the histopathology i.e., funisitis and perivillous fibrin deposition. This may be a result of the systemic procoagulable state that the SARS-CoV-2 is known to trigger. However, more data on the subject is needed.

CONCLUSION(S)

The SARS-CoV-2 is characterised by the presence of a hypercoagulable state which may exacerbate pregnancy associated hypercoagulability. Histopathological presence of funisitis along with perivillous fibrin

deposition in the above patient may point towards an association with foetal vascular malperfusion.

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