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Original Research Article

Histopathological Findings in Placenta Delivered from SARS-CoV2 Positive Mothers

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Abstract:

Introduction: SARS-CoV-2 infected a wide range of age groups and caused significant morbidity and mortality. Even after constant efforts to combat the virus, it could spread rapidly and infected large groups of people worldwide. Pregnant females were always at risk of acquiring the disease and the coagulopathy caused by the virus may compromise the placental circulation.

Materials and Methods: In this study, we compared histopathological findings of 61 placentas delivered from SARS-CoV2 positive mothers with 34 placentas of SARS-CoV2 negative mothers.

Results: No significant difference was found between SARS-CoV2 positive and SARS-CoV2 negative placentas in terms of period of gestation, placental weight, preterm delivery, or intrauterine fetal death. Microscopically, placentas of SARS-CoV2 positive mothers were significantly associated with features of maternal and fetal vascular malperfusion. The predominant feature of fetal malperfusion was chorangiosis and maternal malperfusion was villous agglutination & atherosis. Increased perivillous fibrin deposition was also found significantly associated with the disease.

Conclusion: COVID 19 disease is related to excessive perivillous fibrin deposition, villous edema, maternal and fetal malperfusion. However, these changes are not specific for the disease, as these changes can be found in other conditions as well. No significant adverse fetal outcome was reported in our study.

Keywords: COVID-19, Fetal vascular malperfusion, Maternal vascular malperfusion, Placenta, perivillous fibrin, SARS-CoV2.

Introduction

A novel coronavirus disease (COVID-19) caused by SARS Coronavirus 2 (SARS-CoV2), was identified in January 2020 as the cause of the outbreak of viral pneumonia in Wuhan, China. Coronaviruses are single-stranded, encapsulated RNA viruses. Bats were the only known hosts for this virus. It is known to primarily affect the gastrointestinal and respiratory systems of its host and is mainly found in the avian and mammalian species [1].

The placenta is considered an immune-privileged organ with an attenuated immune response and a target of several viral infections [2]. Various viral infections during pregnancy are associated with some specific placental findings, like lymphoplasmacytic villitis, associated enlargement of villi, and intra-villous hemosiderin deposition in cases of maternal cytomegalovirus infection [3]. Inter-villositis has been reported in the setting of Zika virus [4] and Dengue virus too [5].

The effect of COVID-19 disease on pregnant females and fetal outcomes emerged as a new field of interest among Pathologists, Obstetricians, and Pediatricians. Morphological and histological examination of the placenta may contribute significant clues about placental susceptibility to viral exposure and its consequences. Recently, it has been found that the virus may be vertically transmitted in a minority of cases infected during the third trimester [6]. Though, owing to insufficient evidence, pregnant women are advised to be extra cautious during this pandemic.

The COVID-19 pandemic became a major community health problem, with a large number of COVID-19-infected pregnant women flooding the hospitals. Histopathological examination of the placenta may help in understanding the effect of this virus on maternal and fetal health. Whether pregnant women are at higher risk of developing more severe complications than the general population is still not known, as there is an absence of comparison with appropriate controls [2]. Some of the reports that have come up during this current pandemic demonstrated various histological findings related to maternal and fetal vascular malperfusion. The effect of duration of SARS-CoV2 infection on morphologic alterations of the placenta is still not clear [2]. No pathognomonic finding has yet been defined in the placenta of COVID-19 positive mothers as per the published literature. This unmet need calls for more studies and thorough evaluation of placental samples [7,8]. However, there is one report describing placental abruption in Covid-19-positive female [9].

Here, we present a study of sixty-one placentas of Covid-positive mothers received in the Department of Pathology, intending to explore the placental Histomorphological changes of SARS-CoV2 virus infection.

Materials and Methods

This study aims at combining the placental findings in COVID-19 positive pregnant women, visiting the Obstetrics and Gynecology Department of AIIMS, Patna from July 2020 to May 2021. A case control study design was used and a total of sixty one cases (COVID-19 positive mothers) and thirty-four controls (COVID-19 negative mothers) were included in the study. All patients were tested for COVID-19 infection using nasopharyngeal and oropharyngeal swab placed in M4 viral transport medium for Reverse transcriptase PCR (RTPCR). Placentas of those who tested positive for COVID-19 RTPCR, were sent

to department of Pathology, AIIMS Patna for histopathological evaluation.

Placentas were placed in 10% buffered formalin, as soon as it was delivered and were processed according to the standard procedure. Evaluation of gross findings of the placenta were done when fresh (unfixed). Measurement of placental disc, trimmed weight, length of cord was noted. Then, it was allowed to fixed fix for minimum seventy-two hours. Thorough examination of fetal and maternal surface was done. Any gross pathological findings were recorded along with the photograph.

Sections submitted included 2 from membrane rolls, 2 of umbilical cord, 3 maternal surface, 2 full-thickness, and representative sampling of any significant lesional area. Sections underwent routine processing, embedding, sectioning at 3-4 μm and staining with hematoxylin and eosin stain. (H&E stain).

Pathological findings were recorded according to the current Amsterdam Placental Workshop Group Consensus Statement to meet an international uniform sampling criterion. It includes gross description, pathologic terminologies, and its diagnostic criteria.

Microscopic evaluation for maternal vascular malperfusion, fetal vascular malperfusion, patterns of ascending intrauterine infection and other individual features were done for both cases and control group. Each parameter and histological feature were then analyzed for each group using the chi square test and independent t-test. P value of <0.05 was considered significant [Refer table 1 and 2]. Findings of this were also compared with the available microscopic findings published in the English literature [Refer table 3].

Results

Sixty-one placentas from patients with SARS-CoV2 were examined and compared with thirty-four placentas of COVID-negative mothers. Mean age of SARS-CoV2 positive and negative females included in study was 28.3 and 25.6 years respectively. For SARS-CoV2 positive patients, 46 delivered at term and 15 were preterm. The mean period of gestation, (at the time of delivery) of SARS-CoV2 positive mothers was 37.2 weeks and SARS-CoV2 negative was 36.9 weeks. However, the difference in period of gestation of both groups were not significant(p=0.691).

In gross examination of placentas, 16.6cm and 16.1cm was the mean cord length of SARS-CoV2 positive and negative placentas respectively. The mean placental weight recorded was 414gm for SARS-CoV2 positive and 397gm for SARS-CoV2 negative placentas. The difference between the mean placental weight was not significant(p=0.461). Marginal insertion of cord in 3 placentas and presence of true knot in 2 placentas was some of the other gross findings recorded in SARS-CoV2 positive placentas. 3 placentas showed firm whitish areas, which came out to be peripheral villous infarction on microscopic examination and 4 showed areas of hemorrhage on the maternal surface.

Three SARS-CoV2 positive mothers presented with Intra uterine fetal death (at 21 weeks, 35 weeks, and 39 weeks). However, no fetal death was reported in SARS-CoV2 negative mothers. Some patients had co morbidities along with SARS-CoV-2 infections. 10 cases were associated with hypothyroidism, 7 cases with intrahepatic cholestasis of pregnancy and 6 cases with gestational diabetes mellitus, 3 cases with pregnancy induced hypertension, 3 cases with meconium-stained liquor and 2 with severe oligohydramnios. One case of each, placenta previa, portal vein thrombosis with obstructed labor, Rheumatic heart disease, hepatitis B infection and HIV infection was found in the study population. One case of twin pregnancy was also included in the study.

Increased peri villous fibrin deposition 34(55.7%) and prominent syncytial knots 21(34.4) were the most common finding

present in placentas of SARS-CoV2 positive mothers [Figure: A&B].

Features of fetal vascular malperfusion were found in a total of 30(49.1%) SARS-CoV2 positive placentas(p<0.001). Such features were absent in SARS-CoV2 negative placental specimens. Chorangiosis were seen involving 21(34.4%) (p=<0.001) of SARS-CoV2 positive placentas followed by clustered avascular villi which was present in 11(18.0%) virus positive placentas(p=0.008), [Figure: C].

However maternal vascular malperfusion was detected in 21(34.4%) SARS-CoV2 positive placentas and 2(5.0%) non SARS-CoV2 placentas. Villous agglutination, atherosis and fibrinoid necrosis were seen in significant number of cases [Figure: D]. Both were detected in 7(11.4%) of SARS-CoV2 positive placentas and none were seen in SARS-CoV2 negative cases (p=0.040).

Villous edema was found in 16(26.2%) covid positive placentas and 1(0.02%) of covid negative placentas (p=0.005).

Signs of inflammation were found insignificant. 2(3.2%) SARS-CoV2 positive and 1(0.02%) SARS-CoV2 negative placentas had features of acute inflammatory pathology (p=0.92). One case of chronic deciduitis with plasma cells was seen in each SARS-CoV2 positive and negative placentas (p=0.67).

Table 1: Comparison of clinical details between SARS-CoV2 positive and negative placenta

	SARS-CoV2 +	SARS-CoV2-	P value
No of placentas	61	34	
Mean age (years)	28.3	25.6	0.007
Mean period of gestation (weeks)	37.2	36.9	0.691
Preterm delivery	15	5	0.257
Term delivery	46	29	
Intrauterine fetal death	3	0	0.189
Mean length of cord (cm)	16.6	16.1	0.287
Mean Placental weight (gm)	414	397	0.461

Table 2: Comparison of histological features between SARS-CoV2 positive and negative placenta

Histological features	SARS CoV2 +	SARS CoV2 -	P value
Central villous infarction	0	0	--
Peripheral villous infarction	3	0	0.189
Villous agglutination	7	0	0.040
Accelerated villous maturation	0	0	--
Atherosis and fibrinoid necrosis	7	0	0.040
Mural hypertrophy of membrane arterioles	2	0	0.286
Absence of spiral artery remodelling	2	0	0.286
Retro placental hematoma	7	2	0.372
MVM -any individual feature	21	2	0.002
Clustered avascular villi	11	0	0.008
Fetal vessels – mural fibrin	4	0	0.127

Delayed villous maturation	0	0	--
Chorangiosis	21	0	<0.001
FVM-any individual feature	30	0	<0.001
Maternal inflammatory response – stage 2	2	1	0.928
Fetal inflammatory response – stage 2	0	0	--
Chronic villitis – Low grade	0	0	--
Chronic deciduitis with plasma cells	1	1	0.672
Basal plate with attached myometrial fibers	1	0	0.453
Microscopic accrete	0	0	--
Villous edema	16	1	0.005
Increased perivillous fibrin	34	3	<0.001
Intervillous thrombus	0	2	0.056
Increased circulating nucleated RBCs	0	0	--
Membrane with haemorrhage	4	0	0.127
Distal villous hypoplasia	8	1	0.105
Increased syncytial knots	21	7	0.156

Table 3: Comparison of histological features among various studies.

Histological features	This study	D.Shanes et al[8]	Natasha et al[10]	Gulersen et al[20]	Zhang et al[19]
No of placentas examined	61	16	50	50	74
Central villous infarction	0	1(7)	13 (26)	4(8)	7(9.5)
Peripheral villous infarction	3(4.9)	3 (20)			
Villous agglutination	7(11.4)	3 (20)	9(18)	--	--
Accelerated villous maturation	0	2(13)	--	0	--
Atherosis and fibrinoid necrosis	7(11.4)	3(20)	--	--	--
Mural hypertrophy of membrane arterioles	2(3.2)	5(33)	--	--	4(5.4)
Absence of spiral artery remodelling	2(3.2)	2(13)	--	--	--
Retro placental hematoma	7(11.4)	1(7)	--	2(4)	--
MVM - any individual feature	21(34.4)	11(73)	--	--	--
Clustered avascular villi	11(18.0)	4(27)	14(28) Small fibrotic villi	--	5(6.8) Avascular villi
Fetal vessels – mural fibrin	4(6.5)	1(7)	--	--	--

Delayed villous maturation	0	4(27)	--	10(20)	--
Chorangiosis	21(34.4)	4(27)	7(14)	3(6)	--
FVM – any individual feature	30(49.1)	12(80)	--	4(8)	18(24.3) Thrombosis
Maternal inflammatory response – stage 2	2(3.2)	1(7)	3(6)	11(22) + 9(18)	48(64.9)
Fetal inflammatory response – stage 2	0(0)	1(7)			
Chronic villitis – Low grade	0(0)	2(13)	--	2(4)	17(23)
Chronic deciduitis with plasma cells	1(1.6)	2(13)	--	--	--
Basal plate with attached myometrial fibers	1 (1.6)	3(20)	--	--	--
Microscopic accrete	0(0)	2(13)	--	--	--
Villous edema	16(26.2)	4(27)	--	--	--
Increased perivillous fibrin	34(55.7)	3(20)	26(52)	6(12)	2(2.7)
Intervillous thrombus	0(0)	6(40)	--	13(26)	--
Increased circulating nucleated RBCs	0(0)	1(7)	--	--	--
Membrane with haemorrhage	4(6.5)	2(13)	--	1(2)	--
Distal villous hypoplasia	8(13.1)	--	--	2(4)	--
Increased syncytial knots	21(34.4)	--	20(40)	--	--

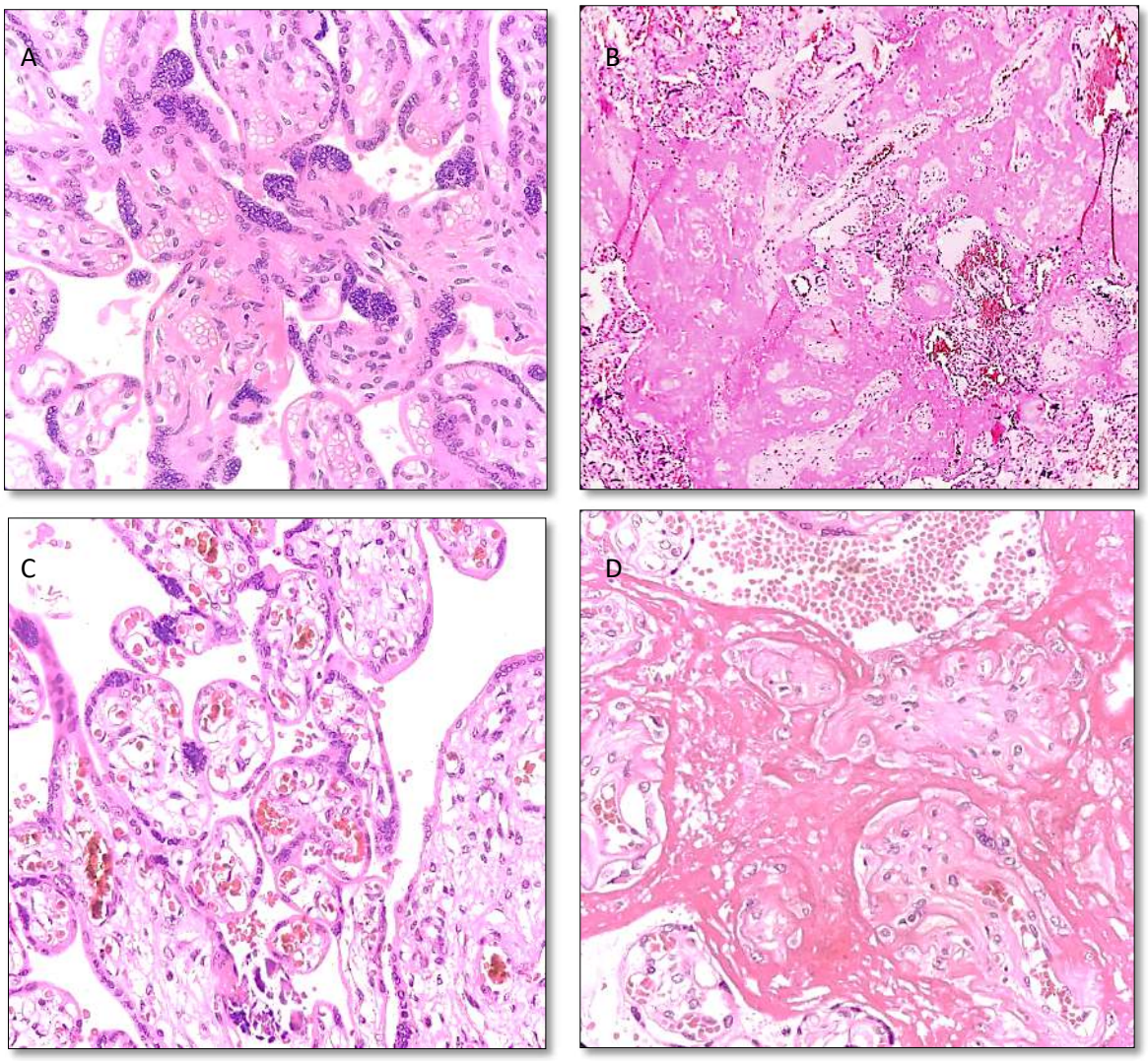

Figure A: Increased syncytial knots seen as small clusters of darkly staining syncytiotrophoblasts. (H&E, 400x)

Figure B: Increased deposition of fibrin in perivillous space. (H&E, 200x)

Figure C: Dilated and congested fetal vessels seen in tertiary villi. Few villi show more than 10 vessels per villi. (H&E, 400x)

Figure D: Agglutination of villi with fibrotic stroma and fibrin deposition in perivillous space. (H&E, 400x)

Discussion

Coronavirus is a family of viruses that is known to cause a wide range of symptoms. In the last few years, three new coronaviruses including MERS, SARS, and SARS-CoV2 have led to severe complications in human beings [10]. Coronavirus disease 2019 (COVID-19), caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV2) virus, has been declared as an emerging public health problem and led to a devastating pandemic. It can manifest as multiorgan damage, the most significant being in the lungs (diffuse alveolar damage in its different phases, microthrombi, bronchopneumonia, necrotizing bronchiolitis, viral pneumonia), heart (lymphocytic myocarditis), kidney (acute tubular injury), central nervous system (microthrombi, ischemic necrosis, acute hemorrhagic infarction, congestion, and vascular edema), lymph nodes (hemophagocytosis and histiocytosis), bone marrow (hemophagocytosis), and vasculature (deep vein thrombosis) [11]. There is only sparse

data available regarding SARS-CoV2 virus effect on the placenta, maternal and fetal outcomes [8].

Data regarding SARS-CoV2 and its impact on pregnancy are evolving very rapidly. Various studies came up with a very different outcome.

In this study, we found increased perivillous fibrin deposition, and features of fetal and maternal vascular malperfusion in a significant number of cases. Severe respiratory illness due to COVID-19 disease may cause circulatory insufficiency in both placenta as well as the fetus. Thus, the maternal COVID-19 infection can lead to hypoxia and decreased oxygen supply which may cause placental insufficiency, Intrauterine growth retardation, fetal distress or demise [12]. A systematic review and meta-analysis concluded that the most common adverse effect of coronavirus is preterm birth. Authors also concluded increased incidence of pre-eclampsia, C-section, and perinatal death [13]. However, we did not find any significant association between COVID-19 disease and preterm delivery, placental weight, intrauterine growth retardation or fetal demise.

Ng et al in 2006, studied 7 placentas from mothers with COVID-19 infection. They found 2 placentas of first trimester with almost normal histology. 3 of the placentas had subchorinonic and intervillous fibrin. They found 2 placentas with features of fetal vasculopathy and both fetuses had intrauterine growth retardation [14].

In some of the recent studies, done by shanes et al in 2019 on 16 placentas reported increased decidual arteriopathy, features of maternal venous malperfusion, intervillous thrombus, chorangiosis and delayed villous maturation as significant finding [8] (Refer table 4). Rebecca et al studied 20 placentas and found intramural fibrin deposition as most common feature of fetal vascular malperfusion. However, in our study chorangiosis and clustered avascular villi were found to be more significant. Chorangiosis occurs due decreased oxygen levels in maternal blood and it is also seen associated with mothers living at high altitude and in diabetes [8]. This cannot be concluded whether it is due to direct effect of SARS-CoV2 or just hypoxic condition induced by COVID-19 vasculopathy. They also found maternal venous malperfusion in 5 cases, features of chronic villitis in 4 cases and acute chorioamnionitis in 1 case [15].

Natasha et al, included 49 placentas and one product of conception in their study. Their results showed increased perivillous fibrin, micro calcification and increased features of malperfusion as compared to controls, which are similar to previous reported findings and also co related with our study [10]. (Refer table 4).

51 placental samples studied by Smith gall et al also reported features of fetal and maternal malperfusion, among them villous agglutination and subchorionic thrombi were the most common [16]. In concordance with this study, we also found villous agglutination in significant number of SARS-CoV2 positive cases ($p=0.04$).

Some more studies done by Chen et al and Menter et al also reported increased perivillous and intravillous fibrin as a common finding. In addition, Menter et al found prominent lymphohistiocytic villitis and intervillositis [2],[17]. Our results did not show any significant features of acute and chronic inflammation, this finding mismatched with the above-mentioned reports.

In contrast to above mentioned studies, some other authors with more number of placental samples found no relevant differences in placental histopathological patterns between SARS CoV2 infected pregnant women versus non infected women [18]. Zhang et al tested 74 placental tissues for SARS CoV2 using in situ hybridization technique. They demonstrated viral particles within syncytiotrophoblasts, atrophic endometrial glandular epithelium and subchorionic plate but did not report any significant histopathological changes and said

no relationship exists between maternal COVID-19 disease and placental pathology [19] (Ref table 4). Gulersen M et al studied 50 placentas in 2020, He M skaria 2 placentas and Hecht JL et al 19 placentas, all of them concluded that COVID-19 is not associated with any specific placental histopathology [20]– [22].

Our study revealed both concordant as well as discordant results when compared with published literature, which may be because we could not compare our findings with proper number of control cases. Also, there is need to evaluate whether increased fibrin deposition, maternal and fetal vascular malperfusion is directly related to virus or is due to coagulopathic changes reported in this viral pathogenesis. Also, no specific pathology was found which could be exclusively associated to COVID 19 disease.

Conclusion

COVID 19 disease is related to excessive perivillous fibrin deposition, villous edema, maternal and fetal malperfusion. The reason behind this may be the coagulopathic effect caused by the virus, which further lead to hypoxia and placental insufficiency. However, these changes are not specific for the disease, as these changes can be found in other conditions as well. No significant adverse fetal outcome was reported in our study. All the above findings need further validation including a greater number of cases. The correlation between histopathological findings and clinical outcomes must be done to understand the importance of changes noted in placentas

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