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SARS-CoV-2 AND PLACENTAL BARRIER: PROSPECT OF MATERNAL-FETAL TRANSMISSION

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Abstract

Severe Acute Respiratory Syndrome (SARS-COV-2) otherwise called coronavirus disease 2019 (COVID-19) is a global pandemic affecting millions of people. The placental barrier separates the fetal circulation from maternal blood in the placenta during pregnancy in humans, which plays an essential role in fetal development and health. This barrier tightly controls the exchange of endogenous and exogenous substances between the mother and the developing fetus. One of the major concerns is whether or not the SARS-COV-2 can be vertically transmitted from a pregnant mother to her developing fetus and what fetal anomalies can result from the infection. This review is aimed at evaluating the current knowledge on maternal, perinatal and neonatal outcomes and transmissibility of SARS-COV-2 infection. We conducted a comprehensive literature search using EMBASE, PubMed, and the Cochrane Library until January 2021. The following keywords were used for the search “SARS-COV-2 and pregnancy, SARS-COV-2 transmission to fetus, vertical transmission of SARS-COV-2”. Some authors proposed no maternal-fetal transmission of SARS-COV-2 while other reports suggested a probable vertical transmission of the novel corona virus based on two major evidences. More research and viable data are needed to establish the fact.

Keywords

SARS-COV-2, SARS-COV-2, Placenta, Vertical transmission, Pregnancy, Fetus

Introduction

One of the main critical aftermaths of infectious diseases is the possible transmission of the disease from the pregnant mothers to the developing fetus; this process is generally referred to as intrauterine vertical transmission.¹ Decades ago, a group of scientists identified the most frequent vertically transmitted infections and developed an acronym for them; TORCH – *Toxoplasma*, Other, Rubella virus, Cytomegalovirus, and the 2 Herpes simplex viruses (type 1 and type 2). This class of infectious diseases has since been extended to encompass Syphilis, Listeriosis, Parvovirus, Coxsackie virus, and *Trypanosoma cruzi*.¹ The vertical transmission from gravid mothers to their fetuses has been a major public health challenge with outbreaks of viral infections such as the HIV (human immunodeficiency virus),^{2,3} Ebola virus,⁴ Zika virus,^{5,6} SARS - Severe acute respiratory syndrome,^{7,8} MERS - Middle East Respiratory Syndrome.⁹ The emergence of these viral infections over the years have resulted in substantial maternal, fetal and neonatal morbidity and death. Due to its impact on the fetus and congenital Zika syndrome development, Zika virus has emerged as the newest TORCH agent.

Fortuitously, not all infectious diseases that affect pregnant mothers are transmitted to the developing fetus, these include majority of bacterial infections as well as a handful of viral pathogens.¹⁰ Most TORCH pathogens and other infectious diseases are transmitted prenatally via the placenta into the fetal circulation.¹¹

Towards the end of 2019, a novel acute respiratory disease was first reported in Wuhan city, Hubei province, China. By 26th December 2019, four separate cases of pneumonia were noticed in a Chinese hospital which was reported to the Chinese Center for Disease Control (CDC) a day after. It was etiologically identified as a

coronavirus, of the RNA viruses, and the genus of Betacoronaviruses. Due to the high contagious rate of the disease, the World Health Organization (WHO), declared the virus as a pandemic on 11th March 2020 and named it severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2).¹² Today, SARS-CoV-2 has spread to all countries of the world, with over 92,493,946 laboratory-confirmed cases and over 1,980,825 deaths globally. The Americas and Europe are the worst hit continents, recording millions of novel SARS-COV-2 positive cases while Africa remains the least affected continent.¹³

SARS-CoV-2 is a zoonotic disease. Earlier reports drew a correlation between the Huanan Seafood Wholesale Market, a popular hub for buying and selling different species of animals and SARS-CoV-2 with 26 of 47 cases in one report and another report, 27 of 41 with epidemiologic links to the same market.^{14,15} The market was finally closed January 1st, 2020 as a result. With more than 85% nucleotide identity with a bat SARS-like CoV, the SARS-CoV-2 is most closely related to the coronaviruses found in bats.^{16,17}

The severe acute respiratory syndrome (SARS-CoV) in 2002 spilled out from Guangdong markets in China and Middle East respiratory syndrome (MERS-CoV) in Saudi Arabia in 2012, both caused by *coronaviridae*. The main reservoir of coronaviruses are bats which transmit the pathogen to intermediate links such as palm civets (SARS) or dromedary camels (MERS) and then to humans. While the SARS was declared contained in 2004,¹⁸ MERS outbreak still continues till today.¹²

The aim of this review study was to evaluate from the available pool of empirical clinical and laboratory data, the current knowledge and prospects of vertical transmission of SARS-COV-2 from pregnant mother to the developing fetus. We conducted a comprehensive literature search using

EMBASE, PubMed, and the Cochrane Library until January 2021. The following keywords were used for the search “SARS-COV-2 and pregnancy, SARS-COV-2 transmission to fetus, vertical transmission of SARS-COV-2”. Inclusion criteria were articles reporting clinically diagnosed and/or laboratory-confirmed SARS-COV-2, patient being pregnant on admission and availability of clinical characteristics, including at least one maternal, perinatal or neonatal outcome. Unreported maternal or perinatal outcomes, non-peer-reviewed or unpublished reports, suspicion of duplicate reporting, and unspecified date and location of the study were excluded. We applied no language restrictions.

Transmission of SARS-CoV-2 infection

The main transmission route for SARS-CoV-2 among humans is similar to transmission of most other respiratory pathogens and influenza that occurs through respiratory droplets formed when an infected person sneezes or coughs and the droplets are inhaled by close contacts, usually within 6 feet. While it is generally not clear yet if SARS-CoV-2 can be transmitted via fomites, reports have indicated a potential fecal-oral transmission as SARS-CoV-2 has also been identified in faecal specimens.¹⁹ Recent evidence suggests that SARS-CoV-2 may have the potential to be transmitted through aerosols^{20,21} as well.

Symptoms of SARS-CoV-2 infection

The frequent early clinical manifestations of SARS-COV-2 infection are cough, fever, myalgia and fatigue, and sometimes headache and diarrhea.²² In pregnant women, cough and fever are the most common symptoms of SARS-CoV-2 infection; other symptoms experienced by pregnant women includes myalgia, headache, dyspnea, chest pain and sore throat.²³

Diagnosis of SARS-CoV-2 infection

A host of factors are considered in diagnosing SARS-COV-2 infection, these are clinical manifestation, epidemiological exposure history, laboratory test results, a positive reverse transcription-polymerase chain reaction (RT-PCR) result and chest computed tomography (CT) findings.^{24,25} It is suggested that the precise number of SARS-CoV-2 infections would be difficult to estimate due to the inability to detect all mild and asymptomatic cases.²⁶

Pathophysiology of SARS-CoV-2 infection in pregnancy

There have been well documented trends postulating that pregnant women have higher tendencies of being severely affected during pandemics.^{27,28} Non-Pregnant women are less vulnerable to respiratory disease-causing agents and pneumonia compared to pregnant women. The susceptibility of pregnant women to respiratory pathogens compared to non-pregnant women may be due to pregnancy physiological adaptation, that is, suppressed immune system, diaphragmatic elevation, increased oxygen consumption and edematous airways in pregnant women.⁸ The earlier severe acute respiratory syndrome (SARS) report suggested that nearly half of pregnant women who caught the SARS virus were admitted in intensive care unit (ICU) due to low oxygen saturation and over 60% of them required mechanical ventilation; more than half of which died in the process.²⁹ Similarly, pregnant women were reported to be four times more likely to be hospitalized and at increased risk of pregnancy complications during the H1N1 influenza virus outbreak in 2009; compared with the general population.³⁰ Also during the influenza pandemic in 1918, there was a very high mortality rate among pregnant women as the proportion of mortality reported among over 1300 cases of pregnant women affected by the influenza virus was 27%.²⁷

SARS-CoV-2 have been associated with severe consequences for pregnant mothers i.e. preterm labor and delivery, asphyxia, membranes premature rupture, pneumonia, intrauterine growth restriction, intrauterine fetal distress, low birth weight, stillbirth, respiratory distress and feeding intolerance.¹² Though casualties among pregnant women from this infection has not yet been fully estimated.

Several pathologies such as low birth weight, neurodevelopmental delay, patent ductus arteriosus, surfactant deficiency, elevated cortisol levels with implications for long-term stress response, chronic inflammatory states, that is, atopic sensitization amongst others have also been discovered in neonates infected with SARS-CoV-2 virus.³¹

The placental barrier

Two cell layers (the fetal capillary endothelium and the placental trophoblast) separate the fetal circulation from maternal blood in the placenta during pregnancy in humans forming the placental barrier, which plays an essential role in fetal development and health.

This barrier tightly controls the exchange of endogenous and exogenous substances between the mother and the developing fetus.³² A multilayered membranous structure consisting of the fetal capillary endothelium and the syncytiotrophoblast separated by a thin interstitium is pivotal to the efficacy of this organ's regulatory roles. This structure separates the maternal intervillous space and fetal circulation, forming a specialized barrier that is responsible for controlling the selectivity and rate of placental transport (Figures 1 & 2). Any alteration in this delicate structural organization results in complications of pregnancy, and possible fatal outcomes for both mother and fetus. Such structural compromise exposes the pregnancy to dangers which might have untoward results both ways, either the mother or the developing fetus. It may cause birth defects, or have rather subtle impacts that can affect adult health later in life via epigenetic developmental programming.³³

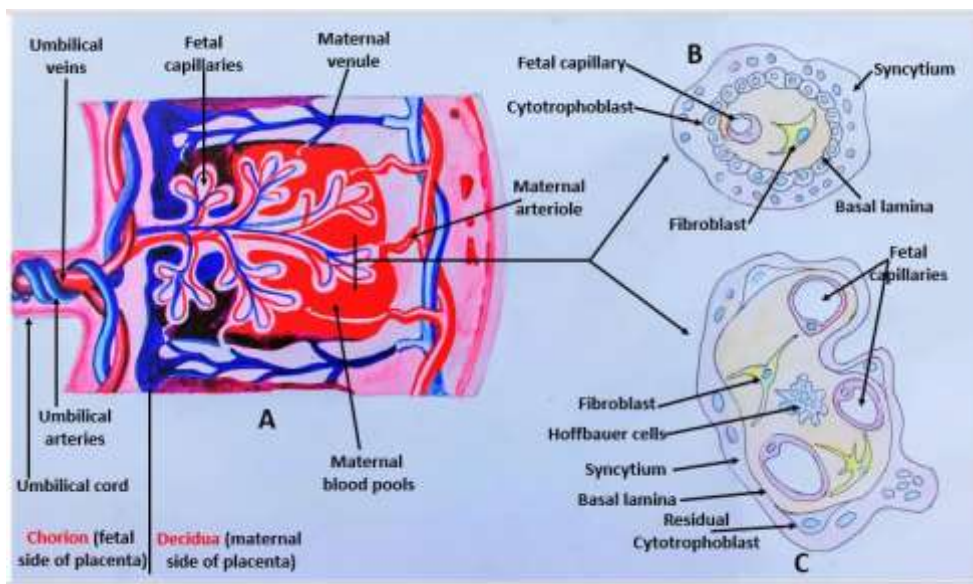


Figure 1: Schematic drawings of the human placenta; a specialized barrier responsible for controlling the selectivity and rate of feto-maternal transport. (A) Fetal placental circulation. (B) Chorionic villous showing the syncytiotrophoblast, a layer of cytotrophoblast cells, the villus stroma with fibroblasts and the fetal capillaries. (C) Term placenta. (Adapted from Murthi et al. 2014)³⁴

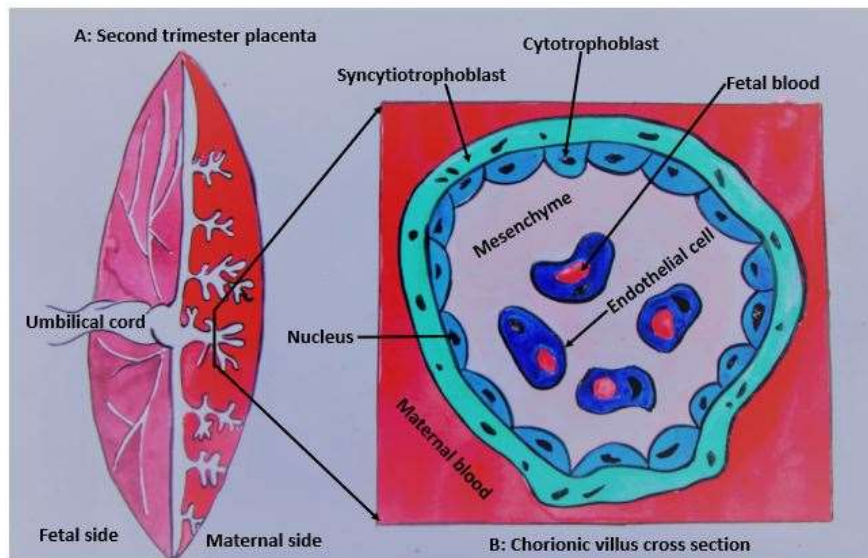


Figure 2: Structural illustration of a second-trimester placenta (A) and cross-section of a chorionic villus (B) placental barrier between fetal and maternal blood. These separate the maternal intervillous space and fetal circulation, forming a specialized barrier that is responsible for controlling the selectivity and rate of placental transport. (Adapted from Hogg et al. 2012)³³

The blood supply to the uterus is through two arteries, the uterine and ovarian arteries, these form the arcuate arteries from which radial arteries enter the myometrial wall. These radial arteries in turn give rise to spiral arteries that supply maternal blood into the intervillous space and bathes the chorionic villi in maternal blood. The fetal internal iliac arteries give rise to two umbilical arteries that carry deoxygenated fetal blood through the umbilical cord to the placenta. Umbilical arteries terminate as capillaries within the villi after dividing into chorionic arteries. The progression follows that substances from the maternal blood pass through the intervillous space via the syncytiotrophoblast, the fetal stroma (connective tissue), and the fetal capillaries endothelium into the fetal circulation. These capillaries in turn drain into chorionic veins which finally empty into a single umbilical vein (Figure 1).³⁵ Substances are thus transported across four tissue levels between maternal and fetal blood: the multinucleated syncytiotrophoblast layer, the

cytotrophoblast cell layer, the villous mesenchyme (fetal stroma), and the endothelial cells lining of the fetal vessels (Figure 2 & 3)^{33,34}.

Only two cell layers separate the fetal and maternal circulations in the human term placenta; the fetal capillary endothelium and the syncytiotrophoblast. The syncytiotrophoblast plays the role of transporting epithelium in the human placenta and has two polarized plasma membranes: a microvillous plasma membrane facing the maternal blood within the intervillous space, and a basal plasma membrane that faces the fetal capillary side (Figure 1).^{36,37} At term, in some parts of the placental membrane regarded as the vasculo-syncytial membrane is very thin, such that the syncytiotrophoblast comes directly in contact with the fetal capillary endothelium (Figure 1).

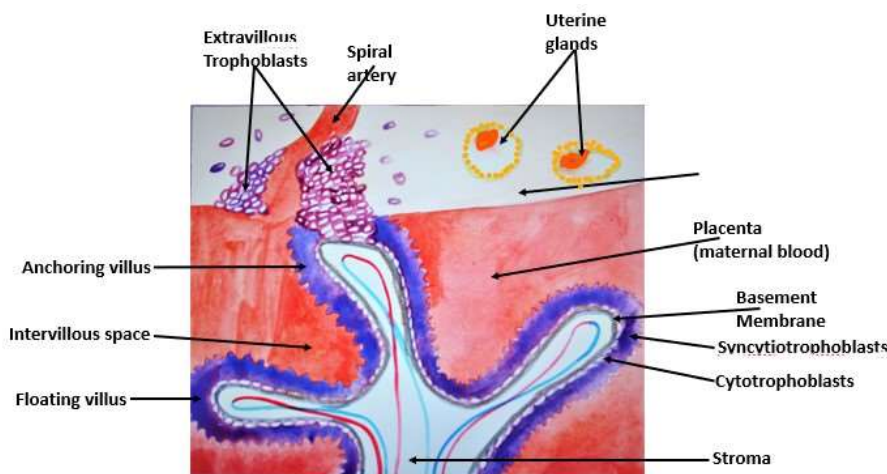


Figure 3: The placental villous tree. Showing the cellular and microscopic interactions among the maternal intervillous space, the placenta, and the fetal circulatory system. (Adapted from Delorme-Axford et al. 2014)³⁸

All through gestation, the placenta serves as both immunological and physical barrier against mother to fetus hematogenous transmission of viruses. The genome size of the SARS-CoV-2 varies from 29.8 kb to 29.9 kb and its genome structure followed the specific gene characteristics to known CoVs³⁸, this in itself may not encourage easy passage through the physical placental barrier. Probable machineries of SARS-CoV-2 vertical transmission include but not limited to transmission through maternal vaginal tract, access via maternal circulation to extravillous trophoblasts and other placental cells, breach via the syncytial layers and direct contagion of syncytiotrophoblasts, and enroute maternal immune cells³⁹, but current evidences suggest that at the maternal-fetal interface, placental trophoblasts and the resident accompanying immune cells such as natural killer cells, macrophages, T regulatory cells, T lymphocytes, and dendritic cells regulate immune defense mechanisms.³⁸ Moreover, emerging evidences also suggest that these defense mechanisms might also include microRNA-mediated antiviral defense in human placental trophoblasts, certain antimicrobial peptides and proteins, and

atypical interferons in the reproductive tracts of both genders. These suggestions may not be in tandem with the popular immunosuppressed state view of gravidity.³⁸

SARS-CoV-2 is believed to gain access into the human cell by utilizing two proteins: the angiotensin-converting enzyme 2 (ACE2), a receptor which enhances viral cell attachment, and an enzyme, the type II transmembrane serine protease (TMPRSS2) that facilitates cell infection progression.⁴⁰ A most recent study gave a preliminary claim that only a few number of cells co-express ACE2 and TMPRSS2 in the lungs, kidney and bladder. Although they warned that infection of the human placenta might occur via alternative routes by interacting with other proteins, such as Basigin (also known as CD147 or EMMPRIN), a transmembrane protein that is capable of providing an alternative cell entry for SARS-CoV-2 when ACE2 and TMPRSS2 are not expressed, however, they reported only four cells (stroma cells and perivascular cells in decidua, villous cytotrophoblast and syncytiotrophoblast in placenta) exhibiting co-expression in any of the gestational trimesters, with an estimated count below 1 per 10,000 cell

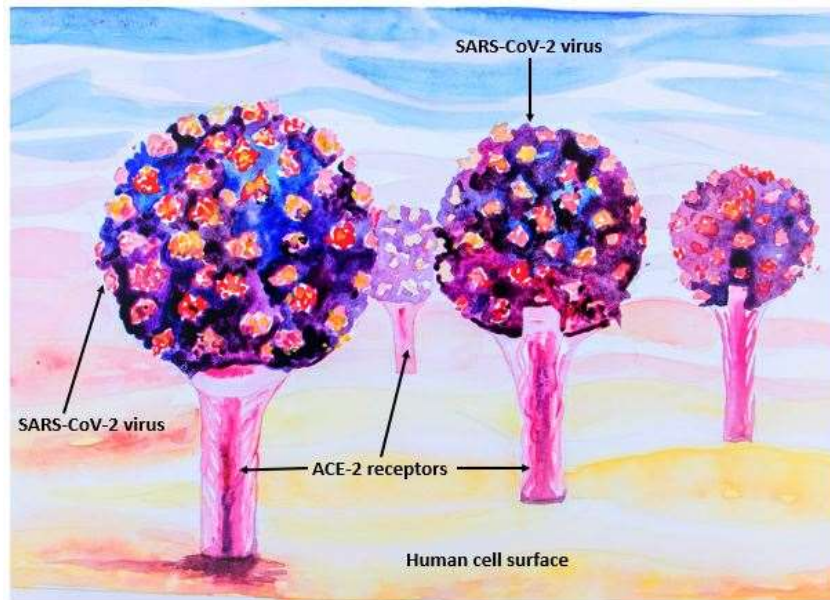


Figure 4: The initiation stage in COVID-19 infection, when SARS-CoV-2 viruses bind to ACE-2 receptors on the human cell (Adapted from Kateryna Kon / Shutterstock).⁴⁰

DISCUSSION

SARS-CoV-2 transmission at conception

Molecular studies have established that SARS-CoV-2 gains entrance into body cells when they bind to host receptor angiotensin-converting enzyme 2 (ACE2), using the spike (S) glycoprotein on its outer surface.^{30,41,42} Host proteases such as transmembrane serine protease 2 (TMPRSS2) are then required to hew the viral S protein to initiate a conformational change to the S that permits for permanent blend of the host and viral cell membranes.^{30,40,41} Downregulation of TMPRSS2 in target lung tissue has shown that SARS-CoV-2 was unable to gain entrance into the lung cells, hence the importance of TMPRSS2 in propelling SARS-CoV-2 into the body cells.³⁰ ACE2 may be one of the main predictors of whether any body cell type is susceptible to SARS-CoV-2 infection or not, TMPRSS2 is more broadly expressed in human tissues than ACE2.³⁰

A study analyzed transcriptomic and proteomic data for identification of cell types that co-express ACE2 and TMPRSS2 genes based on single cell RNA sequencing data, ACE2 and TMPRSS2 co-expression was not observed in testicular spermatogenic series, sperm inclusive; although in non-human primates, a subpopulation of ovarian tissue were observed to express ACE2 and TMPRSS2, nevertheless co-expression was not observed in ovarian somatic cells.⁴² The effects of SARS-CoV-2 on male and female fertility are indeterminate but, findings from the molecular study indicated that SARS-CoV-2 may not have a long-term effect on the structure and function of male and female reproductive system and also quash the possibility of transmitting the virus from gametes to embryos.⁴³

SARS-CoV-2 transmission in pregnancy

The rising cases of neonates testing positive for SARS-CoV-2 have shifted attention to possible vertical transmissibility of the novel SARS-CoV-2. Among the most disturbing outcomes of novel viral disease outbreaks is the potential effects of the virus on pregnancy; thus, ascertaining the potential vertical transmissibility of viral outbreak from mother to child is of paramount importance. Mechanism of vertical transmission include intrauterine infection via the placenta, intrapartum infection during delivery and postpartum infection. The intrauterine transplacental mechanism is the most common cause of viral transfer from mother to developing foetus, especially having been reported to be the main mechanism Zika, hepatitis E, HIV and Ebola viruses were transmitted to developing foetus.^{44,45}

There have been six (6) coronaviruses earlier known to infect people before SARS-CoV-2. Four of those coronaviruses namely; two Alphacoronaviruses (HCoV-229E and HCoV-NL63) and two Betacoronaviruses (HCoVOC43 and HCoV-HKU1) usually cause the common cold and are capable of passing through the placental barrier to the developing fetus, that is, can be transmitted vertically.²⁵ Other Betacoronaviruses, MERS and SARS are capable of bringing about life threatening effects in pregnant women, such as fetal growth restriction, miscarriages, preterm labor and substantial rise in maternal mortality.^{22,25}

Well documented reports have indicated that pregnant women, due to their suppressed immunologic responses, generally makes them a highly vulnerable group for numerous infectious diseases according to earlier experiences with SARS, MERS and influenza. Women during pregnancy are more vulnerable to having life-threatening pneumonia when infected by respiratory pathogens than non-pregnant women.⁴⁶

Despite multiple case reports and cohort studies describing clinical course, laboratory and radiological observations coupled with mode of parturition (Cesarean or vaginal delivery) on pregnant women with SARS-CoV-2 infection, pregnancy-specific data on SARS-CoV-2 are still limited as most of the reported cases are women during their third trimester; there is paucity of data during the first and second trimester of pregnancy.^{23,47}

Breslin et al., studied 18 pregnant women (4 symptomatic; 14 initially asymptomatic) who delivered at Columbia University Irving Medical Center (New York, NY) or the Allen Hospital (New York, NY). Eight (8; 44.5%) had a cesarean delivery while the remaining ten women (10; 55.5%) had normal vaginal deliveries without complications. The APGAR score of all 18 infants were ≥ 7 and ≥ 9 at first (1st) minute and at fifth (5th) minutes respectively and further tested for SARS-CoV-2 infection using PCR nasopharyngeal swab of which all but one out of the 18 infants on day of life 1-2 (DOL 1 - 2) tested negative. The other infants had an “inconclusive” test result, which was clinically managed as a “presumptive negative” diagnosis, as this result may equally indicate low-level detection. None of these neonates was subjected to IgG/IgM SARSCoV-2 testing.²³

Throat swabs from nine (9) neonates of mothers diagnosed with SARS-CoV-2 were tested and all came back negative.⁴⁸ Liu et al.⁴⁹ reported that there were no serologic or clinical evidence for vertical transmission of SARS-CoV-2 infection in neonates given birth to by SARS-CoV-2 positive mother. All the infants got a 1-min Apgar average score of 10.⁴⁹ Weiyong and others got negative SARS-CoV-2 test from maternal vaginal mucus and placenta.⁵⁰ Cord blood, amniotic fluid from six (6) laboratory confirmed SARS-CoV-2 infected women and throat swabs from their neonates were subjected to SAR-CoV-2 test all of which came back negative,⁵¹ and Wang also

obtained a SARS-CoV-2 negative test results from the placenta, amniotic fluid, and cord blood of women already positive to SARS-CoV-2.⁵² These imply that SARS-CoV-2 infection could not have been transferred vertically from an infected mother to her fetus.

Contrarily, Baud and other scientists reported a case of potential vertical transmission of SARS-CoV-2 from mother to the fetus in their observations. A single case of primigravida woman with symptoms of SARS-CoV-2 infection during the third trimester of the pregnancy; a nasopharyngeal swab test conducted came back positive for SARS-CoV-2. A few days later she developed a fever, severe uterine contraction and persistent SARS-CoV-2 symptoms. Physical examination, vaginal examination and ultrasound scanning were conducted of which no sign of pneumonia was observed after physical examination, but vaginal examination shows signs of bulging membranes and ultrasonography revealed fetal tachycardia of 180/min, active fetal movements and normal amniotic fluid, fetal morphology, and growth.³⁴ The patient in the end delivered prematurely through the vagina to a still born infant after long hours of labor. Vaginal swabs and amniotic fluid collected during labor were negative for SARS-CoV-2. Fetal samples such as fetal blood, swabs from mouth, axillae and meconium were obtained within minutes after birth and tested for SARS-CoV-2, all samples came back negative for SARS-CoV-2 infection and bacterial infection. Fetal autopsy revealed no congenital malformations and biopsied fetal tissues, that is, fetal lung, liver and thymus came back negative for SARS-CoV-2 after test.⁴⁷ Maternal blood, urine and vaginal swabs collected after delivery came back negative for SARS-CoV-2 whereas a nasopharyngeal swab remained positive. Swabs and biopsy from the fetal surface of the disinfected placenta both came back positive for SARS-CoV-2 after 24 hours and 48 hours after delivery. Haematoxylin and Eosin-stained

placenta showed signs of inflammation and funisitis. This probably suggests complications from the severe immune reaction or the so called “Cytokine Storm” associated with SARS-CoV-2 Gram and periodic acid–Schiff staining of the placenta, culture and PCR test came back negative for any bacterial or fungal infections.^{47,53,54}

A SARS-CoV-2 positive pregnant woman who presented symptoms such as malaise, low-grade fever, and progressive shortness of breath who ultimately developed respiratory failure that required mechanical ventilation; and eventually gave birth prematurely via caesarian delivery. The infant was immediately separated from the mother and isolated to avert physical transmission; 16 hours post-delivery, the infant nasopharyngeal swab tested for SARS-CoV-2 while real-time polymerase chain reaction (RT-PCR), and immunoglobulin (Ig)-M and IgG for SARS-CoV-2 were negative. On postpartum day 4, maternal IgM and IgG became positive.⁵⁵

Mingyang et al.⁴¹ reported cases of three newborns from mothers with COVID-19 in Henan Province, China. All three women delivered premature babies (30.5-, 36- & 37-week’s gestation) via caesarean section and of all the three neonates delivered, only one tested positive for SARS-CoV-2. The mother of the SARS-CoV-2 positive baby had traveled from Wuhan to Xinyang, Henan province before she was diagnosed with COVID-19 and eventual intubation due to the worsened health condition caused by the viral infection. The baby had Apgar score of 9 and 9 at 1 and 5 minutes respectively and was discharged 11 hours after delivery while the mother was recovering in the hospital. On postnatal day 6, the baby tested positive for SARS-CoV-2 and the caregiver later tested positive for SARS-CoV-2 three days after. This may be a case of probable vertical transmission of SAR-CoV-2 from mother to child, even though no laboratory tests were reported to

have been conducted immediately after the delivery of the newborn to support the claim.⁵⁶

These isolated but consistent cases of SARS-CoV-2 infection in neonates begs the question of vertical transmission of the virus from mothers to fetus prior to parturition. Facchetti and coauthors ran a comprehensive pathological evaluation on the placenta from a term neonate who had tested for SARS-CoV-2 infection accompanied by other systemic diseases and discovered fetal vascular malperfusion, damage to and presence of SARS-CoV-2 in the syncytiotrophoblast and increasing level of immune cells, that is, monocyte-macrophages and mature and immature neutrophils and platelets suggesting inflammatory responses.^{57,58}

Placentas from two SARS-CoV-2 infected mothers who had given birth to neonates who tested positive to SARS-CoV-2 infection postnatally were examined, results from their findings include chronic intervillitis with presence of macrophage CD68+. In addition, SARS-CoV-2 pathogen was discovered in the placenta tissue samples from infected mothers, which is in contrast to non-infected mothers. In the same vein, Penfield et al. also reported the presence of SARS-CoV-2 RNA in three (3) out of eleven (11) placental samples from SARS-CoV-2 positive mothers even though none of the infants demonstrated symptoms or tested positive to SARS-CoV-2 infection.^{59,60,61}

Pre-exposure prophylaxis

Pregnant women presumably have increased risk of developing severe complications of COVID-19 such as respiratory failure, preterm delivery, or death. In spite of the development of COVID-19 vaccines, pregnant women may not be eligible for COVID-19 vaccines at the initial stage as there has not been any special considerations for this population in the

present COVID-19 vaccines trials and safety testing.⁶² The idea of pre-exposure prophylaxis using pregnancy adaptable medications such as hydroxychloroquine as an alternate approach to prevent gestational problems and severe illness in this high-risk population is very essential, which will provide an affordable and safe means of avoiding COVID-19 hitches in pregnancy.⁶² Other drugs such as atovaquone, ivermectin, nitazoxanide, tafenoquine, and mefloquine are unfortunately considered mostly unsafe during pregnancy.⁶²

In contrast to the Pre-exposure prophylaxis concept, a preprint case report and major online news^{63,64,65} reported the case of a 36-week pregnant woman in South Florida, United States, who gave birth to a babygirl with coronavirus antibody three weeks after receiving Moderna COVID-19 vaccine jab. Using the baby's umbilical cord blood taken immediately after delivery prior to placenta ejection, the preprint study stated that they have demonstrated that SARS-CoV-2 IgG antibodies are detectable in a newborn's cord blood sample sequel to maternal's single dose of Moderna COVID-19 vaccine.^{63,64,65} This being the first reported case may not mean much as yet, but aligns with some other vaccines such as the flu vaccine used in pregnant women, and it thus gives a glitter of hope. Several studies are underway involving non-pregnant women, pregnant women and infants.⁶⁵ Results from these studies would give deep insight into the risks, safety and efficacy of the COVID vaccines during pregnancy which needs to be ascertained as soon as possible, hence more research into this possibility becomes a necessity as the global war against SARS-CoV-2 continues.

Conclusion

From the available scientific evidences, one cannot conclusively assert that SARS-CoV-2 infection cannot be vertically transmitted from mother to developing fetuses. Hence, the need for more research and data on the

novel coronavirus, not only to establish facts about its effects on pregnant women and the fate of their respective fetuses but also to curtail its rapidly increasing number of cases, deaths and countries affected. Earlier reports and experiences from SARS, MERS and other respiratory infection-causing pathogens indicated that these infections could severely alter the wellbeing of pregnant women and presently, there is paucity of published data on pregnant women living with SARS-CoV-2 infection on which to proffer recommendations on pregnancy-specific care.

Though, there are no reports that suggest that pregnancy increases vulnerability to SARS-CoV-2; in fact, more men have tested positive for the novel coronavirus than women which might be due to differences in exposure, susceptibility, diagnosis and/or reporting. Also, there is no clinical evidence that pregnant women are more vulnerable to other forms of coronavirus, that is, MERS and SARS. Hence, it is vital to ensure implementation of standard interventions for SARS-CoV-2 control and management of severe respiratory infection in any community.

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Odukoya, S.O.A: Conceptualization, Methodology, Writing, Reviewing and Editing

Lawal, I.A.: Articles sourcing, Writing-Original draft preparation.

Odukoya, A.O.: Writing- Original draft preparation.

Adefolaju, G.A.: Reviewing and Editing

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