The placenta in COVID-19 infection in pregnancy

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Vertical transmission of SARS-cov-2 occurs in approximately 3% (0–9%) of maternal infections, depending on the criteria used (Saadaoui et al. J Pers Med. 2021;11(6):483). However, perinatal mortality and morbidity may not be related to the perinatal infection itself. This is probably due to the absence of SARS-CoV-2 receptors in the vital organs of the fetus, their presence being confined to the kidneys, lungs and intestine (Faure-Bardon et al. Ultrasound Obstet Gynecol. 2021;57(2):242–7). Thus, most adverse effects occur through iatrogenic prematurity to save severely symptomatic mothers with acute respiratory and inflammatory distress syndrome and heart failure (Wong et al. Diagnostics (Basel). 2021;11(1):94).

This dramatic presentation of severe maternal illness has long overshadowed the critical role of placental infection. Angiotensin-converting enzyme (ACE) receptors are strongly present in the syncytiotrophoblast from early pregnancy and the resulting chronic histiocytic intervillositis and trophoblast necrosis are specific placental findings in SARS-CoV-2 (Schwartz and Levitan Arch Pathol Lab Med. 2021;145(8):925–8), (Schwartz and Morotti Viruses. 2020;12(11):1308).

In this issue, Zaigham et al. report a case series that demonstrates a significant pattern of perinatal mortality and morbidity related to placental infection. The 13 cases reported here illustrate four important findings:

- The severity of placental pathology is independent of maternal symptoms and can also develop in asymptomatic women in the second and third trimesters.
- Fetal growth can be severely impaired, as has been demonstrated in two-thirds of cases diagnosed with fetal distress with decreased fetal movements.
- Vertical transmission with fetal or neonatal infection is neither constant nor necessary to precipitate intraterine fetal death (IUFD) or fetal distress.
- An interval of a few days to 25 weeks could separate the diagnosis of maternal infection from the diagnosis of IUFD or fetal distress.

The design of the study does not allow estimation of the prevalence of this severe outcome among mothers with the infection, nor can it shed light on the proportion of IUFDs related to SARS-CoV-2-placental infection among all women with IUFD. The former is unlikely to be assessed accurately, as asymptomatic, and therefore undiagnosed, women may be equally affected. For the latter, it is necessary to recognise the triad of placental pathologies related to SARS-CoV-2 and confirm it by RT-PCR on placental tissue.

The authors did not indicate the vaccination status of these women. It is to be expected that only, or mainly, unvaccinated women would be exposed. However, at this time of a prolonged pandemic, with highly infectious variants and unstable vaccine efficacy, this study, along with a handful of similar case reports, should alert pregnant women and obstetricians to the clinical and ultrasound features of placental insufficiency. One might even suggest that all documented cases of maternal infection should prompt increased surveillance because of placental effects.

CONFLICT OF INTERESTS
None declared. Completed disclosure of interest forms are available to view online as supporting information.

DATA AVAILABILITY STATEMENT
Data sharing not applicable - no new data generated.

SUPPORTING INFORMATION
Additional supporting information may be found in the online version of the article at the publisher’s website.