International Journal of Community Medicine and Public Health
Memon FZ et al. Int J Community Med Public Health. 2026 Jul;13(7):4022-4030
https://www.ijcmph.com
pISSN 2394-6032 | eISSN 2394-6040

DOI: https://dx.doi.org/10.18203/2394-6040.ijcmph20262312

Review Article

Maternal and neonatal biochemical sequelae following prenatal COVID-19 infection: evidence across the peripartum period

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Received: 26 April 2026
Accepted: 22 June 2026

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ABSTRACT

Pregnancy is characterized by dynamic physiological, immunological, and biochemical adaptations that sustain foetal growth and maternal health. Viral infections such as COVID-19 can disrupt this equilibrium, provoking systemic inflammatory, metabolic, and coagulative responses. While clinical complications of prenatal SARS-CoV-2 infection have been widely described, evidence on biochemical sequelae across pregnancy, delivery, and postpartum remains fragmented. Therefore, this scoping review aims to map published evidence on maternal, placental, and neonatal biochemical markers following prenatal COVID-19 infection across the peripartum period. Following PRISMA-ScR guidelines, systematic searches were conducted in PubMed, Google Scholar, and Epistemonikos from January 2020 to March 2025. Observational studies examining biochemical or immunological markers among COVID-19 infected pregnant women and their offspring were included. Data were extracted using a pre-tested charting tool and narratively synthesized during pregnancy, at delivery, and postpartum. Twenty-seven studies met inclusion criteria. Elevated inflammatory, and haematological markers were consistently observed during pregnancy. Neonatal respiratory distress and multisystem inflammatory manifestations were associated with elevated inflammatory markers. Postpartum evidence suggested presence of immunoglobulins in breast milk, thus providing passive neonatal protection. Expression of placental ACE2 and TMPRSS2 suggested viral entry expression, aligning with limited evidence of vertical transmission. Prenatal COVID-19 infection led to a wide spectrum of biochemical alterations in maternal and neonatal systems. However, emerging evidence remains heterogeneous and limited beyond delivery, hence underscoring the need for longitudinal biochemical surveillance across the peripartum period.

Keywords: Biochemical markers, COVID-19, Inflammation, Maternal health, Neonatal outcomes.

INTRODUCTION

Coronavirus disease (COVID-19), caused by the SARS-CoV-2 virus, has had a profound impact on global health. According to WHO, the COVID-19 pandemic resulted in approximately 2 million reported deaths, with a global excess mortality of 14.9 million (uncertainty range: 13.3-16.6 million) during 2020–2021.[1] All subsets of groups were susceptible to COVID-19 infection; however, the clinical outcomes varied significantly due to differences in the immune responses of the hosts. A particular concern group were the pregnant women due to their unique state of physiological changes and immune modulation. Pregnancy is a balanced state of physiological, metabolic, and immunological systems with marked, trimester-dependent biochemical adaptations that regulate nutrient transfer to support foetal growth and development, renal clearance, protein

metabolism and glucose homeostasis, providing the reference frame for interpreting infection-related changes.[2] Maternal viral infection may provoke systemic inflammatory, coagulatory and metabolic responses.

Empirical evidence on prenatal viral infection showed parallel mechanisms: maternal influenza is associated with inflammatory responses and increased risk of preterm birth, while congenital zika viruses induced placental inflammation and metabolic reprogramming impaired foetal growth and development.[3] In case of COVID-19 infection, the literature cited disruption of the normal balance, altering immune responses and metabolic control.[4,5] The virus affects pregnant women and their offspring, not only during the acute phase of infection but also during the postpartum and early childhood periods.

Clinical outcomes such as preterm birth and maternal complications have been widely reported; however, there is a dearth of evidence on biochemical sequelae across the trimester, during delivery and postpartum, particularly regarding heterogeneity of markers, timing and clinical correlations.[4-6]

Thus, it is very critical to map and summarize published evidence on maternal, placental and neonatal biochemical and immunologic markers following prenatal SARS-CoV-2 infection, and to identify gaps for future research.

METHODS

Protocol and registration

The study adopted a scoping review methodology; it incorporates frameworks developed by the Preferred Reporting Items for Systematic Reviews and Meta-analysis Extension for Scoping Reviews (PRISMA-ScR), The final protocol was registered prospectively with the Open Science Framework on 25th August 2025, (https://osf.io/9aw4j).

Eligibility criteria

The PICOS (Population, Intervention, Comparator, Outcome, Study design) eligibility criteria are as follows: Population (P): Women with a history of COVID-19 infection during pregnancy and/or children with in utero exposure to SARS-CoV-2, defined as those born to mothers diagnosed with COVID-19 during pregnancy. Intervention/Exposure (I/E): COVID-19 infection among mothers and/or exposure among their offspring.

Comparator (C): Women without a history of COVID-19 infection during pregnancy and/or their children without in utero exposure to SARS-CoV-2 (unexposed group), or no comparator group in single-arm observational studies. Outcome (O): Any reported changes in biochemical markers in either the mother or the offspring during the perinatal or postnatal period.

Study design

All types of observational studies, including case reports, case series, cross-sectional studies, and cohort studies were involved.

Geography

No geographic restrictions.

Information sources and search strategy

Two authors independently developed our search strategy, which included both controlled Medical Subject Headings (MeSH) terms and free-text terms informed by previously published literature. We searched MEDLINE (PubMed), Google scholar and the Epistemonikos database for articles from January 2020 to March 2025. Our full search strategy for Medline were as follows: ((("COVID-19"(MeSH Terms) OR "COVID-19"(Title/Abstract)) AND ("Pregnancy"(MeSH Terms) OR "Pregnancy"(Title/Abstract)) AND ("c reactive protein"(MeSH Terms) OR ("d dimer"(Title/Abstract) OR "cytokines"(MeSH Terms) OR "alpha 1 acid glycoprotein" (Title/Abstract) OR "hba1c" (Title/Abstract)) OR ("insulin growth factors" (Title/Abstract) OR "angiotensin converting enzyme 2"(MeSH Terms) OR "tmprss2"(Title/Abstract) OR "minerals"(MeSH Terms) OR "vitamin a"(MeSH Terms)))) AND (case reports(Filter) OR classical article(Filter) OR clinical conference(Filter) OR clinical study(Filter) OR comparative study(Filter) OR government publication(Filter) OR meta-analysis(Filter) OR observational study(Filter) OR systematic review(Filter)). The final searches from January 2020 to March 2025, exported the search results into EndNote and removed all duplicates.

Identifying and selecting relevant studies

Duplicated titles between the all-selected databases were removed. To ensure high inter-rater reliability, a training exercise was conducted before the title and abstract screening process began. Using our predefined eligibility criteria, all authors involved in the titles and abstract screening read the titles and abstracts independently and removed those irrelevant articles, leaving 34 articles for full-text review.

Any titles/abstracts that the authors disagreed on were reread by both first and second authors with the feedback of the other author in mind and resolved by discussion or a third adjudicator.

The next step was to read the full-text articles, focusing primarily on the methods, results and discussion sections. The second, third and fourth authors evaluated the articles separately, which resulted in 27 articles to include in the review. The same details are described in PRISMA (SCr) flow chart (Figure 1).

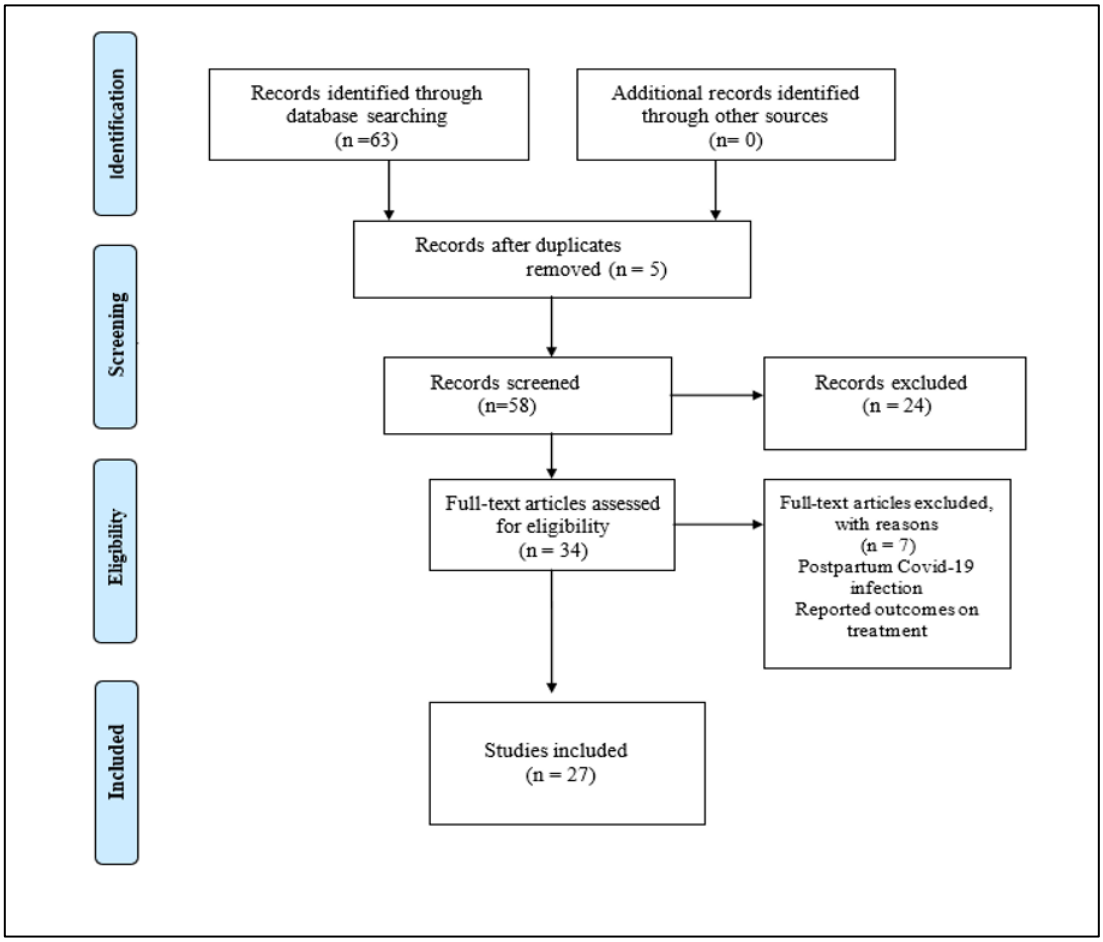

Figure 1: PRISMA flow diagram for the scoping review process.

Table 1: Characteristics of included studies (n=27).

First author	Study design	Sample assessed	Sample size (babies)	Sample size (mother)
Morotti et al (2024)[8]	Case report	In-situ study of expression and gene expression of placentas	4	3
Siddiq et al (2024)[18]	Cross-sectional	Not mentioned	None	278
Tóth et al (2024)[9]	Case report	Venous blood and placental tissue for histochemistry	Intrauterine fetal demise	1
Fernandes et al (2023)[10]	Prospective cross-sectional	Whole blood sample	None	141(89 COVID-19-infected pregnant women and 52 noninfected)
Narayanaswamy et al (2021)[11]	Observational	Colostrum on the day or day after delivery	None	15* (pre-nataly infected) and 8 (not infected).
Lebrão et al (2020)[38]	Case report	Mother's breast milk	1	1
Moriarty et al (2022)[35]	Case report	Placental immunohistochemistry and histopathological analysis	1 twin pregnancy with the death of a twin after birth	1
Babal et al (2021)[37]	Case report	Placenta and fetal lung tissue	Intrauterine fetal death	1
Eman et al (2021)[12]	Retrospective observational	Mother's blood sample	18	19
Bakhle et al (2021)[26]	Case report	Fetal blood sample	1	1

Continued.

First author	Study design	Sample assessed	Sample size (babies)	Sample size (mother)
Shmakov et al (2022)[24]	Prospective observational	Mother's blood examination, COVID testing (NP swabs, vaginal swabs, amniotic fluid, placenta, colostrum)	42	66
Babai et al (2024)[29]	Cross sectional	Blood work and serology of mother	None	135
Atak et al (2022)[16]	Retrospective cohort	Blood examination of mother	None	26 prenatal COVID 19 infected pregnant or immediately postpartum women
Edlow et al (2020)[25]	Longitudinal	Mother's blood examination, infant's blood and cord sample	Cord blood: 32 (exposed), 7 (non-exposed) collected at delivery blood specimens: 45 (exposed), 14 (non-exposed), collected at day 1 of life	93 infected pregnant women v/s 18 non-infected pregnant women
Edlow et al (2020)[25]	Cohort	Maternal plasma, maternal nasal and oropharyngeal swabs, maternal saliva, maternal sputum, placenta pathology specimen and neonate nasal, oropharyngeal swabs and blood were used.	47mother-neonate dyads with SARS-COV-2 infection	127 enrolled pregnant women: - 64 infected and 63 non-infected
Faure-Bardon et al (2021)[34]	Case series	Fetal organ and placental tissue samples	6 samples of non-COVID-19 exposed fetus and 1 sample of in utero COVID-19 exposed fetus	None
Chen et al (2020)[4]	Retrospective observational	Blood examination	None	For markers: 20 infected pregnant women and 159 infected non-pregnant women. For immune responses: 4 infected pregnant and 6 infected non-pregnant women. While 4 pregnant healthy controls and 3 non-pregnant healthy controls
Ozer et al (2021)[23]	Retrospective cross-sectional	Laboratory examination through blood samples	None	15 infected pregnant women and 19 infected non-pregnant women
Cui et al (2021)[36]	Observational	Placenta samples in all three trimesters	None	9
Raoufi et al (2024)[22]	Retrospective cross-sectional	Laboratory examination	None	66 infected pregnant women and 107 infected non-pregnant women
Gong et al (2020)[20]	Retrospective observational	Blood examination	None	10
Liu et al (2024)[15]	Retrospective cohort	Nasopharyngeal swab specimens	None	90 infected pregnant women, 278 infected non-pregnant women
Tjahyadi et al (2023)[33]	Observational retrospective	Laboratory examination	None	31 infected pregnant women with preeclampsia and 113 infected pregnant women without preeclampsia.

Continued.

First author	Study design	Sample assessed	Sample size (babies)	Sample size (mother)
Mao et al (2020)[32]	Case report	Placenta sample	Stillborn fetus	1
Mohr-Sasson et al (2020)[21]	Retrospective cohort	Laboratory examination	None	11 infected pregnant women and 25 infected non-pregnant women
Kinsey et al (2020)[31]	Case report	Bloodwork	None	1
Craina et al (2022)[19]	Case series	Peripheral bloodwork, np, op, rectal and vaginal swab of mother; np of neonate	3	3

*(11 of the 15 participants tested positive for COVID-19 near the time of delivery, and the participants had their most recent positive test 16 to 116 days before delivery).

Data charting

The Google Excel data extraction sheet was developed a priori and pilot-tested by the primary author on the three randomly selected included articles. Through an iterative process, the data extraction form was revised to include information specific to study title, first author name and publication year, type of article or study, study period, study location/settings, study population characteristics (eg: In utero COVID 19 exposed Babies/ prenatal COVID 19 exposed women), sample size, study assessment duration, reported outcomes especially marker characteristics, pathophysiological characteristics and other findings.

Synthesis of results

The synthesis of results has been focused on narrative synthesis, describing biochemical alterations during or after pregnancy in mothers with a history of COVID-19 and in utero-exposed children. This is achieved by an iterative process of summarizing the literature according to data charting. A wide range of biomarkers was found, which were assessed in the selected studies to understand maternal and neonatal outcomes following prenatal exposure to COVID-19. The detailed characteristics of the included studies are described in Table 1.

DISCUSSION

The findings are discussed thematically across five main categories, including inflammatory and cytokine markers, coagulation markers, haematological markers, viral entry and receptor markers, and immunoglobulins associated with COVID-19.

Inflammatory and cytokine markers

Interleukins

A pattern of elevated pro-inflammatory markers was evident in multiple studies. Proinflammatory interleukins mainly IL-1, IL-2, IL-6, IL-8, IL-12, IL-15, IL-17, and TNF are mainly involved in the acute-phase response of infection.[7] Elevated level of IL-6 was the most consistent finding among all the studies.[8-13] IL-1 and TNF-α were reported less frequently but when measured were often higher in symptomatic/severe cases compared with milder disease. High levels of IL-18, IL-1b, was seen in term COVID-19-exposed infants who developed Respiratory distress.[14] One of the studies observed elevated levels of leptin, a distinctive interleukin, possibly reflecting a physiological adaptation in pregnancy aimed at providing antiviral protection in the mother.[15] The pro-inflammatory microenvironment is associated with the risk of adverse pregnancy outcomes, including pregnancy loss, preeclampsia, and gestational diabetes mellitus.[10-16] In contrast, to the proinflammatory cytokines, anti-inflammatory interleukins, such IL-4, IL-5, IL-9, IL-10, IL-13 are characterized by their ability to suppress pro-inflammatory signalling pathways and dampen immune responses.[7] IL-10 was reported in a minority of studies and showed variable behaviour in some IL-10 rose alongside IL-6 suggesting compensatory regulation, while in others IL-10 changes were inconsistent.[8-10] One of the studies found elevation in a distinct cytokine-IL-1RA, an anti-inflammatory cytokine that blocks IL-1α and IL-1β functions and modulates their biological effects.[10] IL-4 and IL-5 were rarely measured and when reported showed limited or variable changes.[10,11]

Chemokines

The chemokines are proteins that play a vital role in the homeostasis of the immune system, involving in all protective or destructive immune and inflammatory responses. These mainly stimulate the migration of cells, especially white blood cells.[17] Alterations in chemokine expression were described in study, with increased levels of CXCL8, CCL11, CCL2, and CCL3 in COVID-19 exposed pregnant women, while levels of CCL4, CCL5, and CXCL10 were comparatively lower during the later trimesters as compared to the non-infected pregnant women.[10]

Interferons

These are signalling proteins secreted from infected cells which activate innate immune response that promotes cytokine production, natural killer cell functions and antigen presentation. A study detected notable increase in IFN-γ levels in COVID-19–exposed pregnant women.[10] Additionally, in severe or critical COVID-19–positive pregnancies, elevated levels of IFN-λ and its receptor

IFNLR1 were detected, potentially explaining the relatively rapid viral clearance observed in these cases and their possible role in preventing vertical transmission of SARS-CoV-2. The marker IFN played a crucial role in preventing vertical transmission of Zika virus (ZIKV) during prenatal infection.[14]

C-reactive protein

C-Reactive Protein is an acute phase reactant, whose level rises in response to inflammation and infection. CRP levels were elevated in COVID-19 pregnant patients in several studies and correlated with disease severity or critical illness in some cohorts.[12,18-20] C-reactive protein was higher in the pregnant patients compared to the non-pregnant patients indicating pregnancy might contribute to the elevated levels.[13] However, other studies found no significant difference in the levels of CRP between the two groups.[21-23] Findings across studies indicate that CRP is a reliable predictor of disease severity in COVID-19.[24-25] Furthermore, CRP was also elevated in the foetal blood sample. Elevated levels had pulmonary thromboembolic phenomenon as a consequence of multisystem inflammatory syndrome in neonate resulting in the rare CT picture of nodular and cavitary lung lesions.[26]

Procalcitonin

An acute phase reactant synthesized by thyroid C cells for the early detection of infections specifically systemic bacterial infections. It helps to differentiate bacterial vs Viral infections.[27] Procalcitonin increases were uncommon and when present were often used to evaluate bacterial co-infection.[12-22] One of the studies found elevated procalcitonin marker in the foetal blood sample indicating infection in the COVID 19 exposed neonate.[26]

Lactate dehydrogenase

A non-specific tissue injury marker released into the blood when cells are damaged.[28] Increase levels were reported in a small number of studies.[19,24] Few studies also reported elevated levels of LDH in pregnant women who died as a consequence of prenatal exposure COVID-19 infection.[16,29]

Coagulation markers

The coagulation system is a host defence response against injury, bleeding, and the invasion of infectious agents in the body. The most important factors involved in coagulation are platelets, endothelial cells, and coagulation factors. Infections cause alterations in the markers of the coagulation system.[30]

D-dimer

D-Dimer is produced when plasmin degrades cross-linked fibrin. It is a marker of both an active coagulation process as well as fibrinolysis.[30] The repeated observation of increased D-dimer supports activation of a typical acute-phase inflammatory response in a subset of infected pregnant patients followed with activation of the coagulation system.[9,12,19,20,24,29] In one case report, intraoperative coagulopathy was observed at the time of delivery, associated with markedly increased D-dimer concentrations.[31] Additionally, it is important to emphasize that pregnancy is a hypercoagulable state. Hence, COVID in addition to pregnancy can cause marked alteration in the levels of D-Dimer as compared to non-pregnant women with COVID19.[13,22] In contrast, one study found a distinct result- The mean D-Dimer values were lower in pregnant women as compared to non-pregnant women. This was statistically significant difference. The study explains that the lower D-dimer values among pregnant women may explain their lower mortality and morbidity rates.[23] The levels of D-Dimer were also elevated in COVID 19 foetus consequently leading to multisystem inflammatory syndrome in neonate.[26]

Other than D-Dimer, alterations in other markers were less reported in these studies. A markedly decreased fibrinogen levels was detected in one study.[9] However, it was elevated in other study.[19] Prothrombin time was shorter PT in pregnant patients as compared to non-pregnant patients with COVID19.[13] Another study reported the mortality rate in pregnant women with COVID 19 increased with decreasing Hb and increasing PT.[29]

Platelets are also an important part of the coagulation cascade. Studies have shown that among COVID-19 patients, severe hematologic changes lead to thrombocytopenia.[30] Few studies also found decreased platelet count in pregnant women exposed with COVID 19.[22,23]

Haematological markers

The main haematological changes reported in the studies were white blood cells.[12] And to some extent ferritin levels. Under infectious disease conditions, an increase in observed the number of WBCs, which are important biomarkers in suspecting various types of infections.[30] A study by Li and Tan showed that the level of total leukocytes increased in severe patients with COVID-19 while the level of lymphocytes decreased.[32] Similar pattern was detected in the studies included in our review.[13,16,19-21,29] An important biomarker of systemic inflammatory response-neutrophil-to-lymphocyte ratio (NLR) was identified in few studies. High neutrophil-to-lymphocyte ratio count were found in pregnant women with COVID-19 correlating with poor outcomes in patients.[22,29,33] Moreover, Ferritin an indirect marker of the total amount of iron stored in the body which rises in the course of infection.[30] It was found elevated in pregnant patient exposed to covid 19.[12,16,19] as well as covid 19 exposed foetus.[26]

Viral entry and receptor markers

The SARS-CoV-2 virus enters into target cells by the presence of angiotensin-converting enzyme 2 (ACE2) on the membrane of the host cell. The Transmembrane cellular protease serine 2 (TMPRSS2) primes the viral spike protein enabling membrane fusion and viral entry.[34] These markers were mainly expressed and analysed in placental samples of COVID 19 infected pregnant women.[8] Immunohistochemistry localized ACE2 mainly in syncytiotrophoblasts and decidual cells, with minimal staining in necrotic trophoblasts or endothelial cells.[35] The co-expression of ACE2 and TMPRSS2 was linked to viral susceptibility and oxidative stress.[36] However, the expression ACE2 and TMPRSS2 suggested a potential protective mechanism against vertical transmission of the infection.[25]

Some studies reported low ACE2 and TMPRSS2 but high CD147 expression, indicating an alternative viral entry pathway.[37] Circulating ACE2 levels did not differ significantly between COVID-19–exposed and healthy pregnant women, although symptomatic cases showed higher levels compared to asymptomatic ones.[9,34,14]

Immunoglobulins associated with COVID-19

Colostrum and breast milk samples from COVID-19–positive mothers demonstrated strong reactivity to the SARS-CoV-2 Receptor-binding domain (RBD), with detection IgA and IgG suggesting a potential protective effect against infection and disease severity in infants.[11,38]

One study reported detectable IgM in COVID 19 exposed neonates exhibited, likely due to placental damage allowing transplacental passage of larger antibodies. However, transfer of protective anti-RBD IgG and anti-N IgG antibodies was significantly reduced, indicating limited passive immunity in newborns.[25] Additionally, pregnant women showed elevated SARS-CoV-2 S-RBD IgG and N protein IgG antibody levels compared to non-pregnant women.[15]

Other markers

Several other nonspecific markers were identified in different studies. Elevated growth factors, altered creatinine and urea levels indicating renal status during the infection, hepatic markers such Aspartate aminotransferase, Alanine aminotransferase, Bilirubin were identified to be elevated in a few studies. Higher ALT levels predicted the severity of the disease.[24,25]

Vertical transmission

Several studies reported no transmission of virus consistent with the existing to the literature available.[9,12,24,39] This protective effect may be due to specific markers such as ACE2 and TMPRSS2, which limit placental infection and tissue damage.[25] Nonetheless, evidence suggests that the possibility of vertical transmission cannot be entirely excluded.[40] intrauterine infection or breastfeeding or transplacental infection routes remain possible.[24,25]

CONCLUSION

In conclusion, prenatal COVID-19 infection is associated with a wide spectrum of biochemical alterations in mothers and neonates. Consistent patterns included elevated inflammatory cytokines, altered coagulation profiles and haematological markers, and variable expression of viral entry receptors in placental tissues. However, most literature outcomes predominantly focused on the infection period, limiting studying of long-term consequences of COVID 19 infection during pregnancy. Overall, the findings emphasize underscore the need for longitudinal biochemical surveillance across the peripartum period.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Memon FZ, Parekh L, Mehta V, Patel E, Memon F. Maternal and neonatal biochemical sequelae following prenatal COVID 19 infection: evidence across the peripartum period. Int J Community Med Public Health 2026;13:4022-30.