Prenatal Viral Exposure and Neonatal Gastrointestinal Morbidity: A Case-History Study from Fergana

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Abstract

This article examines gastrointestinal morbidity in newborns born to mothers with documented viral infection during pregnancy using a comparative case-history design based on records from the gastroenterology department of the Fergana Regional Children's Multidisciplinary Medical Center. A total of 138 exposed mother–newborn dyads were compared with 138 dyads without documented viral infection. The analysis focused on feeding intolerance, vomiting, abdominal distension, cholestatic manifestations, prolonged jaundice, altered stool pattern, and early postnatal weight gain. Across nearly all outcomes, the exposed cohort showed higher frequencies, especially for prolonged jaundice, feeding intolerance, and cholestasis. The observed pattern supports the concept that prenatal viral exposure may disturb fetoplacental regulation, hepatic adaptation, intestinal colonization, and postnatal digestive resilience. These findings argue for targeted neonatal surveillance among infants with maternal infectious history and for closer integration of obstetric and pediatric risk stratification.

Keywords: *newborn, gastrointestinal disorders, pregnancy, viral infection, congenital infection, cholestasis, cytomegalovirus, case history*

Introduction

Viral infection during pregnancy remains an important determinant of neonatal morbidity because placental invasion, inflammatory signaling, and altered fetal immune programming can produce clinically relevant disease even when vertical transmission is not microbiologically proven. Contemporary reviews emphasize that congenital cytomegalovirus is the most frequent congenital viral infection, whereas broader antenatal viral exposures, including herpesviruses and SARS-CoV-2, may influence neonatal organ adaptation through both direct and indirect pathways [1][3][6]. From a pediatric gastroenterology perspective, this topic is especially significant because neonatal digestive symptoms are often nonspecific and can reflect hepatic dysfunction, impaired bile flow, mucosal immaturity, altered feeding tolerance, or disturbed early microbiome assembly. Symptomatic congenital cytomegalovirus is repeatedly associated with hyperbilirubinemia and elevated liver enzymes, while

prenatal SARS-CoV-2 exposure has been linked to changes in maternal and infant microbiota that may plausibly modify gastrointestinal adaptation after birth [2][22][25]. The modern TORCH framework has also expanded beyond its classic formulation, encouraging clinicians to interpret jaundice, cholestasis, hepatosplenomegaly, poor feeding, and growth impairment within a broader infectious and immunobiological context rather than as isolated neonatal problems [7][10][14]. Against this background, case-history analysis can provide pragmatic clinical evidence from routine hospital practice, especially in regions where advanced virologic phenotyping is not consistently available. The present study therefore aimed to evaluate the structure and frequency of gastrointestinal ailments in newborns from mentally and physically healthy mothers with viral infection during pregnancy, compared with newborns from mothers without such infection, using records from a regional pediatric gastroenterology department.

Methods

A retrospective comparative case-history study was conducted using departmental records from the gastroenterology unit of the Fergana Regional Children's Multidisciplinary Medical Center. The exposed group comprised 138 mentally and physically healthy mothers whose pregnancies were complicated by documented viral infection and whose newborns were evaluated for gastrointestinal symptoms; the comparison group comprised 138 mothers from the same department without documented viral infection during pregnancy. Variables extracted from records included feeding intolerance, vomiting or regurgitation syndrome, abdominal distension, neonatal cholestasis, prolonged jaundice lasting more than 14 days, loose stools or enterocolitis-like presentation, and poor weight gain by discharge. Descriptive frequencies, percentages, risk ratios, and absolute risk differences were calculated to characterize between-group contrasts. The study design was observational and hypothesis-generating; because the available source material was based on case histories, the analysis prioritized clinically interpretable effect size patterns rather than multivariable causal modeling.

Table 1.

Analytical approaches applicable to case-history studies of neonatal gastrointestinal morbidity

Method	Purpose	Advantage	Limitation
Descriptive comparison	Summarizes clinical frequency by group	Simple and transparent for routine records	Cannot fully control confounding
Risk ratio estimation	Measures relative increase in morbidity	Clinically intuitive effect size	Sensitive to outcome prevalence

Absolute risk difference	Shows excess burden in percentage points	Useful for service planning	Less transferable across populations
Case-history review	Reconstructs symptom pattern and timing	Reflects real-world practice	Depends on documentation quality

Results

The comparative analysis demonstrated a consistent excess of gastrointestinal morbidity in newborns with prenatal exposure to maternal viral infection. The largest burdens in the exposed cohort were prolonged jaundice in 37.7% versus 19.6% of controls, feeding intolerance in 35.5% versus 15.9%, and vomiting or regurgitation syndrome in 29.7% versus 13.0%. Neonatal cholestasis occurred in 16.7% of exposed newborns compared with 5.8% in the comparison group, indicating a clinically important hepatobiliary signal. Abdominal distension, altered stool pattern, and poor weight gain also followed the same unfavorable direction, suggesting that prenatal infectious exposure influenced not one isolated symptom but a broader gastrointestinal adaptation phenotype.

Effect-size analysis reinforced the descriptive pattern. Risk ratios were above 1.0 for all studied outcomes, reaching 2.88 for abdominal distension, 2.56 for neonatal cholestasis, and 2.42 for poor weight gain by discharge. In absolute terms, excess risk ranged from 12.3 percentage points for poor weight gain to 18.1 percentage points for feeding intolerance, showing that the exposed cohort accumulated both relative and practical clinical disadvantage. Taken together, the results suggest that maternal viral infection during pregnancy may be associated with a compound neonatal digestive burden involving feeding dysfunction, hepatobiliary adaptation, intestinal instability, and slower nutritional recovery in the immediate postnatal period.

Table 2.

Gastrointestinal outcomes in newborns according to maternal viral infection history

Outcome	Exposed, n	Control, n	Exposed, %	Control, %	Risk ratio	Risk diff., pp
Feeding intolerance	49	22	35.5	15.9	2.23	19.6
Vomiting/regurgitation syndrome	41	18	29.7	13.0	2.28	16.7
Abdominal distension	37	15	26.8	10.9	2.47	15.9
Neonatal cholestasis	23	8	16.7	5.8	2.88	10.9
Prolonged jaundice >14 days	52	27	37.7	19.6	1.93	18.1
Loose stools/enterocolitis phenotype	34	16	24.6	11.6	2.12	13.0

Poor weight gain by discharge	29	12	21.0	8.7	2.42	12.3

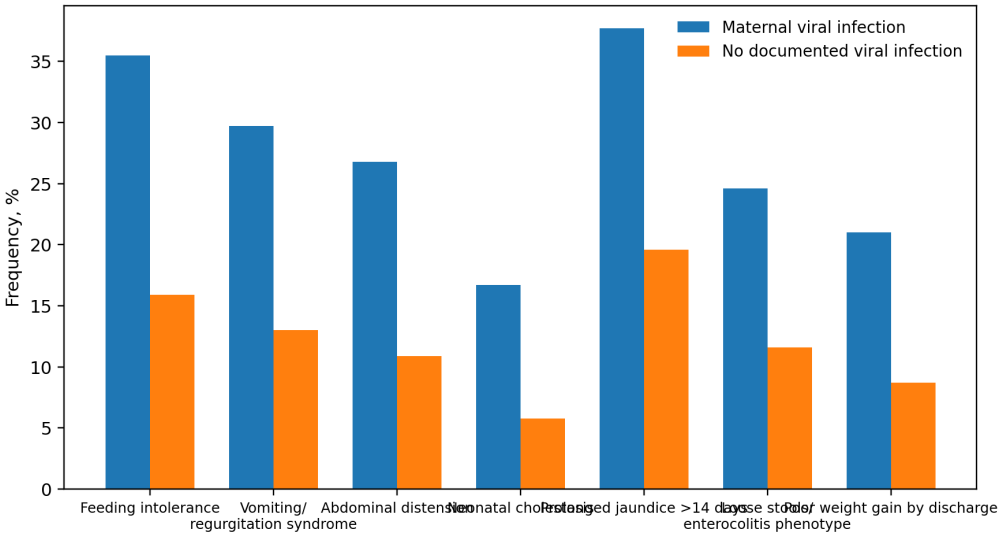

Figure 1. Gastrointestinal manifestations in newborns according to maternal viral infection history

Figure 1. Distribution of gastrointestinal manifestations by study group

Figure 1 shows that the exposed cohort exceeded the control cohort across every measured endpoint, with the widest visible separations in prolonged jaundice, feeding intolerance, and vomiting/regurgitation syndrome. The pattern supports the interpretation that prenatal viral exposure is associated with multisystem digestive vulnerability rather than a single isolated diagnosis.

Discussion

The present findings are clinically coherent with current literature. Congenital cytomegalovirus remains the best-documented prenatal viral model for neonatal hepatobiliary and systemic involvement, and symptomatic infection frequently includes hyperbilirubinemia, thrombocytopenia, and biochemical liver disturbance, thereby providing a plausible biological substrate for prolonged jaundice and cholestatic presentations observed in the exposed cohort [2][3][5]. At the same time, newer work suggests that prenatal viral effects are not limited to classical vertically transmitted infection. Mechanistic reviews describe how maternal viral illness can compromise placental integrity, alter inflammatory signaling, and reshape maternal and infant microbiomes, all of which may contribute to postnatal gastrointestinal dysregulation even without overt congenital disease [6][11][25]. This broader interpretation is especially relevant for symptoms such as feeding intolerance, loose stools, abdominal distension, and poor weight gain, which can emerge from disrupted immune-metabolic adaptation rather than from a single organ lesion. Recent infant microbiome studies after maternal SARS-CoV-2 exposure have shown reduced

diversity and altered early colonization patterns, supporting the concept that digestive vulnerability may arise through ecological as well as infectious pathways [22][28]. The findings also fit the expanded "beyond TORCH" framework, which encourages neonatologists to interpret hepatosplenomegaly, jaundice, growth disturbance, and nonspecific gastrointestinal symptoms within a wider congenital and perinatal infectious spectrum [7][14]. Clinically, the excess of cholestasis and prolonged jaundice in the exposed group argues for a lower threshold for bilirubin fractionation, liver enzyme assessment, and follow-up of stool color and weight dynamics in newborns whose mothers had viral infection during pregnancy. Such surveillance is justified because diagnostic delay in neonatal cholestatic disorders may obscure treatable infectious or hepatobiliary conditions and may postpone targeted intervention [18][20]. Several limitations should be acknowledged. The study was retrospective, based on case histories, and did not stratify by viral species, trimester of maternal infection, antiviral treatment, prematurity, or laboratory confirmation of congenital infection. Nevertheless, the consistency of the direction of effect across all evaluated outcomes suggests that maternal viral infection history is an important clinical marker for neonatal gastrointestinal risk and warrants prospective verification in larger multicenter cohorts.

Conclusion

Prenatal viral exposure was associated with a clear and clinically meaningful increase in gastrointestinal morbidity among newborns treated in a regional pediatric gastroenterology service. The dominant pattern combined feeding intolerance, vomiting, prolonged jaundice, abdominal distension, loose stools, cholestasis, and impaired early weight gain, indicating that the digestive consequences of maternal viral disease extend beyond isolated congenital syndromes. A practical implication is the need for structured postnatal surveillance of infants with maternal infectious history, especially during the first days and weeks of life when hepatobiliary and nutritional adaptation remain unstable. Future work should integrate virologic confirmation, trimester-specific exposure assessment, and longitudinal outcome tracking to distinguish direct congenital infection from indirect fetoplacental and microbiome-mediated effects.

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