Neurologic Findings and Cerebrospinal Fluid Biomarkers in Neonates With SARS-CoV-2 Infection

A Multicenter National Cohort Study

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Background: Neurologic manifestations of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection in neonates are poorly characterized, and central nervous system (CNS) involvement may be difficult to recognize due to nonspecific symptoms. The role of cerebrospinal fluid (CSF) biomarkers in detecting CNS inflammation or neuronal injury in this population remains unclear. We aimed to characterize neurologic manifestations and evaluate CSF findings in affected neonates.

Methods: In this prospective multicenter national cohort study conducted in Spain (October 2020–July 2022), neonates 0–28 days old with confirmed SARS-CoV-2 infection and fever and/or neurologic symptoms were included. Neurologic evaluation, CSF analysis, and neuroimaging were performed when clinically indicated. CSF biomarkers (β2-microglobulin [B2M] and neuron-specific enolase [NSE]) were analyzed and compared with age-matched controls.

Results: Sixty-one neonates were included (median age 16 days). Neurologic symptoms were observed in 27 (44.3%), mainly irritability, lethargy, tone alteration or apnea, and were transient in all cases. Lumbar puncture was performed in 20 neonates; no microbiologically confirmed meningitis or CSF SARS-CoV-2 detection was identified. CSF B2M levels were significantly higher in infected neonates compared with age-matched controls, whereas NSE levels were similar. No severe neurologic complications or deaths occurred.

Conclusion: Neonatal SARS-CoV-2 infection was associated with mild and transient neurologic symptoms without evidence of meningoencephalitis. Elevated CSF B2M with normal NSE suggests subclinical CNS immune activation without neuronal injury.

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Since the emergence of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), neurologic involvement has been increasingly recognized in coronavirus disease 2019 (COVID-19). In children, manifestations range from mild symptoms to severe complications such as encephalopathy and seizures, particularly in multisystem inflammatory syndrome.[1]

Neurologic manifestations in neonates remain poorly characterized. Clinical presentation is often nonspecific, including feeding difficulties, hypotonia, apnea or irritability, complicating recognition of central nervous system (CNS) involvement. Most evidence derives from isolated case reports describing seizures, encephalitis or ischemic lesions, frequently without structured neurologic evaluation. Recent reviews highlight important gaps in epidemiology, clinical characterization and long-term outcomes, while routine cerebrospinal fluid (CSF) findings are usually normal in reported neonatal cases.[2–4] In addition, concerns have been raised regarding possible neurodevelopmental consequences following neonatal infection or *in utero* exposure to SARS-CoV-2, although current evidence suggests that severe or persistent deficits are uncommon.[5]

In neonates presenting with fever and/or neurologic symptoms, lumbar puncture is commonly considered as part of the diagnostic evaluation to exclude potentially treatable conditions such as bacterial meningitis or meningoencephalitis, in which early diagnosis and timely initiation of therapy are critical to prevent neurologic sequelae. However, beyond conventional cytologic and biochemical analysis, the role of CSF biomarkers in detecting subclinical CNS involvement in neonates with SARS-CoV-2 infection remains largely unexplored.[4]

CSF biomarkers may help identify subclinical CNS involvement when clinical findings are nonspecific. Beta-2 microglobulin (B2M) reflects intrathecal immune activation and has been used as a marker of CNS inflammation in neonatal brain disorders.[6–8] Neuron-specific enolase (NSE), a marker of neuronal injury, may complement inflammatory biomarkers by distinguishing immune activation from structural damage.[9]

To our knowledge, no multicenter studies have systematically evaluated neurologic manifestations and CSF inflammatory biomarkers in neonates with SARS-CoV-2 infection using age-matched controls. This study aimed to characterize neurologic manifestations and assess CNS involvement through conventional CSF analysis and relevant biomarkers.

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METHODS

Study Design and Population

This multicenter observational cohort study was conducted within the Spanish Neonatal Neuro-COVID Group between October 2020 and July 2022. The study was conducted across level 2 and 3 neonatal units in Spain, with 20 hospitals contributing eligible patients during the study period.

Inclusion criteria were (1) neonates 0–28 days of age; (2) confirmed SARS-CoV-2 infection (2 positive reverse transcription polymerase chain reaction [RT-PCR] tests in nasopharyngeal swabs); and (3) fever and/or symptoms potentially suggestive of neurologic involvement (lethargy, irritability, altered consciousness, abnormal muscle tone, apnea, seizures). Exclusion criteria were asymptomatic neonates, absence of consent or pre-existing neurologic disease.

The study was approved by the Ethics Committee (CEIC 2347), and written informed consent was obtained.

Neurologic Assessment

All neonates underwent structured neurologic evaluation at presentation and during hospitalization. Neurologic symptoms were systematically recorded, including lethargy, irritability, abnormal muscle tone, apnea, seizures or other abnormal neurologic signs. When symptoms were persistent or clinically significant, neuromonitoring with amplitude-integrated electroencephalography and neuroimaging was recommended. Cranial ultrasound served as first-line imaging; conventional electroencephalography and brain magnetic resonance imaging were recommended for seizures or worrisome progression.

Lumbar puncture was recommended by the study protocol in neonates presenting with fever and/or neurologic symptoms to exclude potentially treatable CNS infections such as bacterial meningitis. Routine CSF analysis included cell count, protein and glucose. Interpretation of CSF cytochemical parameters was performed using age-adjusted neonatal reference values. Normal CSF values were defined according to published neonatal reference ranges: white blood cell <10 cells/μL (term) and <12 cells/μL (preterm); protein <110 mg/dL (term) and <210 mg/dL (preterm); glucose (reported mean values) 51.2 mg/dL (term) and 51.6 mg/dL (preterm).[10] Infectious workup included bacterial culture and molecular testing for herpes simplex virus, cytomegalovirus, enterovirus and parechovirus. Confirmed meningitis was defined by microbiologic identification of a pathogen in CSF by culture or PCR. Suspected meningitis was defined by a compatible clinical presentation and abnormal CSF parameters in the absence of microbiologic confirmation.

Two CSF aliquots were stored at −80°C for SARS-CoV-2 PCR and biomarker analysis (NSE and B2M) at the coordinating laboratory. These biomarkers were selected to explore immune activation (B2M) and neuronal injury (NSE) within the CNS. Sample handling, storage conditions, and analytical procedures followed previously validated methods.[11] The CSF SARS-CoV-2 PCR was done using the Xpert Xpress SARS-COV-2 test (Cepheid, Sunnyvale, CA) according to the manufacturer's instructions. Biomarker analyses were performed blinded to clinical data.

An age-matched control population was used for biomarker comparison. Controls were drawn from a prospective neonatal neurobiomarker cohort and included neonates evaluated for suspected infection due to fever or feeding difficulties. All were clinically stable, without signs of severe infection or multiorgan dysfunction, and did not require respiratory or hemodynamic support. CNS infection was ruled out based on normal CSF parameters and negative microbiologic studies. None had neurologic symptoms, and all had an uneventful clinical course.

Systemic Assessment

Systemic clinical manifestations—respiratory, gastrointestinal, cutaneous and others—were assessed in all neonates. The need for respiratory support, hemodynamic instability and admission to the intensive care unit were also evaluated. Additional imaging studies—echocardiography, lung and abdominal ultrasound—were performed according to clinical indications. Blood samples were collected at admission and at 48 hours, 7, 14 and 21 days if the neonate remained hospitalized for complete blood count, inflammatory markers (C-reactive protein, procalcitonin, interleukin (IL)-6, ferritin), hepatic function and coagulation parameters using age-adjusted reference ranges. Age-adjusted neonatal reference ranges were used to define normal values as follows: absolute neutrophils $>1.5 \times 10^3$/μL; absolute lymphocytes $>2 \times 10^3$/μL; neutrophil-to-lymphocyte ratio (NLR) 0.10 – 0.91; platelets-to-lymphocyte ratio (PLR) 29–115; prothrombin time 10.1–16 s; prothrombin activity >40%; activated partial thromboplastin time <54.3 s; fibrinogen 150–300 mg/dL; D-dimer <2500 ng/mL; aspartate aminotransferase <60 IU/L; alanine aminotransferase <50 IU/L; gamma-glutamyl transferase <50 IU/L; creatine phosphokinase 56–68 IU/L; C-reactive protein <20 mg/dL; procalcitonin <0.5 ng/mL; IL-6 <12 pg/mL; ferritin 25–200 ng/mL. Blood and urine cultures were obtained at admission as part of the initial infectious workup.

Statistical Analysis

Continuous variables are presented as median and interquartile range (IQR). Categorical variables are expressed as counts and percentages. Biomarker comparisons between groups were performed using the Mann–Whitney *U* test. A *P* value <0.05 was considered statistically significant. Statistical analyses were performed using SPSS version 26. Figures were generated using GraphPad Prism version 8.0.1 and BioRender.

RESULTS

Study Population

Sixty-one neonates were included (36 males, 25 females). Median age at presentation was 16 days (IQR 11.5–20). Clinical evaluation was prompted by fever (n = 34), neurologic symptoms (n = 7) or both (n = 20). Fifty-two neonates were admitted, while 9 were evaluated and discharged without admission.

Obstetric and perinatal characteristics are summarized in Table 1 and were largely unremarkable.

Neurologic Manifestations

Neurologic symptoms were observed in 27 neonates (44.3%), most of which were mild and transient, including irritability, lethargy, tone alteration and apnea (Fig. 1). No cases of severe encephalopathy or persistent altered consciousness were identified.

One neonate had suspected seizures at home; however, during hospitalization, no further events occurred, and neurologic investigations (amplitude-integrated electroencephalography, electroencephalography, cranial ultrasound and CSF analysis) were normal. In the remaining neonates, neurologic symptoms were transient and clinically mild, and none were considered clinically significant. All neonates had normal neurologic examinations at discharge.

Neuromonitoring was performed in 2 neonates and showed normal findings. Cranial ultrasound was performed in 5 neonates and was normal in all cases. Brain magnetic resonance imaging was not considered clinically indicated in any patient, given the mild and transient neurologic manifestations.

TABLE 1. Maternal, Perinatal and Neonatal Characteristics of the Study Cohort

Variable	N	Median (IQR) or n (%)
Maternal characteristics		
Maternal age, yr	55	33 (27–36)
Gravidity	57	2 (1-3)
Adequate prenatal care	61	60 (98.4)
Preeclampsia	59	0 (0)
Gestational diabetes	58	6 (10.3)
Placental abruption	59	4 (6.8)
Multiple gestation	61	2 (3.3)
Perinatal characteristics		
Gestational age, wk	61	39.00 (38.5–40.0)
Cephalic presentation	61	59 (96.7)
Abnormal cardiotocography	59	1 (1.7)
Vaginal delivery	61	53 (86.9)
Advanced neonatal resuscitation	2	2 (100)
Birth weight (g)	61	3300 (3035–3512)
Head circumference (cm)	52	34.5 (34.0–35.5)
Apgar score at 1 min	57	9 (9;9)
Apgar score at 5 min	57	10 (10;10)
Umbilical cord pH	42	7.26 (7.20–7.32)
Meconium-stained amniotic fluid	61	6 (9.8)
Chorioamnionitis	61	1 (1.6)
Rupture of membranes >18 h	53	5 (9.4)
Neonatal period		
Breastfeeding	61	46 (75.4)
Household SARS-CoV-2 contact at diagnosis	61	46 (75.4)

Lumbar puncture was performed in 20 neonates (32.8%). CSF leukocytes were elevated in 3 of 19 neonates (15.8 % with 41, 17 and 11 cells/μL). The corresponding CSF red blood cell counts were 4200, 0 and 1350 cells/μL, respectively. The median CSF protein concentration was 59 mg/dL (IQR 47.0–76.9), with no neonate presenting hyperproteinorrachia. Glucose levels were 51.0 mg/dL (IQR 46.0–56.0), both values within normal limits. No cases of microbiologically confirmed meningitis were identified, and microbiologic studies were negative in all samples. Bacterial cultures and viral PCR were negative in all samples, and CSF SARS-CoV-2 PCR was negative in all 12 tested patients.

Median age at lumbar puncture for biomarker analysis was 16 days (IQR 11–20) in cases and 17 days (IQR 14–21) in controls ($P = 0.265$). CSF B2M levels were significantly higher in infected neonates (median 4.16 mg/L, IQR 3.17–5.56, n = 9) than in age-matched controls (median 2.37 mg/L, IQR 1.64–3.28, n = 21; $P = 0.011$). However, no significant differences in B2M levels were observed between neonates with and without neurologic symptoms ($P = 0.286$). In contrast, CSF NSE levels did not differ significantly between infected neonates (median 7.40 ng/mL, IQR 2.70–13.13, n = 8) and controls (median 7.42 ng/mL, IQR 5.78–10.25, n = 20; $P = 0.859$) (Fig. 2).

To assess potential selection bias in the subgroup undergoing lumbar puncture, we compared the frequency of fever and neurologic symptoms between neonates with and without lumbar puncture. Neonates undergoing lumbar puncture had a similar frequency of fever compared with those without lumbar puncture (90% vs. 88%), while neurologic symptoms were more frequent in the lumbar puncture group (60% vs. 37%), although this difference was not statistically significant ($P = 0.084$).

Systemic Involvement

No neonates required mechanical ventilation, inotropic support or transfusions. Supplemental oxygen was required in 12

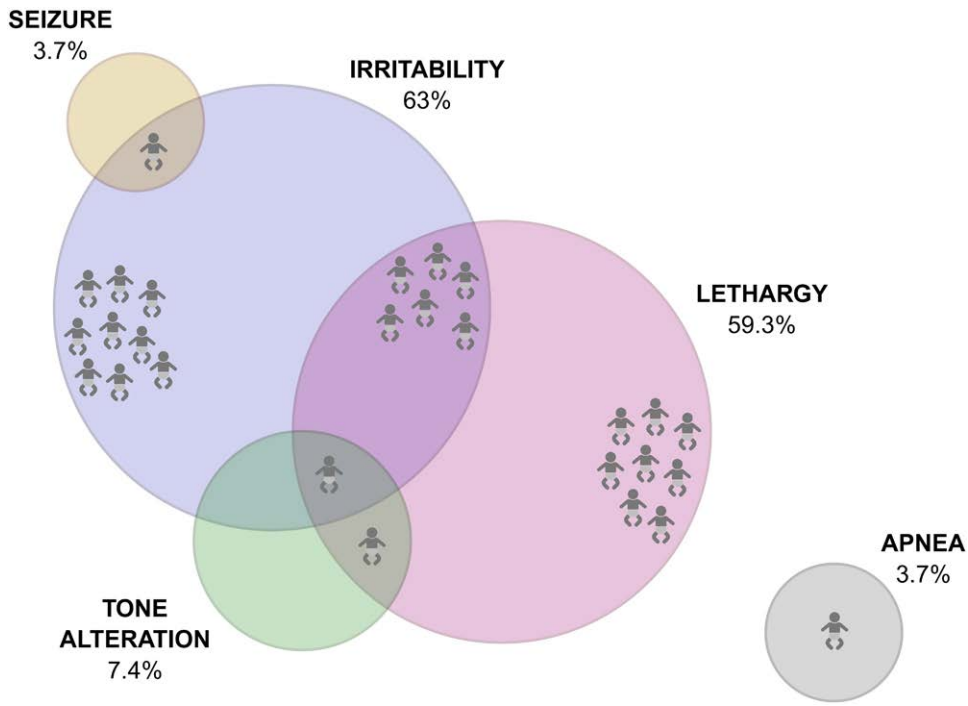

FIGURE 1. Distribution and overlap of neurologic symptoms in neonates with SARS-CoV-2 infection. The Venn diagram illustrates the relative frequency of seizures, irritability, lethargy, tone alteration and apnea, as well as their areas of overlap. The neonate icons represent the number of patients presenting each symptom, and the percentages indicate the proportion of patients within each category. In total, 27 neonates exhibited neurologic symptoms, while 34 did not show any neurologic manifestations. Created in BioRender[11] (https://BioRender.com/2p8ieay).

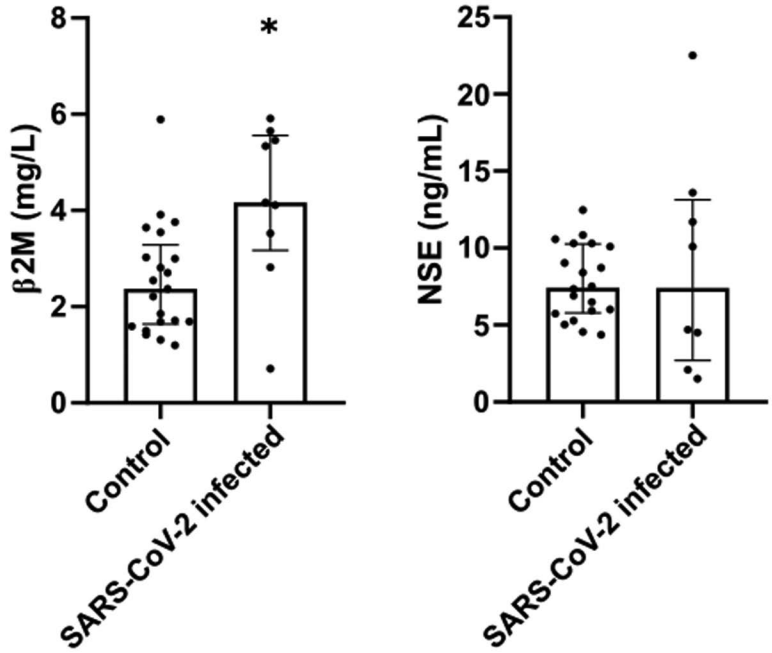

FIGURE 2. Cerebrospinal fluid B2M and NSE levels in neonates with SARS-CoV-2 infection and age-matched controls. Bars represent the median with interquartile range; dots indicate individual values. Statistical comparisons were performed using the Mann–Whitney *U* test (*$P < 0.05$).

neonates (20%) for a median of 3 days (IQR 2–3.75). Fluid and electrolyte therapy was provided to 35% of neonates, while antibiotics and antipyretics were administered to 34.4% and 50% of patients, respectively. Cardiac, pulmonary and abdominal ultrasounds performed in subsets of neonates were all normal.

All neonates with blood tests had elevated ferritin. Elevated fibrinogen and D-dimer were observed in 50% and 47.4%, respectively. Coagulation abnormalities (prothrombin activity, prothrombin time and/or activated partial thromboplastin time) occurred in 17.4%. Neutropenia was present in 39.2%, lymphopenia in 19.6%, abnormal NLR in 47.1%, abnormal PLR in 41.2%, and elevated transaminases (aspartate aminotransferase, alanine aminotransferase and/or gamma-glutamyl transferase) in 51.5%. Elevated IL-6 was observed in 72.2%. No significant complications or deaths occurred.

DISCUSSION

Concerns have been raised regarding possible neurodevelopmental consequences following neonatal SARS-CoV-2 infection or *in utero* exposure. Some studies have reported mild delays in language or motor domains during early childhood, although severe or persistent deficits appear uncommon.[12,13] In contrast, our findings focus on the acute neurologic phase of neonatal infection and show no evidence of meningoencephalitis, neuronal injury biomarkers or clinically significant neurologic dysfunction. These findings suggest that acute CNS involvement in neonatal SARS-CoV-2 infection is uncommon.

This multicenter cohort supports previous observations that neonatal SARS-CoV-2 infection is generally associated with mild and transient neurologic symptoms without evidence of meningoencephalitis or persistent neurologic impairment. The observed increase in CSF B2M without NSE elevation suggests intrathecal immune activation rather than neuronal injury. B2M has previously been associated with inflammatory CNS processes in neonates, including neonatal infections and hypoxic-ischemic encephalopathy, where it reflects activation of the immune response within the CNS.[6–8] In contrast, NSE is a marker of neuronal injury, and its normal levels in our cohort support the absence of structural neuronal damage.[9] Similar patterns have been described in adults with COVID-19 and neurologic symptoms, where elevated CSF inflammatory markers were observed without typical viral meningitis features.[14,15] This pattern may explain the benign clinical course and complete recovery observed.

Three neonates showed elevated CSF leukocytes. Although these neonates presented with fever, microbiologic studies were negative, and no additional clinical findings suggestive of meningitis were identified. Negative SARS-CoV-2 CSF PCR and the absence of confirmed meningitis suggest direct CNS viral invasion is uncommon. In this context, neurologic involvement in neonatal SARS-CoV-2 infection is likely mediated by indirect mechanisms, including systemic inflammation (elevated ferritin, coagulation abnormalities, and abnormal NLR and PLR) and cytokine release (IL-6 increase), which may increase blood–brain barrier permeability and promote neuroimmune activation without viral neuroinvasion.[16] Additional mechanisms such as endothelial dysfunction, microglial activation, and immune-mediated responses have also been described in COVID-19–related neuroinflammation.[17]

The limited use of neurologic investigations in this study reflects real-world clinical practice in neonatal care. Although the study protocol recommended lumbar puncture and neuroimaging in neonates presenting with fever and/or neurologic symptoms, the mild and transient nature of the clinical manifestations often led clinicians to adopt a conservative approach, and the protocol recommendations were not systematically followed in routine practice. Conducting multicenter clinical research in neonates poses inherent challenges, as diagnostic procedures must remain guided by clinical judgment and patient safety rather than research requirements. Consequently, achieving systematic neurologic investigations in

this population may be difficult when the clinical course is generally benign. Nevertheless, this pragmatic approach provides valuable insight into the neurologic presentation of neonatal SARS-CoV-2 infection in routine clinical settings. Importantly, despite the limited and clinically driven number of lumbar punctures, this study represents one of the largest multicenter neonatal cohorts with CSF analysis in SARS-CoV-2 infection and, to our knowledge, the first to assess CSF inflammatory (B2M) and neuronal injury (NSE) biomarkers in this population, providing novel and clinically relevant information on subclinical CNS involvement.

Importantly, the study period included neonates born before and after the introduction of the official recommendation of COVID-19 vaccination during pregnancy in Spain (August 2021). Although maternal vaccination status was not systematically collected, the overall benign neurologic course observed across the cohort suggests that severe acute neurologic involvement is uncommon even in the absence of vaccination. Finally, our cohort included neonates evaluated for possible CNS infection (fever and/or neurologic symptoms) and therefore does not represent all neonatal SARS-CoV-2 infections.

CONCLUSIONS

Neonatal SARS-CoV-2 infection in this multicenter cohort was associated with mild and transient neurologic symptoms, without evidence of meningoencephalitis or persistent neurologic impairment. The observed elevation of CSF B2M suggests subclinical CNS immune activation, highlighting the potential role of CSF biomarkers in improving our understanding of neonatal neuro-COVID. Further longitudinal studies are warranted to clarify the clinical significance of these findings and their potential relevance for long-term neurodevelopmental outcomes.

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