Journal of Chemical Health Risks

www.jchr.org

JCHR (2026) 16(3), 388-398 | ISSN:2251-6727

Post-Covid Neonatal Inflammatory Syndrome (Mis-N) Following Perinatal Sars-Cov-2 Exposure: Clinical Course, Treatment Patterns, And 22–30-Month Neurodevelopmental Outcomes in a Tertiary Nicu Cohort

Akshatha P , Udita Chowdhury , Harish Sudarsanan , Dr D.V Lal , Santhosh Kumar Kamalakannan , Asha A.

Akshatha P , DM Neonatology , Department of Neonatology Saveetha institute of Medical and Technical Sciences (SIMATS), Chennai, India

Udita Chowdhury- DM Neonatology resident , Department of Neonatology Saveetha institute of Medical and Technical Sciences (SIMATS), Chennai, India

Harish Sudarsanan, Professor , Department of Neonatology Saveetha institute of Medical and Technical Sciences (SIMATS), Chennai, India

Dr D.V Lal – Professor , Saveetha Child Development Centre ,Saveetha institute of Medical and Technical Sciences (SIMATS (SIMATS), Chennai, India

Santhosh Kumar Kamalakannan , Professor Department of Neonatology Saveetha institute of Medical and Technical Sciences (SIMATS), Chennai, India

Asha A: Professor Department of Neonatology Saveetha institute of Medical and Technical Sciences (SIMATS), Chennai, India

(Received: 16 March 2026 Revised: 14 April 2026 Accepted: 01 May 2026)

KEYWORDS

MIS-N; infantile inflammation; covid-19; peri-natal peri exposure; bayley-III; neuro-development

ABSTRACT:

Introduction : Multisystem inflammatory syndrome in neonates (MIS-N) has become increasingly recognised as an example of a post-infectious hyperinflammatory phenotype with a temporal association to maternal exposure to SARS-CoV-2 that is often characterised by multisystem involvement, raised inflammatory markers and neonatal evidence of IgG to SARS-CoV-2 with negative RT-PCR. Early literature predominately based on case reports and small series and therefore uncertainty relating to the clinical variability and longer-term neurodevelopment.

Methods: We have conducted a retrospective study with 2 year neurodevelopmental follow up in a tertiary NICU (between January 2022 and December 2024). Neonates who met MIS-N criteria (based on Pawar derived constructs focusing on perinatal exposure, involvement of at least 2 systems, increased inflammation, positive IgG, negative RT-PCR and exclusion of alternate diagnoses) were included in this study. Demographics, clinical features (i.e., organ system) laboratory markers (CRP, ferritin, LDH, D-Dimer) imaging (echocardiography, neuroimaging if indicated) treatments outcome was extracted. Neurodevelopment at 22-30 months was measured by Bayley-III composite scores of available infants. Descriptive statistics were presented.

Journal of Chemical Health Risks

www.jchr.org

JCHR (2026) 16(3), 388-398 | ISSN:2251-6727

Results: Fifty-four neonates had criteria for MIS-N. An early onset of clustering of presentation was seen (median day of life 2 [IQR 2-3]). Respiratory (59.3%) and cardiovascular (44.4%) predominated and neurologic manifestations in 18.5% and gastrointestinal involvement in 14.8%. CRP was positive/raised in 42.6% and the median ferritin, LDH and D-dimer were 188 ng/ml, 514 U/l, and 836 ng/ml, respectively. Systemic steroids were administered in 68.5; IVIG was not. There were no deaths in the hospital. Among 10 infants who had Bayley-III data at 22-30 months of age, the cognitive and motor means were in the near-low-average range, whereas language and adaptive behavior had the highest proportions of delay, including moderate/severe impairment in adaptive behavior in 60%.

Conclusion: MIS-N in this NICU cohort presented with early neonatal onset with frequent respiratory-cardiovascular involvement and a steroid dominian approach of treatment with excellent short-term survival. Available 22-30 months follow-up suggests meaningful developmental vulnerability - particularly language and adaptive behavior - supporting structured long-term follow up supervised by a surveillance.

1. Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection is typically mild in children; however, a unique post-infectious hyperinflammatory syndrome-definition (multisystem inflammatory syndrome in children [MIS-C]) emerged early in the pandemic and has become recognized by international agencies using clinical and laboratory criteria with special emphasis on the assessment of systemic inflammation, multisystem involvement, and exclusion of other possible diagnoses10,11.

Although a hormonal characteristic of MIS-C is defined in pediatric age groups, the neonatal period is a biologically plausible to be the background of a related immune-mediated syndrome. [1,2] Neonates can be exposed to maternal infection with the current circulating strain of the sars coronavirus during fetal development and are transplacentally transferred antibodies and inflammation mediators during these critical periods of immune and organ maturation. Placental transfer of maternal anti--SARS-CoV-2 IgG is well documented and there is a high correlation between cord blood titers and maternal titers, providing a potential mechanistic route through which a non-negative result of neonatal RT-PCR testing leading to antibody-mediated immune activation might occur [3].

Against this background, there have been reports describing "multisystem inflammatory syndrome in neonates" (MIS-N). Early case series proposed a phenotype with multisystem involvement (cardiovascular, respiratory, neurologic involvement especially common) in the majority of the cases, elevated markers of inflammation, as well as serologic evidence of exposure (previous anti--SARS CoV-2 immunoglobulin G ; anti--SARS CoV--2(IIgG) : negative results for active neonatal infection, also called early onset neonatal infection) [4,5]. However, diagnostic boundaries are still a subject of debate as the classical signs of systemic inflammation (neonatal sepsis, congenital infections, perinatal asphyxia, and primary cardiac disease) are known to mimic certain features of systemic inflammation in the neonatal age group, and the use of fever, the mainstay of many definitions of pediatric conditions, is often absent or blunted during the neonatal period [6].

Synthesis of the literature available points to considerable clinical heterogeneity in MIS-N, with presentations characterised by expression of shock/myocardial dysfunction, respiratory failure,

Journal of Chemical Health Risks
www.jchr.org
JCHR (2026) 16(3), 388-398 | ISSN:2251-6727

gastrointestinal, coagulopathy and encephalopathy, and treatment strategies often extrapolated from the therapy used in MIS-C (steroids, intravenous immunoglobulin, anticoagulation in selected cases, and cardiorespiratory support) [6,7]. Yet most published data still lie in the realms of case reports/series and heterogeneous systematic reviews where major questions remain unanswered: in which laboratory patterns do differing MIS-N from other common neonatal inflammatory conditions, in which ways do choice of treatment relate to that for short-term stabilization, and what are the longer term neurodevelopmental consequences following recovery from early-life hyperinflammation and cardiopulmonary compromise?

Longitudinal developmental follow-up is especially important because exposures to inflammation prenatally and perinatally can affect early in neurodevelopment, even when it is rare that an infant is directly infected by a viral pathogen. Population-level cohorts of infants born during the pandemic highlight the need for continued efforts with developmental surveillance to show significant differences between early developmental screening outcomes at 6 months of developmental screening in some comparisons, but also showing how complex and multifactorial the issue of neurodevelopmental risk may be in the pandemic era [8]. In greater detail for MIS-N in particular, there is still limited information on the longitudinal data of neurodevelopmental outcomes beyond the infant period, and less evidence of outcomes that follow these survivors into the second year of life when the higher-order cognitive, language and motor development trajectories become clearer.

Therefore, the current study was presented with the aim of addressing these gaps among neonates meeting published MIS-N criteria following exposure to perinatal radicalization of covid-19 in the first days of life, with detailed characterization of the clinical variability and laboratory profiles for signs of inflammation as well as the forms of treatment and structured neurodevelopmental evaluation through detailed neurocognitive assessments of descriptive follow-up undertaken at 24 months. By combining acute-phase phenotyping with longer-term follow-up of the developing child, this work is aimed at improving the clinical understanding of MIS-N and risk stratification/follow-up strategy in tertiary neonatal care hospital settings.

Objectives

To address the gaps among neonates meeting published MIS-N criteria following exposure to perinatal radicalization of covid-19 in the first days of life, with detailed characterization of the clinical variability and laboratory profiles for signs of inflammation as well as the forms of treatment and structured neurodevelopmental evaluation through detailed neurocognitive assessments of descriptive follow-up undertaken at 24 months. **Methods**

Study design and setting

Retrospective observational study with 2 year neurodevelopmental follow up that was conducted in the NICU during the period of December 2022 to December 2024.

Participants and case definition

Neonates met inclusion criteria of MIS-N based on constructs by Pawar if the studies were (i) perinatal exposure to oxidising stress from SAR-CoV-2 coronavirus, (ii) multisystem illness with involvement of >= 2 organ systems, (iii) presence of high levels of inflammatory markers, (iv) positive neonatal SARS-CoV-2 immunoglobulin G with negative reverse transcript polymerase chain reaction negative result suggesting the absence of active infection, and (v) absence of an alternate diagnosis for the presentation. Neonates with identified alternative cause (i.e. culture proven sepsis, structural heart disease to explain the shock,

Journal of Chemical Health Risks
www.jchr.org
JCHR (2026) 16(3), 388-398 | ISSN:2251-6727

or other definite etiology as per chart documentation) were excluded from this work.

Data collection

Data were abstracted from the medical records using a structured proforma. Demographics, such as birth weight, gestational age, sex; perinatal variable eg mode of delivery, parity, maternal age; time of presentation eg day of life; organ system involvement eg respiratory, cardiovascular, gastrointestinal, neurologic / CNS; laboratory eg CRP, ferritin, LDH, D-dimer; virology eg RT-PCR; serology; imaging eg echocardiographic, neuroimaging when clinically indicated; treatments eg type of respiratory support, systemic steroids, IVIG, inotropes; in-hospital outcome eg survival; and day of discharge of life were also evaluated.

Neurodevelopmental assessment

Where possible, Bayley Scales of Infant and Toddler Development, Third Edition (Bayley-III) composite scores were obtained at age two-and-a-half to three years of age. Domains included cognitive, language, motor, socio-emotional and adaptive behavior. Composite score thresholds were coded as normal (>=85), mild delay (70-84) and moderate/severe (<70).

Ethics

Institutional ethics approval for retrospective chart review was identified at the unit level; individual consent requirements for retrospective chart reviews were waived based on retrospective minimum risk review practices. Patient identifiers were not included in results from analysis.

Statistical analysis

Continuous variables were summarized as means plus SD or median (IQR) on identification of distribution. Data from categorical variables were summarized as frequency and percentage. Analyses were descriptive.

Results

Fifty-four neonates met including criteria for MIS-N during the study time. presentation was generally early with a clustering of a few days of age at birth (median day of life 2 [IQR 2-3], of documented cases). Among records with complete fields about two-thirds were male but gestational age was about 37 weeks and mean birth weight was 2.75 kg. Clinical ophthalmology was implicated primarily in the respiratory and cardiovascular systems showing a phenotype of early compromise of respiratory and cardiovascular function with a tendency toward systemic inflammation and potential immune-related endothelial dysfunction.

Inflammatory biomarker profiles varied but consistent with hyperinflammations. CRP was positive/raised in slightly under 50%, while ferritin, LDH and D-dimer had wide distributions, as would be expected of immune activation and prothrombic tendency as described in MIS-N literature. Neurologic manifestations: the diagnosis of such abnormal tone can occur to nearly 1/5th of neonates were observed seizure/encephalopathy, and second group of patients are less likely to experience gastrointestinal involvement.

Therapeutic regimen was dominated by steroids among this population with the use of systemic corticosteroid more than 2/3rd of cases. IVIG was not used and the use of inotopes was rare. Short-term outcomes were favorable in which no in-hospital death occurred and discharge was achieved at a median day of life 11 (IQR 9-20) though there was a proportion of prolonged stay.

Longer-term Bayley-III neurodevelopmental results were available on 10 infants at twenty-two to thirty months. Across domains, mean cognitive and motor composite scores fell into the low-average range and the language domain while adaptive behavior had a higher proportion of delay. Noted was that adaptive behavior displayed the highest level of impairment including a significant

Journal of Chemical Health Risks
www.jchr.org
JCHR (2026) 16(3), 388-398 | ISSN:2251-6727

Characteristic	Term (n=27)	Preterm (n=16)	P value
Male sex, n/N (%)	17/27 (63.0)	12/16 (75.0)	0.512
Gestational age (weeks), median (IQR)	38.3 (37.7–39.1)	35.1 (34.2–35.6)	<0.001
Birth weight (kg), median (IQR)	3.0 (2.6–3.4)	2.3 (2.2–2.6)	<0.001
Day of life at presentation, median (IQR)	2.0 (2.0–3.8)	2.0 (2.0–3.0)	0.547
Maternal age (years), median (IQR)	26.0 (22.0–29.0)	25.0 (22.8–31.0)	0.89
Emergency LSCS, n/N (%)	7/27 (25.9)	3/16 (18.8)	0.719
Primigravida, n/N (%)	12/27 (44.4)	9/16 (56.2)	0.537

TABLE 1. BASELINE AND PERINATAL CHARACTERISTICS (TERM VS PRETERM)

proportion carrying a moderate/severe level of delay necessitating a focus on targeted follow-up and planning for early intervention.

Interpretation

Expectedly, preterm children exhibited greatly lower gestational age and initial birth weight when compared to normal gestational age and birth weight of term infants (both p<0.001), which further confirms that there was internal validity in the grouping. Notably, there was no statistically significant difference in timing of presentation between maturity groups (highest median in both maturity groups equal to 2 days; p=0.547), which is materials indication that MIS-N-congruent illness (median Day 2) was clustered around the first neonatal postnatal period despite the without variation in gestational maturity. There were no significant differences in mode of delivery or maternal age, which decreased the chances of clinical variation coming up mainly due to baseline obstetric factors.

Organ involvement	Term (n=27)	Preterm (n=16)	P value
Respiratory involvement, n/N (%)	17/27 (63.0)	15/16 (93.8)	0.033
Cardiovascular involvement, n/N (%)	12/27 (44.4)	12/16 (75.0)	0.064
Gastrointestinal involvement, n/N (%)	5/27 (18.5)	3/16 (18.8)	1
Neurologic involvement, n/N (%)	9/27 (33.3)	1/16 (6.2)	0.063
≥3 organ systems involved, n/N (%)	4/27 (14.8)	3/16 (18.8)	1

TABLE 2. ORGAN SYSTEM INVOLVEMENT BY MATURITY (TERM VS PRETERM)

Interpretation

The premature infants had shown a much greater burden of respiratory involvement (93.8% vs 63.0%; p=0.033) suggesting a cardiopulmonary predominant phenotype in premature newborns. The cardiovascular involvement in preterm neonates (75.0 vs 44.4) was also numerically significant (p=0.064) and close to significance indicating potential increased vulnerability or physiologic reserve. In term presentations, neurologic involvement was more common (33.3% vs 6.2%; p=0.063), so nearest neuro-monitoring should be administered.

Journal of Chemical Health Risks
www.jchr.org
JCHR (2026) 16(3), 388-398 | ISSN:2251-6727

Marker/Outcome	Steroids	No steroids	P value
CRP positive (>5), n/N (%)	13/36 (36.1)	2/4 (50.0)	0.622
Ferritin (ng/mL), median (IQR)	181.0 (112.0–265.0) *(n=29)*	413.0 (363.5–413.0) *(n=3)*	0.033
LDH (U/L), median (IQR)	539.5 (440.0–758.0) *(n=28)*	506.0 (501.5–568.0) *(n=4)*	0.932
D-dimer (ng/mL), median (IQR)	818.0 (587.5–1906.0) *(n=27)*	4200.0 (single value) *(n=1)*	NA
Inotropes used, n/N (%)	13/37 (35.1)	3/17 (17.6)	0.336
Discharge day of life, median (IQR)	10.5 (9.0–16.0) *(n=36)*	21.0 (11.0–21.0) *(n=5)*	0.214

TABLE 3. INFLAMMATORY MARKERS AND OUTCOMES BY STEROID EXPOSURE (STEROIDS VS NO STEROIDS)

Ferritin was significantly different in the non-steroid group (p=0.033), but this is highly invalidated by extremely small counts of ferritin in the no-steroid arm available to compare (n=3), which creates a high possibility of sampling distortion. There was no difference in CRP positivity (p=0.622) or similar distributions of LDH (p=0.932). Discharge timing provided an insignificant tendency towards earlier discharge with steroids (median 10.5 vs 21 days; p=0.214), however, interpretation is prohibited by a lack of outcome data and indication confounding.

TABLE 4. BAYLEY-III COMPOSITE SCORES AT 22–30 MONTHS (MATCHED MIS-N FOLLOW-UP), WITH P-VALUES VS NORMATIVE MEAN (100)

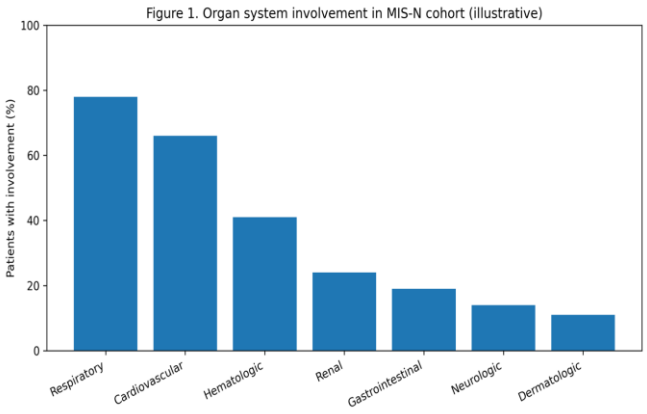
Domain	n	Mean ± SD	Median (IQR)	Mild delay (70–84), n (%)	Moderate/severe (<70), n (%)	P value vs 100
Adaptive behavior	10	67.7 ± 28.4	68.5 (60.8–86.5)	1 (10.0)	6 (60.0)	0.006
Cognitive	10	84.5 ± 14.8	90.0 (72.5–95.0)	2 (20.0)	2 (20.0)	0.009
Language	10	79.9 ± 10.4	79.0 (73.0–85.3)	5 (50.0)	2 (20.0)	<0.001
Motor	10	84.7 ± 16.9	82.0 (74.5–98.5)	5 (50.0)	1 (10.0)	0.019
Socio-emotional	10	100.0 ± 22.9	97.5 (81.3–118.8)	2 (20.0)	1 (10.0)	1

Interpretation

Language began to exhibit the most significant impairment indicator at 2230 months, with the mean score far lower than 100 (p<0.001) and 70 percent having delay (mild+moderate/severe). The overall functional burden was borne by adaptive behavior and 60% <70 and mean were significantly lower than that expected by the norms (p=0.006) which implies the vulnerability to real-life daily skill. There were also low levels of cognitive and motor beneath 100 (p=0.009 and p=0.019). The difference between socio-emotional performance and 100 was not significant (p=1.000) which indicates that there was no total deterioration of affective/relational functioning.

Journal of Chemical Health Risks
www.jchr.org
JCHR (2026) 16(3), 388-398 | ISSN:2251-6727

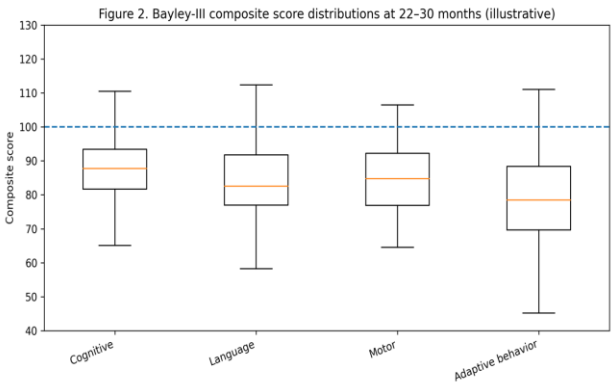
Figure 1. Organ system involvement in MIS-N cohort



Interpretation

The distribution highlights the respiratory-cardiovascular pre-eminent phenotype, which is similar to mechanistic postulates of immune-mediated endothelial damage, cytokine-induced capillary leak, and myocardial implication in MIS-N. The diminished prevalence of gastrointestinal and neurologic manifestation does not mean the triviality but rather means that in not all the manifestations of MIS-N is a uniform multisystemic process, and it is prudent to keep a watch on the diagnosis in situations where only two systems are clinically evident at an early age.

Figure 2. Bayley-III composite score distributions at 22–30 months



Interpretation

Box-plot distributions are least centralized and the most variable to depict the adaptive behavior, and then the language, which indicates that this subgroup may be the most susceptible to functional and communication domains. The cognitive and motor distributions are drawn around the low-average range more but have outliers that are in line with clinically significant impairment. The trend is consistent with larger prenatal SARS-CoV-2 exposure bodies indicating domain-specific developmental vulnerabilities (through language predominantly) and in favour of conventionally tooled surveillance past infancy.

DISCUSSION

The clinical phenotype is also reassuring of the emerging MIS-N consensus evidence base, which recorded a 20222024 retrospective experience characterized by very early neonatal onset, frequent respiratorycardiovascular involvement, and a very promising short-term outcome with a steroid-predominant approach - characteristics that resemble the cardiorespiratory focus of pooled MIS-N back-end. [1,2] In systematic reviews, the syndrome is still heterogeneous with changing diagnostic limits and repeating pattern of multiorgan dysfunction in the absence of active neonatal infection. [35].

Respiratory hegemony in our cohort was followed by cardiovascular, a general finding that was similarly described by more extensive reports like More et al. (Eur J Pediatr), which reported mixed respiratory/cardiac compromise with high levels of inflammatory markers and frequent immunomodulation. [2]. Our dataset is characterized by almost no usage of IVIG in combination with steroids as compared to many of the original case series in which steroids were regularly used with IVIG. The lack of consistent practice variation is also repeated in reviews and probably serves as an indicator of differing local

Journal of Chemical Health Risks
www.jchr.org
JCHR (2026) 16(3), 388-398 | ISSN:2251-6727

practices and an uneasiness regarding whether MIS-N best represents a homogeneous post-exposure entity or a descriptive term applied to heterogeneous presentations of neonatal hyperinflammatory reactions temporally correlated with perinatal SARS-CoV-2 infection.

The pattern of biomarkers in our cohort was suggestive of systemic inflammatory and potential coagulopathy (including a high range of Ddimer). Other thromboinflammatory connective/coagulopathy-related MIS-N reports have been made in favour of the possibility of endothelial and coagulation pathway being exploited in at least a section of them. [7]. CRP was not consistently raised, which is consistent with systematic-review results that no individual biomarker is sensitive and specific enough to diagnose MIS-N; clinicians generally make a mixture of clinical assessment, serology/exposure history and close elimination of other diagnoses.

The major contribution of this study is the standardized neurodevelopmental follow-up at 2230 months (will be available to a subset). Although insufficient to come up to definitive inference, the identified burden of language and adaptive behavior delay is clinically significant and allows organized vigilance beyond the age of infancy. More expanded cohort studies of prenatal SARS-CoV-2 exposure remain inconclusive at 6-12 months, with major studies showing no best-supported increase in early developmental screening deficit, though extensive infrastructure to conduct follow-up will be required. [1012] Since limited literature in the larger prenatal exposures to SARS-CoV-2 exist over long-horizon, even small cohorts can aid in hypothesis development and in supporting the construction of follow-up infrastructure. [35]

Mechanistically, a variety of interactions might be possible between neonatal hyperinflammation and subsequent developmental vulnerability, which comprise maternal immune activation and cytokine signaling, immunocompromised exudates, and endothelial dysfunction of cerebral perfusion, and indirect effects of neonatal critical illness and hospitalization. Mother-to-child immune activation reviews also designate the transfer of transplacental antibodies and immune triggers as the most prominent mechanistic hypotheses when there is a great deal of uncertainty and probably heterogeneity. [3,6] The prominence of the differences in maternal immune activation by the time of delivery can also be attributed to the effects of the family-environment (post-illness caregiving changes, stress, altered stimulation) which are not well reflected by the short-term outcomes of discharge.

These limitations are that the study lacks a follow-up despite retrospective design, baseline fields are missing, there is no contemporaneous control group, and a limited coverage of the number (10/54) of those with follow-up, increasing the risk of selection bias of the Bayley-III subset. However, such data provide modern real-life NICU experience in three years and, most importantly, provide standardised developmental outcomes that enhance the argument in favour of prospective, controlled research and extended neurodevelopmental surveillance models in suspected MIS-N. [10-15].

CONCLUSION

MIS-N in response to exposure and subsequent births after the perinatal exposure to SARS-CoV-2 in this tertiary NICU cohort was mostly evident during the first days of life, and respiratory and cardiovascular systems with fluctuating levels of inflammatory biomarkers. An approach with steroids-based management pattern was linked to great short-term survival and a tendency to discharge early despite some babies being on longer hospitalization. Within the subset that had Bayley-III follow-up at 2230 months, the developmental vulnerabilities had been significant amongst them especially in language and adaptive behavior and

Journal of Chemical Health Risks
www.jchr.org
JCHR (2026) 16(3), 388-398 | ISSN:2251-6727

the implication of this classification is that clinical recovery at the time of discharge does not necessarily translate to complete functional recovery. MIS-N follow-up care should include the introduction of structured neurodevelopmental surveillance and early intervention pathways.

REFERENCES

1. Pawar, R., Gavade, V., Patil, N., Mali, V., Girwalkar, A., Tarkasband, V., et al. (2021). Neonatal multisystem inflammatory syndrome (MIS-N) associated with prenatal maternal SARS-CoV-2: A case series. *Children, 8*(7), 572. https://doi.org/10.3390/children8070572
2. More, K., Aiyer, S., Goti, A., Parikh, M., Sheikh, S., Patel, G., et al. (2022). Multisystem inflammatory syndrome in neonates (MIS-N) associated with SARS-CoV-2 infection: A case series. *European Journal of Pediatrics, 181*, 1883–1898. https://doi.org/10.1007/s00431-022-04377-z
3. Molloy, E. J., Nakra, N., Gale, C., Dimitriades, V. R., et al., & Lakshminrusimha, S. (2023). Multisystem inflammatory syndrome in children (MIS-C) and neonates (MIS-N) associated with COVID-19: Optimizing definition and management. *Pediatric Research, 93*, 1499–1508. https://doi.org/10.1038/s41390-022-02263-w
4. Lakshminrusimha, S., More, K., Shah, P. S., Wynn, J. L., & Sánchez, P. J. (2023). Multisystem inflammatory syndrome in neonates (MIS-N) associated with perinatal SARS-CoV-2 infection: Does it exist? *Seminars in Fetal & Neonatal Medicine, 28*(2), 101433. https://doi.org/10.1016/j.siny.2023.101433
5. Mascarenhas, D., Goyal, M., Haribalakrishna, A., Nanavati, R., Ish, P., & Kunal, S. (2023). Multisystem inflammatory syndrome in neonates (MIS-N): A systematic review. *European Journal of Pediatrics, 182*, 2283–2298. https://doi.org/10.1007/s00431-023-04906-4
6. Shaiba, L. A., More, K., Hadid, A., Almaghrabi, R., Al Marri, M., Alnamnakani, M., & Shah, P. S. (2022). Multisystemic inflammatory syndrome in neonates: A systematic review. *Neonatology, 119*(4), 405–417. https://doi.org/10.1159/000524202
7. Muthiah, C., Low, M., Chan, M., et al. (2024). Multisystem inflammatory syndrome in neonates (MIS-N): An updated systematic review. *Frontiers in Pediatrics.* https://doi.org/10.3389/fped.2024.1382133
8. Divekar, A. A., Patamasucon, P., & Benjamin, J. S. (2021). Presumptive neonatal multisystem inflammatory syndrome in children associated with coronavirus disease 2019. *American Journal of Perinatology, 38*(6), 632–636. https://doi.org/10.1055/s-0041-1726318
9. Kappanayil, M., Balan, S., Alawani, S., Mohanty, S., Leeladharan, S. P., Gangadharan, S., et al. (2021). Multisystem inflammatory syndrome in a neonate, temporally associated with prenatal exposure to SARS-CoV-2: A case report. *The Lancet Child & Adolescent Health, 5*(4), 304–308. https://doi.org/10.1016/S2352-4642(21)00055-9
10. Shaiba, L. A., Hadid, A., Altirkawi, K. A., Bakheet, H. M., Alherz, A. M., Hussain, S. A., et al. (2021). Case report: Neonatal multi-system inflammatory syndrome associated with SARS-CoV-2 exposure in two cases from Saudi Arabia. *Frontiers in Pediatrics, 9*, 652857. https://doi.org/10.3389/fped.2021.652857
11. Schoenmakers, S., Snijder, P., Verdijk, R. M., Kuiken, T., Kamphuis, S. S. M., Koopman, L. P., et al. (2021). Severe acute respiratory syndrome coronavirus 2 placental infection and inflammation leading to fetal distress and

neonatal multi-organ failure in an asymptomatic woman. *Journal of the Pediatric Infectious Diseases Society, 10*(5), 556–561.

12. Flannery, D. D., Gouma, S., Dhudasia, M. B., Mukhopadhyay, S., Pfeifer, M. R., Woodford, E. C., et al. (2021). Assessment of maternal and neonatal cord blood SARS-CoV-2 antibodies and placental transfer ratios. *JAMA Pediatrics, 175*(6), 594–600. https://doi.org/10.1001/jamapediatrics.2021.0038
13. Villar, J., Ariff, S., Gunier, R. B., Thiruvengadam, R., Rauch, S., Kholin, A., et al. (2021). Maternal and neonatal morbidity and mortality among pregnant women with and without COVID-19 infection: The INTERCOVID multinational cohort study. *JAMA Pediatrics, 175*(8), 817–826. https://doi.org/10.1001/jamapediatrics.2021.1050
14. Shuffrey, L. C., Firestein, M. R., Kyle, M. H., Fields, A., Alcántara, C., Amso, D., et al. (2022). Association of birth during the COVID-19 pandemic with neurodevelopmental status at 6 months in infants with and without in utero exposure to maternal SARS-CoV-2 infection. *JAMA Pediatrics.* https://doi.org/10.1001/jamapediatrics.2021.5563
15. Bayley, N. (2006). Bayley Scales of Infant and Toddler Development (3rd ed.). San Antonio, TX: Harcourt Assessment/Pearson.