

Clinical Profile and Outcomes of Suspected Multisystem Immune Response Syndrome in Neonates with Elevated SARS-CoV-2 IgG Antibodies: A Retrospective Observational Study from a Tertiary NICU

Nilesh Darvhekar¹, Kush Jhunjunwala², Kirti³, Suchita Khadse⁴, Satish Deopujari⁵

¹ Department of Pediatrics and Neonatology, Nelson Hospital, Nagpur, Maharashtra, India

² Academic Counsel of Nelson Hospital, Nagpur, Datta Meghe Medical College, Nagpur, Maharashtra, India

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Corresponding Author: Dr. Nilesh Darvhekar

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Abstract:

Introduction: Multisystem immune response syndrome in neonates is increasingly recognized as a hyperinflammatory condition associated with trans placentally acquired maternal antibodies against SARS CoV2. The complete clinical spectrum of this condition is still evolving, and standardized diagnostic criteria and management strategies remain poorly defined.

Aim: This study aims to evaluate the clinical features, inflammatory markers and treatment responses of neonates who are suspected to have multisystem inflammatory syndrome. It also aims to identify recurring patterns of organ involvement and laboratory abnormalities, while examining short term outcomes following immunomodulatory therapies to improve early recognition and guide more consistent diagnostic and management approaches.

Results: A total of 35 neonates met the inclusion criteria, with most becoming symptomatic within the first 48 hours after birth. Respiratory manifestations were the most common presentation, occurring in 82.8% cases, while neurological features such as seizures were observed in 45.7%. Thrombocytopenia was the most frequent laboratory abnormality, identified in 77.1% of the neonates studied. SARS CoV2 IgG antibody levels were elevated in nearly all infants, though antibody titres showed no significant association with disease severity. Neonates who received intravenous immunoglobulin earlier in the disease course demonstrated faster clinical improvement. Due to death of two neonates the overall mortality rate in the cohort was 5.2%.

Conclusion: Suspected neonatal inflammatory syndrome usually presents early, frequently with multisystem involvement and elevated inflammatory markers. Antibody titres alone do not reflect disease severity. Early immunomodulatory therapy may lead to better short-term recovery. Larger prospective studies are required to clarify pathophysiology, establish diagnostic criteria and guide evidence-based management strategies.

Keywords: Multisystem Inflammatory Syndrome in Neonates (MISN), COVID-19/SARS-CoV-2 IgG antibodies, Neonatal hyperinflammation, Intravenous immunoglobulin (IVIG), Neonatal intensive care.

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Introduction

Multisystem inflammatory syndrome in children has been increasingly recognized worldwide yet its exact pathophysiology is still incompletely understood [14,15]. Available evidence indicates that the syndrome is primarily driven by immune dysregulation following exposure to the SARS CoV 2 virus rather than direct viral tissue injury [14]. Despite ongoing investigation, the condition is widely considered a post infectious hyperinflammatory response that may follow either symptomatic or asymptomatic COVID 19 infection [14,15].

Neonatal multisystem inflammatory syndrome (MISN) represents a similarly evolving clinical

entity for which published data remain limited [1,3,13]. Current understanding is largely derived from case reports and small observational studies, and universally accepted diagnostic or treatment guidelines are still lacking [1,13]. Proposed mechanisms suggest that immune mediated inflammation, possibly triggered by maternally derived antibodies, may contribute to disease development in susceptible neonates [2,3,11,13].

Clinical presentation in affected newborns can be highly variable and frequently coincides with more common neonatal disorders, particularly sepsis and metabolic disturbances [5]. This diagnostic overlap, together with limited awareness creates a risk of

delayed recognition that may adversely influence outcomes [5]. Nevertheless, emerging reports indicate that early initiation of immunomodulatory therapies, including intravenous immunoglobulin and corticosteroids, may be associated with clinical improvement [7,8]. Given these uncertainties, systematic evaluation of clinical features, immune markers, and therapeutic responses is essential to improve diagnostic clarity and inform management strategies [1,13].

Materials and Methods

Study Design and Setting: This retrospective observational study was conducted in a tertiary care neonatal unit located in Nagpur, Maharashtra, India. The study covered a three-year period extending from year 2022 to 2024. Ethical approval was obtained from the Institutional Ethics Committee of Datta Meghe Medical College, Nagpur, prior to study initiation. Informed consent was obtained from parents or legal guardians permitting the use of clinical data and relevant photographs for academic purposes.

Eligibility criteria:

- Involvement of at least two organ systems.
- Unexplained temperature instability with reasonable exclusion of alternative diagnoses such as sepsis, birth asphyxia, or inborn errors of metabolism.
- Positive SARS CoV2 IgG antibodies in neonatal serum detected by chemiluminescent microparticle immunoassay on the Abbott Architect platform.
- IgG antibody levels >50 IU/mL .
- Presence of at least one elevated inflammatory marker (C reactive protein, procalcitonin, serum ferritin, serum lactate dehydrogenase or D dimer)
- Requirement of NICU admission

Exclusion Criteria

- Culture proven sepsis was excluded from the study.
- History of severe birth asphyxia were excluded from the study.
- Confirmed inborn metabolic disorder.
- Major congenital anomalies explaining illness

Operational Definition: For this study, suspected neonatal hyperinflammatory syndrome was defined as unexplained severe multisystem illness with raised inflammatory markers and positive SARS-CoV-2 IgG antibodies after exclusion of major alternate causes [1,13].

Data Collection: Retrospectively data was collected which included demographic data, age at symptom onset, referral status, antenatal history, organ system involvement, clinical outcome, laboratory findings,

imaging results, echocardiographic parameters, treatment interventions, timing of immunomodulatory therapy and overall patient prognosis was noted.

Laboratory Assessment: Laboratory investigations primarily assessed systemic inflammation and organ function using a panel of haematological and biochemical parameters relevant to neonatal critical care. These investigations included complete blood count, C reactive protein, procalcitonin, liver function tests, renal function tests, lactate dehydrogenase, ferritin, D dimer, and serum albumin levels [1,5,13]. SARS CoV2 IgG antibody levels were measured using the chemiluminescent microparticle immunoassay method on the Abbott Architect platform [2,3,13]. In neonates presenting with neurological manifestations such as seizures or altered sensorium, cerebrospinal fluid analysis and neuro-sonography were performed to exclude alternative diagnoses including meningitis or intracranial haemorrhage. Two-dimensional echocardiography was performed in all suspected cases. Ventricular function, valvular regurgitation, coronary changes, and pulmonary pressures were documented.

Treatment Variables: Therapeutic interventions were documented, including the timing and administration of intravenous immunoglobulin, corticosteroids, antibiotics, inotropes and anticonvulsant medications when indicated [7,8]. Details regarding respiratory support, whether invasive or non-invasive, were also recorded along with other supportive measures such as albumin supplementation or electrolyte correction. Response to treatment was assessed through continuous clinical evaluation and serial laboratory measurements to monitor changes in inflammatory markers and organ functions.

Outcome Measures: Primary outcomes included survival, need for invasive ventilation, inotrope requirement, seizure control, and time to clinical stabilization. Secondary outcomes included laboratory improvement trends and cardiac function recovery.

Statistical Method: Data were analysed descriptively using proportions and summary statistics. Due to sample size, no inferential statistical comparisons were performed.

Results

Total 48 neonates were screened for eligibility at the beginning of the study. Three infants with culture confirmed sepsis were excluded and 10 neonates who took discharged against medical advice were excluded from analysis. The final cohort comprised of 35 neonates meeting the criteria. Most developed symptoms within first 48 hours of life, with noticeable male predominance.

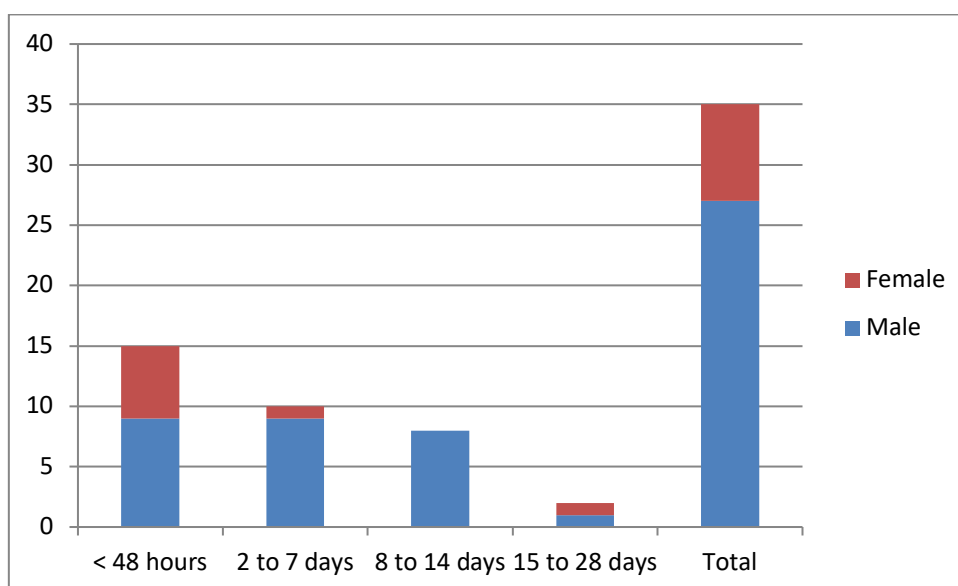


Figure 1: Age at the time of admission and gender

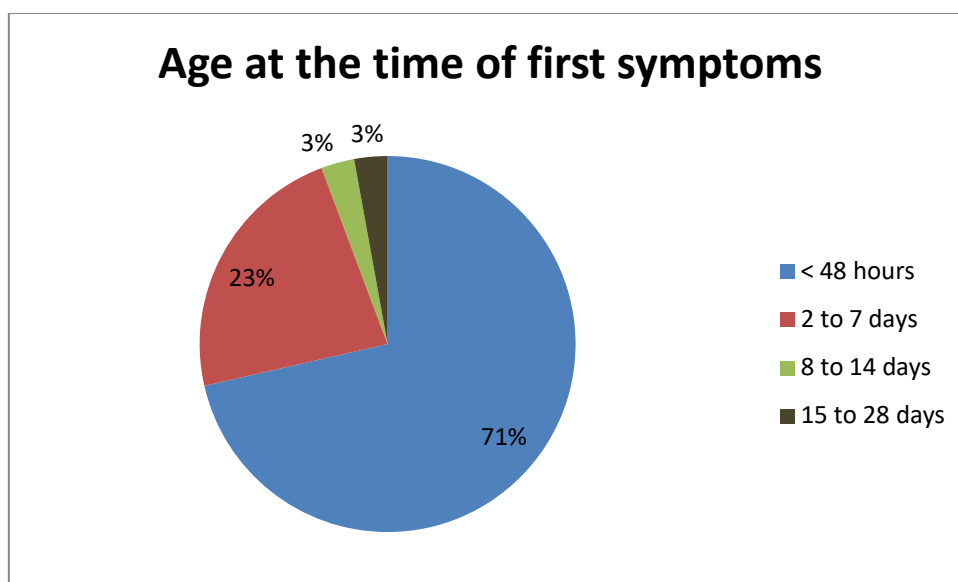


Figure 2: Age at the time of first symptoms

Most patients develop symptoms in the first 48 hours of life and almost all of them become symptomatic in first 7 days. We observed that males were affected more than female newborns (77%).

Table 1: Systems involved

Systems involved		Number of patients
Gastrointestinal system	Total	16
	Feeding intolerance	13
	Abdominal distension	10
	Ryles tube aspirate	5
	Vomiting	1
	Pathological Jaundice	2
	Hepatomegaly	2
	Splenomegaly	1
	Ascites	3
	Abdominal tenderness	1
Cardiovascular system	Total	13
	Shock	3

	Mitral valve regurgitation	4
	Cardiac dysfunction	3
	Unexplained PPHN	5
	Pericarditis	0
Central nervous system	Total	16
	Unexplained irritability	3
	Unexplained seizures	16
	Unexplained encephalopathy	2
Pulmonary system	Total	29
	Distress requiring noninvasive support like CPAP, High flow nasal cannula	15
	Oxygen requirement more than 35%	16
	Invasive ventilation	14

All neonates showed multisystem involvement consistent with working criteria and respiratory manifestations predominated at 82.8% followed by gastrointestinal and neurological features. Cardiovascular involvement was less frequent at 37.1% yet clinically more specific, while many infants required ventilatory support despite generally modest oxygen needs.

Table 2: Investigations

Blood investigations	Number of patients
Anemia	11
Thrombocytopenia	27
Thrombocytosis	0
TLC > 20000	4
TLC < 4000	7
Raised CRP	20
More than 2-fold increase in SGOT/ SGPT	2
Hyponatremia	6
Hypernatremia	1
Hypokalemia	1
Hyperkalemia	0
Raised Lactate	4
Hypoalbuminemia	9
Raised creatinine level	6
Intra cranial bleed on neuro-sonogram	4
Hypoglycemia requiring Glucose delivery rate > 8mg/kg/min	13
Consolidation on chest X ray	1
Dilated bowel loops on abdominal Xray	5
Collapse on chest X ray	2

In this study most common laboratory parameter was thrombocytopenia (77.1%). Hypoglycemia was present in significant number of patients (37.14%). Anemia was present in 31.52% of patients. CRP was not uniformly elevated, indicating that normal CRP does not exclude inflammatory presentation in this group [1,5].

Table 3: Covid antibodies titer (by CMIA, Architect)

In this study Covid IgG antibodies were very high in almost all cases.

Covid antibodies IgG (AU/ml). (<50AU/ml-negative)	Number of patients
< 100	1
100 to 500	1
> than 500	33

Table 4: Inflammatory markers

Inflammatory markers	< 300	300 to 500	>500
Serum Ferritin (ng/mL)	0	7	28
Serum LDH (U/L)	0	2	33
D DIMER (ng/mL FEU)	0	0	4

Inflammatory markers were also significantly increased in all the cases in this study [1,13]. D DIMER was done only in 4 patients which was very high.

Table 5: Complications

Complications	Number of patients
Invasive ventilation	14
Ventilator associated pneumonia	2
Need of higher antibiotic	32
Inotropes requirement	14
Steroid requirement	13
Anticonvulsant requirement	21
Need of antithrombotic agent	1
NBM for more than 48 hours	22
Retinopathy	8
Death	2

Sepsis was initially considered in most infants, leading to higher line antibiotic use in 91.4% of the cases [5]. Approximately 63% neonates required prolonged nil by mouth status, while 60% received anticonvulsants which were later stopped in many of them. Inotropic support was needed in 40% neonates. Retinopathy treatment was unexpectedly frequent, and most referrals originated from Madhya Pradesh.

Table 6: Clinical and biochemical response to IVIG:

IVIG started after admission	Clinical response			Blood report response		
	<48 hours	2 to 7 days	8 to 14 days	<48 hours	2 to 7 days	8 to 14 days
<48 hours	10	5	0	7	8	0
2 to 7 days	8	10	0	2	16	0
8 to 14 days	2	0	0	2	0	0

Among the 35 neonates, 15 received intravenous immunoglobulin within 48 hours of admission and nearly two thirds of them showed clinical improvement during the subsequent 48 hours. 18 infants treated between day two and day seven improved less frequently and showed delayed laboratory recovery. Two infants treated later improved after 48 hours. Thus, earlier therapy correlated with faster recovery. Two late presenting infants died within 36 hours of admission due to severe cardiogenic shock with encephalopathy.

Discussion

During the study period, a noticeable increase in neonates presenting with culture negative sepsis and multisystem involvement was observed. These clinical presentations were not adequately explained by conventional neonatal diagnoses, prompting consideration of an alternative inflammatory mechanism [1,5,13]. Based on clinical patterns and laboratory findings, these cases were interpreted as a possible abnormal immune response associated with maternally derived antibodies [2,3,11,13]. A large proportion of cases originated from Madhya Pradesh, accounting for approximately 74% of the evaluated cohort. This geographic predominance may reflect referral bias toward more severely ill neonates or regional variations in disease expression and recognition. Differences in circulating infectious triggers or healthcare access patterns could also plausibly contribute to this observed distribution.

Most neonates were clinically stable at birth yet developed symptoms within the first 48 hours of postnatal life. Nearly all affected neonates became symptomatic within the first 7 days, suggesting an early neonatal inflammatory process rather than late onset pathology [4,5]. Such early presentation is consistent with mechanisms involving transplacental factors rather than postnatal adaptive immune responses [2,3,13].

Male neonates were more frequently affected than females, a finding that aligns with recognized sex related differences in neonatal immune regulation [6]. Experimental and clinical observations have previously suggested that male newborns may exhibit heightened vulnerability to dysregulated inflammatory responses [4,6]. Hormonal influences and genetic factors have been proposed as potential contributors to these well documented differences in outcomes.

No single antenatal risk factor showed consistent association with disease occurrence across the cohort, indicating multifactorial influences. However, maternal hypothyroidism and pregnancy induced hypertension were documented in a notable proportion of pregnancies within the study population. These associations should be interpreted cautiously, as causal relationships cannot be established within the constraints of a descriptive study design.

Respiratory manifestations constituted the most frequent clinical presentation, followed by gastrointestinal and central nervous system

involvement. Cardiovascular abnormalities were less common yet provided more specific diagnostic indicators, particularly in the absence of structural cardiac disease. Unexplained myocardial dysfunction, valvular regurgitation, and hemodynamic instability frequently raised suspicion for an underlying inflammatory process [1,13]. Neurological manifestations, including seizures and unexplained irritability, were also prominent features in a significant subset of neonates.

Extensive evaluation was performed to exclude infectious and structural causes thereby strengthening the consideration of systemic inflammatory mechanisms [1,5,13]. Similar group of findings have been described in prior neonatal inflammatory case series and observational reports [1,3,13].

Thrombocytopenia was the most common laboratory abnormality, supporting its potential relevance as an adjunctive marker of systemic inflammation [1,5]. Elevated inflammatory markers were documented in most neonates, although the magnitude of elevation demonstrated considerable interindividual variability [1,13]. Interpretation of certain biomarkers requires caution, as reference ranges for neonates may differ substantially from older paediatric populations.

For instance, serum lactate dehydrogenase levels can be physiologically elevated in neonates, limiting specificity when considered in isolation [12]. Therefore, laboratory parameters should be integrated with clinical findings rather than used as independent diagnostic determinants. Variation in clinical severity may reflect heterogeneity in neonatal immune responses to maternally derived immunological stimuli [4,5].

Intravenous immunoglobulin was administered based on clinical judgment, with observed

improvements suggesting a potential immunomodulatory effect in selected cases [7,8]. This therapeutic approach is biologically plausible, given the established role of immunoglobulin preparations in immune mediated and inflammatory disorders [7]. Abnormal immune activation may also contribute to certain phenotypes of neonatal sepsis, potentially explaining inconsistent responses reported in earlier studies [5].

COVID IgG antibody titres were markedly elevated in most evaluated neonates, consistent with widespread maternal seropositivity following vaccination or prior exposure [2,3,13]. No clear correlation between antibody levels and disease severity was identified, indicating that antibody presence alone may not determine clinical expression [2,3,13].

Similar neonatal inflammatory phenomena have been reported in association with other maternally transmitted viral infections, including dengue and chikungunya [9,10]. It is important to recognize that elevated maternal antibodies do not uniformly result in neonatal disease highlighting the complexity of host immune interactions. The recent increase in similar clinical presentations may reflect broader shifts in infectious epidemiology or post epidemic immunological dynamics.

Thus, we propose the term “Hyper Immune Response Syndrome in Neonates” to describe this observed clinical pattern without presuming a singular aetiology. To further explore the contribution of maternal antibodies, cord blood antibody levels were evaluated in healthy neonates lacking clinical symptoms. These babies were followed up for 3 weeks and if they had any symptoms, they were excluded from the study. We found that Covid Ig G levels were significantly high even in healthy neonatal cord blood [2,3].

Table 7: Level of IgG Covid antibodies in term healthy newborn cord blood

S.N.	Gender	Level of Covid IgG antibodies (AU/ml). (< 50 AU/ml: negative)	Neonatal period
1	Male	2119.7	Normal
2	Female	2780.3	Normal
3	Female	1169.7	Normal
4	Female	569.5	Normal
5	Male	829.3	Hyperbilirubinemia
6	Male	261.6	Normal
7	Male	20564.9	Hyperbilirubinemia
8	Male	1882.2	Normal
9	Female	1636.1	Normal
10	Female	900.1	Normal

Antibody titres were frequently elevated in this comparison group, reinforcing the interpretation that antibody transfer alone is insufficient to explain disease occurrence [2,3,11]. These findings

emphasize the need for cautious interpretation of serological data within neonatal inflammatory contexts.

Emerging evidence from experimental studies suggests that neonatal immune responses may be particularly susceptible to dysregulated proinflammatory activation [4,5]. Immature adaptive regulatory mechanisms and heightened innate responsiveness may collectively cause exaggerated inflammatory cascades in neonates [4,5]. Such biological considerations may partly account for the heterogeneous clinical manifestations and variable treatment responses observed in this cohort.

Intravenous immunoglobulin may exert beneficial effects through modulation of immune pathways rather than solely by passive augmentation of host defences [7]. Re-examination of prior studies

evaluating immunoglobulin therapy in neonatal sepsis through this conceptual framework may yield valuable insights [5,7,8]. Future investigations should prioritize prospective designs, standardized diagnostic criteria, and mechanistic studies to clarify underlying immunopathological processes [1,13].

Development of a structured “Hyper immune response score” as given in this table may assist clinicians in identifying neonates who could benefit from targeted immunomodulatory interventions [1,13]. Such an approach represents a potential avenue for research and may improve diagnostic precision in neonates presenting with unexplained multisystem involvement.

Table 8: Hyper immune response score for neonates

S. No.		0	1	2
1	Gender	Female	Male	
2	System involved	1	2	> 2
3	First symptoms (normal at birth)	After 7 days	2 to 7 days	< 48 hours
4	*Unexplained cardiac dysfunction	Absent	Shock, mitral regurgitation	Cardiac dysfunction
5	*Unexplained seizures, irritability, encephalopathy	Absent	irritability	Convulsions, encephalopathy
6	Inflammatory markers (ferritin, LDH)	Normal	High (<500)	Very high/ rising
7	Thrombocytopenia	No	< 1 lakh	
8	Raised CRP	No	>5-fold increase	>100

Maximum score is 13. Any score of 8 or more is considered as significant. Unexplained by any prenatal event like birth asphyxia.

Considering the very small number of patients, we need further case control studies to find the role of immunomodulatory treatment in neonates with suspected hyper immune response.

Limitations: This study was limited by its single-centre, retrospective design, small sample size, and lack of a definitive control group, which may affect the generalizability and strength of the conclusions.

Conclusion

MISN should be considered in neonates presenting with unexplained multisystem involvement and elevated COVID-19 antibodies [1,13]. Hyper immune response syndrome should be the appropriate term for these types of clinical presentations as it is independent of level of COVID-19 antibodies [2,3,11]. Similar presentation can be there in other infective conditions like sepsis and viral infections [5,9,10]. Early recognition and treatment with IVIG can improve clinical outcomes [7,8]. Larger prospective studies are warranted to validate the proposed scoring system and treatment protocols [1,13].

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