Original Research

CLINICAL PROFILE AND OUTCOME OF CHILDREN ADMITTED WITH COVID-19 RELATED MULTISYSTEM INFLAMMATORY SYNDROME IN CHILDREN (MIS-C): A HOSPITAL-BASED RETROSPECTIVE COHORT STUDY

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ABSTRACT

Background: Multi-system Inflammatory Syndrome in Children (MIS-C) is an extremely rare but dangerous hyper-inflammatory disease linked to COVID-19 infection that results in fever, gastrointestinal and skin/mucous membrane symptoms and is also accompanied by cardiovascular issues such as shock and myocarditis.[1,2] Below are the demographic data, clinical manifestations, treatments and clinical outcomes of the children diagnosed with MIS-C who were admitted to our tertiary hospital. Materials and Methods: The medical files of all children under the age of 18 who met the World Health Organization (WHO) criteria for MIS-C admitted at our tertiary hospital between 2020–2021 were retrospectively examined. Information regarding demographics, clinical manifestations, laboratory and echocardiographic tests, treatments and clinical outcomes were collected from the files. Statistical analysis was performed with SPSS version 20. Differences among categorical variables were calculated with the chi-square (χ^2) test or Fisher's exact test and differences among continuous variables were tested with Student t-tests or Mann-Whitney U tests. Because of the number of statistical tests, a Bonferonni adjustment for multiple comparisons was made. Results: A total of forty patients (62.5% males) were evaluated (mean age 8.16 ± 2.62 years). Most patients (nearly 90%) experienced fever, and gastrointestinal symptoms (e.g., abdominal pain, vomiting, diarrhea). Commonly observed mucocutaneous signs (e.g., rash = 50%, oral changes = 45%, conjunctivitis = 47.5%) and lymphadenopathy occurred commonly. Eight patients (20%) developed shock (cardiogenic/vasoplegic), and nine patients (22.5%) developed myocarditis. The mean ages of patients developing shock and myocarditis did not differ ($p > 0.05$). However, patients with shock had higher levels of cardiac troponin and N-terminal pro b-type natriuretic peptide (NT-proBNP) than non-shock patients (mean troponin level 1.04 vs. 0.12 ng/mL, $p = 0.003$). Echocardiographic abnormalities were present in 47.5% of the patients overall; six (15%) patients had coronary artery dilation or ectasia. Eleven patients (27.5%) required inotropic support and three (7.5%) patients required mechanical ventilation. Intensive care unit admission was necessary for seventeen (42.5%) patients. Immunotherapy was universally administered: 95% received aspirin, 50% received intravenous immunoglobulin (IVIG), and 75% received corticosteroids (37.5% were given pulse dose steroids). Thirty percent of the patients received both IVIG and steroid therapy. Patients who developed shock were more likely to have been treated with IVIG (100% vs. 37.5%, $p = 0.006$) and with combined IVIG and steroid therapy (87.5% vs. 15.6%, $p < 0.001$). One patient (2.5%) died (in the shock group). At 6 weeks all 39 discharged patients were clinically stable with improvement on echocardiography; 34 completed 3- and 6-month follow-up and had no residual echocardiographic changes. One child had permanent neurological sequelae from hypoxic brain injury. **Conclusion:** In this study, most of the children who had MIS-C presented with fever, gastrointestinal and mucocutaneous symptoms, and approximately one fifth of them developed shock. Cardiac injury biomarkers were substantially higher in patients with shock, indicating cardiac damage. Therefore, we believe that when these children are identified early as having shock and myocardial dysfunction, aggressive immunomodulatory therapy should be initiated. With timely immunomodulatory treatment, immediate and 6-month outcomes were favorable for most children; however, rare severe neurological sequelae can occur.

Keywords: MIS-C, COVID-19, Children, Shock, Myocarditis, Outcome.

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INTRODUCTION

Multisystem Inflammatory Syndrome in Children (MIS-C) is an inflammatory reaction which has been associated with various infections including COVID-19, however the association with SARS-CoV-2 has become well established due to its emergence during the COVID-19 pandemic. MIS-C has been noted to occur approximately weeks following the initial SARS-CoV-2 infection and is characterized by fever, increased inflammatory markers and involvement of multiple organs systems. Both the Centers for Disease Control and Prevention (CDC) and the World Health Organization (WHO) have developed case definitions for MIS-C to assist clinicians in diagnosing this illness. While COVID-19 typically presents less severely in pediatric populations than it does in adult populations, MIS-C can be potentially life threatening and may mimic conditions such as Kawasaki disease or toxic shock syndrome.[1,2] Reports early on noted gastrointestinal symptoms, rash, conjunctivitis, and shock as common manifestations of MIS-C. A systematic review of 662 MIS-C cases noted that all 662 patients had fever and >70% of them experienced gastrointestinal symptoms, while >50% of the patients had echocardiogram findings consistent with ventricular dysfunction.[6] Of those children who were included in larger MIS-C series (i.e., Feldstein et al, NEJM), >80% of children were admitted to the ICU; 20% were mechanically ventilated and approximately 2% died [7].

Reports from India are currently limited. Jain et al. identified 23 MIS-C cases from Mumbai, with a mean age of 7.2 years; 65% of patients were admitted with shock and there was 1 fatality.[8] An additional report of 41 children with MIS-C from Kerala reported that 88% of the children required ICU admission; 20% of the children required mechanical ventilation, and 5% of the children died.[9]. With prompt recognition and medical attention, the majority of children with MIS-C survive; long-term outcomes are still under study.

The purpose of this manuscript is to describe the clinical presentation, laboratory and cardiac findings, treatment modalities, and short-term outcomes of children who were admitted to our tertiary-care hospital with MIS-C. Additionally, we evaluated the medium-term outcomes of children admitted with MIS-C. Also we compared the characteristics of children with MIS-C who presented in shock to those who did not to determine distinguishing factors.

MATERIALS AND METHODS

This retrospective study was conducted at a tertiary-care pediatric hospital. All children (<18 years) admitted with a diagnosis of MIS-C between 2020 and 2021 were included. MIS-C was defined according to WHO criteria: children with fever, elevated inflammatory markers, and ≥2 organ systems involved (e.g. gastrointestinal, cardiac, mucocutaneous), with evidence of SARS-CoV-2 infection or exposure. Data were extracted from medical records, including demographics, comorbidities, COVID-19 testing (RT-PCR or antibody), symptom onset, clinical findings, laboratory and radiologic results, echocardiography, treatments, and outcomes (need for intensive care, ventilation, inotropes, and survival). Discharged patients were followed up clinically and with echocardiography at 6 weeks, 3 months, and 6 months to assess medium-term outcomes.

Laboratory values recorded included complete blood count, inflammatory markers (C-reactive protein, erythrocyte sedimentation rate), coagulation (D-dimer), and cardiac markers (troponin I, NT-proBNP). Echocardiographic data were reviewed for ventricular function and coronary artery abnormalities. Patients were categorized into two groups: with shock (cardiogenic or vasoplegic shock requiring inotropic support) and without shock.

Statistical analysis was performed using SPSS v20 (IBM Corp). Continuous variables are reported as mean±SD or median (IQR) and compared using independent t-tests or Mann–Whitney U tests. Categorical variables are reported as counts (%) and compared by Pearson χ^2 or Fisher's exact test. A p-value <0.05 was considered significant, with Bonferroni correction applied for multiple comparisons.

RESULTS

Demographics: Forty children met MIS-C criteria. The mean age was 8.16±2.62 years (range 2–12); 25 (62.5%) were male. Nutritional status was normal in most; only 6 (15%) were underweight or overweight. Three patients (7.5%) had non-severe comorbidities (e.g. mild asthma, seizure disorder). All had laboratory evidence of prior SARS-CoV-2 exposure (RT-PCR or antibody positive). The median interval from COVID-19 exposure or illness to MIS-C presentation was ~21 days. There was no significant age difference between males and females (mean 8.5 vs 7.9 years, p>0.3).

Clinical Presentation: Fever was the most common feature, occurring in 39 patients (97.5%). Other frequent symptoms included fatigue (37.5%), abdominal pain (32.5%), vomiting (20%), and rash (50%). Oral mucosal changes (red lips/tongue or cheilitis) were seen in 18 (45%), and non-purulent conjunctivitis in 19 (47.5%). Lymphadenopathy (mostly cervical) occurred in 10 (25%). Three patients had respiratory symptoms (cough or dyspnea), and 3 had headache or irritability. The mean duration of fever before admission was 4.3±1.1 days.

Patients were divided into those with shock (n=8, 20%) and without shock (n=32, 80%). Table 1 compares their characteristics. Children with shock were slightly older (mean 7.6 vs 8.3 years) and more often female (62.5% vs 31.3%), but these differences were not statistically significant (p>0.1). Fever was equally common in both groups (p=0.78). Gastrointestinal symptoms (abdominal pain, vomiting, diarrhea) and rash/conjunctivitis occurred in all subgroups, with no significant differences. However, myocarditis (clinical or echocardiographic) was far more frequent in the shock group (75% vs 9.4%, p<0.001). Accordingly, markers of cardiac injury were higher in shock patients.

Laboratory and Echocardiographic Findings: At presentation, inflammatory markers were markedly elevated in all MIS-C patients. The mean CRP level was 3.98±3.04 mg/dL (median 3.6); ESR median was 50 mm/hr. Troponin I was profoundly higher in the shock group (mean 1.04±0.95 ng/mL) than in the no-shock group (0.12±0.17 ng/mL, p=0.003). Similarly, NT-proBNP was elevated (mean 540 pg/mL shock vs 180 pg/mL non-shock). Ferritin, D-dimer, and LDH levels were raised in most patients, but did not differ significantly between groups. The mean hemoglobin was 10.7±1.2 g/dL; 40% were anemic (Hb<11). About one-third had neutrophilia and lymphopenia. Coagulation and metabolic parameters were otherwise unremarkable except mild hyponatremia (mean Na 132±4 mEq/L) in 60%.

Echocardiography (acute phase) was abnormal in 19 (47.5%) patients. Six (15%) had coronary artery dilatation or ectasia (z-score>2), and 2 had small aneurysms (z>2.5). Myocardial dysfunction (global or segmental hypokinesia) was noted in 7 (17.5%), and 10 (25%) had pericardial effusion. All 8 shock patients had some echo abnormality (reduced left ventricular ejection fraction, pericardial effusion, or coronary changes), whereas only 11/32 non-shock patients did (p<0.001). Table 2 summarizes key laboratory and echo findings by group.

Treatment: All patients received immunomodulatory therapy. Intravenous immunoglobulin (IVIG, 2 g/kg) was given to 20 (50%) patients; corticosteroids (methylprednisolone or dexamethasone) to 30 (75%) – of these 15 (37.5%) received high-dose pulse steroids. Thirty-eight (95%) also received low-dose aspirin. Eight (20%) required a second dose of IVIG due to incomplete response. Use of IVIG was significantly higher in the shock group (100% vs 37.5%, p=0.006). Combined therapy (IVIG plus steroids) was used in 12 (30%): this included 7/8 (87.5%) shock patients versus only 5/32 (15.6%) non-shock (p<0.001). Other therapies (e.g. immunomodulators) were not used.

Outcomes: The mean hospital stay was 9.2±3.5 days for survivors. Seventeen patients (42.5%) required intensive care admission: all 8 with shock and 9 without shock (28%) (p=0.001). Inotropic support was needed in 11 (27.5%): all 8 shock patients and 3 non-shock (9.4%) (p<0.0001). Mechanical ventilation was used in 3 patients (7.5%): 2 with shock and 1 without (not statistically significant). Five patients (12.5%) had acute kidney injury requiring temporary dialysis (all in the shock group). Overall, 39 patients (97.5%) recovered and were discharged home; one child in the shock group (with severe myocarditis) died (overall mortality 2.5%).

Follow Up: All 39 discharged patients were followed up at 6 weeks; at that visit all were clinically stable and demonstrated improvement on echocardiography. At 3 months and 6 months, 34 patients (85%) completed follow-up. None of the 34 had persistent echocardiographic abnormalities at either 3-month or 6-month assessment. One child (initially complicated by encephalopathy and refractory seizures requiring mechanical ventilation) had permanent neurological sequelae: spastic quadriparesis with MRI evidence of white-matter changes consistent with hypoxic brain injury.

Shock vs. Non-Shock Comparisons: Patients with shock had significantly higher markers of myocardial injury (troponin, pro-BNP) and required more aggressive therapy and support (see Table 1–4). Shock was strongly associated with myocarditis and ventricular dysfunction (p<0.001). In contrast, inflammatory markers like CRP and ferritin tended to be higher in the shock group but did not reach significance (possibly due to small sample size). No deaths occurred among children without shock, whereas one death (12.5%) occurred in the shock group.

Tables and Figures

Table 1. Baseline Demographic and Clinical Characteristics of MIS-C Patients (Overall and by Shock Status)

Variable	Overall (n=40)	Shock (n=8)	No Shock (n=32)	p value
Age (years), mean ± SD	8.16 ± 2.62	7.6 ± 2.4	8.3 ± 2.7	0.48
Male sex, n (%)	25 (62.5)	3 (37.5)	22 (68.8)	0.11
Duration of fever (days), mean ± SD	4.3 ± 1.1	4.6 ± 1.3	4.2 ± 1.0	0.32
Fever, n (%)	39 (97.5)	8 (100)	31 (96.9)	0.78
Gastrointestinal symptoms*, n (%)	26 (65)	6 (75)	20 (62.5)	0.52

Rash, n (%)	20 (50)	5 (62.5)	15 (46.9)	0.43
Conjunctivitis, n (%)	19 (47.5)	5 (62.5)	14 (43.8)	0.34
Oral mucosal changes, n (%)	18 (45)	4 (50)	14 (43.8)	0.75
Myocarditis, n (%)	9 (22.5)	6 (75)	3 (9.4)	<0.001
ICU admission, n (%)	17 (42.5)	8 (100)	9 (28.1)	0.001
Mechanical ventilation, n (%)	3 (7.5)	2 (25)	1 (3.1)	0.06
Mortality, n (%)	1 (2.5)	1 (12.5)	0	0.18

*Includes abdominal pain, vomiting, diarrhea p values by Chi-square/Fisher's exact test or independent t-test

Table 2. Laboratory Parameters at Admission (Shock vs No Shock)

Parameter	Shock (n=8) Mean ± SD	No Shock (n=32) Mean ± SD	p value
Hemoglobin (g/dL)	10.4 ± 1.3	10.8 ± 1.1	0.29
Total leukocyte count (×10³/µL)	13.2 ± 3.5	11.8 ± 3.1	0.21
Lymphocyte count (cells/µL)	950 ± 420	1350 ± 510	0.08
CRP (mg/dL)	5.6 ± 3.8	3.3 ± 2.6	0.06
ESR (mm/hr)	58 ± 14	47 ± 18	0.11
Ferritin (ng/mL)	610 ± 240	480 ± 210	0.14
D-dimer (µg/mL)	2.9 ± 1.1	2.3 ± 0.9	0.17
Troponin I (ng/mL)	1.04 ± 0.95	0.12 ± 0.17	0.003
NT-proBNP (pg/mL)	540 ± 180	180 ± 95	<0.001
Sodium (mEq/L)	130 ± 4	133 ± 3	0.04

Independent sample t-test; Bonferroni correction applied for multiple comparisons.

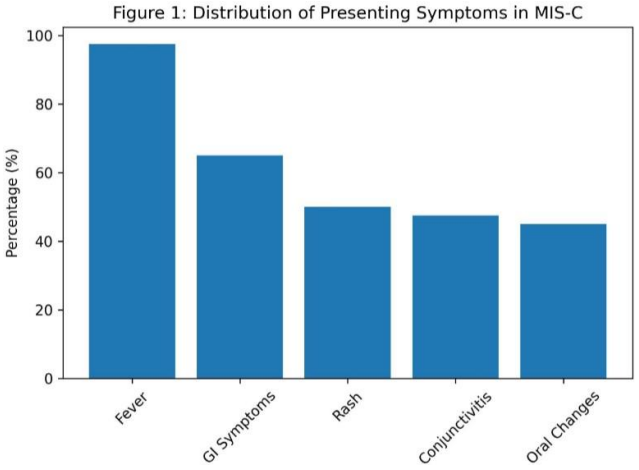
Table 3. Echocardiographic Findings in MIS-C

Echocardiographic Finding	Overall (n=40) n (%)	Shock (n=8) n (%)	No Shock (n=32) n (%)	p value
Any abnormality	19 (47.5)	8 (100)	11 (34.4)	<0.001
LV systolic dysfunction	7 (17.5)	6 (75)	1 (3.1)	<0.001
Coronary dilation (Z >2)	6 (15)	3 (37.5)	3 (9.4)	0.05
Coronary aneurysm	2 (5)	1 (12.5)	1 (3.1)	0.32
Pericardial effusion	10 (25)	5 (62.5)	5 (15.6)	0.01
Global hypokinesia	6 (15)	5 (62.5)	1 (3.1)	<0.001

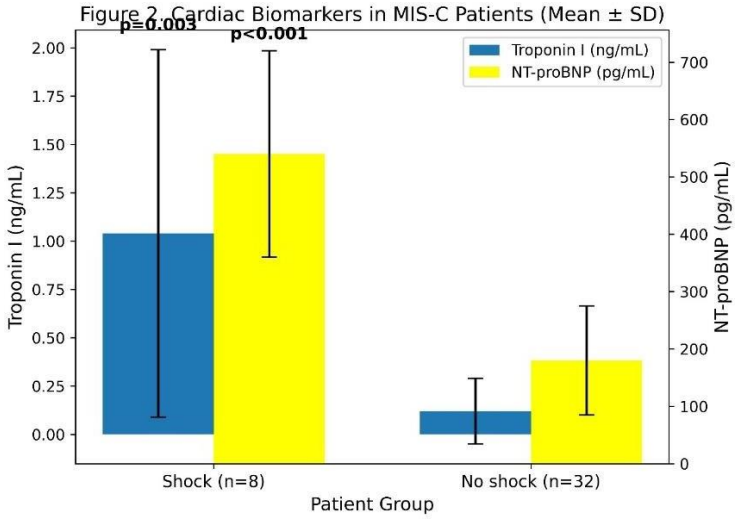
Chi-square/Fisher's exact test

Table 4. Treatment Modalities and Outcomes

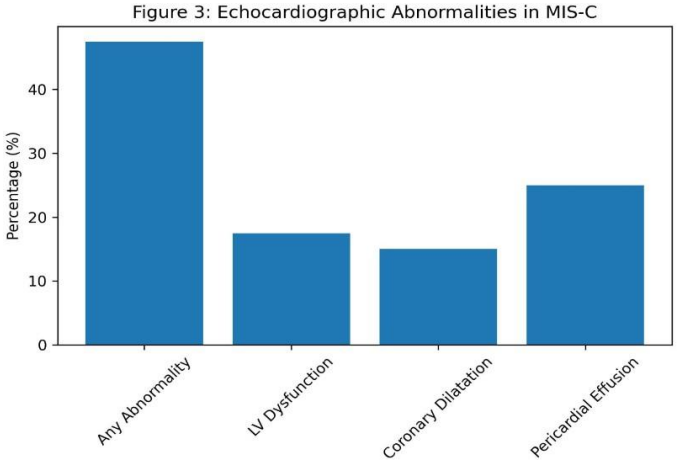
Variable	Overall (n=40) n (%)	Shock (n=8) n (%)	No Shock (n=32) n (%)	p value
IVIG	20 (50)	8 (100)	12 (37.5)	0.006
Steroids (any)	30 (75)	8 (100)	22 (68.8)	0.08
Pulse steroid	15 (37.5)	6 (75)	9 (28.1)	0.02
IVIG + Steroid combination	12 (30)	7 (87.5)	5 (15.6)	<0.001
Aspirin	38 (95)	8 (100)	30 (93.8)	0.44
Inotropic support	11 (27.5)	8 (100)	3 (9.4)	<0.001
Mechanical ventilation	3 (7.5)	2 (25)	1 (3.1)	0.06
Dialysis (AKI)	5 (12.5)	5 (62.5)	0	<0.001
Mean hospital stay (days)	9.2 ± 3.5	12.4 ± 3.2	8.3 ± 2.9	0.004
Mortality	1 (2.5)	1 (12.5)	0	0.18



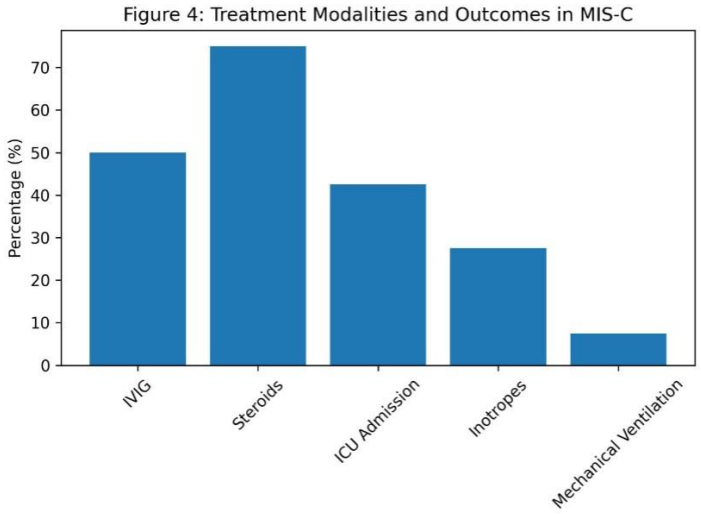
Bar diagram showing the frequency (%) of major presenting symptoms among children diagnosed with MIS-C. Fever was the most common presenting feature, followed by gastrointestinal manifestations, mucocutaneous findings, and conjunctival involvement.



Bar diagram comparing mean troponin I and NT-proBNP levels in children with MIS-C presenting with shock versus those without shock. Significantly elevated cardiac biomarkers were observed in the shock group, indicating greater myocardial involvement.



Bar diagram depicting the proportion of children demonstrating echocardiographic abnormalities during the acute phase of MIS-C. Left ventricular systolic dysfunction and coronary artery involvement were more frequent among severe cases.



Bar diagram illustrating the percentage of children receiving various treatment modalities and requiring critical care support. Use of intravenous immunoglobulin, corticosteroids, intensive care admission, and inotropic support were higher among children with severe disease.

DISCUSSION

This paper presents a cohort of 40 pediatric MIS-C patients at a single tertiary hospital. Pediatric MIS-C patients included mostly school-aged (mean 8.2 years) and predominantly males (62.5%), unlike some reports indicating MIS-C is primarily seen in females [11]. Like previous series, fever and GI symptoms (vomiting, diarrhea, abdominal pain) were the most common presenting complaints. Approximately half of the cohort experienced rash and/or conjunctivitis/oral mucous membrane changes indicative of Kawasaki-like symptoms described in other studies. Additionally, fatigue ("dull activity") and decreased oral intake were also frequent presentations in agreement with Sai et al. [11].

Only approximately 20% of the cohort initially presented in shock, which is less than the 35-40% reported in other series [12]. Children who presented in shock were somewhat older and more likely to be female, however this difference was not statistically significant. Furthermore, shock patients exhibited significantly increased levels of cardiac biomarkers: troponin I levels in shock patients were >9 times greater than in non-shock patients ($p = 0.003$). These results are consistent with myocardial injury being the underlying mechanism responsible for shock in MIS-C patients, as previously shown by Jain et al., and Gupta et al. (40% shock, higher IL-6, D-dimer, troponin in shock patients) [8, 12]. Additionally, myocarditis (either clinical or echocardiogram-based) was demonstrated in 75% of shock patients and only 9% of non-shock patients ($p < 0.001$). Shock patients required inotrope support and ICU admission. The data illustrate the necessity for cardiac assessment in MIS-C patients.

Abnormalities detected by echocardiography were common: almost half of all patients demonstrated pathology. Similar to other Indian studies (20%), coronary artery dilation or ectasia was evident in 15% of patients [11]. There were fewer coronary aneurysms noted in this cohort than expected given the high prevalence of myocardial injury. The degree of left ventricular dysfunction was significant in the shock group: 7 of 8 patients demonstrated moderate to severe dysfunction, while no patients without shock demonstrated such dysfunction ($p < 0.001$). Therefore, it is understandable that shock patients required additional support.[3,4,5]

The laboratory findings in this cohort represented a hyperinflammatory response, including universal elevations in CRP, ESR, ferritin, and D-dimer. However, while there was a trend towards greater elevation of CRP in shock patients (mean 5.6 vs 3.3 mg/dl, $p \approx 0.06$), troponin and NT-proBNP were significantly higher in shock patients. Additionally, lymphopenia and neutrophilia were prevalent, but failed to differentiate between shock and non-shock patients. Furthermore, procalcitonin, which has been reported by others to be predictive of shock [12], was not uniformly measured in our cohort.

All patients were treated according to established MIS-C treatment guidelines, which included steroid administration to all patients and IVIG administration to half of them. The use of IVIG was significantly higher in the shock group (100% vs 38%) and combined IVIG + steroid was administered to 88% of shock patients compared to 16% of non-shock patients ($p < 0.001$), demonstrating a more aggressive approach to treating the severe cases. These treatments are consistent with other reports (for example, the combination of steroids + IVIG was used in 85% of Indian MIS-C cases) [9]. Additionally, aspirin was

utilized as an anti-platelet agent in every patient. Biologic agents (such as IL-1 or IL-6 inhibitors) were not utilized in this cohort.

Outcomes in this cohort were largely favorable. Only one child (2.5%) in this cohort expired, which is consistent with low mortality rates in many MIS-C cohorts [11]. This deceased patient suffered from refractory shock and cardiac failure despite receiving maximum therapy. The survival rate (97.5%) in this cohort is equivalent to the 98.3% reported by Ahmed et al. [6] and the 98.7% (77/78) in the South India series [10]. A total of 27.5% of the patients in this cohort required inotropic support, which is consistent with the approximately 50% rate of inotropic requirement in other series. Additionally, mechanical ventilation (7.5%) and dialysis (12.5%) were necessary for a minority of patients, who were typically those with the most severe initial presentations.

Limitations to this study include its retrospective design and single center nature, both of which may reduce the generalizability of this data. Additionally, the number of patients in each category (especially the shock subgroup), is limited, which reduces the statistical power to detect some differences. Lastly, long term follow up of cardiac sequelae was not part of the scope of this study. Nonetheless, this study adds to the growing body of literature regarding MIS-C in India.

CONCLUSION

Children with MIS-C in our cohort typically presented several weeks after COVID-19 exposure with high fever, gastrointestinal symptoms, and mucocutaneous signs. A subset (20%) developed shock with significant myocardial involvement, marked by elevated troponin and ventricular dysfunction. Treatment with steroids and IVIG led to recovery in nearly all cases. The overall mortality was low (2.5%), reflecting that with early recognition and aggressive management, even severe MIS-C can be survived. In this cohort, medium-term outcomes at 6 months were excellent with resolution of echocardiographic abnormalities in those followed; however, rare but severe sequelae (post-MIS-C hypoxic brain injury) may be permanent and warrant long-term surveillance. Close cardiac monitoring and follow-up are advisable given the frequency of coronary and myocardial involvement in MIS-C.

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