

Case Report on a Young Patient with Multisystem Inflammatory Syndrome in Adult (MIS-A)

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Abstract

Background: A healthy 22 year old man presented with clinical and laboratory characteristics of multisystem inflammatory syndrome resembling MIS-C with cardiac dysfunction requiring intensive care support that included mechanical ventilation and vasopressor support. **Aim:** To present a rare case of Multisystem Inflammatory Syndrome in Adult (MIS-A). **Case Report:** The patient presented with 5 days of fever, profuse sweating, vomiting and loose stools. Patient was otherwise healthy with no prior medical illness and was never a diagnosed case of COVID 19. At the time of admission, patient was found to be in shock with leukocytosis, conjunctivitis, acute kidney injury, cardiac dysfunction, acute hepatitis with both COVID antigen and RT-PCR negative. The patient required ICU admission with ventilatory and vasopressor support. MIS-A secondary to COVID19 was suspected on the basis of his clinical presentation and a positive COVID Antibody report. Patient improved clinically with IV steroids, IVIG, heparin, and Critical Care management and support. **Conclusion:** A small portion of patients present with late onset complications following asymptomatic COVID 19 infection. This Stage includes Multiorgan dysfunction in the setting of severe inflammation. MIS-C has become a recognized syndrome whereas a parallel syndrome in adults has not been well defined. So MIS-A should be considered in adults presenting with features compatible with MIS-C like illness, including shock and cardiac dysfunction.

Keywords: Multisystem Inflammatory Syndrome in Adult (MIS-A); COVID19; Hyper inflammatory; Extra pulmonary organ dysfunction; SARS COV-2 Antibody; IVIG; Methylprednisolone; MIS-C; Kawasaki disease; Shock; Anti coagulation

Introduction

Multisystem Inflammatory Disease in Adults (MIS-A) is a rare but serious condition seen in adults associated with Covid-19 infection, characterized by a hyper inflammatory response and multi organ dysfunction. Although hyperinflammation and extrapulmonary organ dysfunction have been reported in hospitalised adults with severe Covid-19 infections, these conditions are generally accompanied by respiratory failure. However in patients with MIS-C like symptoms have minimal respiratory symptoms, which often lack intrinsic respiratory disease.^[1]

Case Report

A 22 year old healthy male patient presented with fever, profuse sweating, and passage of loose stool since 5 days, vomiting since 2 days. He had recent history of travel.

He was not on any regular medication and had no allergy history. The patient had no preceding respiratory symptoms.

Patient was hemodynamically unstable; Spo2 was 100% on 4 L oxygen by mask.

Skin rash and petechiae present over chest and limbs, with conjunctival congestion.

Laboratory parameters shown in Table 1

Anti SARS COV-2 Antibody was positive.

All cultures were negative.

CT thorax showed very minimal bibasilar infiltrates with septal thickening.

Patient was immediately resuscitated according to the surviving sepsis guidelines and started on high vasopressor support and corticosteroids.

Patient had a seizure on Day 1, following which he was put on invasive ventilation for fall in GCS and oxygen saturation.

Patient improved clinically and weaned off from ventilatory support gradually and extubated on Day 3.

Due to the concern for inflammatory multisystem organ involvement similar to that seen in MIS-C and risk of progression to more florid cardiac involvement, a risk benefit discussion was held with the patient's attendants regarding treatment with IVIG including potential risk of hypercoagulability.^[2,3]

IVIG was started @2 gm/kg on Day 2 and was given over 3 days. IV methylprednisolone and Aspirin was also started based

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Table 1: Laboratory data.

Parameter	D ₀	D ₁	D ₂	D ₃	D ₄	D ₅	D ₆	D ₇	D ₁₂	D ₁₄
TC/Cumm	17100	23900	26500			14600		19400		
CRP (mg/L)	520	531.5				98.4		63.5		6.3
Procalcitonin (ng/ml)	6.81	3.63				1.06		1.23		
FERRITIN (ng/ml)	>1000		>1000				1000	1000		
D-dimer (ng/ml FEU)	2044		3852	3634	3060		5000	5000	4551	2560
AST(U/L)	5840		3649		726		274			
ALT(U/L)	3704		2040		1734		774			
TROP-I (ng/ml)	51.1		26.5		3.4					
NT-proBNP (pg/ml)	>30000		>30000		12200					1920

TC: Total Count, CRP: C Reactive Protein, AST: Aspartate Aminotransferase, ALT: Alanine Aminotransferase, Trop I: Troponin I, NT pro BNP: N Terminal Pro hormone B type Natriuretic Peptide

on the treatment courses suggested for MIS-C or KAWASAKI Disease. [4-6]

His hemodynamics status improved gradually over the next few days and the vasopressor support was gradually tapered and stopped on Day 5.

Point of care ECHO done on Day 1 showed global hypokinesia, with LVEF 35%-40%. Repeat ECHO on Day 7 showed LVEF 50%-55%, with preserved LV systolic function.

There was a downtrend seen in his inflammatory lab markers from day 4 with improvement in his clinical symptoms and was discharged on day 15.

Discussion

Multisystem Inflammatory Syndrome in Children (MIS-C) has become a recognized syndrome, whereas a parallel syndrome in adults has not been well defined. MIS-C was reported in April 2020 as a hyper inflammatory syndrome with variable features of Kawasaki disease. [1]

The pathophysiology of MIS in both children and adults is currently unknown. SARS-CoV-2 has been identified in multiple organs including the heart, liver, brain, kidneys and gastrointestinal tract. The proposed mechanisms for extrapulmonary dysfunction in COVID 19 include endothelial damage, thromboinflammation, dysregulated immune responses and dysregulation of the renin-angiotensin-aldosterone system. [2] The interval between infection and development of MIS-A is unclear, adding to the uncertainty regarding whether MIS-A represents a manifestation of acute infection or an entirely postacute phenomenon. [7,8] In patients who reported typical COVID-19 symptoms before MIS-A onset, MIS-A was experienced approximately 2-5 weeks later. [3,9]

Patients who fulfilled the working MIS-A case definition, included the following five criteria: [3]

- Severe illness requiring hospitalization, age \geq 21 years
- Positive test result for current or previous SARS-CoV-2 infection (nucleic acid, antigen, or antibody) during admission or in the previous 12 weeks

- Severe dysfunction of one or more extra pulmonary organ systems (e.g. hypotension or shock, cardiac dysfunction, arterial/venous thrombosis/thromboembolism, or acute liver injury)
- Laboratory evidence of severe inflammation (e.g. elevated CRP, ferritin, D-dimer, or interleukin-6) and
- Absence of severe respiratory illness (to exclude patients in which inflammation and organ dysfunction might be attributable simply to tissue hypoxia). [3]

Patients with mild respiratory symptoms who met these criteria were also included. Patients were excluded if alternative diagnoses such as bacterial sepsis were identified.

Three published case series were identified describing adult patients with manifestation consistent with MIS-A. [7-9] One series describes seven previous healthy, young adult men aged 20-42 yrs, who experienced mixed cardiogenic and vasoplegic shock along with high SARS CoV-2 IgG antibody titers. [7] All had markedly elevated inflammatory markers and required inotropes or vasopressors and 3 required intra-aortic balloon pumps. All were treated with corticosteroids and therapeutic anti coagulation.

The second case series described 2 patients aged 21 and 50 yrs with positive SARSCoV-2 test, who came to medical attention because of large vessel strokes. [8]

The third case series describes the pathologic findings of endothelialitis and complement deposition in the vessels of 2 patients with illness resembling MIS-A. [9]

Hékimian et al presented few cases which were an important addition to descriptions of MIS-A. [10]

The clinical presentation in some patients with severe COVID-19 could overlap with MIS-A, but the pathophysiology may be different. Dual SARS-CoV-2 RT-PCR and antibody testing, and a thorough history can provide supportive evidence for suspected MIS-A. Several features of MIS-A require urgent attention.

First, a lack of clear guidance regarding diagnosis highlights the need to establish MIS-A case definitions and testing algorithms.

Although antibody testing is strongly recommended by the Infectious Diseases Society of America in the setting of MIS-C, the role of antibody testing for cases of suspected MIS-A needs to be defined. [11]

Second, optimal treatment is still unclear given the rarity of this syndrome. IV immunoglobulin, steroids, and other immunomodulatory agents have been used to treat suspected cases of MIS-A.

Third, the potential for chronic sequelae is not yet known and could affect long-term follow-up care and monitoring.

Finally, further studies on the immunopathogenesis of this syndrome are needed. If MIS is postinfectious or antibody mediated, there could be important implications for vaccine safety.

MIS-A is likely a rare but important syndrome that can be difficult to distinguish from severe COVID-19, particularly in older patients with multiple comorbidities and needs further research in the adult population to understand the full range of clinical manifestations and to identify potential opportunities for targeted treatment of inflammatory processes.

Conclusion

A small portion of patients present with late onset complications following asymptomatic COVID 19 infection. This Stage includes Multiorgan dysfunction in the setting of severe inflammation. MIS-C has become a recognized syndrome whereas a parallel syndrome in adults has not been well defined. So MIS-A should be considered in adults presenting with features compatible with MIS-C like illness, including shock and cardiac dysfunction.

Competing Interests

The authors report no competing (commercial/academic) interests.

References

- Riphagen S, Gomez X, Gonzalez-Martinez C, Wilkinson N, Theocharis P. Hyperinflammatory shock in children during COVID-19 pandemic. *The Lancet*. 2020;395:1607-1608.
- Gupta A, Madhavan MV, Sehgal K, Nair N, Mahajan S, Sehrawat TS, et al. Extrapulmonary manifestations of COVID-19. *Nat Med*. 2020;26:1017-1032.
- Morris SB, Schwartz NG, Patel P, Abbo L, Beauchamps L, Balan S, et al. Case series of multisystem inflammatory syndrome in adults associated with SARS-CoV-2 infection—United Kingdom and United States, March–August 2020. *Morb Mortal Wkly Rep*. 2020;69:1450.
- Hennon TR, Penque MD, Abdul-Aziz R, Alibrahim OS, McGreevy MB, Prout AJ, et al. COVID-19 associated Multisystem Inflammatory Syndrome in Children (MIS-C) guidelines; a Western New York approach. *Progr Pediatr Cardiol*. 2020.
- Dallaire F, Fortier-Morissette Z, Blais S, Dhanrajani A, Basodan D, Renaud C, et al. Aspirin dose and prevention of coronary abnormalities in Kawasaki disease. *Pediatrics*. 2017;139.
- Platt B, Belarski E, Manaloor J, Ofner S, Carroll AE, John CC, et al. Comparison of risk of recrudescence fever in children with Kawasaki disease treated with intravenous immunoglobulin and low-dose vs high-dose aspirin. *JAMA Netw Open*. 2020;3:e1918565-.
- Chau VQ, Giustino G, Mahmood K, Oliveros E, Neibart E, Oloomi M, et al. Cardiogenic shock and hyperinflammatory syndrome in young males with COVID-19. *Circ Heart Fail*. 2020;13:e007485.
- Chau VQ, Giustino G, Mahmood K, Oliveros E, Neibart E, Oloomi M, et al. Cardiogenic shock and hyperinflammatory syndrome in young males with COVID-19. *Circ Heart Fail*. 2020;13:e007485.
- Magro C, Mulvey JJ, Berlin D, Nuovo G, Salvatore S, Harp J, et al. Complement associated microvascular injury and thrombosis in the pathogenesis of severe COVID-19 infection: a report of five cases. *Transl Res*. 2020;220:1-3.
- Tenforde MW, Morris SB. Multisystem inflammatory syndrome in adults: coming into focus. *Chest*. 2021;159:471-472.
- Hanson KE, Caliendo AM, Arias CA, Englund JA, Lee MJ, Loeb M, et al. Infectious diseases society of America guidelines on the diagnosis of COVID-19. *Clin Infect Dis*. 2020.