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Case Report

A young adult with multisystem inflammatory syndrome (MIS-A) following COVID-19 infection: A case report

Suresh Ramasubban[1], Chadrashish Chakravarty[1], Lawni Goswami[1], Subhajit Sen[1,*], Debasis Rout[1], Anupam Mohapatra[1]

[1] *Dept. of Respiratory Medicine and Critical Care, Apollo Multispeciality Hospital, Kolkata, West Bengal, India*

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ABSTRACT

Recent reports have described a secondary Multi system Inflammatory Syndrome in adult (MIS-A)and its temporal association with COVID-19 infection. Here, we report the case of a 25-year-old male who presented with shock and multi organ failure. Three-week prior to present admission he developed an Influenza like illness, secondary to SARS-CoV2 infection as detected by a reverse-transcription polymerase chain reaction (RT-PCR). Three weeks later, patient had recurrence of fever associated with throat pain, neck swelling, and a rash followed by shock and multiorgan failure. Laboratory work revealed elevated inflammatory markers. His throat swab for SARS-CoV-2 by RT-PCR was negative and SARS-CoV-2 IgG antibody was reactive.In view of presentation like multi-inflammatory syndrome in children (MIS-C) a probable diagnosis of MIS-A was made. He was successfully treated with intravenous Immunoglobulin and pulse dose Methylprednisolone. This case highlights the need to remain vigilant for multisystem inflammatory syndromes in the COVID-19 affected patients.[

1. Introduction

As the coronavirus 2019 pandemic has spread across the globe, new complications in patients of covid-19 infection have surfaced. Multisystem inflammatory syndrome in adults(MIS-A)is a severe complication that has been seen in patients with SARS COV 2 infection across the globe. Case reports has been published in different literatures regarding task of 2 and recently the CDC has come up with a new Diagnostic criteria for multisystem inflammatory syndrome in adults.(https://www.cdc.gov/mis/mis-a/hcp.html#) In this case report represent one such case of multisystem inflammatory syndrome in adults who has presented to to us with multisystem complaints that was eventually attributed to MIS A.

2. Case Presentation

A 25-year-old male was admitted to our Intensive care unit (ICU) with Shock. He was well three-week prior to admission when he developed an Influenza like illness, secondary to SARS-CoV-2 infection as detected by a reverse-transcription polymerase chain reaction (RT-PCR). He had an unremarkable course & illness was short lived and did not require any therapy. Three weeks later, patient had recurrence of fever, with chills and rigors and associated with throat pain and painful neck swelling and a faint rash over his neck and upper chest and was admitted to a hospital. During his stay he worsened and developed altered mental status and hypotension, and transferred to ICU.

On presentation he was febrile, tachycardic, tachypnoeic and had profound hypotension requiring high dose norepinephrine at 1mcg/kg/min as a vasopressor. His oxygen saturations were maintained at 95% on room air.

* *Corresponding author*.
E-mail address: bingos.sen@gmail.com (S. Sen).

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He was awake, not fully alert and had periods of agitation with frequent non purposeful movements. He appeared toxic and had tender bilateral cervical lymphadenopathy, tongue was red with white spots. Conjunctival hyperemiaand icterus were also noted. His chest was clear to auscultation and there were no murmurs or gallop rhythm and no organomegaly was noted.

Laboratory results showed leucocytosis and elevated inflammatory markers like C-reactive protein (CRP), ferritin and D-dimer (Table 1). His throat swab for SARS-CoV2 by RT-PCR was negative and SARS-CoV2 IgG antibody was reactive. Blood and urine cultures were negative. Serology for malaria, dengue, and typhoid was also negative. Chest X ray and High-resolution CT scan (HRCT) of the chest without contrast were unremarkable. A fine needle aspirate cytology (FNAC) from the left submandibular lymphnode demonstrated reactive hyperplasia. A magnetic resonance imaging (MRI) of the brain revealed cytotoxic lesions in splenium of corpus callosum and mild meningeal enhancement (Figure 1). Cerebrospinal fluid analysis was non-contributory. He also had features of acute kidney injury (AKI) and jaundice with transaminitis.

In the ICU, his blood pressure failed to stabilize with volume resuscitation. Invasive hemodynamic monitoring with a pulse contour analysis of the stroke volume (SV) revealed SV of 70 ml and a cardiac index of 4.2 lit/min/m2 with a stroke volume variation of 7%. His vasopressors were optimized, and he was initiated on vasopressin and stress dose of hydrocortisone. He developed atrial fibrillation with rapid ventricular response. His troponins and N-terminal brain natriuretic peptide levels were also elevated. He developed pulmonary oedema and was intubated and ventilated to maintain adequate oxygenation. He was cardioverted to attain hemodynamic stability and after cardioversion sinus rhythm was maintained with Amiodarone infusion. His laboratory parameters showed worsening of AKI, transaminitis and a new thrombocytopenia with elevated prothrombin time. His blood cultures repeatedly remained negative and a work-up for vasculitis with ANA, dsDNA, complement levels and ANCA levels were all normal. Serology for EpsteinBarr Virus (EBV IgM) and a PCR for Cytomegalovirus were also negative.

In view of progressive multiorgan failure with elevated inflammatory markers, evidence of recent COVID-19 infection, and a presentation like multi-inflammatory syndrome in children (MIS-C) a probable diagnosis of MIS-A was made. A risk benefit decision was held with the patient family regarding treatment with intravenous immunoglobulins (IvIG). He was treated with IvIG (2mg/kg) split over three days to decrease risk of thrombosis.[1,2] The stress dose hydrocortisone was converted to pulse dose methylprednisolone once the course of IvIG was completed as patient was still in shock. The steroids were tapered to oral prednisolone at a dose of 1mg/kg after the pulse dose steroids. His haemodynamic and cardiac function improved by day 9 and he was weaned off ventilator subsequently over next three days.

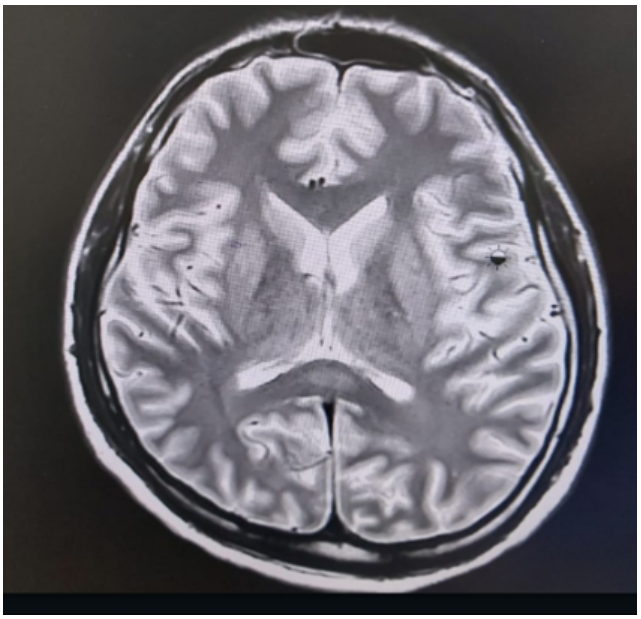

Fig. 1: MRI Brain (Cytotoxic lesions in the splenium of corpus callosum probably secondary to SARS COVID-19 with mild meningeal enhancement)

3. Discussion

Recently, reports of a Kawasaki like disease[3] has been seen in children with COVID-19. A multi-inflammatory syndrome in children (MIS-C) has been defined using the following criteria: a) occurring in individuals less than 21 years of age presenting with fever; b) laboratory evidence of inflammation; c) evidence of clinically severe infection leading to hospitalization with greater than 2 organ system involvement; d) no other plausible diagnosis and e) a recent SARS-CoV2 infection confirmed by RT-PCR.[4–6] Similarly, adults with current or previous SARS-CoV2 infection with a hyperinflammatory syndrome resembling MIS-C, have been reported. The centers for disease control (CDC) has developed a working definition of multi-inflammatory syndrome in adults (MIS-A) which includes the following criteria: a) Severe illness in a person greater than 21years of age requiring hospitalization; b) a positive test result for current or previous SARS-CoV2 infection during admission or in the previous 12 weeks; c) Severe dysfunction of one or more extrapulmonary organ systems (hypotension or shock, cardiac dysfunction, arterial or venous thrombosis or acute kidney injury); d) laboratory evidence of severe inflammation.[7] Our patient as described in the case met the criteria for MIS-A. MIS-A has been reported only in

Table 1: Laboratory Datas:

	On Admission	On Discharge	
White blood cells (/cumm)	29.1	15.6	4000-10000
Differential Count (%) :-			
Neutrophils	90	84	40- 80
Lymphocytes	07	11	20- 40
Monocytes	02	04	02- 10
Eosinophils	01	01	01- 06
Hemoglobin (g/dl)	11.1	9.2	13- 17
Platelets (lakh/cumm)	1.40	3.93	1.5- 4
Sodium (mEq/L)	139	130	136- 145
Potassium (mEq/L)	3.5	4.5	3.5- 5.2
Calcium (mEq/L)	7.7	7.8	8.6- 10.2
Chloride (mEq/L)	102	115	96- 106
Carbon Dioxide	38	33	35- 45
Urea (mg/dl)	85	33	17- 49
Creatinine (mg/dl)	3.7	0.7	0.8- 1.3
Alanine Transferase (U/L)	26	106	10- 40
Aspartate Transferase (U/L)	28	226	10- 42
Bilirubin (mg/dl):-			
Total	4.9	1.2	Upto 1
Direct	3.3	0.3	Upto 0.2
Serum Ammonia()	194	42	< 90
Serum Procalcitonin (ng/ml)	0.03	<0.03	< 0.5
B Natriuretic Peptide (pg/ml)	51000	6962	< 300
C Reactive Protein (mg/dl)	85	1.9	< 0.5
Ferritin (ng/ml)	6250	2224	20- 250
Creatinine Phosphokinase (U/L)	646	12454	39-308
Lactate Dehydrogenase (U/L)	354	265	100-190
D Dimer (ng/ml)	2265	1932	Upto 500
Fibrinogen (mg/dl)	870	383	200-400
INR	1.15	NA	< 1.28

small case reports and a few case series, wherein the age range of the affected patients was between 21 and 50 years, majority were females and predominantly non-Caucasians. A majority had no comorbidities and obesity was the major risk factor. All patients have cardiac abnormalities such as arrhythmias, elevated troponin levels or echocardiographic evidence of left or right ventricular dysfunction. All patients had markedly elevated markers of inflammation (CRP and ferritin) and markers of coagulopathy (D-dimer). In the case series, majority of the patients have recent COVID-19 infection suggesting a post-acute phenomenon. Most of the patients were treated with intravenous immunoglobulins and steroids and most of them made good recovery despite being critically ill.[8]

4. Conclusions

This case report highlights the need for awareness of the possibility of MIS-A in adults. An acute febrile illness in an adult with multiple organ dysfunction should prompt a clinician to consider MIS-A, which is mostly a post-acute phenomenon to COVID-19. The relevance of recognition of MIS-A is due to the change in management that the diagnosis brings about. However, treatment protocols at present are anecdotal and need evaluation.

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6. Conflict of Interest

The authors declare that there is no conflict of interest.

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None.

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Author biography

Suresh Ramasubban, Senior Consultant Intensivist https://orcid.org/0000-0002-9874-0968

Chadrashish Chakravarty, Senior Consultant Intensivist

Lawni Goswami, Consultant Intensivist

Subhajit Sen, Post Doctoral Trainee https://orcid.org/0000-0001-8226-6678

Debasis Rout, Post Doctoral Trainee (Critical Care Medicine) https://orcid.org/0000-0001-8391-1827

Anupam Mohapatra, Consultant Intensivist

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