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Post-COVID-19 Multisystem Inflammatory Syndrome in a Young Adult: A Case Report

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Abstract

Background: Post-COVID-19 infection syndrome has been described in children, known as Multisystem Inflammatory Syndrome in Children temporally associated with SARS-CoV-2. Clinical signs and symptoms include conjunctivitis, rash, fever, hypotension, gastrointestinal symptoms, myocarditis and coronary abnormalities. Most case reports were described in children, and there are very few case reports of Post-COVID-19 Multisystem Inflammatory Syndrome in Adults (MIS-A).

Case report: We describe a case of a healthy 21-years-old man with symptoms of fever, myalgia and loss of taste, followed by severe dyspnea and hypoxemia. Chest computed tomography scan revealed extensive bilateral lung infiltrates. Shock and acute kidney injury followed. Echocardiography showed severe left ventricular dysfunction. The patient was intubated and ventilated. Due to cardiogenic shock and hypoxemia, veno-arterial Extracorporeal Membrane Oxygenation (ECMO) therapy was initiated. A diagnosis of multisystem inflammatory syndrome due to past COVID infection was made. The patient received methylprednisolone pulse therapy and intravenous immunoglobulin therapy. His condition improved quickly, and the patient was weaned off ECMO support.

Conclusion: We presented a case of severe MIS-A in a young adult, with recent asymptomatic COVID-19 infection, manifested as cardiogenic shock and pulmonary edema. As MIS-A presentation may mimic other pathologic conditions; high index of suspicion is required.

Keywords: MIS-A • COVID-19 • Cardiogenic shock • Pulmonary edema

Introduction

Post COVID-19 infection, a Kawasaki-like syndrome has been described in children and adolescents. This syndrome is also known as Multisystem Inflammatory Syndrome in Children (MIS-C) or Pediatric Multisystem Inflammatory Syndrome temporally associated with SARS-CoV-2 (PIMS-TS) [1]. In the United Kingdom, the diagnostic criteria for PIMS-TS include organ dysfunction, evidence of recent COVID-19 infection, as well as exclusion of any other infectious disease [2,3]. In the United States of America, the diagnosis is based on clinical presentation, the evidence of severe illness with multisystem (at least two) organ involvement, a positive test for current or recent SARS-CoV-2 infection or COVID exposure to SARS-CoV-2 within the past 4 weeks as well as the absence other an alternative etiology [4]. Clinical signs and symptoms include bilateral non-purulent conjunctivitis, rash, and fever, hypotension with or without shock, gastrointestinal symptoms (diarrhea, vomiting, abdominal pain), myocarditis, pericarditis, valvulitis or coronary abnormalities. In addition, the following laboratory variables are required: elevated inflammatory markers (C-reactive protein, ferritin, D-dimer and interleukin-6) for at least 3 days, as well as elevated troponin levels and an abnormal coagulation profile [5]. While most case reports or case series of Post-COVID-19 multisystem inflammatory syndrome were described in children or adolescents, there are very few case reports of Post-COVID-19 multisystem inflammatory syndrome in adults (MIS-A). We describe a case of 21-year-old man with severe MIS-A, admitted to the Intensive Care Unit (ICU) in our hospital.

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Case Report

A healthy 21-years-old man received his first dose of COVID-19 vaccine on February 24th 2021. On the following day he developed symptoms of high fever, myalgia, fatigue, sore throat, dry cough and loss of taste. Due to worsening of symptoms he presented to the emergency department on March 1st, 2021. His temperature was 39° Celsius. However, clinical examination revealed no pathology. Arterial blood oxygenation and chest x-ray were both normal; except for mildly elevated liver enzymes all laboratory investigations including a COVID-19 test were not pathological. He was discharged home in good clinical condition. He returned to the emergency department on March 3rd, 2021 due to a fever of 40° Celsius, extreme fatigue tachypnea and dyspnea. Laboratory parameters revealed increased inflammatory indices: leukocytosis of 13.65 k/microl with left shift and lymphopenia, increased liver enzymes with Aspartate Aminotransferase (AST) of 62 U/l and Alanine Aminotransferase (ALT) of 76 U/l, and increased C-Reactive Protein (CRP) of 20 mg/dl. Chest x-ray and arterial blood gas were normal. He was admitted to an internal medicine department. On further and specific questioning, the patient revealed that, in the past 8 weeks, there were several cases of COVID-19 in his family. A second COVID-19 test was negative.

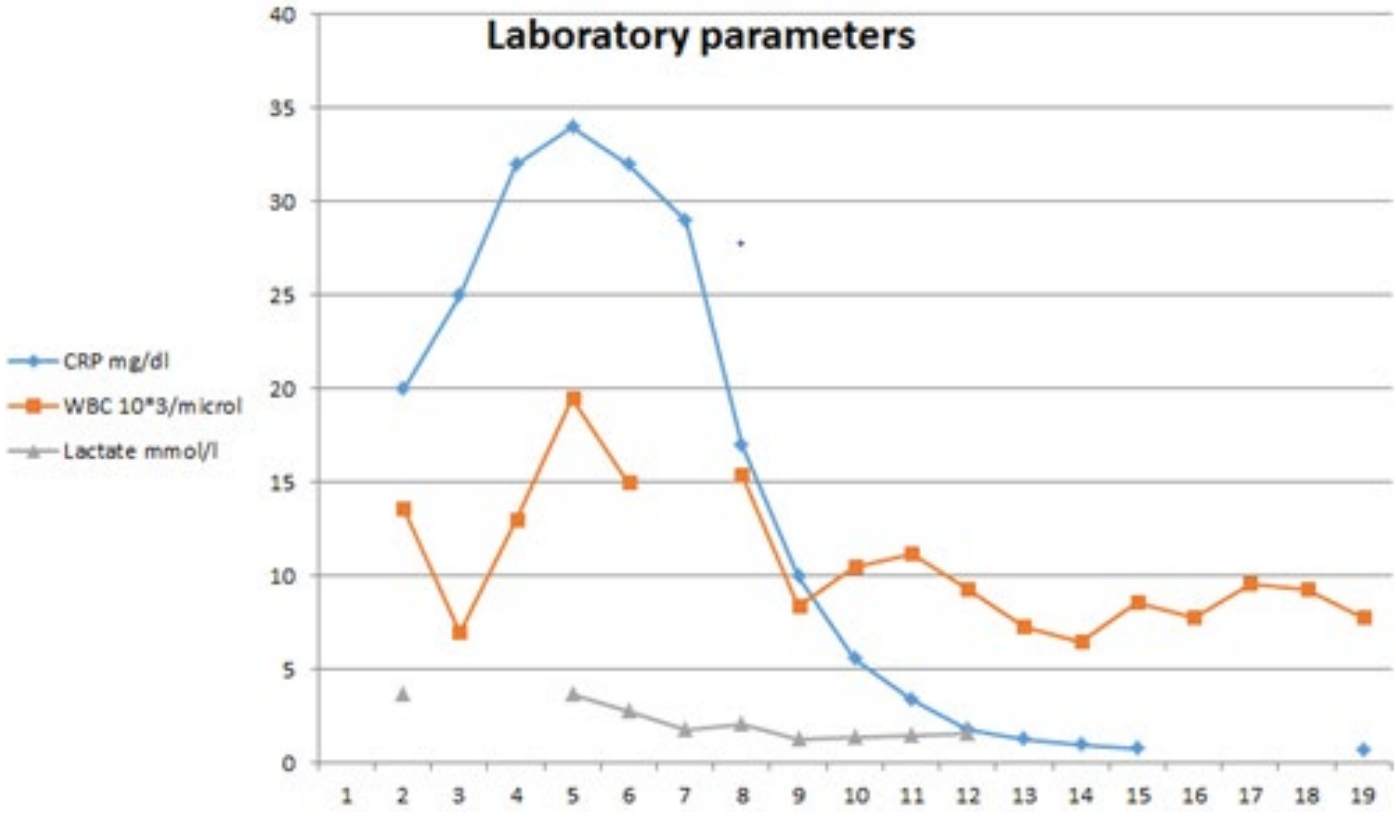
In the internal medicine department he developed severe hypoxemia, (P/F ratio=200), worsening tachypnea and dyspnea, sinus tachycardia, high fever and an increase in inflammatory markers, acute kidney injury with creatinine of 1.4 mg/dl and low blood pressure. Antibiotic therapy with ceftriaxone and azithromycin was initiated. Chest Computed Tomography (CT) scan revealed extensive bilateral lung infiltrates with ground-glass appearance. Blood, urine and sputum culture were negative, as was a GeneXpert test for COVID-19. Due

to severe hypoxemia, shock and acute kidney injury he was transferred to the ICU on March 5th, 2021. Upon ICU admission he was tachypneic, dyspneic and had very severe hypoxemia (P/F ratio=80), sinus tachycardia (140 BPM), low blood pressure (70-80/40-50 mmHg), metabolic acidosis (lactate=3.7 mmol/l; bicarbonate=18 mmol/l), and acute kidney injury (creatinine of 1.4 mg/dl) (Table 1 and Graph 1). Echocardiography showed severe left ventricular dysfunction with preserved right ventricular function, mild pulmonary hypertension and mild mitral regurgitation. No pericardial effusion was observed. Due to severe hypoxemia with lactic acidosis the patient was intubated and ventilated. After intubation, the hypoxemia worsened (P/F ratio <55), and the chest x-ray revealed diffuse bilateral pulmonary infiltrates suggestive of pulmonary edema. The patient was in mixed cardiogenic and septic shock, extreme tachycardia and hypoxemia, low blood pressure and lactic acidosis. He did not respond to furosemide and nitric oxide. Echocardiography showed severe global left ventricular dysfunction. He was placed in the prone position shortly after intubation with marked improvement in P/F ratio, but the blood pressure continued to decrease despite extreme vasopressor and inotropic support. Patient's temperature was 40° Celsius. The Biofire® FilmArray pneumonia panel and GeneXpert COVID-19 tests that were performed on sputum taken from the endotracheal tube and were both negative. Similarly, a HIV test was

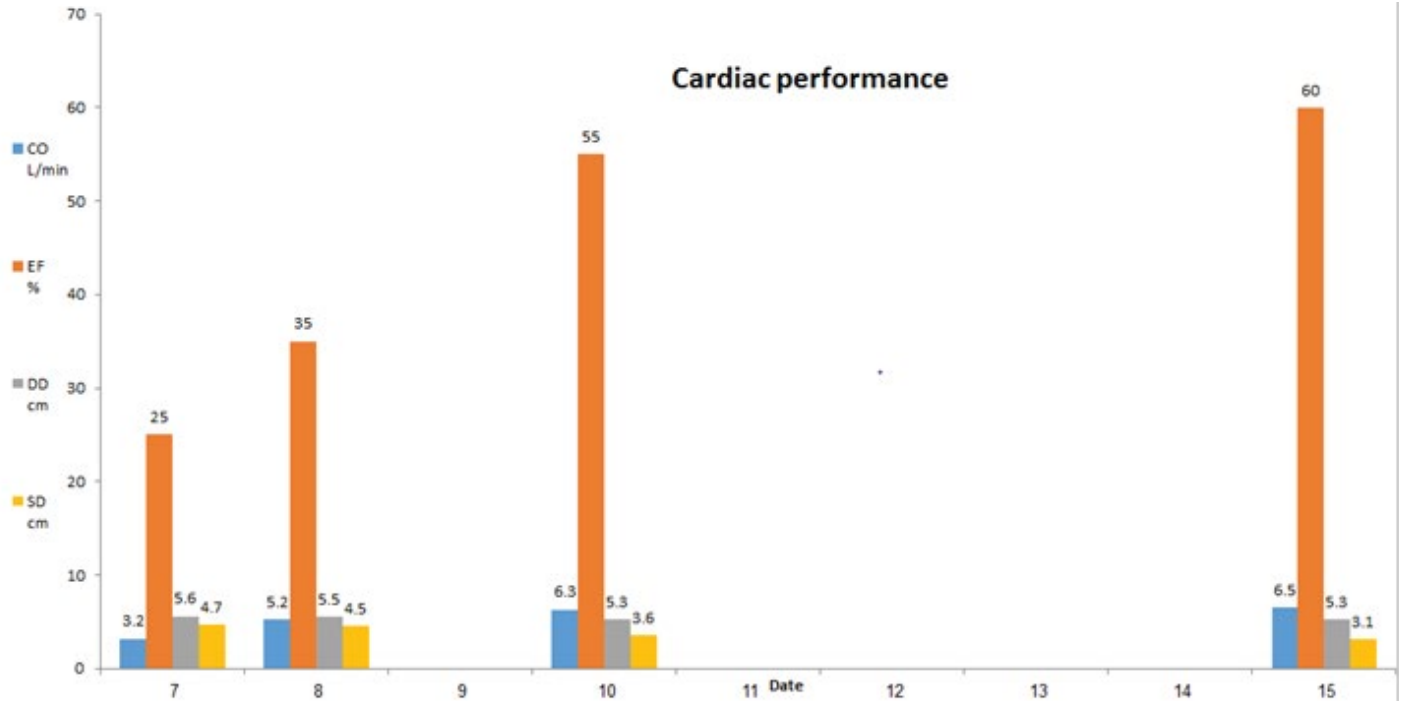
Table 1. Serial laboratory and cardiac parameters.

Date	CO (l/min)	EF (%)	DD (cm)	SD (cm)	Lactate (mmol/l)	WBC (10^3/microl)	CRP (mg/dl)	CPK (u/l)	LDH (u/l)	Cr (mg/dl)
3.3					3.7	13.6	20			1
4.3						7	25			0.9
5.3						13	32		455	1.1
6.3		20			3.7	19.5	34	189	802	1.14
7.3	3.2	25	5.6	4.7	2.8	15	32	328	734	0.87
8.3	5.2	35	5.5	4.5	1.8		29	309	658	0.64
9.3					2.1	15.4	17	130	728	0.56
10.3	6.3	55	5.3	3.6	1.3	8.4	10	49	559	0.55
11.3					1.4	10.5	5.6	66	593	0.68
12.3					1.5	11.2	3.4	80	615	0.57
13.3					1.6	9.3	1.8	64	581	0.43
14.3						7.3	1.3	44	587	0.45
15.3	6.5	60	5.3	3.1		6.5	1			0.53
16.3						8.6	0.8			0.58
17.3						7.8		22	419	0.52
18.3						9.6				
19.3						9.3				
20.3						7.8	0.7			0.58

CO: Cardiac Output; EF: Ejection Fraction; DD: Diastolic Dimension (normal 3.7-5.2 cm); SD: Systolic Dimension (normal 2.5-4 cm); WBC: White Blood Cell Count; CRP: C-Reactive Protein; CPK: Creatine Phosphokinase; LDH: Lactate Dehydrogenase; Cr: Creatinine; mmol: millimole; l: liter



Graph 1. Serial laboratory parameters.



Graph 2. Serial cardiac parameters.

negative. Electrocardiography showed sinus tachycardia without evidence of ischemia, and troponin T level was 275 ng/l/.

Due to cardiogenic shock and hypoxemia, Veno-Arterial (VA) Extracorporeal Membrane Oxygenation (ECMO) therapy was initiated and the patient was transferred to a tertiary medical center while on ECMO. The patient had severe myocarditis with ejection fraction of 20% and diastolic dysfunction Grade 2. He required inotropic and vasopressor therapy while on ECMO. Sars-CoV-2 serology was performed: Anti S IgG was positive- 40,000 AU/ml, with positive Anti N IgG, indicative of recent COVID infection. He tested negative for legionella pneumophilia, pneumocystis jiroveci, bartonella, brucella, Q fever, leptospirosis and ricketssia. He had normal levels of rheumatoid factor, immunoglobulins and complement, and Anti-Nuclear Antibody (ANA) was negative. According to the clinical presentation and serology tests, a diagnosis of adult multisystem inflammatory syndrome due to past COVID infection (MIS-A) was made. Antibiotic therapy was stopped. The patient received three days of methylprednisolone pulse therapy, 1 gram per day, and three days of Intravenous Immunoglobulin (IVIG) therapy, 0.4 gram per kilogram per day. Afterwards 300 mg of intravenous hydrocortisone were given daily with gradual tapering down of steroid dose administered. His condition improved quickly with gradual increase of ejection fraction (Graph 2). The patient was weaned off ECMO support on March 10, 2021 and cannulas were removed. Successful extubation took place on March 11, 2021. Ejection fraction gradually improved to a level of 60% with normal diastolic function. Pharmacologic therapy with beta blockers, Angiotensin Converting Enzyme (ACE) inhibitors and aspirin was initiated. He was transferred to the cardiology ward on March 13. Two days later he had complained of right leg pain. On vascular examination Dorsalis Pedis and Tibialis Posterior pulses were absent. However, Doppler signals were normal. He underwent successful right percutaneous thrombectomy of the right femoral artery. He was discharged home on March 21, 2021.

Discussion

Post-COVID Multisystem Inflammatory Syndrome in Adults (MIS-A) is a rare disease., Since June 2020, several case reposts have described cardiovascular, gastrointestinal, dermatologic and neurologic symptoms without severe respiratory illness, with positive test results for antibody assays indicating recent COVID infection [6]. In review of 27 cases of MIS-A in the literature, MIS-A was experienced approximately 2-5 weeks post COVID infection, although many patients had asymptomatic COVID infection, which make the timeline unclear [6]. The majority (24/27) of patients survived,. Clinical manifestations included sore throat, rash, conjunctivitis, fever, diarrhea, shock (both cardiogenic, vasoplegic and mixed), myocarditis, left ventricular systolic dysfunction, acute kidney injury, nausea, abdominal pain, fatigue, myalgia and. All patients had markedly elevated laboratory markers of inflammation, including CRP and ferritin, as well as D-dimers. Half of them had lymphopenia. Respiratory symptoms were absent or minimal, excluding pulmonary edema as result of cardiac failure. Many of the patients had history of recent COVID-19 infection and had positive SARA-CoV-2 antibody tests, with fewer having positive SARS-CoV-2 reverse transcription polymerase chain reaction tests. Recommended therapy, in addition to supportive care, is extrapolated from MIS-C therapeutic guidelines, and includes IVIG, corticosteroids, and possibly Tocilizumab. IVIG is recommended for all patients who meet diagnostic criteria for MIS-A. Corticosteroid therapy is recommended for patients with moderate or severe manifestations, concomitantly with IVIG. The therapy is given initially intravenous, with methylprednisolone [7]. When clinical improvement is observed, the therapy can be transitioned to an equivalent oral dose of prednisolone or prednisone by the time of discharge and then tapering-off over 3-4 weeks. In extreme cases, methylprednisolone is given as pulse therapy, at a dose of 30 mg/kg/dose [7].

Interleukin-1 inhibitor (Anakinra), interleukin-6 inhibitor (Tocilizumab) or convalescent plasma from recovered COVID-19 patients are controversial, and may be considered as an option for patients who cannot receive corticosteroid therapy or who are refractory to this treatment [8]. In addition, low-dose aspirin therapy is recommended. Although most patients with MIS-A present with mild or even absent respiratory symptoms, in the described case the most prominent clinical symptom was acute respiratory failure with severe hypoxemia and diffuse pulmonary infiltrates in chest CT. Therefore, our first assumption was acute COVID infection. COVID tests may be false negative, especially at the beginning of acute illness, and sometimes only has the third or fourth test turned out positive. Due to high index of suspicion- clinical symptoms and confirmed exposure, we repeated the tests four times, including GeneXpert test from sputum derived from the endotracheal tube which is more sensitive. A high index of suspicion is required in order to diagnose patients with MIS-A, as this syndrome may mimic other, more common conditions, such as septic shock, acute coronary syndrome, severe acute COVID-19 infection, toxic shock syndrome, other viral or bacterial infections, vasculitis and hemophagocytic lymphohistiocytosis syndrome. As a result, we suggest, that of MIS-A may be misdiagnosed. As the COVID-19 pandemic expands, we may see more cases of this syndrome. When recognized and treated early, the

prognosis of MIS-A, is good. Prompt recognition and intensive specific therapy are required in order to avoid mortality and severe morbidity.

Conclusion

In conclusion, we presented a case of severe MIS-A in a young adult, with recent asymptomatic COVID-19 infection, manifested as cardiogenic shock and pulmonary edema. As MIS-A presentation may mimic other pathologic conditions, high index of suspicion is required.

References

1. Li Jiang, Kun Tang, Mike Levin and Omar Irfan. "COVID-19 and Multisystem Inflammatory Syndrome in Children and Adolescents." Lancet Infect Dis 20 (2020): 276-288.
2. Nameet Jerath and Tania Oberoi. "Pediatric Inflammatory Multisystem Syndrome-Temporally Associated with SARS-CoV-2: A Severe Disease Syndrome in Children Temporally Associated with SARS-CoV-2." *Apollo Med* 39 (2014): 346.
3. Ashlesha Kaushik, Sandeep Gupta, Mangla Sood and Seema Sharma, et al. "A Systematic Review of Multisystem Inflammatory Syndrome in Children Associated With SARS-CoV-2 Infection." *Pediat Infect Dis J* 30 (2020): 340.
4. Sarah, Long. "Compelling Evidence of a Novel Multisystem Inflammatory Syndrome in Children (MIS-C) Associated with SARS-CoV-2 Infection." *J Pediat* 224 (2020): 1-3.
5. http://www.who.int/publications/i/item/multisystem-inflammatory-syndrome-in-cholldren-and-adolescents-with-COVID-19.
6. Sapna Bamrah Morris, Noah G. Schwartz, Pragna Patel and Lilian Abbo, 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* 69 (2020): 1450.
7. Lauren A. Henderson, Scott W. Canna, Kevin G. Friedman and Mark Gorelik, et al. "American College of Rheumatology Clinical Guidance for Multisystem Inflammatory Syndrome in Children Associated with SARS-CoV-2 and Hyperinflammation in Pediatric COVID-19: Version 1." *Arthritis Rheumatol* 72 (2020): 1791.
8. Feldstein, Levin. "Multisystem Inflammatory Syndrome in US Children and Adolescents." *N Engl J Med* 383 (2020): 334.

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