ISSN: 2754-4516

Journal of Medicine and Healthcare

SCIENTIFIC Research and Community

Case Report

Open Access

Fatal Multisystem inflammatory syndrome (MIS) in a young adult following a recent mild Covid-19

Mahathi Gopalakrishnan, Dineshbabu Sekar*, Ravi Pradeep, Vijayanarayanan Aditya and Molly Mary Thabah

Department of Medicine Jawaharlal Institute of Postgraduate Medical Education and Research Puducherry- 605006, India

ABSTRACT

Corona virus disease (COVID19) has been evolving with different spectrum of illness over time. Recently, Multi system Inflammatory syndrome (MIS), a hyper inflammatory immune response has been observed after recovery from COVID, and is described in children. Here we present a young adult with Multi system inflammatory syndrome post an asymptomatic COVID infection with a rapidly worsening clinical course. Shock, gastrointestinal symptoms are described in most adults with multisystem inflammatory syndrome (MIS-A), while respiratory involvement is minimal, whereas the patient we are reporting here had severe respiratory involvement and fulminant course. As the case reports of MIS-A is being reported majority of cases are not diagnosed in time. Increased awareness about this condition is essential, as timely immunomodulatory therapy is therapeutically life saving.

***Corresponding author**

Dineshbabu Sekar, Assistant Professor, Department of Medicine Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER) Puducherry- 605006, India. Tel: +91 9582318223; Email: babu.dhinuu@gmail.com

Received: July 05, 2021; **Accepted:** July 13, 2021; **Published:** July 15, 2021

Keywords: COVID19, Viral infection, Immune response, Treatment

Case description

A 28-year-old man presented to the emergency because of severe throat pain, difficulty in swallowing for one week, generalized erythematous rash, and high-grade fever for five days. A month ago, he had cough, fever, and throat swab was positive for severe acute respiratory syndrome novel coronavirus (SARS-CoV2) by RT-PCR. He was home quarantined, did not have hypoxemia, and recovered. At presentation his appearance was toxic, temperature 1030 F, pulse 110/min, blood pressure 74/40 mm Hg, oxygen saturation 98% while breathing room air. There were petechial lesions over the feet and arms, multiple erythematous urticated papules and plaques with confluence over the back (figure 1). Oral cavity examination showed mucosal erythema, petechiae over the palate and buccal mucosa, congestion of uvula, tonsils, and posterior pharyngeal wall. There was purulent exudate in the posterior pharyngeal wall. The neck was tender to palpation with restriction of movements. Ultrasonography neck showed multiple cervical lymphadenopathy. There was mild diffuse tenderness of abdomen. He received intravenous (IV) ceftriaxone empirically for streptococcal pharyngitis, IV vancomycin and IV piperacillin-tazobactam for presumed streptococcal toxic shock syndrome. There was transient hemodynamic improvement with IV fluids, but few hours later he had hypoxemia, hypotension again set in requiring vasopressors. Total CK was 1500 IU/L (normal 0-128), Troponin T was 3400 ng/mL (normal 0-14 ng/mL) indicating myocarditis. Left ventricular function couldn't be assessed because of tachycardia. Hemogram showed neutrophilic leukocytosis, platelets 80,000/mm3. Contrast enhanced computed tomography (CT) of neck, CT pulmonary angiography did not show pulmonary embolism nor collections in the neck. High resolution CT chest showed dense consolidation involving bilateral lower lobes (figure 1). Because of fever, rashes, lymphadenopathy, oral and pharyngeal mucosal inflammatory change, leukocytosis, thrombocytopenia, lung consolidation with hypoxemia, and shock in the background of recent coronavirus disease 2019 (Covid-19) a diagnosis of multisystem inflammatory syndrome in adults (MIS-A) was made. Intravenous Immunoglobulin (IVIG) 2 gm/kg was given. Vasopressors were continued with intermittent intravenous furosemide. Despite IVIG and supportive therapy the patient rapidly deteriorated and he succumbed 36-hours after arrival to hospital. Serum ferritin was 6938 ng/mL, CRP 38.4 mg/dL, ESR 110 mm 1st hour. Throat swab returned negative for streptococci, Corynebacterium diphtheriae. Blood cultures showed no growth at 48 hours and 5 days. Antinuclear antibodies, and antineutrophil cytoplasmic antibodies were negative. IgG antibodies to SARS-CoV-2 nucleocapsid (ELISA) was strongly positive.

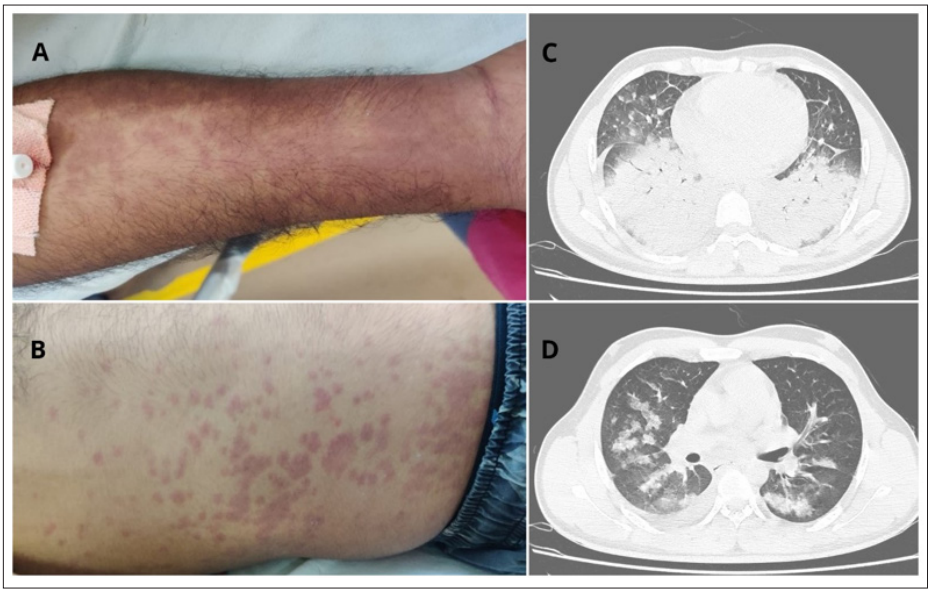

Figure 1: Petechial lesions over the right arm **(A)** with multiple erythematous urticated papules and plaques with confluence over the back **(B)**, high resolution CT chest showed dense consolidation with air bronchogram involving bilateral lower lobes **(C)**, and right pleural effusion with areas of consolidation **(D)** in right upper and middle lobes.

Discussion

The clinical features of MIS-A described in the CDC series namely odynophagia, throat pain, swelling of neck, lymphadenopathy, maculopapular rash were observed in our patient too [1]. MIS-A related to SARS-CoV2 typically occurs a median of 23 days after exposure to Covid -19.2 Twenty percent have shock at presentation [2]. Indeed, profound cardiogenic and vasodilatory shock in young males could be a late manifestation of Covid-19 [3]. IV methylprednisolone results in clinical improvements and reversal of shock.3 However in pediatric-MIS both steroids and IVIG are useful, so we used IVIG because sepsis was not yet ruled out in our patient. He had rapid deterioration despite use of IVIG because of lung involvement and severe hypoxemia requiring mechanical ventilation. Previous series have described good outcome of MIS-A because respiratory involvement is minimal [2]. As MIS-A is a new syndrome it is not clear whether this severe hypoxemia is an extension of covid-19 or a manifestation of MIS-A [4]. The pathophysiology of MIS-A could be SARS-CoV2 non-neutralizing antibodies leading to antibody dependent enhancement in immune cells resulting in hyperinflammation [5]. The optimal management of fulminant cases such the one we have described need to be studied, because MIS-A are expected to rise. It is important to research the risk factors of post Covid-19 MIS-A so that treatment directed to control hyperinflammation can be instituted early.

Acknowledgements: We thank Dr. Tamilarasu Kadhiravan, Professor of Medicine JIPMER for constant support and encouragement.

Consent: The patient's family has granted permission to write this report.

References

1. Morris SB, Schwartz NG, Patel P, Abbo L, Beauchamps L, et al. (2020) Case Series of Multisystem Inflammatory Syndrome in Adults Associated with SARS-CoV-2 Infection-United Kingdom and United States, March-August 2020. MMWR Morb Mortal Wkly Rep. 69: 1450-1456.
2. Davogustto GE, Clark DE, Hardison E, Yanis AH, Lowery BD, et al. (2021) Characteristics Associated With Multisystem Inflammatory Syndrome Among Adults With SARS-CoV-2 Infection. JAMA Netw Open. 4: e2110323
3. Chau VQ, Giustino G, Mahmood K, Oliveros E, Neibart E, et al. (2020) Cardiogenic Shock and Hyperinflammatory Syndrome in Young Males With COVID-19. Circ Heart Fail. 13: e007485.
4. Julius MA, Cantrell D, Sharif S, Zelnik Yovel D, Rapoport MJ et al. (2021) The first fatal post-covid-19 adult patient with multi-system inflammatory syndrome in Israel. Isr Med Assoc J. 23: 212-213.
5. Iwasaki A, Yang Y (2020) The potential danger of suboptimal antibody responses in COVID-19. Nat Rev Immunol. 20: 339-341.