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Case Study

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POST COVID-19 MULTISYSTEM INFLAMMATORY SYNDROME IN ADULT: A CASE REPORT

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ABSTRACT

Covid 19, a severe acute respiratory syndrome, which evolved during the time of late 2019, is principally transmitted through respiratory droplets, aerosols as well as mucosal membrane contact with fomites. Multiple organ inflammatory syndrome in adults, a serious complication of covid 19 increases the duration of hospitalization and mortality rate. Here we present the case of a 39 year old male patient who had a history of covid 19, admitted to the neurology department with the complaints of fever, myalgia, loss of consciousness associated with frothing from the mouth and postictal confusion was diagnosed with post covid multiple inflammatory syndrome in adults, status epilepticus, sepsis and acute kidney injury. The patient responded to IV antibiotics, antiepileptic, sodium valproate, IVIG as well as methylprednisolone and was discharged in a stable condition**.**

KEYWORDS: Covid 19, Multisystem Inflammatory Syndrome, Multiorgan Dysfunction, SARS-CoV2.

INTRODUCTION

Coronavirus disease 2019 (COVID-19) is a disease caused by a novel Coronavirus called severe acute respiratory syndrome coronavirus 2 (SARS-CoV 2) which spreads rapidly leading to a worldwide pandemic. As of February 15th, 2022, around 412 million covid-19 infection cases and nearly 6 million deaths have been reported. The principal mode of transmission of SARS-CoV 2 in persons is through exposure to respiratory droplets carrying infectious virus (generally within a space of 6 feet) as well as through contact transmission (example, shaking hands) and airborne transmission of droplets that remain in the air over long distances. Clinical manifestations of Covid-19 range from asymptomatic or mild symptoms to severe illness and mortality. Fever or chills, sore throat, cough, muscle or body aches, headache and shortness of breath are the common symptoms associated with mild covid-19 infection.[1] Covid 19 can progress as a severe condition with hypoxemia and dyspnea and rapidly causes acute respiratory distress syndrome (ARDS).[2] Advanced age, patients with commodities such as hypertension, diabetes mellitus, cardiovascular disease, etc. have been the independent risk factors for severity and mortality.[3]

One of the serious complications linked to covid-19 infection is MIS-C (in children) which is identified as inflammation leading to organ damage. The Centers for Disease Control and Prevention described the same phenomenon that takes place in adults as a multisystem inflammatory syndrome in adults (MIS-A).[4] Adult patients of all ages with current or previous SARS-CoV 2 infections can develop a hyper-inflammatory syndrome resembling MIS-C. Hyper-inflammation, pulmonary diseases and extrapulmonary organ dysfunction such as acute kidney injury, acute liver failure, cardiovascular disease, hematological abnormalities, neurological disorders, gastrointestinal symptoms, hyperglycemia and ketosis, ocular symptoms, dermatologic complications can be experienced in hospitalized adults with severe Covid-19.

The plausible mechanisms that may contribute to the Covid-19 and multiple organ dysfunction include the expression of ACE-2 which is an entry receptor of SARS- CoV 2 in the lung, heart, kidney, testis, liver, lymphocytes and nervous system, direct viral damage, endothelial damage, thrombo-inflammation, dysregulation of immune responses and maladaptation of ACE-2 related pathways.[5] The involvement of multiple organs in Covid-19 patients increases the length of hospitalization and mortality rate.[2]

CASE DESCRIPTION

A 39-year-old male, with a history of covid 19, was admitted on 17-10-2021 to the department of Neurology with complaints of fever and myalgia for 1 week, one episode of loss of consciousness associated with frothing from the mouth and postictal confusion. He received the first dose of covid vaccination on 07-09-2021.

He was admitted to ICU and was evaluated for new-onset seizure with an etiological possibility of meningoencephalitis. CSF study on the day of admission recorded total WBC count - 06 (0-5 cells/ul), sugar - 99 (50-80 mg/dl), protein - 38 (20-40 mg/dl), and chloride – 126 (96-106 mmol/L). CSF culture showed no growth. The initial EEG was normal. Plain CT of the brain was done and was reported normal. He was treated initially with antibiotics (Inj. Ceftriaxone 2 g IV BD) and antiepileptics (Inj. Levitiracetam 1 g/day). He had an episode of status epilepticus in ICU after which Inj. Sodium valproate (1000 mg/day) was added. He did not have further episodes of seizure but had a worsening sensorium (GCS E4V2M5). Anti-covid IgG antibody titre was elevated (2574 AU/ml, against a normal cut-off of 50 AU/ml). CRP was raised – 40.1, D-dimer was 263 and procalcitonin was negative.

With a possibility of post covid multisystem inflammatory syndrome of adult (MIS-A) or Autoimmune encephalitis, he was started on IVIG (2 g/kg) and methylprednisolone pulse therapy (1 g for 5 days). IVIG total dose of 120 g was administered over 4 days. He had recurrent seizures in ICU, after which anti-epileptics were optimized sequentially after a repeat EEG showed electrographic status epilepticus with PLEDs (Phenobarbitone 120 mg/day, Levetiracetam 2 g/day, Valproate 1 g/day and Midazolam infusion 4 mg/hour). Since he had a count of 22400 for his covid antibody as well as his MRI showed status epilepticus, physician consultation was obtained and advised to continue the same line of management. He developed a worsening sensorium with desaturation. Critical care consultation was taken, and he was intubated and mechanically ventilated after a detailed discussion with bystanders. He had 2 episodes of fever and a repeat lumbar puncture on 25/10/2021 showed 28 cells (all lymphocytes), protein 28 mg/dL and sugar 112 mg/dL. Inj. Acyclovir (500 mg IV Q8H) was added. MRI brain with contrast on 26/10/2021 showed bilateral temporal lobe and hippocampal FLAIR hyperintensity with minimal diffusion restriction and gyral edema was noted in the right temporal lobe with no evidence of abnormal post-contrast enhancement. He was noted to have decreased urine output (1960 ml) and a serum creatinine value of 2.3 mg/dl on 28/10/2021 which was raised from 0.7 mg/dl at the time of admission. Nephrology opinion was sought and was managed as per instruction by giving IV fluid at a rate of 100 ml/hr (0.9% NS 2 L/day). Pulmonology opinion was sought and was advised to add Inj. Amikacin 500 mg IV OD for VAP. Cardiology consultation was done and was advised to add Inj. Tocilizumab 400 mg IV (stat), Inj. Clexane 0.4 ml SC and Inj. Solumedrol 125 mg IV BD to the ongoing medications. His blood culture showed growth of Pseudomonas species and urine culture yielded Candida species growth. Appropriate culture-sensitive antibiotics and antifungals were started. After stabilizing his condition, he was shifted to the ward. Psychiatric opinion was sought for altered behaviour and their advice was followed. His neurological status improved and he was conscious, oriented, moving all 4 limbs and recovered motor aphasia during discharge.

Table 1: Patient's EEG Result.

EEG REPORT	
DATE	**DESCRIPTION**
22-10-2021	Abnormal record. Recorded 1 episode of electrographic seizure noted for more than 90 seconds. In addition, PLED (Periodic Lateralised Epileptiform Discharges) were noted.
25-10-2021	Abnormal record. Recorded electrographics status epilepticus.
27-10-2021	Abnormal record. Suggestive of epileptiform discharges. Recorded multiple episodes of electrographic seizure noted.
02-11-2021	Abnormal record. Suggestive of epileptiform discharges.
01-11-2021	Abnormal record. Suggestive of epileptiform discharges.
08-11-2021	The record shows no epileptiform or localization-related abnormalities.
19-11-2021	Normal record.

DISCUSSION

Covid 19, severe acute respiratory syndrome coronavirus 2, emerged in Wuhan, China, during the time of late December 2019.[3] This disease-causing virus is an enveloped, nonsegmented positive-sense RNA virus that belongs to β coronaviridae family.[6] As the epidemic progressed, person-to-person passing on is the mode of spread through respiratory droplets, aerosols as well as mucosal membrane contact with fomites. Also, faecal-oral transmission is speculated, but no evidence of intrauterine or transplacental transmission has been reported. The most recurrent serious demonstration of covid 19 infections seems to be pneumonia which is characterized by cough, fever, dyspnea and bilateral infiltrates displayed on radiographic chest imaging. Although most patients will only experience mild symptoms of the disease, some patients may experience rapid progression of their symptoms over the span of a week.[3]

Multiple system inflammatory syndrome is a serious life-threatening health condition, which is not commonly seen in adults[7] is characterized by acute lung failure, acute liver failure, acute kidney injury cardiovascular disease as well as a wide spectrum of hematological abnormalities and neurological disorders.[2] A published case report was identified describing a 22 years old male patient, who required ICU admission with ventilatory

and vasopressor support. He was diagnosed with MIS-A secondary to covid 19 infection with a positive covid antibody report and IVIG at a rate of 2g/kg was given for 3 days along with other critical care management.[8] The pathophysiology of MIS in both adults as well as children, is unknown and the common treatment methods include intravenous immunoglobulin, corticosteroids, interleukin 6 inhibitors, tocilizumab and so on. In a case series of MIS-A associated with SARS-Cov 2 infection conducted in UK and USA during March to August 2020, various inflammatory markers like CRP, D-dimer and so on were elevated. Among the total patients 7 were treated with immunoglobulins, 10 with corticosteroids and 2 with interleukin 6 inhibitors and tocilizumab.[9]

Covid 19, initially described purely as a respiratory disease is now characterized by multi-organ dysfunction, which may include acute lung failure, acute liver failure, acute kidney injury, cardiovascular disease, hematological abnormalities as well as neurological disorders.[10] Since the time period between the infection and the development of MIS-A is not clear, it adds to the uncertainty regarding whether MIS-A represents a demonstration of acute infection or complete post-acute phenomena.[2] Even though the etiopathogenesis of MIS is unknown, an antibody-mediated process, as well as dysregulated immune response, is suspected since it occurs in the post-acute covid 19 period. The incidence of acute kidney injury after covid 19 is 8.9% and 2.8% in adults as well as children.

CONCLUSION

Multisystem inflammatory syndrome in adults (MIS-A), a delayed immunologic response to SARS-Cov-2 infection in adults, is a rare but serious hyperinflammatory condition that occurs approximately 4 weeks after the onset of COVID-19 with extrapulmonary multiorgan dysfunction. So, it is essential for the public to suspect and identify the MIS-A and should be aware of the empirical treatment in order to reduce the morbidity and mortality associated with MIS-A. Preventing the SARS-CoV-2 infection and transmission by following the appropriate advice provided by health authorities is the primary way to overcome the COVID-19 infection.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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