

Epstein-Barr Virus and COVID-19 Induced Systemic Inflammatory Response Syndrome and Guillain-Barré Syndrome - A Novel Case Report

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Abstract: A 51-year-old gentleman without any comorbidities was admitted to the hospital with complaints of fever and sore throat. On examination and investigation, he was found to have jaundice and acute kidney injury with biochemical pancreatitis. He was being treated conservatively with IV Fluids and IV Antibiotics. Investigations revealed that he was suffering from the Epstein-Barr virus, and he improved with the ongoing treatment. But on day 5 of admission, he suddenly developed bilateral facial nerve palsy with weakness of the bilateral lower limbs. The nerve conduction velocity test was suggestive of Guillain-Barré syndrome. Incidentally, on the same day, he also tested positive for COVID-19. He was then initiated on IV Immunoglobulin and showed a remarkable response, with improvement in limb power and facial weakness. He was discharged after completing 5 days of IV immunoglobulin treatment for further outpatient care, including continuation of physiotherapy at home.

Keywords: Guillain-Barre Syndrome; Epstein-Barr Virus; COVID-19; Case Report.



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1. Introduction

Guillain-Barré syndrome is an acute immune-mediated polyneuropathy triggered by an immune response to an antecedent infection or any event that cross-reacts with shared epitopes on the peripheral nerves. It has an overall incidence of 1 to 2 cases per 10000 population per year. Patients usually present with symptoms of progressive, ascending, and symmetric muscle weakness and reduced deep tendon reflexes; the most severe symptoms include respiratory muscle weakness, which might require ventilatory support. Nerve conduction studies, Electromyography, and CSF studies help establish the diagnosis [1].

Epstein-Barr Virus is a herpes virus that is mainly spread by intimate contact between susceptible persons and asymptomatic EBV shedders. Most primary EBV infections are subclinical and inapparent but can present with Infectious Mononucleosis, B-cell Lymphoma, T-cell Lymphoma, Hodgkin's Lymphoma, and nasopharyngeal carcinoma. Treatment is mainly supportive, along with antiviral Acyclovir and corticosteroid Prednisolone in selected cases, along with management of the complications [2].

COVID-19 is caused by Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2), a novel coronavirus detected first in the city of Wuhan in Hubei Province of China, causing a cluster of pneumonia cases. Understanding of the disease is still evolving and can cause mild to critical disease. The presence of co-morbidities and other risk factors increases the probability of severity of the disease, most common being the respiratory complaints of fever, cough, myalgia, and presence of pneumonia in chest radiographs [3].

2. Case Report

A 51-year-old gentleman with complaints of fever and sore throat for 7 days and discolouration of eyes and urine for 3 days from Kolkata was admitted through the Emergency department. The fever was insidious in onset, high-grade, and gradually progressive with intermittent spikes along with complaints of a sore throat for the same duration. He also developed yellowish discolouration of eyes and urine in the last 3 days, which was insidious in onset and gradually progressive. He developed altered sensorium and incoherent talk for the last 1 day, following which the family of the patient got him admitted. There were no complaints of nausea and vomiting, loss of consciousness, headache, neck pain, cough, palpitations, chest pain, abdominal pains, night sweats, reduced appetite, or any similar illness in the family and neighbourhood.

On examination, he was alert, conscious, and cooperative but was speaking without context. Pulse rate 110 per min, regular in rhythm, normal in volume and character. Respiratory rate 25 per min, regular, no accessory muscles of respiration working. BP 110/70 mm Hg. SpO₂ 97% in Room air. The temperature is 100°F. Systemic examination was all within normal limits. He was initiated, including IV Fluids, IV Antibiotics, and other supportive measures were taken. He improved with the improvement of his sensorium, fever remitted 3 days later, and a reduction of Total Leukocyte count with the improvement of renal function and liver function tests with conservative management. In view of multi-systemic organ dysfunction, he was worked up with cultures and a tropical fever workup, which were all negative. 2D ECHO was normal. He was then evaluated for viral fevers, and quantitative EBV DNA PCR came out to be positive- his treatment was continued conservatively, and he made gradual improvement.

On day 5 of admission, he suddenly developed complaints of pain and weakness of the muscles of the face. On examination, he was found to have bilateral facial nerve palsy, normal sensory examination, normal power in upper limbs, reduced power in lower limbs with reduced deep tendon jerks in lower limbs, and normal superficial reflexes. He was urgently referred to a Neurologist and underwent Nerve Conduction Studies and a Repetitive Nerve Stimulation test, which were suggestive of Guillain-Barre Syndrome. A SARS-CoV-2 RT-PCR was sent, which came out positive. Lumbar puncture was also done, which showed slightly elevated protein levels. There was no evidence of any infection or significant albumin-cytological dissociation. The investigation chart of the patient has been given in table 1 in detail.

Table 1. Investigation Chart.

| Parameter | Day 1 | Day 3 | Day 5 | Day 6 | Day 8 | Day 10 |
|--------------------------------------|--------------|---------------|---------------|-----------------|----------------|-----------------|
| Hemoglobin (mg/dL) | 11.6 | 11.4 | 11.1 | 10.7 | 11.5 | 9.8 |
| TLC (per cu mm) | 21,6 | 22,5 | 20,1 | 21,1 | 11,8 | 5,5 |
| Platelets (lac/dL) | 1.16 | 0.89 | 0.87 | 1.03 | 1.88 | 3.22 |
| Sodium / Potassium (mEq/dL) | 131 / 4.0 | 132 / 3.5 | 136 / 3.3 | 138 / 3.1 | 134 / 3.3 | 140 / 3.8 |
| Urea / Creatinine (mg/dL) | 124 / 2.5 | 96 / 2.3 | 66 / 2.0 | 54 / 1.4 | 45 / 1.2 | 33 / 1.1 |
| Total / Conjugated Bilirubin (mg/dL) | 6.5 / 4.1 | 5.5 / 3.7 | 3.6 / 2.2 | 2.6 / 1.4 | 2.5 / 1.3 | 1.4 / 0.9 |
| Albumin / Globulin (g/dL) | 2.3 / 2.9 | 2.4 / 3.4 | 2.3 / 3.7 | 2.6 / 3.9 | 2.7 / 4.2 | 2.6 / 6.2 |
| SGOT / SGPT / ALP (U/dL) | 78 / 46 / 94 | 67 / 45 / 199 | 98 / 85 / 224 | 100 / 104 / 217 | 122 / 92 / 212 | 161 / 145 / 149 |

| | | | | | | |
|------------------------|-----------|---|---|------|---|---|
| Amylase / Lipase (U/L) | 225 / 943 | – | – | – | – | – |
| Ferritin (ng/mL) | – | – | – | 1020 | – | – |
| LDH (U/L) | – | – | – | 217 | – | – |

All initial investigations, including the Widal test, CMV PCR, and blood and urine cultures (cultures and tropical fever workup), were negative. EBV PCR showed 75,000 copies per milliliter. On day 5, SARS CoV 2 RT PCR returned positive. On day 6, CSF study revealed a cell count of 5 with 100% lymphocytes, normal glucose and LDH levels, and borderline elevated protein at 62. A four-limb nerve conduction study was suggestive of Guillain Barré Syndrome, with conduction slowing, prolonged distal latency, and prolonged F waves predominantly in the lower limb nerves.

The repetitive nerve stimulation test and MRI brain were within normal limits, and the comprehensive CNS infection panel PCR was negative. On day 9, SARS CoV 2 IgG was 11,788. He was initiated on intravenous immunoglobulin and showed marked improvement in muscle strength, eventually being discharged in a hemodynamically stable condition with advice for physiotherapy at home after completing the IV immunoglobulin course.

3. Discussion and Conclusion

A 51-year-old gentleman presented with Systemic Inflammatory Response Syndrome caused by Epstein-Barr virus, which was managed conservatively. On day 5 of admission, he had a sudden onset of facial weakness, diagnosed as Guillain-Barré Syndrome and was also detected to be suffering from COVID-19. He was then initiated on IV Immunoglobulin and physiotherapy, and he had a complete recovery.

Systemic Inflammatory Response Syndrome (SIRS) is an aberrant, exaggerated defense response of the body to any endogenous or exogenous noxious stimuli, which can cause autonomic, endocrine, hematologic, and immunological alterations via acute phase reactants. Although this mechanism is defensive, the dysregulated cytokine storm can lead to a catastrophic inflammatory cascade. Systemic Inflammatory Response Syndrome can be diagnosed if there is any 2 of the 4 -body temperature above 38 C or below 36 C; Heart rate more than 90/min; Respiratory rate more than 20/min or PCO₂ less than 32 mm Hg; TLC more than 12K or less than 4K or more than 10% of band forms [4]. Systemic Inflammatory Response Syndrome is caused by a variety of infections, with previous documentation of causative organisms being Epstein-Barr Virus, as described by Spivack et al [5] and by COVID-19, as described by Masi et al [6].

Guillain-Barré Syndrome (GBS) or autoimmune demyelinating polyradiculopathy is an immune-related disorder characterized by acute, flaccid neuromuscular disorder and is usually post-infectious. Physical examination can detect reduced power with absent deep tendon reflexes and is diagnosed by Nerve conduction velocity and treated by IV Immunoglobulin [7]. There are many identified predisposing infections causing Guillain-Barré Syndrome, among them 5% to 28% of a mild form of Guillain-Barré Syndrome is caused by Epstein Barr Virus as described by Kim et al [8] and is also reported after COVID-19 infections as described by Khan et al [9] but could not find any report of Guillain-Barré Syndrome in which EBV and COVID-19 were identified simultaneously.

In this patient, we could not find any evidence as to which caused the Guillain-Barré Syndrome or the probable pathophysiology. We can only assume that complex molecular mechanisms of both infections might have triggered the disease. There is no definite treatment for viral illnesses, and Guillain-Barré Syndrome was treated as per protocol with Intravenous Immunoglobulin. Hence, we report this patient who developed Systemic Inflammatory Response Syndrome and Guillain-Barré Syndrome following a simultaneous infection of EBV and COVID-19.

We thus present a patient with Systemic Inflammatory Response Syndrome who developed Guillain-Barré Syndrome later. EBV was initially diagnosed and was later found

to be COVID-19 positive; both can cause the illnesses. It is challenging to pinpoint the cause and pathophysiology due to the complex molecular mechanisms involved.

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Research Ethics Committee Approval: We declare that the patient approved the study by signing an informed consent form and the study followed the ethical guidelines established by the Declaration of Helsinki.

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Conflicts of Interest: All other authors declare no conflicts of interest.

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