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"AIDP: Another aspect of COVID 19"

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Introduction

COVID-19 has presented predominantly with respiratorydisease, but neurological manifestations are being increasingly recognized including stroke, encephalitis, acute disseminated encephalomyelitis, and myelitis along with symptoms of peripheral nervous system, and as muscle diseases. The proposed mechanisms of neurological involvement are direct infection by the virus or inflammation of the nervous system and vasculature, which may be para-infectious or post infectious. [1]

This case of a 55-year-old woman from Central India presented with acute flaccid paralysis as Guillain-Barré syndrome due to COVID 19 infection. Guillain-Barré syndrome (GBS) is an acute, frequently severe, and fulminant polyradiculoneuropathy that is autoimmune in nature. It manifests as a rapidly evolving, ascending, areflexic motor paralysis with or without sensory disturbance. [2]

Case presentation

Our patient, a 55-year-oldfemale, resident of central India, presented with complaints of generalized weakness and body ache of 3 days. She alsohad mild nonspecific abdominal pain associated with nausea. 2 days later, she had complaints of difficulty in walking and mild weakness of arms.Weakness was sudden in onset and was progressing. This was associated with pain in the limbs. There was no history of fever, headache, vomiting or convulsions, involvement of facial or other cranial nerves, trauma to spine or similar episodes in the past. No history of bowel or bladder involvement were present. There was no history of recent vaccination.

She was a diagnosed hypertensive, diabetic but well controlled on oral drugs.

On admission, she had a GCS of E1V1M1, hemodynamically stable with blood pressure 130/85 mm Hg, heart rate 70 beats/minute, respiratory rate 15/minute, and maintaining oxygen saturation of 96% on Oxygen 6 L/min, unequal pupil (right-3mm not reacting to light while left-2mm and reacting to light), she had weakness of both distal and proximal lower extremities, associated with unsteady gait. She had with no sphincter affection or symptoms suggestive of dysautonomia. Physical examination showed normal temperature of 37°C, with normal respiratory and cardiac findings. On neurological exam, the patient was alert, conscious, and oriented with normal speech and higher mental functions. Cranial nerves were normal.

Motor examination showed reduced tone, muscle strength examination weakness in 4 limbs with a Medical Research Council scale of grade 4+/5 in proximal muscles, grade 3/5 in distal muscles of the upper extremities, grade 4/5 in proximal muscles, and grade 3/5 in distal muscles of both lower extremities. Deep tendon reflexes were absent all over, and planter response was flexor bilaterally. She was tested positive for COVID 19. CT thorax also suggested bilateral lower lobe infiltrates.

Initial laboratory investigations were as follows; white blood cells (WBCs) count of 8.6 cells per microliter (neutrophils = 62.2%; lymphocytes = 23.4%), RBCs of 4.47 million cells/mcL, hemoglobin 11. 9 g/dL, and platelet count is 180,000 platelets/mcL. Erythrocyte sedimentation rate of 16 mm/h and C-reactive protein was 114 mg/L. His international normalization ratio was 1.34, serum glucose 8.4 mmol/L, BUN 4.6 mmol/L, Cr 84 umol/L, alanine aminotransferase 46 U/L, potassium 4.8 mmol/L, sodium 131 mmol/L, and HbA1c was 6.1%

Cerebrospinal fluid (CSF) analysis showed high protein; 623 mg/L, normal glucose levels: 4.3 mmol/L, no WBCS, negative culture and sensitivity, and gram stain for bacterial infection. Virology PCR screening for neurotropic viruses was negative in serum and CSF.

On further investigation, she was found to have Guillain-Barré syndrome (AIDP) by Nerve conduction study (predominantly sensorimotor axonal polyradiculoneuropathy) and was treated for the same with doses of IVIG following which her weakness improved with nofurther progression of weakness and was discharged later with no residual weakness of limbs.

Discussion

Coronaviruses (CoVs) belong to the subfamily Orthocoronavirinae in the family Coronaviridae, Order Nidovirales. The CoV genome is an enveloped, positive-sense, single-stranded RNA. The mechanisms by which SARS-CoV-2 causes neurological damage include direct damage to specific receptors, cytokine-related injury, secondary hypoxia, and retrograde travel along nerve fibres. [3]

The SARS-CoV 2 virus attaches to angiotensin converting enzyme 2 receptor and to gangliosides containing sialic acid. [4, 5] The mechanism of GBS following COVID-19 is suggested to be due to molecular mimicry between the viral protein associated gangliosides and peripheral nerve gangliosides. [6] Alternatively, the mechanism of nerve damage is due to inflammatory mediators released by macrophages following T-cell activation. [1] Also some authors suggest that this could be due to the generalised, hyper inflammatory response that happens with COVID-19 illness as a parainfectious mechanism. [7, 8] This is seen in our case as well, as evidenced by the elevated levels of Inflammatory markers.

Most cases reported gave typical features of GBS, such as generalised weakness, (which is so far reported to occur within 5days - 3weeks of COVID infection), demyelination on nerve conduction study, albumino-cytological dissociation in CSF; CE-MRI studies were not conclusive in some of the cases. [7, 9, 10, 11, 13] While, CE-MRI spine couldn't be performed in our case, other features correlated well with the general picture of reported GBS cases. Preceding the onset of weakness our patient had antecedent illness as abdominal pain and nausea, and little respiratory symptoms of COVID 19.

Treatment for GBS associated with COVID-19 is same as with other cases of GBS; which included IVIG, plasmapheresis and other supportive treatment. Excellent response to IVIG is seen in many cases reported, further emphasizing the immunological mechanism of the disease. [11, 12, 13] Similarly in our case also, further progression of weakness was halted and considerable improvement in tone of limb muscles noted.

Conclusion

Physicians must be well informed about the possibility of such a case to occur and consider GBS as a differential diagnosis in a similar scenario. Overlap of respiratory paralysis in GBS and COVID-19 makes it even more important for the physicians to diagnose and manage GBS early

in patients of COVID-19. [6] To the best of our knowledge, this is the first case of Guillain-Barré syndrome associated with COVID-19 reported from Central India. Further reports of such cases and case studies would lead to a better understanding of the disease, so that patients might be diagnosed early and receive appropriate treatment.

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