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Post Covid–19 Guillain Barre Syndrome: A Case Report

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Abstract

A 43 year male presented to the Medicine department with complaint of weakness in all of his four limbs. The patient had history of covid 19 infection 40 days prior to this event. His laboratory findings showed albumino–cytological dissociation in CSF, a decrease in nerve conduction velocity, unremarkable renal function test, liver function test, serum electrolytes and normal brain spine imaging. The cause was considered to be post covid acute inflammatory demyelinating polyneuropathy (AIDP) or Guillain Barre Syndrome (GBS) and he was treated with intravenous immunoglobulin (I.V. Ig).

Keywords: Covid19, Guillain Barre Syndrome, intravenous immunoglobulin.

Introduction

The novel virus, Severe acute respiratory syndrome corona virus 2 (SARS–CoV–2) which originated from

Wuhan, China in 2019 led to an acute respiratory infectious disease which spread across the globe and rendered WHO to declare it a global pandemic. India witnessed its first confirmed case of Covid 19 infection in January 2020[1].(1) It is an enveloped, positive sense and single stranded RNA virus. The most common presentation in Covid–19 infection included dry cough, fever, headache, difficulty in breathing, sore throat, headache and even diarrhea[2]. The severity of the disease was closely related to the age group of the patient , the duration of illness and the co–morbidities which all resulted in the need of an ICU setup for monitoring[3,4]. COVID 19 virus infection can be classified into 3 stages viz Stage 1 which is asymptomatic, Stage 2 which lead to upper airway and conduction system infection and the later stage 3 which included hypoxia, ground glass haziness on imaging and may progress to Acute respiratory distress syndrome[5]. But many of the covid recovered patients also suffered from different symptoms similar to the post recovery manifestations, which happened after Severe acute respiratory syndrome (SARS) attack in 2003[6]. The primary system involved is respiratory but neurological, cardiovascular and gastrointestinal involvement have also been reported in different literature. Kamal et al concluded in their study that majority of the post covid patients suffered from fatigue (72.8 %), anxiety (38 %), joint pain (31.4 %), continuous headache (28.9 %), chest pain (28.9 %), dementia (28.6 %) depression (28.6 %), and dyspnea (28.2%)[7]. In a study from France on 58 ICU patients, 49 (84%) had neurological complications, including 40 (69%) with encephalopathy and 39 (67%) with coticospinal tract signs[8]. In a study reported from Wuhan, 78 (36.4%) out of 214 admitted patients had neurological symptoms like anosmia, stroke and seizures[9]. The neurological complications commonly noted were cerebrovascular accident, acute transverse myelitis, Guillain barre syndrome (GBS) and acute encephalitis[10].

Case Presentation

A 43 year old male presented to the medicine department with chief complaint of tingling sensation and weakness in all four limbs. The patient's complaint started with tingling over his lower limbs that ascended to involve his upper limbs. Few hours later the patient started feeling weakness in his lower limbs which within 12 hours progressed to involve his both upper limbs. Simultaneously the patient also developed difficulty in speaking and drinking water. 40 days prior to this event, the patient tested RT-PCR positive for COVID 19, was home isolated with supportive medication and became RT-PCR negative for COVID 19 after 10 days. On examination, the patient was afebrile with blood pressure of 116/80, pulse rate of 70/min, oxygen saturation 98% on room air and no chest discomfort. He was conscious oriented to time place & person and was well cooperative. The patient had left side lower motor neuron (LMN) type 7th cranial nerve palsy and absent palatal and pharyngeal reflex owing to bulbar involvement. The tone was decreased and the power was limited to MRC Grade 1 in all 4 limbs. The sensory modalities viz. fine touch, pain, temperature, proprioception and vibration were normal. The deep tendon reflexes were absent and the bilateral plantar was mute. There were no signs of meningeal irritation and no bladder and bowel involvement. According to the history given and the clinical findings of LMN type quadriparesis without sensory and bladder involvement, a provisional diagnosis of Post COVID Acute inflammatory demyelinating polyneuropathy (AIDP) or Guillain Barre syndrome (GBS) was made.

His blood examination showed CRP 66 IU/ml, S.LDH 286 IU/L,S.D Dimer 0.42 mcg/ml, S.ALP 99 IU/L, S Urea/ Creatinine 42/0.8 mg/dl, S. total bilirubin/ direct bilirubin 0.7/0.3 mg/dl, S. SGOT/SGPT 24/18 IU/L, S. Na+/k+ 144.14 / 4.90 mmol/L and total calcium 10.87 mg/dl. His hemogram showed WBC 11,400 with DLC per microlitre as neutrophil 10,410 (90.8%) ; lymphocyte 810 (7.1%) ; monocytes 190 (1.7%) ; eosinophil 20 (0.2%) ; basophil 20 (0.2%); hemoglobin 11.9 gm/dl with hematocrit 45.2 and MCV 84.2 fl; total platelet count 2,01,000 per microlitre. Nerve conduction study showed axonal degeneration with demyelinating neuropathy. CSF analysis showed glucose level of 91 mg/dl, protein level of 238 mg/dl with 5 cells per cumm consisting of 100% lymphocytes which was consistent with the albuminocytological dissociation. His MRI Brain and Spine do not reveal any significant abnormality. Thus based on the clinical scenario and the investigational findings a final diagnosis of Guillain Barre syndrome succeeding COVID 19 infection with level 1 diagnostic certainty according to Brighton Criteria was made. The patient was treated with intravenous immunoglobulin (I.V. Ig) with a total dose of 2gm/kg over 5 days. The patient improved symptomatically and was discharged after 10 days on proper physiotherapy and follow up. (Figure – 1)

Discussion

Guillain Barre Syndrome is an autoimmune entity which occurs mostly following an infectious cause, usually respiratory or gastrointestinal, the most common agent being Campylobacter jejuni. About 75% of GBS patients have history of antecedent infection and almost every febrile illness or immunization has at one time being reported to precede GBS. It occurs globally affecting all ages with no gender bias. The overall incidence of GBS ranged between 1.1 per lac / year to 1.8 per lac / year but recent outbreaks have been reported following covid19 infection[11]. Earlier Lormeau et al in their study on French population during outbreak of Zika virus infection during 2013–14 concluded an increase in GBS cases suggesting a possible association between Zika virus and GBS[12]. GBS occurs as a result of molecular mimicry in which non self-antigens of infectious agents causes cross reactive cellular and humoral immune response leading to demyelination and axonal nerve injury in susceptible individual. It leads to symmetrical ascending flaccid paralysis with areflexia and without bladder and bowel involvement. In about 5% of patients, the weakness may progress to total motor paralysis and respiratory failure in a couple of day. Even ocular and pupillary muscles can be involved in severe cases. The pattern is monophasic with onset to plateau phase around 12 hrs – 28 days. The other variants of GBS are Miller Fischer syndrome (MFS), Acute motor axonal neuropathy (AMAN) and Acute motor sensory axonal neuropathy (AMSAN). The electrophysiologic study and CSF study are used to confirm the diagnosis in view of clinical presentation. The albumin-cytological dissociation onsets about 48 hrs after weakness and is usually elevated by the end of 1st week. The various antibodies found in AIDP, AMAN and MFS are GM1, GD1a and GQ1b respectively. The mainstay of treatment is I.V. Ig with dose of 2gm/kg over 5 days and if needed plasmapheresis. Steroids are not recommended and the patient is provided proper physiotherapy for the underlying weakness.

Conclusion

The dramatically emerged corona virus named SARS–CoV–2 caused severe pneumonia leading to worldwide mortalities and leaving significant impact even post

recovery. A differential diagnosis of AIDP should be considered if patient present with motor weakness following a covid19 infection. Early treatment with intravenous immunoglobulin should be started with proper monitoring of other parameters.

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Legend Figures

Figure 1: Showing left side LMN type facial palsy