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A Case Report of a Quinquagenarian Patient Suffering with Rare Neural Disorder Guillain Barre Syndrome

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Abstract: ***Guillain - Barré syndrome is an acute onset ascending paralysis and sensory disturbances with autoimmune disease often following infections such as gastrointestinal disease. A 58 year old male patient, following gastrointestinal infection, had progressive weakness in the lower limbs which ascended into the upper limbs. On clinical examination patient showed bilateral weakness of the lower extremities with decreased reflexes and sensory deficits but no cranial nerve involvement. Electromyography and nerve conduction studies affirmed the CSF diagnostic work - up as one of the most common subtypes of GBS: acute inflammatory demyelinating polyneuropathy. IVIG combined with supportive treatment, consisting of help in respiration and physiotherapy, produced dramatic improvement. By day five, enough independent respiratory function had returned to allow the patient to be moved for rehabilitation, thus demonstrating early intervention and multidisciplinary management. Continued long - term follow - up revealed the trend of continued motor recovery and independent ambulation, thus requiring further neurological assessment and intensive care management in an attempt to maximize the benefit. The case highlights the fact that GBS requires early diagnosis and IVIG therapy followed by multifaceted management at the earliest.***

Keywords: Guillain - Barré Syndrome, Autoimmune disorder, Intravenous immunoglobulin, Multidisciplinary management

1. Introduction

Guillain - Barré syndrome (GBS) is the most frequent cause of acute neuromuscular paralysis. The immunopathology of GBS, having wonderfully developed and bridged well over a century, has remarkably advanced in terms of clinical features, diagnosis, and management by their immune - mediated pathophysiology. GBS being suspected among physicians saves possible preventable morbidity if treated. [1, 2, 3] GBS and its forms are the commonest kind of immune - mediated neuropathies, mostly preceded by gastrointestinal or respiratory infections. It closely follows the mode of infections like Campylobacter jejuni and those vaccinations. Therefore, GBS hints at its multi - factorial etiology. It is a very rare condition: incidence of 0.4 to 2 cases/100000 individuals with a high service expenditure and long recoveries. This disease has also been more predominant in males than females, and it is estimated that 100, 000 new cases take place annually worldwide. [6]. In the GBS population, up to 70% have reported the presence of antecedent infections supporting molecular mimicry. The variability in the presentation of GBS may also be considered with the various antibodies directed to the different components of the peripheral nerves. [7, 8]. The complement cascade is also implicated in the pathogenesis of GBS but many more remain yet to be discovered concerning the acute inflammatory demyelinating polyneuropathy variant.

Patients with GBS usually have the following: Ascending weakness and sensory symptoms usually peak four weeks [9] since the onset. The condition is often marked by the symmetric involvement of flaccid paralysis and deeply weak, which conditions usually require admission to a hospital. The most critical clinical variant of GBS is respiratory failure, which has appeared in as many as 30% of cases that would require long hospital stays. Other clinical variants of GBS include acute motor axonal neuropathy and the pharyngeal - cervical - brachial variant which are tested through electromyography, nerve conduction studies, cerebrospinal fluid analysis with albuminocytologic dissociation and ganglioside antibody. Other causes must be excluded through MRI, and serial monitoring of respiratory functions must be performed in all high - risk patients. [14]. GVB is essentially treated with IVIG or plasma exchange. Both are established to be of benefit in randomized controlled trials [15]. The mechanisms of IVIG include immune modulation and plasma exchange acts by physically removing the pathogenic antibodies and complement proteins. Both treatments are best initiated in the first two weeks of symptom onset. Most of these patients, however will experience significant long - term disability despite treatment [16, 17]. Utilizing Erasmus GBS Outcome Score and other predictors will be able to spot early on those destined to have a poor outcome in order to initiate interventions sooner. Other ongoing clinical trials include IVIG and more courses, plus complement inhibitors. Most patients recover well, but with a high percentage who could achieve independent ambulation at six months, some may remain debilitated by continued disabilities and complications that include respiratory compromise and bulbar palsies [11, 18].

Here is a case of Guillain barre syndrome wherein the causative etiology being a gastrointestinal disorder resulted in neuronal dysfunction, thus requiring ICU admission and management.

2. Case Report

A 58 - year - old male who came to the clinic with progressive weakness of the lower limbs that ascended to the upper limbs within a week. This was accompanied by disturbances of sensation. Ten days ago, he had an infection of the intestines with diarrhea and abdominal cramping, which resolved spontaneously. He denied significant illness, medicine, or known allergies.
He was weak in the lower limbs and had a decreased deep tendon reflex both in the upper and the lower limbs. On examination, the sensation at his knees were decreased. The four limbs had decreased muscle tone. There was no meningismus or encephalopathy in the examination of cranial nerve. CT of the brain also was negative for stroke or hemorrhage. The patient was hemodynamically stable, but it was noticed that the patient had a degree of breathlessness and was not able to get an adequate breath without much exertion.

All the investigations, including count, liver, and kidney functions, came within normal limits. The cerebrospinal fluid analysis showed high levels of protein at 135 mg/dL with cell count being normal; hence, albuminocytologic dissociation could be ruled out. No abnormality was shown in the MRI of the brain and spine. Electromyography and nerve conduction studies revealed demyelination in all limbs, and thus, this confirmed Guillain - Barré Syndrome.

On the first day, the patient's condition worsened with disturbances of gait and major sensory disturbances. Treatment initiated as per NINDS guidelines. The first five days of intravenous immunoglobulin IVIG are initiated at a dose of 0.4g/kg/day. Thiamine 100mg twice a day, Librium 10mg for seizures, and Clexane 40mg daily for DVT prophylaxis are initiated. General supportive care is non - invasive ventilation, physiotherapy, and pain control with analgesics.

He continued to regain muscle strength and breathing capacity. He was then moved to the rehabilitation ward on the fifth day of his ventilator. On rehabilitation, he continued to acquire demyelination. Day eight: he left with anti - epileptic - Librium 10mg; vitamin supplements - Thiamine, Meganeuron; anticoagulant - Apixaban 2.5 mg; for review of neurology in two weeks. He started walking with aid at six weeks and had remarkably improved Follow up for recurrence and remaining impairments.

3. Discussion

A 58 - year - old male with progressive ascending weakness and sensory disturbance following gastrointestinal infection with diarrhoea and cramping. The above is the classical presentation of GBS that usually happens after infections, most commonly by Campylobacter jejuni. Such sudden advancement mandates detailed diagnostic workup including physical examination, CSF examination and EMG, that's all positive for GBS. Diminished deep tendon reflexes and decreased muscle tone coupled with absence of cranial nerves also provide supporting evidences to this diagnosis.

In GBS, inappropriate immune response occurs in which the body's immune system goes about attacking peripheral nerves; this is normally due to infection by molecular mimicry. The earlier gastrointestinal infection in this patient most probably elicited an immune reaction that cross - reacted with neural antigens and gave rise to demyelination with the subsequent neurological deficits [4]. Further, normal cell counts with albuminocytologic dissociation characteristic of the inflammatory process results in the deposition of other proteins at higher concentrations in CSF [19]. Such immune - mediated demyelination disrupts nerve conduction and explains the characteristic motor and sensory symptoms seen in GBS.

Management of GBS will include prompt intravenous immunoglobulin at a dose of 0.4 g/kg/day for five days, which essentially neutralized the immune attack. To follow through with this, care will consist of respiratory, physiotherapeutic supports, and analgesics supplemented with thiamine, Librium, and Clexane nutritional supplements, seizure prophylaxis, and DVT prophylaxis.

She slowly improved over days and regained independent breath support by day five and was sent to rehabilitation for further physiotherapy. She had been supported with a great deal of recovery through early and intensive physiotherapy having been mobilised at six weeks with minimal support, and therefore, this would have been a good use of timely intervention and rehabilitation.

Long - term follow - up is very essential in the case of GBS. The patient may need to be followed up for possible relapse and residual deficits. The patient was started on drugs and set up for neurological assessment bi - weekly. Support and rehabilitation should go on long after the acute illness to bring about the best possible recovery with favorable long - term outlook.

4. Conclusion

Again, this patient of GBS puts forward the point of early diagnosis and full treatment. GBS was diagnosed upon progressive ascending weakness following the isolation of a gastrointestinal infection from analysis of cerebrospinal fluid and electromyography. Early initiation of IVIG with provisions for respiratory support, physiotherapy, and pain management stabilized the patient further to his recovery. Improvement at discharge is testimony to the requirement of timeliness for intervention.

This therefore highlighted the management of GBS with multidisciplinary care and rehabilitation. Improved motor function as well as walking within six weeks indicate effective follow - up of comprehensive treatment that resulted in positive outcomes for better recovery and reduced the potential complications. This case should require early intervention with multidisciplinary support to ensure optimal recovery outcome in GBS long term.

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