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Case Report

Expect the unexpected: a case report of bacterial meningitis causing Guillain-Barré syndrome in an immunocompetent person

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ABSTRACT

This is the case report of a 36-year-old male presenting as acute onset of progressive bilateral lower extremity weakness, initially managed as a case of Guillain-Barré Syndrome (GBS). However, his cerebrospinal fluid tested positive for *Neisseria meningitidis* Y, *Neisseria meningitidis* W135, *Streptococcus* group B antigen, and *Streptococcus pneumoniae*. The patient was later treated for bacterial meningitis. Most cases of GBS are preceded by an upper respiratory infection or gastroenteritis. There are only a few reported cases of bacterial meningitis occurring coincidentally with GBS, much more in an immunocompetent individual. This is a rare case of such with good patient recovery and outcome.

Keywords: Meningitis, Guillain-Barre syndrome, Polyradiculoneuropathy, CNS infection

INTRODUCTION

GBS is the most common form of acute inflammatory demyelinating polyneuropathy. *Campylobacter jejuni* is the most frequent antecedent infectious cause, other agents include cytomegalovirus, Epstein-Barr virus, and *Mycoplasma pneumoniae*.[1] It is usually preceded by a respiratory or gastrointestinal infection although in recent times, there are a lot of reported cases of post-immunization GBS.[2] Up to 70% of patients have reported a preceding illness at least 1-6 weeks prior to the onset of symptoms.[3] There is no definite age or sex predilection with cases ranging from infants to elderly, but it is more common in people aged 50 years and older.[4] In a systematic review by Wachira et al 2022 the estimated incidence rate worldwide is approximately 0.30 to 6.08 cases per 100,000 people.[5]

GBS is characterized by ascending motor weakness over a period of several days to weeks, initially affecting the lower extremities. Depending on the variant, it can also present with sensory deficits, hyporeflexia/areflexia, and cranial nerve palsies. Cranial nerve involvement, if present, would occur later in the disease. Severe forms of GBS are characterized by dysautonomia and respiratory involvement leading to a need for mechanical ventilation.

There are different variants of GBS depending on the symptomatology and clinical picture. The most common form is the demyelinating type, presenting with ascending motor weakness starting from the legs to the trunk to the arms, then involving the cranial nerves. There is also a variant which predominantly affects the pharyngeal-cervical-brachial muscles leading to upper extremity weakness, dysphagia and ophthalmoplegia. Another variant is the Miller-Fisher Syndrome, manifesting with ophthalmoplegia, ataxia, and areflexia. Other forms of the disease exist such as purely motor, purely sensory, or one with both motor and sensory symptoms.

The tests that can aid in the diagnosis of GBS is the cerebrospinal fluid analysis done thru a lumbar tap and

electrodiagnostic studies such as the electromyography and nerve conduction velocity (EMG-NCV) studies. In the CSF, it is expected to see albuminocytologic dissociation, where the CSF has little to no cells, but with elevated protein count.[6] Most patients would present with abnormal EMG-NCV findings after 1 week and usual findings include prolonged distal latencies, reduction in the amplitude of muscle action potentials, slowed conduction velocity, and conduction block in motor nerves.[7]

Treatment includes general medical care and support. Ideally, these patients should be monitored closely for any sign of impending respiratory failure, dysautonomia, or rapidly progressing motor weakness. Specific treatment therapies include plasma exchange and IVIg especially in cases with rapid deterioration.

Majority of patients improve with only residual motor or sensory deficits. Around 2-10% of patients die during the first days of illness owing to respiratory compromise, infection, dysautonomia, or cardiac arrest.[8] Early initiation of treatment with rehabilitation should improve prognosis and recovery for most patients.

Bacterial meningitis, on the other hand, is a neurological disease characterized with inflammation of the meninges and invasion of the bacteria into the subarachnoid space. In severe cases of meningitis, the bacteria can spread to include the brain parenchyma (encephalitis), can seep into the ventricles (ventriculitis), and even the spinal cord.[9]

The classic triad of fever, headache, and altered mental status are present in 41% of cases of bacterial meningitis, and more commonly seen in the elderly.[10] Some present with focal neurologic deficits, behavioral changes, and seizure. The pathogens causing meningitis vary by age group, with *S. pneumoniae* and *N. meningitidis* being the most common among adults.

The diagnosis is made through CSF analysis or by clinical suspicion. Typical CSF findings include an elevated opening pressure, turbid appearance of the fluid, increased protein, decreased glucose, and neutrophilic predominance.[11] Cultures are not always positive. Treatment includes immediate antibiotic therapy and adjunctive use of corticosteroids.

CASE REPORT

We have presented with the case of a 36-year-old male from the Philippines who was admitted at East Avenue Medical Center due to a 5-day history of ascending bilateral lower extremity weakness. He initially reported a feeling of numbness and “pins-and-needles” sensation on both feet which rapidly progressed to motor weakness of both legs. After 3 days, the weakness involved his hips and upper extremities as well, so much that he could not stand up anymore. He also developed loose stools and urinary incontinence. This patient had no known illnesses and only underwent herniorrhaphy for inguinal hernia years back.

He arrived at the emergency room with stable vital signs but with noted 1/5 strength on both lower extremities and 4/5 on both upper extremities. Patient had intact sensory examination tested on different modalities such as touch, pain, temperature, and vibration, but noted to be areflexic.

He was managed as a case of GBS, started immediately on IVIg at 0.4 g/kg/day for 5 consecutive days. The paralysis progressed to involve the facial muscles as well leading to facial diplegia, but fortunately there was no respiratory compromise.

EMG-NCV was done with results consistent of an acute, severe sensorimotor polyradiculoneuropathy involving the upper and lower extremities consistent with that of GBS. Patient’s sensory nerve action potentials of the median and ulnar nerves are reduced while that of the radial and sural, superficial peroneal nerves were normal. The conduction velocities of the sural nerves and the superficial peroneal nerves were also decreased. Motor conduction was abnormal, showing reduced conduction velocity, prolonged distal latencies, and decreased amplitude. F-waves were prolonged and tibial H-reflex responses were absent on both sides. Needle EMG also showed increased duration, polyphasic motor unit potentials and decreased recruitment in the upper and lower extremities.

He underwent lumbar tap for CSF examination which showed yellowish turbid color, with an opening pressure of 16, and a closing pressure of 10. CSF analysis revealed 30×10^6 /l RBC, pleocytosis at 120 10^6 /l WBC with a total cell count of 150 10^6 /l and a 94% predominance of lymphocytes. Protein count was elevated at 2377 with a CSF/serum glucose ratio of 0.53.

It is not common to see this kind of CSF picture in cases of GBS. The CSF was then sent for bacterial panel testing where it tested positive for *N. meningitidis* Y, *N. meningitidis* W135, *Streptococcus* group B antigen, and *S. pneumoniae*. At this point, the patient had already completed 5 days of IVIg with significant improvement in motor strength. There were no signs and symptoms of infection present like fever or headache, and patient did not present with any meningeal signs as well. Patient was then requested HIV screening to rule out an immunocompromised state, but he tested negative. His CD4 count was 656 as well.

He was then treated to 10 days duration of ceftriaxone 2 gm every 12 hours and was subsequently discharged after 3 weeks with almost full recovery.

DISCUSSION

While any infectious agent can cause GBS, it is uncommon for bacterial meningitis to present symptomatically as ascending motor weakness, much more in an immunocompetent person. There are only a few reported cases of bacterial meningitis occurring concurrently with GBS. There is one case report published last 2018 citing development of GBS after a bout of bacterial meningitis from chronic suppurative otitis media (CSOM).[12] But the CSF analysis and blood cultures tested negative. The patient was started on IVIg on day 20 of hospital stay with significant improvement upon discharge.

Another case report published last 2022 documented a 49-year-old male developing GBS after *N. meningitidis* infection. He was also treated with a 5-day course of IVIg, although his stay at the hospital was much longer because he was intubated and stayed in the ICU. He was discharged after 21 weeks with MRS 1 functional outcome 2 years after that GBS bout. This patient had no other predisposing illnesses or condition that could have contributed to the development of meningococcemia.[13]

Another case report published last 2019 reported a woman developing GBS after Zika-virus associated aseptic meningitis diagnosed on CSF analysis and clinically. The patient was discharged well without any need for IVIg.[14]

For our patient, he did not present with any symptoms of meningitis and the disease was only discovered in the CSF analysis. It was unknown what predisposed him to developing meningitis, and why it did not present symptomatically. However, the temporal relationship between the development of GBS and the meningitis cannot be assumed and can only be speculated upon. This was one of the few cases of bacterial meningitis presenting as GBS in an immunocompetent person.

The exact pathophysiology was unknown. It may be due to invasion of the bacteria from the meninges then spreading to involve the spinal cord and peripheral nerves. Developing polyradiculopathy in the form of an acute inflammatory demyelinating disease is a severe yet rare consequence of bacterial meningitis, approximately 2% in some case series.[15] There was a reported case of *P. meningitis* presenting as GBS but patient had poor outcomes.

CONCLUSION

In conclusion, this kind of cases are rare. It is prudent to treat both the GBS and the bacterial meningitis to ensure good recovery and prevention of neurologic deterioration. In all cases of GBS, it may be beneficial to do a lumbar tap and send the CSF for complete analysis to ensure that doctors do not miss a diagnosis such as this especially in immunocompetent patients who do not present with the typical meningeal symptoms.

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