Guillain-Barre Syndrome Presenting with Acute Motor Sensory Neuropathy following a MRSA Infection: A Case Report

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ABSTRACT

A case report is presented of a 3-year-old boy who came with the complaints of weakness of lower limbs since 5 days and upper limbs since 3 days. On examination he had generalized hypotonia and areflexia. Power of the limbs was low with weak grip. Sensory and motor nerve conduction studies revealed acute motor axonal neuropathy involving both upper and lower limbs. Culture of blood and sputum revealed MRSA infection. Patient was managed with IV fluids and broad spectrum IV antibiotics. He was started on IvIg. The clinical manifestation of Guillain-Barre syndrome followed a combined MRSA infection. So this case report reveals an evolutionary significanceantecedent infection to the cause of Guillain-Barre syndrome.

Key words: Acute motor axonal neuropathy, Guillain-Barre Syndrome, IvIg, MRSA infection, Neurological disorder.

INTRODUCTION

Guillain-Barre Syndrome is an acute demyelinating disorder of the peripheral nervous system that results from an aberrant immune response directed at peripheral nerves. Guillain-Barre syndromes is an acute, immune-mediated disorder of peripheral nerves, spinal roots, and cranial nerves, commonly presenting as a rapidly progressive, areflexive, relatively symmetric ascending weakness of the limb, truncal, respiratory, pharyngeal, and facial musculature, with variable sensory and autonomic dysfunction. According to the Centers for Disease Control and Prevention (CDC), USA, approximately 1 to 2 individuals per 100,000 are affected by the syndrome. The UK National Health Service (NHS) reports that about 1,500 British people are affected annually, out of a population of 61 million. It is a little more common in men than women and can affect humans of all ages. Autonomic abnormalities in Guillain-Barre syndrome are usually transient and reversible.

CASE REPORT

History

A 3 years boy presented with history of weakness of lower limbs since 5 days followed by weakness of upper limbs since 3 days, associated with change of voice since 2 days, mild to moderate low grade intermittent fever, cough since 1 day, for the above complaints he was admitted.

Examination

He was a febrile, maintaining saturations at room air and hemodynamically stable heart rate was 130/min, blood pressure-68/30 mm hg and RR-20/min. CRT<2sec. Peripheries were warm and pulses were well felt. On auscultation of chest air entry was bilaterally equal with normal heart sounds abdomen was soft with no organamegally. On neurological examination, child was conscious, coherent and interactive pupils were bilaterally equal and reacting to light child had generalised hypotonia and areflexia. Power in lower limb and upper limb was 2/5 proximally and grip

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was weak, no meningeal signs and no signs of raised intracranial pressure. Examination of other systems was normal.

Management

He was admitted in ward and was started on IV fluids and broad spectrum IV antibiotics. He was started on IvIg (total dose -2 gm/kg).

His initial heamogram revealed Hb-9.0 gm%, WBCs–11,200 cells/cumm, platelets–3.88 lakh/cumm and CRP–42.9 mg/l. Serum electrolytes showed Na-135, K-5.0 and Cl-96. Serum creatinine was 0.57.

He was closely monitored in ward for any neurological and respiratory deterioration. As he developed respiratory distress (progressive respiratory muscle involvement), he was shifted to PICU for further management

He was shifted to PICU in view of progressive illness with possible respiratory compromise. As shoulder abduction was not possible with respiratory muscle involvement he was put on C-PAP support he was comfortable but he developed respiratory distress whenever he was off C-PAP. As he had high grade fever with right side patch on chest x ray, his antibiotics were upgraded after toileting, he was intubated and put on PCV-VG mode of ventilation with minimal possible settings his ventilator settings were optimised as per ABG`s. ET culture grew MRSA and hence gram positive cover was started.

Nerve conduction study showed significantly decreased CMAP amplitude with normal DLs and CVs in all tested nerves which is s/o acute motor axonal neuropathy involving both upper limbs (Table 1-3).

Close neurological monitoring was done throughout PICU stay. Parents were counselled regarding his condition and were regularly updated about the further management plan.

Gradually he improved and his fever spikes reduced with gradual improvement in power of shoulder internal and external rotators and his chest x ray was also improved. He was extubated after 48 hrs of ventilation and kept on low flow oxygen he tolerated extubation well and able to maintain saturation with low flow oxygen. After observing for 24 hrs, he was shifted to ward for further management.

During ward stay he remained neurologically stable and had no other neurological symptoms. Gradually his oxygen was tapered and stopped he started accepting orally and NG tube was removed. He improved with the above line of management and was discharged.

DISCUSSION

Guillain-Barre syndrome is a non-familiar inflammatory demyelinating disease of peripheral nerve that may be associated with extensive secondary axonal and even anterior horn cell degeneration.[1] GBS can cause life-

Table 1: Motor Nerve Conduction

Nerve and site	Latency	Amplitude	Segment	Latency Difference	Distance	Conduction Velocity
Median. L						
Wrist	1.0 ms	2.2 mV	Abductor pollicis breviswrist	1.0 ms	0 mm	0 m/s
Elbow	3.9 ms	1.2 mV	Wrist-elbow	2.9 ms	120 mm	41 m/s
Ulnar. L						
wrist	1.0 ms	0.1 mV	Abductor digiti minimi(manus)-wrist	1.0 ms	0 mm	0 m/s
Elbow	3.3 ms	0.4 mV	Wrist-elbow	2.3 ms	130 mm	57 m/s
Peroneal. L						
Ankle	2.3 ms	0.3 mV	Extensor digitorum brevisankle	2.3 ms	0 mm	0 m/s
Fibula	7.0 ms	0.1 mV	Ankle-Fibula(head)	4.7 ms	170 mm	36 m/s
Tibial. L						
Ankle	1.3 ms	2.4 mV	Abductor hallucis-Ankle	1.3 ms	0 mm	0 m/s
Popliteal fossa	6.3 ms	2.4 mV	Ankle-Popliteal fossa	5.0 ms	170 mm	34 m/s

Table 2: F-Wave Studies

Nerve	M-Latency	F-Latency
Median. L	3.9	-
Ulnar. L	3.3	-
Peroneal. L	7.0	-
Tibial. L	6.3	30.3

Table 3: Sensory Nerve Conduction

Nerve and Site	Onset Latency	Peak Latency	Amplitude	Segment	Latency Difference	Distance	Conduction Velocity
Median. L							
Digit II(index finger)	1.3 ms	2.0 ms	24 micro V	Wrist-Digit II (index finger)	1.3 ms	60 mm	45 m/s
Ulnar. L							
Digit V (little finger)	1.1 ms	3.3 ms	45 micro V	Wrist-Digit V(little finger)	1.1 ms	50 mm	46 m/s
Sural. L							
Lower leg	1.2 ms	1.6 ms	46 micro V	Ankle-Lower leg	1.2 ms	50 mm	42 m/s

threatening complications if the respiratory muscles are affected or if autonomic nervous system is involved. Epidemiology suggests annual incidence of 1.55/100,000 and incidence increases from 0.8/100,000 in patients younger than 35 to 4.67/100,000 in patients older than 75 years. Men are affected approximately 1.5 times more than women. There are no incidence studies of GBS in Indian population, but some case based studies have been reported.[2]

The onset of symptoms was abrupt following an upper respiratory tract infection. Our differential diagnosis pointed to a type of GBS, which initially appeared with a sensory disturbance (hot and cold sensation) but was followed by a quite atypical descending progression, or other post-infectious neurologic diseases, including HIV, CMV, HSV, EBV, WNV, C. jejuni, Haemophilus influenzae, Lyme disease and TBC. As suggested by Kaida et al. in a recent review, the mechanism of action of infectious microorganisms that induce GBS may be attributed to the molecular mimicry of lipo-oligosaccharide genes that are responsible for the formation of human ganglioside-like lipo-oligosaccharide structures. Other causes, such as Multiple sclerosis (MS) or other demyelinating syndromes, diabetes mellitus, B12 vitamin deficiency, drugs and chemical neuropathies were excluded following historical, clinical, laboratory and imaging tests. Examination for vasculitides, Sjögren's syndrome, sarcoidosis and paraneoplastic neuropathy proved negative.[3]

Most patients develop a weakness which tends to begin in the lower extremities due to demyelination of the peripheral nerves resulting in ascending paralysis and also a loss of cranialnerve function. Manifestations may be acute or chronic, and temporary or permanent, depending upon the degree of neuronal destruction. Muscle stretch reflexes are depressed in most patients and the sensory loss is variable. Difficulty with walking, running, climbing stairs, and getting up from a chair are usual early complaints. This weakness is usually symmetric and can also involve the upper extremities.[4] Treatment for patients with Guillain-Barre syndrome depends on whether they have mildly acute, severely acute or chronic involvement. The incidence of death in one study was 1.5% to 8% of patients.[5]

GBS is the most common peripheral neuropathy causing respiratory paralysis. Despite advances in respiratory management and immunotherapy, mortality from GBS is as high as 20% for ventilated patients. Mechanical ventilation is usually required by one third of the patients.[6]

Immunotherapy therapy has not reduced the mortality in GBS.[7] In a Cochrane systematic review of 6 trials with 587 patients it has been shown that corticosteroid therapy is ineffective for treating GBS.[8] IVIg, in a regimen of 0.4 g/kg bodyweight daily for 5 consecutive days, has replaced PE as the preferred treatment in many centers, mainly because of its greater convenience and availability.[9]

CONCLUSION

Guillain-Barre syndrome is a neurological disorder causing muscle paralysis. Patients may be mildly or

seriously affected by paralysis in which some percentage people will lead to death. The clinical manifestation of Guillain-Barre syndrome in our patient followed a combined MRSA infection. So this case report reveals an antecedent infection to the cause of Guillain-Barre syndrome.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

ABBREVIATIONS

ABG:	Arterial Blood Gases
CDC:	Centers for Disease Control and Prevention
CPAP:	Continuous positive airway pressure
CMV:	Cytomegalovirus
CMAP:	Compound Muscle Action Potential
EBV:	Epstein-Barr virus
GBS:	Guillain-Barre Syndrome
HIV:	Human immunodeficiency virus
HSV:	Herpes simplex virus
IVIG:	Intravenous immunoglobulin
MS:	Multiple sclerosis
NG:	Nasogastric
NHS:	National Health Service
WNV:	West Nile Virus

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