CASE REPORT

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Acute Motor Sensory Axonal Neuropathy following Viral Encephalitis: Sequential Immune Dysregulation

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Introduction

Guillain-Barré Syndrome(GBS) is recognised as an acute, post-infectious, immune-mediated polyneuropathy. It often occurs after an antecedent mucosal infections such as respiratory or gastrointestinal infection[1]. Triggers for the development of GBS include *Campylobacter jejuni*, Cytomegalovirus (CMV), and influenza[2]. Based on electrodiagnostic findings and pathological features, GBS can be broadly subclassified into Acute Inflammatory Demyelinating Polyradiculoneuropathy (AIDP), Acute Motor Axonal Neuropathy (AMAN), Acute Motor Sensory Axonal Neuropathy. Other related variants include Miller-Fischer Syndrome, Bickerstaff Brainstem encephalitis, Cervico-Pharyngeal-Brachial variant[3].

The proportional incidence of GBS subtypes varies significantly; while the demyelinating form is predominant in North America and Europe, the axonal variants are more prevalent in Asia and Central/South America. AMSAN is one of the rarest forms of GBS, accounting for less than 5% of all cases[4]. The axonal variants present with more profound deficits having a less favourable prognosis compared to demyelinating forms as the recovery in axonal variants involves incomplete axonal regeneration rather than simple remyelination[5].

Case Presentation

A 77-year-old female, presented with a 4-day history of fever followed by altered mental status, characterised by irrelevant speech, and aggressive behaviour. Physical Examination revealed a conscious delirious patient with positive signs of meningeal irritation such as neck rigidity. Motor strength in all four limbs was found to be MRC grade 5/5. The initial Diagnostic Workup including routine labs and fever panel were unremarkable, ruling out common bacterial or septic causes. Magnetic Resonance Imaging (MRI) of brain with contrast showed no acute intraparenchymal lesions or significant leptomeningeal enhancement (Fig. 1). Cerebrospinal Fluid (CSF) Analysis demonstrated lymphocytic pleocytosis (250 cells/μL) with an elevated protein level (95.4 mg/dL) Table I.

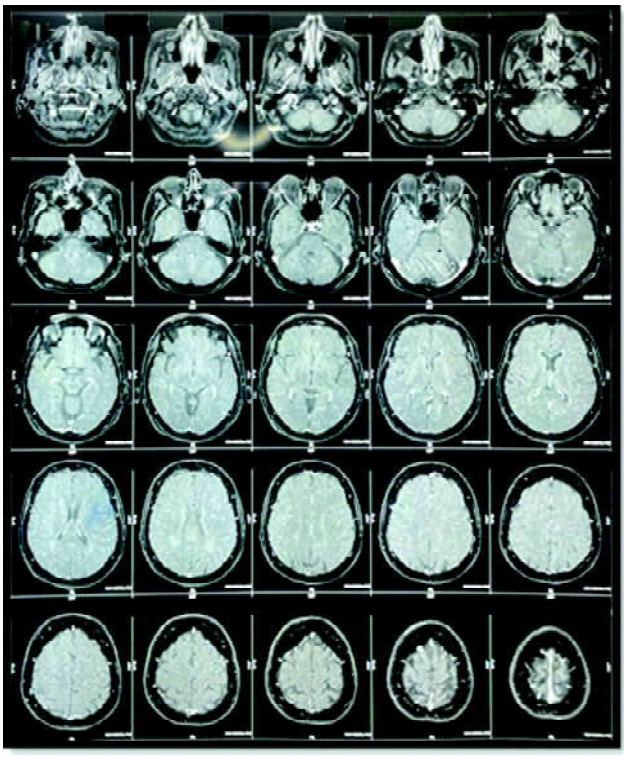

Fig. 1: *Initial MRI Brain with contrast study.*

Table I: CSF analysis.

Initial CSF analysis			
Parameter	**Patient result**	**Reference range**	**Unit**
CSF cell count	250	0 - 5	cells/μL
CSF protein	95.4	15 - 45	mg/dL
Repeat CSF analysis			
Parameter	**Patient result**	**Reference range**	**Unit**
CSF cell count	50	0 - 5	cells/μL
CSF protein	98.6	15 - 45	mg/dL

**Abbreviations* CSF: Cerebrospinal fluid.*

Based on the clinical syndrome (fever, encephalopathy, meningeal signs) and the CSF profile with elevated proteins

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and cell count, a presumptive diagnosis of Viral Encephalitis was made. Intravenous Acyclovir (10 mg/kg body weight) was initiated. By day 2 of hospitalisation the patient responded well to Acyclovir. Her mental status had improved, she was oriented and obeying commands. The patient was shifted to ward and continued on intravenous Acyclovir.

On Day 7 of hospitalisation, the patient experienced an acute-onset ascending motor weakness. She subsequently developed urinary retention requiring catheterisation and labile blood pressure (indicating an autonomic dysfunction).

On examination: hypotonia was noted in all 4 limbs with reduced Motor Strength: Bilateral Lower Limbs MRC Grade 3/5 and Bilateral Upper Limbs MRC Grade 4/5. The new symptom constellation prompted re-evaluation for a post-infectious process. MRI Brain with whole spine was performed and the study remained unremarkable, excluding acute stroke, myelitis or compressive lesions (Fig. 2, 3). Repeat CSF analysis was performed and the analysis showed protein: 98.6 mg/dL and WBC Count: 50 cells/μL (Table II).

Table II: Nerve conduction study.

Motor Nerve Conduction Study					
Nerve	**Recording Site (Muscle)**	**Stimulation Site**	**Latency (ms)**	**Amplitude (mV/μV)**	**Conduction Velocity (m/s)**
Median (L)	APB	Wrist	3.25	4.38 mV	68.97
		Elbow	6.88	3.48 mV	68.97
Median (R)	APB	Wrist	3.13	2.44 mV	68.97
		Elbow	6.75	1.62 mV	68.97
Peroneal (L)	EDB	Ankle	0.00	0.00 μV	–
		Knee	0.00	0.00 μV	–
Peroneal (R)	EDB	Ankle	2.50	0.91 mV	54.90
		Knee	8.88	0.68 mV	54.90
Tibial (L)	EHL	Ankle	0.00	0.00 μV	–
		Popliteal Fossa	0.00	0.00 μV	–
Tibial (R)	EHL	Ankle	14.00	2.83 mV	95.48
		Popliteal Fossa	17.88	0.73 mV	95.48
Sensory Nerve Conduction Study					
Nerve	**Side**	**Recording Site**	**Peak Latency (ms)**	**Amplitude (mu\μV)**	**Interpretation**
Superficial Peroneal	Left	Ankle	NR	0.0	Absent Response (Severe Axonal Loss)
Superficial Peroneal	Right	Ankle	NR	0.0	Absent Response (Severe Axonal Loss)
Sural	Left	Ankle	2.15	11.4	Normal
Sural	Right	Ankle	NR	0.0	Absent Response (Severe Axonal Loss)
Median	Left	Digit 2	2.40	30.7	Mild slowing, Amplitude preserved
Median	Right	Digit 2	2.80	30.6	Mild slowing, Amplitude preserved
Ulnar	Left	Digit 5	2.45	26.3	Mild slowing, Amplitude preserved
Ulnar	Right	Digit 5	2.70	24.4	Mild slowing, Amplitude preserved
Radial	Left	Forearm	1.20	25.0	Normal
Radial	Right	Forearm	1.60	18.1	Normal

The combination of rising protein and resolving pleocytosis formed the basis for albuminocytological dissociation, a cardinal feature of GBS. Electrophysiological studies (Table III, IV) revealed uniformly absent or reduced muscle action potential amplitudes in Peroneal and Tibial nerve and reduced sensory nerve action potential in the superficial peroneal and sural nerves. These findings are consistent with acute motor and sensory axonal polyradiculoneuropathy, a severe axonal variant of GBS.

A final diagnosis of Post-Viral Encephalitis AMSAN variant of GBS was established. The patient was initiated

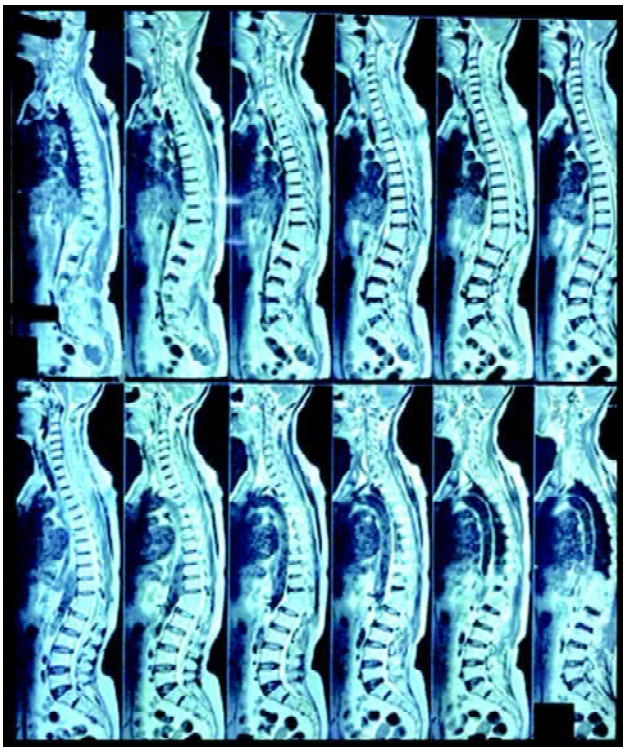

Fig. 2: *MRI whole spine screening.*

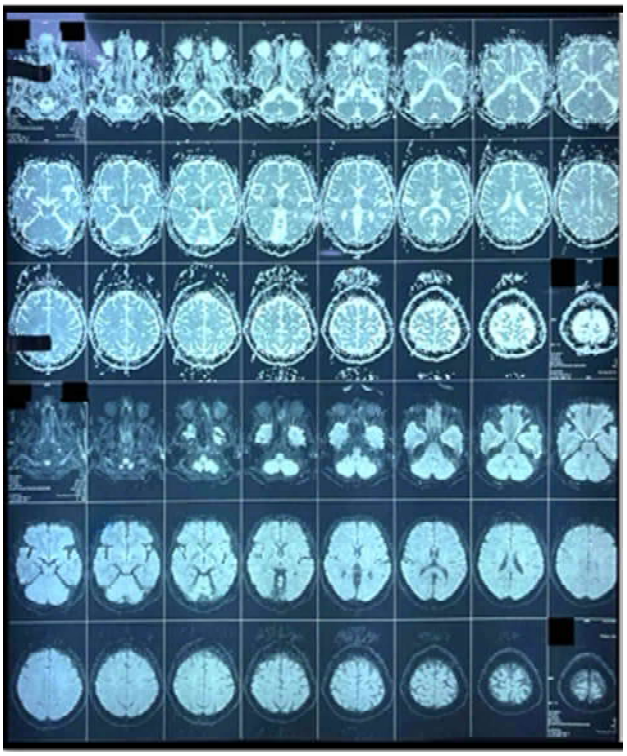

Fig. 3: *Repeat MRI brain study.*

Intravenous Immunoglobulin (IVIg)(at the standard total dose 2 g/kg body weight, totalling 120 g) administered over 5 days. By the completion of the IVIg course, the patient demonstrated gradual clinical improvement in motor strength in the bilateral lower limbs with power of MRC Grade 4/5.

Discussion

The occurrence of GBS following severe CNS inflammation as seen in our patient, challenges the conventional mucosal para-infectious paradigm.

The rapid transition noted in our patient from encephalitis to the AMSAN variant of GBS can be speculated to be a result of either a shared antigenic determinant between the CNS and PNS or a profound immune response resulting in disruption of the blood brain barrier thus allowing a CNS inflammation to trigger a secondary cascade in the periphery.

The hypothesis of 'immune spillover' is further strengthened by case reports documenting development of GBS following intense CNS insults such as cerebrovascular accidents. The common pathological link in these analogous cases is disruption of blood brain barrier hypothesised to permit an immune spillover[6].

A study conducted by Yang *et al* demonstrated varying degrees of axonal degeneration and demyelination in sciatic nerve of a mouse model infected with Japanese encephalitis. This study established a link between and a primary CNS inflammation triggering an immune response in the periphery[7]. Similarly, Tan *et al* conducted a sural nerve biopsy on a patient who developed GBS one week after traumatic brain injury. The presence of foamy macrophages in the endoneurium provided a direct histopathological evidence of immune mediated destruction in PNS following an acute CNS insult[8].

The most instructive element of our case is the evolution of the CSF profile. The initial CSF analysis, characterised by marked lymphocytic pleocytosis (250 cells/muL) and elevated protein (95.4 mg/dL), strongly indicated an active viral neurotropic infection despite lack of leptomeningeal enhancement in the MRI study. Improvement in patient's sensorium following Acyclovir also strengthened the diagnosis of viral encephalitis. A study conducted by Sukumaran *et al* (2024) demonstrated that 38.1% patients with infectious encephalitis presented with normal MRI findings, underscoring the importance of biochemical assessment, especially in hyperacute phase of illness[9].

The new onset neurological deterioration on day 7 of hospitalisation in the backdrop this unique CSF picture of resolving CNS inflammation juxtaposed with rising protein effectively ruled out ongoing encephalitis as the cause of the new weakness. Furthermore, the Electrodiagnostic

study revealed an absent motor and sensory responses in left peroneal and tibial nerve, with reduced amplitude in the right leg confirming the diagnosis of Acute Motor and Sensory Axonal Neuropathy.

The constellation of progressive ascending flaccid quadriparesis (MRC Grade 3/5), intense dysesthesia, and rapid progression was consistent with the severe axonal pathology. The development of urinary retention with labile blood pressure highlights significant autonomic dysfunction – an indicator of the severity of the disease. Autonomic dysfunction occurs in approximately 70% of patients with axonal variants of GBS[10].

The rapid clinical response following IVIG, with bilateral lower limb power improving from MRC Grade 3/5 to MRC Grade 4/5 by the end of the treatment course underscores the importance of high clinical suspicion and timely intervention.

Conclusion

This case expands the clinical spectrum of GBS from a mucosal para-infectious immune-mediated response by documenting AMSAN variant GBS following viral encephalitis in an elderly patient. The temporal evolution from an initial CNS pleocytosis to a subsequent albumino-cytological dissociation supports the possibility of CNS mediated immune response contributing to a PNS injury. Though such causality cannot be established from a single case, such observations highlight the need to explore the spectrum of post CNS insult immune dysregulation as an uncommon but possible pathway of GBS.

References

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