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RESEARCH ARTICLE
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LOCALIZED SCALP RASH FOLLOWING RITUXIMAB INFUSION IN AN INFANT WITH ACUTE FLACCID MYELITIS: A CASE REPORT

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Abstract

Background: Acute Flaccid Myelitis (AFM) is a rare, severe neurological disorder characterized by sudden limb weakness, usually with presumed autoimmune etiology. Immuno-modulatory therapy, including rituximab, is sometimes used off-labely. Cutaneous adverse drug reactions to rituximab can occur but are less frequently reported as localized scalp rashes, particularly in infants.

Case Presentation: A 3-month-24-day-old male infant weighing 5kg developed progressive left lower limb monoparesis. Nerve conduction study showed severe axonal and demyelinating neuropathy. MRI and negative CSF PCR showed, anti-AQP4, and anti-MOG antibody confirmed a probable post-viral or autoimmune etiology of AFM diagnosis. The patient was started on intravenous rituximab (100 mg), but within the initial first 10–20 mL of infusion, the infant developed a hypersensitivity reaction. The reaction was erythematous, pruritic maculopapular rash localized to the scalp, with no systemic symptoms. The infusion was immediately stopped, and the rash resolved completely within 48–72 hours following treatment with intravenous antihistaminics and corticosteroids. The adverse drug reaction was subsequently reported to VigiFlow.

Conclusion: This report presents a unique, localized cutaneous hypersensitivity reaction to rituximab in an infant, and attention to the importance of careful monitoring during initial infusions and highlighting the value of ADR reporting in paediatric biologics therapy.

Keywords: Rituximab, Acute Flaccid Myelitis, Scalp Rash, Drug Eruption, Infant, Adverse Drug Reaction, Paediatric, Hypersensitivity

Introduction

Rituximab is a chimeric monoclonal antibody against the CD20 antigen expressed on the surface of B lymphocytes and functions by causing depletion of B-cells via antibody-dependent cellular cytotoxicity, complement-mediated cytolysis, and apoptosis. Rituximab is utilized in the treatment of several B-cell malignancies and autoimmune diseases [1]. Its application is in adults, in diseases such as non-Hodgkin lymphoma, rheumatoid arthritis, and neuromyelitis optica spectrum disorders (NMOSD). However, its use within the paediatric population remains limited and frequently off-label, held in reserve for resistant or severe immune-mediated illness [2,3].

In paediatric neurology, Rituximab has been used to manage diseases like autoimmune encephalitis, MOG antibody-associated condition, and post-infectious transverse myelitis [4] . The initiation of rituximab, immunotherapeutic agent requires a judicious risk-benefit analysis, for usage in infants. In paediatric patients immaturity of the immune system can affect efficacy and susceptibility to toxicities. Hypersensitivity reactions are one of the most frequent infusion-related adverse reactions to Rituximab, usually manifesting with the first infusion because of cytokine release or immune complex formation. [5]

The case describes a 3-month-24 days-old male child who had progressive left lower limb monoparesis, diagnosed eventually as a likely post-infectious or autoimmune inflammatory myelopathy. Rituximab was started as immunomodulatory treatment. Nevertheless, an acute hypersensitivity reaction was encountered shortly after the infusion was started, highlighting the need for careful monitoring and premedication measures in biologic therapy, especially in extremely young children.

Case presentation

A 3-month-24 days -old male infant, weighing 5 kg with a body surface area of 0.28 m^2, was brought to the hospital with a history of progressive weakness in the left lower limb. The weakness was insidious in onset and had been worsening over the course of several days. On examination, the child was hemodynamically stable with a heart rate of 112 beats per minute, respiratory rate of 28 breaths per minute, and capillary refill time of less than 3 seconds. Neurological assessment revealed diminished movement of the left lower limb, which remained in an abducted posture. Sensory examination was intact. No other neurological deficits were noted in the upper limbs or right lower limb, and cranial nerves were grossly normal.

To evaluate the motor deficit, nerve conduction studies were conducted. Motor nerve conduction study showed no response (N.R.) from the left peroneal and left tibial nerves, suggesting significant motor pathway involvement. The Left Median Nerve (APB) and the Left Ulnar Nerve (ADM) appears largely within normal limits, with a distal motor latency (DML) of 2.04 ms and 1.56 ms respectively. However, the Right Ulnar Nerve (ADM) displays a significantly prolonged distal motor latency of 7.44 ms and a very low amplitude of 1.1 mV. The reduced amplitude indicates significant axonal loss. In contrast, the lower limb motor nerves, specifically the Left Peroneal - EDB and Left Tibial - AH, show "No Response" (NR) for all measured parameters (latency, amplitude, velocity) from both distal (ankle) and proximal (fibular head/knee) stimulation points. This indicates a severe, possibly complete, absence of motor responses in these nerves, strongly suggesting significant axonal neuropathy or severe demyelination, preventing measurable conduction.

Sensory nerve conduction studies reveal severely abnormal findings for the left median and left ulnar nerves, both demonstrating significantly prolonged latencies and markedly reduced conduction velocities (7 m/s for median, 6 m/s for ulnar), indicating severe demyelination or axonal loss affecting these nerves. In contrast, the left sural nerve shows a relatively preserved response with a normal amplitude (111.9 μV) and a conduction velocity (29 m/s) that is within the lower range of normal or mildly reduced, suggesting it is relatively spared compared to the upper limb nerves.

These nerve conduction studies point towards a severe peripheral neuropathy, predominantly axonal and demyelinating, affecting the bilateral lower extremities, with an absence of motor responses in the left peroneal and tibial nerves. In the upper extremities, there is evidence of severe right ulnar motor neuropathy, left median sensory neuropathy, and left ulnar sensory neuropathy, all indicating

significant involvement of these peripheral nerves. The prolonged F-waves further suggest proximal involvement or a generalized polyneuropathy **[Fig 2].**
Further workup included cerebrospinal fluid (CSF) analysis, which revealed 2 mL of clear fluid with lymphocytic pleocytosis, indicating a possible viral or autoimmune etiology. A meningitis-encephalitis PCR panel was performed and was negative for common pathogens including *Haemophilus influenzae*, *Listeria monocytogenes*, *Neisseria meningitidis*, cytomegalovirus (CMV), and Enterovirus. Serum procalcitonin measured on 26th April 2025 was negative, helping to rule out an acute bacterial infection.

Given the neurological presentation, autoimmune markers were evaluated. Serum anti-aquaporin 4 (NMO) IgG and anti-myelin oligodendrocyte glycoprotein (MOG) IgG antibodies were both negative. MRI of the spine revealed a mild fusiform expansion of the cervical and lower dorsal spinal cord, including the conus medullaris, along with subtle intramedullary hyperintensity on T2-weighted images—findings consistent with an inflammatory myelopathy.

A provisional diagnosis of acute flaccid myelitis (AFM), likely post-viral or autoimmune in origin, was made based on the clinical presentation, neurophysiological findings, spinal MRI, and negative infectious and autoimmune serologies, including CSF PCR, anti-AQP4, and anti-MOG antibodies. Differential diagnoses such as Guillain-Barré Syndrome, transverse myelitis, and poliomyelitis were considered but were deemed less likely due to asymmetric weakness, preserved reflexes, characteristic MRI findings, and negative viral PCR.After reviewing the MRI findings and excluding infectious etiologies, a decision was taken to initiate immunomodulatory therapy with intravenous Rituximab.

A dose of 100 mg Rituximab was infused over 4 hours. However, within the first 10–20 mL of infusion, the child developed a hypersensitivity reaction characterized by erythematous rashes over the head **[Fig 1]**. The rash appeared within first 10–20 mL of infusion of initiating the rituximab infusion and was characterized by multiple erythematous, mildly edematous maculopapular lesions localized to the scalp, predominantly over the vertex and bilateral parietal regions. The lesions were discrete to mildly confluent, warm to touch, and mildly pruritic, without scaling, vesiculation, or crusting. There was no mucosal involvement, facial swelling, or systemic signs such as fever, respiratory distress, or hypotension.

The remainder of the body surface was unaffected. The infusion was immediately discontinued, and the patient was managed with intravenous pheniramine and hydrocortisone (50 mg). The rash resolved completely within 48–72 hours without sequelae, and the patient remained stable thereafter. The child's condition stabilized by the evening with no further adverse effects. The child's condition stabilized with no further adverse effects. The ADR was formally submitted to VigiFlow via the Pharmacovigilance Programme of India.

Discussion

Rituximab, a chimeric monoclonal antibody targeting the CD20 antigen on B lymphocytes, has become an important therapeutic agent in the management of various autoimmune diseases, hematologic malignancies, and inflammatory conditions such as nephrotic syndrome and myelitis. Although generally well tolerated in paediatric populations, infusion-related hypersensitivity reactions remain a well-documented adverse effect, especially during the first exposure.

In this case, a 3-month-24 days -old male infant with suspected autoimmune or post-infectious inflammatory myelopathy developed an acute hypersensitivity reaction within the initial 10–20 mL of his first Rituximab infusion. The reaction was characterized by erythematous rashes over the scalp and was promptly managed with intravenous antihistamines and corticosteroids, leading to stabilization of the patient's condition.

Infusion reactions associated with Rituximab are most commonly attributed to cytokine release and can range from mild cutaneous manifestations to severe anaphylaxis. These reactions typically occur within the first few hours of the initial dose and are thought to be mediated by a combination of immune complex formation, complement activation, and direct cytokine release from lysed B cells.

In infants and younger children, the immaturity of the immune system may alter the typical presentation and severity of such reactions, necessitating heightened vigilance. As per the WHO-Uppsala Monitoring Centre (WHO-UMC) causality assessment system, the adverse drug reaction in this case was classified as Probable. This was based on the clear temporal relationship between the administration of rituximab and the onset of the rash, the absence of alternative explanations such as infection or concurrent medications, and the resolution of symptoms upon withdrawal of the drug (positive dechallenge). No re-challenge was attempted due to the patient's age and the nature of the reaction. The patient was discharged in stable condition without any post-infusion complications **[Fig 3].** .

Pre-medication with corticosteroids and antihistamines is generally recommended to reduce the risk of infusion reactions. However, in very young patients like this infant, the use of Rituximab is often off-label and evidence regarding appropriate pre-medication protocols remains limited[11,12]. Furthermore, underlying inflammation of the central nervous system may predispose to an exaggerated immune response.

Although cutaneous hypersensitivity reactions are usually non-life-threatening, they should prompt immediate cessation of the infusion and appropriate management to prevent escalation. Re-challenging with Rituximab in such patients requires careful risk-benefit analysis and consideration of desensitization protocols or alternative immunotherapies.

Conclusion

This case highlights a rare but important adverse reaction to Rituximab in a 3-month-24 days -old infant with suspected autoimmune inflammatory myelopathy. While Rituximab is increasingly used as an off-label immunomodulatory agent in paediatric neuroinflammatory conditions, clinicians should remain vigilant for hypersensitivity reactions, even during the initial infusion. Early identification and prompt management of such reactions are critical to patient safety. This case emphasizes the critical need for close monitoring during biologic therapy, readiness to manage potential adverse reactions and the need for further studies evaluating the safety profile of Rituximab in infants and young children.

Authors' contributions SM managed the clinical case. AK and HS collected data, reviewed the case, and prepared the initial manuscript draft. VV and SG provided intellectual input, supervision, and critically revised the manuscript. All authors contributed to manuscript revision, approved the final version, and agree to be accountable for all aspects of the work.

Conflict of interest None

Funding None.

Availability of data and materials

All the data used and or analyzed during case report development have been included in the case presentation.

Consent

Written informed consent for publication of this case report and accompanying images was obtained from the patient's parent/legal guardian.

Ethics approval Not applicable.

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Abbreviations:
AFM: Acute Flaccid Myelitis; ADR: Adverse Drug Reaction; AQP4: Aquaporin-4; BSA: Body Surface Area; CD20: Cluster of Differentiation 20; CMAP: Compound Muscle Action Potential; CSF: Cerebrospinal Fluid; EDB: Extensor Digitorum Brevis; ENMG: Electroneuromyography; IV: Intravenous; ICSR: Individual Case Safety Report; MOG: Myelin Oligodendrocyte Glycoprotein; MRI: Magnetic Resonance Imaging; ms: Millisecond; µV: Microvolt; NCS: Nerve Conduction Study; NMOSD: Neuromyelitis Optica Spectrum Disorder; NR: No Response; PvPI: Pharmacovigilance Programme of India; APB: Abductor Pollicis Brevis; ADM: Abductor Digiti Minimi; DML: Distal Motor Latency

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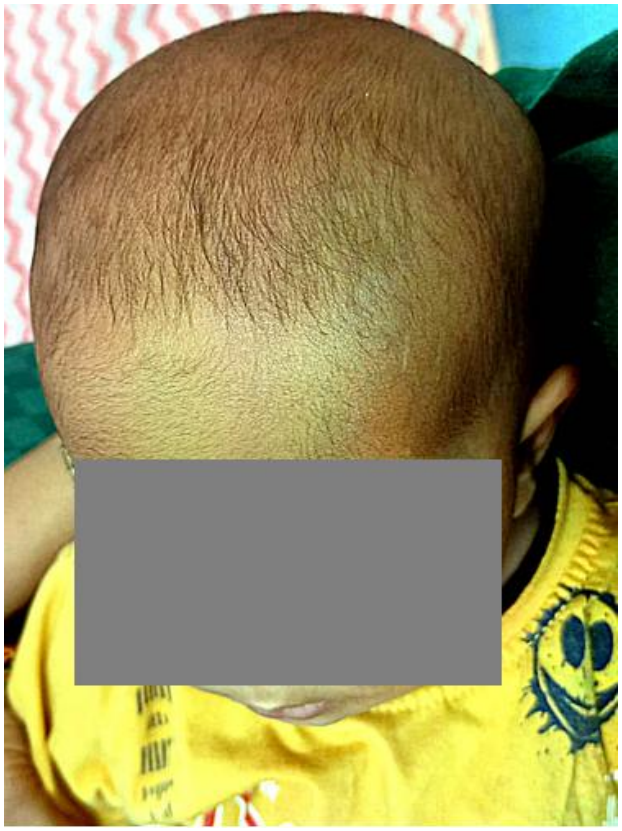

FIGURE 1: Erythematous rash localized over the scalp observed shortly after initiating rituximab infusion in the infant.

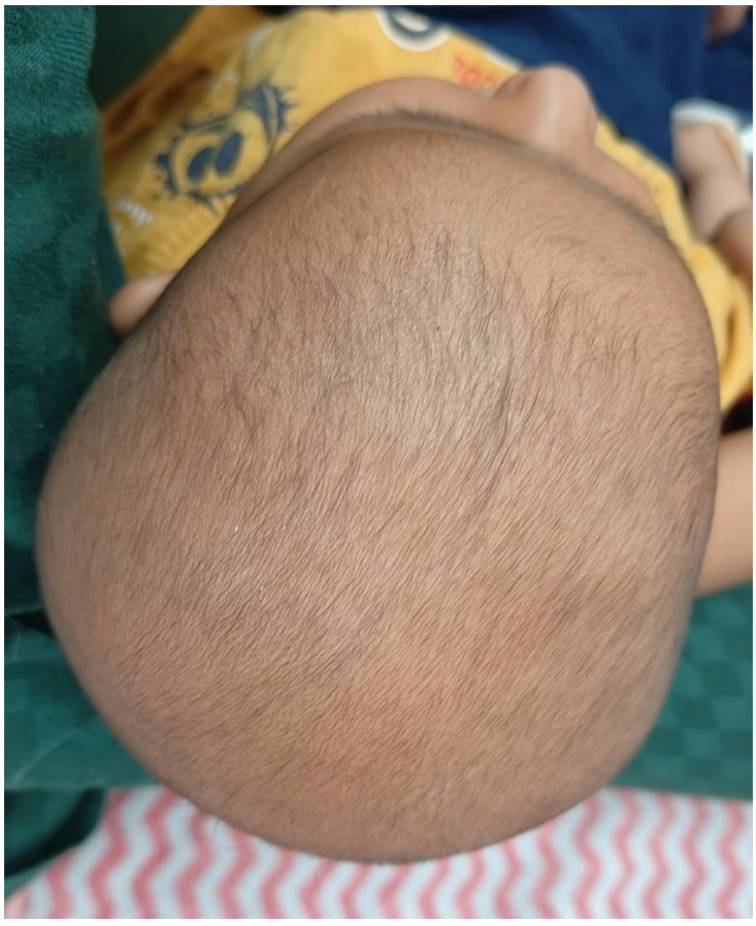

FIGURE 2: Follow-up image showing resolved scalp rash 72 hours post infusion

Figure 3a) Motor nerve conduction study (Motor nerve conduction study showing normal responses in the left median and ulnar nerves, but significantly prolonged latency and reduced amplitude in the right ulnar nerve, indicating axonal loss. No responses were recorded from the left peroneal and tibial nerves, suggestive of severe axonal neuropathy or demyelination.**)**

NERVE/Sites	Muscle	Latency ms	Amplitude mV	Segments	Distance mm	Lat Diff ms	Velocity m/s
Left Median-APB							
Wrist	APB	2.04	5.5	Wrist-APB			
Elbow	APB	3.88	3.1	Elbow-Wrist	80	1.83	44
Right Ulnar- ADM							
Wrist	ADM	7.44	1.1	Wrist-ADM	80		
B.Elbow	ADM	11.25	0.5	B.Elbow-Wrist		3.81	
Left Ulnar- ADM							
Wrist	ADM	1.56	2.8	Wrist-ADM			
B.Elbow	ADM	3.83	1.7	B.Elbow-Wrist	80	2.27	35
Left Peroneal- EDB							
Ankle	EDB	NR	NR	Ankle-EDB	80		
B.Fib Head	EDB	NR	NR	B.Fib Head-Ankle		NR	
Left Tibial-AH							
Ankle	AH	NR	NR	Ankle-AH	80		
Knee	AH	NR	NR	Knee-Ankle		NR	

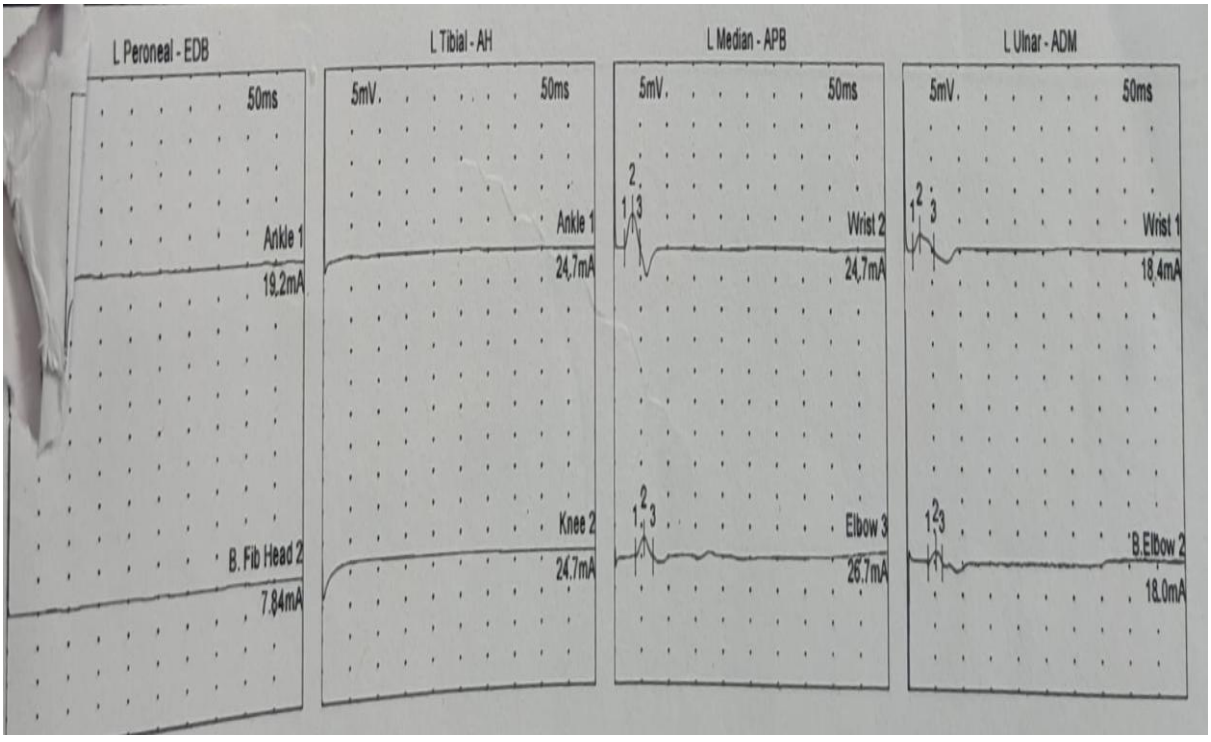

Figure 3b) Sensory Nerve Conduction Study (Sensory nerve conduction study showing prolonged latencies and reduced amplitudes in the left median and ulnar nerves, consistent with demyelination and axonal loss. The left sural nerve shows relatively preserved amplitude and velocity, suggesting sparing or only mild involvement.)

NERVE/Sites	Rec. Site	Latency ms	PP Amplitude (µV)	Segments	Distance mm	Velocity m/s
Left Median-Dig II (Antidromic)						
Wrist	Index	7.66	20.5	Wrist-Index	50	7
Left Ulnar- Dig V (Antidromic)						
Wrist	Dig V	8.70	48.4	Wrist-Dig V	50	6
Left Sural- (Antidromic)						
Calf	Ankle	4.79	111.9	Calf-Ankle	140	29

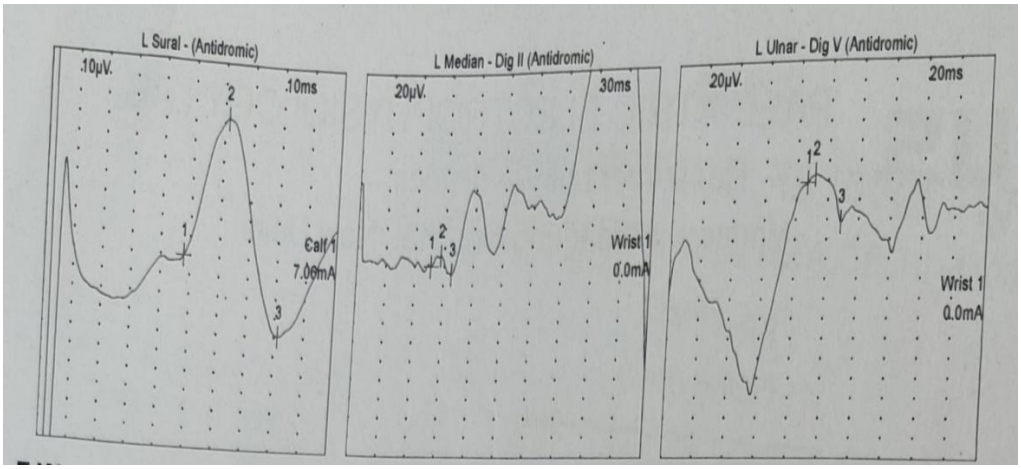

Figure 3c) F Waves (F-wave studies showing prolonged latencies in the left median and ulnar nerves, indicating proximal nerve involvement and supporting a generalized polyneuropathy.)

Nerve	M Latency ms	F Latency ms	F-M Lat ms
L Median-APB	2.5	26.7	24.2
L Ulnar- ADM	3.3	31.9	28.5

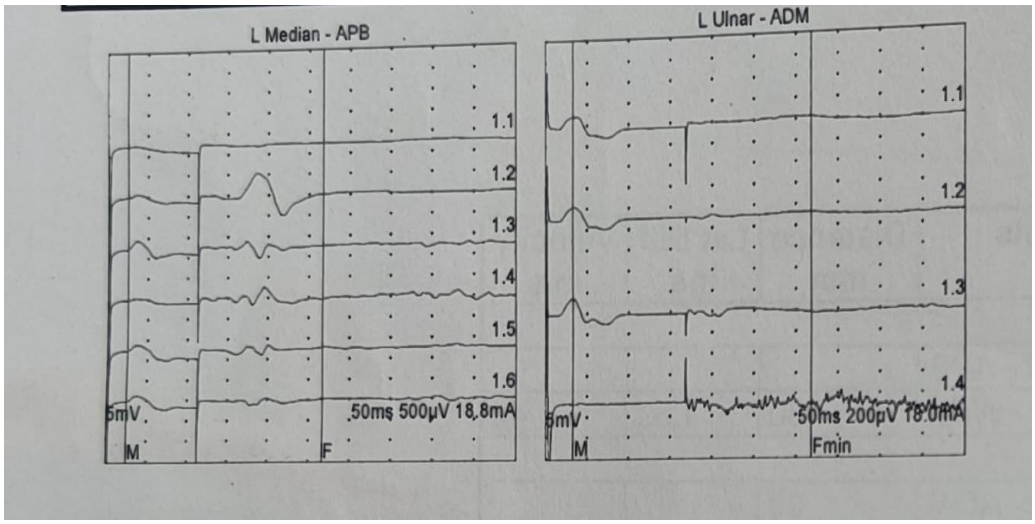

FIGURE 3: Nerve conduction study points to a widespread, severe polyneuropathy with predominant demyelinating features- axonal loss.