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Guillain-Barré Syndrome with Overlapping Features of AMAN and PCB Variant : A Case Report

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Introduction

Guillain-Barré syndrome (GBS) encompasses acute immune mediated neuropathies. A rare variant, pharyngeal-cervical-brachial (PCB), produces oropharyngeal and cervicobrachial weakness while sparing the lower limbs. Diagnosis is challenging, particularly when overlapping with acute motor axonal neuropathy (AMAN). Early electrophysiology may show reversible conduction failure (RCF), mimicking demyelination before axonal degeneration. We report a case of AMAN-PCB overlap in a patient with gastric cancer, initially obscured but clarified through rehabilitation assessment and serial electrophysiological monitoring.

Case report

A 76 years old male with stage IIIC gastric cancer was admitted for a scheduled chemotherapy cycle. Three days earlier, after an episode of diarrhea, he developed rapidly progressive muscle weakness, prompting deferment of chemotherapy and transfer to neurology. Examination revealed disproportionate weakness. Upper limbs (medical research council (MRC) 1–2) were more affected than lower limbs (MRC 3–4). Initial nerve conduction studies (NCS) on hospital day 2 showed widespread conduction blocks and slowed velocities (Table 1), initially interpreted as acute inflammatory demyelinating polyneuropathy (AIDP). He was treated with a five day course of intravenous immunoglobulin. On hospital day 9, he was transferred to rehabilitation medicine. Assessment revealed persistent weakness of the neck and shoulder girdle with relatively preserved lower limbs.

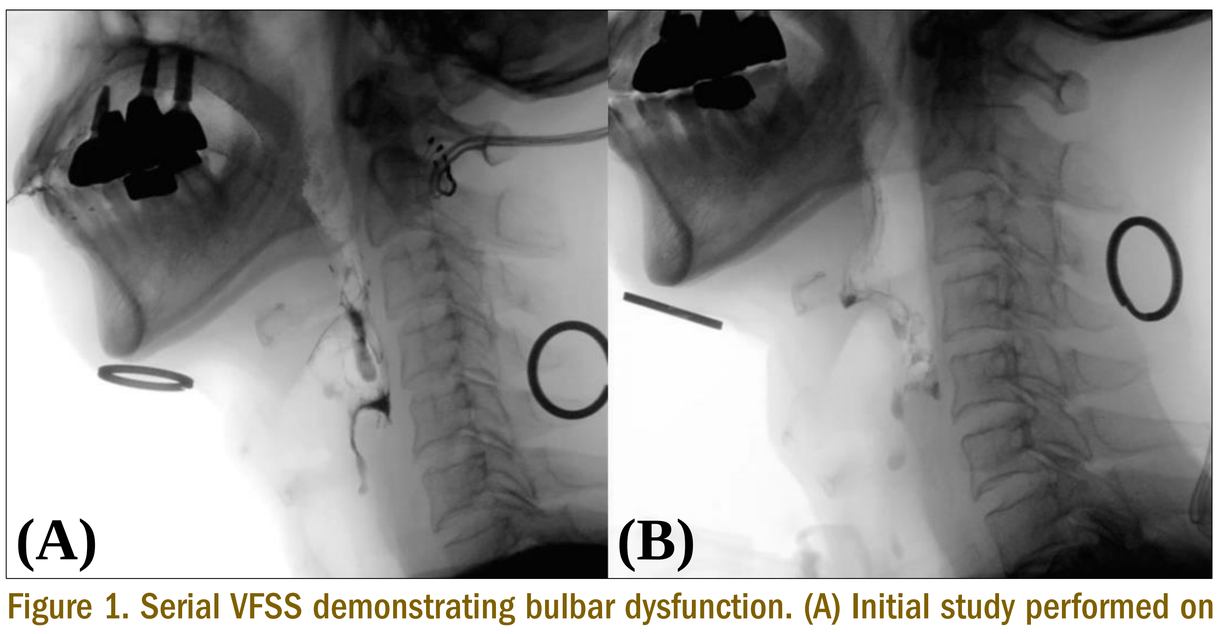

Figure 1. Serial VFSS demonstrating bulbar dysfunction. (A) Initial study performed on February 5, 2025 showing liquid tracheal aspiration, (B) Follow up study on January 13, 2026 confirming persistent and severe aspiration, indicating unresolved bulbar palsy despite the recovery of neck muscle strength

Videofluoroscopic swallowing study (VFSS) showed bulbar dysfunction with tracheal aspiration (Fig. 1), and pulmonary function tests indicated critical respiratory compromise (maximal inspiratory pressure 35%, maximal expiratory pressure 30%), suggesting phrenic nerve involvement. Anti-ganglioside antibody assays and follow up electrodiagnostic studies were performed. The patient developed acute respiratory failure with hypercapnia, requiring intubation and tracheostomy. Follow up NCS showed previously blocked nerves had become electrically inexcitable with loss of compound muscle action potential amplitudes (Table 2), and diaphragm electromyography confirmed active denervation. These findings reclassified the pathology from AIDP to AMAN, confirming that the initial conduction blocks were RCFs. Serology was positive for anti-GM1 and anti-GD1b antibodies, supporting a diagnosis of AMAN-PCB overlap syndrome.

Table 1. Initial nerve conduction study

Sensory nerve action potential

Nerve	Stimulation site	Peak latency (ms)	Amplitude (μV)	Site 1	Site 2	Velocity (m/s)
Left superficial peroneal anti sensory	14 cm	3.9	10.6	14 cm	Ant Lat Mall	36
Right superficial eroneal anti sensory	14 cm	4.1	11.5	14 cm	Ant Lat Mall	34
Left sural anti sensory	Calf	2.8	12.2	Calf	Lat Mall	50
Right sural anti sensory	Calf	3.0	12.9	Calf	Lat Mall	47
Right median ortho sensory	2nd digit	2.3	26.1	2nd digit	Rec	61
	Palm	1.7	62.4	Palm	Rec	47
	Wrist	4.5	18.1	Wrist	Rec	53
	Elbow	1.9	65.8	Elbow	Rec	58
Right ulnar ortho sensory	5th digit	2.2	18.8	5th digit	Rec	41
	Wrist	4.0	21.3	Wrist	Rec	65
	Elbow	1.8	60.1	Elbow	Rec	67

Compound muscle action potential

Nerve	Stimulation site	Onset latency (ms)	Amplitude (μV)	Site 1	Site 2	Velocity (m/s)
Right median	Wrist	3.3	5.9	Wrist	Elbow	45
	Elbow	8.8	1.5	Elbow	Axilla	48
	Axilla	11.3	0.8			
Right ulnar	Wrist	2.3	5.3	Wrist	B Elbow	51
	B Elbow	7.2	3.6	B Elbow	A Elbow	48
	A Elbow	9.5	2.0	A Elbow	Axilla	143
	Axilla	10.2	2.0			
Left peroneal	Ankle	3.7	2.8	Ankle	B Fib	38
	B Fib	12.5	1.5	B Fib	Poplt	180
	Poplt	13.0	1.2			
Right peroneal	Ankle	4.1	3.1	Ankle	B Fib	36
	B Fib	13.2	2.1	B Fib	Poplt	90
	Poplt	14.2	2.0			
Left tibial	Ankle	3.7	8.9	Ankle	Knee	40
	Knee	12.5	4.7			
Right tibial	Ankle	4.2	8.2	Ankle	Knee	40
	Knee	12.8	3.5			

F wave / **H-reflex**

Nerve	F-Lat (ms)	Nerve	H-Lat (ms)
Right median	NR	Left tibial	NR
Left peroneal	62.98	Right tibial	NR
Right peroneal	56.09		
Left tibial	56.00		
Right tibial	50.67		
Right ulnar	NR		

Ant Lat Mall; anterior lateral malleolus, Lat Mall; lateral malleolus, Rec; recording site, B Elbow; below elbow, A Elbow; above elbow, B Fib; below fibular head, Poplt; popliteal fossa, NR; no response, Lat; latency.

Table 2. Follow up nerve conduction study

Sensory nerve action potential

Nerve	Stimulation site	Peak latency (ms)	Amplitude (μV)	Site 1	Site 2	Velocity (m/s)
Left superficial peroneal anti sensory	14 cm	2.8	6.5	14 cm	Ant Lat Mall	50
Right superficial peroneal anti sensory	14 cm	2.5	4.8	14 cm	Ant Lat Mall	56
Left sural anti sensory	Calf	3.2	12.1	Calf	Lat Mall	44
Right sural anti sensory	Calf	2.8	16.1	Calf	Lat Mall	50
Right median ortho sensory	2nd digit	3.1	11.4	2nd digit	Rec	45
	Palm	2.0	22.7	Palm	Rec	35
	Wrist	5.5	11.3	Wrist	Rec	51
	Elbow	2.4	31.9	Elbow	Rec	54
Right ulnar ortho sensory	5th digit	2.9	8.4	5th digit	Rec	41
	Wrist	5.6	10.5	Wrist	Rec	50
	Elbow	2.2	25.4	Elbow	Rec	55

Compound muscle action potential

Nerve	Stimulation site	Onset latency (ms)	Amplitude (μV)	Site 1	Site 2	Velocity (m/s)
Right median	Wrist	NR		Wrist	Elbow	
	Elbow	NR		Elbow	Axilla	
	Axilla	NR				
Right ulnar	Wrist	NR		Wrist	B Elbow	
	B Elbow	NR		B Elbow	A Elbow	
	A Elbow	NR		A Elbow	Axilla	
	Axilla	NR				
Left peroneal	Ankle	NR		Ankle	B Fib	
	B Fib	NR		B Fib	Poplt	
	Poplt	NR				
Right peroneal	Ankle	NR		Ankle	B Fib	
	B Fib	NR		B Fib	Poplt	
	Poplt	NR				
Left tibial	Ankle	4.9	1.8	Ankle	Knee	27
	Knee	17.3	1.1			
Right tibial	Ankle	4.9	1.6	Ankle	Knee	34
	Knee	12.8	1.0			

F wave / **H-reflex**

Nerve	F-Lat (ms)	Nerve	H-Lat (ms)
Right median	NR	Left tibial	NR
Left peroneal	NR	Right tibial	NR
Right peroneal	NR		
Left tibial	69.65		
Right tibial	66.03		
Right ulnar	NR		

Ant Lat Mall; anterior lateral malleolus, Lat Mall; lateral malleolus, Rec; recording site, B Elbow; below elbow, A Elbow; above elbow, B Fib; below fibular head, Poplt; popliteal fossa, NR; no response, Lat; latency.

Conclusion

This case highlights the diagnostic challenge of AMAN-PCB overlap syndrome, where cervicobrachial and bulbar weakness suggests PCB, but serology and electrophysiological progression indicate AMAN. The shift from conduction blocks to axonal loss demonstrates pseudo-demyelination, in which antibody mediated attacks at the nodes of Ranvier mimic demyelination before axonal degeneration. Clinicians should not exclude axonal variants based solely on early demyelinating patterns. The triad of bulbar dysfunction, acute respiratory failure, and upper limb predominant weakness should prompt early recognition to ensure timely respiratory management.

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