"QUADRIPARESIS BEYOND THE USUAL "A COMPARATIVE CASE SERIES OF FIVE DISTINCT ETIOLOGIES

DR SREEDEVE SHAMRITHA,DM NEUROLGY RESIDENT ,THANJAVUR MEDICAL COLLEGE
DR ARUN RAJ.E,PROFESSOR AND HEAD OF DEPARTMENT
DR SEKAR ,ASSISTANT PROFESSOR,THANJAVUR MEDICAL COLLEGE

INTRODUCTION

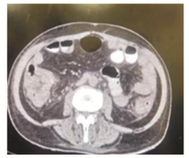
Acute Flaccid Paralysis is a neurological emergency characterized by rapidly evolving weakness with Hypotonia and Areflexia. While Guillain-Barré syndrome (GBS) is the most common etiology, various Neuropathic, Myopathic, and Systemic inflammatory conditions may mimic its presentation. Early differentiation is crucial to guide appropriate therapy and improve outcomes. This case series highlights five distinct causes of Acute Flaccid Paralysis that initially resembled GBS.

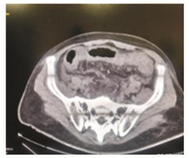
METHODS AND METHODOLOGY

We assessed a series of five patients presenting with Acute Quadriparesis. Parameters evaluated included age, sex, onset pattern, MRC grading at admission, Nerve conduction studies, Ventilatory requirements, CSF analysis, Additional investigations as indicated and neurological recovery. Clinical, Radiological and laboratory evaluations were conducted to confirm the diagnosis in each case.

RESULTS

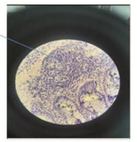
A 56-year-old male developed quadriparesis with bulbar involvement over 2 weeks following a cough. Examination showed MRC grade 2–3 weakness. NCS revealed demyelinating polyradiculoneuropathy and CSF showed albuminocytological dissociation. He also had abdominal distension; stool for Campylobacter jejuni was negative. Ascitic fluid demonstrated lymphocytic predominance with high SAAG, negative for malignancy and tuberculosis. CT abdomen showed diffuse colonic inflammation, and colonoscopy with biopsy confirmed colitis of infective/inflammatory etiology





HPE:
NORMAL COLONIC MUCOSA WITH GOBLET CELLS AND CHRONIC NON SPECIFIC INFLAMMATION IN LAMINA PROPRIA.

NO EVIDENCE OF DYSPLASIA



- Neurological manifestations, though rare are important extraintestinal complications of inflammatory bowel disease (IBD), with peripheral nervous system involvement more frequent in ulcerative colitis. Guillain-Barré Syndrome (GBS) is uncommon but may arise from immune dysregulation, infections, malabsorption, or immunotherapy. Reported cases usually occur during remission, often associated with anti-TNF therapy or steroid tapering. Our case is distinct, presenting as GBS during active colitis, suggesting intestinal inflammation itself may act as an immune trigger.

CASE 2

A 45-year-old female intubated on day 2 of illness had quadriparesis (MRC 1–2), Neck, Bifacial and Bulbar weakness with Bilateral abduction restriction and Autonomic dysfunction. By day 3, she developed coma with absent brainstem reflexes not attributable to hypoxia. CT,MRI brain, Routine Investigations, Echo and EEG were normal. CSF showed Albuminocytological dissociation. NCS initially demonstrated demyelination with conduction block and temporal dispersion; Repeat studies showed absent motor and sensory responses. She was started on IV immunoglobulin but succumbed on day 4 to sudden cardiac arrest. This represents Fulminant GBS, a rare entity with very few cases reported in literature.

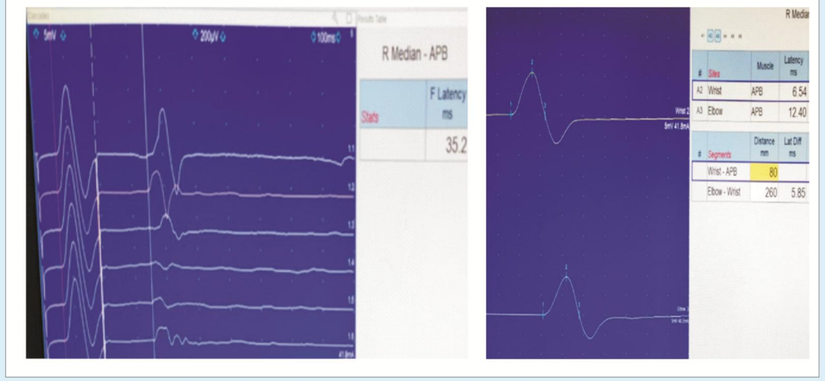
Final Laboratory Report

Name : [redacted] Sex/Age : Female / 41 Years Lab ID : 50330400440
Ref. By : SRF ID : Ref. ID :
Corporate : UHID :
Col Dt. Time : 12-Mar-2025 17:01 Recv Dt. Time : 12-Mar-2025 17:01 Sample Type : Serum
Reg.Dt. Time : 12-Mar-2025 17:01 Report Released @ : 15-Mar-2025 15:43 Report Printed : 15-Mar-2025 15:59

TEST	RESULTS	UNIT	BIOLOGICAL REF RANGE	REMARKS
BIOCHEMICAL INVESTIGATIONS				
Ganglioside Profile (IgM) By Immunoblot				
Monosialoganglioside GM1	Negative			
Monosialoganglioside GM2	Negative			
Monosialoganglioside GM3	Negative			
Disialoganglioside GD 1a	Negative			
Disialoganglioside GD 1b	Negative			
Trisialoganglioside GT1b	Negative			
Tetrasialoganglioside GQ1b	Negative			

CASE 3

A 17-year-old male with antecedent history of diarrhea presented on day 10 of illness with acute flaccid quadriparesis (MRC 3) and Areflexia. NCS showed Demyelination with prolonged distal latencies and reduced conduction velocity. MRI cervical spine was normal; CSF revealed Albuminocytological dissociation. He was started on IVIG but worsened with new Bulbar and Bifacial weakness, declining to MRC 1–2 with respiratory distress. A repeat short course of IVIG was ineffective with progression of symptoms. He was diagnosed as **Acute-onset CIDP**, initiated on Plasmapheresis and subsequently improved. On follow-up, he continues on monthly pulse steroids with recovery to MRC 5.Acute-onset CIDP, seen in ~16% of CIDP, often mimicking Fluctuating GBS. Unlike typical reports of lesser cranial nerve involvement, our patient had prominent bulbar features, but showed good steroid responsiveness on follow-up.



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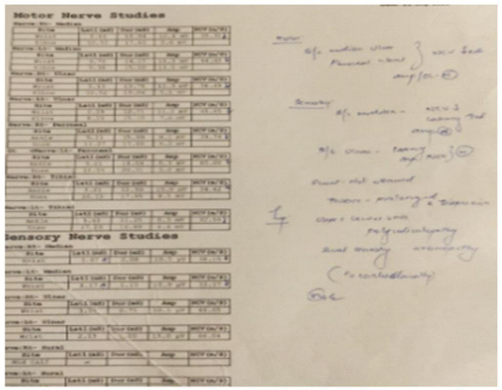
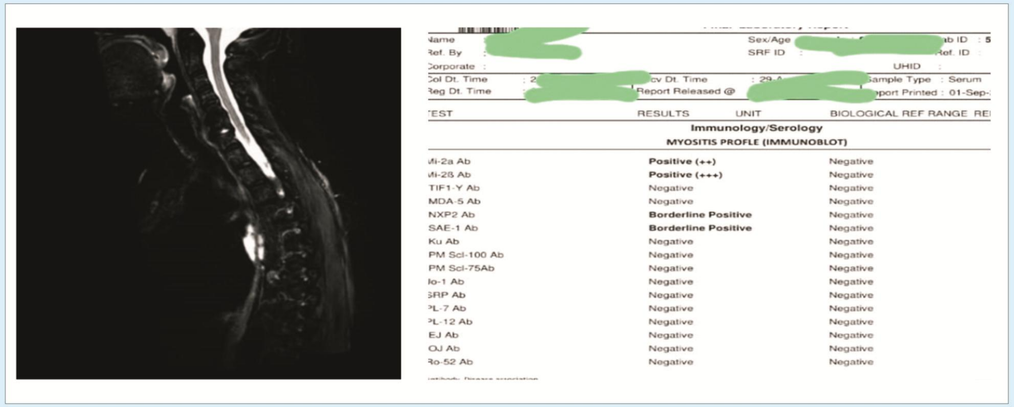
CASE 4

A 40-year-old male with no Co morbidities had recurrent episodes of acute limb weakness over 2 years. The first episode 2 years earlier was an AIDP treated with IVIG with full recovery at 6 months. Eleven months later, he had a similar relapse diagnosed as recurrent GBS, treated with IVIG but with incomplete recovery.In the current admission, he developed acute ascending weakness over 8 days (MRC 3–4) with gait unsteadiness, without sensory, cranial nerve or autonomic involvement. No preceding infection or vaccination was reported. Routine labs, renal function, thyroid profile, ANA, protein electrophoresis, and CT brain,chest,abdomen were normal. CSF showed Albuminocytological dissociation (protein 60 mg/dL, 2 cells). NCS revealed prolonged distal latencies, slowed conduction velocities, and absent F waves, raising the possibility of Nodopathy due to recurrence. He was treated with Plasmapheresis with clinical improvement but was subsequently lost to follow-up.



CASE 5

A 30-year-old male developed Progressive Quadriparesis with neck weakness following antecedent diarrhea. Initially treated as GBS with IVIG, he deteriorated (MRC 1–2) with severe limb pain and Areflexia. NCS showed Demyelinating Polyradiculoneuropathy; MRI spine revealed **Paraspinal edema**. He worsened with respiratory distress requiring ventilation. Thyroid and Paraneoplastic workup were normal, but serum CPK was elevated (3097). A diagnosis of GBS with inflammatory Myopathy was considered and Myositis profile was sent. He was treated with steroids followed by Plasma exchange, with gradual improvement.This rare overlap of GBS with inflammatory Myopathy has scant literature support and association with Dermatomyositis remains unclear



Name		Sex/Age		Lab ID	: 5
Ref. By		SRF ID		Ref. ID	
Corporate				UHID	
Col Dt. Time	: 2	Rcv Dt. Time	: 29-A	Sample Type	: Serum
Reg Dt. Time		Report Released @		Report Printed	: 01-Sep-

TEST	RESULTS	UNIT	BIOLOGICAL REF RANGE
Immunology/Serology			
MYOSITIS PROFLE (IMMUNOBLOT)			
Mi-2a Ab	**Positive (++)**		Negative
Mi-2ß Ab	**Positive (+++)**		Negative
TIF1-Y Ab	Negative		Negative
MDA-5 Ab	Negative		Negative
NXP2 Ab	**Borderline Positive**		Negative
SAE-1 Ab	**Borderline Positive**		Negative
Ku Ab	Negative		Negative
PM Scl-100 Ab	Negative		Negative
PM Scl-75Ab	Negative		Negative
Jo-1 Ab	Negative		Negative
SRP Ab	Negative		Negative
PL-7 Ab	Negative		Negative
PL-12 Ab	Negative		Negative
EJ Ab	Negative		Negative
OJ Ab	Negative		Negative
Ro-52 Ab	Negative		Negative

CONCLUSION

Acute Quadriparesis warrants a broad differential beyond GBS. Comprehensive clinical assessment, Electrophysiology, Imaging and systemic Evaluation are essential for timely diagnosis and appropriate Management

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