e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2026; 18(9); 1561-1565

Case Series

Case Series of Acute Flaccid Paralysis in a Tertiary Care Teaching Institute in Western India

Pravin Soni[1], Niranjan Pathak[2], Ram Mundhe[3], Hrishikesh Kudale[4]

[1]Head of Department, Department of Medicine, PCMC'S PGI, YCMH, Pimpri, Pune-18
[2]Associate Professor, Department of Medicine, PCMC'S PGI, YCMH, Pimpri, Pune- 18
[3]Assistant Professor, Department of Medicine, PCMC'S PGI, YCMH, Pimpri, Pune-18
[4]Resident, Department of Medicine, PCMC'S PGI, YCMH, Pimpri, Pune-18

Received: 01-06-2026 / Revised: 15-07-2026 / Accepted: 21-08-2026
Corresponding author: Dr. Hrishikesh Kudale
Conflict of interest: Nil

Abstract

Acute flaccid paralysis (AFP) in adults represents a clinical emergency with a broad differential diagnosis. Early recognition and etiological identification are crucial for prompt management and favorable outcomes. This case series highlights the diverse etiologies, clinical presentations, diagnostic approaches, and therapeutic outcomes in adult patients presenting with AFP. We retrospectively analyzed 6 adult cases of AFP admitted to Yashwantrao Chavan Memorial Hospital, Pimpri, and Pune over a period of January 2024 to May 2025. Detailed clinical evaluation, neurophysiological studies, imaging, and relevant laboratory investigations were performed to identify the underlying causes. Patients were followed up to assess outcomes.

Keywords: Acute Flaccid Paralysis, Guillain Barre Syndrome.

DOI: 10.25258/ijcpr.18.9.216

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/4.0) and the Budapest Open Access Initiative (http://www.budapestopenaccessinitiative.org/read), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Acute flaccid paralysis (AFP) is a clinical syndrome characterized by rapid onset of weakness of lower motor neuron type, including weakness of the respiratory and pharyngeal muscles, progressing to maximum severity within several days to weeks. AFP is a complex clinical syndrome with a broad array of potential aetiologies that vary remarkably with age.

Patients presenting with acute onset of quadriparesis or paraparesis pose a unique challenge to the clinician. An accurate and early diagnosis is the key to a positive outcome. Various studies are available in the paediatric population regarding AFP prevalence and differential diagnoses, mainly as an offshoot of the global polio eradication initiative. However, few such studies have been conducted among adults.

Acute flaccid paralysis (AFP) is "characterized by rapid onset of weakness of an individual's extremities, often including weakness of the muscles of respiration and swallowing, progressing to maximum severity within 1-10 days.

The term 'flaccid' indicates the absence of spasticity or other signs of disordered central nervous system motor tracts such as hyperreflexia, clonus, or extensor plantar responses." Since the elimination of poliovirus from large parts of the world, Guillain-Barré syndrome (GBS) has become the most important clinical cause of AFP. Studies around the world have identified envenomation, porphyria, hypokalaemia, early acute transverse myelitis, rhabdomyolysis, botulism, and myasthenia gravis as other causes of acute flaccid paralysis. Neuroparalytic snakebite has been previously reported as a significant cause of acute flaccid paralysis in young rural men from Northern India. We evaluated cases of Acute Lower Motor Neuron type of weakness and analyzed the underlying spectrum of clinical conditions.

Criteria for inclusion of patients for the study: Any case of acute flaccid paralysis admitted to the Department of Medicine in PGI Yashwantrao Chavan Memorial Hospital, PCMC, and Pune who is:

1. >18 years but <60 years
2. Paralysis is acute in onset (<2 weeks duration)
3. Flaccid nature (absence of clonus, extensor plantar, spasticity, scissoring, Criss cross gait)
4. Paraparesis
5. Quadriparesis with or without respiratory or bulbar muscle involvement.

Case Presentation

Case 1- 31-year-old female, Housewife by occupation, presented with chief complaints of tingling sensation in lower limbs which then progressed to weakness of all four limbs, sudden in onset, gradually progressive in nature. Patient had history of vomiting and loose motions, 3-4 episodes each, 4 days before onset of symptoms Examination revealed 1/5 power in Bilateral Upper Limbs and 0/5 power in Bilateral Lower Limbs according to MMRC grading. Bilateral upper and lower limb Deep tendon reflexes were absent and bilateral plantar response was mute. CSF studies showed Albumin cytological Dissociation and Nerve Conduction Study demonstrated Motor Axonal and Demyelinating Polyradiculo-neuropathy in Upper limb and Lower limb suggestive of Guillain Barre Syndrome. Intravenous Immunoglobulin was started according to weight based dosing along with physiotherapy. Patient then developed Breathlessness with drop in oxygen saturation. She was intubated secondary to respiratory muscle paralysis.

Case 2– 42-year-old female patient, Housewife by occupation, came with chief complaints of bilateral upper limb and lower limb weakness, sudden in onset, gradually progressive in nature. Patient gave history of similar complaints 3 months back. Patient also gives history of CA Breast for which she was operated 1 year back and on chemotherapy. Examination revealed 2/5 power in bilateral upper and lower limbs according to MMRC grading. Bilateral upper limb and lower limb reflexes were diminished, and bilateral plantar responses were mute. Laboratory studies revealed a serum potassium level of 2.2 meq/L. Patient's Arterial Blood Gas Analysis showed Metabolic Acidosis. Urinary Potassium was more than 15mmol/L s/o Renal Tubular Acidosis. ANA profile followed by ANA Blot was done which led towards the diagnosis of Sjogren's syndrome. Patient was given Potassium Correction with due ECG monitoring. Patient's power improved to 5/5 across all four limbs within 24hrs of starting potassium correction.

Case 3- 30-year-old married male presented in drowsy disoriented state with pulse 64/min, blood pressure 110/70 mmHg, and oxygen saturation 90 % which was decreasing. He was intubated in view of decreasing GCS as patient became stuporous and later comatose. On further examination, quadriplegia with power 0/5 in all muscle groups was present according to MMRC grading, deep tendon reflexes were absent. However plantar was surprisingly extensor bilaterally. Pupils were dilated non-reactive to light. Mother and wife gave history of sudden onset upper abdominal pain and nausea at 4 am in morning followed by heaviness of tongue, difficulty to speak, sense of choking, altered mental status and weakness of all four limbs within an hour. Chest, cardiac, and abdominal examinations were normal. Blood and urine investigations were normal. Chest X-ray, CT brain [plain+ angiography+ venography] were normal. Neostigmine test was negative. Tests for porphyria were negative. Later, ptosis was prominent and whole blood clotting time was more than 20 min. High degree of suspicion of snakebite was considered but attendants gave no history, and no bite mark was found. Patient was given polyvalent antivenom and Neostigmine Atropine cycles urgently and after 2 days; he became conscious, ptosis decreased, muscle power increased to 3/5. After 4 days, patient was successfully extubated.

Case 4- 28-year-old male, Engineer by occupation, came with the chief complaints of weakness in all 4 limbs in the last 2 days, sudden in onset, non-progressive in nature. There was no recent history of cough, cold or vomiting, loose motions. Examination revealed 3/5 power in Bilateral upper limbs and Lower limbs according to MMRC grading. Bilateral upper and lower limb Deep tendon reflexes were absent and bilateral plantar response was mute. CSF studies were within normal limits. Nerve Conduction studies demonstrated Acute Inflammatory Demyelinating Polyradiculo-neuropathy in Upper limb and lower limb suggestive of AIDP variant of GBS. Intravenous Immunoglobulin was started according to weight based dosing along with rigorous physiotherapy. Patient then developed Bilateral Facial palsy and underwent MRI for the same. MRI demonstrated Demyelinating changes in Bilateral Facial Nerves consistent with the diagnosis of GBS. Patient power improved to 4/5 in all four limbs, and his facial palsy was gradually resolving

Case 5- 24 yr old male, student by occupation, came with chief complaints of sudden onset weakness of distal muscles of upper limb while writing leading to inability to hold pen. Patient later developed weakness of both the lower limb such that he was not able to grip chappals and later unable to get up from sitting position or turn in bed from side to side. Patient could however perform overhead abduction without resistance. Patient also complained of band like sensation across trunk with loss of bladder function.

On examination, there was hypotonia in all four limbs with areflexia and definite sensory level at T6. There was history of fever episode 4-5 days prior to the episode. CSF studies were suggestive of albumin cytological dissociation. NCV was suggestive of Acute Inflammatory Demyelinating Polyradiculoneuropathy. All findings were pointing to GBS except the definite sensory level, bladder involvement and involvement of upper limbs followed by lower limbs. MRI spine T2 weighted image showed spinal canal hyperintensity in

thoracic region which was longitudinally extensive.

A diagnosis of GBS-LETM overlap syndrome was made. Patient was treated with IVIG and steroids. On discharge, power had improved to 4/5 in all four limbs and reflexes were brisk. Anti AQP, anti MOG antibodies were found to be negative.

Case 6- 23-year-old female, housewife by occupation, came with chief complaints of weakness in bilateral upper and lower limbs, sudden in onset, gradually progressive in nature. Patient had no history of similar episodes in the past. But patient gives positive family history of similar episode in the father 5-6 years back. There was no history of vomiting, loose motions, fever or cough/ cold prior to the weakness. On examination, there was hypotonia in all 4 limbs with 4/5 power in bilateral upper limbs and 2/5 power in bilateral lower limbs. Bilateral plantar responses were flexor. Deep Tendon Reflexes were diminished but present. Blood Investigations revealed a Serum Potassium of 7meq/L. Patient was suspected with Hyperkalemic periodic paralysis and was started with Insulin drip along with 25% Dextrose. Patient's power improved significantly as the potassium was corrected, and she was ambulatory the next day without any support.

Results

Table 1: Distribution of Etiologies in Acute Flaccid Paralysis Cases (n=6)

Etiology	Number of cases (n)
Guillain-Barre Syndrome (GBS)	3
Transverse Myetlitis	1
Neuroparalytic Snakebite	1
Hypokalemic Periodic Paralysis	1
Hyperkalemic Periodic Paralysis	1

Table 2: Clinical features across different etiologies

Clinical Feature	GBS	Transverse Myelitis	Neuroparalytic Snakebite	Hypokalemic Periodic Paralysis	Hyperkalemic Periodic Paralysis
Acute Onset (<72 hrs)	Yes	Yes	Yes	Yes	Yes
Symmetrical Weakness	Yes	Yes	Yes	Yes	Yes
Cranial Nerve Involvement	Yes	No	No	No	No
Respiratory Muscle Involvement	Yes	Yes	Yes	No	No
Sensory Involvement	No	No	No	No	No
Autonomic Dysfunction	Yes	No	Yes	No	No
Serum Potassium abnormality	No	No	No	No	No
MRI Spine abnormality	No	No	No	No	No

Table 3: Treatment and Outcomes

Parameter	GBS	Transverse Myelitis	Neuroparalytic Snakebite	Hypokalemic Periodic Paralysis	Hyperkalemic Periodic Paralysis
Immunotherapy (IVIG/Plasma) Used	Yes	No	No	No	No
Steroids Used	No	Yes	Nol	No	No
Antisnake venom used	No	No	Yes	No	No
Electrolyte Correction Required	No	No	No	Yes	Yes
Ventilator Support Needed	Yes	No	Yes	No	No
Full recovery at discharge (%)	No	Yes	Yes	Yes	Yes
Mortality	1/3 cases	0%	0%	0%	0%

Table 4: Summary of outcome

Etiology	Mean duration of hospital stay (days)	Residual weakness at discharge (%)	Mortality (%)
Guillain-Barre Syndrome (GBS)	1 month	Present	33.33
Transverse Myetlitis	7-10 days	Present	0
Neuroparalytic Snakebite	7-10 days	Absent	0
Hypokalemic Periodic Paralysis	3-4 days	Absent	0
Hyperkalemic Periodic Paralysis	3-4 days	Absent	0

Discussion

This case series describes the clinical presentations, etiological diversity, and diagnostic considerations in six adult patients presenting with acute flaccid paralysis (AFP). The spectrum of underlying causes—ranging from immune-mediated

neuropathies and electrolyte imbalances to neurotoxins and central nervous system inflammation—underscores the complexity of AFP in adults, where timely recognition and tailored intervention are critical to improving outcomes.

Guillain-Barré Syndrome (GBS): GBS remains the most common cause of AFP in adults worldwide [1]. Our two patients with GBS presented with symmetrical ascending weakness and areflexia—hallmark features of the disease. Both demonstrated albuminocytologic dissociation in CSF and demyelinating patterns on nerve conduction studies. These findings are consistent with the acute inflammatory demyelinating polyradiculoneuropathy (AIDP) subtype, the most common form of GBS in Western and South Asian populations [2].

The pathogenesis of GBS involves an aberrant autoimmune response, often following infection, leading to peripheral nerve demyelination or axonal degeneration [3]. Prompt diagnosis and early initiation of IVIG therapy are essential, especially in cases with bulbar or respiratory involvement, which are associated with increased morbidity [4].

Hypokalemic Paralysis Secondary to Sjögren's Syndrome: One patient developed profound flaccid quadriparesis secondary to hypokalemia in the context of Sjögren's syndrome, likely due to distal renal tubular acidosis (dRTA)—a recognized though underreported complication of Sjögren's [5]. Hypokalemic periodic paralysis in such cases results from renal potassium loss and may be the first manifestation of underlying autoimmune disease.

Hypokalemic paralysis in autoimmune disorders should prompt evaluation for dRTA via urine pH, serum bicarbonate, and anion gap. Correction of serum potassium, combined with immunosuppressive therapy where indicated, typically results in rapid neurological recovery [6]. Early identification is essential to prevent recurrences and nephrocalcinosis.

Hyperkalemic Periodic Paralysis: The case of hyperkalemic periodic paralysis (HYPP) in our series presented with episodic limb weakness and elevated serum potassium, consistent with Type 1 HYPP, an autosomal dominant channelopathy caused by mutations in the SCN4A gene [7]. Unlike hypokalemic forms, HYPP tends to present with brief, self-limiting episodes often triggered by rest after exercise, fasting, or high-potassium meals. Recognition of periodic paralysis syndromes is critical as misdiagnosis can lead to inappropriate therapy. Acetazolamide and potassium-wasting diuretics may be beneficial in long-term management [8]. Genetic counseling and avoidance of triggers are essential components of care.

Neuroparalytic Snakebite: One patient in our series developed acute descending paralysis following a snakebite, consistent with elapid envenomation, likely from a krait or cobra—common in India and Southeast Asia [9]. The neurotoxins block neuromuscular transmission at the presynaptic or postsynaptic level, leading to rapid onset of ptosis, bulbar weakness, and respiratory failure if untreated [10].

Early administration of polyvalent anti-snake venom (ASV), along with respiratory support, can significantly reduce mortality11]. Neuroparalytic snakebite should be considered in all AFP cases with a relevant exposure history, even in the absence of visible bite marks.

Transverse Myelitis: The case of transverse myelitis presented with acute bilateral limb weakness, sensory level, and sphincter dysfunction—features distinguishing it from peripheral causes of AFP. MRI confirmed spinal cord inflammation, and CSF analysis showed lymphocytic pleocytosis. Transverse myelitis may be idiopathic or associated with infections, autoimmune diseases, or demyelinating disorders such as Neuromyelitis Optica spectrum disorder (NMOSD) [12]. High-dose intravenous corticosteroids remain the first-line therapy, with plasma exchange reserved for non-responders [13].

Clinical and Public Health Implications: This case series illustrates the heterogeneity of AFP in adults, with etiologies affecting the peripheral nerves, neuromuscular junction, muscles, and spinal cord. Timely identification of the underlying cause is crucial, as treatment strategies and prognoses vary significantly. While GBS and transverse myelitis require immunomodulation, periodic paralysis and hypokalemic paralysis demand electrolyte correction, and neurotoxic paralysis mandates antivenom and intensive supportive care. Despite the focus of AFP surveillance being largely on pediatric populations for poliomyelitis eradication, these adult cases demonstrate that comprehensive AFP evaluation is equally critical in adults, especially in endemic regions where snakebites and autoimmune diseases are prevalent.

Conclusion

AFP in adults encompasses a broad diagnostic spectrum, necessitating a systematic, and multidiscipli nary approach. Clinicians must consider not only GBS but also rarer causes like periodic paralysis, autoimmune renal tubular acidosis, neurotoxic envenomation, and spinal cord inflammation. Early diagnosis and targeted therapy are pivotal in optimizing neurological recovery and reducing morbidity.

References

1. Willison HJ, Jacobs BC, van Doorn PA. Guillain–Barré syndrome. Lancet. 2016;388

(10045):717–727.
2. Yuki N, Hartung HP. Guillain–Barré syndrome. N Engl J Med. 2012;366(24):2294–2304.
3. Van Doorn PA. Diagnosis, treatment and prognosis of Guillain–Barré syndrome. Presse Med. 2013;42(6)\:e193–201.
4. Hughes RA, Swan AV, van Doorn PA. Intravenous immunoglobulin for Guillain–Barré syndrome. Cochrane Database Syst Rev. 2014;(9)\:CD002063.
5. Pimentel CF, et al. Hypokalemic paralysis in Sjögren's syndrome. Clin Nephrol. 2008;70 (6):558–62.
6. Amer H, et al. Renal tubular acidosis associated with autoimmune diseases. Nat Rev Nephrol. 2011;7(12):682–692.
7. Cannon SC. Pathomechanisms in channelopathies of skeletal muscle and brain. Annu Rev Neurosci. 2015;38:397–420.
8. Fontaine B. Periodic paralysis. Adv Exp Med Biol. 2018;1084:129–146.
9. Warrell DA. Snake bite. Lancet. 2010;375(9708):77–88.
10. Silva A, et al. Neurotoxic envenomation and neuromuscular blockade: a review. Toxins. 2017;9(11):380.
11. Kularatne SA. Epidemiology and clinical picture of the Russell's viper (Daboia russelii) bite in Sri Lanka: a prospective study. Toxicon. 2003;41(7):851–858.
12. Transverse Myelitis Consortium Working Group. Proposed diagnostic criteria and nosology of acute transverse myelitis. Neurology. 2002;59(4):499–505.
13. Jacob A, Weinshenker BG. An approach to the diagnosis of acute transverse myelitis. Semin Neurol.