

Case Report

Rehabilitation challenges in a polytrauma patient with discordant neurological findings: coexistence of traumatic brachial plexus injury and bilateral ankle clonus – a case report

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ABSTRACT

Traumatic brachial plexus injury (BPI) typically produces lower motor neuron (LMN) findings, whereas exaggerated reflexes and ankle clonus suggest upper motor neuron (UMN) pathway involvement. Their coexistence following polytrauma presents a diagnostic and rehabilitation challenge. A 23-year-old man sustained a road traffic accident while riding a two-wheeler, resulting in severe left upper-limb weakness and a comminuted distal femur fracture. Following open reduction and internal fixation, he was referred for rehabilitation. Examination revealed marked hypotonia, with muscle power of 1/5 in the left shoulder and elbow and 2/5 in the finger flexors and abductors. Left biceps and triceps reflexes were absent, while sensation was impaired in the C5 and C6 dermatomes. Bilateral Hoffmann signs, brisk knee jerks, and sustained ankle clonus were present. There was no definite sensory level, and bladder and bowel functions were preserved. Nerve conduction studies and electromyography suggested left brachial plexopathy with axonal involvement. Computed tomography of the brain and spine showed no acute abnormality. Spinal MRI was not obtained because of concerns regarding the femoral fixation implants. A multidisciplinary rehabilitation programme included positioning, splinting, passive mobilisation, electrical stimulation, lower-limb exercises, progressive mobilisation, transfer training, and functional retraining. Functional Independence Measure score improved from 45/126 at admission to 90/126 after one month. Upper-limb weakness persisted, while ankle clonus decreased on serial examination. This case highlights the importance of repeated neurological assessment and individualized multidisciplinary rehabilitation when peripheral and possible central motor pathway findings coexist following polytrauma.

Keywords: Brachial plexus injury, Ankle clonus, Polytrauma, Rehabilitation, Spinal cord injury

INTRODUCTION

Traumatic brachial plexus injury (BPI) is a severe peripheral nerve injury commonly associated with high-energy trauma, including road traffic accidents. Clinical findings typically reflect LMN dysfunction and may include flaccid weakness, hypotonia, reduced or absent reflexes, sensory impairment and muscle wasting.^{1,2}

In contrast, brisk deep tendon reflexes and ankle clonus are UMN signs that raise concern for corticospinal tract involvement.³

Coexistence of peripheral and central neurological signs may occur after high-energy trauma and can make neurological localization difficult.

Concomitant traumatic spinal cord and brachial plexus injuries have been described and may be overlooked when one neurological lesion dominates the clinical picture.^{4,5}

Such diagnostic uncertainty is particularly relevant to rehabilitation because the goals and precautions for a flaccid peripheral nerve lesion differ from those for a central motor pathway lesion.

We present a young polytrauma patient with severe traumatic left upper-limb weakness consistent with brachial plexopathy and simultaneous bilateral ankle clonus, highlighting the diagnostic uncertainty, limitations of imaging, and rehabilitation considerations in a patient with discordant neurological findings.

CASE REPORT

A 23-year-old man sustained injuries in a road traffic accident while riding a two-wheeler on 18 February 2026. Immediately after the trauma, he developed severe weakness of the left upper limb and inability to bear weight on the left lower limb. Imaging demonstrated a left comminuted distal femur fracture. Open reduction and internal fixation with plating was performed on 9 March 2026. He was referred to the Department of Physical Medicine and Rehabilitation on postoperative day 35 (13 April 2026) for comprehensive rehabilitation and discharged on 16/5/26 after 33 days rehabilitation at PMR department. There is no relevant pre-existing neurological disorder in the past and in the family members.

At rehabilitation admission, the patient was conscious, oriented and hemodynamically stable. Left upper limb was hypotonic with severe weakness. According to the Medical Research Council (MRC) scale, muscle power was 1/5 in the left shoulder flexors and extensors, elbow flexors and extensors, and 2/5 in left wrist flexors and extensors. Hand grip was absent. Left biceps and triceps reflexes were absent. Sensation was preserved over the left upper limb except for C5, C6 dermatome where it was found impaired. The right upper limb was neurologically normal.

Examination of the lower limbs showed restricted movement of the left lower limb because of pain and recent

fixation. Left knee range of motion was 0-20°. Right lower-limb motor power was preserved. Bilateral knee jerks were exaggerated and sustained ankle clonus was present bilaterally. Hoffmann sign was present and no definite sensory level was identified. Bladder and bowel functions were normal.

Routine haematological and biochemical investigations were within normal limits. Thyroid function, serum calcium, liver and renal function tests, HIV serology and hepatitis screening were unremarkable. Cerebrospinal fluid analysis was also reported as unremarkable. These investigations did not identify a metabolic or infectious explanation for the clonus.

Nerve conduction studies showed markedly reduced compound muscle action potential amplitudes in the left musculocutaneous and axillary nerves with reduced sensory amplitude in the corresponding sensory nerves. Needle EMG shows fibrillation potentials, positive sharp waves, reduced recruitment of motor units and reduced interference pattern. Overall, the electrodiagnostic findings were suggestive of a left brachial plexopathy with upper trunk involvement. Non-contrast computed tomography (NCCT) of the brain showed no acute intracranial abnormality (Figure 1). Computed tomography of the whole spine showed preserved vertebral alignment without acute osseous abnormality (Figure 2). Spinal MRI was not obtained because of concerns related to the metallic femoral fixation implants. Therefore, occult cervical spinal cord pathology could not be definitively excluded. The combination of bilateral ankle clonus and exaggerated knee jerks in the absence of a definite sensory level or bladder/bowel dysfunction raised clinical suspicion of possible central motor pathway involvement.

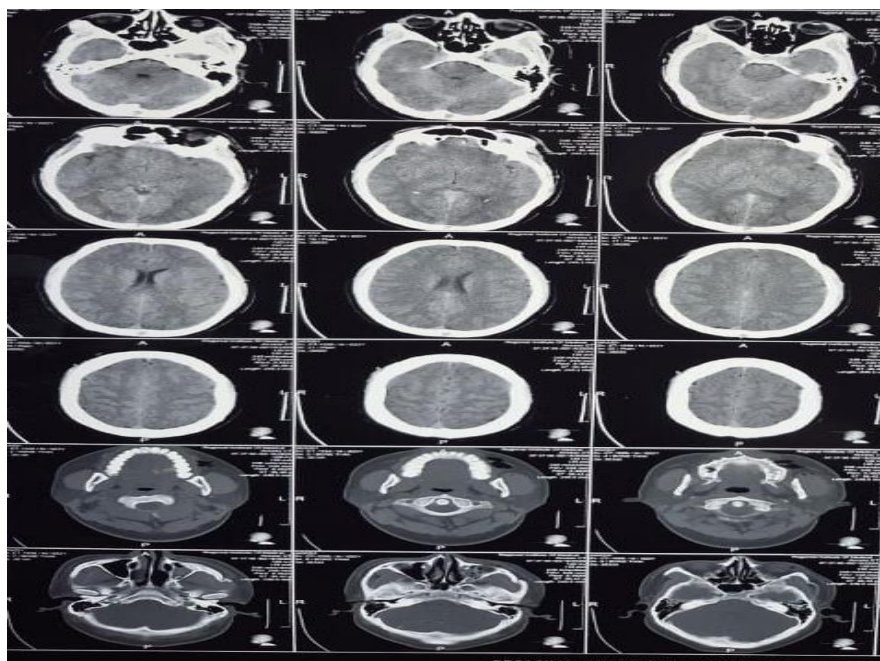


Figure 1: NCCT brain showing no acute intracranial abnormality.

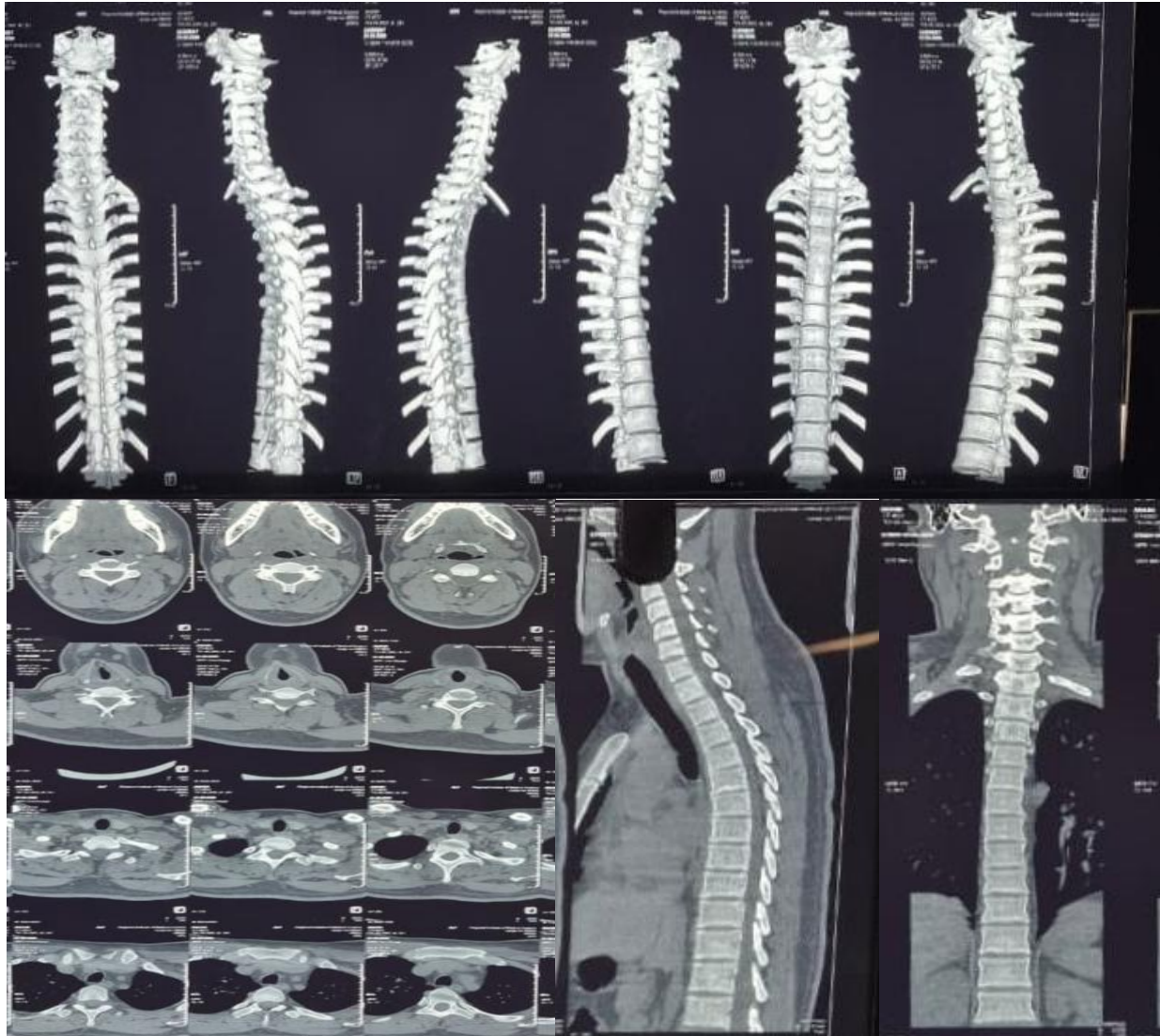


Figure 2: CT of whole spine showing preserved vertebral alignment without acute osseous abnormality.

Functional assessment using the functional independence measure (FIM) showed complete dependence at admission on 13/04/2026 with a reported total score of approximately 45/126. The FIM score improved to 90/126 (moderate dependence) at discharge on 16/05/2026 following rehabilitation at PMR Department RIMS

A multidisciplinary rehabilitation programme was initiated. The left shoulder was positioned in abduction and external rotation, and a shoulder sling was used to support the limb and reduce the risk of glenohumeral subluxation during mobilisation. A resting hand splint was prescribed to reduce the risk of contracture. Passive range-of-motion exercises were performed for the shoulder, elbow, wrist and fingers. Electrical stimulation was applied to the supraspinatus and deltoid muscles for 2 weeks. For the lower limbs, gradual knee mobilisation, assisted range-of-motion exercises, quadriceps strengthening, ankle pumps and gastrosoleus stretching were performed, within the restrictions advised following femoral fixation. Tilt-table training and progressive

mobilisation were introduced gradually. Bed mobility, sitting balance, transfer training and bedside standing activities were emphasized. Psychological counselling and nutritional support were also provided. The rehabilitation program was conducted at PMR department for 33 days.

During rehabilitation, the patient showed gradual improvement in bed mobility, transfers, sitting tolerance and upright positioning. The reported FIM score improved to approximately 100/126 at follow-up after 2 weeks. Upper-limb weakness persisted, whereas bilateral ankle clonus gradually decreased on serial neurological examinations.

DISCUSSION

Traumatic BPI classically presents as a LMN lesion characterized by weakness, hypotonia, and absent reflexes.¹ The presence of bilateral ankle clonus and exaggerated knee jerks in our patient suggested additional

involvement of UMN pathways, producing a discordant neurological presentation.

Combined peripheral nerve and cervical spinal cord injuries may occur following high-velocity trauma due to traction or compressive forces acting on the cervical region.^{5,6} Mild spinal cord contusion or incomplete spinal cord injury may present with hyperreflexia and clonus despite preserved bladder and bowel function.⁶

Preganglionic brachial plexus injuries are anatomically close to the cervical spinal cord and may coexist with root avulsion or cord pathology.⁷ Such overlap may complicate clinical localization and delay diagnosis. In the present case, MRI spine could not be performed because of metallic implants, limiting definitive evaluation of spinal cord involvement. Under such circumstances, serial neurological examination becomes particularly important in monitoring progression and recovery.

Alternative metabolic and infectious causes of clonus were considered less likely because laboratory investigations were unremarkable and symptoms developed immediately after trauma. The gradual reduction of ankle clonus during rehabilitation may indicate transient post-traumatic dysfunction involving descending motor pathways.

Although the most likely explanation for bilateral ankle clonus in the present case is associated cervical cord involvement, previous reports have described abnormal spinal excitability phenomena following peripheral nerve injury. Caviness described the neurophysiological basis of myoclonus and spinal hyperexcitability, while Shimizu et al reported spinal myoclonus following peripheral nerve injury.^{8,9} Although clonus and myoclonus are distinct clinical entities, these reports suggest that altered spinal reflex excitability may occur in selected neurological conditions involving peripheral nerve trauma.

From a rehabilitation perspective, this case posed several important challenges. Simultaneous management of severe flaccid upper limb weakness and possible central motor pathway involvement required individualized rehabilitation planning. Prevention of shoulder subluxation, contractures, joint stiffness, deep vein thrombosis, and deconditioning was essential. Orthopedic restrictions following femoral fixation further complicated mobilization and gait training. Despite diagnostic uncertainty, early multidisciplinary rehabilitation contributed to meaningful functional improvement. Improvement in FIM scores reflected gains in transfers, bed mobility, sitting balance, and upright tolerance, emphasizing the importance of structured rehabilitation even when neurological recovery remained incomplete.

This case highlights the importance of careful neurological examination and serial monitoring in polytrauma patients with mixed neurological findings. Recognition of possible combined central and peripheral nervous system involvement is essential for appropriate rehabilitation planning and prognostic assessment.

CONCLUSION

The coexistence of BPI and bilateral ankle clonus is uncommon and presents significant diagnostic and rehabilitation challenges. This case emphasizes the importance of serial neurological examination, clinical reasoning, and individualized rehabilitation planning in polytrauma patients with discordant neurological findings. Early multidisciplinary rehabilitation may improve functional outcomes even when definitive neuroimaging is limited.

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