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JCIMCR OPEN ACCESS Journal of Clinical Images and Medical Case Reports

ISSN 2766-7820

Short Report

Open Access, Volume 6

Case report: A rare and interesting case of progressive quadriparesis

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Received: May 08, 2025

Accepted: Jun 03, 2025

Published: Jun 10, 2025

Archived: www.jcimcr.org

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DOI: www.doi.org/10.52768/2766-7820/3628

Abstract

Progressive quadriparesis involves the gradual weakening of all four limbs due to diverse etiologist affecting the nervous system, muscles, or connective tissues. Common causes include spinal cord compression, neurodegenerative disorders, autoimmune diseases, or metabolic abnormalities. A detailed clinical evaluation, supported by imaging and laboratory investigations, is crucial for identifying the underlying pathology. Key diagnostic tools include MRI, electromyography (EMG), and Cerebrospinal Fluid (CSF) analysis. Early recognition of treatable conditions like cervical spondylotic myelopathy or vitamin deficiencies can significantly enhance outcomes. This report highlights the diagnostic challenges, therapeutic interventions, and prognosis in a case of progressive quadriparesis, emphasizing the role of nerve biopsy in establishing the diagnosis.

Keywords: Progressive quadriparesis; Neurological disorders; Nerve biopsy; Diagnostic approach; Therapeutic interventions.

Case description

50-year-old female homemaker mandya district, presented with progressive weakness in all four limbs, starting with the left upper limb 9 months prior, followed by the right upper limb 3 months later. Subsequently, weakness developed in the lower limbs, first in the left leg (1 month) and later in the right leg (15 days).

Clinical progression

- **Upper limbs:** Initial weakness in fingers progressed over 3-4 months to involve the arms, resulting in difficulty performing daily activities like lifting objects and washing utensils.
- **Lower limbs:** Gradual weakness led to difficulty gripping slippers and rising from a chair or bed.

Notable exclusions

No history of neck pain, sensory abnormalities, cranial nerve involvement, bowel/bladder dysfunction, trauma, or heavy metal exposure.

Medical history: Hypertension for 5 years (on medication).

Examination

General: Middle-aged female, conscious, cooperative, moderately built, well-oriented. BP: 180/110 mmHg.

CNS: Normal higher mental functions; no cranial nerve abnormalities.

Motor system:

- Wasting of thenar and hypothenar muscles noted.
- ***Power:*** Severe weakness (1/5 in proximal muscles, 0/5 in wrist flexion/extension).
- ***Reflexes:*** Deep tendon reflexes absent; plantar responses mute.
- ***Tone:*** Hypotonia in all limbs.

Sensory system: Normal for all modalities (touch, pain, temperature, vibration).

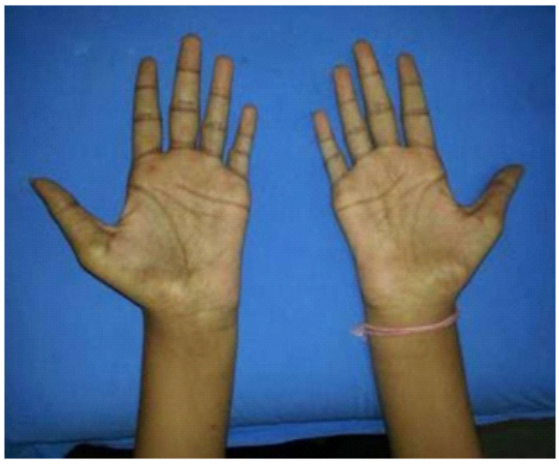


Figure 1: Clinical image.

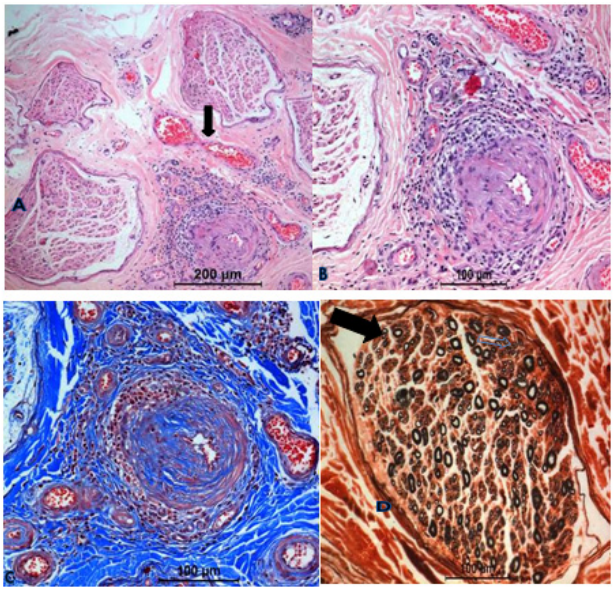
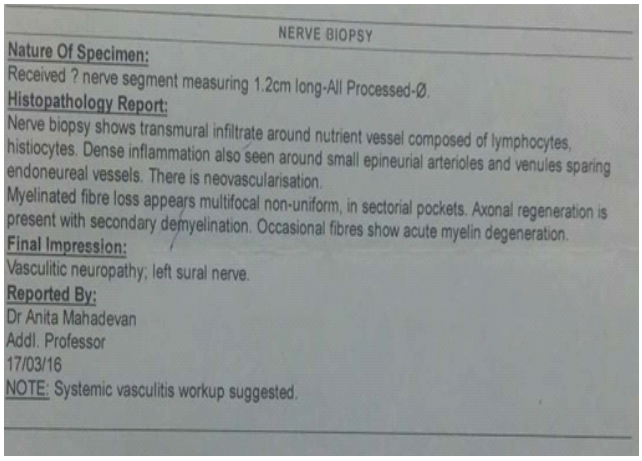


Figure 2: **(A)** Shows transmural infiltrate around a nutrient vessel (arrow) and dense inflammation around small arterioles, venules. **(B)** Higher magnification highlighting lymphohistiocytic infiltrate and neovascularisation around the nutrient vessel. **(C)** Masson trichrome stain highlighting the vascular changes. **(D)** Kulchitsky-Pal stain for myelin shows fiber loss in pockets with regenerating clusters (arrow).



NERVE BIOPSY

Nature Of Specimen:
Received ? nerve segment measuring 1.2cm long-All Processed-Ø.

Histopathology Report:
Nerve biopsy shows transmural infiltrate around nutrient vessel composed of lymphocytes, histiocytes. Dense inflammation also seen around small epineurial arterioles and venules sparing endoneureal vessels. There is neovascularisation.
Myelinated fibre loss appears multifocal non-uniform, in sectorial pockets. Axonal regeneration is present with secondary demyelination. Occasional fibres show acute myelin degeneration.

Final Impression:
Vasculitic neuropathy; left sural nerve.

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17/03/16

NOTE: Systemic vasculitis workup suggested.

Figure 3: Biopsy.

Investigations

1. Blood tests:

ESR: 140 mm/hr.

Hemoglobin: 10.6 g/dL (microcytic hypochromic anemia).

Other parameters: Normal.

2. Nerve conduction studies:

Upper limbs: Pure motor neuropathy.

Lower limbs: Axonopathy in common peroneal, sural, and superficial peroneal nerves.

3. CSF analysis: Elevated protein (214.2 mg/dL); otherwise, unremarkable.

4. Nerve biopsy (left sural nerve): Vasculitic neuropathy characterized by transmural inflammation, myelinated fiber loss, and secondary demyelination.

Diagnosis

Isolated Vasculitic Peripheral Neuropathy (IVPN).

Treatment

1. Acute management:

- Intravenous methylprednisolone (1 g/day).
- Antihypertensives: Amlodipine, Metoprolol, and Prazosin.

2. Long-term management:

- Immunosuppressives: Hydroxychloroquine (100 mg BD) and Methotrexate (7.5 mg weekly).
- Neuropathic pain: Amitriptyline (25 mg) and Pregabalin (75 mg).

3. Supportive care: Physiotherapy for motor recovery.

Discussion

Peripheral neuropathy due to isolated vasculitis was first described in detail by Dyck et al. (1987), who documented its pathological alterations and natural history [1]. This condition, also known as nonsystemic vasculitic neuropathy (NSVN), can mimic other neuropathies, making early diagnosis challenging [2,3]. The diagnosis heavily relies on nerve biopsy, which provides definitive pathological evidence, such as vascular inflammation, axonal loss, and secondary demyelination [4]. Patients often present with indolent, progressive motor deficits, and elevated ESR, as seen in this case. Management revolves around immunosuppressive therapy. Corticosteroids remain the first-line treatment, with methotrexate and hydroxychloroquine as effective adjuncts to prevent relapses [5,6]. Prognosis depends largely on the timeliness of diagnosis and intervention, as untreated cases risk permanent neurological deficits [7].

Conclusion

Isolated vasculitic peripheral neuropathy is often underdiagnosed due to its nonspecific presentation and the invasive

nature of nerve biopsy. Awareness and timely intervention are crucial, as early immunosuppressive therapy can lead to significant clinical improvement.

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