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AXONAL MONONEURITIS MULTIPLEX WITH INFLAMMATORY MYOPATHY AS VASCULITIC MANIFESTATION OF RHEUMATOID ARTHRITIS: A CLINICAL CASE ANALYSIS

KEY WORDS: Rheumatoid Vasculitis; Mononeuritis Multiplex; Inflammatory Myopathy; Axonal Neuropathy; IVIG

Dr Mogaparthi Harshini* Resident, Department of General Medicine, Dr. DY. Patil University, School of Medicine, Nerul, Maharashtra, India*Corresponding Author

Dr Kandimalla Sri Kalyani Resident, Department of General Medicine, Dr. DY. Patil University, School of Medicine, Nerul, Maharashtra, India

Dr Archana Bhate Department of General Medicine, Dr. DY. Patil University, School of Medicine, Nerul, Maharashtra, India

ABSTRACT

Background: Rheumatoid arthritis (RA) is a chronic systemic autoimmune disorder primarily characterized by symmetrical inflammatory polyarthritis. Beyond joint involvement, RA can manifest with extra-articular complications that significantly increase morbidity and functional disability. Rheumatoid vasculitis is an uncommon but severe manifestation, usually seen in seropositive or active disease, affecting small and medium vessels and leading to ischemic injury of peripheral nerves and muscles. Mononeuritis multiplex is a distinctive neurological presentation characterized by acute, painful, asymmetric sensory-motor deficits involving multiple non-contiguous nerves. When associated with inflammatory myopathy, the clinical presentation becomes complex and may mimic demyelinating neuropathies or metabolic myopathies, often delaying timely diagnosis. Early recognition based on clinical examination and prompt immunomodulatory therapy are essential to prevent irreversible neurological sequelae. This case highlights the importance of bedside clinical assessment in identifying vasculitic neuropathy in RA.

Aim:
To emphasize a clinically driven diagnosis of rheumatoid vasculitis presenting as mononeuritis multiplex with associated inflammatory myopathy.

Objectives:
To describe the pattern and progression of neurological deficits in rheumatoid vasculitis.

To correlate clinical findings with laboratory and electrophysiological evidence.

To evaluate short-term therapeutic response to immunosuppressive therapy and IVIG.

Material and Methods:
This is a single-patient descriptive case study conducted at a tertiary care hospital involving a 58-year-old female with known rheumatoid arthritis presenting with acute progressive limb weakness and neuropathic pain. A detailed clinical neurological examination was performed assessing muscle power, deep tendon reflexes, sensory modalities, cranial nerve involvement, gait, and distribution of deficits. Laboratory investigations included complete blood counts, serum electrolytes, renal function tests, rheumatoid factor, anti-CCP antibodies, and serum creatine phosphokinase (CPK). Electrophysiological evaluation with nerve conduction studies (NCV) and electromyography (EMG) was undertaken to characterize the neuropathy. A left sural nerve biopsy was performed for histopathological confirmation. Multidisciplinary management involved neurology and rheumatology teams. Treatment consisted of pulse intravenous methylprednisolone, transition to oral corticosteroids, disease-modifying antirheumatic drugs (methotrexate and hydroxychloroquine), and intravenous immunoglobulin (IVIG) administered over five days. Clinical status and biochemical parameters were monitored during hospitalization.

RESULTS:
The patient presented with acute onset asymmetric distal weakness, reduced hand grip, difficulty in dorsiflexion of the foot, and patchy sensory loss involving both upper and lower limbs. Neuropathic burning pain and muscle tenderness were prominent symptoms. Reflexes were diminished in affected limbs without cranial nerve or sphincter involvement. The neurological deficit evolved from focal nerve involvement to a generalized yet asymmetric sensorimotor neuropathy over several days, consistent with mononeuritis multiplex.

Laboratory evaluation revealed strongly positive rheumatoid factor with negative anti-CCP antibodies. Hematological parameters showed anemia and leukocytosis with preserved renal function. Serum CPK was markedly elevated, rising from 601 IU/L to 5396 IU/L, suggestive of concurrent inflammatory myopathy. NCV indicated mononeuritis multiplex, EMG showed widespread active and chronic motor axonal denervation. Sural nerve biopsy confirmed vasculitic neuropathy with vascular inflammation and axonal degeneration.

Following administration of high-dose corticosteroids and IVIG, the patient demonstrated significant reduction in neuropathic pain, stabilization of muscle strength, and gradual biochemical improvement, supporting an immune-mediated inflammatory etiology rather than demyelinating neuropathy.

DISCUSSION AND CONCLUSION:
This case illustrates rheumatoid vasculitis as a severe extra-articular manifestation presenting with mononeuritis multiplex and inflammatory myopathy. The diagnosis was primarily guided by clinical features—acute asymmetric weakness, neuropathic pain, multi-nerve involvement, and muscle tenderness-supported by elevated muscle enzymes and electrophysiological evidence of axonal neuropathy. Strong rheumatoid factor positivity further reinforced the autoimmune basis. Differential considerations such as Guillain–Barré syndrome and compressive radiculopathies were less likely due to the asymmetric progression, preserved cranial nerve function, and axonal rather than demyelinating pattern on studies. Early aggressive immunosuppression combined with IVIG played a pivotal role in halting neurological deterioration and improving functional outcomes.

Rheumatoid vasculitis should be suspected in RA patients presenting with acute painful asymmetric neuropathy accompanied by elevated CPK levels. A clinically focused

approach with timely immunomodulatory therapy can prevent permanent disability and improve prognosis. Awareness of this rare yet treatable complication is essential for physicians managing systemic autoimmune diseases.

REFERENCES

1) Scott DL, Wolfe F, Huizinga TW. Rheumatoid arthritis. Lancet. 2010;376(9746):1094-1108.
2) Said G, Lacroix C. Primary and secondary vasculitic neuropathy. J Neurol. 2005;252(6):633-641.
3) Voskuyl AE. The heart and cardiovascular manifestations in rheumatoid arthritis. Rheumatology. 2006;45(suppl 4):iv4-iv7.
4) Collins MP, Dyck PJ. Vasculitic neuropathies. Continuum (Minneap Minn). 2014;20(5):1334-1349.
5) Guillevin L, Pagnoux C. Treatment of systemic vasculitides. Nat Rev Rheumatol. 2009;5(5):237-243.