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INTERNATIONAL JOURNAL OF MULTIDISCIPLINARY EDUCATIONAL RESEARCH
ISSN:2277-7881(Print); IMPACT FACTOR :10.16(2026); IC VALUE:5.16; ISI VALUE:2.286
Peer-Peer Reviewed, Refereed & Open Access International Journal - As per UGC Norms
(Fulfilled Suggests Parameters of UGC by IJMER)
Volume:15, Issue:7(1), July 2026
Scopus Review ID: A2B96D3ACF3FEA2A
Article Received: Reviewed: Accepted
Publisher: Sucharitha Publication, India
Online Copy of Article Publication Available: www.ijmer.in

P-ANCA ASSOCIATED VASCULITIS PRESENTING WITH PULMONARY INFILTRATES AND MONONEURITIS MULTIPLEX: A CASE REPORT

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Abstract

Introduction

Antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis comprises a group of autoimmune disorders characterized by necrotizing inflammation of small blood vessels with multisystem involvement. Perinuclear ANCA (P-ANCA), directed predominantly against myeloperoxidase (MPO), is commonly associated with microscopic polyangiitis and eosinophilic granulomatosis with polyangiitis. Pulmonary and peripheral nervous system involvement may be the presenting manifestations, making early diagnosis challenging.

Case Report

A 46-year-old female working as a palliative caretaker presented with persistent dry cough for one year, followed by inflammatory polyarthralgia involving the wrists and ankles for two months. She subsequently developed progressive weakness and numbness of both lower limbs, predominantly on the right side, along with paresthesia involving the medial aspect of both forearms and hands. Neurological examination revealed bilateral foot drop, more severe on the right side, sensory loss over the superficial and deep peroneal nerve distribution, bilateral ulnar nerve dysesthesia with clawing of the hands, absent ankle jerks, and a high-stepping gait suggestive of mononeuritis multiplex. Laboratory investigations demonstrated normocytic normochromic anemia, elevated erythrocyte sedimentation rate (100 mm/hour), positive rheumatoid factor, elevated anti-cyclic citrullinated peptide antibody, strongly positive P-ANCA (95 U), and negative C-ANCA and antinuclear antibody. Chest radiography and contrast-enhanced computed tomography of the thorax demonstrated pulmonary infiltrative nodular lesions with tree-in-bud opacities. Tuberculosis was excluded by negative sputum acid-fast bacilli smear and cartridge-based nucleic acid amplification test. Nerve conduction studies demonstrated axonal neuropathy involving the common peroneal nerves. The patient was treated with corticosteroids and rituximab along with supportive therapy and showed clinical stabilization.

Conclusion

This case highlights the importance of considering ANCA-associated vasculitis in patients presenting with pulmonary infiltrates and mononeuritis multiplex. Early recognition, prompt immunological evaluation, and initiation of immunosuppressive therapy are essential to prevent irreversible neurological deficits and systemic complications.

Keywords: P-ANCA vasculitis, ANCA-associated vasculitis, mononeuritis multiplex, pulmonary infiltrates, microscopic polyangiitis, eosinophilic granulomatosis with polyangiitis, rituximab.

INTRODUCTION

Antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis represents a heterogeneous group of autoimmune disorders characterized by pauci-immune necrotizing inflammation affecting predominantly small blood vessels. The major clinicopathological entities include microscopic polyangiitis (MPA), granulomatosis with polyangiitis (GPA), and eosinophilic granulomatosis with polyangiitis (EGPA). Perinuclear ANCA (P-ANCA), directed predominantly against myeloperoxidase (MPO), is frequently associated with microscopic polyangiitis and eosinophilic granulomatosis with polyangiitis.

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Pulmonary manifestations, including pulmonary infiltrates, nodules and diffuse alveolar hemorrhage, together with peripheral neuropathy presenting as mononeuritis multiplex, are well-recognized manifestations of ANCA-associated vasculitis. Since these manifestations may mimic infectious or connective tissue disorders, diagnosis is often delayed. Early recognition and timely immunosuppressive therapy are essential to prevent irreversible organ damage.

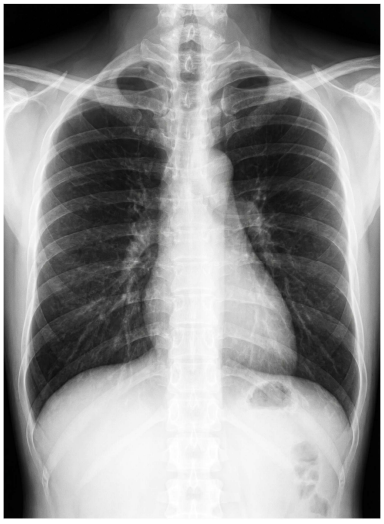
We report a case of P-ANCA-associated vasculitis presenting with chronic pulmonary symptoms and mononeuritis multiplex, emphasizing the importance of early diagnosis and treatment.

CASE REPORT

A 46-year-old female working as a palliative caretaker presented with persistent dry cough for one year. Two months before presentation, she developed progressively worsening pain involving both wrist and ankle joints. She subsequently noticed progressive weakness of both lower limbs, more pronounced on the right side, followed by numbness and weakness involving the medial aspect of both forearms and hands over the preceding one month.

On neurological examination, she had bilateral foot drop, more severe on the right side, with weakness of ankle dorsiflexion. Sensory examination demonstrated impaired touch, pain, and temperature sensation over the superficial and deep peroneal nerve distributions bilaterally. Upper limb examination revealed dysesthesia in the bilateral ulnar nerve distribution with clawing of both hands. Deep tendon reflexes showed absent ankle jerks bilaterally, and gait examination demonstrated a high-stepping gait suggestive of mononeuritis multiplex.

Laboratory investigations revealed normocytic normochromic anemia (hemoglobin 9.4 g/dL) with elevated erythrocyte sedimentation rate (100 mm/hour). Rheumatoid factor and anti-cyclic citrullinated peptide antibody were positive. Antinuclear antibody was negative. P-ANCA was strongly positive (95 U), whereas C-ANCA was negative. Peripheral blood smear demonstrated normocytic normochromic anemia with neutrophilic leukocytosis and thrombocytosis. Serum calcium was 10.3 mg/dL, serum phosphate 5.07 mg/dL, serum ferritin 372 ng/mL, creatine phosphokinase 119 U/L, and gamma-glutamyl transferase 141 U/L.



Chest radiograph and contrast-enhanced computed tomography of the thorax demonstrated small infiltrative nodular parenchymal changes in the right lung and tree-in-bud nodular opacities in the left lower lobe (Figure 1).

Nerve conduction studies revealed reduced compound muscle action potential amplitudes involving the common peroneal nerves, consistent with axonal neuropathy.

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The differential diagnosis included microscopic polyangiitis, eosinophilic granulomatosis with polyangiitis, granulomatosis with polyangiitis, and infective pulmonary disease. During readmission for worsening respiratory symptoms, sputum acid-fast bacilli smear and cartridge-based nucleic acid amplification testing were negative for tuberculosis. Repeat chest radiography demonstrated left lower lobe consolidation.

Based on the clinical presentation, electrophysiological findings, pulmonary imaging, and positive P-ANCA serology, a diagnosis of ANCA-associated vasculitis presenting with pulmonary involvement and mononeuritis multiplex was established.

The patient received intravenous rituximab (1 g administered as two doses) along with systemic corticosteroids. She was discharged on oral prednisolone (Wysolone 20 mg once daily), metformin 500 mg twice daily, trimethoprim-sulfamethoxazole prophylaxis, calcium supplementation, gabapentin 300 mg at bedtime, and duloxetine 20 mg once daily. She was advised regular follow-up for monitoring of neurological recovery and disease activity.

Figure 1. Chest radiograph demonstrating pulmonary infiltrative changes during evaluation for chronic respiratory symptoms.

DISCUSSION

ANCA-associated vasculitis is an uncommon autoimmune disorder characterized by necrotizing inflammation of small blood vessels involving multiple organ systems. Neurological involvement occurs in approximately one-third of patients and commonly manifests as mononeuritis multiplex due to ischemic injury of the vasa nervorum.

Pulmonary involvement is frequently observed in microscopic polyangiitis and eosinophilic granulomatosis with polyangiitis and may present with pulmonary infiltrates, nodules, consolidations, or diffuse alveolar hemorrhage. Since these radiological findings closely resemble pulmonary tuberculosis and other infections, a high index of suspicion is required.

Our patient presented with chronic pulmonary symptoms followed by rapidly progressive mononeuritis multiplex, elevated inflammatory markers, and strongly positive P-ANCA, supporting the diagnosis of ANCA-associated vasculitis. Although eosinophilic granulomatosis with polyangiitis remained an important differential diagnosis because of pulmonary involvement, the absence of documented asthma, peripheral eosinophilia, and histopathological confirmation precluded a definitive diagnosis. Microscopic polyangiitis therefore remained an equally plausible diagnostic consideration.

Prompt initiation of immunosuppressive therapy with corticosteroids and rituximab resulted in stabilization of the patient's neurological and pulmonary manifestations. Early diagnosis and treatment remain essential to reduce morbidity and prevent irreversible neurological disability.

CONCLUSION

This case emphasizes the importance of considering ANCA-associated vasculitis in patients presenting with chronic pulmonary infiltrates associated with mononeuritis multiplex. A multidisciplinary approach involving detailed clinical evaluation, immunological testing, electrophysiological studies, and radiological assessment is essential for timely diagnosis. Early initiation of corticosteroids and rituximab may prevent irreversible neurological deficits and improve long-term outcomes. Continued follow-up is required to monitor disease activity, treatment response, and relapse.

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