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Case Study

A Case Report on Polymyositis

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ABSTRACT

Polymyositis is a chronic autoimmune inflammatory myopathy marked by symmetrical proximal muscle weakness due to immune-mediated muscle fiber damage.The disease predominantly affects adults, exhibiting a higher incidence in females, and is characterized by immune activation involving cytotoxic T lymphocytes and inflammatory mediators.Polymyositis symptoms include trouble getting up from a chair, climbing stairs, or lifting things. These symptoms are often accompanied by fatigue, muscle pain, trouble swallowing (dysphagia), and possible breathing problems. Patients exhibit progressive muscle weakness, dysphagia, and systemic manifestations which can severely affect quality of life.Diagnosis entails clinical assessment, laboratory analyses including elevated creatine kinase levels, autoantibody testing, imaging modalities, electromyography, and muscle biopsy for validation. The main goal of management is to make muscles stronger and improve functional outcomes through drug treatments. Corticosteroids are the first-line treatment, and immunosuppressive drugs like methotrexate and azathioprine are also used. .To improve the prognosis and the quality of life for patients, it is important to diagnose them early and use a multidisciplinary approach to treatment.

INTRODUCTION

Polymyositis is a chronic autoimmune inflammatory myopathy characterized by immune-mediated destruction of skeletal muscle fibers, resulting in symmetrical proximal muscle weakness.It is an uncommon disorder with an estimated annual incidence of approximately 1–8 cases per million population and a prevalence ranging from 5 to 22 cases per 100,000 individuals. The disease predominantly affects adults between 30 and 60 years of age and occurs

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nearly twice as frequently in females compared to males. Although uncommon, polymyositis contributes significantly to morbidity because of progressive muscle weakness, disability, and involvement of extramuscular organs such as the lungs, heart, and gastrointestinal tract.[2]The precise etiology remains unknown; however, genetic susceptibility combined with environmental triggers appears to initiate autoimmune activation. Viral infections, certain medications, connective tissue diseases, malignancies, and immune dysregulation have been implicated as potential precipitating factors. Patients frequently demonstrate myositis-specific autoantibodies, including anti-Jo-1, PL-7, PL-12, EJ, OJ, and Ro-52, which help define clinical subsets and predict disease manifestations.[3]**.** Persistent inflammation causes progressive muscle weakness, muscle atrophy, fibrosis, and impaired physical function. Elevated serum muscle enzymes such as creatine phosphokinase (CPK), aldolase, AST, ALT, and LDH reflect ongoing muscle injury.[4]Patients usually present with gradually progressive symmetrical proximal muscle weakness affecting the shoulder and pelvic girdle muscles. Difficulty climbing stairs, rising from chairs, lifting objects, combing hair, and walking are common complaints. Additional manifestations include fatigue, myalgia, dysphagia, weight loss, interstitial lung disease, arthritis, Raynaud phenomenon, and constitutional symptoms.[5]Diagnosis is based on the integration of clinical findings with laboratory and imaging investigations. Markedly elevated serum creatine phosphokinase remains one of the most sensitive laboratory markers. MRI identifies active muscle inflammation and guides muscle biopsy when required. Myositis-specific antibodies improve diagnostic accuracy and aid disease classification. Electromyography demonstrates myopathic changes, while muscle biopsy confirms inflammatory infiltration and muscle fiber necrosis when diagnostic uncertainty exists.[6]

High-dose corticosteroids remain the cornerstone of initial treatment. Intravenous methylprednisolone pulse therapy is frequently administered in severe disease, followed by oral corticosteroid tapering. Steroid-sparing immunosuppressive agents including methotrexate, azathioprine, mycophenolate mofetil, tacrolimus, rituximab, and intravenous immunoglobulin are employed depending on disease severity and response. Physical rehabilitation, nutritional support, infection management, and regular monitoring of muscle enzymes are integral components of comprehensive care.**[7]**

CASE PRESENTATION

Patient Information

A 45-year-old female was admitted to the Department of General Medicine on with progressively worsening proximal muscle weakness. She complained of difficulty in walking with unsteadiness, climbing up and down stairs, and rising from a sitting position for one month. During the week before admission, her symptoms worsened significantly, interfering with daily activities. She also complained of pain during urination, suggesting a concurrent urinary tract infection. There was no history of fever, trauma, recent strenuous exercise, or exposure to myotoxic drugs. Her past medical history, medication history, family history, and social history were unremarkable.

Clinical Examination

On admission, the patient was conscious, alert, and oriented. Vital signs were stable throughout hospitalization.

- Temperature: **98.4°F**

- Blood pressure: **110/70 mmHg**
- Pulse rate: **84 beats/minute**
- Respiratory rate: **16 breaths/minute**

Systemic examination revealed:

- Central nervous system: No focal neurological deficits
- Cardiovascular system: Normal heart sounds (S1 and S2)
- Respiratory system: Bilateral equal air entry with clear lung fields
- Gastrointestinal system: Soft, non-tender abdomen

Laboratory Investigations

Baseline hematological investigations revealed mild anemia with hemoglobin of 10.6g/dL, which slightly decreased to 10.1 g/dL before discharge. The total leukocyte count remained within the normal range. ESR was markedly elevated (95 mm/hour) initially and decreased to 58 mm/hour following treatment, indicating improvement in inflammatory activity. Platelet count remained within normal limits.

Liver function tests demonstrated significant elevation of hepatic transaminases with AST measuring 207 U/L and ALT 326 U/L, while bilirubin and serum albumin remained within normal limits. These findings suggested acute liver injury associated with active muscle inflammation rather than primary hepatic disease.

Urinalysis showed numerous pus cells and microscopic hematuria at admission, confirming urinary tract infection. Repeat urine examination showed marked improvement following antibiotic therapy.

Renal function remained preserved throughout hospitalization with normal serum creatinine and electrolyte concentrations.

Lipid profile revealed elevated triglycerides (228 mg/dL) and VLDL with reduced HDL cholesterol. Glycemic evaluation demonstrated HbA1c of 6.2%, indicating impaired glucose metabolism.

Inflammatory and muscle injury markers were markedly abnormal. C-reactive protein measured 24.4 mg/dL, lactate dehydrogenase was 561 U/L, and serum creatine phosphokinase (CPK) reached 22,566 IU/L, representing extensive skeletal muscle injury. Follow-up CPK decreased dramatically to 630 IU/L, reflecting an excellent response to immunosuppressive therapy. Serum IgE was also elevated.

Imaging Studies

Magnetic resonance imaging (MRI) of both thighs demonstrated diffuse patchy inflammatory signal abnormalities involving the gluteal and thigh musculature bilaterally, consistent with acute inflammatory myositis. Minimal fatty replacement involving the gluteus maximus, gluteus medius, gluteus minimus, and quadratus femoris muscles suggested early chronic muscle involvement. Echocardiography demonstrated preserved left ventricular systolic function without regional wall motion abnormalities.

Immunological Evaluation

The myositis-specific antibody panel demonstrated positivity for multiple antisynthetase antibodies including **PL-7**, **PL-12**, **EJ**, **OJ**, and **Ro-52**, supporting the diagnosis of autoimmune inflammatory myopathy. These serological findings, together with MRI findings, markedly elevated muscle enzymes, and characteristic clinical manifestations, confirmed the diagnosis of polymyositis.

Therapeutic Management

Considering the severity of muscle inflammation, intravenous pulse methylprednisolone therapy was initiated. The patient received 750 mg intravenously on the first day, followed by 1 g intravenously daily for three consecutive days.

Supportive treatment included intravenous fluids, cefoperazone-sulbactam for urinary tract infection, esomeprazole for gastric protection, acetylcysteine for liver support, ursodeoxycholic acid, rifaximin, vitamin E with levocarnitine, bisoprolol, olmesartan, urinary alkalizer, and symptomatic medications according to clinical requirements.

After clinical stabilization, the patient was discharged on oral prednisolone, which was gradually tapered, together with mycophenolate mofetil as maintenance immunosuppressive therapy. Calcium and vitamin D supplementation were prescribed to reduce the risk of corticosteroid-induced osteoporosis. Additional medications for associated conditions were continued, and follow-up was scheduled in the General Medicine outpatient department.

Clinical Outcome

Following pulse corticosteroid therapy, the patient demonstrated progressive clinical improvement with reduction in proximal muscle weakness and better functional mobility. Laboratory evaluation showed a dramatic fall in serum CPK concentration from 22,566 IU/L to 630 IU/L, accompanied by reductions in ESR and CRP, indicating effective suppression of muscle inflammation. Urinary symptoms resolved following antimicrobial therapy, and liver enzyme abnormalities showed improvement. The patient was discharged in stable condition with instructions for continued immunosuppressive therapy, regular outpatient follow-up, monitoring of muscle enzymes, liver function tests, and physiotherapy to improve muscle strength and prevent disability

DISCUSSION

Polymyositis (PM) is a rare autoimmune inflammatory myopathy characterized by chronic inflammation of skeletal muscles resulting in progressive, symmetrical proximal muscle weakness. It belongs to the spectrum of idiopathic inflammatory myopathies (IIMs), together with dermatomyositis, immune-mediated necrotizing myopathy, antisynthetase syndrome, and inclusion body myositis. Although polymyositis accounts for only a small proportion of inflammatory myopathies, delayed diagnosis remains common because of its insidious onset and nonspecific clinical manifestations. Early recognition and prompt immunosuppressive therapy are crucial for preventing irreversible muscle damage, improving muscle strength, and reducing long-term disability [1,2].

The present case involved a 45-year-old female presenting with progressive proximal muscle weakness, manifested by difficulty in walking, climbing stairs, and rising from a sitting position. These symptoms are considered the classical manifestations of polymyositis and are consistent with the 2017 EULAR/ACR Classification Criteria, which identify symmetrical proximal muscle weakness as one of the strongest predictors of idiopathic inflammatory myopathies [1]. Similarly, Dalakas reported that weakness involving the shoulder and pelvic girdle muscles is the most characteristic clinical feature of polymyositis and usually progresses over weeks to months [2,3]. The absence of sensory deficits and preserved tendon reflexes in the present patient further supported a primary inflammatory myopathy rather than a neuropathic disorder.

One of the most remarkable laboratory findings in this patient was the marked elevation of serum creatine phosphokinase (CPK) to 22,566 IU/L, together with elevated lactate dehydrogenase (LDH), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP). Elevated muscle enzymes remain among the most sensitive indicators of active muscle inflammation and muscle fiber necrosis. Serum CPK may increase

up to 50-fold above normal during active disease and usually declines rapidly following effective immunosuppressive therapy [2,4]. In the present case, serum CPK decreased dramatically from 22,566 IU/L to 630 IU/L after corticosteroid therapy, indicating excellent biochemical response and effective suppression of muscle inflammation. Similar reductions in muscle enzyme levels have been reported as reliable markers of treatment response in inflammatory myopathies [6,10].

Magnetic resonance imaging has become an indispensable non-invasive investigation for inflammatory myopathies because it detects muscle edema, inflammation, and fatty replacement before irreversible muscle atrophy develops. MRI also assists in selecting the most appropriate site for muscle biopsy and monitoring treatment response [12]. Another important diagnostic feature of this case was the positivity for multiple myositis-specific antibodies, including PL-7, PL-12, EJ, OJ, and Ro-52. Autoantibody profiling has significantly improved the diagnosis and classification of idiopathic inflammatory myopathies by identifying clinically distinct subgroups with characteristic prognostic features [9,10]. Anti-PL-7 and anti-PL-12 antibodies belong to the antisynthetase antibody family and are frequently associated with severe muscle disease, arthritis, and interstitial lung disease. Ro-52 positivity has also been associated with increased disease activity and poorer prognosis [9,22]. Although the present patient did not demonstrate pulmonary involvement during hospitalization, long-term monitoring for interstitial lung disease remains essential because pulmonary manifestations may develop later during the disease course

CONCLUSION

Polymyositis is a rare autoimmune inflammatory myopathy that should be considered in adults presenting with progressive symmetrical proximal muscle weakness and markedly elevated muscle enzymes. Early diagnosis requires a comprehensive evaluation incorporating clinical findings, serum muscle enzyme analysis, magnetic resonance imaging, and myositis-specific autoantibody testing. In the present case, the combination of characteristic clinical features, significantly elevated creatine phosphokinase levels, MRI evidence of diffuse inflammatory myositis, and positive PL-7, PL-12, EJ, OJ, and Ro-52 antibodies established the diagnosis of polymyositis associated with urinary tract infection and acute liver injury.

Prompt initiation of pulse intravenous methylprednisolone followed by oral prednisolone and mycophenolate mofetil resulted in marked clinical improvement together with substantial reduction in serum creatine phosphokinase levels, indicating successful suppression of inflammatory muscle injury. Concurrent management of urinary tract infection and supportive care contributed to the favorable clinical outcome.

This case highlights the importance of multidisciplinary management involving physicians, rheumatologists, neurologists, physiotherapists, and clinical pharmacists to optimize patient care. Long-term follow-up is essential to monitor disease activity, treatment-related adverse effects, relapse, and potential complications such as interstitial lung disease, dysphagia, osteoporosis, and cardiovascular involvement. Early recognition and individualized immunosuppressive therapy remain the cornerstone of improving functional recovery and quality of life in patients with polymyositis.

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