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Case Report

A Case of Pulmonary Embolism in Polymyositis

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Abstract

Polymyositis is an uncommon autoimmune inflammatory myopathy. It is characterized predominantly by proximal muscle weakness with potential involvement of multiple organ systems. Despite ongoing research, its pathogenesis remains incompletely understood. Venous thromboembolism (VTE) is a major health problem, sometimes complicated by pulmonary embolism. Systemic inflammation modulates thrombotic responses by supressing fibrinolysis, upregulating procoagulant, and downregulating anticoagulants. VTE is a multifactorial disease manifesting as either deep vein thrombosis (DVT) or pulmonary embolism (PE). It is the third most common vascular disorder in Western countries, preceding only myocardial infarction and stroke. PE, a serious and potentially fatal condition caused by thrombotic obstruction of the pulmonary arteries, is a rare occurrence in patients with polymyositis. The co-existence of these two conditions represents a clinical entity that warrants further investigation. Autoimmune and immune mediated disorders such as systemic lupus erythematosus, inflammatory bowel disease, polyarteritis nodosa and polymyositis/dermatomyositis have been linked to an increased risk of venous thromboembolism. Chronic inflammatory disorders present with an increased risk of VTE (six to eight times) compared to the general population. The case being presented describes a patient, who was first diagnosed to have pulmonary embolism, followed by a diagnosis of inflammatory myositis due to combination of persistently high creatine kinase levels, radiological findings of inflammatory myositis and abnormal electroencephalogram (EEG). The patient was started on high dose steroid therapy followed by immunosuppressive therapy which led to clinical improvement. It highlights the importance of suspecting venous thromboembolism in cases of inflammatory myositis or other autoimmune conditions.

Keywords: inflammatory myopathy; polymyositis; pulmonary embolism; venous thromboembolism; autoimmune disorders

Introduction

Autoimmune conditions have specific pathophysiologic mechanisms which can lead to an increased risk of arterial or venous thrombosis [1]. Polymyositis (PM) and dermatomyositis (DM) are the primary forms of idiopathic inflammatory myopathies in adults[2]. They are characterized by chronic muscle inflammation and progressive weakness [3]. “The estimated prevalence ranges from 1 to 10 cases per million adults and 1 to 3.2 cases per million children [4]”.Clinical manifestations include symmetric, painless, and rather proximal then distal limb paresis that occurs within weeks to months [2]. Pulmonary involvement is a common complication in PM/DM and is associated with poor clinical outcomes and elevated mortality rates [5]. The overall prognosis of PM/DM remains unfavourable. The reported mortality rates range from 50% to 61%. In addition to reduced quality of life, affected individuals are at an elevated risk for various comorbidities, including osteoporosis and cardiovascular disease [3]. Emerging evidence indicates that individuals with inflammatory conditions are predisposed to a hypercoagulable state, particularly during the initial stages of disease onset [6]. Venous thromboembolism (VTE), comprising deep vein thrombosis (DVT) and pulmonary embolism (PE), is the third most prevalent cardiovascular disorder, following myocardial infarction and stroke [7]. VTE affects about 1-2 per 1,000 individuals per year [8].According to Virchow’s triad, VTE results from stasis, changes in blood coagulability and alterations in vessel wall structure [8]. The risk of VTE in patients with inflammatory myopathies has been largely underrecognized.

We report a case of pulmonary embolism in a patient diagnosed with previously unrecognized polymyositis. This case highlights a clinically significant, yet underreported, association between inflammatory myopathies and venous thromboembolic events. It emphasizes the importance of considering underlying inflammatory myopathies in patients presenting with unexplained pulmonary symptoms and contributes to the growing body of evidence guiding the evaluation and management of such cases.

Case Presentation:

A male in his 40's having a medical history of hypertension and hyperuricemia presented with an 8-day history of acute dyspnea and pleuritic chest pain. The symptoms began shortly after returning from a recent trip to Europe. He reported a dry cough but denied fever, muscle weakness, gastrointestinal or lower urinary tract symptoms, and recent weight loss. There was no history of allergies. Both surgical and family histories were unremarkable. The patient was a non-smoker, consumed alcohol occasionally.

On physical examination, the patient was awake, alert, and fully oriented. Pulmonary auscultation revealed clear bilateral breath sounds. The abdomen was soft, non-tender, and bowel sounds were present. Cardiovascular examination was normal, with no peripheral edema. Vital signs were as follows: temperature 37.8 °C, blood pressure 154/83 mmHg, heart rate 108 beats/min, respiratory rate 16 breaths/min, and oxygen saturation 100% on room air.

Initial laboratory evaluation revealed elevated levels of D-dimer, total creatine kinase (CK), and lactate dehydrogenase (LDH) (Table 1). Given the elevated D-dimer in the context of pleuritic chest pain and recent long-haul travel, an urgent computed tomography pulmonary angiogram (CTPA) was performed (Figure 1), which confirmed the presence of multiple pulmonary emboli. The patient was promptly initiated on therapeutic anticoagulation therapy with low-molecular-weight heparin (LMWH), which was subsequently transitioned to rivaroxaban, a direct oral anticoagulant. The patient was closely monitored during follow-up and demonstrated clinical improvement.

Labs	Reference range	Results
HB	13.0-17.5 g/dL	10
WBC	4-11 $10^3/\mu L$	11
Platelets	150-450 $10^3/\mu L$	278
D-dimer	0.00-0.50 ug/ml	2.63
Sodium (Na)	136-145 mmol/L	138
Potassium (K)	3.5-5.1 mmol/L	4.0
Creatine Kinase (CK)	30.00-200.00 U/L	4861
ESR	0-20 mm/Hr.	64
Ck-MB	0-5.1 ug/L	47
TROPONIN-I	0.00-0.0342 ng/ml	0.003
ALT	0-55 U/L	137
AST	0-55 U/L	140
ALBUMIN	35.00-52.00 g/L	29
CRP	0.0-5.0 mg/L	11
LDH	125-220 U/L	487
FERRITIN	30.00-274 ng/L	712
ANTICARDIOLIPIN IgM	< 20: Negative	2.3
TSH	0.350-4.9 µIU/ml	1.39
FREE T_3	2.64-5.43 pmol/L	3.30
FREE T_4	9-19 pmol/L	15

Table 1: Laboratory investigations

Abbreviations: HB, hemoglobin; WBC, white blood cell; ESR, erythrocyte sedimentation rate; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRP, C-reactive protein; LDH, lactate dehydrogenase; TSH, thyroid-stimulating hormone.

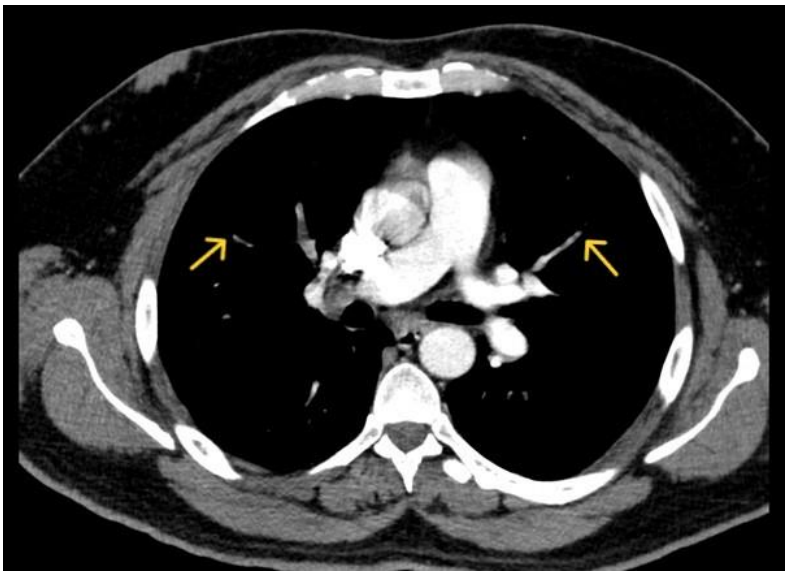

Figure 1: CT pulmonary angiogram showing filling defects in second and third generation branches of both lungs indicating bilateral lower lobe pulmonary embolism (Yellow arrows)

Transthoracic echocardiography was performed and revealed no evidence of right ventricular strain. Given the presence of cough, a respiratory pathogen panel was performed and returned positive for *Bordetella pertussis*. The patient was treated with the macrolide antibiotic Azithromycin. Considering the markedly elevated creatine kinase (CK) levels, intravenous fluid therapy was initiated for suspected rhabdomyolysis. A repeat CK measurement after 24 hours showed a reduction to 3000 U/L. However, CK levels remained fluctuating during the hospital course. Rheumatology consultation was obtained, and an autoimmune workup was initiated prior to discharge.

At the follow-up appointment, results of the autoimmune workup (Table 2) revealed a positive antinuclear antibody (ANA) with a diffuse granular cytoplasmic pattern.

Labs	Results	Reference range
ANA screen (IFA)	Positive (2+)	Negative
Nucleosomes antibody	Negative	Negative
Ds-DNA	Negative	Negative
Histones antibody	Negative	Negative
Sm antibody	Negative	Negative
RNP (68kd/A/C) antibody	Negative	Negative
Sm/RNP antibody	Negative	Negative
SSA/Ro-60 antibody	Negative	Negative
SSA/Ro-52 antibody	Negative	Negative
SS-B antibody	Negative	Negative
Scl-70 antibody	Negative	Negative
Ku antibody	Negative	Negative
PM-Scl 100	Negative	Negative
Mi-2 antibody	Negative	Negative
Jo-1 antibody	Negative	Negative
PL-7 antibody	Negative	Negative
PL-12 antibody	Negative	Negative
SRP-54 antibody	Negative	Negative
Ribosomal P antibody	Negative	Negative
CENP-A/B (Centromere) antibody	Negative	Negative
PCNA antibody	Negative	Negative
Sp100 antibody	Negative	Negative
Gp210 antibody	Negative	Negative
AMA-M2 (recombinant) antibody	Negative	Negative
AMA-M2 (native) antibody	Negative	Negative
MPO-ANCA	3.6 CU	< 20 CU
PR3-ANCA	< 2.3 CU	< 20 CU
Rheumatoid Factor	< 9 IU/mL	Negative <30/ Weakly positive 30-50/ Positive > 50 IU/mL
Angiotensin-1-converting enzyme (ACE)	11.42 U/L	20-70 U/L
Endomysium-IgA antibody	Negative	< 1:10: Negative
Beta-2 Glycoprotein I-IgG antibody	7.1 CU	< 20 CU
Beta-2 Glycoprotein I-IgM antibody	1.7 CU	< 20 CU

Table 2: Laboratory investigation

Abbreviations: ANA (antinuclear antibody); ds DNA (double stranded deoxyribonucleic acid); Sm (smooth muscle); RNP (ribonuclear protein); SSA/SSB (anti-Sjogren's syndrome related antigen A/B); Scl (systemic sclerosis); PM (polymyositis); PCNA (proliferating cell nuclear antigen); AMA (antimitochondrial antibody); ANCA (antineutrophil cytoplasmic antibody); MPO (myeloperoxidase); PR3 (proteinase 3).

Based on elevated CK levels and a positive autoimmune serology, an initial diagnosis of polymyositis was considered. Magnetic resonance imaging (MRI) of the thighs demonstrated diffuse muscle edema and T2-weighted hyperintensity involving the bilateral obturator externus, pectineus, iliopsoas, adductor and quadriceps muscles (Figure 2 and 3).

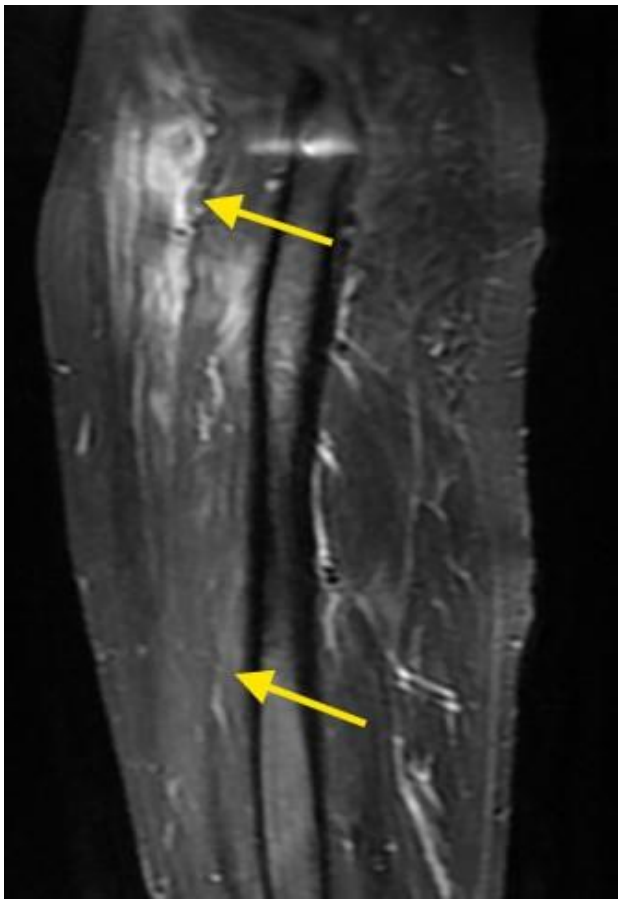

Figure 2: MRI scan of right thigh showing edema and inflammation of thigh muscles (yellow arrows).

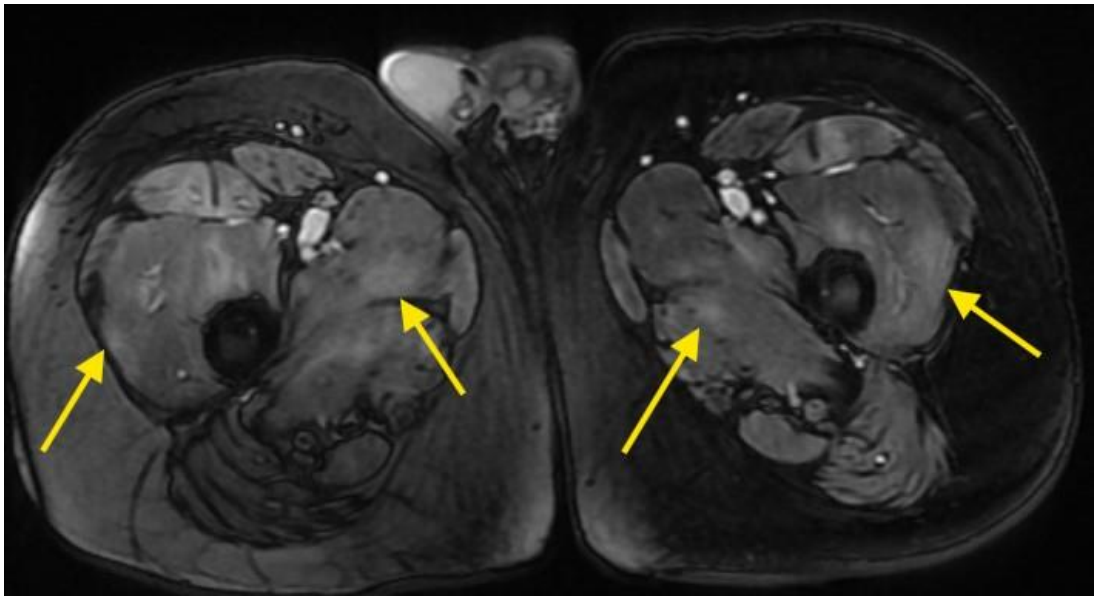

Figure 3: MRI scan of bilateral thighs showing diffuse edema and inflammation of bilateral thigh muscles (Obturator externus, pectineus, iliopsoas, adductor muscles and quadriceps muscles) indicated by yellow arrows.

MRI also revealed a multifocal, patchy distribution of muscle edema, findings highly suggestive of inflammatory myopathy consistent with polymyositis. High-resolution computed tomography (CT) of the chest showed no evidence of interstitial lung disease. Electromyography (Table 3) demonstrated a myopathic pattern characterized by spontaneous activity, further supporting the diagnosis of inflammatory myositis. In the absence of muscle biopsy facilities, the patient was initiated on a three-day course of high-dose corticosteroids, followed by maintenance immunosuppressive therapy with azathioprine. Over a course of ne year the CK levels normalized, and the immunosuppressant therapy was stopped.

Muscle	Nerve	Roots	IA	Fib	PSW	Fasc	H.F.	Amp	Dur.	PPP	Pattern
R. Deltoid	Axillary	C5-C6	1+	1+	None	None	None	N	N	N	N
R. Triceps brachii	Radial	C6-C8	N	None	None	None	None	N	N	N	N
R. Biceps brachii	Musculocut-aneous	C5-C6	N	1+	1+	None	None	Reduced	Reduced	N	N
R. Vastus medialis	Femoral	L2-L4	N	None	None	None	None	N	N	N	N
R. Tibialis anterior	Deep Peroneal (Fibular)	L4-L5	N	1+	None	None	None	Reduced	Reduced	N	N
L. Deltoid	Axillary	C5-C6	N	None	None	None	None	Reduced	Reduced	N	N
L. Biceps brachii	Musculocut-aneous	C5-C6	N	2+	None	None	None	N	N	N	N
L. Vastus medialis	Femoral	L2-L4	N	1+	None	None	None	Reduced	Reduced	N	N
L. Tibialis anterior	Deep peroneal (Fibular)	L4-L5	N	1+	None	None	None	Reduced	N	N	N

Table 3: Electromyogram study

Abbreviation: R (Right); L (Left); N (Normal); C (Cervical); L (Lumbar)

Discussion:

The diagnosis of polymyositis was established based on elevated creatine kinase (CK) and lactate dehydrogenase (LDH) levels, electromyographic evidence of myopathic changes, and MRI findings consistent with muscle inflammation. Although a muscle biopsy was not performed, the clinical and diagnostic findings strongly supported the diagnosis. The underlying etiology of polymyositis remains unclear [1]. As systemic autoimmune rheumatic diseases, polymyositis (PM) and dermatomyositis (DM) are associated with significant morbidity, elevated mortality, and considerable healthcare burden [7]. Prevalence of PM and DM is 21.5/100000, found to be higher in women and older individuals [8]. The overall prognosis of these conditions remains poor.PE itself is associated with mortality rate of 15% in the first three month of diagnosis [7].

Current literature on the association between PM/DM and venous thromboembolism (VTE) remains limited [3]. Altered immune responses—particularly to viral infections—are thought to contribute to vascular immune complex and complement deposition, leading to endothelial injury and small vessel obstruction [4]. Although pulmonary involvement is reported in only a subset of PM cases, its presence is associated with a more severe disease course and worse prognosis [4]. Several plausible mechanisms have been proposed to explain the increased VTE risk in autoimmune inflammatory diseases. Virchow's triad—comprising venous stasis, hypercoagulability, and endothelial injury—provides a framework for understanding this risk [8]. Inflammatory processes can enhance thrombotic potential by upregulating procoagulant factors and inhibiting fibrinolysis, thereby contributing to a hypercoagulable state [7].

The association between polymyositis (PM) and pulmonary embolism (PE) remains poorly characterized, with limited data available—most of which are derived from studies in Caucasian populations. Li et al. [3] reported a significant association between PM/dermatomyositis (DM) and venous thromboembolism (VTE) risk in a Caucasian cohort. Similarly, Antovic et al. [6] demonstrated a markedly elevated relative risk and hazard ratio of 7.81 (95% CI: 4.74–12.85) for VTE in patients with inflammatory myopathies compared to the general population. Carruthers et al. [7] also found a significantly increased incidence of VTE, including PE, among patients with PM/DM. A meta-analysis conducted by Young et al. [9] further confirmed this association, reporting a relative risk (RR) of 4.364 (95% CI, $p = 5.8 \times 10^{-5}$). Another metanalysis including 9045 patients affected by PM and DM showed an overall risk of VTE Odds Ratio (OR) of 4.31; in particular, PM-associated OR of VTE was estimated at 6.87, and DM-associated risk at OR of 8.67 [1].

In a Chinese cohort, Chen et al. [10] observed thromboembolic events in 54.2% (13/24) of patients within six months before or after the diagnosis of inflammatory myopathy. Chung et al. [11], in a large-scale study of 2031 patients with PM/DM and 8124 matched controls, reported an 11.1-fold increased risk of VTE in the PM/DM group. More recently, Li et al. [12] examined mortality trends across two time cohorts (1997–2005 and 2006–2014) and found declining mortality rates in patients with PM and DM. However, a significant residual mortality gap persisted in the more recent cohort. Multivariable hazard ratios for PM were 2.4 (95% CI: 1.7–3.4) and 2.0 (95% CI: 1.4–2.9) in the earlier and later cohorts, respectively. In a study by Li et al. [3] significant relationship between PM/DM and VTE risk was observed among the Caucasian population. These findings underscore the ongoing burden of disease and the need for further research into the thromboembolic risks associated with inflammatory myopathies.

In patients with inflammatory myopathies, dyspnea is often attributed to interstitial lung disease (ILD), a known pulmonary manifestation of these conditions. However, in the present case, imaging revealed no evidence of ILD. This case underscores the importance of considering alternative etiologies, such as pulmonary embolism, in the differential diagnosis of respiratory symptoms in patients with inflammatory myopathies. It highlights the need for comprehensive evaluation and may contribute to improving the clinical management of such presentations.

Conclusion:

This case highlights a rare but clinically significant association between polymyositis and pulmonary embolism. Evidence from existing studies suggests that the risk of pulmonary embolism is highest during the early phase following the diagnosis of polymyositis. Therefore, patients with inflammatory myopathies presenting with respiratory symptoms such as chest pain or dyspnea should be promptly evaluated for thromboembolic complications. Conversely, in patients diagnosed with pulmonary embolism, the presence of signs or symptoms suggestive of inflammatory myopathy or arthropathy should prompt further rheumatologic assessment to ensure timely diagnosis and management.

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