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CASE REPORT

A Case Report of Inflammatory Myositis in a Patient with Uncontrolled Type 2 Diabetes and Hypertension

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Abstract: Inflammatory myositis presents a significant clinical challenge when occurring alongside metabolic disorders. We report a case of a 48-year-old male who presented with acute onset proximal muscle weakness, calf swelling, and intermittent fever. The patient had a history of uncontrolled Type 2 Diabetes Mellitus (T2DM) with an HbA1c of 10.1% and hypertension (HTN). Laboratory investigations revealed elevated CPK-NAC and LDH levels, while nerve conduction studies demonstrated motor axonal and demyelinating neuropathy affecting bilateral peroneal, tibial, and median motor nerves. Histopathological examination confirmed the diagnosis of myositis. The patient also developed cellulitis, necessitating broad-spectrum antibiotic therapy. Treatment protocol included glycemic control with insulin (both regular and long-acting) and oral hypoglycemics, antihypertensive medication, corticosteroids, and hydroxychloroquine. The case revealed notable interactions between chronic metabolic disorders and inflammatory muscle disease, where persistent hyperglycemia potentially contributed to immune dysregulation and microvascular damage. The patient showed improvement with the implemented therapeutic regimen, though a muscle biopsy was recommended for further diagnostic clarity.

Keywords: Inflammatory Myositis; Type 2 Diabetes Mellitus; Hypertension; Cellulitis; Immune Dysregulation.

1. Introduction

Inflammatory myositis represents a group of rare muscle disorders characterized by chronic inflammation and progressive weakness of skeletal muscles [1]. The condition's pathogenesis involves complex immunological mechanisms, where autoimmune responses target muscle fibers, leading to inflammation, necrosis, and impaired regeneration [2, 3]. While the primary etiology often remains unclear, various factors including autoimmune disorders, infections, genetic predisposition, and metabolic disturbances contribute to its development [4].

The relationship between inflammatory myositis and metabolic disorders, particularly Type 2 Diabetes Mellitus (T2DM), presents a unique clinical challenge. Persistent hyperglycemia induces systemic inflammation through advanced glycation end-products formation and oxidative stress pathways [5]. These mechanisms can trigger immune dysregulation and enhance susceptibility to inflammatory conditions, including myositis [6]. Additionally, diabetic microangiopathy compromises muscle perfusion, potentially exacerbating muscle inflammation and weakness [7]. The presence of Hypertension (HTN) adds another layer of complexity in this case. Chronic elevation in blood pressure promotes endothelial dysfunction and vascular remodeling, leading to tissue hypoxia and inflammation [8]. The combination of HTN and T2DM accelerates microvascular damage through shared pathways involving oxidative stress and pro-inflammatory cytokine production [9].

Recent molecular studies have identified specific inflammatory mediators common to both metabolic disorders and myositis. These include TNF-α, IL-6, and other cytokines that perpetuate muscle inflammation and insulin resistance [10]. The presence of these inflammatory markers suggests a bidirectional relationship between metabolic dysfunction and muscle inflammation [11]. The clinical management of concurrent myositis, T2DM, and HTN requires careful consideration of multiple factors.

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Immunosuppressive therapy, while necessary for controlling muscle inflammation, can affect glycemic control and potentially increase infection risk [12]. Similarly, the presence of HTN may influence the choice of corticosteroid dosing and duration [13].

2. Case Report

2.1. Clinical Presentation

A 48-year-old male presented to the male medicine ward in December 2024 with complaints of progressive proximal muscle weakness, accompanied by swelling and erythema in the calf region. The patient reported being asymptomatic until seven days prior to admission when he developed intermittent fever. His medical history included Type 2 Diabetes Mellitus and hypertension, both diagnosed five years ago, with suboptimal control despite prescribed medications.

2.2. Physical Examination

Upon admission, vital signs revealed hypertension (150/98 mmHg), mild tachycardia (heart rate 90 beats/minute), normal respiratory rate (20 breaths/minute), and normal body temperature (98.4°F). Physical examination demonstrated bilateral proximal muscle weakness, more pronounced in the lower extremities, with a muscle strength grade of 3/5. The calf regions showed signs of cellulitis with local warmth, tenderness, and erythema.

2.3. Laboratory Investigations

Initial laboratory investigations revealed significant metabolic derangement, with an HbA1c of 10.1% and random plasma glucose of 406 mg/dL. Muscle enzyme studies showed markedly elevated CPK-NAC (2,845 U/L) and LDH (456 U/L) levels. Additional laboratory findings included reduced hemoglobin (10.2 g/dL), low total protein (5.8 g/dL), and elevated serum lipase (182 U/L). Peripheral blood smear analysis confirmed normocytic normochromic anemia, while inflammatory markers showed elevation with ESR at 68 mm/hr and CRP at 24 mg/L.

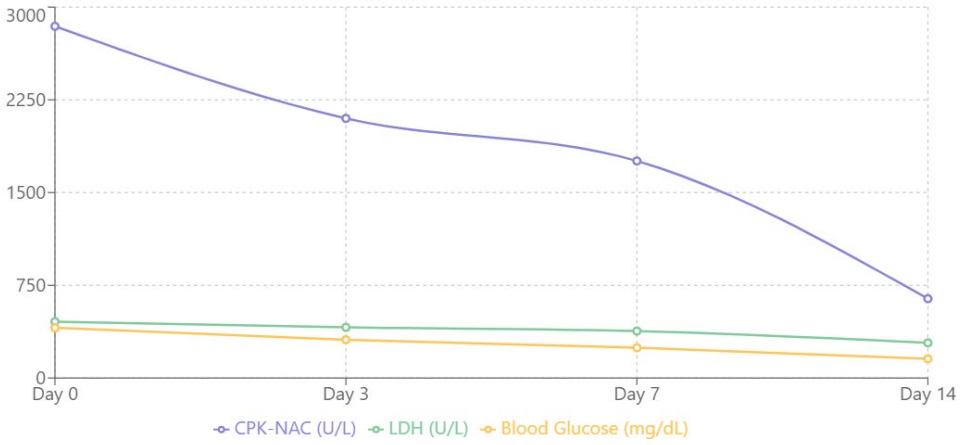

Figure 1. Inflammatory and metabolic markers during hospital stay

2.4. Diagnostic Studies

Nerve conduction studies revealed motor axonal and demyelinating neuropathy affecting bilateral peroneal, tibial, and median motor nerves. The histopathological examination of muscle tissue demonstrated inflammatory infiltrates with muscle fiber degeneration and regeneration, confirming the diagnosis of myositis.

2.5. Treatment and Management

The patient received intravenous regular insulin according to sliding scale, with insulin glargine (Lantus) initiated at 10 units and subsequently increased to 14 units. Oral antidiabetic therapy consisted of metformin and vildagliptin (500/50mg) administered twice daily. Blood pressure management included amlodipine 5mg daily. For the inflammatory component, prednisolone was administered at 40mg in the morning and 30mg in the evening, along with hydroxychloroquine 200mg twice daily. Cellulitis was treated with intravenous piperacillin-tazobactam 4.5g administered thrice daily. The supportive care regimen included normal saline infusion at 30ml/hr, with ondansetron for nausea control, paracetamol 650mg for pain management, and clonazepam at bedtime for sleep regulation.

2.6. Clinical Course and Follow-up

Throughout the hospital stay, the patient received comprehensive dietary counseling and medication education. Regular monitoring of blood glucose levels, blood pressure, and muscle strength showed gradual improvement. Prior to discharge, the patient was advised to maintain regular follow-up with his primary care physician and was recommended for further muscle biopsy.

Table 1. Clinical and Laboratory Parameters Throughout Hospital Stay

Parameters	Day of Admission	Day 3	Day 7	Day of Discharge (Day 14)
Muscle Strength (MRC Scale)				
Proximal Upper Limb	3/5	3/5	3/5	4/5
Proximal Lower Limb	2/5	2/5	3/5	4/5
Distal Upper Limb	4/5	4/5	4/5	5/5
Distal Lower Limb	3/5	3/5	4/5	4/5
Laboratory Parameters				
CPK-NAC (U/L)	2,845	2,100	1,756	642
LDH (U/L)	456	410	380	285
Random Blood Glucose (mg/dL)	406	310	245	156
HbA1c (%)	10.1	-	-	9.2
ESR (mm/hr)	68	55	45	32
CRP (mg/L)	24	20	18	10
Vital Signs				
Blood Pressure (mmHg)	150/98	145/90	142/88	130/82
Heart Rate (beats/min)	90	86	84	78
Temperature (°F)	98.4	98.3	98.2	98.0
Medication Dosage				
Prednisolone (mg/day)	70	70	60	50
Insulin Glargine (units)	10	10	12	14
Amlodipine (mg)	5	5	5	5

MRC: Medical Research Council; CPK-NAC: Creatine Phosphokinase N-acetyl L-cysteine; LDH: Lactate Dehydrogenase; ESR: Erythrocyte Sedimentation Rate; CRP: C-Reactive Protein

3. Discussion

The presented case shows the interrelation between inflammatory myositis, Type 2 Diabetes Mellitus (T2DM), and hypertension (HTN). The patient's initial presentation of proximal muscle weakness, accompanied by significantly elevated CPK-NAC and LDH levels, aligns with classical myositis manifestations [14]. The concurrent presence of poorly controlled diabetes, evidenced by an HbA1c of 10.1%, suggests a potential pathophysiological link between metabolic dysregulation and muscle inflammation [15].

Chronic hyperglycemia induces a pro-inflammatory state through multiple pathways, including advanced glycation end-products formation and oxidative stress generation [16]. These mechanisms can trigger autoimmune responses and exacerbate muscle inflammation. The presence of motor axonal and demyelinating neuropathy in this patient further supports the role of diabetic complications in neuromuscular dysfunction [17]. Additionally, hypertension-induced endothelial dysfunction may compound microvascular damage, potentially contributing to muscle ischemia and inflammation [18].

The patient's presentation of proximal muscle weakness, coupled with elevated muscle enzymes and confirmatory histopathological findings, established the diagnosis of myositis [19]. The normocytic normochromic anemia observed likely reflects chronic disease processes and inflammatory mediators' effects on erythropoiesis [20]. The development of cellulitis highlights the increased susceptibility to infections in diabetic patients, necessitating prompt antimicrobial intervention [21].

Glycemic control was achieved through a combination of basal-bolus insulin therapy and oral hypoglycemic agents, addressing the underlying metabolic dysfunction [22]. The use of corticosteroids for myositis management required careful consideration due to their potential impact on glycemic control. The addition of hydroxychloroquine served dual purposes - as an immunomodulator for myositis and a potential glucose-lowering agent [23].

Antihypertensive therapy with amlodipine is used for blood pressure control while considering the patient's concurrent conditions. The choice of calcium channel blocker was appropriate given its neutral metabolic profile and effectiveness in reducing cardiovascular risk in diabetic patients [24]. Motor neuropathy findings in this case emphasize the importance of regular neurological assessment in diabetic patients with muscle symptoms [25, 26].

4. Conclusion

This case serves as an example of complex relation between inflammatory myositis, T2DM, and HTN and demonstrates the challenges in managing concurrent autoimmune and metabolic disorders. The successful management required a delicate balance between immunosuppression, glycemic control, and cardiovascular risk reduction. The case also indicates the importance of a comprehensive approach to patient care, considering the potential interactions between multiple disease processes. Early recognition of inflammatory myositis in patients with underlying metabolic disorders, coupled with appropriate therapeutic intervention, can significantly improve clinical outcomes. Long-term follow-up remains crucial for monitoring disease progression and adjusting treatment strategies accordingly

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