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Glucocorticoid-Induced Myopathy in a Patient with Systemic Lupus Erythematosus (SLE): A Case Report and Review of the Literature

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Patient: Female, 35
Final Diagnosis: Glucocorticoid-induced myopathy
Symptoms: Generalized weakness
Medication: Prednisone
Clinical Procedure: —
Specialty: General and Internal Medicine

Objective: Unusual clinical course

Background: Chronic intake of high-dose corticosteroids is associated with multiple adverse clinical effects, including hypertension, insulin resistance, impaired wound healing, immunosuppression, myopathy, and osteoporosis. In cases of autoimmune disease, use of steroid-sparing treatment modalities is preferred over chronic steroid therapy to limit these side effects. Glucocorticoid-induced myopathy is a less common side effect of chronic steroid use in patients treated with <10 mg/day of prednisone. However, doses exceeding 40–60 mg/day can induce clinically significant myopathy and weakness.

Case Report: A 35-year-old woman with a past medical history of hypothyroidism, systemic lupus erythematosus (SLE), and end-stage renal disease secondary to lupus nephritis, on hemodialysis, presented to the local emergency department with progressive bilateral proximal lower extremity weakness. Three months before admission, when her insurance company prematurely discontinued her monthly cyclophosphamide injections, at which time, she was treated with prednisone 60 mg daily. Two months before hospital admission, she reported increasing fatigue, weight gain, difficulty in standing from a seated position and climbing stairs.

Conclusions: Elucidating the etiology of progressive neuromotor deficit in immunosuppressed patients can be difficult. The management of SLE and other autoimmune diseases with chronic high-dose steroids is associated with recognized side effects. Differentiating natural disease progression from iatrogenic etiologies is important in this subset of patients, particularly to reduce prolonged clinical management and hospital admissions.

MeSH Keywords: Glucocorticoids • Lupus Erythematosus, Systemic • Muscular Diseases

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Background

The term, myopathy, refers to the loss of contractile function of muscle that results in weakness. Myopathies are distinct from neuromuscular junction disease and neuropathic causes of weakness, in that the primary defect is within the muscle tissue. The etiology of myopathy varies widely and includes endocrine disorders, nutritional deficiency, metabolic disease, drug-induced or toxic causes, congenital disease, inflammatory, and infectious processes.

Endocrine disorders of the thyroid, parathyroid, adrenal, and pituitary glands can produce hormonally-mediated alterations in metabolism that can disrupt myocyte function, leading to weakness. Electrolyte and vitamin abnormalities including hypokalemia (serum potassium <2.5 mEq/l), hypercalcemia (serum calcium >14 mg/dl), and vitamin D deficiency (25(OH) D3) (<10 ng/ml) may also lead to symptomatic weakness [1]. Metabolic myopathies include disorders of carbohydrate, purine, and lipid metabolism [1]. The presentation of metabolic myopathies can vary, and affected individuals may present with dynamic symptoms, such as exercise intolerance, acutely reversible weakness, and myoglobinuria, or may present with static symptoms, including fixed weakness, cardiomyopathy, and neuropathy.

Iatrogenic causes of myopathy include the chronic use of corticosteroids, colchicine, antimalarials, and statins, and are often reversible with cessation of use of the drug. Illicit drug use and alcohol abuse have recognized associations with myopathy. Specifically, cocaine is proposed to induce muscle injury through increased sympathomimetic activity and arterial vasoconstriction [2]. Alcohol and its metabolites can disrupt intracellular metabolism and cellular membrane structures, and there are reported cases of both acute and chronic myopathy following 'binge' drinking and chronic consumption, respectively [3].

Congenital myopathies include central-core disease, nemaline and myotubular myopathies, which are usually present at birth or infancy. Affected individuals can present with variable degrees of non-progressive or slowly progressive generalized weakness, hypotonia and, in severe cases, may present as a 'floppy infant.' The diagnosis of congenital myopathies requires histological examination to identify pathological features of type-1 muscle fibers that are unique to these diseases. Conversely, inflammatory myopathies more commonly present in adulthood and fall into three distinct categories: infectious, noninfectious inflammatory, and systemic inflammatory disease. Infection-related myopathies are often viral in origin, and are due to influenza, parainfluenza, human immune deficiency virus (HIV), Epstein Barr virus (EBV), and Echovirus, but may also be due to bacterial myositis (Lyme disease) and parasitic (toxoplasmosis) etiologies. Noninfectious inflammatory myopathies include dermatomyositis, polymyositis, and inclusion body myositis (hereditary inflammatory myopathy). Systemic inflammatory myopathies are often associated with autoimmune diseases and include systemic lupus erythematosus (SLE), Sjögren's syndrome, rheumatoid arthritis, and scleroderma.

SLE is a prototypic multisystem disease of autoimmune origin and is characterized by a fundamental failure of mechanisms that maintain self-tolerance. Autoimmune injury leads to tissue damage and apoptosis, together with an inadequate clearance of nuclear material, that may result in the exposure of a large burden of nuclear antigens to the immune system. Underlying abnormalities in B-lymphocytes and T-lymphocytes allow for the survival of self-reactive lymphocytes, which are then stimulated by 'self' nuclear antigens, allowing antibody production against these antigens. The net result is a cycle of antigen release and immune cell activation resulting in the production of high-affinity autoantibodies [4]. As a result, virtually any organ in the body may be affected in SLE, including skeletal muscle.

The mainstay of treatment of inflammatory myopathies is immune suppression, including the use of corticosteroids. However, the prolonged use of corticosteroids is associated with multiple adverse effects, including hypertension, insulin resistance, poor wound healing, immunosuppression, myopathy, and osteoporosis. Glucocorticoids have been shown to induce myopathy by their direct catabolic effect on skeletal muscle, leading to increased levels of amino acids that can be utilized by the liver for gluconeogenesis. Also, glucocorticoid-induced suppression of the serine/threonine-protein kinase, AKT1, has been shown to increase ubiquitin-ligase atrogin-1 (MAFbx) that targets skeletal muscle proteins for degradation [5]. In cases of autoimmune disease, use of steroid-sparing treatment modalities is preferred over chronic steroid therapy, to limit the side effects.

Glucocorticoid-induced myopathy is a less frequent effect of chronic steroid use in patients treated with <10 mg/day of prednisone. However, doses exceeding 40–60 mg/day can induce clinically significant weakness in less than two weeks [6]. The likelihood of developing glucocorticoid-induced myopathy increases with duration of use. However, the overall frequency of this adverse effect is not well defined.

This case report describes a 35-year-old woman who presented with progressive bilateral lower extremity weakness in the setting of hypothyroidism, nephritis due to SLE, and chronic steroid use.

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Case Report

A 35-year-old woman with a past medical history of hypothyroidism, systemic lupus erythematosus (SLE), and end-stage renal disease secondary to lupus nephritis was treated with hemodialysis on alternate days, three days a week. She presented to the emergency department of her local hospital with progressive bilateral proximal lower extremity weakness.

The patient had been in her usual state of health until approximately three months before admission when her insurance company prematurely discontinued her monthly cyclophosphamide injections for treatment of lupus nephritis. She was then started on prednisone 60 mg daily. Two months before admission, she reported increasing fatigue, weight gain and muscle weakness with increasing difficulty standing from a seated position and difficulty climbing stairs. At this time, she also reported increasing numbness and tingling in her distal lower extremities. One month before admission her weakness progressed, and she was no longer able to ambulate independently. Her symptoms were reportedly constant, without aggravating or alleviating factors. On admission, she denied any previous episodes of weakness, pain, urinary or fecal incontinence, recent illness, fever, chills, nausea, vomiting, or diaphoresis.

On examination, the patient appeared well with a Cushingoid habitus. Her blood pressure was 127/59 mmHg, pulse rate 110 beats per minute, temperature 37.1°C, respiratory rate 18 breaths per minute, oxygen saturation 98% while on room air, and she rated her level of pain as 0 out of 10. Cardiac and respiratory examination findings were normal. Examination of the abdomen showed central obesity with multiple linear striae along the bilateral flanks but was otherwise normal. Inspection of her lower limbs showed symmetrical muscle tone without atrophy. Her skin was intact without erythema, ecchymoses, or signs of skin thinning. Palpation of the lower limbs showed no tenderness or myalgia. There was decreased sensation to light touch in the L4/L5 dermatomes. Motor function in all four extremities was grossly intact, but on strength testing, findings included, bilateral dorsiflexion +5, plantarflexion +5, and hip flexion +1. Biceps, triceps, patellar, and Achilles reflexes were 2+ and normal.

Laboratory test results showed hematocrit (35%), hemoglobin (9.4 g/dl), mean corpuscular volume (MCV) 95 μm^3, white blood cell count (WBC) 6.5 per mm^3, platelets 73,000 per mm^3, urea nitrogen 44 mg/dl, and creatinine 6.23 mg/dl. Blood levels of electrolytes, glucose, total protein, total bilirubin, alanine aminotransferase (ALT), and alkaline phosphatase (ALP) were normal. An electrocardiogram (ECG) showed sinus tachycardia.

Magnetic resonance imaging (MRI) of her lumbar spine was obtained and showed a compression deformity of the superior endplate of L1, with approximately 40% height loss and 1mm fragment retropulsion without evidence of significant stenosis of the central spinal canal or neural foraminal stenosis. MRI T2-weighted imaging showed increased signal intensity in the disc space and adjacent endplates above L1, which were findings that were supportive for spondylodiscitis (discitis). Neurosurgery consultation resulted in the decision to treat the L1 fracture conservatively with a thoracolumbosacral orthosis (TLSO) brace. Following this hospital admission, the patient resumed her previous medications that included hydroxychloroquine, nifedipine, levothyroxine, and prednisone, and she resumed her regular hemodialysis schedule. Cyclophosphamide treatment was reinitiated for management of lupus nephropathy.

On the third hospital day, a nuclear medicine three-phase bone scan was performed to further evaluate discitis as a cause of her persistent weakness. The bone scan showed a mild increase in uptake at the L1 vertebral body, but overall findings were nonspecific. In the absence of fever, leukocytosis, or back pain in the area of the compromised vertebrae, her radiculopathy symptoms were considered to be unlikely for underlying discitis or epidural abscess.

With low clinical suspicion for spinal cord compression, the diagnostic workup proceeded to evaluate a myopathic etiology for the symptoms of weakness in this patient. On the fourth hospital day, chemistry, urinalysis, and serologic tests for infectious, inflammatory, and endocrine myopathies were obtained. Serum creatine kinase (CK), lactate dehydrogenase (LDH), and aldolase were normal, as shown in Table 1. Urine myoglobin was not detected. Serological studies for antibodies to for influenza, parainfluenza, adenovirus, Epstein-Barr virus (EBV), hepatitis B virus (HBV), hepatitis C virus (HCV), and human immune deficiency virus (HIV) were negative, and the rapid plasma reagin (RPR) test for syphilis was non-reactive. The patient's history of hypothyroidism raised the possibility of hypothyroid myopathy. However, the patient reported no other symptoms of hypothyroidism, and serum thyroid-stimulating hormone (TSH) was found to be within normal limits. Anti-Jo-1 and anti-Mi2 serologies were obtained to evaluate for inflammatory myositis (dermatomyositis, polymyositis) and were non-reactive. Autoimmune serology included measurement of anti-Ro (Sjögren's), anti-La (Sjögren's), anti-cyclic citrullinated peptide (CCP) (rheumatoid arthritis), anti-centromere (scleroderma), and anti-Scl-70 (scleroderma) were negative, and are listed in Table 2.

On the tenth hospital day, electromyography (EMG) was performed and showed low-amplitude motor unit potentials, consistent with a myopathic process. On the eleventh hospital day the patient was scheduled for a biopsy of her proximal thigh muscle; however, a left anterior tibialis biopsy was

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Table 1. Laboratory data (with reference ranges) on the day of admission.

Variable	Reference range (adults)	On admission
WBC count (per mm^3)	4,500–11,000	6,700
Differential count		
Neutrophils (%)	54–62	64
Lymphocytes (%)	25–33	26.8
Monocytes (%)	3–7	7.6
Eosinophils (%)	1–3	1.2
Basophils (%)	0–0.75	0.4
Hemoglobin (g/dL)	12.0–16.0	9.4
Hematocrit (%)	36–46	35
RBC (10^{12}/L)	3.94–5.66	4.22
MCV (fL)	80–96	97.7
Platelets (mm^3)	150,000–400,000	73,000
Sodium (mmol)	136–145	147
Potassium (mmol)	3.6–5	4.5
Calcium (mg/dL)	8.5–10.1	9.0
Magnesium (mg/dL)	1.8–2.4	2.0
Phosphorous (mg/dL)	2.5–4.9	2.4
Urea nitrogen (mg/dL)	7–18	44
Creatinine (mg/dL)	0.6–1.2	6.23
Hemoglobin A1c (%)	≤6	5.6
B12 (pg/mL)	>250	523
Vitamin D	N/A	N/A
Folate (ng/dL)	3.1–17.5	12
TSH (μU/mL)	0.5–5.0	3.4
Serum creatine kinase (CK) U/L	10–70	62
Lactate dehydrogenase (LDH) (U/L)	45–90	59
Aldolase (U/L)	<7.5	4.1
Copper (μg/dL)	63–140	82
Fe (μg/dL)	40–155	92
TIBC (μg/dL)	250–450	127
Ferritin (μg/L)	15–150	682
C-reactive protein (CRP) (mg/L)	0–10	1.45
Erythrocyte sedimentation rate (mm/hr)	<30	36

WBC – white blood cells; RBC – red blood cells; MCV – mean cell volume; TSH – thyroid stimulating hormone; Fe – iron; TIBC – total iron-binding capacity.

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Table 2. Serology data during admission.

Antigen	Result
Anti-Sm	1.1 Positive
Anti-Sm RNP	>8 Positive
Anti-DS DNA	9 Positive
ANA	1: 320
RPR	Non-reactive
HIV	Non-reactive
Influenza	Non-reactive
Parainfluenza	Non-reactive
Adenovirus	Non-reactive
Epstein-Barr virus	Non-reactive
Hepatitis B virus	Non-reactive
Hepatitis C virus	Non-reactive
Anti-Jo-1	Non-reactive
Anti-Mi2	Non-reactive
Anti-Ro	Non-reactive
Anti-La	Non-reactive
Anti-CCP	Non-reactive
Anti-centromere	Non-reactive
Anti-Scl-70	Non-reactive

Sm – smooth muscle; RNP – ribonucleoprotein; ANA – antinuclear antibody; RPR – rapid plasma reagin; HIV – human immune deficiency virus; CCP – cyclic citrullinated peptide.

performed instead, and a proximal muscle biopsy was not obtained. Histological examination showed no necrosis or inflammatory infiltrates to suggest myositis. In the absence of serological or pathological evidence of myopathy, the patient was continued on high doses of steroids due to concerns for an atypical SLE myelopathy (transverse myelitis). Treatment with intravenous immunoglobulins (IVIG) was initiated for possible idiopathic immune-mediated neuromuscular disease but without clinical improvement.

On the eighteenth hospital day, the patient was transferred to the intensive care unit (ICU) with acute encephalopathy and hypotension. Computed tomography (CT) and MRI of the brain were negative for acute intracranial abnormalities. Anti-ribosomal P protein serology was negative, which excluded neuropsychiatric manifestations of SLE. Cerebrospinal fluid (CSF) and blood cultures were obtained and found to be positive for Group B *Streptococcus* (GBS), *Streptococcus agalactiae*. The patient received appropriate antibiotic treatment, and all subsequent blood cultures were negative for GBS. In the following

Table 3. Medical problems prior to hospital admission.

Medical condition	Complications
• Lupus nephritis with ESRD • L1 compression fracture • CMV colitis and gastritis • Hypertension • Thrombocytopenia • Normocytic anemia • Discoid lupus under right breast • Oral herpes simplex virus • Hypothyroidism	• Seizure-like activity during hemodialysis • GBS meningitis and bacteremia; Resolved • Lower GI bleeding • Repeated transient hypoglycemia • VRE UTI • Klebsiella UTI • Hospital-acquired pneumonia • Steroid-induced myopathy

UTI – urinary tract infection; ESRD – end-stage renal disease; CMV – cytomegalovirus; VRE – vancomycin-resistant *E. coli*; GI – gastrointestiunal; GBS – Group B *Streptococcus*.

weeks, the patient's hospital course was complicated by extended ICU care, cytomegalovirus (CMV) gastritis, and colitis. Her clinical complications while in hospital are listed in Table 3.

After two months in the hospital, the patient was severely debilitated, and on examination, her lower extremity weakness remained unchanged. Although physical therapy had been attempted, this had been limited due to previous spinal surgery. A repeat MRI of her lumbar spine showed a stable compression deformity of the L1 vertebrae without evidence of osteomyelitis or discitis. In the absence of conclusive evidence of spinal cord compression, neuropathic, or myopathic disease, the patient was titrated off of her steroid regimen in an attempt to exclude glucocorticoid-induced myopathy. Intensive twice-daily physical therapy was initiated in the following weeks. The patient progressed from sitting at the edge of the bed to brief periods of standing with assistance. The correlation between the improvement in her symptoms and tapering of steroids supported a diagnosis of glucocorticoid-induced myopathy; however, repeat EMG was not performed.

Following a complicated three-month hospitalization, the patient was discharged to a subacute level of care for continued intensive physical rehabilitation. She was discharged in a stable condition with persistent paresthesiae, but with improved bilateral lower extremity motor strength.

Discussion

Understanding the pathophysiology of glucocorticoid-induced myopathy is essential for distinguishing organic versus

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iatrogenic etiologies of new-onset muscle weakness. Physical examination, measurement of serum creatine kinase (CK) or aldolase, electrophysiologic studies, and muscle biopsy findings can provide important information regarding the etiology of patients presenting with these symptoms. In patients with systemic lupus erythematosus (SLE), it is important to exclude reversible causes, including glucocorticoid-induced muscle weakness [7,8]. The differential diagnosis of progressive bilateral lower extremity may vary widely in patients with autoimmune disease. In this case, in the absence of apparent spinal cord compression, the differential diagnosis of myopathy included drug-induced myopathy, SLE neuropathy, SLE myopathy, SLE myelopathy, uremic polyneuropathy, and vitamin D deficiency.

The clinical manifestations of glucocorticoid-induced myopathy include proximal muscle weakness and atrophy of proximal muscle groups without myalgia or muscle tenderness. Weakness most commonly appears in the lower extremities prior to affecting the upper extremities. The onset of weakness may be dose-dependent and duration-dependent, and studies have shown that doses of 40–60 mg/day can induce weakness within two weeks [6]. Muscle atrophy may be exacerbated by inactivity, which sensitizes skeletal muscle to the catabolic effects of steroids. Serum CK or aldolase levels are typically within normal limits and are rarely elevated [6,9]. However, serum lactate dehydrogenase (LDH) levels may be elevated. Electromyography studies often show no abnormalities in conduction rates. Muscle biopsy of the affected muscle groups may show increased numbers of sarcolemmal nuclei, loss of fiber cross-striations of type IIb fibers, without necrosis or inflammation [6,9,10]. The diagnosis of glucocorticoid-induced myopathy is most often based on history and physical examination, as there is no definitive test for this condition. Symptoms of weakness commonly show improvement after three to four weeks following discontinuation of steroid therapy.

Chloroquine and hydroxychloroquine are commonly prescribed for the treatment of SLE. These medications impair complement-dependent antigen-antibody reactions and provide a useful tool in the management of severe or refractory SLE. However, chronic use of chloroquine and hydroxychloroquine can lead to proximal muscle myopathies, with associated weakness and atrophy. Muscle enzyme levels, particularly CK levels, are usually normal. The gold standard method for the diagnosis of drug-induced myopathy is muscle biopsy. Microscopic findings include presence of autophagic membrane-bound vacuoles containing membranous debris within myocytes. There are characteristic curvilinear bodies with short curved membranous structures with alternating light and dark zones most commonly seen in type 1 fibers [4]. Treatment includes cessation of the causative drug.

In between 10–15% of patients with SLE, peripheral neuropathy secondary to vasculopathies of small arteries supplying the affected nerves can be a complication [11]. Clinically, patients often present with asymmetric paresthesiae and numbness of the digits that is worse at night [12]. Acute forms of peripheral neuropathy can present with ascending areflexic motor weakness without sensory loss. The diagnosis of peripheral neuropathy is made with electromyography and nerve conduction studies, which show polyphasic conduction velocities of long duration and high amplitude, consistent with generalized sensorimotor peripheral neuropathy. This type of neuropathy responds well to glucocorticoid treatment, usually within three weeks. A rare form of SLE neuropathy is chronic inflammatory demyelinating polyradiculoneuropathy (CIPD) that presents with recurrent episodes of Guillain-Barré syndrome-like symptoms, mononeuritis multiplex, or symmetric polyradiculopathy, over a period of weeks to months [13,14]. Treatment may involve glucocorticoids and intravenous immunoglobulins (IVIG) or plasmapheresis.

Up to 70% of patients with SLE may present with myalgia, muscle tenderness, or weakness [15,16]. However, severe muscle weakness, atrophy, or myositis are uncommon, occurring in between 7–15% of patients [15,16]. Typically, diagnosis is made with muscle biopsy which shows perivascular and perifascicular mononuclear cell infiltrates in 25% of patients, or other inflammatory cell infiltration characteristic of SLE [17]. The diagnosis is supported by elevations in serum CK and aldolase. Conversely, antimalarial and glucocorticoid-induced myopathies show no inflammation, and serum CK and aldolase are usually normal. Treatment may involve glucocorticoids, with symptomatic improvement seen within two to three weeks.

SLE myelopathy involving the central nervous system (CNS) rarely occurs in SLE, affecting between 1–2% of patients [18]. Symptoms include the acute or subacute development of neurological signs and symptoms consistent with motor, sensory and/or autonomic dysfunction. Sudden onset of lower extremity weakness or sensory loss are common reasons for patients to seek medical evaluation, and fecal and urinary incontinence is usually present. The pathogenesis is thought to be secondary to arteritis, resulting in ischemic necrosis of the spinal cord. MRI, with and without gadolinium, should be used to exclude other causes of spinal cord compression. On T2-weighted MRI, hyperintense foci and localized edema around the spinal cord is a characteristic diagnostic feature. Analysis of cerebrospinal fluid (CSF) should be performed to rule out infection. However, CSF findings are abnormal in only half of all patients diagnosed with transverse myelitis [19]. Findings may include moderate lymphocytosis (typically <100/mm^3) and elevated protein (100–120 mg/dl) with normal glucose levels. Treatment may include the combination of prednisone, plasmapheresis, and cyclophosphamide [19].

The prevalence of uremic polyneuropathy increases linearly with increasing serum creatinine [16]. Initial sensory symptoms include

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paresthesiae involving the distal lower extremities that ascend with disease progression. In more advanced stages, patients may experience motor symptoms, including distal weakness, myoclonus, and potential paralysis [19]. Physical examination in early disease shows loss of position sense and vibration sense with decreased deep tendon reflexes. Late findings may include muscle atrophy and motor symptoms. The diagnosis of uremic polyneuropathy usually requires electrophysiologic studies to measure motor conduction velocity of the peroneal nerve and sensory conduction velocity of the sural nerve [20]. Treatment may range from medical management, dialysis, or renal transplantation.

Hypovitaminosis D is prevalent in more than half of patients with SLE, leading to loss of trabecular bone density [21,22]. In the setting of osteomalacia, vitamin D deficiency-induced myopathy is associated with proximal muscle weakness, hypotonia, and muscle wasting [23]. In patients with end-stage renal disease, serum levels of 25-hydroxyvitamin D, or 25(OH)D, below 50 nmol/l has been correlated with decreased quadriceps strength [24]. Vitamin D sufficiency can be assessed by measuring serum levels of 25(OH)D or calcidiol, and can be supplemented orally. In this case, measurement of vitamin D levels would have been useful in helping determine whether vitamin D insufficiency was a contributing factor to this patient's condition, but the findings may not have changed patient management.

Conclusions

This report is of a case of a patient who was first diagnosed with an incidental L1 compression fracture that prompted hospital admission and evaluation for discitis as a cause of bilateral lower extremity weakness. This case has been presented to raise the clinical suspicion for glucocorticoid-induced myopathy in patients presenting with muscle weakness in the context of chronic high-dose steroid use. Also, this case highlights the use of steroid-sparing modalities when appropriate, and recognizes the need for a multidisciplinary approach to the evaluation and treatment of patients with complex, multisystem autoimmune diseases, such as SLE.

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Conflict of interest

None.

References:

1. Inzucchi SE: Understanding hypercalcemia. Its metabolic basis, signs, and symptoms. Postgrad Med, 2004; 115(4): 69–70, 73–76
2. Zamora-Quezada JC, Dinerman H, Stadecker MJ, Kelly JJ: Muscle and skin infarction after free-basing cocaine (crack). Ann Intern Med, 1988; 108(4): 564–66
3. Haller RG, Knochel JP: Skeletal muscle disease in alcoholism. Med Clin North Am, 1984; 68(1): 91–103
4. Vinay K, Abbas AK, Fausto N et al: Robbins and cotran pathologic basis of disease. 8th ed. Philadelphia: Elsevier Saunders, 2010; 219–21
5. Stitt TN, Drujan D, Clarke BA et al: The IGF-1/PI3K/Akt pathway prevents expression of muscle atrophy-induced ubiquitin ligases by inhibiting FOXO transcription factors. Mol Cell, 2004; 14(3): 395–403
6. Bowyer SL, Lamothe MP, Hollister JR: Steroid myopathy: Incidence and detection in a population with asthma. J Allergy Clin Immunol, 1985; 76(2 Pt 1): 234–42
7. Isenber DA, Snaith ML: Muscle disease in systemic lupus erythematosus: A study of its nature, frequency and cause. J Rheumatol, 1981; 8(6): 917–24
8. Stevens MB: Musculoskeletal manifestations. In: The clinical management of systemic lupus erythematosus, Schur PH (ed.), Grune and Stratton, New York, 1983
9. Khaleeli AA, Edwards RH, Gohil K et al: Corticosteroid myopathy: A clinical and pathological study. Clin Endocrinol (Oxf), 1983; 18(2): 155–66
10. Afifi AK, Bergman RA, Harvey JC: Steroid myopathy. Clinical, histologic and cytologic observations. Johns Hopkins Med J, 1968; 123(4): 158–73
11. Florica B, Aghdassi E, Su J et al: Peripheral neuropathy in patients with systemic lupus erythematosus. Semin Arthritis Rheum, 2011; 41(2): 203–11
12. Omdal R, Henriksen OA, Mellgren SI, Husby G: Peripheral neuropathy in systemic lupus erythematosus. Neurology, 1991; 41(6): 808–11
13. Lewis M, Gibson T: Systemic lupus erythematous with recurrent Guillain-Barré-like syndrome treated with intravenous immunoglobulins. Lupus, 2003; 12(11): 857–59
14. Vina ER, Fang AJ, Wallace DJ, Weisman MH: Chronic inflammatory demyelinating polyneuropathy in patients with systemic lupus erythematosus: Prognosis and outcome. Semin Arthritis Rheum, 2005; 35(3): 175–84
15. Dubois EL, Wallace DJ: Clinical and laboratory manifestations of SLE. Dubois LE, Wallace DJ, Dubois EL (eds.), Lea and Febiger, Philadelphia, 1987; 317
16. Ropes MW: Systemic lupus erythematosus, Harvard University Press, Cambridge, 1976
17. Rothfield N: Clinical features of systemic lupus erythematosus. In: Textbook of rheumatology. Kelley WN, Harris ED, Ruddy S, Sledge CB (eds.), WB Saunders, Philadelphia, 1981
18. Beatriz Lavras C, Ferreira DM, Lavras Costallat LT, Appenzeller S: Myelopathy in systemic lupus erythematosus: Clinical, laboratory, radiological and progression findings in a cohort of 1,193 patients. Rev Bras Reumatol Engl Ed, 2016; 56(3): 240–51
19. Bertsias GK, Ioannidis JP, Aringer M et al: EULAR recommendations for the management of systemic lupus erythematosus with neuropsychiatric manifestations: Report of a task force of the EULAR standing committee for clinical affairs. Ann Rheum Dis, 2010; 69(12): 2074–82
20. Aggarwal HK, Sood S, Jain D et al: Evaluation of spectrum of peripheral neuropathy in predialysis patients with chronic kidney disease. Ren Fail, 2013; 35(10): 1323–29
21. Ruiz-Irastorza G, Egurbide MV, Olivares N et al: Vitamin D deficiency in systemic lupus erythematosus: Prevalence, predictors and clinical consequences. Rheumatology (Oxford), 2008; 47(6): 920–23
22. Bhattoa HP, Kiss E, Bettembuk P, Balogh A: Bone mineral density, biochemical markers of bone turnover, and hormonal status in men with systemic lupus erythematosus. Rheumatol Int, 2001; 21(3): 97–102
23. Lips P, van Schoor NM, Bravenboer N: Vitamin D-related disorders. In: Primer on the metabolic bone diseases and disorders of mineral metabolism, 8th ed, Rosen CJ (ed.), John Wiley & Sons, Inc., Ames, 2013
24. Boudville N, Inderjeeth C, Elder GJ, Glendenning P: Association between 25-hydroxyvitamin D, somatic muscle weakness and falls risk in end-stage renal failure. Clin Endocrinol (Oxf), 2010; 73(3): 299–304

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