

Case Report

Lupus with myositis in an adult Indian male

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ABSTRACT

Systemic lupus erythematosus (SLE) is a chronic inflammatory disease that involves various organs and has a vast spectrum of multi system involvement. SLE is an autoimmune illness which is rarely found in males but when present holds a poor prognosis with a predisposition to develop various manifestations. This case involves a 50-year-old male who developed myositis as a complication of lupus despite adequate immunosuppressive therapy.

Keywords: Lupus, Myositis, Muscle involvement

INTRODUCTION

SLE myopathy is found to be present in individuals who have elevated creatine kinase, with evidence of muscle edema on MRI, biopsy, or electromyography. It is most commonly seen in the Afro- American population.¹ Mostly the myopathy resolves by prompt treatment with immunosuppressives. The most dangerous complication of SLE remains lupus nephritis mortality. Other than lupus nephritis, the physicians should also be aware of the overlap syndromes with myositis in SLE, which have a poorer prognosis and may require aggressive therapy. This is a case report of a middle-aged Indian male who had SLE, chronic liver disease and developed painful swelling of his muscles, but improved with addition of immunosuppressives.

CASE REPORT

A 50-year-old male who is a known case of SLE with lupus nephritis, decompensated chronic liver disease with portal hypertension was in his usual state of health 15 days back when he developed bilateral painful swelling on anterolateral side of distal thigh, more on the right side, tender to touch, associated with overlying skin erythema for 1 week. He described the pain as continuous, dull-

aching sensation, increased on walking. He also complained of high-grade fever for 1 week.

He was started on intravenous antibiotic clindamycin and other supportive managements. On investigations, he was found to have anaemia; Hb 8 g/dl, TLC 4.20 thous/cumm, thrombocytopenia; platelets 110 thous/cumm. Prothrombin time- 23 seconds. INR-2.4. Activated partial thromboplastin time- 33.5 seconds. Blood urea nitrogen- 8.56 mg/dl, creatinine-0.57 mg/dl, Na⁺/K⁺ 139/3.68 mEq/L Total bilirubin-0.77 mg/dl. Direct Bilirubin-0.37mg/dl. Total protein-5.92 gm/dl. Albumin-2.86 gm/dl, SGOT/PT 28/15 IU/L, alk. phos. /GGT 106/36 IU/L.

Inflammatory marker- ESR 68 mm, normal procalcitonin-0.10ng/ml, CRP-37mg/l.

MRI thigh was done which showed myositis involving the anterior compartment muscles on the right side, predominantly the vastus lateralis and intermedius with interfacial and subcutaneous oedema. Focal area of myositis involving the distal vastus lateralis and intermedius muscles in the left thigh. No obvious drainable collection noted in the thigh on either side. MRI findings revealed bilateral avascular necrosis of the femoral heads with the right femoral head showing a larger area of involvement and subjacent edema in the femoral head and

neck. Bilateral lower limb venous doppler was done which showed no evidence of DVT. NCV /EMG was done which showed bilateral common peroneal motor axonal + demyelinating neuropathy, left>right, mild degree of bilateral carpal tunnel syndrome right>left and myopathy without active denervation.

CPK- 52IU/l, LDH-204 IU/L. Extensive myositis panel was negative. Patient was already on rituximab for lupus, additional immunosuppressive in the form of Mycophenolate mofetil 360mg thrice daily was added. Patient's condition improved and was discharged. Patient is currently doing well on follow up.



Figure 1 (A and B): Bilateral lower lateral margin of bilateral thigh swollen with right side more than left and MRI image showing myositis of vastus lateralis and intermedialis muscle.

DISCUSSION

Myositis is prolonged inflammatory state of muscles in the body. SLE is a multi-spectral disease that can involve muscles causing extensive inflammation of muscular system. Studies have displayed that SLE causing muscle disease has a prevalence of around 3% and 8% of adult patients¹⁻³ and as high as 31% in paediatric population.⁴ Pathological features on muscle biopsy include extensive atrophy of the fascicles of muscles, with inflammation of the veins, peri muscular inflammation, neurogenic muscle injury, and necrosis of the muscles.^{5,6} In a cohort study involving adult and paediatric patients with SLE, an incidence of 6.3% inflammatory myositis was documented.⁷

Inflammatory myositis with SLE induced muscular fatigability or a myopathy due to drugs like lipid lowering agents such as statins or steroids can be achieved through diagnostic modalities like biopsy of the pathogenic muscle, electromyographic studies, and increased muscle breakdown products like serum creatine phosphokinase.⁸ A sensitive and specific marker to measure the range of

muscle fascicular injury is creatinine kinase. However, in our case these values were normal.⁹

Symptoms of myositis include weakness in performing routine activities, muscular pain on movement as well as on rest, difficulty and weakness while standing or walking.¹⁰ There are three types myopathies present in patients of lupus which are polymyositis, inclusion body myositis and dermatomyositis. Muscle weakness involving axial muscles of the torso primarily, leading to difficulty in sitting up or lifting is a well-known feature of polymyositis. Inclusion body myositis is associated with a slow prolongation of muscular atrophy and fatigability. Muscle weakness of polymyositis in addition to a scaly, erythematous skin rash is hallmark feature of dermatomyositis.¹¹

Treatment of myopathy is generally challenging and involves usage of both immunosuppressives as well as cytotoxic drugs. Initial choice of drugs have always been corticosteroids but addition of other immune suppressing agents such as methotrexate, rituximab, cyclophosphamide and mycophenolate mofetil have been used with success leading to reduced relapse rates.¹²

CONCLUSION

SLE is an important cause of morbidity as encountered by family physicians. Myopathy can be induced as a part of the disease process or as an overlapping myopathic syndrome secondary to any autoimmune pathology. Prompt diagnosis with MRI scan and addition of immune regulating drugs can salvage patient from developing complications.

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