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Case report

Unusual trigger for dermatomyositis: Atorvastatin

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Introduction

Malignancies, infections and rarely drugs have been identified as the trigger for dermatomyositis which presents with proximal muscle weakness, myalgia and photosensitive skin rashes[1]. HMG-CoA reductase inhibitors or statins are frequently prescribed for primary and secondary prevention of atherosclerotic cardiovascular disease. Derangement of liver enzymes and myotoxocity is commonly encountered following treatment with statins but association with dermatomyositis is indistinct.

Case report

A 49-year-old heavy vehicle driver presented with gradual onset of difficulty in walking and facial discoloration for two months after treatment for hyperlipidaemia with atorvastatin. After one month patient perceived gradual onset of pain and weakness of all four limbs predominantly affecting the proximal muscles. Subsequently he became completely bed bound and suffered severe myalgia but swallowing was intact. Face, neck and lateral aspects of the trunk showed dark discoloration with poikiloderma. Evolving shawl sign was noted on the upper back and cuticular dystrophy was noted on nail beds. But there was no eyelid edema, heliotrope sign, nail fold telangiectasia or Gottron's sign. He has had type II diabetes mellitus for six years and a good glycemic control with metformin hydrochloride without any micro or macrovascular complications. In neurological examination, muscle power was 2, 3 and 4 out of 5 in proximal limb muscles, distal muscles and neck muscles respectively. Palatal and tongue movements were spared. Other central and peripheral nervous system examination findings were normal. Respiratory, cardiovascular and abdominal examination did not reveal any abnormality. Neither history nor examination revealed evidence of a malignancy.

Full blood count revealed neutrophil leucocytosis with normal haemoglobin and platelet count. Blood picture showed mild to moderate rouleaux formation and neutrophil toxic changes. Serum creatinine and electrolytes were within normal limits. Erythrocyte sedimentation rate was 56 mm/hour. His glycemic control was good and TSH was within normal limits. LDH was 700 U/L. Coagulation profile was normal and mild reduction of serum albumin (34 g/l) was noted. CPK was high (5578 U/L). Liver transaminases were high (AST: 1630 U/l and ALT: 2490 U/l). ALP was normal. ANA was positive in homogenous florescence pattern, with a titer of more than 1/80. Rheumatoid factor was negative. Antibodies to extractable nuclear antigen were negative. HIV 1 and 2 antibodies, syphilis serology, hepatitis B surface antigen and hepatitis C antibody were negative. Prostate specific antigen and carcinoembryonic antigen were negative.

Electromyogram (EMG) revealed asymmetrical myopathic changes, which were more pronounced in upper limbs than lower limbs. Contrast enhanced computerized tomography of chest, abdomen, pelvis, colonoscopy and upper gastro intestinal endoscopy did not reveal any abnormality. Muscle biopsy revealed focal degenerative changes with cytoplasmic swelling, intense eosinophilia, loss of striations and fragmentation of myofibrils. These foci showed infiltration by chronic inflammatory cells. In skin biopsy, epidermis showed mild hyperkeratosis and focal vacuolar changes of basal cells. The upper dermis was slightly oedematous and contained a few scattered chronic inflammatory cells. Aclain blue stain showed increased mucin in the upper dermis. Diagnosis of dermatomyositis was made based on clinical presentation, serum biochemistry, electromyography, skin and muscle biopsy findings. Atorvastatin was identified as the possible trigger for dermatomyositis. Atorvastatin was withheld and remarkable symptomatic improvement, gradual reduction of CPK and liver transaminases were observed over one week. Subsequently

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low dose steroids were commenced where complete resolution of the dermatological manifestations and muscle weakness occurred after five weeks.

Discussion

Dermatomyositis is a connective tissue disorder characterized by peri-vascular inflammatory lesions with immune complex deposition in the capillaries of the endomysium and inflammatory cell accumulation, mainly with T-CD4+ and B lymphocytes, which infiltrate myocytes leading to its vacuolization and degeneration in skeletal muscles and rarely in smooth muscles. Muscle cells are damaged by focal phagocyte infiltrations and this leads to muscle fiber loss. Besides viral infections or cancer as a trigger, clinical reports showed relationship of the disease with medications[1,2]. Atorvastatin was identified as a possible trigger of dermatomyositis in previous reports[3-5]. Statins are myotoxic and classically causes necrotizing skeletal muscle lesions. But this patient demonstrated dermatological, rheumatological and histological findings supportive for dermatomyositis. Atorvastatin therapy was identified as the trigger for dermatomyositis. Statin induced dermatomyositis is rare but potentially reversible with discontinuation of the drug and with steroids. This may be the first clinical report where dermatomyositis was triggered by atorvastatin in Sri Lanka.

Conflicts of interest

Authors declare that they have no conflicts of interest.

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