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Case Report

An Exceptionally Rare Case of Statin Induced Autoimmune Necrotizing Myopathy treated with Intravenous Immunoglobulin

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ABSTRACT

The distal triceps tendon rupture is a relatively rare injury, accounting for less than 1% of all tendon injuries. Several risk factors have been described. In this article, we present the case of a 34-year-old athlete with a complete rupture of the distal triceps tendon of his left arm that took place during the Cross-Fit national games. The patient presented reduced range of motion, swelling, a palpable gap over the olecranon and inability to extend his left elbow against gravity. Ultrasound and MRI revealed a complete rupture of the distal triceps tendon. Surgery was performed a week after the injury. The central stump of the tendon was reattached at his anatomic position, while the sutures were passed through 2 transosseous drill holes in the olecranon and stabilized with an anchor. A fast-track postoperative protocol including a supervised gradual strengthening program was followed. No complications were noticed during the follow up. The patient returned to full sport activity 4 months postoperatively.

Keywords: Triceps injury; Triceps complete rupture; Sport injuries; Transosseous technique; Knotless anchor

Introduction

Statin induced immune mediated necrotizing myopathy (SIMNM) is a rare entity characterized by progressive muscle weakness. The usual progression of weakness in SIMNM often exhibits a gradual improvement, highlighting the imperative need for early initiation of immunosuppressive therapy to mitigate the related risks of morbidity and mortality.

Case Presentation

We present a case of 74 years old male with history of hypertension, hyperlipidemia presenting with bilateral lower limb edema and intermittent chest pain. Upon arrival at the emergency department, the patient exhibited stable vitals and was not in acute distress[1]. Initial assessments revealed elevated troponin and transaminase levels, while an electrocardiogram

and echocardiogram showed no signs of ischemia[2]. Further imaging done with chest computed tomography angiogram ruled out pulmonary embolism and revealed small pericardial cyst[3]. Elevated liver function tests prompted a creatinine kinase evaluation, resulting in a diagnosis of rhabdomyolysis secondary to statin therapy, indicated by a CK level of 32,400. Throughout the 5-day hospitalization, CK levels gradually decreased from 320,400 to 17,000 with aggressive fluid management[4]. Thyroid panel was normal. Various consultations were sought, including rheumatology, renal, general surgery, and neurology, with electromyography suggesting a myopathic disorder, likely necrotizing[5]. A lower extremity muscle biopsy confirmed autoimmune necrotizing myopathy. Treatment involved IVIG administration (total of 5 doses), continuous renal follow-up to ensure adequate urine output using IV fluids and diuretics, and cessation of amlodipine due to exacerbating leg swelling. Laboratory assessments revealed elevated Anti 3-hydroxy-3-methylglutaryl-coenzyme A (HMG-CoA) antibodies, while other panels and tests including anti Jo 1, antinuclear antibodies came back negative, metabolic myositis panel all came back within normal limits. Additionally, his serum aldolase levels were elevated, making aldolase deficiency extremely unlikely. Muscle biopsy result confirmed autoimmune necrotizing myopathy. Most likely suspicion was that the combination of the new prescription of amlodipine on top of his statin increased the statins risk of myopathy/muscle injury. IVIG therapy led to a gradual improvement in the patient's condition, with CK levels initially stabilizing before slowly declining (**Figures 1 and 2**).

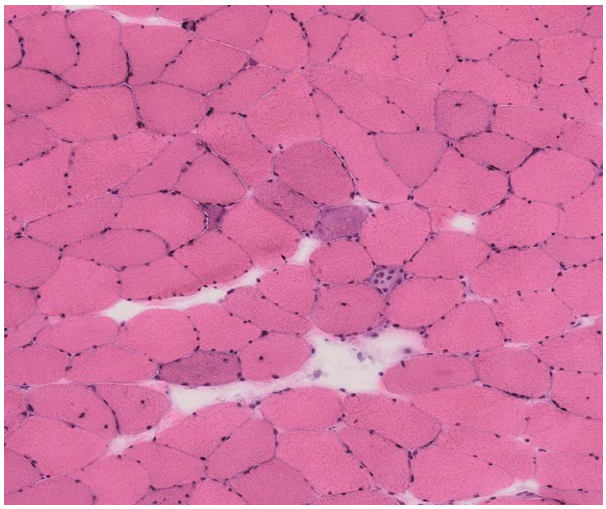

Figure 1. Frozen H&E showing scattered necrotic and regenerating fibers.

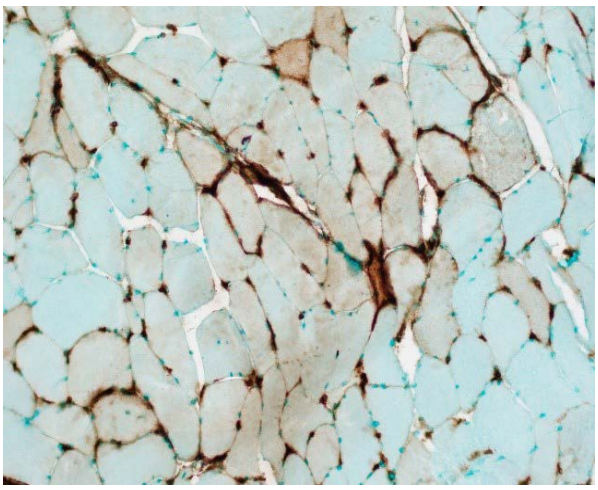

Figure 2. MHC1 IHC shows multifocal sarcolemma staining.

Discussion

Statin is one of the commonly prescribed drugs and its side effects of myopathy is well known. However, diagnosing and treating statin induced immune mediated necrotizing myopathy remains challenging. A broad suspicion is warranted whenever there is profound weakness with slow improvement in CK levels. Immune mediated myopathies are generally treated with immunosuppression and warrants sooner diagnosis to avert considerable morbidity and mortality. Our patient had significant improvement with IVIG but experienced a gradual decline in CK levels over several weeks.

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