A Rare but Reversible Cause of Myopathy: Hydroxychloroquine Induced Myopathy

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Abstract

Hydroxychloroquine is used in the treatment of various rheumatic conditions including systemic lupus erythematosus. Hydroxychloroquine induced myopathy is rare adverse reaction but symptoms are usually recover after discontinuation of the drug. Here we report a case of woman with systemic lupus erythematosus who presented with myopathy after eight months treatment with hydroxychloroquine. Her symptoms were recovered after cessation of the drug. The reported prevalence of antimalarial myopathy varies, i.e. less than 2 – 6.7%. Physicians should be aware of this cause of myopathy because hydroxychloroquine is widely used around the world and symptoms usually recover only after cessation of the drug. 

Keywords: Hydroxychloroquine; Myopathy; Systemic lupus erythematosus

Introduction

Hydroxychloroquine is an antimalarial drug that is also used in the treatment of various rheumatic diseases including systemic lupus erythematosus (SLE). Various adverse reactions include skin rash, diarrhea, nausea and retinopathy were reported [1]. Hydroxychloroquine induced myopathy is a rare side effect and reported prevalence is up to 6.7% [2]. Involvements with respiratory musculature or myocardium were reported [3–5], but myopathy symptoms usually improve only after discontinuation of hydroxychloroquine [2]. Here, we report a case of hydroxychloroquine induced myopathy recovered after cessation of the drug.

Case Presentation

A 59-year-old Japanese woman presented with severe myalgia and weakness of both arms and legs. Seven years ago, the patient was referred to our hospital with malar rash, photosensitivity, arthritis and liver damage. The laboratory examinations revealed positive result of anti-nuclear antibody (1:40, speckled and cytoplasmic pattern), elevated anti-DNA antibody (13 IU/ml, normal < 6.0 Radioimmunoassay), hypocomplementemia (C3 83mg/dL, C4 6mg/dL) and pancytopenia. The patient was diagnosed as SLE based on the American College of Rheumatology criteria [6]. The treatment was commenced with prednisolone and tacrolimus and subsequently mizoribine was added. We changed these immunosuppressants to mycophenolate mofetil because elevated liver enzymes and hypocomplementemia didn’t improve. She had been treated with 5 mg of prednisolone per day and 1500 mg of mycophenolate mofetil per day for a year before myalgia and weakness developed. She was also prescribed 300 mg of hydroxychloroquine per day (200 mg–400 mg alternatively on every other day) for eight months. On physical examination, severe myalgia and weakness of both arms and legs were noted. The neurological examination showed no evidence suggesting peripheral neuropathy. The C-reactive protein level elevated (0.52 mg/dL, normal < 0.3), creatine kinase (CK) was 437 IU/L (normal < 200), anti-DNA antibody was positive (8.3 IU/mL, normal < 6.0), anti-SS-A/Ro antibody was positive (> 240 U/ml, normal < 7.90) and other autoantibodies including myositis-specific antibodies (anti-ARS antibody, anti-Mi-2 antibody, anti-MDA-5 antibody, anti-TIF1-γ antibody) were all negative. MRI of both thighs revealed inflammation of both gluteal muscles and femoral muscles (Figure 1). She rejected electroneuromyography and muscle biopsy. The dose of prednisolone was stable for a year and the patient wasn’t prescribed myotoxic drugs including statins before myopathy developed. The laboratory data including complement and anti-DNA antibody didn’t change before myopathy developed, and temporary increase of prednisolone dose (5 – 20 mg for 2 weeks) didn’t improve symptoms and CKP level. Therefore we suspected hydroxychloroquine induced myopathy rather than glucocorticoid-induced myopathy or myopathy associated to SLE itself. There were no findings suggesting respiratory failure or cardiac involvement. After discontinuation of hydroxychloroquine, her symptoms were gradually relieved. After seven months, CK level returned to normal range and MRI findings improved completely (Figure 2). 

Discussion

Hydroxychloroquine is used in the treatment of various rheumatic diseases including SLE. Hydroxychloroquine induced myopathy is thought to be due to accumulation of hydroxychloroquine in lysosomes [7]. The duration of antimalarial therapy varies widely from less than one year to more than ten years [7]. Muscle enzymes including CK may be normal or mildly elevated. It was reported that there is no correlation between abnormal muscle enzyme levels and the presence of either muscle symptoms or abnormal neuromuscular examination results [8].

Figure 1: MRI of both thighs showing inflammation of gluteus maximus, gluteus medius, iliacus, quadriceps femoris and hamstring of both sides.
The reported prevalence of antimalarial myopathy varies from less than 2 – 6.7% [2,9]. Muscle symptom usually recovers within months after discontinuation of the antimalarial drugs [2]. Hydroxychloroquine induced myopathy is thought to be underestimated because of slightly elevated to normal CK levels [10]. Physicians should be aware of this cause of myopathy because hydroxychloroquine is widely used around the world and symptoms usually recover only after cessation of the drug.

Conflict of Interest

All the authors receive no financial supports and have no conflicts of interest.

Ethical Consideration

Ethics committee approval was not required in accordance with the policy of our institution.

References