Myositis developing after Covid-19 mRNA vaccine: Case Report

Bünyamin Tosunoğlu, Hafize Nalan Güneş, Burcu Gökçe Çokal, Tahir Kurtuluş Yoldaş

Abstract

Vaccine-related side effects are common. Usually, pain, edema, redness and tenderness may be seen at the injection site. Symptoms such as fever, fatigue, myalgia may occur. The coronavirus 2019 disease (Covid-19) has affected many people around the world. Although the vaccines that have been used play an active role in the fight against the pandemic, adverse events still continue to be reported.

We present a 21-year-old patient who was diagnosed as having myositis after receiving covid vaccine with complaints of pain in her left arm two days after the 2nd dose of BNT162b2 mRNA Covid-19 vaccine, followed by inability to stand up from sitting and squatting and difficulty in going up and down stairs.

Keywords: vaccine, myositis, creatine kinase, IVIG

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INTRODUCTION

Vaccine-related side effects are common. Generally, pain, edema, redness and tenderness may be seen at the injection site. Symptoms such as fever, fatigue, myalgia may occur. The coronavirus 2019 disease (Covid-19) has affected many people around the world [1,2] Although the vaccines that have been used play an active role in the fight against the pandemic, adverse events still continue to be reported. Myositis can occur due to trauma, drugs (especially statins), viral infection, vaccines, and autoimmunity. It has been reported that Covid-19 infection causes various skeletal muscle complications ranging from asymptomatic creatine kinase (CK) elevation, myalgia, myositis, and rhabdomyolysis [3,4], Various mechanisms have been postulated, including direct viral invasion of muscle cells or hyperinflammation syndrome [5-7]. Covid-19 myositis may therefore result from accumulation of virus-antibody complexes in myocytes or from virus-induced expression of muscle antigen in the cell membrane and damage by cytokine storm. It can also trigger autoimmunity. Myositis may occur after Covid-19 vaccination, possibly due to the same mechanisms [1,2,4].

CASE REPORT

A 21-year-old right-handed female patient complained of pain in her left arm especially during lifting her arm about 2 days after receiving the covid-19 mRNA BNT162b2 vaccine, but the patient did not apply to any health institution, thinking that these complaints would go away, and she used nonsteroidal anti-inflammatory and muscle relaxants. Despite the fact that severity of pain did not decrease, she applied to our neurology outpatient clinic

From the Department of Neurology, Ankara Training and Research Hospital, Ankara, Turkey
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Correspondence to: Bünyamin Tosunoğlu

after receiving vaccine for twenty days with complaints of difficulty in sitting and getting up, pain in her legs, inability to get up from crouching, and difficulty climbing stairs. She had no known major disease or using regular medication. There was no history of trauma, febrile illness, similar complaints in her family, and substance use. The patient was admitted to our neurology service for further examination and treatment. In her physical examination, her blood pressure was 120/70 mmHg, heart rate was 80/min, temperature was 36.5, and oxygen saturation was 98% in room air. Electrocardiography (ECG) was in normal sinus rhythm. In the neurological examination of the patient muscle strength was 3/5 in bilateral proximal legs and full in both upper limbs using the Medical Research Council Scale. Deep tendon reflexes were normoactive. No pathology was detected in the brain computerized tomography (CT)

and brain magnetic resonance imaging (MRI). Initial blood test showed high levels of creatine kinase (CK, 7793 U/l), alanine aminotransferase (ALT, 129 U/l), and aspartate aminotransferase (AST, 134 U/l).

The results of urinalysis were normal without myoglobinuria. The Covid polymerase chain reaction (PCR) test was negative. In the first electromyography (EMG), it was consistent with myopathic involvement (Shortening of motor unit times accompanied by denervation, decrease in amplitude) accompanied by dominant active denervation findings in the proximal lower extremities.

In laboratory tests, the results of complete blood count, platelet function tests, coagulation tests, routine biochemistry tests, complete urinalysis, protein electrophoresis, antinuclear antibody, vitamin B12, thyroid function tests, HbA1c, erythrocyte sedimentation rate, autoantibody screening (anti-SSA, anti- SSB), antithyroid antibodies, syphilis serology (fluorescent treponemal antibody), Schirmer test, homocysteine, anticardiolipin, rheumatoid factor, Anti-streptolysin O, rheumatoid factor, immunoglobin A,G,M were normal. Elisa tests (Hepatitis A, HIV, hepatitis B, hepatitis C) were negative. When viral meningitis agents (Herpes simplex virus 1-2, varicella zoster virus, enterovirus, parechovirus, ebstein barr virus, cytomegalovirus, adenovirus) were investigated, the agent was not found. Brucella tests were negative. The pathergy test for Behçet was negative. No pathological finding was detected in chest X-ray and abdominal ultrasonography. No significant pathology was found in the muscle biopsy performed from the patient's left biceps brachi muscle. MRI study of both thighs showed abnormal hyperintensity with contrast enhancement especially at the anterior muscle groups, favoring myositis with edematous change. (Figure 1).

The patient's complaints regressed. Follow-up EMG still revealed features of myopathy but active denervating changes disappeared. After discharge, she received 64 mg/day of oral methylprednisolone with stepwise tapering of dosage and her symptoms improved and abnormal CK level reduced gradually.

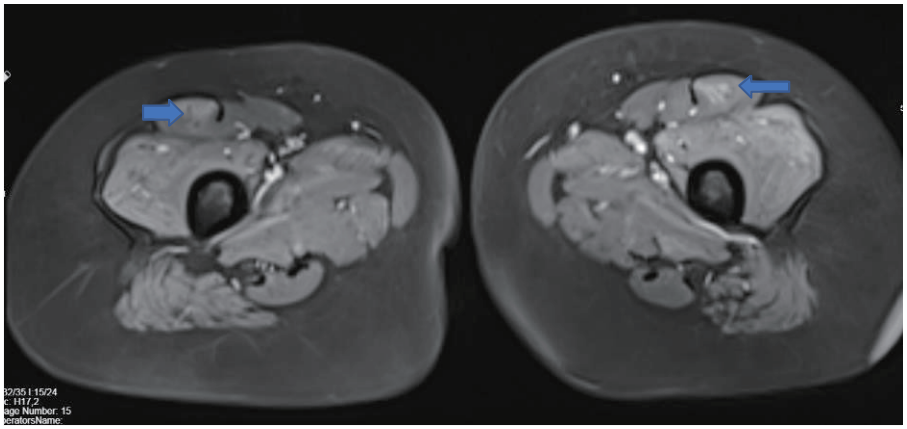

Figure 1. Abnormal contrast enhancement (arrows) especially in anterior muscle groups of both thighs.

Hydration with intravenous saline for 10 days and oral 64 mg/day methylprednisolone treatment was applied to the patient. Although CK tended to decrease, the patient's complaints did not regress. The patient was treated with intravenous immunoglobulin (IVIG) for 5 days.

Discussion: Acute myositis usually presented with proximal muscle weakness and the causes of myositis include adverse effect of specific drugs and vaccines, autoimmunity, viral infection, and trauma [1,2,5]. Our patient developed weakness of bilateral proximal legs while EMG and thigh MRI showed evidence of myositis. There was no obvious etiology, which can explain the occurrence of acute myopathy except injection of Covid-19 vaccination about 2-3 weeks earlier. It is pity that we can only obtained a muscle sample for biopsy from biceps, which did not show obvious infiltration of inflammatory cells. Under a diagnosis of vaccination-related acute myositis, oral methylprednisolone followed by IVIG were given with gradual improvement in clinical manifestations and elevation of serum CK levels. It has been reported that Covid-19 infection itself causes many skeletal muscle complications, and it has been reported in the literature that its mechanism includes direct viral invasion of muscle cells or hyperinflammation syndrome. It may result from accumulation of virus-antibody complexes in myocytes or from virus-induced cell membrane expression of muscle antigen and damage by cytokine storm. It was thought that myositis may occur due to the same mechanisms after Covid-19 vaccination. However, the mechanism of post-vaccine-induced inflammatory myositis is not fully known [3,5-7].

In summary, vaccination-related myositis should be taken into consideration in patients with acute myopathy in the Covid-19 pandemic era. In addition, for patients who have received BNT162b2 vaccination, a potential complication of acute myositis should be noticed.

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