

Heart attack or rhabdomyolysis?

Mustafa Bolatkale¹, Muhittin İşsever¹, Ulaş Karaoğlu¹, Ahmet Cagdas Acara^{2*}, Mehtap Bulut¹

¹Department of Emergency Medicine, Medipol University Hospital, İstanbul, Turkey

²Department of Emergency Medicine, Gaziemir State Hospital, Izmir, Turkey



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***Corresponding author:**

Ahmet Cagdas Acara,
Gaziemir State Hospital, Izmir, Turkey.
Email: cagdasacara@gmail.com

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Abstract

Statins are commonly used drugs in the treatment of hyperlipidemia (HL), despite some undesirable side effects. These range from mild symptoms such as myopathy, muscle weakness and myalgia to severe muscle weakness associated with chronic myopathy and acute renal failure (ARF) as a result of rhabdomyolysis. The most serious and deadly side effect of statins is rhabdomyolysis. The case presented here is of a patient with rhabdomyolysis due to treatment with the antihyperlipidemic drug, atorvastatin.

Keywords: Rhabdomyolysis, Atorvastatin, Heart attack, Statin

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Introduction

Rhabdomyolysis is a clinical condition resulting from skeletal muscle damage due to traumatic or non-traumatic causes. The most common traumatic causes are natural disasters, traffic accidents and occupational accidents. By the same token the most common non-traumatic causes are medications, toxins, infections and electrolyte disorders. Statins, fibric acid derivatives, corticosteroids, colchicine, amphotericin B and many other drugs can cause rhabdomyolysis (1).

Case Presentation

A 53-year-old male patient was admitted to the emergency department with a 3-day history of chest pain, abdominal pain and widespread body pain. The patient described pressure and compressive-type chest pain. He had a medical history of hypertension, coronary artery disease, hyperlipidemia (HL), and prior myocardial infarction. The patient used 40 mg atorvastatin for HL and a self-initiated additional dose of atorvastatin 20 mg tablets without the advice of a physician.

The patient's vital signs were normal. Physical examination revealed tenderness to palpation of the entire body. Tenderness with palpation of the upper right quadrant was present during the abdominal examination. Electrocardiogram (ECG) demonstrated a normal sinus rhythm. The laboratory results were white blood cell (WBC): 13.49 μ /L, hemoglobin: 12.8 g/dL, CK-MB: 17 IU/L, troponin I: 0.082 IU/L, blood urea nitrogen (BUN): 47.12 mg/dL, creatinine: 2.18 mg/dL, alanine

transaminase (ALT): 1056.9 U/L, aspartate transaminase (AST): 2733.5 U/L, K: 5.73 mmol/L, creatinine kinase (CK): 351.000 U/L, lactate dehydrogenase (LDH): 4307 U/L. Venous blood gas analysis showed pH: 7.29, pCO₂: 43.3 mm Hg and HCO₃: 18.2, base deficit (BE): -4.6 mmol/L. In the complete urinalysis; yellow color was seen and pH: 5, density: 1010, ketones (-), protein (-), glucose (-) and hemoglobin (++) and urine microscopy showed 14 RBCs/HPF and 3 leukocytes/HPF.

The patient with chest pain and cardiac enzyme elevation was referred to the cardiology department and a pre-diagnosis of acute coronary syndrome (ACS) was made. Serial ECG monitoring was in normal sinus rhythm. Bedside echocardiography was performed and no wall motion abnormality was detected. The patient was not thought to have ACS by the consultant cardiologist. There were no pathological findings on abdominal ultrasound. The patient was referred to internal medicine, and was diagnosed with rhabdomyolysis associated with the use of statin and was admitted to the internal medicine service. In the 24-hour urine analysis microalbumin and microprotein were measured at 21 and 143 mg/d respectively. Hepatitis and HIV serology were normal. Creatinine clearance by age was calculated as 44.34 mL/min. The patient was considered at risk (R) stage according to the RIFLE (Risk, Injury, Failure, Loss of function, and End-stage renal disease) criteria and atorvastatin-induced rhabdomyolysis and ARF associated with rhabdomyolysis were diagnosed. Atorvastatin treatment was stopped. Adequate fluid replacement was administered. During



hospitalization, the need for hemodialysis arose and dialysis was applied 4 times. The follow-up laboratory test results were BUN: 31 mg/dL, serum creatinine: 1.2 mg/dL, AST: 19 U/L, ALT: 38 U/L, CK: 284 U/L, Na: 141 mmol/L, K 3.4 mmol/L, P 3.4 mg/dL. Polyuria developed but decreased during the follow-up and renal function tests returned to normal levels. When an improvement was determined in the general symptoms and laboratory findings, the patient was discharged with a treatment plan and an outpatient clinic follow-up examination for 10 days later.

Discussion

Statins are widely used in the treatment of HL and they are usually safe drugs. However, findings ranging from non-specific symptoms such as myalgia and muscle weakness to rhabdomyolysis-induced ARF can be seen in patients taking statins. This situation is associated with elevation of CK. Clinical symptoms and serum CK levels improve quickly after discontinuation of the drug. The risk of developing statin-associated myalgia has been reported as 2%-11%, risk of myositis at 0.5%, and the risk of rhabdomyolysis at approximately 0.1% (2).

Rhabdomyolysis is defined as a severe myopathy with muscle symptoms and elevation of CK more than ten times the upper limit of normal (3). Clinical signs of rhabdomyolysis include pain in the muscles, tenderness, skin discoloration in the affected regions, pain mostly in the waist and thigh, fatigue, tachycardia, nausea and vomiting, fever, and palpitations (4). The most common presentation of rhabdomyolysis is muscle weakness, pain, cramps and edema with tea colored (red-brown) urine (5). ARF occurs in approximately 30% of rhabdomyolysis cases. Rhabdomyolysis is the cause in 8% of all cases of ARF. Rhabdomyolysis-induced ARF is caused by hypovolemia related to rhabdomyolysis or the direct toxic effect of myoglobin if there is no underlying cause (6).

In the current case, the patient had been taking additional treatment without medical advice and statin-induced rhabdomyolysis and ARF developed.

In conclusion, statins are in current widespread use and because of the potential fatal side effects, these

medications should be chosen carefully. These drugs must be used for the correct indications and at the right dose and there should be regular monitoring of kidney and liver functions and muscle enzymes. Patients must be evaluated for rhabdomyolysis facilitating factors, and they should be informed about the side effects and symptoms of myopathy.

Ethical issues

The study adhered strictly to the principles of the Declaration of Helsinki as revised in 2008.

Authors' contributions

All authors have equal contribution to write this article.

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