

Opium-Induced Rhabdomyolysis and Acute Renal Failure in a Patient Taking Opium Habitually: a Case Report

Opium Kullanma Alışkanlığı Olan Bir Hastada Opiumun Neden Olduğu Rabdomyoliz ve Akut Böbrek Yetmezliği: Olgu Sunumu

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CASE REPORT
OLGU SUNUMU

ABSTRACT ÖZET

A 60-year-old male hypertensive and diabetic patient who took opium habitually for six months was sent to our hospital from a private hospital because of muscle weakness, rhabdomyolysis and acute renal failure. The laboratory tests revealed high serum creatine kinase, creatinine, myoglobin and lactate dehydrogenase. Intravenous hydration, bicarbonate and mannitol treatment were applied. During the follow-up period, the serum creatine kinase level and renal function tests gradually normalized. Although acute opiate drug intoxication can cause rhabdomyolysis, one of the causes of rhabdomyolysis is taking opium habitually. Here, we report a patient who presented with rhabdomyolysis and acute renal failure while using opium regularly. Physicians should keep in mind that habitual opium use can cause rhabdomyolysis and associated acute renal failure.

Son 6 aydır sürekli afyon kullanan ve kas güçsüzlüğü şikâyeti olan 60 yaşındaki hipertansif ve diyabetik erkek hasta, rabdomyoliz ve akut böbrek yetmezliği nedeniyle hastanemize gönderildi. Laboratuvar incelemesinde serum kreatinin kinaz, kreatinin ve laktat dehidrogenaz enzim yüksekliği saptandı. İntravenöz hidrasyon, bikarbonat ve mannitol tedavisi uygulandı. Takipler esnasında serum kreatin kinaz düzeyleri ve böbrek fonksiyon testlerinde gerileme oldu. Opiyat içeren ilaçların akut zehirlenmeleri rabdomyolize neden olabilirken; sürekli afyon kullanımı da rabdomyoliz nedenlerinden biridir. Bu yazıda afyon kullanımı sırasında rabdomyoliz ve akut böbrek yetmezliği gelişen bir olgu sunuluyor ve sürekli afyon kullanımının rabdomyoliz ve onunla ilişkili akut böbrek yetmezliğine neden olabileceği vurgulanıyor.

Key words: Acute kidney injury, opium, rhabdomyolysis

Anahtar kelimeler: Akut böbrek hasarı, opiyum, rabdomyoliz

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Introduction

Rhabdomyolysis is a biochemical and clinical syndrome resulting from skeletal muscle injury and the release of muscle cell components into the extracellular compartment (1). The presenting clinical features are myalgia, myoglobinuria and elevated serum creatine kinase (2). Myoglobinuria is the most significant consequence, leading to acute renal failure (ARF) in 15%-33% of patients with rhabdomyolysis. Rhabdomyolysis results from inherited muscle enzyme deficiencies, toxins such as alcohol, opiates and cocaine, trauma, medications such as fibrates and statins, muscle overexertion, infections, hypothyroidism and other disorders (3-5). Rhabdomyolysis has been reported in 22 of 188 consecutive patients with acute opiate intoxication (6). On the other hand, one of the causes of rhabdomyolysis among acute human poisoning cases has been opium (7). Here, we report a patient who presented with rhabdomyolysis and acute renal failure while taking opium resin habitually.

Case Report

A 60-year-old unconscious male patient with diabetes mellitus and hypertension had been admitted to a private hospital. A cerebrovascular event had been excluded by computerised tomography and Doppler ultrasonography. He had used aspirin (300 mg/day), metformin (2000 mg/day), pioglitazone (30 mg/day), metoprolol (100 mg/day) and amlodipine (10 mg/day). He had been taking opium resin habitually for a period of six months. The patient was sent to our hospital because of rhabdomyolysis and acute renal failure.

The physical examination was completely normal except for muscle weakness. Systolic/diastolic blood pressure was 145/90 mmHg. Routine laboratory tests revealed a creatine kinase (CK) level of 10796 U/L (normal range: 24-195 U/L), blood urea nitrogen (BUN) 140 mg/dL (normal range: 7-18 mg/dL), creatinine 6.3 mg/dL (normal range: 0.7-1.3 mg/dL), myoglobin 643 ng/mL (normal: 17.4-105.7 ng/mL), fasting blood glucose 190 mg/dL (normal range: 65-100 mg/dL), albumin 4 g/dL (normal range: 3.5-5 g/dL), sodium 150 mmol/L (normal range: 136-145 mmol/L), potassium 4.0 mmol/L (normal range: 3.5-5.1 mmol/L), calcium 9.0 mg/dL (normal range: 8.2-10.9 mg/dL), phosphorus 4.6 mg/dL (normal range: 2.7-4.5 mg/dL), aspartate aminotransferase (AST) 401 U/L (normal range: 5.0-45 U/L), alanine aminotransferase (ALT) 490 U/L (normal range: 5.0-5 U/L), lactate dehydrogenase (LDH) 670 U/L (normal range: 100-210 U/L), haemoglobin 16.8 g/dL, white blood cell count 8500/mm³ and platelet count 157000/mm³. The erythrocyte sedimentation rate was 15 mm/h.

Urine pH was 5.6, urine density was 1025. A urinary dipstick test was positive for protein (+), erythrocyte (++) and negative for glucose. Microscopic examination of urine showed a total of 8 erythrocytes high power field. Fractional excretion of Na (FENa) was calculated at 1.3%. The serum concentration of thyroid stimulating hormone (TSH) was 1.2 uIU/mL (normal range: 0.34-5.6 uIU/mL), free T3 3.1 pg/mL (normal range: 2.5-3.9 pg/mL) and free T4 0.74 ng/dL (normal range: 0.54-1.12 ng/dL). The urine myoglobin level was not investigated due to the unavailability of the test in the laboratory.

The patient was diagnosed with rhabdomyolysis and acute renal failure. There was no medication, disease or condition (except for habitually taking opium) that was suspected of causing rhabdomyolysis. The patient was hospitalised for three days and given intravenous hydration, bicarbonate and mannitol for the treatment of rhabdomyolysis and acute renal failure. Kidney size, parenchymal echogenicity and the pelvicaliceal system were normal by ultrasonography. Urine output was 3000 mL/day. Arterial blood pH was 7.31; pCO₂ was 38 mmHg and bicarbonate was 21 mmol/L during the follow-up period. Serum CK, BUN and creatinine levels gradually decreased to 180 U/L, 36 mg/dL and 3.1 mg/dL, respectively, after three days.

Discussion

Rhabdomyolysis is a biochemical and clinical syndrome resulting from skeletal muscle injury and the release of muscle cell components into the extracellular compartment. The presenting clinical features are myalgia, myoglobinuria and elevated serum creatine kinase. Creatine kinase is the most sensitive enzyme marker of muscle injury. The serum myoglobin level and/or the presence of myoglobinuria are neither practical nor accurate criteria for the diagnosis of rhabdomyolysis (7). Myalgia and high levels of serum creatine kinase and myoglobin were observed although the urine myoglobin level was not investigated in the presented case. Fulminant rhabdomyolysis may be associated with tubular necrosis, acute renal failure, excessive hyperkalaemia and hypocalcaemia, which may induce further life-threatening complications. Irrespective of the cause of rhabdomyolysis, the mortality rate may be as high as 8% (8). Therefore, early diagnosis of rhabdomyolysis is the most important for prevention of its potentially life-threatening sequelae such as acute renal failure, electrolyte imbalance and shock.

A transient elevation of serum creatinine disproportionate to the elevation of blood urea nitrogen (BUN) is common in early acute rhabdomyolysis. Presumably, this results from the release of creatine from injured muscle, which is spontaneously dehydrated to creatinine. The usual ratio of urea nitrogen to creatinine in serum is 10:1. Ratios of 5 or less shortly after onset suggest acute rhabdomyolysis. In oliguric patients, a urine sodium concentration above 20 mEq/L suggests tubular injury. However, the urine sodium concentration may be low in cases of myoglobinuria and, accordingly, this finding in pigment nephropathy may be less helpful than in other oliguric settings. Hyperkalaemia is often observed as a consequence of the release of potassium from damaged muscle cells. Profound hypocalcaemia, with serum calcium values below 3.0 mg/dL, may result from hyperphosphataemia and trapping of calcium in injured muscle. Hypercalcaemia may occur later, especially

during the diuretic phase of acute renal failure. Usually, this is seen in patients who have been given calcium salts earlier in the illness.

Rhabdomyolysis results from inherited muscle enzyme deficiencies, toxins such as alcohol and opiates, trauma, medication such as fibrates and statins, muscle overexertion, infections, hypothyroidism and other disorders (3-5). Alcohol abuse, trauma, the use of fibrates, statins, antipsychotics, selective serotonin reuptake inhibitors, or lithium, infection and hypothyroidism were excluded in our patient.

Opiate intoxication can cause coma, respiratory and cardiac arrest, aspiration of gastric contents, cardiac dysrhythmia, myocarditis, pulmonary oedema, convulsions, left ventricular dysfunction, lesions of the peripheral nervous system, acute and chronic nephropathies, electrolyte abnormalities, disseminated intravascular coagulopathy and mortality. Rhabdomyolysis has been reported in 22 of 188 consecutive patients presenting with acute opiate intoxication (6). Although acute opiate drug intoxication can be a cause of rhabdomyolysis, one of the causes of rhabdomyolysis is taking opium habitually (7). Here, we report a patient who presented with rhabdomyolysis and acute renal failure while using opium. He had been taking opium resin habitually for 6 months. We speculate that rhabdomyolysis may have occurred because he either used opium at varying doses or in combination with metformin/pioglitazone.

Therapy for drug-induced rhabdomyolysis consists of supportive and specific measures. The mainstay of treatment is hospitalisation with aggressive intravenous fluid resuscitation with the correction/prevention of dehydration and electrolyte abnormalities. Alkalinisation of the urine with sodium bicarbonate, diuretic therapy or a combination of both may protect the kidneys from acidosis and the precipitation of myoglobin in the tubules. Especially in patients with acute renal failure, haemodialysis is necessary (3, 8). Intravenous hydration and mannitol treatment were applied to our patient. Serum CK, BUN and creatinine levels gradually decreased within three days. Haemodialysis treatment was not required.

Conclusion

Especially in countries in which opium is commonly used, physicians should keep in mind that habitual opium use can be cause rhabdomyolysis and associated acute renal failure, especially in patients with diabetes mellitus and hypertension.

Authors' contributions

Conceived and designed the study: FS, MS, RC, AJG, AO, EU. Examination and follow-up of the patient: FS, MS, RC, AJG, AO, EU. Analyzed the data: FS, MS, RC, AJG, AO, EU. Wrote the paper: FS, RC. All authors read and approved the final manuscript.

Conflict of interest

No conflicts of interest were declared by the authors.

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