


A 23-year-old Healthy Male with Syncope after Playing Futsal: A Case of Exertional Rhabdomyolysis

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ABSTRACT

Introduction: Exertional rhabdomyolysis (ER) is a medical condition resulting from strenuous exercise characterized by skeletal muscle damage followed by the release of intracellular components into the circulation. ER has various clinical manifestations and is associated with various complications that increase morbidity and mortality. **Case:** A 23-year-old male came to the emergency department with decreased consciousness after playing futsal. After urinary catheter insertion, brown-colored urine was observed. Laboratory examination showed increased serum CK level (199,011 IU/L), increased serum creatinine, hyperkalemia, and metabolic acidosis. The diagnosis was exertional rhabdomyolysis, acute kidney injury, hyperkalemia, and metabolic acidosis. **Conclusion:** The condition of the patient improved after aggressive intravenous rehydration, intravenous insulin, and sodium bicarbonate therapy. Prompt diagnosis and appropriate management can reduce complications, morbidity and mortality.

Keywords: Exertional rhabdomyolysis, futsal, syncope.

ABSTRAK

Pendahuluan: Rabdomiolisis eksersional (RE) merupakan kondisi medis akibat olahraga berat yang ditandai oleh kerusakan otot rangka diikuti dengan terlepasnya komponen intraseluler ke sirkulasi. RE memiliki gejala klinis yang bervariasi dan dapat menimbulkan berbagai komplikasi yang meningkatkan morbiditas dan mortalitas. **Kasus:** Pria berusia 23 tahun datang ke UGD dengan penurunan kesadaran setelah bermain futsal. Setelah pemasangan kateter urin, ditemukan urin berwarna coklat. Pemeriksaan laboratorium menunjukkan peningkatan kadar *creatine kinase* (CK) serum (199,011 IU/L), peningkatan kreatinin serum, hiperkalemia, dan asidosis metabolik. Pasien didiagnosis rabdomiolisis eksersional, gangguan ginjal akut, hiperkalemia, dan asidosis metabolik. **Simpulan:** Kondisi pasien dan fungsi ginjal pasien membaik setelah terapi rehidrasi intravena agresif, insulin intravena, dan natrium bikarbonat. Diagnosis yang cepat dan penatalaksanaan yang tepat dapat mengurangi komplikasi, morbiditas dan mortalitas. **David Kristianus, Jonkie Budi Tirtadjaja. Pria Sehat 23 Tahun Dengan Sinkop setelah Bermain Futsal: Kasus Rabdomiolisis Eksersional.**

Kata Kunci: Rabdomiolisis eksersional, futsal, sinkop.

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INTRODUCTION

Rhabdomyolysis is a syndrome in which intracellular muscle components are released into the circulation as a result of damage and necrosis of skeletal muscle tissue.[1] The release of intracellular components in the form of myoglobin, enzymes and electrolytes causes systemic complications and causes various clinical manifestations ranging from asymptomatic increases in bloodstream muscle enzymes to life-threatening conditions such as acute kidney injury.[2] Rhabdomyolysis can be caused by various conditions such as direct myocyte damage due to severe traumatic injury or caused by disorders of myocyte metabolism in the form of an imbalance between ATP supply and demand due to strenuous activity/exercise, certain drugs, infections, and genetic disorders in myocytes or myopathy.[2]

Recently, the incidence of exertional rhabdomyolysis (ER) has shown an increase in the healthy general population due to the increasing awareness about healthy lifestyles such as exercise, but the exact incidence is still unknown.[1,3] Exercises that are heavy/excessive, prolonged, unaccustomed, repetitive, and involving eccentric contractions may cause ER.[4] Systemic complications that occur in rhabdomyolysis, especially acute kidney injury, are associated with morbidity and mortality, so preventing ER is important.[2,4]

This article presents a case of rhabdomyolysis following vigorous exercise with an atypical initial manifestation of syncope.

CASE

A 23 year-old male was brought to the emergency room with a sudden loss of consciousness while playing futsal. No history of fever, headache, weakness, paralysis, and loss of appetite. The patient was known to be in good health, no history of diabetes mellitus

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or other diseases. The patient was stuporous (E2M4V2). Blood pressure 110/80 mmHg, pulse rate 120x/minute, body temperature 40.7oC, respiratory rate 24x/minute. Physical examination of the head, neck, chest, abdomen, and extremities revealed no abnormalities. The heart rhythm showed sinus tachycardia. Approximately 300 mL of brown, tea-colored urine was observed after urinary catheterization **(Figure)**.

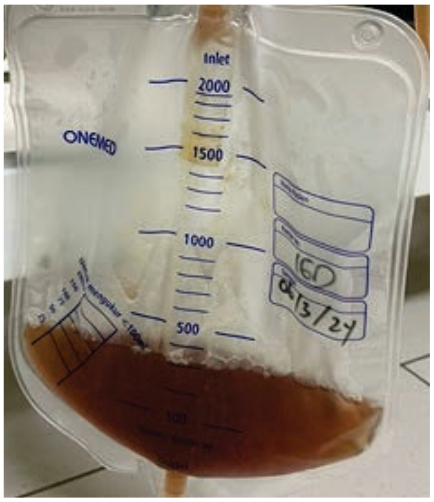

Figure. 300 mL of initial brown-colored urine.

Chest x-ray and head CT scan did not show any abnormalities.Abnormal renal function, increased blood potassium level, metabolic acidosis, and hematuria were found in laboratory examination; serum creatine kinase (CK) was elevated **(Table).**

The patient was diagnosed with exertional rhabdomyolysis, acute kidney injury, hyperkalemia, and metabolic acidosis. The patient was treated in the intensive care unit. He was given aggressive intravenous rehydration therapy with NaCl 0.9%, paracetamol 1 gram IV, calcium gluconate 3x1 gram IV, insulin aspart 10 units IV + dextrose 40% 50 mL IV for hyperkalemia correction, oral calcium polystyrene sulphonate 3x5 grams, and oral sodium bicarbonate 3x500 mg. Urine output was adequate at a rate of 100 mL/hr (1.67 mL/kg/hour, with a target of 1-3 mL/kg/hour).

His consciousness improved and became alert after 1 day of hospitalization. He complained muscle pain throughout the body and weakness. He said he was playing futsal for 3 consecutive days for a long duration and did not drink enough before being hospitalized. He usually plays futsal only 1-2 times a month. He had experienced brown urine and muscle pain throughout the body after strenuous exercise. On the 3rd day of hospitalization, kidney function and blood potassium levels improved. Muscle pain and weakness subsided after 5 days. He was discharged and advised to avoid strenuous exercise.

DISCUSSION

Disturbances in cellular metabolism in ATP production or direct injury to skeletal muscle cause rhabdomyolysis.[2] In high-intensity and prolonged exercise, continuous muscle contractions cause ATP deficiency.[5] ATP deficiency disrupts the function of the Na+/K+-ATPase ion pump resulting in an accumulation of intracellular Na+ ions which causes an increase in intracellular Ca2+ ions through the Na+/Ca2+-exchanger.[6] Intracellular calcium ions will activate protease and phospholipase A2 enzymes, causing cell membrane damage and myocyte necrosis which triggers an inflammatory response.[5-7] Intracellular proteins (myoglobin, CK, lactate dehydrogenase (LDH), aspartate aminotransferase (AST), aldolase, and nucleic acids) and intracellular metabolites (potassium, urate, phosphate) will be released into the circulation, causing exertional rhabdomyolysis (ER).[6]

ER mainly occurs in sports with high intensity and long duration, involving eccentric contractions and excessive muscle activity.[1,5,7] Rhabdomyolysis has been observed in several high-intensity and prolonged sports such as football, marathons, triathlons, bodybuilding, and CrossFit.[5] ER also occurs more often in individuals with less physical fitness or those who are less trained/experienced with the sport.[5,7] Other factors such as hot temperatures, high humidity, dehydration, male gender, infections (influenza, Epstein-Barr virus, *S.aureus, S.pyogenes*), genetic (enzyme deficiency in energy metabolism in muscles), consumption of certain drugs/substances (terbinafine, statins, alcohol, cocaine, heroin), and smoking increase the risk of rhabdomyolysis.[2,5,7] Alharbi (2023) reported

Table. Laboratory examination results during hospitalization.

	Day 1	Day 3	Normal Values
Hemoglobin (g/dL)	16.4	-	13.2 - 17.3
Hematocrite (%)	48	-	40 - 52
Leukocyte (10^3/µL)	**20.7**	-	3.8 - 10.6
Thrombocyte (10^3/µL)	446	-	150 - 450
Random blood glucose (mg/dL)	149	-	70 - 200
Ureum (mg/dL)	32	48	19 - 49
Creatinine (mg/dL)	**1.84**	**1.47**	0.9 - 1.3
Potassium (mmol/L)	**5.8**	5.0	3.5 - 5.0
Sodium (mmol/L)	137	138	135 - 145
Calcium (mg/dL)	8.4	-	8.3-10.6
Phosphate (mg/dL)	2.6	-	2.4-5.1
CK (U/L)	**199,011**	-	38-175
Lactate (mmol/L)	1.6	-	0.7 - 2.5
Blood Gas Analysis:			
pH	**7.335**	-	7.35 - 7.45
pCO2 (mmHg)	**25.5**	-	35 - 45
HCO3 (mmol/L)	**13.8**	-	22.0 - 26.0
Base excess (mmol/L)	**-9.80**	-	-2.0 - +2.0
Urinalysis:			
Color	Brown	-	
pH	6.0	-	
Protein	1+	-	
Glucose	Negative	-	
Blood	2+	-	
Bilirubin	Negative	-	


a case of rhabdomyolysis following vigorous exercise (3 sets of 24 push-up cycles) after 6-8 weeks of no exercise.[8] This case played futsal consecutively for several days; dehydration, and hot environmental temperatures increased the patient's risk of developing ER.

Clinical symptoms of rhabdomyolysis vary and can be asymptomatic.[2,6] Myalgia, weakness, and tea-colored urine constitute the classic triad of rhabdomyolysis symptoms.[6,9] However, less than 10% patients show this classic triad, and more than 50% patients do not complain of myalgia or weakness but notice changes in urine color as an initial symptom.[3,6,9] Respectively, myalgia, weakness, and tea-colored urine were found in 23%, 12%, and 10% patients.[6] Muscle swelling and tenderness may also be found.[6] Non-specific systemic symptoms, due to intracellular muscle components in the circulation, may include malaise, fever, nausea, vomiting, tachycardia, fatigue, and headache.[3,5,6,9] In severe cases, confusion, agitation, and delirium may occur.[3]

This case presented with symptoms of tea-colored urine, myalgia, and weakness, which are the classic triad of rhabdomyolysis, as well as fever. Syncope was not a typical symptom of rhabdomyolysis. Therefore, decreased consciousness in this case may be due to other conditions related to exercise.

Decreased consciousness during exercise can be caused by several conditions such as exertional heat stroke, exercise-associated collapse, cardiac arrest, or exercise-associated hyponatremia.[10] Exertional heat stroke can be triggered by strenuous physical activity in a hot or humid environment and is characterized by hyperthermia (body temperature >40°C or an increase >2.5°C from the baseline) and central nervous system dysfunction/altered mental state (e.g., delirium, coma, or seizures).[11,12] Risk factors for exertional heat stroke include dehydration, obesity, aging, and immune system disorders such as diabetes mellitus.[11] Signs and symptoms include tachycardia, hypotension, sweating, nausea, headache, confusion, and loss of consciousness.[13] Risk of multiorgan dysfunction include rhabdomyolysis, acute liver injury or failure, acute kidney injury, electrolytes depletion, central nervous system impairment, cardiovascular dysfunction (ischemic changes), systemic inflammatory response syndrome, and disseminated intravascular coagulation.[14] This case presented with decreased consciousness and a body temperature of 40.7°C after strenuous exercise and inadequate fluid intake. This condition is consistent with exertional heat stroke. Exertional heat stroke and dehydration were suspected as the cause of syncope due to playing futsal in a hot environment.

Serum CK levels are a sensitive indicator of muscle damage, it begin to increase within 2-12 hours after muscle damage and reach their peak within 24-72 hours.[6] Serum CK levels more than 5x the upper limit of normal or >1000 IU/L after heavy physical activity accompanied by appropriate clinical symptoms can confirm the diagnosis of RE.[3,7,9] Serum CK concentration is proportional to the extent of muscle damage.[6]

Apart from serum CK increase, rhabdomyolysis can also caused an increase of potassium, creatinine, myoglobin, AST and LDH blood levels.[2] Other laboratory findings are leukocytosis and increased ESR and CRP.[15] This case had a serum CK level of 199,011 IU/L, a leukocyte count of 20,700/µL, hyperkalemia, and increased serum creatinine level. Sunder (2019) reported a similar case in a 35 year-old male who experienced rhabdomyolysis after heavy exercise with a serum CK level of 2937 IU/L, a leukocyte count of 19400/µL, and a serum creatinine level of 12.2 mg/dl.[16] An observational study in Singapore on 62 patients with rhabdomyolysis reported higher mean serum CK levels in RE compared with rhabdomyolysis due to other causes (15,343 IU/L vs 6,007 IU/L).[17]

Various complications may occur in ER, including acute kidney injury (AKI), compartment syndrome, hyperkalemia, hypocalcemia, hypercalcemia, hyperphosphatemia, hypovolemia, and disseminated intravascular coagulation.[9,12] AKI with 10%-30% incidence, is a complication that may increase morbidity and mortality in rhabdomyolysis.[2,7] Mortality in ER patients without AKI treated in the intensive care unit is 22% and increases to 59% if accompanied by AKI.[16] The risk of AKI in rhabdomyolysis increases in patients with CK levels >5000 IU/L.[8,9] An observational study in Dubai of 25 ER patients showed AKI in 5 (20%) patients with CK levels 17,080-33,030 IU/L, blood potassium levels 5.7-6.1 mmol/L; all patients experienced metabolic acidosis and oligoanuria and required hemodialysis.[18] AKI in ER occurs due to the toxic effect of myoglobin on proximal tubular cells through the formation of free radicals, distal tubular obstruction due to the formation of myoglobin casts (in acidic urine), and dehydration due to exercise.[3] This case experienced complications in the form of AKI, hyperkalemia, and metabolic acidosis with a serum CK level of 199,011 IU/L.

Management of ER includes intravenous rehydration to prevent AKI using crystalloid fluids (Ringer's lactate or NaCl 0.9%) to achieve the target urine output of 1-3 ml/kg/hour.[2,3] Fluid administration can be started at a rate of 400 ml/hour (200-1000 ml/hour) and titrated according to urine output.[2] Renal replacement therapy is considered if the patient remains anuric despite fluid rehydration.[2] Sodium bicarbonate is administered if the patient experiences acidosis, with a urine pH target of >6.5 to minimize kidney damage due to myoglobin.[7] Hyperkalemia and hyperphosphatemia were corrected with medications or hemodialysis.[2,9] Correction of hypocalcemia should be avoided because calcium levels will increase as the disease progresses.[2] If the patient is not hospitalized, it is recommended to rest for 72 hours and sleep at least 8 hours every night.[19] Patients are allowed to return to exercise after >4 weeks and are no longer symptomatic, starting with light exercise.[19] This case received aggressive intravenous rehydration, correction of hyperkalemia, and sodium bicarbonate therapy for correction of metabolic acidosis.

Various measures can be taken to prevent ER. Warming up before exercise can help the muscles to adapt, thereby reducing muscle damage risk during exercise.[5] Gradual increase of exercise intensity and adequate water intake during exercise can reduce the risk of rhabdomyolysis.[4,5] Avoiding exercise while suffering from communicable diseases due to viruses, such as influenza, diarrhea, or vomiting, can also reduce the risk of ER.[5] Adequate intake of fluids, electrolytes, and nutrients (carbohydrates and protein) after exercise helps regenerate damaged muscles and prevent rhabdomyolysis.[5]

CONCLUSION

Rhabdomyolysis has various clinical

manifestations. It's important to consider exertional rhabdomyolysis as a differential diagnosis in patients with brown-colored urine after exercise. Prompt diagnosis and appropriate management can reduce complications, morbidity and mortality. It's also crucial to educate the public about preventive measures to minimize the risk of exertional rhabdomyolysis.

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