



Case Report

A rare autopsy case of traumatic rhabdomyolysis associated with intermittent assault



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ABSTRACT

Traumatic rhabdomyolysis generally occurs after severe blunt trauma and is acute in onset, associated with severe disease, and potentially lethal. Accordingly, diagnosis of traumatic rhabdomyolysis in patients without massive subcutaneous or intramuscular hemorrhage is difficult, especially in the postmortem period, which is limited in terms of the availability of biochemical examination tools and accurate history of illness. To the best of our knowledge, there are no previous reports of death from traumatic rhabdomyolysis among individuals who did not pursue medical consultation. A previously healthy man in his early sixties had been punched and kicked several times in the previous 2 months, but he had not gone to a hospital. He suddenly lost consciousness at his workplace approximately 5 days after the most recent assault, and cardiopulmonary arrest occurred when the emergency service arrived. He died the same day, and a medicolegal autopsy was performed. Although several sites of minor subcutaneous and muscle hemorrhage were observed, the cause of death was unclear upon macroscopic assessment. Immunohistochemical staining revealed acute renal failure caused by rhabdomyolysis. We herein report a rare case of fatal traumatic rhabdomyolysis, seemingly associated with minor and apparently nonlethal muscle injury.

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1. Introduction

Rhabdomyolysis, or the dissolution of skeletal muscle, is characterized by the leakage of intramyocellular substances such as myoglobin, electrolytes, and other sarcoplasmic proteins into the circulation [1–3]. The development of rhabdomyolysis has many causes, including trauma, excessive muscular activity (e.g., exercises and seizures), prolonged immobilization, muscle ischemia, infection, excess body heat (e.g., heat stroke and malignant syndrome), metabolic and electrolyte disorders, drugs and toxins, and genetic defects [4–9]. In severe cases, rhabdomyolysis causes life-threatening complications such as acute renal failure, hyperkalemia-induced arrhythmia, and disseminated intravascular coagulation [10–14]. In contrast, patients with less severe forms of rhabdomyolysis or chronic or intermittent muscle destruction have few symptoms and do not develop renal failure [15]. Conse-

quently, traumatic rhabdomyolysis with a fatal outcome typically involves severe blunt injury and takes an acute course; therefore, diagnosis of rhabdomyolysis as the cause of death is thought to be less difficult [15].

We herein describe a rare autopsy case of kidney injury induced by traumatic rhabdomyolysis that was difficult to diagnose because of minor muscular hemorrhage. To the best of our knowledge, severe traumatic rhabdomyolysis without significant muscle hemorrhage has not been previously reported.

2. Case report

A male hairdresser in his early sixties suddenly lost consciousness at his workplace (a barber's shop). He had no history of medications for internal use. Cardiopulmonary arrest occurred upon arrival of the emergency service. Resuscitation was unsuccessful, and he was pronounced dead within 1 h of the emergency call. A recent police investigation revealed that he had been punched and kicked several times in the previous 2 months and that the most recent assault was approximately 5 days before his death.

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Fig. 1. Discolored skin suggesting subcutaneous hemorrhage distributed over the whole body.

At 2 and 3 days before his death, he had complained of fatigue and missed work, but he had not gone to a hospital. Whether he had developed anuria or oliguria was unclear. Two coworkers were present when he lost consciousness, and no seizure was observed. One of the two coworkers was later arrested for assault and found guilty.

His rectal temperature, measured 3 h after death, was 35.4 °C. It was early summer and the daily maximum temperature was 26.7 °C. Weak postmortem lividity was present over the back of his body excluding the pressure points. His height was 165 cm and weight was 61.5 kg. Areas of discolored skin (apparent bruises) were distributed over the whole body, and their marginal regions were partially brown (Fig. 1). At autopsy, subcutaneous hemorrhage in the process of absorption was observed on the legs and lumbar region; no obvious subcutaneous hemorrhage was present in other parts of the body, including the chest, abdomen, back, and upper limbs (Fig. 2). The subcutaneous hemorrhage involved about 30% of the left leg and about 20% of the right. The subcutaneous hemorrhage was considered to be undergoing healing because of the process of absorption and the color of the marginal regions of the bruises.

Intramuscular hemorrhage was observed in the femoral and lower back muscles; however, it appeared to be nonlethal (Fig. 2). The blood volume of the heart was 50 ml and the blood in the liver and kidneys was moderately congested. The weight of the lungs was increased by edema and congestion (right lung, 900 g; left lung,

875 g). Gastric ulceration was found in the pyloric section, and a small amount of bleeding in the ileum was observed. There was no perforation of the digestive tract. There was a small amount of pleural effusion (right, 6 ml; left, 27 ml) and a small amount of pericardial effusion (6.5 ml). There were no macroscopic abnormalities of the epicardium or cardiac muscle. No coronary stenosis or occlusion was present. There was no systemic edema. Both kidneys were normal in size (right, 180 g; left, 185 g). No other macroscopic abnormalities were present. The blood urea nitrogen level was 108.8 mg/dl and the serum creatinine level was 8.64 mg/dl, indicating the presence of antemortem renal failure [16]. The hemoglobin A1c level was within normal limits (4.7%), and the C-reactive protein level was modestly elevated (4.7 mg/dl). Ethanol was not detected in the blood or urine. Screening tests for common and illicit drugs by liquid chromatography–tandem mass spectrometry in the blood and urine showed negative results. Histological investigation of whole organs and the quadriceps and gastrocnemius muscles was performed. The striated muscles showed no inflammatory cell infiltration or necrosis. There was no suggestive feature of fat embolism in the lung. Although no glomerular changes were observed under hematoxylin–eosin staining, immunohistochemical staining of kidney sections with myoglobin antibody revealed distinct staining of tubular casts (Fig. 3). There were no other histological abnormalities in the organs examined. The urine was not cola-colored, but yellow and slightly cloudy, and the urinary myoglobin level was >3000 ng/ml.



Fig. 2. Upper photograph, left leg. Lower photograph, right leg. The solid lines show areas of muscular hemorrhage. The dashed lines show subcutaneous hemorrhage in the healing process.

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