

Subacute Flask Paralysis Developed after Falling from High; Gullain Barre Syndrome? Or Rhabdomyolysis?

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Abstract

Rhabdomyolysis is a condition in which intracellular components undergo systemic circulation leading to impairment in clinical and laboratory findings due to striated muscle destruction due to traumatic or traumatic causes. The most common clinical symptoms of rhabdomyolysis are muscle weakness, muscle pain and dark urinary tract. The most important complication is acute renal failure.

A 32-year-old female patient was brought in by an immediate family member with complaints of fatigue, muscle aches, and wandering in the morning. The patient had a history of psychosis and the use of antipsychotic medication. In the patient's anamnesis, it was learned that she jumped about six meters in height 10 days ago for suicide but was able to walk completely independently and without support. There was flask paralysis in the lower extremity on the neurological examination performed. She was begun to be followed by Guillain barre syndrome (GBS) by preliminary diagnosis. In the biochemical analysis results, creatine kinase 50,902 U/L (24-190), AST 871 U/L (10-40) and lactate dehydrogenase 1617 U/L (220-450) were detected. According to the clinical and laboratory results, acute renal insufficiency (ARI) developed in the patient despite diagnosis of rhabdomyolysis and intensive hydration therapy. ARI was completely improved with appropriate treatment. Patient was mobilized and discharged.

In this article, a case is presented in which GBS was considered with the initial examination findings in the emergency department and clinical and laboratory findings and rhabdomyolysis were detected in follow-up. It was emphasized the importance of seeing rhabdomyolysis, which is among the neuromuscular emergencies, in mind for the rare occurrence. The presence of flask paralysis in our case clinic and the development of rhabdomyolysis after falling from the top have the feature of being first.

Keywords: Rhabdomyolysis; Guillain barre syndrome; Flask paralysis; Trauma; Acute renal failure

Introduction

Rhabdomyolysis (RML) is a clinical condition resulting from the release of plasma intracellular material (myoglobin, creatinine phosphokinase, aldolase, lactate dehydrogenase, aspartataminotransferase, and potassium) into the plasma which destroys sarcolemma integrity. Clinical and laboratory findings and complications arise as a result of destruction of muscle cells [1].

In RML etiology; various factors such as post-traumatic muscle compressions, alcoholism, drug use, intoxications, heavy exercise, status epilepticus, delirium tremens, viral infections, electrolyte imbalances (especially hypopotassemia, hypophosphatemia), hypothermia and hyperthermia are involved [2].

Clinically, in RML patients; findings such as muscle pain and tenderness, fever, malaise, sensory abnormalities of the affected case can be found. The most stable laboratory finding of RML is increased serum myoglobin level. However, since the half-life of myoglobin is very short (1-3 h), this increase has no diagnostic value. Serum creatine kinase (CK) level is the most typical indication of muscle damage and is the most practical method used for diagnosis. The most important complication is the impairment of renal perfusion and the development of acute renal failure (ARI) due to myoglobin damage [3].

In our study, we presented a case with the first neurological examination finding Gullain Barre syndrome (GBS) with traumatic trauma and sudden gait difficulty in follow-ups, laboratory and clinical findings and rhabdomyolysis diagnosis in follow-up.

Case Report

A 32-year-old female patient was brought in by an immediate family member with complaints of fatigue, muscle aches, and wandering in

the morning. In the patient's anamnesis [1,2] it was learned that she jumped about six meters in height 10 days ago for suicide but was able to walk completely independently and without support. There was no evidence of bone pathology in the direct radiographs and that there was an emergency center admission on the day of trauma. A few days before the emergency service was brought on, the gradual onset of fatigue, muscle aches and walking polio complaints began, and the last day was well settled. On entry physical examination blood pressure was: 130/80 mmHg, heart rate: 120 pulses/min, fever: 36.2°C, number of breaths: 14/min. Neurological examination; conscious, cooperative, oriental. In the motor examination, the upper limbs had 4/5 motor power while the lower extremities had flask paralysis. Deep tendon reflexes (DTR) were hypoactive at the upper foci and abolished at the lower side. The base skin was taken as a bilateral flexor. The patient's self-history has long been known to require help in out-of-home work, while maintaining her home function, which is diagnosed with sciatica (paranoid type (DSM IV-TR)). As medical treatment, he used risperidone for psychosis 4 mgk/day and essitolopram 20 mg/day. There were no characteristics in his family history.

Because of the trauma and fast walking walking strength of our patient's history, our emergency department initially had direct

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Received December 14, 2017; Accepted January 20, 2018; Published January 27, 2018

Citation: Ulusoy EK (2018) Subacute Flask Paralysis Developed after Falling from High; Gullain Barre Syndrome? Or Rhabdomyolysis?. J Clin Case Rep 8: 1075. doi: 10.4172/2165-7920.10001075

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radiographs, thoracolumbar CT and Lomber MR for trocolumbar and musculoskeletal pathologies. Routine blood tests were sent. The pathology of the musculoskeletal system was not determined in the patient who was evaluated by neurosurgeon and orthopedic. In the laboratory review; Leukocyte was: 8100/mm³ Hb: 12.4 g/dL, AST; 1146 u/l (N 13-41), ALT: 368 u/l (N 7-35), LDH: 2085 u/l (N 140-280), K: 5.1 mmol/l (N3.5-5.1), BUN: 29 mg/dl (N: 2-20), Kr: 1,1 mg/dl. Her liver enzymes were high in her tests and abdominal USG was withdrawn and reported as normal. The result was consulted with gastroenterology clinic and acute pathology was not considered. The patient was admitted to our service with the preliminary diagnosis of GBS for the reason that the patient had a rapid and progressive walking gait.

On the first day of hospitalization, extensive biochemistry and hemogram tests were sent. Electromyographic examination was performed in our electroneurophysiology laboratory. However, the pathological findings of the nerves and muscles examined were not observed. The muscle pain of the patient became clearer. Laboratory values were AST; 871 u/l, ALT: 380 u/l, LDH: 1617 u/l, K: 5,0 mmol/l BUN: 50 mg/dl, Kr: 2.54 mg/dL, and CK was 50,902 u/l. Urine density was 1013 and was dark brown. The patient was thought to have rhabdomyolysis due to trauma, height of CK, urine color and anuria. The patient was began intense hydration and was counseled by nephrology. Hemodialysis (HD) was performed on the patient whose ongoing anuria had significant renal function tests. Psychiatric counseling was used to adjust dosing of antipsychotic medications that could cause rhabdomyolysis, and medications were discontinued early. Day 2 blood values were BUN: 43 mg/dL, Kr: 2.2 mg/dl, CK: 25,900 u/l, respectively. There was a decrease in the muscle mass of the patient. Her muscles moved her feet on a flat surface, but she could not overcome the gravity. Day 3 blood values were BUN: 18 mg/dL, Kr: 1,5 mg/dl, CK: 7,910 u/l. Patient who had increased urine output and urine color did not need HD. The patient was completely bed rested in the early period. At week 1, the CK value of the patient was measured as 250 u/l, and the patient was able to support the upper extremity without any loss of strength, while the lower extremity supported the standing. At month 1 control, CK: 219 u/l, the patient had mild muscle pain but could mobilize without support.

Discussion

RML may occur due to traumatic or non-traumatic causes. Because of the striated muscle damage from the spleen, intracellular components pass through the systemic circulation leading to impairment of clinical and laboratory findings. However, when rhabdomyolysis is mentioned, first the muscle damage table that develops after crush syndrome comes to mind. Approximately 40% of patients with rhabdomyolysis suffer from muscle compression. Rhabdomyolysis can occur after trauma, excessive physical activity, epileptic seizures, alcohol and other medications, infections [1,4]. There was a history of high-to-low trauma and antipsychotic medication about ten days before the onset of clinical symptoms.

Guillain-Barre Syndrome (GBS) is an acute inflammatory polyneuropathy characterized by rapid progressive symmetrical muscle weakness and loss of deep tendon reflexes. Symptoms usually start from the lower extremities, progress to the trunk and upper extremities over the days. Progression is usually symmetric [5]. In the differential diagnosis, rhabdomyolysis between the neuromuscular vacuoles should be considered.

The complaints and findings of patients diagnosed with rhabdomyolysis vary. The most common clinical symptoms of

rhabdomyolysis include muscle weakness, muscle pain, dark urine excretion. In a comprehensive study, muscle pain and weakness were detected in about 50% of patients with rhabdomyolysis [6]. Although the exact diagnosis of rhabdomyolysis depends on laboratory tests, it is necessary to pay attention to the clinical symptoms. Rhabdomyolysis mild forms may not have any muscle symptoms. Electrolyte disturbances may occur in the blood circulation due to destruction of muscle tissue and concomitant nausea, vomiting, confusion, coma or heart rhythm problems may occur [2,7]. In addition, although the patients are not mentioned, the most important clinical finding, urine color, should be questioned in detail. Due to myoglobinuria, a dark brown color of the urine color should be considered in the diagnosis of rhabdomyolysis as well as renal pathologies. [7] There was muscle weakness, which began at the lower extremity and was rapid progression. At first, GBS was thought to be due to the fact that there was a high-fall story and flank paralysis, no mention of dark urine, and the fact that the DTRs were abolished. Serum CK concentration was not consulted in ER. However, the follow-up treatment was started immediately after CK was over 50 thousand, dark colored urine, an aneurysm, and rhabdomyolysis. Out of the cases of rhabdomyolysis occurring during hypokalemic periodic paralysis, none of the case reports in the literature screening showed no flank paralysis or rhabdomyolysis developed after a high fall [8]. Although the patient's serum potassium concentration is normal, flank paralysis is seen in her clinic and he is the first to have rhabdomyolysis after heights fall.

Complications include electrolyte imbalance, compartment syndrome, disseminated intravascular coagulation (DIC), and peripheral neuropathy, with acute renal failure and hyperpotasia being the most important complications. The incidence of acute renal failure after rhabdomyolysis ranges from 17 to 40%. The reason for ARF is the accumulation of myoglobin and similar proteins in the kidney and direct nephrotoxic effect [9,10]. Acute renal failure developed on the first day of admission in our case, despite intensive hydration. The patient was referred to the nephrology clinic and her renal function after hemodialysis was completely normalized. Urine output increased, and urine color came to normal color from the bay. This complication did not develop in our case, which is a case of compartment syndrome.

The increase in serum CK level is the most typical indication of muscle damage. For the diagnosis of rhabdomyolysis, it has been suggested that the CK value should be above 500 units, 1000 units per 3000 units. The enzyme reaches the top of the serum usually within the first 12-24 hours after trauma and normally has a half-life of 48 hours. However, the height of CK in rhabdomyolysis does not have a fully predictive value for the development of ARF [11,12]. Our patient was over 50 thousand (u/l) on the first day of admission to CK level. There was a decline in CK levels with improvement in the clinic. However, even at 1 month's control, it was over 200.

There is no precisely defined algorithm in the treatment of rhabdomyolysis. Diagnosis is advised for aggressive fluid therapy ARI. There is no specific fluid therapy. Sodium bicarbonate, mannitol and furosemide are recommended in the treatment of rhabdomyolysis. The events should be followed carefully for hypercalcemia [13]. In our case, the ARI chart developed early in the patient. However, there was intense fluid replacement and short-term improvement with HD. Electrolyte in balance did not develop.

Conclusion

Diagnosis may be missed in emergency departments if the neuromuscular emergencies are not in the routine of laboratory tests

for rhabdomyolysis, and this may be confused with GBS. Since the rhabdomyolysis clinic is variable, the physical examination and the tale do not give enough clues to diagnosis, the suspicion of rhabdomyolysis and the importance of remembering rhabdomyolysis gain importance. CK, potassium, etc. laboratory tests need to be looked at for diagnosis. Detailed anamnesis should be taken in patients presenting with walking pain and muscle pain reasons, trauma and drug use narrative should be questioned. In these patients, acute renal insufficiency should be awakened as a complication and early hydration should be initiated. It is also the first feature of flank paraparesis in our patient's clinic and the development of rhabdomyolysis after a fall from the top.

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