Research Paper

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"Rhabdomyolysis"; Elevated CPK values following Seizures

Medical Science

KEYWORDS : Rhabdomyolysis; High CPK ; Seizures ; Acute Renal Failure

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ABSTRACT

Male aged 40 years was admitted with H/o fall , seizures& fever. Patients blood sample was received in the laboratory for investigations. Pts total leukocytic count was more than normal and CPK values were extremely high (51000 U/L). The patient had elevated serum Creatinine, Blood Urea Nitrogen (BUN) Serum Uric acid, Serum Sodium, Serum Phosphorous, Bilirubin, SGOT, SGPT , deranged prothrombin time and decreased potassium, calcium, protein and serum albumin. Also the urine colour and dipstix test suggested presence of myoglobin in urine. Retrospectively the clinical details of the patient mentioned that he had history of fall with three episodes of generalized tonic clonic convulsions. Also, the patient was hypertensive and a known case of chronic liver disease secondary to alcoholism and had no history of cardiac or renal disease. The above investigations along with the clinical details suggested that the patient might be a case of Rhabdomyolysis leading to acute renal failure with extremely high CPK values. In spite of repeated haemodialysis and electrolytes management, the patient could not recover and succumbed to death on third day of admission.In case of very high levels of CPK values in patients of Rhabdomyolysis leading to acute Renal Failure, a conclusive diagnosis should be made at the earliest.

INTRODUCTION

Creatine phosphokinase (CPK) is an enzyme expressed by various tissues and cell types. CPK catalyses the conversion of creatine and consumes adenosine triphosphate (ATP) to create phosphocreatine (PCr) and adenosine diphosphate (ADP). Elevation of CPK is an indication of damage to muscle. It is therefore indicative of injury, rhabdomyolysis, myocardial infarction, myositis and myocarditis. The raised CPK level is the most sensitive laboratory finding for rhabdomyolysis.[1]Rhabdomyolysis means destruction or disintegration of striated muscle .The disease was first described in the 20th century, and important discoveries as to its mechanism were made during the Blitz of London in 1941.[6]The breakdown products when muscle is damaged include a protein called myoglobin.(**Figure 1)** Myoglobin is related to hemoglobin. Both are proteins in nature. Hemoglobin transports oxygen in the blood; myoglobin stores oxygen in muscles. When myoglobin is released into the blood after muscle injury, it is filtered out of the body by the kidneys. Since myoglobin is toxic to the small tubules of the kidneys, high levels of this protein can damage the kidneys and may result in acute renal failure.[2]This syndrome ranges from an asymptomatic illness with elevation in the CPK level to a life-threatening condition associated with extreme elevations in CPK, electrolyte imbalances, acute renal failure (ARF) and disseminated intravascular coagulation.

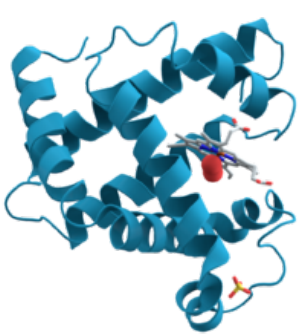

Figure 1
[Schematic diagram of myoglobin, a heme-containing protein that participates in oxygen storage in normal muscle but is responsible for kidney damage in Rhabdomyolysis]

Figure 2
[Patient with rhabdomyolysis showing the characteristic brown discoloration of urine as a result of myoglobinuria .]

CASE REPORT

We received a blood sample of a forty years old male patient for complete blood investigations along with the fever profile. **On first day** he had elevated WBC count of 15,700 cells per cubic mm, Blood Urea Nitrogen (BUN) **34 mg/dl** ,Serum Creatinine **1.3** mg/dl, Serum Uric acid **14.2mg/dl**, Serum Sodium 152 meq/l, Serum Potassium 2.8 meq/l, Serum Calcium **10.5 mg/dl** , Serum Phosphorous **4.6 mg/dl** , Serum Cholestrol 252 mg/dl , Total Bilirubin **3.71 mg/dl** , Direct Bilirubin **1.7 mg/dl,** Total protein **10.0 gm/dl**, Albumin 4.9 g/dl, SGOT **477 IU/L** and SGPT **124 IU/L** deranged Prothrombin time of **17.7** seconds and INR of 1.2. **The tests for Malaria and Dengue were negative.**

On second day he had elevated WBC count of 12100 cells per cubic mm, Serum Creatinine of **2.6**mg/dl was increased to **5.2 mg/dl** in the later half of the day ,Blood Urea Nitrogen (BUN) **52 mg/dl**, Serum Uric acid **18.5 mg/dl** Serum Sodium 151 meq/l, Serum Potassium 2.7 meq/l, Serum Calcium **5.9 mg/dl**, Serum Phosphorous **8.7 mg/dl** Total Bilirubin **5.5 mg/dl** , Direct Bilirubin **4.6 mg/dl,** Total protein **6.5gm/dl**,Albumin 3.1 g/dl, SGOT **1466 IU/L** and SGPT **225 IU/L** ,deranged Prothrombin time of 17.9 seconds, INR of 1.2 and the Creatine Phoshokinase(**CPK**) was found to be **51,000 U/L** which was unusually high. Retrospectively the clinical details of the patient mentioned that he had history of fall with three episodes of generalized tonic clonic convulsions with tongue bite, blood tinged vomiting, decreased urinary output was febrile and was having altered sensorium. On exploring the history further it was found that the patient was hypertensive and a known case of chronic liver disease secondary to alcoholism. However patient had no symptoms pertaining to cardiac problem as well as no past history of renal disease. So the positive findings in patient were **extremely high CPK levels, acute elevation of serum creatinine, hypocalcemia, hyperuricemia, hyperphoshatemia,deranged prothrombin time, electrolyte imbalance, high SGOT , high SGPT, high total protein on the first day , leucocytosis and high coloured urine with proteinuria .** Myoglobin has a short half-life, and is therefore less useful as a diagnostic test in the later stages. [3]

That is why total protein values in our case had fallen from 10 gm/dl to 6.5 gm/dl on second day. The above investigations along with the clinical details suggested that the patient might be a case of Rhabdomyolysis leading to acute renal failure with extremely high CPK values. Rhabdomyolysis was confirmed with a positive dipstix test on urine .

DISCUSSION

Rhabdomyolysis in seizures can occur due to fall, leading to muscle trauma and fractures. Muscle forces generated during tonic-clonic seizures alone can also cause severe muscle injury. The risk of renal failure increases with co-morbid conditions such as sepsis, dehydration, and acidosis. The diagnosis is usually straightforward with typical clinical and biochemical features such as hyperkalemia, hyperphosphatemia, hypocalcemia, and elevations in serum uric acid and creatine kinase (MM isoenzyme) levels at presentation suggest a diagnosis of Rhabdomyolysis as were seen in our case .[4]Rhabdomyolysis occurs frequently but is usually asymptomatic. However, in more serious cases, severe electrolyte disorders and acute renal failure may occur, leading to life-threatening situations. The most sensitive laboratory finding of muscle injury is an elevated CPK level.[5] CPK elevations are frequently classified as mild, moderate, or severe. These classifications roughly correspond to less than 10 times the upper limit of normal (or 2,000 IU/L), 10 to 50 times the upper limit of normal (or 2,000 to 10,000 IU/L), and greater than 50 times the upper limit of normal (or greater than 10,000 IU/L), respectively. The risk of renal failure increases above 5,000 to 6,000 IU/L. In the absence of myocardial or brain infarction, CPK >5000 U/l indicates serious muscle injury.In our patient, typical clinical features and **CPK** value of **51,000 IU/L** strongly suggested the diagnosis of Rhabdomyolysis which was further confirmed in urine by dipstix test.

The management of patients with rhabdomyolysis includes advanced life support (airway, breathing and circulation) followed by measures to preserve renal function – the latter includes vigorous hydration. The use of alkalizing agents and osmotic diuretics, while commonly used, remains of unproven benefit. In this case, in spite of repeated haemodialysis and electrolytes management, the patient could not recover and succumbed to death on third day of admission.

CONCLUSION

In case of very high levels of CPK values in patients of Rhabdomyolysis leading to acute Renal Failure, a conclusive diagnosis should be made at the earliest. Such cases should be closely supervised by the nephrologists or the treating physicians as rigorous hemodialysis and strict vigil on electrolytes can markedly reduce the mortality rate .

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