Calcium Kinetic in a patient with acute renal failure due to Rhabdomyolysis: A Case Report and Review of Literature

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Abstract

Hypercalcemia may follow hypocalcemia in the course of acute renal failure (also named now as acute kidney injury) secondary to rhabdomyolysis. The clinician should be aware of calcium kinetics to avoid the complications of both hypocalcemia and hypercalcemia which may occur at few days interval during the recovery phase. This report presents a case of a young male who developed anuric Acute Renal Failure due to strenuous exercise induced rhabdomyolysis. He was treated with supportive, corrective and dialysis measures. The progress was favorable with a diuretic phase. During the diuretic phase, he developed progressive hypercalcemia reaching up to 3.54 mEq/l with constipation and drowsiness. Investigations showed besides stigmata of rhabdomyolysis and Acute Renal Failure, low initial levels of vitamin D metabolites. The calcemia eventually normalized with fluids, dialysis and a single dose of Pamidronate Sodium. The patient was discharged 3 weeks after admission with a recovered clinical condition, improved renal functions and normal calcemia. The biphasic kinetics of calcium in this setting is documented. We conclude that serum corrected calcium should be monitored in the context of Acute Renal Failure due to rhabdomyolysis.

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Introduction

Rhabdomyolysis is a known cause of Acute Renal Failure (ARF). It may be due to different causes which are in common muscle injury and necrosis.[1] Calcium kinetics presents a particular pattern in this type of ARF. The clinician should be well aware of this specific entity to give proper care and to avoid the complications associated with hypo and or hypercalcemia. We present a case which illustrates the biphasic pattern of calcium kinetics and the management of the patient.

Case Report

A 28 year old Omani male was admitted to the nephrology department for the management of ARF. He did not have a history of any particular medical condition. He complained of fever and severe myalgia that followed shortly after strenuous exercise in the form of prolonged excessive exercise in the gymnasium lasting for approximately 2 hours.

The next morning, he noticed that his urine was dark and decreased in quantity. These symptoms and signs progressed over a period of one week. Initially, he attended private clinics where he was prescribed symptomatic treatment including non-steroidal anti -inflammatory drugs (NSAIDs). On admission, physical examination showed a young male with good stature. He presented with symptoms of nausea, constipation and loss of appetite. The vital signs were stable, but he was afebrile. His pulse rate was regular at 82/m, while his blood pressure was 140/80 mmHg. He was also found to be anuric. The other elements of physical examination were unremarkable. Laboratory investigations showed that urea was 35.5 mmol/l (N: 3.3-7.0), serum creatinine was 1615 mmol/l (N: 45-100), and creatine kinase (CK) was 14424 mmol/l (N: 25-70). While serum calcium was 2.02 mmol/l (N: 2.1-2.6), hemoglobin was 14.6 g/dl (N: 14-18.1), and white blood cell count was 10.4 x 10 pwr 9 (N: 3.6-11.5). The ultrasound showed normal kidneys without obstruction. In view of these elements, the clinical diagnosis of acute renal failure (acute kidney injury) due to rhabdomyolysis secondary to strenuous exercise was made.

Treatment and management consisted of vital signs monitoring, urine output and input charting, calcium and intravenous fluids. Hemodialysis through a temporary vascular access was instituted. On day two, the urine output was nil. He received dialysis and other supportive measures for two weeks. During this period, he improved clinically and started to produce urine. The last dialysis was performed at day 15. Calcium monitoring during the diuretic phase showed that it had increased to a peak value of 3.54 mmol/l at day five, (Fig. 1). He complained of vague abdominal pain, constipation and fatigue, which could be attributed to the hypercalcemia. Hypercalcemia during the recovery phase of ARF due to rhabdomyolysis was suspected. Further investigations showed the following:

1. 25 (OH) Vitamin D3 36 nmol/l (N: 53-150);
2. 1,25 (OH)2 vitamin D3 11 pmol/l (N: 43-148)
3. Parathormone (PTH) 0.8 pmol/l (N: 0.8-8.5).

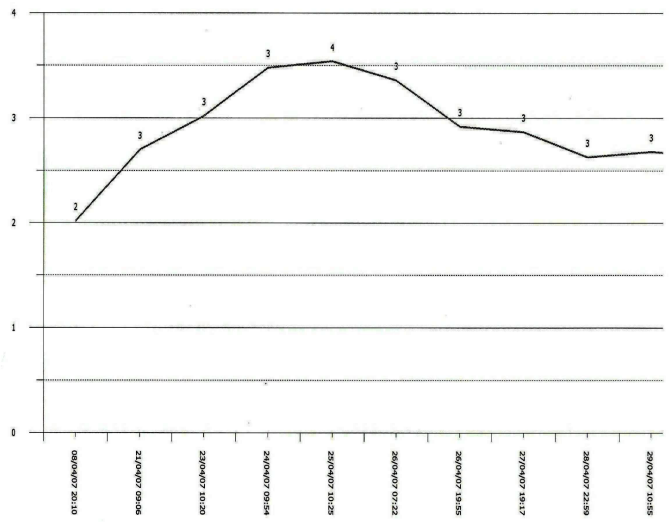

Figure 1: kinetics of the calcemia during the recovery of the ARF. After 3 weeks of treatment the patient's serum creatinine was 288 mmol/l (N: 45-100), and serum calcemia was 2.63 mmol/l.

He was discharged on day 21 in a very good general condition with improved renal functions. He was advised follow up at the nephrology out-patient clinic.

Discussion

The terminology of ARF (Acute Renal Failure) is now being replaced by the term Acute Kidney injury.[1,2] It is defined as either an abrupt increase in serum creatinine level of more than 0.3 mg/dl (26.5 mmol/l), or a percentage increase of more than 50% (by a factor of 1.5 from the baseline), or a reduction in documented urinary output (<0.5 ml/kg body weight /hour for more than 6 hrs). The causes of Acute Kidney Injury are divided into pre-renal, intra-renal and post-renal. Pre-renal factors range from obvious renal hypoperfusion in patients with hypotension or hemorrhage to more subtle hypoperfusion such as that seen in patients with heart failure or cirrhosis. Post-renal acute kidney injury is caused by the blockade of urinary flow. While Intra-renal causes of acute kidney injury can be divided into diseases of the vasculature, tubulo-interstitial compartment or the glomeruli.[1,2] Rhabdomyolysis may lead to myoglobinuric acute renal failure and is the etiology of 5-25% of cases of acute renal failure.[1,2] The first documented case of rhabdomyolysis causing acute renal failure was described by Bywaters and Beall in1941.[3] The most common etiological factors are traumatic crush injuries during wars and natural disasters especially earthquakes, electric shock, pressure necrosis, central occlusion, and surgery.[4,5] Other reported etiologies in order of apparent prevalence include alcoholism, muscle compression, seizures, metabolic derangements, drugs, infection, muscular dystrophies and Strenuous exercise has been reported to cause rhabdomyolysis with ARF in some cases.[1,2,4,5] Abnormal calcium metabolism is a specific complication of rhabdomyolysis induced acute renal failure.[6] During the initial and oliguric phase, patients are frequently hypocalcemic. The hypocalcemia can sometimes be extreme.[6,7] Nevertheless, care should be taken in the management of initial hypocalcemia, as overzealous correction may lead to hypercalcemia. Calcium deposition in the muscles, hyperphosphatemia and skeletal resistance to the action of the parathyroid hormone are believed to be the possible underlying mechanisms.[6] It is possible that the initial calcium intake participates in the development of the hypercalcemia. During the recovery phase, hypercalcemia may develop in up to 40% of patients.[6,7] The pathophysiology of the hypercalcemia observed in the recovery phase may be due to several mechanisms including mobilization of calcium from injured muscles, soft tissue deposits, to the secondary hyperparathyroidism which developed during the initial phase and to the elevated levels of 1,25 dihydroxy vitamin D that also developed during the initial phase as a corrective response to the hypocalcemia.[8]

Most cases of hypercalcemia are self-limiting and do not require major interventions other than observation and proper hydration. Nevertheless, some cases may require specific measures including dialysis intensification, biphosphonate and diuresis with loop diuretics.

Conclusion

Hypercalcemia (during the diuretic phase) following hypocalcemia (in the initial phase) may occur during the course of ARF associated with rhabdomyolysis. In most instances, these conditions are self limited and do not require specific measures other than observation and proper hydration, but may require intervention when severe and symptomatic. The clinician should be aware of the dual kinetics of calcium in this situation to plan proper monitoring and intervention.

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