Abstract:

Introduction: The reported incidence of acute kidney injury after rhabdomyolysis is as high as 65%[1] which occurs as a result of multiple mechanisms including tubular obstruction, direct and ischemic tubular injury, or intrarenal vasoconstriction. In this report, we present a case of acute kidney injury induced by rhabdomyolysis in a young male post-trauma.

Presentation: A 37 yr old male patient presented to the emergency department with a history of trauma and physical assault 4 days back, multiple wounds all over the body and reduced urine output for 1 day. On examination he was found to have generalised swelling all over the body, multiple bruises and abrasions along with fracture of right 4th metatarsal bone. Laboratory studies revealed CPK: 17345 IU/L; BUN: 79mg/dl; Serum creatinine: 11.35mg/dl; Serum potassium: 4.1meq/L; Urine Ph: 5. Adequate hydration was started with balanced salt solution and 0.9% NS also haemodialysis was done with which the patient's renal functions improved gradually.

Conclusion: Rhabdomyolysis induced AKI in a patient with no co morbidities resolves gradually with adequate hydration and urine output monitoring.

Introduction:

Rhabdomyolysis is a clinical condition in which skeletal muscles break down rapidly releasing toxic substance like creatine kinase and myoglobin into the blood stream which in turn leads to kidney injury. This can happen because of sustained trauma, sepsis, heat stroke or even drugs.

Pathophysiology:

Rhabdomyolysis involves direct sarcolemic injury leading to depletion of ATP within the myocyte which impairs the function of ATPase pump. This causes an increased sarcoplasmic calcium levels leading to a state of persistent contraction of the myofibril. This sustained contraction leads to further energy depletion.

After myocyte injury, intracellular contents are released into the circulation.[2, 3] Hyperkalaemia, hyperuricaemia, and hyperphosphataemia can develop rapidly. High levels of phosphate released in blood; bind to calcium, and calcium–phosphate deposition occur in soft tissue, resulting into hypocalcaemia. Ischemic muscle is

also forced to utilise anaerobic metabolism leading to metabolic acidosis.

During the recovery phase of rhabdomyolysis, significant number of patients develop hypercalcaemia, due to the release of vitamin D stores from injured muscle, providing substrate for the production of excess 1,25-dihydroxyvitamin D[4.] Hypercalcaemia may be further exacerbated if excess calcium is administered during the acute hypocalcaemic phase.

Myoglobin a dark red haem-containing protein stores and transports oxygen in muscle. Only small levels of it are normally present in plasma. It has a small molecular weight and is easily filtered. When the renal threshold for free myoglobin is exceeded it appears in urine.[5] This then interacts with the Tamm–Horsfall proteins to form brown granular casts resulting in tubular obstruction. The process is favoured when the urine is acidic and may have no nephrotoxic effect when the urine is alkaline.

Myoglobin also causes deficit of nitric oxide leading to renal vasoconstriction which further compounds to kidney injury. Renal blood flow is further compromised by hypovolaemia, activation of the renin–angiotensin system, and additional vascular mediators. [2]

Clinical features:

Severity of symptoms depends upon the degree of muscle damage and extent of kidney injury. Rhabdomyolysis may be asymptomatic or associated with muscle pain, nausea, sepsis, dyselectrolytemia, arrythmia, acute renal failure and even coma. A high degree of suspicion is needed from prompt management and avoidance of complications arising. Severe pain may at times limit limb movement and additionally in compartment syndrome or crush injury the muscles may get tense and swollen, leading to a sensoryneural loss. In diffuse muscle injury such as which occurs with drugs, there may be a generalised malaise with diffuse myalgia. Rarely, patients may volunteer that their urine has changed to a red or brown colour.

Lab findings:

Rhabdomyolysis is typically diagnosed when the CK is more than 5000 units/litre or five times its normal upper limit. Myoglobin levels peak before increases in CK, however, myoglobin is metabolised rapidly at sites outside of the kidney hence CK a

more reliable marker of rhabdomyolysis. The absence of myoglobinuria does not rule out the possibility of rhabdomyolysis. In a study of 475 patients with rhabdomyolysis diagnosed by CK levels, myoglobinuria was only detected in 19%[6] A Metabolic Acidosis with high anion gap is usually observed. Arterial blood gases also reveal trend of serum lactate and pH and serves as a guide for fluid replacement. Regular observations including hourly urine output are required to detect any deterioration promptly.

Case Report:

A 37 year old male, with no comorbidities, presented to the emergency department with complains of reduced urine output for 1 day, there was an associated history of physical assault 4 days back in which he suffered multiple contusions and abrasions all over his body along with a fracture of right 4th metatarsal bone, for which orthopaedic consult was taken. He was admitted to the critical care department and his initial laboratory studies revealed CPK: 17345 IU/L, BUN: 79mg/dl, Serum creatinine: 11.35mg/dl, Serum Potassium: 4.1meq/L, Urine Ph: 5, Serum Calcium: 7.2mg/dl, Serum Phosphorus: 7.6mg/dl.

Foley's catheterization was done and central venous access was taken. Keeping myogologinuria induced rhabdomyolysis in mind he was thereafter managed primarily with fluid resuscitation with a target urine output of 3 ml/kg/hr or 300 ml/hr. 0.9% Normal saline, balanced salt solution was used while monitoring potassium and arterial blood gases to look for hyperchloraemic acidosis.

Continuous infusion of soda bicarbonate was initiated to alkalinise the urine to reduce the precipitation of Tamm–Horsfall protein complexes. A urinary pH of 7 was achieved on day 2 of the treatment. We also initiated infusion of Inj furosemide at 5-10 mg/hr to maintain an adequate urine output.

The patient was evaluated by a nephrologist and haemodialysis was initiated on day 2 and day 4 of admission, soda bicarbonate and furosemide was gradually tapered off with close monitoring of urine output. Fluid resuscitation continued, as guided by IVC dimensions. The patients renal functions improved gradually and he was later discharged in a stable condition with adequate urine output, with a serum creatinine of 2.59mg/dl and CPK: 150 IU/L.

Discussion:

We have reported a case of Acute kidney injury due to trauma induced rhabdomyolysis, patient here landed up into AKI due to dehydration and inadequate fluid resuscitation, which eventually resolved with intravenous hydration therapy. Another contributing factor of adequate recovery was young age of the patient with no history of any comorbidities or drug intake which could have been be a secondary cause of renal dysfunction.

Evidence for the use of sodium bicarbonate as a therapy to prevent AKI in rhabdomyolysis is lacking. A recent systematic review found no level 1–3 evidence to support its use.

Mannitol also has theoretical benefits by flushing nephrotoxic agents through the tubules, it extracts fluid that has accumulated in injured muscle, and acts as a free radical scavenger. Again there was no level 1–3 evidence to support the use of mannitol in the prevention of AKI[7.]

Considering the benefit achieved by alkalising the urine and forced diuresis soda bi carbonate and furosemide infusion was used in this patient. Further studies may be needed to clarify this effect.

Hence, we conclude stating the primary treatment of Acute kidney injury induced by rhabdomyolysis being vigorous fluid resuscitation with intensive urine output monitoring.

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Abbreviations :

AKI : Acute Kidney Injury

CPK : Creatinine Phospho Kinase

CK : Creatinine Kinase

BUN : Blood Urea Nitrogen

ATP : Adenosine Tri Phosphate

NS : Normal Saline

IVC : Inferior Vena Cava

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