Case Report

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An Unusual Cause of Myoglobinuria

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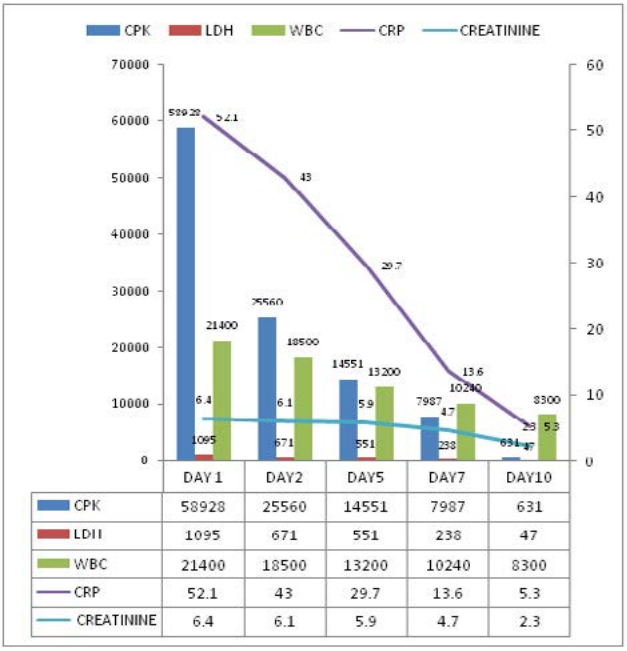
Rhabdomyolysis is characterized by the acute breakdown of skeletal muscle, resulting in the release of muscle cell contents like myoglobin, creatine phosphokinase (CK) and lactate dehydrogenase, which can lead to acute kidney injury in severe cases. A number of etiologies have been identified in acute rhabdomyolysis including hereditary and acquired of which drugs and trauma account for the majority of cases [1]. Physical therapy is frequently prescribed and generally considered safe for weakness; deconditioning and non - specific muscle aches. Rhabdomyolysis following a massage session is unheard of. However we report a rare case of rhabdomyolysis with acute kidney injury following an aggressive massage session.

A 57 year old gentleman, who has been diagnosed to have Diabetes and Hypertension for 5 years prior, and who is reasonably well controlled on medications, presented to the hospital with fever (39°C), breathlessness and decreased urine output for a day. On examination the blood pressure was 170/100 mm of Hg, saturation of 94% and bibasilar crepitations on auscultation. He was hospitalized for his fever, breathlessness and oliguria. On investigations it was found that his serum creatinine was 5.7 mg%. The baseline creatinine was 1.1mg% just 2 months prior to this. Further investigations revealed a Hemoglobin of12.7gm% and the total leucocyte count was 21400 /cu mm. His other blood tests revealed: aspartate aminotransferase (AST) 240 U/l, alanine aminotransferase (ALT) 550 U/l, lactic dehydrogenase (LDH) 1095 U/l, creatine phosphokinase (CPK) 58928 U/l with 100% of CPK-MM form, potassium 6.7 mEq/l, creatinine 6.3 mg/dl and urea nitrogen 76 mg/dl. Considering the possibility of acute pyelonephritis hisurine analysis and urine culture was also sent. Urine routine examination showed1-2 RBC, 1-2 WBC, protein trace and no casts. Spot Urine Protein to Creatinine ratio was 2.5 and urine cultures were sterile. Blood cultures were also sterile and the test for Leptospirosis was negative.

In view of the hyperkalemia, fluid overload and uremia he was dialyzed once and the potassium was corrected. The qualitative test of urine for myoglobin was positive. Consequently he was monitored daily for creatinine levels, LDH, CRP, TLC and CPK levels. The time-concentration curve of CPK and LDH was similar to that of WBC of peripheral blood and serum C-reactive protein (CRP) level, (close correlation between the extent of inflammation & rhabdomyolysis process). Downward trend of creatinine also associated with fall in CPK, LDH, CRP and WBC levels. After 2 weeks, the patient was discharged with a stable creatinine. On follow a week later, his serum creatinine was 1.2 mg/dl.

To determine the etiology of raised CPK a detailed history was taken and he confessed that he regularly received body massage for 1 hour. However 1 day prior to hospitalization, he received a prolonged body massage session for 2 h served by two masseurs simultaneously. The strength and intensity of this massage session was also significantly higher. He was not taking statins. He also gave history of diarrhea and vomiting 2 days prior and additionally he was also taking Olmesartan 40 mg daily for hypertension. Generalized muscle pain developed one night prior to admission but was ignored. He developed oliguria and dyspnea the next day and he was hospitalized.



	DAY 1	DAY2	DAY5	DAY7	DAY10
CPK	58928	25560	14551	7987	631
LDH	1095	671	551	238	47
WBC	21400	18500	13200	10240	8300
CRP	52.1	43	29.7	13.6	5.3
CREATININE	6.4	6.1	5.9	4.7	2.3

Graph: The time-concentration curve of CPK was almost parallel to that of WBC and CRP, indicating that the extent of inflammation was closely related to the rhabdomyolysis process. Note that creatinine and LDH levels also declined along with CPK levels.

Compression or pressure-induced rhabdomyolysis has been reported in coma or immobilized patients, prolonged cardiopulmonary resuscitation and obese men who received bariatric surgery, but it has rarely been associated with body massage [2]. Myoglobinuria is a key player in the complex pathogenesis of AKI only in presence of hypovolemia, hypotension, and aciduria [3]. Sinert et al. reported that AKI is not observed when nephrotoxic cofactors are absent. The 3 different pathological mechanisms involved in the development of AKI are: (i) renal hypo perfusion resulting from hypovolemia-induced renal vasoconstriction and myoglobin scavenging of nitric oxide. (ii) Heme protein exerts direct toxicity on tubular epithelial cells and free radical release by myoglobin causes oxidative injury to the tubular epithelial cells. (iii) The precipitated myoglobin, Tamm-Horsfall protein, uric acid crystals in presence of acidic urine causes tubular obstruction and decreases glomerular filtration rate [4]. Elderly and diabetic patients need to be warned that vigorous body massage may lead to this potentially dangerous. In addition, the people receiving body massage should drink adequate amount of water before and after the massage session so as to prevent unusual episodes of rhabdomyolysis-associated AKI, which is exacerbated by volume depletion [5].

References

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