Massive Rhabdomyolysis in Cirrhosis of Liver

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Cirrhosis of the liver is a chronic parenchymal disease which has multiple etiologies. Cirrhosis results in damage to the liver parenchyma and can lead to multiple complications, generally involving either parenchyrnal liver failure or portal hypertension with its consequences[1]. As the disease is chronic in nature, it can develop many complications along with various metabolic abnormalities. as a result of treatment with various medications. Here we describe three patients with liver cirrhosis who developed massive rhabdomyolysis and had no definite identifiable cause.

Case Report

Case 1

A 4 years old male had been diagnosed to have cirrhosis of the liver and had grade Ill esophageal varices and had undergone esophageal variceal sclerotherapy for bleeding several months ago. He had been taking Lactulose and Silvmarin and had been doing well until 3 days prior to present admission when he presented with fever and weakness. On admission, his blood pressure was 125/75 mm Hg, pulse 96/mm. respiration 24/mm. temperature 98° F. He had no ascites. hepatomegalv or edema. Mild jaundice was noted. His laboratory findings were as follows: Sodium 133.Potassium 7.3, random blood sugar 163, serum creatininc 4.0. Prothrom bin time 1 5 sec. total bilirubbin 3.8. direct bilirubin 1.5. AST 3745. ALT 706. CBC showed WBC count 20300. hemoglobin 15.3. hematocrit 42.2. platelet count 152000, neutrophils 89% lymphocytes 10% monocytes 1%. Recent HCV RNA was negative. Chest x-ray showed mild cardiomeglay. ECG showed tall T-waves.

Patient was admitted to intensive care unit and soon after his CPK was found to be 74144. Blood culture was drawn wich did not grow any organism. Repeat CPK within two hours was 142915. His treatment consisted of intravenous fluid and supportive care with Calcium Gluconate and Sodium Bicarb. An arterial blood gases showed PH of 7.3. PCO2: 37, P02:81. saturation 93°/o. Appropriate care in the intensive care was provided but within hours patients developed bradvcardia and could not be resuscitated.

Case 2

This was a 47 years old male who had history of liver disease and had developed jaundice one week ago. He had also complained of swelling of abdomen. No fever was noted. On physical examination blood pressure was 112/70. pulse 80/mm, respiration 14. temperature 98° F. Moderate jaundice was noted. Also mild ascites and edema were present. Laboratory findings on admission showed, sodium 127. potassium 5.1, glucose 123. creatinine 1.0. blood urea nitrogen 25, total bilirubin 22.5. direct bilirubin 15.8. AST 75, ALT 39, alkaline phosphotase 89, prothrombin time 20 sec. CBC showed WBC count 12100. hemoglobin 10.8, hematocrit 30.2. platelet count 99000.

He underwent treatment with Adactone and Silvmarin. Liver biopsy was performed and showed portal inflammation grade III and fibrosis stage IV which indicated cirrhosis. His HCV RNA was negative. His general condition remained well for first three days but then he developed generalized body pain and muscle weakness.

His CPK at this time was performed and was found to be 15925. Aldactone was discontinued. His potassium was 3.7, sodium was 128. Inspite of treatment, patient became hypotensive and could not be revived and expired.

Case 3

This was a 65 years old female, who had cirrhosis for number of years. She came with hematemesis and malena. Her HCV RNA was positive but due to her age she was not treated with Interferon. On

admission, her blood pressure was 110/60, pulse 100/min, respiration 18/min and temperature 98° F. She had no jaundice, ascites or edema. Only tenderness of neck muscles was noted. Laboratory findings revealed a sodium of 137. potassioum of 5.7, BUN of 24 and creatinine of 1.8. Her prothrombin time was 20.4 seconds, total bilirubin 2.5, AST 1674, ALT 388 and Alkaline phosphatase was 175. CRC showed a hemoglobin of 9.5, WBC 10800 and platelet of 67000.
She was given fresh frozen plasma and i/v octreotide and stabilized. Her CPK was fOund to be 237783 and next day it was 1794 18. She had lots of muscle aches now. Her BUN now was 65, creatinine 4.3 and potassium was 6.7. Dialysis was performed but she did poorly and expired. Summary of these three cases is given in Table.

Table. Pertinent Clinical Data of Cases.

	Age/Sex	Total Bilirubin	AST	ALT	Na	K	Creat	CPK
Case 1	41/M	3.8	3745	706	133	7.3	4.0	74144
								142915
Case 2	47/M	22.5	34	81	131	5.1	0.6	15925
Case 3	65/F	2.5	1674	388	137	5.7	1.8	237783
								179418

Discussion

Acute rhabdomyolysis is the most acute and severe form of necrotizing myopathy which occurs from many causes[2]. The muscle damage leads to influx from extracellular compartment into the muscle cells of water, sodium chloride and calcium which lead to hypovolemia. hemodynamic shock, renal failure and hypocalcemia[3]. At the same time. there is efflux from the damaged muscle cells of potassium, purine, phophate. lactic acid. myoglobin and thromboplastin and these lead to hyperkalemia. hyperuricernia, hvperphosphatemia, metabolic acidosis, nephrotoxicitv and disseminated intravascular coagulation[3]. One of these patients had high potassium at the time of presentation and no other significant abnormalities mentioned above were noted. Probably the time needed for these abnormalities to appear had not taken place in this patient and the course was too rapid.
Patients with cirrhosis are known to develop rhabdomvolvsis from various causes which include vasopressin infusion[4], various infections[5], electrolyte imbalance consisting of hyponatremia and hypokalemia[6-8] and drugs[9]. Serum electrolytes in our patients were at reasonable levels and one patient had fever and elevated leukocyte count of which no infection could be localized and the blood culture were found to be negative. However, the presence of infection cannot be entirely ruled out. These patients present very specific management problems because patients of cirrhosis already have ascites where a large amount of fluid can not be administered. Patient with rhabdomyolysis need large amounts of fluid to be administered rapidly[3,10], otherwise they develop renal failure and further complications. In these patients with massively elevated CPK levels indicating severe rhabdomyolysis in presence of liver disease and in one patient ascites. the amount of fluid administered was limited.

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