Wet Beriberi associated with Rhabdomyolysis

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Abstract

Malnutrition and undernutrition are frequently encountered in everyday practice. Thiamin deficiency resulting in Beriberi disease is underrecognized. Here we report a case of cardiac Beriberi associated with rhabdomyolysis, both successfully treated with high dose Thiamin. We also provide possible explanation of the underlying mechanism based on literature review.

Case presentation

Here we report a case of a 20 year-old African American male who presented with severe depression and lower extremity edema.

Patient suffered from depression after traumatic event. He lost 25 pounds in the month prior to presentation. Patient on examination looked emaciated and had feeble voice. He had BMI 16kg/m^2. Regular pulse at 140BPM. Blood pressure 120/91mmHg. Patient afebrile and saturating on room air. He had bilateral calf tenderness. Neurological exam significant for lower extremities hypersensitivity to light touch. EKG showed sinus tachycardia. His workup is shown in **Table 1**and remarkable for Total CK level of 8000 U/L. Hepatitis serology negative. CT angiogram chest/abdomen/pelvis was significant for extensive pneumomediastinum with small pleural effusions. Patient was admitted for further evaluation and was started on fluids for Rhabdomyolysis. Spontaneous pneumomediastinum was asymptomatic and likely related to asthma. On day 2, total CK level went up to 41,000U/L and started to become hypoxic. X-ray showed worsening pleural effusion. Echocardiogram showed generalized hypokinesis and ejection fraction of 15-20%. AST and ALT elevation felt to be secondary to rhabdomyolysis. His total CK remained relatively unchanged. B12 and folic acid level were normal. B1 level was checked and was undetectable. On Day 5, patient was seen by new attending who suspected Beriberi and patient was started on Thiamin (500mg IV three times/day). CK level fell from 30,000U/L to 6,000U/L. patient tachycardia resolved. He started to feel better with more energy on day 7, however, he remained depressed. On day 8, repeat echocardiogram showed significant improvement of ejection fraction to 45-50%. Patient edema resolved and he was discharged home on day 9 in stable condition.

Discussion

Thiamin functions as a coenzyme in the metabolism of carbohydrates and branched-chain amino acids. It is vital for two types of reactions in human body; decarboxylation of α-ketoacids and transketolation. Thiamin has not been linked to toxicity.[1] With limited body storage, Thiamine deficiency can occur in as short as 14 days.[2]

Thiamin deficiency can present as Beriberi in its two types wet and dry, or the Wernicke-Korsakoff syndrome. Dry Beriberi present as symmetrical sensory and motor peripheral neuropathy. Wet type mainly has cardiac involvement with cardiomyopathy.[1] presentation can be as severe as multiorgan failure.[3]

Nowadays this disease is uncommon in developed countries. Most cases seen are related to patients who underwent bariatric surgeries or who suffer from alcoholism and severe malnutrition.[4–6]Thiamin deficiency

has been described in patients on total parenteral nutrition (TPN) if adequate vitamin replacement is not provided.[7]

Cardiac Beriberi can be a challenging diagnosis for patients presenting with dilated cardiomyopathy. The diagnosis can be made based on clinical signs and symptoms associated with dilated cardiomyopathy, peripheral neuropathy, malnutrition and absence of other with response to Thiamin supplementation.[8]

Thiamin plays an integral role in TCA cycle. Its deficiency leads to Adenosine accumulation and decreased ATP production. This can be seen in Beriberi heart disease. It can be chronic which starts as high output state secondary to peripheral vasodilation and decreased vascular resistance. If this state continues along with decreased myocardial contractility, it can lead to low output state.[8–12]Acute Beriberi (Shoshin beriberi) can present with low cardiac output and even cardiogenic shock. This deficiency can lead to myonecrosis which was described in two autopsy studies[13]Aggressive repletion of thiamine results in dramatic improvement in systolic heart function[8,11,14], this was also seen in our patient.

In our review in literature, we didn't find any cases where Thiamin was linked to rhabdomyolysis. Suspected mechanism likely similar to cardiomyopathy. Dramatic improvement in total CK level along with improved energy level and decreased calf tenderness clinically support this theory.

In conclusion, patients presenting with rhabdomyolysis and signs of heart failure who are malnourished might be experiencing Beriberi disease. Thiamin, water soluble vitamin, is not associated with toxicity. Aggressive replacement of Thiamin should be attempted given dramatic improvement if this is the etiology.

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Table 1: Laboratory tests done during admission

Test	1/22/2020	1/23/2020	1/27/2020	1/28/2020
Sodium	139	141	138	
Potassium	5.0	3.9	3.7	
Chloride	104	109	107	
CO2	28	25	27	
BUN	49	24	10	
Creatinine Serum	1.3	1.0	0.8	
Glucose	116	131	82	
Alkaline Phosphatase	85	51	50	
Albumin	4.7	2.7	2.2	
Total protein	9.4	5.7	5.9	
AST	316	601	588	
ALT	97	126	233	
Iron Saturation	31			
Acetaminophen	<3			
TSH	2.19			
Ethanol	<3			
Folate	11			
Iron				
Magnesium	3.2			
TIBC	165			
Transferrin	116			
Vitamin B1, LCMSMS	<6			
Vitamin B12	1,014			
Total CK	8,105	41,376	30,884	6,790
Vitamin D, 25-OH				13