



## MIXED BERIBERI – A COMBINATION OF CARDIAC, NEUROLOGICAL AND GASTRIC BERIBERI- A RARE PRESENTATION

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**ABSTRACT** We present a rare case of mixed beriberi with cardiac, neurological manifestations and gastric Beriberi. Prolonged Thiamine deficiency causes beriberi, presenting as wet or dry Beriberi but a rare coexistence with gastric beriberi is being reported here. Patients with Beriberi generally present with pain, paraesthesia, shortness of breath and irritability. Abdominal pain is a rare manifestation of gastric beriberi. In developing countries apart from nutritional causes, alcoholic abuse is the leading cause of beriberi. Gastric pain due to metabolic acidosis is a rare manifestation of gastric beriberi. This disorder is a metabolic emergency and requires prompt recognition and treatment with intravenous thiamine. Sudden appearance of right heart failure and pulmonary hypertension is common. Death occurs quickly from right heart failure and the patient usually dies fully conscious if this condition is not recognised clinically. We report a case of gastric Beriberi that was successfully treated with intravenous thiamine.

**KEYWORDS :** Cardiac Beriberi, Neurological Beriberi, Metabolic Acidosis, Gastric Beriberi.

### INTRODUCTION:

Beriberi is a treatable condition with insidious onset, rarely presenting with acute abdominal pain and heart failure. If the condition is not recognised promptly mortality occurs within hours. With the increasing incidence of alcoholic abuse incidence of Gastric beriberi has increased around the world. Only a few case reports were documented in India. Prakasha<sup>1</sup> et al. reported three cases of dry and wet-beriberi mimicking critical illness polyneuropathy. Rathi<sup>2</sup> et al reported a case of infantile beriberi. Pothukuchi Venkata Krishna<sup>3</sup> et al reported a sub acute cardiac beriberi with neurological involvement. Here we are reporting a very rare case not reported so far, of mixed beriberi with acute abdominal pain and metabolic acidosis along with neurological signs and right heart failure

### CASE REPORT:

A 34-year-old male who was chronic alcoholic and smoker presented with epigastric pain, chest pain for 2 days with breathlessness of NYHA class-III for past 24 hrs. He had generalized weakness and myalgias and difficulty in getting up from the squatting posture for 5 days, swaying while walking, paraesthesia's in the form of tingling and numbness for one week.

### PHYSICALEXAMINATION:

Physical examination revealed tachycardia of 100/min, regular low volume pulse, blood pressure was 90/70mmHg, and he was afebrile. JVP was elevated with prominent 'V' wave. Apical impulse was felt in the left sixth intercostal space in the mid clavicular line. CVS examination revealed sinus tachycardia, a third heart sound. A systolic murmur of grade 2/6 is heard in tricuspid area. Respiratory system examination revealed tachypnea with bilateral fine inspiratory basal crepitations. On per abdomen examination tenderness over epigastrium and right hypochondrium and hepatomegaly present CNS examination revealed patient conscious, and irritable with speech and cranial nerve being normal. Motor system examination revealed power of grade 3/5 in both lower limbs and deep tendon reflexes were absent in both lower limbs and bilateral plantar reflexes were flexor. Sensory examination revealed vibration and fine touch sensation were lost in both lower limbs and ataxic gait present.

### INVESTIGATIONS:

Hemogram showed normocytic hypochromic anaemia and Haemoglobin- 12.3g/dl, RBC count- 3.4 M/cu mm, WBC count-18.9/cu mm, Platelets count- 3.5 lakhs/cu mm, ESR- 9mm/1st hr, RBS-150mg/dl, Blood Urea- 33mg/dl, Serum Creatinine -0.8mg/dl, Serum electrolytes sodium-135 meq/L, Potassium-4.0 meq/L, Chlorides-101

meq/L, ABG analysis-

PH-6.9

PO<sub>2</sub>-60mmHg

PCO<sub>2</sub>-20mmHg

Bicarbonate- 6meq/L

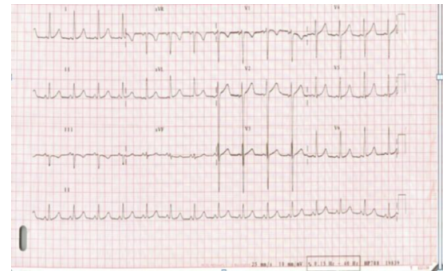
Serum amylase- Normal Limits

Serum lipase – Normal Limits

Liver Function Test – Normal limits

Blood Thiamine Level – <0.06 µ/dl (0.2-2.0)

Ecg :Fig: 1). Sinus rhythm ,no st-t changes



**Fig:1** ECG – sinus rhythm, no st-t changes

Xray chest pa view(fig:2) showed ctr:60%,left ventricular configuration



**Fig:2**

ECHO – (fig:3)

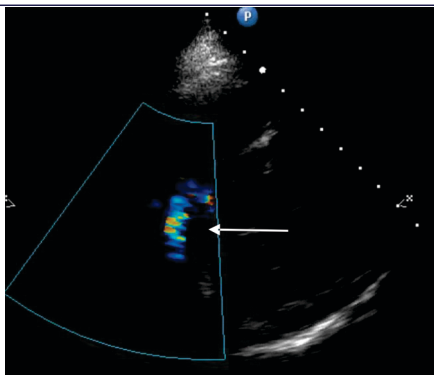
showed Dilated right atrium, right ventricular

Mild to Moderate TR

Mild PAH, PASP (40-45 mmHg)

IVC plethora present

Good LV systolic function



**Fig .3-2D ECHO Color Doppler showing mild Tricuspid regurgitation(white arrow)**

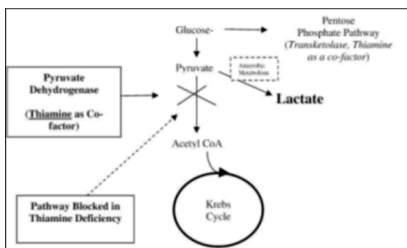
**NERVE CONDUCTION STUDIES:**

Showed mixed sensory and motor axonal neuropathy in lower limbs without changes of demyelination.

**DISCUSSION:**

Thiamine deficiency or beriberi, refers to the lack of thiamine pyrophosphate, the active form of vitamin B1<sup>4</sup>.

Thiamine pyrophosphate is the biologically active form of thiamine, acts as a coenzyme in carbohydrate metabolism through the decarboxylation of alpha ketoacids (table:1) It also takes part in the formation of glucose by acting as a coenzyme for the transketolase reactions in the pentose mono phosphate pathway. The body cannot produce thiamine and can only store up to 30mg in tissues. Thiamine is mostly concentrated in the skeletal muscles. Other organs in which it is found are the brain, heart, liver, and kidneys.



**Table :1**

In developed nations beriberi is reported in alcoholism and fad diets, long term peritoneal dialysis, long term starvation and person on receiving high glucose containing IV fluids. In developing nations mostly due to nutritional deficiency in poor, people in refugee homes. Diuretic usage leads to thiamine deficiency due to increased excretion of thiamine.

In chronic alcoholics, thiamine deficiency due to decreased B1 absorption and storage dysfunction. Due to thiamine deficiency there may be increased lactic acid production leading to metabolic acidosis.

**PATHOPHYSIOLOGY:**

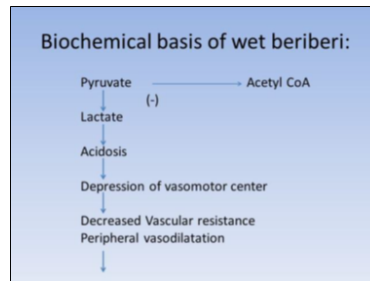
When healthy individuals are deprived of thiamine, thiamine stores will be depleted within one month, however within one week of stopping thiamine intake healthy people develop a resting tachycardia, decrease in short term memory, weakness and decreased deep tendon reflexes. Some people develop peripheral neuropathy. Nervous system involvement due to with thiamine deficiency is termed as dry beriberi. Cardiovascular involvement due to thiamine deficiency is termed as wet beriberi.

**Wet Beriberi:**

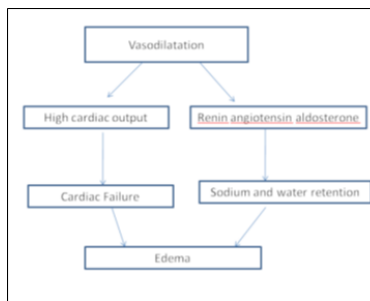
Wet Beriberi is one of the clinical syndromes associated with thiamine deficiency. Thiamine in its phosphorylated form (TPP) is the precursor, co-factor of both pyruvate dehydrogenase and alpha ketoglutarate decarboxylase which are both key enzymes of kerbs cycle.

Decrease in the activity of these two enzymes due to thiamine deficiency may lead to the accumulation of pyruvate and lactate<sup>5</sup> in the tissues. Which leads to the decrease in the peripheral resistance and

increases the venous blood flow and increase in the cardiac preload. Increase preload and myocardial dysfunction leads to congestive heart failure. (Table:2,3) Wet beriberi<sup>6</sup> usually associated with right heart failure and moderate pulmonary arterial hypertension.



**Table:2**



**Table:3**

Some patients come to the hospital with low cardiac output, some with ST elevation myocardial infarction, some with non-ST MI, pericardial effusion or severe pulmonary arterial hypertension.

Wet beriberi has a rare and more severe form known as “Shoshin Beriberi”<sup>7,8</sup> or acute fulminant cardiovascular beriberi. It is due to the predominant injury to the heart and rapid deterioration follows the inability of the heart muscle to satisfy the body’s demand. They present with cyanosis of palms and soles, distended neck veins, restlessness, tachycardia and anxiety. oedema may not be present.

In unexplained causes of metabolic acidosis specifically lactic acidosis, it is important to consider beri-beri due to thiamine deficiency. In gastro intestinal beri-beri<sup>9</sup> abdominal pain is due to lactic acidosis.

Watson<sup>10</sup> et al reported 39.4% (13/33) of wet beriberi presented with acute renal failure with high levels of pyruvate and lactate. It is due to the peripheral vasodilatation leading to activation of renin angiotensin aldosterone system which leads to the decreased glomerular filtration rate. Acute liver failure may be another complication of wet beriberi due to right heart failure.

**DIAGNOSIS:**

The Laboratory diagnosis of thiamine deficiency usually made by a functional enzymatic assay of transketolase activity measured before and after the addition of thiamine pyrophosphate. A>25% stimulation in response to the addition of thiamine pyrophosphate (i.e. an activity coefficient of 1.25) is interpreted as abnormal. However, this test is expensive and time consuming. If laboratory confirmation is required, we need to measure blood thiamine, pyruvate, alpha ketoglutarate, lactate, glyoxylate levels, urinary excretion of thiamine and its metabolites. Serum TSH is needed to rule out thyrotoxicosis induced high output heart failure.

**TREATMENT:**

In suspected cases of thiamine deficiency, prompt administration of parenteral thiamine is indicated. The recommended dose in 50 mg given intramuscularly for several days. The duration of therapy depends on the symptoms, and therapy is indicated until all symptoms have disappeared. Maintenance is recommended at 2.5-5 mg per day orally unless a malabsorption syndrome is suspected.

In acute thiamine deficiency with either cardiovascular or neurologic signs, 200 mg of thiamine three times daily should be given intravenously until there is no further improvement in acute

symptoms; oral thiamine (10 mg/d) should subsequently be given until recovery is complete.

Lactic acidosis associated with acute pernicious beriberi has been treated with thiamine alone sodium bicarbonate is also usually recommended.

The return of normal metabolic and hemodynamic status reduced lactate production and allowed metabolism of excess lactate to provide an acid base status.

For Shoshin beriberi patient after B1 administration the hemodynamic indices dramatically improved within minutes to hours.

#### **CONCLUSION:**

We present a rare case of mixed beriberi (Neurological/Cardiac) (wet beriberi) along with gastric beriberi in a patient with history of heavy alcohol consumption.

Patients with malnourished diet who have unexplained heart failure, metabolic acidosis (lactic acidosis) with or without multi organ failure should be empirically administered thiamine without delay.

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