ROLE OF THIAMIN DEFICIENCY IN THE HEART FAILURE

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Annotation: *Thiamine is a water soluble B1 vitamin available in cereal grains, egg, nuts and meat products. Thiamin is a required coenzyme in the energy-producing reactions that fuel myocardial contraction. So, thiamin deficiency may contribute to myocardial weakness by limiting the energy available for contraction. Studies have reported a wide range in the prevalence of thiamin deficiency in patients with heart failure (3–91 %). The impact of thiamin supplementation in patients with heart failure is inconclusive. Because, studies conducted to date are limited by their small sample size, indirect methods of assessing thiamin concentration, methodological inconsistencies, use of impractical means of thiamin supplementation, a focus on hospitalized patients. This article illustrates the role of thiamine in the body and the importance of thiamine deficiency in heart failure.*

Key words: *Thiamine deficiency (TD), heart failure (HF), adenosine triphosphate (ATP), glycolysis, cardiovascular system, adenosine, beriberi, cardiomyocyte.*

INTRODUCTION

After consuming, vitamin B1 which is included in vegetables, cereals or meat products is absorbed and combines with adenosine triphosphate in liver, kidneys and leukocyte to form thiamine pyrophosphate, a coenzyme in carbohydrate metabolism. Its active form thiamin pyrophosphate (TPP) serves as a coenzyme for pyruvate dehydrogenase complex and α-ketoglutarate dehydrogenase in the Krebs cycle, thereby playing a vital role in aerobic metabolism and ATP production. Decreasing of some useful products such as meat, egg, fish, nuts, broccoli during daily diet causes to thiamin deficiency.

Thiamin deficiency can lead to two different diseases: dry and wet forms of beriberi. Dry beriberi is associated with a violation of the central and peripheral nervous system, whereas wet beriberi is manifested by damage to the cardiovascular system. Thiamine deficiency impairs production of adenosine triphosphate, leading to accumulation of adenosine. This increase causes reduction in systemic vascular resistance via direct vasomotor depression, leading to a compensatory high-output state with increased blood volume.

Dry beriberi is characterized by polyneuropathies with symmetrical changes in sensory, motor and reflex functions. Another manifestation of damage to the nervous system is Wernicke syndrome – Korsakov, who represents the classical triad, including confusion, ataxia and oculomotor abnormalities. The probable pathogenesis of wet beriberi is the depletion of ATP in cardiomyocytes and an increase in the formation of adenosine in the myocardium. A decrease in ATP causes a weakness of contractile function of the myocardium, which ultimately leads to HF. A decrease in ATP synthesis in cardiomyocytes

causes an increase in adenosine monophosphate, which is converted into adenosine. The accumulation of adenosine in cardiomyocytes leads to its release into plasma followed by systemic vasodilation. Thiamine is a coenzyme of mitochondrial glucose oxidation. TD, causing a blockade of the conversion of pyruvate to acetyl-CoA, reduces the formation of ATP and causes cellular acidosis with an increase in the level of free fatty acids. The lack of ATP causes the body to activate glycolysis and mobilize fat resources. The accumulation of pyruvate due to a slowdown in the transformation into acetyl-CoA and increased glycolysis leads to the conversion of pyruvate into lactate and the development of lactic acidosis. These biochemical processes lead to pathological changes in the cardiovascular system. The end-diastolic pressure in the right and left ventricles increases with an increase in oxygen consumption.

Peripheral vascular resistance decreases, which is accompanied by arteriovenous blood shunting, increased cardiac output and venous insufficiency.

MATERIAL AND METHODS

We took experience of another country. After obtaining the ethical clearance from the Institutional Ethical Committee, the present study was conducted in the Department of Cardiology, SKIMS Srinagar. 50 patients were enrolled in this study.

RESULTS

All the included patients were admitted in the Cardiology department with the diagnosis of heart failure made using Framingham criteria2 requiring the presence of two major or one major and two minor criteria.

The study participants were randomly allocated to one week of inpatient double blind intravenous therapy with either placebo or thiamine given as two daily I/V doses of either normal saline or 100 mg thiamine HCl. Each patient underwent two dimensional echocardiographic examination before the intravenous treatment and at the end of inpatient week using a commercially available system.

Thiamine system was evaluated by measuring thiamine pyrophosphate effect (TPPE) or erythrocyte transketolase activity prior to and at the end of the week of intravenous treatment by semi-automatic method adopted by JN Mount et all3. In addition to thiamine status, the following clinical parameters were daily assessed for 1 week: blood pressure, heart rate, body weight and 24 hour urinary output. Further, NYHA class, complete blood count, serum glucose, serum Na+/K+, blood urea, serum creatinine and urine analysis were compared before and after one week of treatment. All patients who were taking drugs 5 days before entering the hospital for the study continued them at the same dosages throughout the full 7 weeks of the study3.

The issue of age as a factor contributing to the prevalence of thiamine deficiency was evaluated by Levy et all7 who conducted a prospective trial of patients with a mean age of 47+10 years, NYHA class of 2.5+0.6 and mean LVEF of 22+9%. No evidence of thiamine deficiency was fund in their population of heart failure patients4.

In a recent study, Smithline et al.5 examined the effect of thiamin (100 mg thiamin or placebo) in a small number of acute heart failure patients (55 in the control group and 63 in the treatment group). Thiamin values increased significantly in the treatment group and

were unchanged in the control group. They concluded that, "in mild-moderate acute heart failure patients without thiamine deficiency, a standard dosing regimen of thiamine did not improve dyspnea, biomarkers, or other clinical parameters."

CONCLUSION

Studies on thiamin supplementation have shown mixed results. In conclusion, it can be said that treatment with thiamine in patients with chronic heart failure is important in restoring heart health and generating energy, although it is not recommended by most medical professionals in a specific treatment.

LITERATURES:

1.American journal of clinical nutrition. Volume 110, issue 6, 2019; p.1271

2. Ho KKL et al. Framingham Criteria for diagnosis of congestive heart failure. Circulation 1993; 88: 107.

3. Role of thiamine deficiency and efficacy of thiamine in treatment of patients with heart failure. International journal of advanced research 2020.

4. Levy WC, Soine LA et al. Thiamine deficiency in congestive heart failure (Letter). Am J Med 1992; 93: 705.

5. Smithline HA. Supplemental thiamine for the treatment of acute heart failure syndrome: a randomized controlled trial. BMC Complement Altern Med. 2019; 19: 96