Increased troponin I in "wet" beriberi

Troponin I (TnI) elevation is the direct result of cardiomyofibril damage and subsequent release into the circulation. There are many causes for TnI elevation¹ but no previous reports have addressed the metabolic problem of beriberi or thiamine deficiency and TnI elevation. This report highlights two cases of beriberi in the presence of beriberi.

Investigations in both cases were undertaken in the same laboratory. TnI was analysed using the Beckman Coulter Access II platform with a coefficient variation of 5.6%. The detection limit is 0.01 µg/l and the upper reference limit is 0.03 µg/l at the 97.5th centile.²

Case 1

Patient 1 was a 64 year old widow with chronic alcoholic cirrhosis and ongoing alcohol consumption at around 40 to 60 g/day. She presented with a sudden collapse without any preceding symptoms. Her blood pressure was posturally low at 140/80 lying and 110/70 sitting, with moderate dehydration and evidence of mild biventricular failure. Her TnI was 0.35 µg/l (normal <0.1 µg/l), thiamine 46 nmol/l (reference range, 0 to 75 nmol/l), erythrocyte transketolase activity 5% (normal <5%), brain natriuretic peptide (BNP) 88 pg/ml (normal <20 pg/ml), and serum magnesium 0.55 mmol/l (reference range, 0.60 to 0.95 mmol/l). Transthoracic echocardiography showed diffuse global dyskinesia with a left ventricular ejection fraction (LVEF) of 45%, consistent with "wet" beriberi. Her coronary angiogram showed minor atherosclerotic disease in the right coronary artery. She was treated with oral thiamine and cardiac failure drugs. Her heart failure had clinically resolved at a six week follow up visit. Repeat echocardiography was not undertaken.

Case 2

A 48 year old man of no fixed address presented with fulminant (Shoshin) beriberi including severe peripheral dropsy and abdominal pain. He was clinically found to have evidence of chronic alcoholic cirrhosis and was in severe biventricular failure with pulmonary congestion, with 10 cm elevation of jugular venous pressure and pitting oedema to mid-thigh. His blood pressure was 100/60 with a pulse rate of 112 beats/min. His TnI was 0.48 µg/l, thiamine 18 nmol/l, erythrocyte transketolase activity 34%, BNP 458 ng/ml, and serum magnesium 0.44 mmol/l. Transthoracic echocardiography showed marked left ventricular dyskinesia with an ejection fraction of 32%. He was treated with intravenous frusemide and thiamine after dextrose supplementation and improved steadily thereafter with oral thiamine. Follow up echocardiography showed a left ventricular ejection fraction of 42%.

Comment

The cases are representative of a typically high risk group of thiamine deficient patients, with chronic alcoholism, social isolation, and presumably a poor dietary intake.³ As the body storage of thiamine is often small, with a high turnover rate and a

Figure 1 The multiple intracellular functions of thiamine. Thiamine is involved in the various energy generating pathways at the cellular level as well as in the formation of intracellular and intercellular products. These are critical for the optimal function of the whole heart (adapted from Russell,³ no permission required; and Alberts *et al.*, in: *Molecular biology of the cell*, 4th edition, chapter 14. New York: Garland Science, 2002:773 (with permission)). The electron microscopic picture insert shows an abundance of mitochondria in the myocardium, where thiamine is pivotal in providing a constant source of energy for optimal myocardial function.

half life of 10 to 18 days, high risk patients can enter into thiamine deficiency rapidly. Coexisting hypomagnesaemia, a likely result of chronic alcohol abuse, further aggravates the myocardial damaging effect of thiamine deficiency.⁴

At the precellular level, chronic alcoholism causes thiamine deficiency by reducing intestinal absorption.⁵ Intracellularly, thiamine and magnesium normally function as co-factors for critical enzymatic activities in the pentose phosphate and glycolytic pathways and the citric acid cycle, which involve the enzymes transketolase, pyruvate dehydrogenase, and α-ketoglutarate dehydrogenase, respectively (fig 1). Because these pathways are pivotal for cellular function, thiamine deficiency will be markedly disruptive, leading to eventual myocardial fibrillar apoptosis and death. This is a particular problem for the continually active and energy demanding myocardium. In addition, thiamine is also important for the generation of glutathione, an antioxidant, which is needed to counteract the highly reactive free radicals. In the relative absence of thiamine and hence of glutathione supply, cells will malfunction because of excessive oxidative stress, leading to premature death.⁴ Hypothetically, chronic or recurrent episodic thiamine deficiency will lead to cumulative permanent damage causing cardiomyopathy in the long term, as in patient 2, independent of the direct effect of alcohol.

In the absence of atherosclerotic disease, TnI can be significantly raised in thiamine deficiency, which is particularly common in

alcoholics. However, where this problem is suspected there is no harm in treating the high risk patient empirically with thiamine, even though the clinical index of suspicion may be low.

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