Increased serum levels of troponin I and lesions in coronary angiography in hemodialysed patients

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Abstract

Purpose: Calcium-phosphate disorders are a frequent finding in HD patients, and, in some cases, may cause an abnormal coronary calcification. Some of the HD patients have increased serum cTnI level without evidence of acute coronary syndrome. The aim of this study was to determine if there is a correlance between increased cTnI levels and presence of stenotic changes in coronary arteries in asymptomatic HD patients.

Material and methods: In 13 of 119 HD patients (M:F 10:3) a coronary angiography was performed. The mean age of the patients was 53 years (33-76) and the mean HD duration was 55 months (3-156). cTnI was analyzed by AxSYM system and, subsequently, by VIDAS system.

Results: A constant or intermittent elevation of cTnI was detected in 5 of 13 patients. In 10 of 13 pts a critical stenosis of at least 1 coronary artery was found. A critical stenosis was found in 4 of 5 cTnI (+) patients and in 6 of 8 cTnI (-) patients. An excess calcification of coronaries was observed in 7 patients, including 1 cTnI positive patient with no evidence of coronary stenosis.

Conclusions: 1. The elevation of cTnI in asymptomatic HD patients is observed when there is: (I) excess calcification accompanied by a critical stenosis of at least 1 coronary artery, (II) a critical stenosis of 2 or more coronaries with no evidence of calcification. 2. We suggest that excess cardiovascular calcification in HD patients may be one of the major factors responsible for the troponin release.

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Introduction

Cardiovascular diseases are a main cause of mortality among end-stage renal disease (ESRD) patients. There are methods and markers to be found that could possibly predict the risk of life threatening cardiovascular events, especially in HD patients. In the latest data, troponin I (cTnI) was found to be one of the most specific markers of cardiac injury [1,2]. Troponin I is a marker with high specifity to cardiomyocyte injury and plays a major role in acute myocardial infarction (AMI) diagnosis [2]. Some of the HD patients have increased serum cTnI levels without evidence of acute coronary syndrome (ACS) [3,4]. The reason for this phenomenon is unknown. Factors that may possibly predict the elevation of serum cTnI are left ventricular hypertrophy (LVH) [5,6], coronary artery disease (CAD) [5,7], HD duration and calcium-phosphate disorders [3,8]. In HD patients with a long history of therapy, asymptomatic cardiomyocyte injury caused by heart calcification may occur [9]. In some cases, an abnormal calcification of coronary arteries was observed in this group of patients. The aim of the study was to determine if there is a correlance between increased cTnI levels and stenotic changes in coronary arteries in asymptomatic HD patients.

Material and methods

We examined a total of 119 patients (M:F 69:50, age range 40.5 (20-78) years) haemodialyzed in the dialysis unit in Toruń. In patients with no history of angina during the previous one month, serum concentration of cTnI was determined before dialysis. During the 28 month follow-up period, five determinations of cTnI were performed at 6 month intervals. In 13 of 119 patients (M:F 10:3) a coronary angiography (CA) was

Table 1. **Comparison of coronary artery stenosis (CAS) and coronary artery calcification (CAC) in analyzed group of HD patients**

	cTnI (+) positive pts n = 5	cTnI (-) negative pts n = 8
Sex (m:f 10/3)	4 / 1	2 / 6
Age, median (years)	60.0	49.8
HD duration, median (months)	70.8	45.1
Coronary artery lesions:		
CAS only	1	3
CAS and CAC	3	3
CAC only	1	-
no lesions	-	2

performed. The indications for CA were ischaemic heart disease (IHD) history or a positive exercise ECG test in asymptomatic potential renal recipients. The mean age of the patients was 53 years (33-67) and the mean HD duration was 55 months (3-156). The following kidney diseases as a cause of renal failure were noted: diabetes – 2, glomerulonephritis – 7, tubulointerstitial nephropathy – 2 and others in 2 patients. The results of the CA were compared with serum cTnI concentrations. The Troponin I level was analyzed by MEIA technology with AxSYM system (Abbott Diagnostic, IL, USA) and, subsequently, by ELFA technology with VIDAS system (Bio-Merieux). Concentrations of cTnI >0.3 μg/L for the AxSYM method and >0.1 μg/L for the VIDAS method were concluded to be positive.

Results

A constant or intermittent elevation of serum cTnI concentration was detected in 5 out of 13 patients (38%). In 10 of 13 patients (77%) a critical stenosis of at least 1 coronary artery was found. After comparing the results, a critical stenosis was found in 4 out of 5 cTnI (+) positive patients and in 6 out of 8 cTnI (-) negative patients. An excess calcification of coronary arteries was observed in 7 patients, including 1 cTnI (+) positive patient with no evidence of coronary stenosis. In 2 cTnI (-) negative patients no coronary lesions were found (*Tab. 1*).

Discussion

Studies carried out in medical centers around the world indicate a prognostic value of the cTnI increase in HD patients. According to some authors, the increased cTnI level predicts ACS and one year cardiac death [6-8,10]. Our findings indicated that 77% of the HD patients with an ischaemic heart disease history or with a positive exercise ECG test had a significant lesions in coronary arteries. An increase of serum cTnI levels was found in 38% of the patients. This suggests that not in all of the patients with lesions narrowing coronary arteries, a constant or intermittent increase of cTnI occurs. There are also other factors that may damage the cardiomyocytes, independently of the coronary artery lesions. It seems that cardiomyocyte injury in HD patients occurs when a long-lasting effect of a number of kidney disease related factors as the course and HD complications take place. It should be noted that a serum increase of cTnI appeared in our patients almost in all cases when CA revealed coronary artery calcification. Arterial calcification is a frequent finding in ESRD patients. Calcification develops at two sites of the arterial wall; arterial intima calcification (AIC) represents an advanced stage of atherosclerosis and is associated with the development of plaques and occlusive lesions [11]; arterial media calcification (AMC) is commonly associated with aging, presence and duration of diabetes and is common in ESRD [11,12]. In ESRD patients, AMC may occur without AIC, and this phenomenon is a consequence of calcium-phosphate disorders. Also, arterial calcification may cause arterial stiffness; however, the mechanism of disturbances varies from obstructive coronary artery disease. It has been shown that arterial wall stiffness, as assessed by an aortic pulse wave velocity is correlated independently with vascular calcification [9]. The elevation in serum phosphate, calcium-phosphate product and increased calcium load are found to be a risk factors for vascular calcification [13,14]. The progress of arterial calcification occurs especially when mineral metabolism is not well controlled and the intake of calcium-based phosphate binders as well as sevelamer is inadequate [15].

Conclusions

The elevation of cTnI level in asymptomatic HD patients is observed when there is: (I) an excess calcification accompanied by a critical stenosis of at least 1 coronary artery, (II) a critical stenosis of 2 or more coronaries with no evidence of calcification. We suggest that excess cardiovascular calcification in HD patients may be one of the major factors responsible for the troponin release.

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