Cardiac troponin I in patients with chronic kidney disease treated conservatively or undergoing long-term haemodialysis

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Abstract

Background: Cardiac troponin I (cTnI) has been shown to be a specific marker of myocardial damage in the general population. In patients suffering from chronic kidney disease (CKD) cTnI may be increased in serum without other signs of acute myocardial damage confusing the diagnosis.

Aim: To compare cTnI concentration in CKD patients, treated conservatively or with haemodialysis, with healthy controls, and to evaluate the cardiovascular risk factor profile in these groups.

Methods: The study population consisted of three groups: group I (n=10, 5 women, 5 men, mean age 32±4 years) – healthy, young volunteers without kidney diseases with creatinine clearance (CrCl) 97.13±23.24 ml/min; group II (n=21, 8 women, 13 men, mean age 51±15 years) – patients with CKD in stages 3-5 with CrCl=34.04±18.34 ml/min; and group III (n=30, 14 women, 16 men, mean age 50±14 years) – patients on long-term haemodialysis. The cTnI level was measured using an AxSYM analyzer (Abbott). In group III blood was taken before the haemodialysis session. The high sensitivity C-reactive protein (hsCRP), haemoglobin, parathyroid hormone (PTH) and phosphorus levels were determined. Blood pressure was also recorded. Echocardiography was performed and left ventriclular mass index (LVMI) was calculated on the basis of the Devereux and Reichek formula.

Results: Compared with controls, the cTnI values were significantly higher in patients from group III and tended to be higher in patients from group II (0.01±0.03 vs. 0.063±0.08 and 0.066±0.162 ng/ml, respectively, $p < 0.05$ and NS). In 46% of haemodialysed patients cTnI concentration was above the value of the 99th percentile in the apparently, healthy population but did not exceed the acute myocardial infarction diagnostic cut-off. The high sensitivity C-reactive protein value was significantly higher in groups III and II versus controls (4.92±5.12 and 2.26±2 vs. 0.85±0.48 mg/dl, $p < 0.05$ respectively). The LVMI values were significantly higher in groups III and II than in controls (159±46 and 113±35 vs. 81±14 g/m^2, respectively). There was a significant correlation between hsCRP and LVMI in group II ($r=0.49$, $p < 0.05$). Blood pressure was significantly higher in groups III and II compared to controls (129±25 and 137±19 vs. 116±7 mmHg, respectively). Patients from group III had significantly decreased haemoglobin value and increased PTH as well as phosphorus concentration compared to subject from group II and controls.

Conclusion: Chronic kidney disease is associated with accumulation of cardiovascular risk factors and increased cTnI concentration.

Key words: troponin I, chronic kidney disease, haemodialysis, LVMI, hsCRP

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Introduction

It has been shown that cardiovascular morbidity and mortality increase with decreasing glomerular filtration [1]. The observation is all the more important as the probability of death due to cardiovascular causes in patients with chronic kidney disease (CKD) receiving conservative treatment is higher than initiation of renal replacement therapy [2, 3]. That is why so many patients die before reaching indications for initiation of renal replacement therapy.

Haemodynamically significant atherosclerotic lesions in epicardial arteries are found to be present in 24% of younger patients waiting for renal transplantation, and the prevalence as well as severity of atherosclerotic lesions increase with age of patients and the duration of renal replacement therapy [4]. Factors responsible for high prevalence of cardiovascular diseases in patients with CKD include standard cardiovascular risk factors such as diabetes mellitus (DM), hypertension, hypercholesterolaemia, obesity and cigarette smoking, as well as atypical but specifically associated with CKD factors, such as anaemia, left ventricular hypertrophy (LVH), secondary hyperparathyroidism, proinflammatory states, oxidative stress, high homocysteine level and calcium-phosphate abnormalities.

Diagnosis of coronary artery disease (CAD) in patients with CKD is hampered by atypical clinical presentation. Angina can occur in young patients treated with haemodialysis, even in the absence of atherosclerotic lesions in coronary arteries, and then it is the result of impaired reactivity of coronary vessels, secondary to endothelial dysfunction and reduced cardiac microcirculation density [5]. These abnormalities, along with LV hypertrophy and fibrosis, lead to the reduction of coronary flow reserve and to an imbalance between myocardial oxygen supply and demand, and constitute cardiac X syndrome [6]. Patients with CKD more often experience silent ischaemia due to autonomic neuropathy, as well as present with atypical types of CAD [7, 8].

The course and the symptoms of CAD in patients with CKD differ from those in the general population, and the sensitivity and specificity of diagnostic tests are also lower. There have been many controversies regarding the measurement of cardiac troponins in the diagnosis of acute coronary syndromes in patients with CKD, since their concentrations tend to be non-specifically elevated in such patients. The majority of studies measuring serum troponin levels in patients with CKD dealt with troponin T (TnT) and were performed in patients receiving renal replacement therapy. Increased serum concentrations of cardiac troponin T (cTnT) in patients with CKD might result from a cross reaction of used reagents (particularly when using older assays) with TnT isoform released from skeletal muscles. Increased cTnT levels were also correlated with age of patients, the presence of multiple cardiovascular risk factors, LVH, CAD and DM, and associated with increased total and cardiovascular mortality [9–12]. Serum concentrations of cardiac troponin I (cTnI) in patients with CKD are elevated less often than cTnT, suggesting its higher specificity as a myocardial necrosis marker.

Because of many controversies regarding serum concentration of cTnI in patients with CKD and very few reports on investigations done before renal replacement therapy initiation, the current study was undertaken to evaluate cTnI concentrations in healthy volunteers as well as in patients with CKD without clinical symptoms of CAD, heart failure, or DM, receiving conservative treatment or treated with haemodialysis.

Methods

Patients

The study included patients with CKD, treated in the Nephrology Outpatient Department and Dialysis Unit of the Department of Nephrology of the University Hospital in Bydgoszcz. The exclusion criteria were as follows: age <18 years, diagnosis of DM, CAD, heart failure (NYHA class III or IV) and acute infection within 2 weeks before enrolment. Creatinine clearance (CrCl) formed was calculated according to the Cockcroft-Gault formula. Three study groups were formed:

Group I (n=10) – control group composed of healthy volunteers, 5 men and 5 women, mean age 33±4 years, without a history of hypertension or CKD, with a mean CrCl of 97.13±23.24 ml/min, not receiving any medications for any chronic condition.

Group II (n=21) – patients with CKD receiving conservative treatment, 13 men and 8 women, mean age 52±15 years, with a mean CrCl of 34.04±18.34 ml/min. Because of the limited number of patients in this study group, they were not stratified according to the CKD stage.

Group III (n=30) – patients treated with haemodialysis (HD) three times a week; there were 16 men and 14 women, at the mean age of 51±15 years, with a mean HD treatment duration of 80±73 weeks and a mean Kt/V (dialysis adequacy index) of 1.26±0.23. Residual diuresis was absent in 90% of patients and the remaining 10% of patients had diuresis of less than 200 ml/24 h.

The cause of CKD in group II was hypertension (in 60%), chronic glomerulonephritis (in 20%), pyelonephritis (in 10%), nephrolithiasis (in 5%) and ischaemic nephropathy (in 5%).

The cause of CKD in group III was chronic glomerulonephritis (in 43%), hypertension (in 12%), secondary amyloidosis (in 10%), nephrolithiasis (in 7%), polycystic kidney disease (in 7%), Wegener granulomatosis (in 7%), systemic lupus erythematosus (in 5%), Schonlein-Henoch disease (in 2%), interstitial tubulonephritis (in 2%), diathesis urica (in 2%), vesicoureteral reflux (in 2%) and trauma (in 2%).

Laboratory tests

In all patients serum concentrations of cTnI, creatinine, inorganic phosphate, haemoglobin, high sensitivity C-reactive protein (hsCRP) and parathormone (PTH) were measured. Blood samples were collected in the morning after overnight fasting. In patients from group III blood samples were collected immediately before HD, 3 days after the previous HD session. Blood samples were then centrifuged for 10 minutes at 3500 rps and supernatant frozen at –80°C until the time of biochemical measurements.

The cTnI concentration was measured using an immunoenzymatic assay with microparticles (Microparticle Enzyme Immunoassay – MEIA) and ABBOTT AxSYM immunology analyser. It uses cTnI-C complex as an antigen. The cut-off value to diagnose myocardial infarction (MI) was 0.40 ng/ml (as recommended by WHO). The maximum sensitivity of the test was 0.02 ng/ml. The value representing the 99th percentile of cTnI concentration in the healthy population is 0.04 ng/ml and was regarded in the present study as the upper normal limit.

Serum concentrations of creatinine, inorganic phosphate and haemoglobin were measured using calorimetric methods and Roche reagents with the Hitachi 912 analyser. Serum concentration of hsCRP was assessed with the Dade Behring tests in a BN TM Systems analyser. The sensitivity of this method was 0.175mg/l. Serum concentration of PTH was measured by chemiluminescent immunoassay using DiaSorin reagents in a LIASON device. The reference normal range for this method was 17.3-72.9 pg/ml.

Blood pressure measurement

Blood pressure (BP) measurements were performed with a mercury sphygmomanometer, with the cuff wrapped around the left arm, in a sitting position, after a 10-minute rest period.

Patients from group II had their BP measured twice during one visit. Fifty-seven percent of these patients were treated with angiotensin-converting enzyme inhibitors (ACE-I) or angiotensin receptor blockers (ARB), while 48% of subjects were treated with beta-blockers (BB).

Patients from group III had BP measured at the beginning of, during, and at the end of haemodialysis. The mean value of these three measurements was calculated. Thirty-seven percent of patients in this group received ACE-I or ARBs, and 27% BBs. Groups II and III did not significantly differ in terms of cardioprotective medications used (ACE-I, ARB or BB).

Echocardiography

In all patients two-dimensional echocardiography was performed using a Hewlett Packard Sonos 2000 device (Phillips Medical). Left ventricular mass (LVM) was calculated by the Devereux and Reichek formula, and adjusted for body surface area to obtain LVM index (LVMI).

Statistical analysis

The results are presented as means ± SD or numbers and percentages. The Shapiro-Wilk test was used to verify normal distribution of variables. Significance of differences between mean values was tested by Student's t-test. A Chi-square test (χ^2) was used to analyse differences of distribution in groups. The correlation between analysed variables was assessed by Pearson linear correlation index. A p value <0.05 was considered significant.

Results

The results of laboratory tests, blood pressure measurements and LVMI in each group are presented in Table I.

Serum concentration of cTnI was significantly higher in patients treated with HD than in control subjects. In 46% of patients cTnI values were found to be above the upper limit of normal, but they did not exceed the cut-off value for acute coronary syndrome diagnosis (cTnI=0.4 ng/ml). The difference between group II and controls did not reach statistical significance, most likely due to the limited number of patients

In the control group, cTnI concentration was found to be 0.01 ng/ml in one patient and 0 ng/ml in the others. No statistically significant correlations were found between cTnI and variables such as glomerular filtration rate, LVH, inflammation, anaemia or hyperphosphataemia in all study groups.

In group III, a significant negative correlation was found between the duration of renal replacement therapy and systolic ($r=-0.47$, $p<0.01$) or diastolic ($r=-0.42$, $p<0.05$) BP values.

Left ventricular mass index increased with increasing severity of CKD and showed a statistical difference between the examined groups. The LVMI was found to

Table I. Results of laboratory tests, blood pressure measurements and LVMI

Parameter	Group I n=10	Group II n=21	Group III n=30
Troponin I [ng/ml]	0.010±0.031	0.066±0.162	0.063±0.080 *
Hig-sensitivity C-reactive protein [mg/dl]	0.853±0.482	2.264±2.071 *	4.926±5.151 * #
Haemoglobin [g/dl]	13.48±0.97	12.72±1.83	9.85±1.04 * #
Parathormone [pg/ml]	not done	82±66	374±433 #
Phosphates [mmol/l]	1.08±0.20	1.24±0.32	1.91±0.56 * #
Systolic blood pressure [mmHg]	116±7	137±19 *	129±25 *
Diastolic blood pressure [mmHg]	73±6	81±11 *	76±13
Left ventricular mass index [g/m^2]	81±14	113±35 *	159±46 * #

** p <0.05 vs. group I, # p <0.05 vs. group II*

correlate with hsCRP concentration (r=0.499, p <0.05) in group II. The multiple regression analysis performed in this group revealed the presence of a correlation between LVMI (dependent variable) and hsCRP (beta=0.41), systolic BP (beta=0.31), age (beta=0.287) and haemoglobin concentration (beta=0.094) (R^2 =0.45, p<0.05).

Discussion

Our study showed that 46% of patients with CKD receiving renal replacement therapy had cTnI levels exceeding the upper limit of normal for healthy persons. However, these levels did not exceed the threshold value for the diagnosis of an acute coronary syndrome (cTnI=0.4 ng/ml). The mean cTnI concentration in patients with CKD receiving conservative treatment tended to be higher than in controls, but the difference was not significant.

Similar results were presented by Wayand et al. [13], who demonstrated elevated cTnI concentrations in 48% of haemodialysed patients with LVH but without a history of CAD. In haemodialysed patients with CAD, however, increased concentration of cTnI was found in 50% of cases [13]. Khan et al. in a group of 126 haemodialysed individuals, found cTnI values exceeding the 99th percentile in 20% of subjects [8].

In contrast, a study with 63 patients, treated with HD, without any symptoms of CAD, revealed that the concentrations of cTnI exceeded the cut-off value for diagnosis of MI (0.4 ng/ml) in 22% of examined subjects and were elevated above 0.08 ng/ml in as many as 75% of individuals [14]. The differences in the prevalence of elevated cTnI concentrations in haemodialysed patients, observed in various studies, may be due to the use of different diagnostic tests. Currently, there are many available tests to measure cTnI concentration, which vary in terms of the type of reagents and monoclonal antibodies used, various epitopes of cTnI recognised and cut-of values for the MI diagnosis [15].

Also the biochemical features of the cTnI particle might affect its detection. Cardiac troponin I is a protein with molecular mass of 24 kDa and is an inhibitory subunit of a thin filament composing a contractile unit – the troponin-tropomyosin complex. Cardiac isoform cTnI is absent in skeletal muscles [15]. The majority of cTnI is located in sarcomeres and only 3-8% is dispersed in myocyte cytosol. The consequence of acute MI is the release of cTnI binary complex (cTnI-cTnI), cTnI-TnC binary complex (cTnI-TnC) and smaller amounts of cTnT-cTnI-TnC complex (cTnT-cTnI-TnC). Only about 3-10% of cTnI is released as a free particle. Cardiac troponin I is hydrophobic and therefore it may bind with serum proteins or with the surface area of the dialyser. In the blood stream several biochemical modifications of cTnI take place, including phosphorylation, oxidation and proteolysis, potentially affecting its detection by immunoenzymatic methods. This concept was confirmed by Nakai et al. study, which demonstrated increased serum concentration of cTnI-I complex in 39.5% of patients treated with HD and the absence of free cTnI in all subjects [16]. In patients with CKD and acute MI, serum half-life of cTnI tended to be longer [17]. Haemodialysis caused an insignificant decrease of cTnT concentration and did not affect cTnI concentration [18], whereas decreased concentration of cTnI following HD was found in another study [13]. Therefore, the type of dialyser may influence the results of analysis.

Increased concentrations of cTnI were also identified by currently used immunoassays in healthy sportsmen after strenuous exercise [19]. Strenuous exercise may thus cause clinically silent cardiomyocyte damage which presumably results from increased concentrations of catecholamines. Catecholamines might be responsible

for spasm of coronary vessels in the case of endothelial dysfunction.

Endothelial dysfunction and hyperactivity of the sympathetic system are present in early stages of CKD and lead to premature occurrence of hypertension, LVH and increased myocardial oxygen demand [20]. Heart muscle damage, secondary to hyperactivity of the sympathetic system, might therefore result in increased troponin concentrations in early stages of CKD.

The current study revealed that the concentrations of cTnI in patients with CKD receiving conservative treatment (group II) tended to be higher than in controls. There were too few subjects in group II and so the concentrations of cTnI in patients with given stages of CKD were not evaluated. Abbas et al. [21] showed, however, that cTnI and cTnT concentrations were elevated in patients with stage 3 of CKD with no symptoms of CAD. A correlation between increased concentrations of cTnI and LVMI, PTH, age and anaemia was also demonstrated in this study. Moreover, the incidence of increased cTnI concentration (above the 99th percentile) was noted to be higher with decreasing values of glomerular filtration rate (GFR). The concentration of cTnI was found to be elevated in 12% of patients with stage 3 or 4 of CKD and in 26% of patients with stage 5, receiving conservative treatment. The study of Lamb et al. [22] of 222 patients with stage 3-5 of CKD, receiving conservative therapy, revealed that the concentration of cTnI, measured by ultra-sensitive assay, was increased above the 99th percentile in 33% of subjects. Contrary to the results presented by Abbas et al., Lamb et al. did not demonstrate a higher incidence of increased cTnI concentrations in patients with more seriously depressed GFR. Similarly to Abbas et al., however, a significant correlation between cTnI and age, LVMI, PTH and CRP was demonstrated.

In our study we did not observe a direct correlation between cTnI and LVMI or PTH, but a correlation between hsCRP and LVMI was found. Premature occurrence of atherosclerosis in patients with CKD is well known [23]. Atherosclerotic lesions in patients with CKD tend to be calcified. Also, increased thickness of the intima-media complex in big vessels was found. All these changes lead to decreased vessel relaxation, increased systolic BP and pulse pressure as well as to LVH. A correlation between hsCRP and LVMI, found in group II, may reflect LV pressure overload, secondary to increased thickness of arteries and to hypertension.

Studies conducted so far have shown the presence of an inflammatory state in early stages of CKD (mean CrCl 36.3±23.1 ml/min) [24]. Our findings are similar, showing elevated concentrations of hsCRP in group II (CrCl = 34.04±18.34 ml/min) and even higher in group III.

Wong et al. [25] found significantly higher concentrations of acute-phase proteins and cTnT in patients receiving renal replacement therapy than in those on conservative treatment. The inflammatory state present in CKD may thus be a factor responsible for increased cardiovascular mortality observed in these patients and this observation is confirmed by the study of Kanwar et al. [26]. They showed in a group of 58 patients treated with HD without a history of CAD, that a concentration of hsCRP ≥3 mg/l, independently of troponin I concentrations, was associated with increased mortality during a 27-month follow-up period. In the study of Apple et al. of 399 patients with stage 5 of CKD, treated with renal replacement therapy, cTnI concentration was found to be elevated in 19% of subjects. The concentration of hsCRP, cTnT and cTnI were independent predictors of a 2-year survival. Two--year mortality was 6% if the concentrations of hsCRP, cTnI and cTnT were all within the normal ranges, 44% if the concentrations of hsCRP and cTNT were increased and cTnI within the normal range, and as high as 47-61%, depending on the assay used (Beckman or Dade), if only cTnI concentrations were found to be elevated [27]. It has also been demonstrated that increased concentrations of hsCRP in patients treated with HD may precede or even predict myocardial hypertrophy [28]. Furthermore, hsCRP is associated with LV systolic function and might be a useful marker of LV remodelling [29, 30]. Such an association can also appear in the course of conservative treatment of CKD. This may be reflected by the correlation between CRP and LVMI in group II observed in our study.

Our study demonstrated that LVMI increased with the progression of CKD. In patients from group II, LVM, despite values being lower than required for the diagnosis of LV hypertrophy (defined as LVMI >131 g/m^2 for men and >100 g/m^2 for women) [31], was significantly higher than in controls. Increased concentrations of hsCRP and elevated systolic BP in patients from group II, in comparison to controls, may be responsible for increased LVMI in these patients. Group III presented with considerable LV hypertrophy as compared with the other groups. It may be the consequence of coexistence of several abnormalities in these patients (fluid overload, hypertension, anaemia, secondary hyperparathyroidism, hyperactivity of the sympathetic nervous system and rennin-angiotensin-aldosterone system). Systolic BP was considerably higher in patients with CKD, in both groups II and III, than in the control group. A negative correlation between both systolic and diastolic BP and the duration of renal replacement therapy was found in group III. Significant

differences in diastolic BP values were found only between group II and group I. Thus, the role of pressure overload in group II and predominant volume overload in group III seems clear.

The reason for a non-specific increase of cTnI concentration in patients with CKD remains unknown. There are several hypotheses such as microdamage of the myocardium, inflammatory state, cardiotoxic effects of electrolyte abnormalities and osmolarity changes, increased afterload, increased myocardial wall tension because of fluid overload and heart damage independent from ischaemia, caused by abnormalities of calcium-phosphate balance [32]. Some studies suggest that cTnI and cTnT fragments might be released from cardiomyocytes also when rupture of a cell membrane does not occur. The increase of troponin T concentration in the group of 615 patients was caused by acute coronary syndrome in 53% of cases and in the remaining subjects a non--specific increase was recognised. The predictive value of the test in acute coronary syndrome diagnosis was quite low in patients with borderline levels of troponin T concentrations, older than 70 years and with chronic renal failure [33]. Cardiac Troponin I may thus be a marker of increased stretching and tension of the myocardium, both acute – as in pulmonary thromboembolism, septic shock and acute heart failure – and chronic – as in chronic heart failure and liver or renal disease. Increased cardiac wall tension and inappropriate adaptation to such stress may lead to myofibril damage and cardiomyocyte lysis, and result in the release of cTnI or antigen fragments of this protein, which are detected by currently available assays.

It has been demonstrated that LV hypertrophy correlates with increased serum concentrations of cTnT and cTnI, as well as with decreased concentrations in cardiomyocytes. Relative content of troponin isoforms changes with myocardial hypertrophy; thus it is possible that remodelling and heart failure may trigger the mechanisms responsible for the release of troponin to the plasma and reflect the changes in protein metabolism within cardiomyocytes [34, 35]. This fact has been confirmed by Sharma et al. [36] in a prospective 2-year observation of 126 patients with a mean creatinine concentration of 608 µmol/l, selected for renal transplantation. Fifty-five percent of these patients were treated with dialysis; the remainder received conservative therapy. A positive correlation between increased concentration of troponin T and LV dilatation, LVM, impaired systolic function and increased LV filling pressure was demonstrated. Furthermore, a positive correlation was found between concentration of cTnT and N-terminal brain-type natriuretic peptide prohormone.

The abnormalities of morphology and function of LV in patients with CKD, particularly in patients treated with renal replacement therapy, may lead to increase of plasma troponin concentration. It should also be pointed out that increased concentrations of troponin I in dialysed patients, independently of the clinical manifestations, are associated with increased 30-day incidence of adverse cardiac events such as death from cardiovascular causes, MI, heart failure de novo and the need for myocardial revascularisation [37].

Summary

Chronic kidney disease is associated with accumulation of cardiovascular risk factors and increased cTnI concentrations. As suggested by available studies, the measurement of cTnI concentration is a more sensitive indicator of myocardial damage caused by various factors than any other biomarker. The assessment of cTnI concentration is thus the first-line evaluation when diagnosis of acute coronary syndrome is considered in patients with CKD [38]. Routine measurements of initial cTnI concentration in patients in whom renal replacement therapy is introduced together with the assessment of cardiovascular risk factors in early stages of CKD may help to select the group of asymptomatic patients who require aggressive primary prevention.

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Stężenie troponiny I u chorych z przewlekłą chorobą nerek leczonych zachowawczo lub hemodializami

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Streszczenie

Wstęp: Frakcja sercowa troponiny I (cTnI) jest czułym i swoistym markerem uszkodzenia mięśnia sercowego w populacji ogólnej. W grupie pacjentów z przewlekłą chorobą nerek (CKD) stężenie cTnI może być podwyższone mimo niewystępowania innych objawów ostrego uszkodzenia mięśnia sercowego. Przyczyna niespecyficznego wzrostu cTnI w CKD wciąż nie jest jasna. Hipotetycznie tłumaczy się go mikrouszkodzeniem miokardium, stanem zapalnym, kardiotoksycznością spowodowaną zaburzeniami jonowymi i zmianą osmolarności, zwiększonym obciążeniem następczym serca, zwiększonym napięciem ściany serca z powodu przeładowania płynami, a także uszkodzeniem serca niezależnym od niedokrwienia, a spowodowanym zaburzeniami gospodarki wapniowo-fosforanowej.

Cel: Porównanie stężenia cTnI w grupie pacjentów z CKD, bez objawów choroby wieńcowej, cukrzycy i przewlekłej niewydolności serca w III/IV klasie wg NYHA, z grupą osób zdrowych oraz ocena czynników ryzyka sercowo-naczyniowego w tych grupach.

Metodyka: Wyróżniono trzy grupy badane: grupa I (5 kobiet, 5 mężczyzn, w średnim wieku 32±4 lata) – zdrowi, młodzi ochotnicy bez CKD z klirensem kreatyniny (CrCl) 97,13±23,24 ml/min; grupa II (8 kobiet, 13 mężczyzn, średni wiek 51±15 lat) – pacjenci z CKD w stadium 3–5 ze średnim CrCl 34,04±18,34 ml/min; grupa III (14 kobiet, 16 mężczyzn, średni wiek 50±14 lat) – pacjenci leczeni hemodializami. Krew do badania pobierano na czczo. W grupie III krew pobierano bezpośrednio przed zabiegiem hemodializy. Stężenie cTnI było oznaczane na analizatorze immunologicznym AxSYM firmy ABBOTT. Oznaczono również stężenie białka C-reaktywnego (hsCRP), stężenie hemoglobiny, parathormonu i fosforanów. Ciśnienie tętnicze mierzono sfigmomanometrem rtęciowym. W grupie pacjentów z grupy II dokonano dwóch pomiarów ciśnienia tętniczego w czasie jednej wizyty. W grupie III wartości ciśnienia mierzono na początku, w połowie i na koniec zabiegu hemodializy. Ze wszystkich pomiarów wyliczono wartość średnią. Wykonano dwuwymiarowe badanie echokardiograficzne, wyliczono indeks masy lewej komory (LVMI).

Wyniki: Stężenie cTnI było istotnie statystycznie podwyższone w grupie III i nieistotnie w grupie II w porównaniu z kontrolną (odpowiednio 0,063±0,08 i 0,066±0,162 do 0,01±0,03 ng/ml). U 46% pacjentów hemodializowanych stężenie cTnI znajdowało się powyżej wartości 99. percentyla w zupełnie zdrowej populacji. W żadnym przypadku nie przekroczyło jednak wartości odcięcia dla ostrego zawału serca. Stężenie hsCRP narastało wraz z pogarszaniem się funkcji nerek i różniło istotnie statystycznie między badanymi grupami – wartości wynosiły odpowiednio 4,92±5,12 mg/dl w grupie III; 2,26±2,0 mg/dl w grupie II i 0,85±0,48 mg/dl w grupie I. Wskaźnik LVMI różnił się istotnie statystycznie między badanymi grupami i wykazywał coraz wyższe wartości wraz z progresją CKD. Wartości LVMI w grupie III, II i I wynosiły odpowiednio 159±46 g/m^2, 113±35 g/m^2 i 81±14 g/m^2. W grupie II wykazano obecność korelacji pomiędzy hsCRP a LVMI ($r=0,49$, $p<0,05$). Wartości skurczowego ciśnienia tętniczego w grupie III wynosiły 129±25, a w grupie II 137±19 mmHg i nie różniły się istotnie pomiędzy sobą. Były istotnie wyższe w porównaniu z grupą I – 116±7 mmHg. W grupie III wykazano istotnie niższe stężenie hemoglobiny oraz podwyższone stężenie PTH i fosforanu w porównaniu z grupą II i kontrolną.

Wnioski: Przewlekła choroba nerek jest skojarzona z nagromadzeniem licznych czynników ryzyka sercowo-naczyniowego oraz podwyższonym stężeniem cTnI.

Słowa kluczowe: troponina I, przewlekła choroba nerek, hemodializoterapia, LVMI, hsCRP

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