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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

High sensitive troponin-I in patients with slow coronary flow pattern

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Abstract

Hypothesis: We examined the hypothesis that a specific myocardial injury marker, namely high sensitive cardiac troponin-I (HsTn-I), is elevated in patients with slow coronary flow (SCF) pattern.

Aim: To examine the above hypothesis by studying a group of patients who had undergone coronary angiography for the detection of their chest pain aetiology with SCF pattern despite an angiographically normal coronary arteriogram.

Methods: We evaluated and performed coronary angiography (CAG) of 97 patients with chest discomfort. The indication for CAG was at least Canada class 3 angina and/or proven myocardial ischaemia according to noninvasive diagnostic tests. We further divided patients into three subgroups according to CAG images and compared HsTn-I plasma levels in 39 patients with SCF pattern, 28 patients with coronary artery disease (CAD), and 30 patients with normal coronary arteries. We researched the association between qualitative HsTn-I positivity and demographic features including cardiovascular risk factors, inflammation markers and TIMI frame count for each of the epicardial coronary arteries.

Results: TIMI frame count for each epicardial coronary artery was significantly higher in patients with SCF pattern than in patients with CAD and normal coronary arteries ($p < 0.001$). HsTn-I positivity was not statistically different between patients with SCF pattern and normal coronary arteries ($p = 512$), but it was significantly higher in the CAD group than the other two group of patients ($p < 0.001$).

Conclusions: In patients with SCF, HsTn-I may be detectable, but it is not elevated as in patients with normal coronary arteries.

Key words: high sensitive troponin-I, slow coronary flow, coronary artery disease

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INTRODUCTION

Slow coronary flow (SCF) is a well-known clinical entity characterised by delayed opacification of coronary arteries in the presence of normal coronary arteries [1]. Although the definite mechanism is not fully understood yet, since its first definition by Tambe et al. [1], microvascular dysfunction has been thought to be the main pathophysiologic mechanism. Recent studies have revealed that endothelial dysfunction, diffuse atherosclerosis, and inflammation might play important roles in the pathogenesis of SCF [2–6]. Also SCF is observed in some patients presenting with acute coronary syndrome (ACS) [7–9]. An abnormal SCF pattern in the coronary artery can cause thrombus formation, distal embolisation and myocardial infarction (MI) [10].

Cardiac troponins are biomarkers of myocardial injury and even mild elevations are associated with increased mortality. High sensitive troponin (HsTn) assays that can measure troponin concentrations one tenth the size of those measureable with conventional assays have been developed in recent years [11].

Our aim was to investigate the clinical value of HsTn-I in SCF and its possible association with cardiovascular risk factors and inflammation markers.

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METHODS

Patient selection

We evaluated 97 patients with symptoms of chest discomfort who were referred from our cardiology outpatient clinic for coronary angiography (CAG) because of suspected coronary artery disease (CAD). We included patients older than 18 years old and who had at least Canada class 3 angina and/or proven myocardial ischaemia according to noninvasive diagnostic tests. All patients gave written informed consent and the study complied with the Declaration of Helsinki and was approved by the Institutional Review Board and Ethics Commitee at Mehmet Akif Ersoy Education and Research Hospital. We excluded patients with a history of acute MI, serious valvular heart disease, rhythm disturbances, heart failure, inflammatory diseases, peripheral arterial diseases, renal failure, liver failure, pregnant patients, patients with ACS and left ventricular hypertrophy. Also we excluded patients subjected to air embolisation during CAG or complicated during catheterisation.

We divided patients into three subgroups according to the CAG findings: Group 1 consisted of patients with SCF pattern; Group 2 consisted of patients with at least 50% lumen narrowing in at least one epicardial coronary artery (the CAD group); and Group 3 consisted of patients with normal coronary arteries (the control group).

Blood samples and analysis

Blood samples were collected from the patients after a 12-h overnight fast. Blood samples for HsTn-I assay were drawn from peripheral veins just after CAG. Venous blood samples were centrifuged at 3,000 rpm for 10 min to collect serum samples. Biochemical tests other than HsTn-I were measured from serum samples with Cobas-C 501 (Roche, USA) biochemical analyser using Roche kits. HsTn-I was measured from venous blood samples collected in K3 EDTA tubes using Triage device (Biosite Incorporated, USA) by the immunofluorescent method using fluorescent antibody conjugates. The analytic sensitivity of HsTn-I was accepted as 0.01 ng/mL for the 95th percentile as recommended by the manufacturer. Values equal to or above 0.01 ng/mL were accepted as positive results.

Coronary arteriography

Coronary arteriography was performed with a femoral approach using Judkins catheters. Coronary arteries were visualised in left and right oblique planes, and cranial and caudal angles. Left ventriculography was performed in left and right anterior oblique views. Injection of contrast medium (Iopromide, Ultravist-370; Schering AG, Berlin, Germany) was carried out by an automatic injector at a speed of 3–4 mL/s for the left coronary artery, and 2–3 mL/s for the right coronary artery (RCA). Arteriographies were recorded at a speed of 30 frames/s.

TIMI frame count and definition of slow coronary flow

Coronary blood flow was measured quantitatively using the Thrombolysis in Myocardial Infarction (TIMI) frame count (TFC) which was derived from the number of cine-frames recorded from the first entrance of contrast to its arrival at the distal end of the left anterior descending artery (LAD), or the circumflex artery (Cx), or the RCA. The TFCs for the LAD were divided by 1.7 to calculate the corrected TFC because the normal frame counts for the LAD artery are 1.7 times greater than the mean for the left Cx and RCA as described earlier. Patients with a corrected TFC greater than two standard deviations from the normal range for the particular vessel were considered as having SCF pattern, while those whose corrected TFC fell within two standard deviations were considered as having normal coronary flow. The mean corrected TFC was further calculated by averaging the sum of the corrected TFCs for each coronary artery [12]. TFCs were evaluated by two experienced observers blinded to the study design.

Statistical analysis

Statistical analyses were performed using SPSS software version 17. The variables were investigated using visual (histograms, probability plots) and analytical methods (Kolmogorov-Smirnov/Shapiro-Wilk's test) to determine whether or not they were normally distributed. Descriptive analyses were presented as mean ± standard deviation (SD) and categorical variables were expressed as percentages. Groups were compared with the Kruskal-Wallis test and χ^2 test. The Mann-Whitney U test was performed to test the significance of pairwise differences using Bonferroni correction to adjust for multiple comparisons. The inter-rater agreement between the two observers in determining the TFC was investigated using the Kappa test. An overall 5% type-I error level was deemed to confer statistical significance.

RESULTS

Clinical features, biochemical analysis and TFC of all three groups are set out in Table 1. Age, sex, body mass index, smoking, hypertension, diabetes, blood pressure, heart rate and biochemical parameters were not statistically different among groups. HsTn-I positivity was not statistically different between patients with SCF pattern and normal coronary arteries ($p = 512$), but it was significantly higher in the CAD group than the other two groups of patients ($p < 0.001$) (Fig. 1). Our TFC results showed excellent agreement between independent observers (Kappa = 0.89).

DISCUSSION

The major findings of our study were that HsTn-I levels showing myocardial damage were detectable in patients with CAD, and that in SCF patients HsTn-I levels were not statistically different from normal individuals.

Table 1. Demographic features, biochemical markers and TIMI frame counts of the groups

	Group 1 (n = 39)	Group 2 (n = 28)	Group 3 (n = 30)	P
Age [years]	48.5 ± 10.3	50.0 ± 6.1	49.3 ± 9.1	0.684
Sex (male)	33 (84.6%)	21 (75%)	18 (60%)	0.068
Body mass index [kg/m^2]	29.1 ± 3.2	28.6 ± 3.9	28.4 ± 3.5	0.736
Diastolic pressure [mm Hg]	73.5±7.1	77.0 ± 8.1	71.9 ± 7.4	0.111
Systolic pressure [mm Hg]	123.0 ± 9.1	126.2 ± 9.0	120.8 ± 10.8	0.218
Heart rate [bpm]	79.1±8.1	75.6±11.3	75.9 ± 8.0	0.238
Smoking	17 (43.6%)	8 (28.6%)	12 (40%)	0.445
Diabetes mellitus	3 (7.7%)	7 (25%)	3 (10%)	0.098
Hypertension	8 (20.5%)	12 (42.9%)	8 (26.8%)	0.131
Haematocrit	42.8 ± 3.7	42.3 ± 5.1	41.2 ± 5.3	0.658
Glucose [mg/dL]	115.3 ± 36.7	111.4 ± 40.0	103.1 ± 13.4	0.633
Creatinine [mg/dL]	0.8 ± 0.2	0.9 ± 0.2	0.8 ± 0.1	0.199
Total cholesterol [mg/dL]	194.5 ± 41.9	214.3 ± 46.1	193.5 ± 40.7	0.138
LDL [mg/dL]	122.8 ± 29.3	141.3 ± 42.7	121.5 ± 33.5	0.117
HDL [mg/dL]	42.2 ± 12.4	39.4 ± 7.3	45.0 ± 12.4	0.160
HsCRP [mg/dL]	3.5 ± 2.8	4.0 ± 2.6	3.6 ± 2.9	0.526
Uric acid [mg/dL]	5.6 ± 1.6	5.5 ± 1.9	5.1 ± 1.0	0.671
TIMI frame count:				
LAD	36.5 ± 16.7	22.3 ± 8.3	19.8 ± 5.5	< 0.001
CX	35.7 ± 11.7	23.1 ± 3.7	20.6 ± 5.9	< 0.001
RCA	37.6 ± 13.9	21.9 ± 4.2	19.6 ± 5.5	< 0.001
Mean	36.6 ± 10.6	22.4 ± 4.0	20.0 ± 4.1	< 0.001
HsTn-I positive	5 (12.8%)	20 (71.4%)	3 (10%)	< 0.001*

*Chi-square test; LDL — low density lipoprotein; HDL — high density lipoprotein; HsCRP — high sensitive C-reactive protein; LAD — left anterior descending artery; Cx — circumferential artery; RCA — right coronary artery; HsTn-I — high sensitive troponin-I

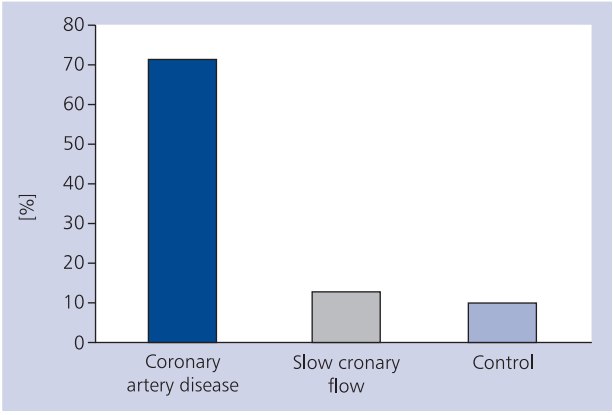

Figure 1. High sensitive troponin-I positivity accordings to groups

Despite all efforts, it remains difficult to determine the group of patients at risk for acute cardiovascular events. Cardiac troponins, simple and cost-effective biomarkers of myocardial injury that are parts of the cardiomyocyte contractile apparatus, are important in this respect [11]. The main concern regarding conventional troponin measurements is the deficiency of sensitivity in the first few hours of acute MI because of late transition to circulation [13]. However, newly developed HsTn assays will improve the risk classifications used in everyday practice because they are able to detect 10-fold lower concentrations than conventional assays and are markers of subclinical myocardial damage and cardiovascular event risk [14, 15].

The only limitation of HsTn assays is their slightly lower specificity than conventional assays [15]. High sensitive cardiac troponins are sometimes detected in healthy adults. The prevelance of HsTn-T in the general population is approximately 0.7% and it can be related to many different conditions such as advanced age, being an athlete, left venticular hypertrophy, left ventricular dysfunction, renal failure or diabetes [16]. Therefore clinicians worried about MI misdiagnosis due to lower diagnostic cardiac troponin thresholds should keep in mind that slight increases in HsTn levels may be the result of the other conditions mentioned above [13, 15]. Therefore cardiac troponins have to be read as quantitative variables.

'Detectable' HsTn should be distinguished from 'elevated' HsTn because the diagnosis is highly dependent to the absolute level. Very low concentrations can be detected in healthy people [13]. Live cardiomyocytes sometimes release troponin as intact proteins without necrosis due to integrin-mediated stretch-related mechanism [14]. The differential diagnosis of slight increases should be made, and higher levels and/or rises within short periods should be ascribed to more serious conditions such as acute MI [13].

Conventional cardiac troponin assays predict short- and long-term outcomes poorly. This observation comes from the results of clinical trials that involved homogeneous high risk patient groups [15]. On the other hand, the clinical importance of slight HsTn elevations in patients with stable CAD was not well known until recently. However several studies have revealed their strong and graded relation with adverse cardiovascular outcome and death [13]. Ndrepepa et al. [11] investigated the prognostic value of HsTn in patients with stable angina pectoris and reported HsTn-T as strong predictors of all-cause and cardiac mortality [11]. Mingels et al. [17] reported HsTn-T as a useful prognostic marker in patients with chest pain and that it is associated with CAD extent assessed by coronary computed tomography-angiography and coronary artery calcium scoring. Ang et al. [18] evaluated the prognostic value of HsTn-T in patients with recent ACS. They measured HsTn-T levels once, seven weeks after the event, and showed that HsTn-T may predict adverse clinical outcomes, left ventricular dysfunction and left ventricular hypertrophy independent of cardiovascular risk factors, increased B-type natriuretic peptide levels, and echocardiographic left ventricular dysfunction.

Low-level troponin release in patients with stable CAD may be because of transient, silent episodes of ischaemia in small vessels causing a mismatch between metabolic demand and supply [16, 17]. Another potential mechanism is the movement of small localised thrombi causing micro-injuries in small vessels [17]. Other possible explanations of detectable HsTn concentrations are: coronary vasospasm, direct coronary injury, chest trauma, intense exercise, inflammatory conditions such as pericarditis, amyloidosis, cardiomyocyte apoptosis, decreased renal clearance and/or increased myocardial strain due to increased pressure or volume overload [16, 18].

SCF is a coronary microvascular disease which may be observed in patients presenting with acute MI, stable angina, unstable angina and hypotension, although rarely. Shirani et al. [19] found the incidence of SCF in patients undergoing selective coronary angiography to be 1%. Gökçe et al. [20] reported SCF incidence in patients with angina of 7%.

The pathophysiology of delayed contrast passage in coronary arteries without obstructive lumen narrowing seen in SCF has not been fully understood yet [12]. In 1972, Tambe et al. [1] declared that the slow flow of the contrast was because of an abnormal increase in small vessel resistance. Mangieri et al. [21] studied endomyocardial biopsies of patients with SCF. They reported vessel wall thickening causing lumen narrowing, mitochondiral abnormalities and decrease in glycogen content in this group of patients. Cannon et al. [22] mentioned excessive sensitisation for vasoconstructive impulses and a decrease in vasodilatator capacity in the microvascular network. Mosseri et al. [23] suggested that SCF may be cause small coronary artery occlusive disease which can lead to the early stages of atherosclerosis. Friedman [24] discussed a neurobiological effect on the pathophysiology of SCF.

In our study, we found statistically significant HsTn-I elevation in stable CAD patients, but in the SCF patient group HsTn-I levels were not different from normal individuals. SCF may reflect impaired coronary vasomotor reflex, but does not cause overt myocardial injury in resting patients. SCF patients may not respond adequately to the conditions necessitating high coronary flow demand. Yaymacı et al. [25] showed that atrial pacing or isoproterenol infusion causes increased myocardial lactate extraction, and that atrial pacing leads to decreased coronary sinus oxygen saturation and/or dipyridamole infusion triggering an abnormal increase in coronary flow.

Limitations of the study

Our study was single-centred and had a relatively small sample size, making the power of the research limited. Furthermore, our study provided no information about long-term outcomes. Another limitation was that we did not evaluate myocardial structure or function and HsTn-I assays were studied once in a resting state.

CONCLUSIONS

HsTn-I levels are detectable in patients with SCF, but not different from normal individuals. Noninvasive diagnosis and management of SCF needs more sophisticated diagnostic tools.

Conflict of interest: *none declared*

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Stężenie troponiny I oznaczane metodą wysokoczułą u chorych z wolnym przepływem wieńcowym

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Streszczenie

Wstęp: Autorzy sprawdzili hipotezę, że swoisty wskaźnik uszkodzenia mięśnia sercowego, stężenie sercowej troponiny I oznaczone metodą wysokoczułą (HsTn-I), jest podwyższone u chorych z wolnym przepływem wieńcowym.

Cel: Zbadanie powyższej hipotezy w grupie chorych poddanych koronarografii w celu ustalenia przyczyny bólu w klatce piersiowej, u których przy prawidłowym obrazie angiograficznym naczyń wieńcowych współistniał wolny przepływ wieńcowy.

Metody: U 97 chorych z objawami dyskomfortu w klatce piersiowej przeprowadzono ocenę kliniczną i koronarografię. Wskazaniami do koronarografii były: co najmniej 3. stopień w skali nasilenia objawów dławicowych Kanadyjskiego Towarzystwa Kardiologicznego (CCS) i/lub niedokrwienie mięśnia sercowego potwierdzone w badaniach nieinwazyjnych. Następnie podzielono pacjentów na 3 grupy w zależności od wyników koronarografii i porównano stężenia HsTn-I w osoczu u 39 chorych z wolnym przepływem wieńcowym, 28 pacjentów z chorobą wieńcową (CAD) i 30 osób z prawidłowym obrazem tętnic wieńcowych. Przeanalizowano zależności między dodatnim wynikiem oznaczenia HsTn-I (zmienna jakościowa) a parametrami demograficznymi obejmującymi m.in. czynniki ryzyka sercowo-naczyniowego, wskaźniki zapalenia i ocenę przepływu w skali TIMI dla każdej tętnicy nasierdziowej.

Wyniki: U pacjentów z wolnym przepływem wieńcowym punktowa ocena w skali TIMI była istotnie wyższa niż u osób z chorobą wieńcową i z prawidłowym obrazem tętnic wieńcowych w przypadku każdej tętnicy nasierdziowej ($p < 0,001$). Nie było statystycznie istotnych różnic w zakresie częstości dodatnich wyników oznaczenia HsTn-I miedzy pacjentami z wolnym przepływem wieńcowym a osobami z prawidłowym obrazem tętnic wieńcowych ($p = 512$). Dodatni wynik oznaczenia HsTn-I stwierdzano natomiast istotnie częściej w grupie osób z CAD niż w pozostałych grupach pacjentów ($p < 0,001$).

Wnioski: U chorych z wolnym przepływem wieńcowym HsTn-I może być wykrywalna, ale jej stężenie może się utrzymywać w granicach normy, podobnie jak u osób z prawidłowymi tętnicami wieńcowymi.

Słowa kluczowe: troponina I oznaczana metodą wysokoczułą, wolny przepływ wieńcowy, choroba wieńcowa

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