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Periprocedural myocardial damage during percutaneous coronary intervention: a point-of-care platelet testing and intravascular ultrasound/virtual histology study

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Abstract

Background: Recent studies have implied that platelet reactivity as well as certain lesion morphology may be linked to myocardial injury during percutaneous coronary intervention (PCI). However, to date the abovementioned features have not been investigated simultaneously in one population.

Aim: To determine if and how high on-treatment platelet reactivity, different lesion morphology, and plaque components are associated with increased risk of periprocedural myocardial injury in patients referred for elective coronary stenting.

Methods: Sixty patients pretreated with aspirin and clopidogrel and undergoing elective PCI with stent(s) implantation were included. On-treatment platelet reactivity was measured with VerifyNow Aspirin and P_2Y_{12} assays (Accumetrics, USA) before PCI. Grey-scale intravascular ultrasound (IVUS) and virtual histology were performed before stent(s) implantation (Volcano, USA). Two levels of myocardial injury were considered: any elevation of troponin I (periprocedural myocardial damage, PMD) and/or > 3 times the upper normal limit (periprocedural myocardial infarction, PMI).

Results: By receiver-operating characteristics analysis, the following factors, ranked from strongest to weakest, were able to distinguish between patients with and without PMD: remodelling index (RI), fibrous tissue, fibro-fatty tissue volume (FFT), plaque and media cross-sectional area, and external elastic membrane cross-sectional area (EEM CSA). Only platelet count and RI could differentiate patients with and without PMI. PMD as well as PMI could not be predicted either by VerifyNow Aspirin or P_2Y_{12} assay. Likewise, there was no association between necrotic core volume and PMD or PMI. In logistic regression analysis, after adjusting for possible clinical and procedural confounding factors, only EEM CSA > 14.6 mm^2 (OR 23.7, 95% CI 1.9–302, p = 0.015), RI > 1.044 (OR 12.3, 95% CI 1.2–121.9, p = 0.032) and FFT > 11.2 mm^3 (OR 13.6, 95% CI 1.1–160.9, p = 0.038) were independent predictors of PMD. Only RI > 1.044 was identified as an independent predictor of PMI (OR 7.5, 95% CI 1.92–29.6, p = 0.004).

Conclusions: Greater total vessel area, positive remodelling at the lesion site, and high volume of FFT in the coronary plaque are independently associated with increased risk of myocardial injury. Only positive RI was an independent predictor of PMI. Simple lesion morphology, rather than more complex VH-IVUS analysis or platelet reactivity, seems to predict myocardial injury after elective PCI.

Key words: periprocedural myocardial injury, stable coronary artery disease, IVUS, VH-IVUS, platelet reactivity

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INTRODUCTION

Troponin elevation after non-emergent coronary interventions is indicative of an increase in long-term all-cause mortality as well as composite adverse events of all-cause mortality and myocardial infarction [1–4]. Therefore, periprocedural myocardial injury has been widely used in numerous clinical trials as a surrogate ischaemic endpoint.

Dual antiplatelet therapy with aspirin (ASA) and clopidogrel is the currently recommended standard therapy for the prevention of thrombotic complications after percutaneous coronary intervention (PCI) [5]. However, it has been proved that the antiplatelet effect of a fixed dose of ASA or clopidogrel is not uniform in all patients and that a substantial interindividual variability in platelet inhibition exists [6, 7]. To date, there is a large body of evidence that high on-treatment platelet reactivity under ASA or clopidogrel therapy assessed with aggregometry as well as point-of-care tests is linked to an increased risk of subsequent cardiovascular events [8, 9]. Alternatively, recent studies with the use of grey scale intravascular ultrasound (IVUS) as well as virtual histology (VH-IVUS) have implied that certain morphology of the lesion treated with coronary stenting can also increase the risk of distal embolisation detected angiographically or by troponin rise [10–12].

However, to date, the influence of platelet reactivity and lesion characteristics have not been investigated simultaneously in one population. Therefore, in the present study we sought to determine if and how high on-treatment platelet reactivity, different lesion morphology, and plaque components are associated with increased risk of periprocedural myocardial injury in patients referred for elective coronary stenting.

METHODS

Study design, inclusion and exclusion criteria

This study was made as a prospective observational trial. Patients > 18 years old with stable coronary artery disease (CAD) referred to elective PCI were included. Main exclusion criteria were: elevated cardiac markers at baseline, unstable CAD, NYHA class III or IV heart failure, anaemia with haemoglobin concentration < 10 g/dL, platelet count outside 100–450 × $10^3/\mu$L range, haemodynamic instability during procedure, chronic total occlusion of the artery treated, and lesion located at the bifurcation. The local medical ethics committee approved the study design and all patients gave informed consent. Clinical information regarding medical history, comorbidities and pharmacotherapy was obtained from each patient on the day of admission.

Antiplatelet therapy

Most patients received ASA and clopidogrel chronically, each 75 mg daily, for a period of at least seven days. In ASA or clopidogrel-naïve patients, loading doses of 300 mg ASA or 600 mg clopidogrel were administered at least six hours before PCI. Glycoprotein IIb/IIIa inhibitors were not used.

Percutaneous coronary intervention

PCI was performed by two experienced operators. Vascular approach, procedure technique, and devices used for procedure were left to the discretion of the physician and were consistent with typical practice in our department.

Blood tests

Cardiac troponin, complete blood count and creatinine concentration were evaluated at baseline. Serial troponin I measurements were made six, 12 and 24 hours after procedure in the central hospital laboratory using heterogeneous immunoassay module (Flex® reagent cartridge) with Dimension® device (Siemens); analytical sensitivity 0.04 ng/mL, upper normal limit 0.1 ng/mL. Platelet reactivity was assessed before PCI, from arterial blood sample with the use of point-of-care VerifyNow™ System (Accumetrics, USA), but at least six hours after clopidogrel and/or ASA loading dose administration and was reported as P_2Y_{12} reaction units (PRU) for clopidogrel and ASA reaction units (ARU) for ASA. High on-treatment platelet reactivity was defined according to previously described cut-off values (PRU > 235 and/or ARU > 550).

IVUS and VH-IVUS

Grey-scale IVUS and VH-IVUS examination were preceded by intracoronary administration of 300 μg nitroglycerine and performed before lesion predilatation and after PCI. A 20 MHz, 3.2-French IVUS imaging catheter (Eagle Eye®, In-Vision Gold/Platinum, Volcano Corp., USA) was advanced > 10 mm distally to the lesion. Automated pullback was used at the speed of 0.5 mm/s drawing back the catheter to > 10 mm proximally to the lesion. IVUS and VH-IVUS quantitative analysis were performed across the entire target lesion. Cross-sectional measurements at minimal lumen, as well as at proximal and distal reference sites, were performed. External elastic membrane (EEM) cross-sectional area (CSA) and lumen CSA were measured. Plaque plus media (P&M) was calculated as EEM CSA minus lumen CSA; plaque burden (PB) was defined as a ratio of P&M and EEM CSA multiplied by 100. Proximal and distal references were single slices with maximum lumen CSA within 10 mm proximally and distally to the lesion, but before any significant side-branches. Remodelling index (RI) was defined as EEM CSA in minimal lumen site divided by the mean of EEM CSA in proximal and distal reference. All definitions were based on the American College of Cardiology Clinical Expert Consensus Document on Standards for Acquisition, Measurement and Reporting of Intravascular Ultrasound Studies [13]. By VH-IVUS analysis, four major plaque components were identified i.e. fibrous tissue (FT), fibro-fatty tissue (FFT), dense calcium, and necrotic core (NC) and were reported as the total volume and percentage of each component. The presence of thin-cap fibroatheroma (TCFA) was defined as NC ≥ 10% of plaque area in at least three consecutive frames without overlying FT in the presence of ≥ 40% plaque burden [14].

Study endpoints

Any troponin I elevation at any time-point after PCI was considered as periprocedural myocardial damage (PMD). Periprocedural myocardial infarction (PMI) was defined as troponin I elevation at any time-point of more than three times the upper normal limit [15].

Statistical analysis

Statistica 10 and Medcalc (ver. 12.2.1) software were used for statistical analysis. Normal distribution of continuous variables was checked with Kolomogorov-Smirnov test. Continuous variables were presented as mean ± 1 standard deviation (SD) or median depending on distribution; comparisons were conducted with t-Student or Mann-Whitney tests, respectively. Discrete variables were presented as percentage and frequencies and comparisons were performed with Fisher's exact test. Receiver operating characteristics (ROC) curve analysis was used to identify factors able to distinguish between patients with and without PMD. Variables significant by ROC analysis (entered as categorical variables above cut off value found by respective ROC analyses) plus several possible clinical and procedural confounding factors (age, female sex, hypertension, diabetes, dyslipidaemia, renal failure, lesion length, predilatation and postdilatation) were selected for stepwise logistic regression modelling to find independent predictors of PMD. P-values lower than 0.05 were considered as statistically significant.

RESULTS

Overall, 60 patients were enrolled and evaluated. Patient characteristics are presented in Table 1. Mean age of the studied population was 60.9 ± 11.7 years and the majority of patients were males (73.3%). A total of 79 stents were successfully implanted (47 [59.5%] bare metal and 32 [40.5%] drug-eluting stents). The study population was divided into groups that were compared to each other i.e. a population with PMI (PMI, n = 26, 43.3%), a population with PMD (PMD, n = 34, 56.7%), and a population with no PMD (nPMD, n = 26, 43.3%). There were no significant differences in age, sex, rate of comorbidities, laboratory findings or pharmacotherapy between the groups. There was a trend toward significant difference in platelet count between the nPMD and the PMI group (208.81 × $10^3/\mu$L vs. 235.23 × $10^3/\mu$L, p = 0.06, respectively). Mean PRU and ARU values were 251.77 ± 71.68 and 456.05 ± 72.34, respectively, and there were no significant differences between groups in terms of predefined high on-treatment platelet reactivity.

Sixty lesions were analysed with grey-scale IVUS. Due to technical problems, VH-IVUS data was only available in 53 patients. Grey-scale IVUS findings are summarised in Table 2 and VH-IVUS data is shown in Table 3. Average minimal lumen area was 3.07 ± 5.5 mm^2 and didn't differ between the nPMD and the PMD groups (3.03 vs. 3.09 mm^2, p = 0.81) or between the nPMD and the PMI groups (3.03 vs. 3.13 mm^2, p = 0.65). P&M CSA at minimal lumen site (11.14 vs. 12.63 mm^2, p = 0.03) and RI (0.91 vs. 1.07, p = 0.0025) were significantly lower in the nPMD compared to the PMD group. RI in the PMI group was also significantly higher than in the nPMD group (1.06 vs. 0.91, p = 0.0069). Lesions in the PMD group had significantly higher FT volume (51.71 vs. 93.52 mm^3, p = 0.03) and FFT volume (10.59 vs. 26.38 mm^3, p = 0.018). Similar differences were found between the nPMD and the PMI groups in terms of fibro-fatty volume (10.59 vs. 24.24 mm^3, p = 0.049) and FT volume (51.71 vs. 91.56 mm^3, p = 0.09). There were no differences in the absolute or percentage volume of other plaque components, and the frequency of TCFAs was similar between groups (Table 3).

ROC curve analysis revealed five variables that predicted the occurrence of PMD: EEM CSA (cut off > 14.6 mm^2, AUC = 0.653, 95% CI 0.519–0.772, p = 0.038) and P&M CSA (> 9.8 mm^2, AUC = 0.664, 95% CI 0.530–0.781, p = 0.024) at minimal lumen site, RI (> 1.044, AUC = 0.724, 95% CI 0.593––0.832, p = 0.0006), FT volume (> 50 mm^3, AUC = 0.716, 95% CI 0.567–0.837, p = 0.0048), FFT volume (> 11.2 mm^3, AUC = 0.704, 95% CI 0.555–0.827, p = 0.0079), and two variables that were able to distinguish between patients with and without PMI: RI (cut off > 1.044, AUC 0.658, 95% CI 0.525–0.776, p = 0.0308) and platelet count (> 168 × $10^3/\mu$L, AUC 0.656, 95% CI 0.522–0.774, p = 0.0269). ROC curves for these variables are shown in Figures 1 and 2.

Multivariate logistic regression analysis was performed to identify independent predictors of PMD and PMI. Variables significant in ROC curve analysis as well as several clinical and procedural factors were included in a multivariate model. According to multivariate analysis, the only independent predictors of PMD were: RI > 1.044 (OR = 13.32, 95% CI 1.25–121.85, p = 0.032), EEM CSA > 14.6 mm^2 (OR = 23.67, 95% CI 1.86––302.01, p = 0.015) and FFT volume > 11.2 mm^3 (OR = 13.60, 95% CI 1.15–160.91, p = 0.038). The performance of the multivariate model with RI and EEM CSA (AUC = 0.73, 95% CI 0.6–0.84) significantly improved after incorporation of FFT volume (AUC = 0.86, 95% CI 0.73–0.94). In multivariate logistic regression analysis, only RI > 1.044 (OR 7.5, 95% CI 1.92–29.6, p = 0.004) was identified as an independent predictor of PMI.

DISCUSSION

In the present study, we analysed the potential impact of different lesion and platelet parameters on myocardial damage during elective coronary interventions. To the best of our knowledge, this is the first attempt to test and compare the independent contribution of on-treatment platelet reactivity as well as IVUS/VH-IVUS lesion characteristics simultaneously in one homogenous population.

The main finding of our study is that simple parameters possible to assess with grey-scale IVUS, namely total vessel area (EEM CSA) and positive remodelling, are the strongest

Table 1. Patient characteristics and platelet reactivity

Variable	Total population (n = 60)	No PMD (n = 26)	PMD (n = 34)	P
Age (mean ± SD) [years]	60.9 ± 11.7	63.0 ±11.0	59.4 ± 12.1	0.24
Male [%]	73.3	65.4	79.4	0.69
Medical history [%]:				
Hypertension	73.3	73.1	73.5	1.00
Type 2 diabetes	23.3	19.2	26.5	0.77
Heart failure	18.3	19.2	17.6	1.00
Renal failure*	33.33	46.15	23.53	0.21
Dyslipidaemia	68.3	57.7	76.5	0.54
Myocardial infarction	51.7	46.2	55.9	0.82
Smoking	33.3	30.8	35.3	1.00
Laboratory findings (mean ± SD):				
eGFR^ [mL/min/1.73 m^2]	68.24 ± 21.00	65.69	70.20	0.41
HGB [g/dL]	13.91 ± 1.26	13.94	13.87	0.84
PLT [×10^3/μL]	220.52 ± 50.76	208.81	229.47	0.12
MPV [fL]	10.39 ± 1.17	10.38	10.39	0.97
Pharmacotherapy [%]:				
Aspirin	100	100	100	1.00
Clopidogrel	100	100	100	1.00
β-blocker	96.6	96.0	97.0	1.00
Statin	98.3	100	97.0	1.00
ACEI	87.9	84.0	90.9	0.85
ARB	8.6	8.0	9.1	1.00
CCB	12.1	12.0	12.1	1.00
Nitrate	8.6	8.0	9.1	1.00
PPI	12.1	8.0	15.2	0.69
Target vessel [%]:				
Left anterior descending	46.7	38.5	52.9	0.64
Right coronary artery	35.0	38.5	26.5	0.60
Circumflex	13.3	15.4	8.8	0.69
Intermediate branch	3.3	3.8	2.9	1.00
Left main	1.7	3.8	0.0	0.44
Percutaneous coronary intervention:				
Mean stents (no.)	1.3	1.3	1.4	1.00
Mean stent(s) (length, mean ± SD) [mm]	26.0 ± 14.25	24.2	27.4	0.39
Predilatation [%]	61.6	53.8	67.6	0.67
Postdilatation [%]	78.2	76.0	79.4	1.00
Platelet reactivity:				
PRU (mean ± SD)	251.77 ± 71.68	247.35	255.15	0.68
PRU > 235 [%]	58.3	50	73.5	0.40
ARU (mean ± SD)	456.05 ± 72.34	461.42	451.94	0.69
ARU > 550 [%]	13.3	15.4	11.8	0.72

PMD — periprocedural myocardial damage; eGFR — estimated glomerular filtration rate; HGB — haemoglobin; PLT — platelets; MPV — mean platelet volume; ACEI — angiotensin converting enzyme inhibitor; ARB — angiotensin receptor blocker; CCB — calcium channel blocker; PPI — proton pump inhibitor; PRU — P_2Y_{12} reaction units; ARU — aspirin reaction units; *eGFR < 60 mL/min/1.73 m^2; ^according to MDRD formula

Table 2. Grey-scale intravascular ultrasound findings

Variable	Total population (n = 60)	No PMD (n = 26)	PMD (n = 34)	P
EEM CSA (ml) [mm^2]	15.06 ± 4.89	14.17	15.74	0.10
EEM CSA (dr) [mm^2]	13.24 ± 5.08	13.90	12.79	0.65
EEM CSA (pr) [mm^2]	17.33 ± 5.50	17.43	17.27	0.94
Minimal lumen area [mm^2]	3.07 ± 0.94	3.04	3.09	0.81
Lumen CSA (dr) [mm^2]	7.79 ± 2.73	7.50	8.00	0.37
Lumen CSA (pr) [mm^2]	9.84 ± 4.19	10.23	9.55	0.88
P&M CSA (ml) [mm^2]	11.98 ± 4.58	11.14	12.63	0.03
P&M CSA (dr) [mm^2]	5.45 ± 3.26	6.37	4.79	0.08
P&M CSA (pr) [mm^2]	7.50 ± 3.83	7.20	7.72	0.61
Plaque burden [mL]	78.54 ± 6.42	77.40	79.40	0.09
Lesion length [mm]	21.00 ± 11.66	18.49	22.75	0.15
Remodelling index	1.00 ± 0.21	0.91	1.07	0.0025

PMD — periprocedual myocardial damage; EEM — external elastic membrane; CSA — cross-sectional area; P&M — plaque and media; ml — minimal lumen; pr — proximal reference; dr — distal reference

Table 3. Virtual histology findings

Variable	Total population (n = 53)	No PMD (n = 22)	PMD (n = 31)	P
Fibrous tissue [mm^3]	76.97 ± 67.52	51.71	93.52	0.03
Fibrous tissue [%]	56.18 ± 9.51	55.45	56.71	0.55
Fibro-fatty tissue [mm^3]	20.13 ± 23.01	10.59	26.38	0.018
Fibro-fatty tissue [%]	13.61 ± 8.67	12.43	14.45	0.58
Dense calcium [mm^3]	10.29 ± 9.00	9.91	10.55	0.61
Dense calcium [%]	9.92 ± 7.53	10.72	9.36	0.32
Necrotic core [mm^3]	23.55 ± 17.18	21.66	24.79	0.63
Necrotic core [%]	20.32 ± 9.00	21.50	19.48	0.43
Thin cap fibroatheroma [%]	62.3	59.1	64.5	1.00

PMD — periprocedual myocardial damage

predictors of PMD. On the other hand, platelet reactivity measured with point-of-care assay could not distinguish between patients with and without PMD/PMI.

In the last decade, it has been shown that response to a fixed dose of clopidogrel is heterogeneous and that, in a substantial proportion of patients, platelet inhibition is inadequate or even absent [7]. Subsequently, it was proved in several observational studies that on-treatment (on-ASA and on-clopidogrel) platelet reactivity may contribute to a poorer outcome in patients undergoing coronary stenting [8, 9]. This association is strongest in the setting of acute coronary syndromes (ACS), but it can be also observed in patients with stable CAD. In our study, we did not find any relation between platelet reactivity and PMD. This may be partly explained by a hypercoagulable state during acute phase of ACS and therefore a greater need for platelet inhibition compared to elective stenting.

Previous studies have found a clear relationship between the severity and direction of arterial remodelling and the clinical presentation of patients with stable CAD [16, 17]. It was shown that positive remodelling is one of the independent predictors of periprocedural complications in patients with ACS undergoing coronary stenting. However, as of now, there is no data available on its association with PMD and PMI in stable patients. Most VH-IVUS studies have shown a link between certain plaque components, mainly NC, and PMD/PMI [11, 12]. In our study, we did not find any differences in total NC volume or the rate of TCFA between nPMD and any level of periprocedural myocardial injury. On the other hand, there were significant differences between groups in FT volume and FFT volume, and the later turned out to be an independent predictor of PMD by multivariate analysis. Fibro-fatty plaques, known also as lipid-rich plaques, are characterised by pronounced positive remodelling due to metalloproteases production induced by oxidised low-density lipoproteins [18]. Plaque damage during coronary stenting causes the release of some factors like tissue-factor as well as some microparticles responsible for distal embolisation [19]. This finding supports and extends

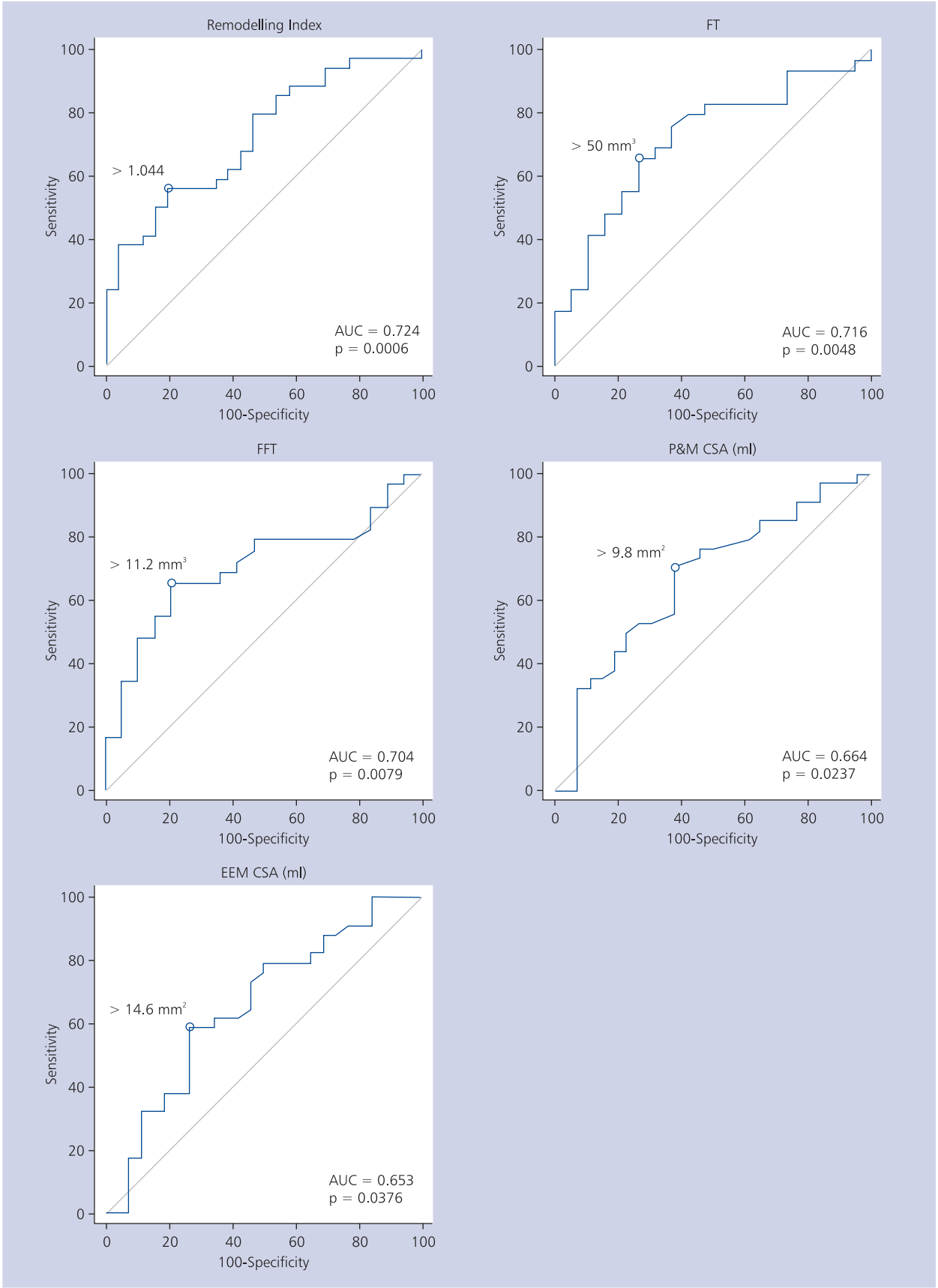

Figure 1. Receiver operating characteristics curve for periprocedural myocardial damage; FT — fibrous tissue; FFT — fibro-fatty tissue; P&M — plaque and media; CSA — cross-sectional area; EEM — external elastic membrane; ml — minimal lumen

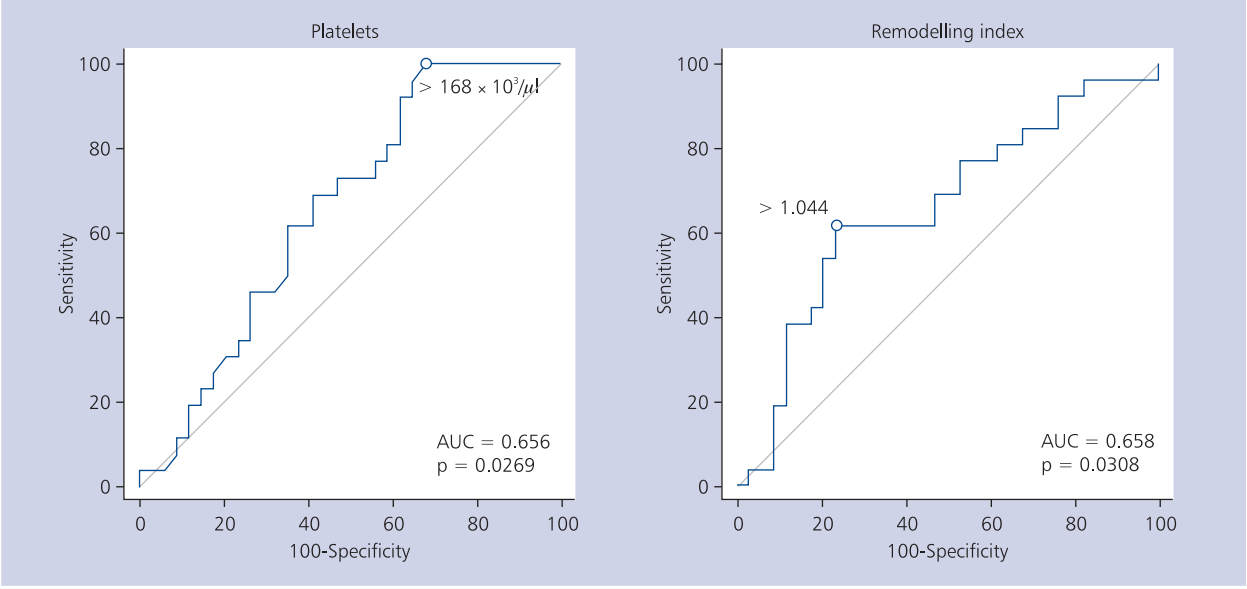

Figure 2. Receiver operating characteristics curve for periprocedural myocardial infarction

the results of previous studies, in which higher fibro-fatty volume was associated with no-reflow phenomenon in ST elevation myocardial infarction patients [20, 21].

In conclusion, it seems that the combined use of grey-scale IVUS (large, positively remodelled vessel) and VH-IVUS (lipid-rich plaque) enables optimal identification of patients prone to any periprocedural myocardial injury. Moreover, further analysis indicates that only a simple grey-scale IVUS parameter, namely RI, is associated with more pronounced myocardial necrosis considered as PMI. The present findings broaden the meaning of the term 'vulnerable plaque' which is considered as NC-rich plaque or TCFA in clinical condition of ACS. Differently, in the setting of stable CAD, large, lipid-rich plaque rather than the presence of TCFA can be perceived as 'vulnerable'.

Limitations of the study

There are several limitations that merit careful consideration. First, the number of patients was small, thus the analysis was underpowered to detect all possible predictors of PMD/PMI, and some selection bias cannot be excluded entirely. Second, the study population consisted of stable patients but with a recent history of ACS. This may have influenced the relatively high percentage of TCFA found by VH-IVUS in the target lesions. Third, the present findings refer only to significant but not critical target lesions allowing IVUS probe insertion before any lesion preparation. Finally, due to technical difficulties, the VH-IVUS analysis was performed only in 88% of the patients.

CONCLUSIONS

Greater total vessel area, positive remodelling at the lesion site, and high volume of FFT in the coronary plaque are independently associated with an increased risk of myocardial injury. Only positive RI was an independent predictor of PMI. Simple lesion morphology, rather than more complex VH-IVUS analysis or platelet reactivity, seems to predict myocardial injury after elective PCI.

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Conflict of interest: *none declared*

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Okołozabiegowe uszkodzenie miokardium podczas angioplastyki tętnic wieńcowych: badanie z wykorzystaniem przyłóżkowych testów oceny funkcji płytek krwi i ultrasonografii wewnątrzwieńcowej z wirtualną histologią

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Streszczenie

Wstęp: Wyniki ostatnich badań sugerują, że reaktywność płytek krwi i określona morfologia blaszki miażdżycowej mogą wpływać na uszkodzenie mięśnia sercowego podczas przezskórnych interwencji wieńcowych (PCI). Jednak dotychczas nie badano jednoczesnego wpływu powyższych parametrów na ryzyko uszkodzenia miokardium w jednej populacji.

Cel: Celem niniejszej pracy była ocena korelacji wysokiej reaktywności płytek krwi podczas leczenia przeciwpłytkowego i równocześnie morfologii oraz budowy blaszki miażdżycowej z ryzykiem okołozabiegowego uszkodzenia mięśnia sercowego u pacjentów kierowanych na planową PCI z implantacją stentu.

Metody: Do badania włączono 60 osób leczonych kwasem acetylosalicylowym i klopidogrelem, których poddano planowej PCI z implantacją stentu(ów). Ocenę reaktywności płytek krwi przeprowadzono przed PCI za pomocą urządzenia VerifyNow (Accumetrics, USA). U wszystkich pacjentów przed PCI oceniano morfologię zmiany w ultrasonografii wewnątrzwieńcowej (IVUS) z wirtualną histologią (VH-IVUS) (Volcano, USA). Wyróżniono 2 poziomy uszkodzenia mięśnia sercowego: jakikolwiek wzrost stężenia troponiny I powyżej normy w seryjnych oznaczeniach w ciągu 24 godzin po zabiegu (okołozabiegowe uszkodzenie miokardium, PMD) i/lub wzrost stężenia troponiny I więcej niż 3 razy powyżej normy (okołozabiegowy zawał serca, PMI).

Wyniki: Następujące parametry, w kolejności od najsilniejszego do najsłabszego, różnicowały na podstawie analizy krzywych ROC pacjentów z PMD i pacjentów bez PMD: wskaźnik remodelingu (RI), objętość tkanki włóknistej (FT), objętość tkanki włóknisto-tłuszczowej (FF), pole powierzchni przekroju blaszki i błony środkowej, pole powierzchni przekroju naczynia w miejscu zwężenia (EEM CSA). Wśród parametrów różnicujących chorych bez PMI od chorych z PMI znalazły się jedynie: liczba płytek krwi oraz RI. Reaktywność płytek krwi zarówno podczas leczenia kwasem acetylosalicylowym, jak i klopidogrelem nie była czynnikiem ryzyka ani PMD, ani PMI. Nie wykazano również związku między objętością rdzenia martwiczego a PMD. W analizie regresji logistycznej, uwzględniającej wiele parametrów klinicznych i zabiegowych jedynymi niezależnymi predyktorami PMD okazały się EEM CSA > 14.6 mm^2 (OR 23,7; 95%CI 1,9–302; p = 0,015), RI > 1,044 (OR 12,3; 95%CI 1,2–121,9; p = 0,032) oraz FF > 11,2 mm^3 (OR 13,6; 95%CI 1,1–160,9; p = 0,038). Z kolei jedynym niezależnym czynnikiem predykcyjnym wystąpienia PMI był RI > 1,044 (OR 7,5; 95%CI 1,92–29,6; p = 0,004).

Wnioski: Duża powierzchnia naczynia w miejscu zwężenia, pozytywny remodeling i duża objętość tkanki włóknisto-tłuszczowej w blaszce miażdżycowej są niezależnymi predyktorami uszkodzenia mięśnia sercowego. Jedynie pozytywny remodeling okazał się niezależnym predyktorem PMI. Wydaje się, że uszkodzenie mięśnia sercowego podczas planowej PCI zależy bardziej od prostych parametrów morfologicznych blaszki miażdżycowej niż jej budowy histologicznej czy reaktywności płytek krwi.

Słowa kluczowe: okołozabiegowe uszkodzenie mięśnia sercowego, stabilna choroba wieńcowa, IVUS, VH-IVUS, reaktywność płytek krwi

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