Novel procedural risk factors for myocardial injury following percutaneous coronary interventions with rotational atherectomy

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INTRODUCTION

Rotational atherectomy (RA) is one of the most widely used methods of plaque modification, especially in the presence of diffuse calcifications [1]. Due to the aggressiveness of the RA procedure and the presence of pulverized microdebris, it is thought to be associated with higher risk of myocardial injury [2]. High-sensitivity cardiac troponin I (hs-TnI) is one of the most sensitive indicators of minor myocardial damage after percutaneous coronary intervention (PCI) [3]. Multiple studies showed a high prevalence of elevated hs-TnI after percutaneous procedures, with discordant effect on clinical results [4–6]. It was also shown that the extent of myocardial injury is related to the complexity of the procedure [2]. However, there are no data on which procedural aspects of RA are associated with myocardial injury. This study aimed to assess the incidence and procedure-specific indicators of myocardial injury.

METHODS

This was a single-center prospective observational study. We included patients with clinical indications for RA in accordance with the European expert consensus document [1]. The exclusion criteria were a prior diagnosis of acute coronary syndrome and elevated baseline hs-TnI. The study protocol was accepted by the local ethics committee (ID no. 239/2016) and was in accordance with the declaration of Helsinki. All patients gave written informed consent for their participation in the study.

Treatment was conducted according to local standards and guidelines using the RotaLink system (Boston Scientific, Marlborough, MA, US). All procedural aspects were left to the discretion of the operator. All patients were on dual antiplatelet therapy during the procedure, all patients received acetylsalicylic acid, 38 (90%) received clopidogrel, and 4 (10%) received ticagrelor. All procedures were performed by a single team of experienced operators to ensure consistent results. Operators and physicians providing care to the patients were blinded for hs-TnI results unless a patient presented clinical signs of myocardial ischemia after the index procedure.

Blood samples were collected before the procedure and on the following morning (12–24 hours post-procedure). Hs-TnI measurements were performed in a local laboratory using the chemiluminescence LOCI™ method. All measurements were performed using a Dimension EXL analyzer (Siemens Healthcare Diagnostics, Erlangen, Germany).

Periprocedural myocardial injury and periprocedural myocardial infarction were defined according to the Fourth Universal Definition of Myocardial Infarction. Myocardial injury was defined as an increase in hs-TnI levels above the 99th percentile upper reference limit (URL), and substantial myocardial injury was defined as an increase in TnI levels more than five times above the 99th percentile URL.

Statistical analysis

No indentation variables with normal distribution were presented as mean and standard deviation. Continuous variables with skewed distribution were presented as median with interquartile range. Categorical variables were presented as numbers and percentages. For continuous variables, intergroup differences were compared using Student's t-test or the Mann-Whitney U test, depending on the type of distribution. The χ^2 test (using Yate's correction for continuity where necessary) was used to compare categorical variables. A *P*-value < 0.05 was considered statistically significant. All statistical analyses were performed using Statistica 10.0 (StatSoft, Tulsa, OC, US) software.

RESULTS AND DISCUSSION

During the study period (September 2016 to April 2018), 110 patients underwent RA in our institution. Forty-three (39%) patients were hospitalized for acute coronary syndrome or had elevated hs-TnI levels before the procedure and were, therefore, excluded from this study. Furthermore, in 25 (22%) patients, full procedural or laboratory data were not available. As a result, 42 patients with complete clinical and procedural data were included in this study.

A substantial increase (≥5 times above the upper limit of normal) in hs-TnI was present in 23 (55%) patients (referred to below as the high TnI group). Median concentration of hs-TnI in this group was 0.28 (0.017–0.04) ng/ml immediately after the procedure and 0.95 (0.44–1.85) ng/ml 12–24 hours later. Non-substantial increase (<5 times above the upper limit of normal) in hs-TnI was present in 19 (45%) patients. Complete demographics, comorbidities, and procedural factors of both groups are presented in Table 1.

Except for a trend toward older age (mean, 73.4 vs. 68.8; *P* = 0.09) and a higher prevalence of diabetes in the high TnI group (74% vs. 47%; *P* = 0.07), there were no differences in common cardiovascular risk factors. Patients with a substantial increase in hs-TnI after RA underwent PCI less often (70% vs. 95%, p=0.03) and trended towards a higher SYNTAX Score (mean, 21.7 vs. 15.8; *P* = 0.051).

There were no differences in the treated vessels between the groups.

We showed that in the high TnI group, total burr use time (median, 194 seconds vs. 82 seconds; *P* <0.001) and the total number of burr runs (median, 7 vs. 4; *P* = 0.01) were significantly higher. There was also a trend toward a larger number of stents implanted (mean, 1.56 vs. 1.16; *P* = 0.058) in that group. Procedural success was high in both groups, however, significantly higher in the high TnI group (100% vs. 84%; *P* = 0.048).

The rate of complications was low and did not differ between the two groups. Despite significant TnI release, after correlation with clinical data, only one patient in the high TnI group fulfilled the criteria for type 4a myocardial infarction diagnosis.

Our study is, to our knowledge, the first to investigate both incidence and procedural indicators of myocardial injury after RA.

We showed that despite a very high occurrence of any (79%) or significant (55%) TnI elevation after RA, the rate of in-hospital complications was very low with only a single type 4a myocardial infarction, and no significant arrhythmias were observed during the procedures.

Previous studies showed that the occurrence of myocardial injury and periprocedural MI related to RA is higher than after traditional PCI [2, 7]. This observation was foreseeable, but until now it could not be connected to specific procedural aspects. Thus far the degree of myocardial injury was correlated mainly with the atherosclerotic burden and the extent of calcification, which provoked the use of more aggressive techniques [8]. Previous publications demonstrated clinical and anatomical (sequential lesions, acute lesion angulation) predictors of MI after RA. However, none of these studies focused on the procedural aspects of the intervention [9, 10]. Other studies showed that during RA procedures radial access and female sex are risk factors for coronary artery perforation and periprocedural complications (including mortality), respectively [11, 12]. In our study there were no differences between the studied groups in this regard, therefore, there is no evidence that these factors could impact myocardial injury after RA.

For the first time, we showed that the total time of burr use and the number of burr runs are correlated with a significant elevation of TnI levels after the procedure. It is worth noting that in most cases, time of burr use and the number of burrs used are, indeed, directly associated with the extent of the calcified plaque, which enforces more aggressive treatment. We also observed higher SYNTAX scores in the high TnI group. These observations suggest a larger impact of overall atherosclerotic burden rather than selected procedural aspects of TnI release after RA.

CONCLUSIONS

Myocardial injury after RA, defined as a substantial increase in hs-TnI levels, is not uncommon. The clinical significance of this finding requires further studies.

Hs-TnI release occurs even when the procedure is performed without any complications. Frequent significant hs-TnI release after RA seems to be clinically silent. Further studies with clinical endpoints are required to create any new recommendations for RA operators.

Article information

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Table 1. Population clinical and procedural characteristics

	All patients	No significant (< 5 × ULN) hs-TnI release	Significant (≥5 × ULN) hs-TnI release	*P*-value
N (%)	42 (100)	19 (45)	23 (55)	
Age, years, mean (SD)	71.3 (8.9)	68.8 (9.6)	73.4 (7.8)	0.09
Male sex, n (%)	31 (74)	16 (84)	15 (65)	0.16
Hypertension, n (%)	39 (93)	17 (89)	22 (96)	0.43
Diabetes mellitus, n (%)	26 (62)	9 (47)	17 (74)	0.07
Hyperlipidemia, n (%)	18 (43)	8 (42)	10 (43)	0.92
Peripheral artery disease, n (%)	9 (21)	4 (21)	5 (22)	0.96
Left ventricular ejection fraction, %, mean (SD)	48.8 (11.3)	46.2 (12.4)	50.9 (10.0)	0.18
Impaired renal function with eGFR <60 ml/min/1.73 m², n (%)	9 (21)	3 (16)	6 (26)	0.41
Prior acute coronary syndrome, n (%)	22 (52)	11 (58)	11 (48)	0.51
Prior PCI, n (%)	34 (81)	18 (95)	16 (70)	0.03
Prior CABG, n (%)	5 (10)	1 (5)	4 (17)	0.23
EuroSCORE, median (IQR)	2.03 (1.2–4.0)	1.65 (1.08–2.83)	2.34 (1.21–5.77)	0.26
SYNTAX score, mean (SD)	19.0 (9.8)	15.8 (9.7)	21.7 (9.2)	0.051
Radial access, n (%)	41 (98)	19 (100)	22 (96)	0.36
GP IIb/IIIa inhibitors used, n (%)	0 (0)	0 (0)	0 (0)	1.0
Target vessel				
RCA, n (%)	15 (36)	8 (42)	7 (30)	0.43
LM, n (%)	5 (12)	3 (16)	2 (9)	0.48
LAD, n (%)	17 (40)	6 (32)	11 (48)	0.29
Cx, n (%)	2 (5)	1 (5)	1 (4)	0.89
OM, n (%)	2 (5)	1 (5)	1 (4)	0.89
Dg, n (%)	1 (2)	0 (0)	0 (0)	0.34
Procedural data				
Total time of burr use, second, median (IQR)	114 (79–222)	82 (60–143)	194 (83–327)	0.007
Total number of burr runs, median (IQR)	5 (3–8)	4 (2–6)	7 (3–12)	0.01
Mean burr speed, RPM × 1000, mean (SD)	145.9 (4.9)	146.2 (4.3)	145.7 (5.5)	0.76
Number of burrs used, mean (SD)	1.21 (0.4)	1.21 (0.4)	1.22 (0.4)	0.95
Burr-to-artery ratio, mean (SD)	0.46 (0.08)	0.46 (0.07)	0.46 (0.08)	0.91
Maximum burr diameter, mean (SD)	1.44 (0.2)	1.45 (0.2)	1.42 (0.18)	0.69
Number of stents implanted, mean (SD)	1.38 (0.7)	1.16 (0.2)	1.56 (0.7)	0.058
Total length of implanted stents, mm, mean (SD)	27.6 (16.2)	25.2 (16.3)	29.4 (16.2)	0.42
Contrast volume, ml, mean (SD)	218 (51)	205 (34)	229 (62)	0.22
Procedural success, n (%)	39 (93)	16 (84)	23 (100)	0.048
Periprocedural complications				
Slow/no-flow, n (%)	0 (0)	0 (0)	0 (0)	1.0
Side branch occlusion, n (%)	0 (0)	0 (0)	0 (0)	1.0
Dissection, n (%)	2 (5)	0 (0)	2 (9)	0.11
Perforation, n (%)	1 (2)	0 (0)	1 (4)	0.36
Emergency CABG, n (%)	0 (0)	0 (0)	0 (0)	1.0
In-hospital outcomes				
Death, n (%)	0 (0)	0 (0)	0 (0)	1.0
Peri-procedural MI, n (%)	1 (2)	0 (0)	1 (4)	0.36
Stroke/TIA, n (%)	0 (0)	0 (0)	0 (0)	1.0
Contrast-induced nephropathy, n (%)	0 (0)	0 (0)	0 (0)	1.0
Hs-TnI immediately post-procedure, ng/ml, median (IQR)	0.022 (0.017–0.039)	0.017 (0.017–0.039)	0.28 (0.017–0.04)	0.06
Hs-TnI 12–24 hours post procedure, ng/ml, median (IQR)	0.371 (0.086–1.181)	0.084 (0.035–0.176)	0.95 (0.44–1.85)	<0.001
Any hs-TnI elevation, n (%)	33 (79)	10 (53)	23 (100)	<0.001

Abbreviations: CABG, coronary artery bypass grafting; Cx, circumflex artery; Dg, diagonal artery; eGFR, estimated glomerular filtration rate; hs-TnI, high-sensitive troponin I; LAD, left anterior descending; LM, left main; MI, myocardial infarction; OM, obtuse marginal artery; PCI, percutaneous coronary intervention; RCA, right coronary artery; RPM, revolutions per minute; TIA, transient ischemic attack; ULN, upper limit of normal

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