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ORIGINAL ARTICLE

Plaque erosion versus rupture characterization by optical frequency domain imaging before and after coronary stenting following successful fibrinolysis for ST-segment elevation myocardial infarction

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Abstract
Intracoronary thrombus burden affects the quality of myocardial reperfusion in the setting of ST-elevation myocardial infarction (STEMI). We aimed to study the characteristics of the plaque and thrombus assessed by intracoronary optical frequency domain imaging (OFDI) according to the presence of plaque rupture or erosion in STEMI patients treated with successful fibrinolysis. Pre-stenting thrombus and post-stenting atherothrombotic burden were compared between plaque rupture and erosion. Twenty-seven consecutive patients were included: 17 (63%) had OFDI-plaque rupture and 10 (37%) had OFDI-erosion. Thrombus volume and burden were significantly higher in case of rupture compared to erosion at baseline (13.4 ± 18.4 vs 2.8 ± 2.3 mm^3; $p = 0.03$ and 33.8 ± 17.5 vs 17.5 ± 9.9%; $p = 0.007$, respectively). In the rupture group, the core of the thrombus consisted dominantly of red thrombus evenly distributed along the entire culprit plaque. In the erosion group, it consisted dominantly of white thrombus with a focal distribution near the minimal lumen area zone. After stenting, the atherothrombotic volume, burden and its distribution, as well as angiographic estimators of myocardial reperfusion were similar between groups. Our study showed that pre-PCI thrombus amount, typesetting and distribution are mainly linked to the underlying mechanism of STEMI. After stenting, the atherothrombotic burden and its distribution were similar between the groups.

Keywords Optical frequency domain imaging · ST-elevation myocardial infarction · Thrombus · Plaque rupture · Plaque erosion

Introduction

The most common underlying mechanisms of myocardial infarction are a rupture or erosion of an atherosclerotic plaque initiating intraluminal thrombus formation and the subsequent partial or complete occlusion of the coronary artery [1]. High thrombus burden is associated with the risk of distal embolization and microcirculatory impairment, reducing the efficacy of myocardial reperfusion by percutaneous coronary intervention (PCI) and increasing infarct size [2–4].

Optical coherence tomography (OCT) and optical frequency domain imaging (OFDI) provide high resolution imaging of the culprit lesion and thrombus [5]. Several studies have described the underlying plaque morphologies and the thrombus burden using OCT [6–9] in patients with ST-segment elevation myocardial infarction (STEMI) treated with primary PCI. However, in all latter studies, OCT has been performed after thrombectomy or balloon pre-dilation as high thrombus burden or severe stenosis is the pitfalls of the technique in the acute phase of STEMI. Hence, features described by such studies may have been modified by such interventions prior to imaging.

In patients with STEMI managed early after symptom-onset, pre-hospital fibrinolysis (FL) within a

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pharmaco-invasive strategy is a valuable alternative to primary PCI with similar early and late mortality rates [10]. At the time of PCI—within 2–24 h after thrombolytic therapy [11]—, the paradoxical pro-thrombotic post-FL status [12] associated with the variability of platelet response and slow onset of action of clopidogrel [13] especially with the low doses [14] recommended in association with FL increase the risk of thrombus formation. On the other hand, thrombus burden depends also on the underlying mechanism of myocardial infarction, plaque rupture being associated with higher thrombus burden as compared to erosion [9].

The aim of our study was to assess and compare the characteristics of thrombus between OFDI-plaque rupture (PR) and OFDI-plaque erosion (PE), before and after PCI following successful FL in STEMI patients treated using OFDI.

Methods

Study population

We investigated patients enrolled in the prospective AODIS study (assessment of intracoronary thrombus by optical frequency domain imaging during percutaneous coronary intervention after successful fibrinolysis and its impact on myocardial reperfusion) between July 2015 and October 2016. The inclusion criteria were: patient ≥ 18 years admitted to our tertiary center for STEMI successfully treated by FL and PCI of a native coronary culprit lesion 3–24 h after FL, with a pre- and post-PCI OFDI imaging. Successful FL was defined as the resolution of both ST-segment elevation (> 50%) and chest pain < 90 min after FL [15]. OFDI (Lunawave®, FastView®, Terumo Europe, Leuven, Belgium) was performed before PCI and any percutaneous intervention and at the end of the procedure. Exclusion criteria were: use of glycoprotein IIb/IIIa inhibitors, cardiogenic shock, stent thrombosis or restenosis, severe renal insufficiency (estimated glomerular filtration rate (eGFR) < 30 mL/min), coagulation disorders and severely calcified or tortuous arteries inaccessible to OFDI.

Informed consent was obtained from all individual participants included in the study and the study was approved by the local ethics committee. The study is registered on ClinicalTrials.gov under the identifier NCT02850315.

Pharmacological regimen

All patients received a 250 mg i.v. bolus of aspirin and a clopidogrel oral loading dose of 300 mg if aged ≤ 75 years or 75 mg if aged > 75 years, followed by 75 mg of clopidogrel and aspirin daily as recommended in our regional STEMI network protocol. Anticoagulation was performed with enoxaparin in all. The fibrinolytic agent was tenecteplase, administered in a weight-adjusted dose [16].

OFDI image analysis

The OFDI procedure was performed as previously described [17]. The images were analyzed by 2 investigators (VR and CB) using previously validated criteria for OCT plaque characterization [18–20]. OFDI-plaque rupture (PR) was defined by the presence of fibrous cap discontinuity with a clear cavity formed inside the plaque [7, 19]. OFDI-plaque erosion (PE) was defined by the presence of attached thrombus overlying an intact plaque or luminal surface irregularity without thrombus and with no detectable signs of fibrous cap rupture. Culprit lesions that did not satisfy these criteria were classified as "other". Stent malapposition was defined as a stent to adjacent vessel lumen distance > 200 µm.

For the thrombus quantification, the whole stented segment was analyzed at each frame on post-PCI images. The corresponding arterial segment on pre-PCI images was identified using anatomical landmarks and analyzed similarly at each frame. On pre-PCI images, thrombus was defined as an irregular intraluminal mass either attached to the vessel wall or floating into the lumen. Thrombus was categorized as either erythrocyte-rich (red) highly backscattering with high attenuation or, platelet-rich (white), less backscattering, homogeneous with low attenuation. At each frame, lumen area (LA) and flow area (FA) were manually measured by planimetry as previously described [21] and thrombus area (TA) was defined as the difference between LA and FA (Fig. 1). The thrombus volume was calculated by numerical integration based on the disk summation method by the proprietary software [17]. Thrombus burden (TB) was calculated as thrombus volume divided by lumen volume multiplied by 100. We also calculated the thrombus burden of the thrombotic area (TB thrombotic area) by restricting the quantification only to the segment containing visible thrombus.

Post-stent atherothrombotic burden (ATB) included tissue prolapse and thrombus, either attached to the vessel wall or the struts or floating. The post-stent LA was measured using stents' struts as limits. In case of strut malapposition, a corresponding point in the vessel wall was traced instead [21]. The calculation of atherothrombotic burden and volume was similar to TB and thrombus volume. The semi-quantitative pre- and post-PCI thrombus scores were calculated by summing the number of involved quadrants

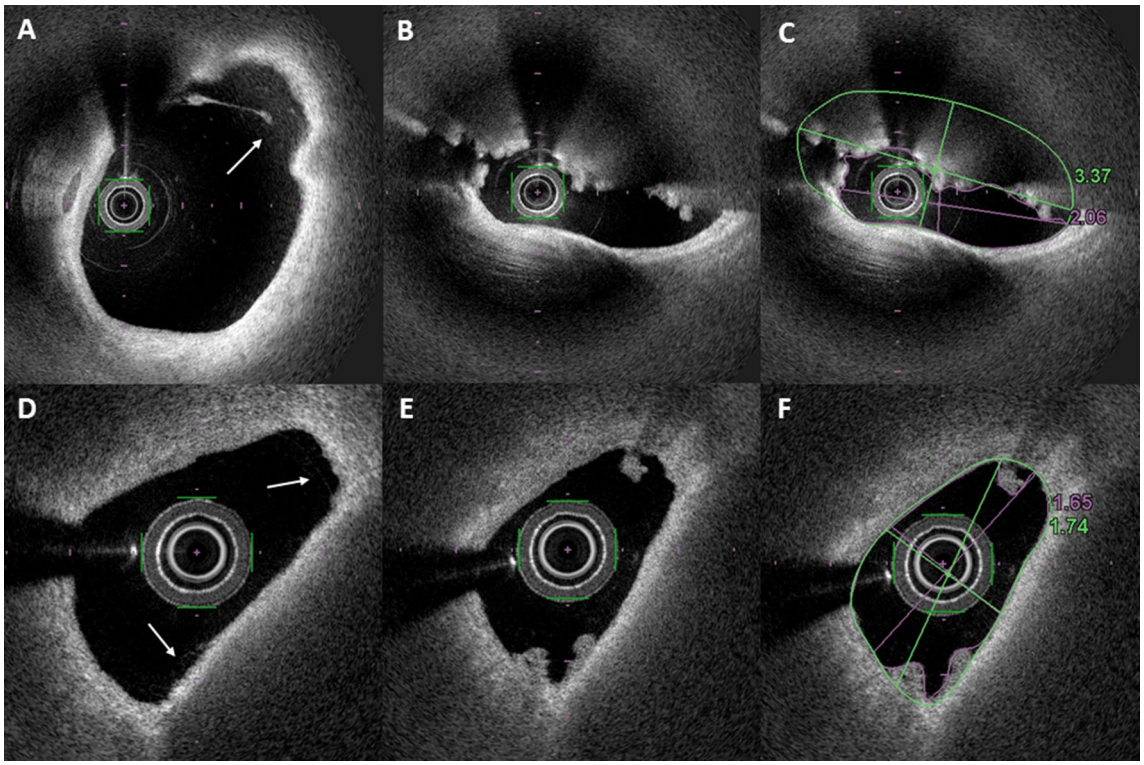

Fig. 1 Optical frequency domain imaging examples of plaque analysis. Plaque rupture (white arrow, **a**) associated with red thrombus 5 mm more proximal (**b**); lumen and flow area delimitations (**c**; thrombus area 1.31 mm^2 or 38.9%). Plaque erosion with mild luminal surface irregularities (white arrow, **d**) and white thrombus 0.2 mm more distal (**e**); lumen and flow area delimitations (**f**; thrombus area 0.09 mm^2 or 5.2%)

containing thrombus at 1 mm intervals [22]. Irregular protrusion was defined as previously described [23].

Angiographic analysis

Coronary angiograms were analyzed before PCI and at the end of the procedure to evaluate basal and post-PCI Thrombolysis in Myocardial Infarction (TIMI) flow grade, angiographic thrombus grade and, post-PCI myocardial blush grade and corrected TIMI frame count (CTFC), as previously described [24, 25].

Statistical analysis

Groups were defined by the identification of PR or PE. Continuous variables were expressed as mean ± standard deviation and compared between groups using Student's *t*, Welch–Satterthwaite or Wilcoxon–Mann–Whitney tests when adapted. Categorical variables were expressed as numbers (percentages) and compared between groups using Fisher's exact test. Univariate correlations were assessed by Pearson's correlation test. A p value of <0.05 was considered statistically significant. SAS 9.4 (SAS Institute Inc., Cary, NC, USA) was used for statistical analysis.

Results

A total of 31 patients underwent OCT evaluation after successful FL but 4 were excluded from the analysis because of poor pre-stent image quality in 2 and un-identification of plaque rupture or erosion in 2. Finally, 27 patients were included in the analysis, 17 (63%) with PR and 10 (37%) with PE.

Baseline demographic and angiographic characteristics (detailed in Table 1) were comparable between groups, except for the culprit coronary lesion which was more often located in the left circumflex in the erosion group ($p=0.04$). No patient had thrombus aspiration.

The OFDI analysis showed that rupture was associated with higher rates of lipid-rich plaque (Table 2). Baseline thrombus volume and burden of the thrombotic area were significantly higher in PR compared to PE (13.4 ± 18.4 vs 2.8 ± 2.3 mm^3; $p=0.03$ and 33.8 ± 17.5 vs 17.5 ± 9.9%; $p=0.007$, respectively). In the rupture group, the core of the thrombus primarily consisted of red thrombus which was evenly distributed over the entire culprit lesion (Fig. 2a). In the erosion group, the core of the thrombus primarily consisted of white thrombus which remained concentrated around the minimal lumen

Table 1 Baseline characteristics of the study population

Baseline patient characteristics	All $n=27$ (100%)	Rupture $n=17$ (63%)	Erosion $n=10$ (37%)	p
Age (years)	59.9 ± 13.9	63.2 ± 15.4	54.2 ± 9.2	0.1
Men	24 (88.9%)	15 (88.2%)	9 (90%)	0.46
Body mass index (kg/m^2)	26.7 ± 4.7	27.9 ± 5.1	24.6 ± 2.9	0.07
Systemic hypertension	11 (40.7%)	7 (41.2%)	4 (40%)	0.3
Hyperlipidemia	12 (44.4%)	7 (25.9%)	5 (50%)	0.3
Active smoker	14 (51.8%)	8 (47%)	6 (60%)	0.5
Diabetes mellitus	2 (7.4%)	1 (5.9%)	1 (10%)	0.5
History of myocardial infarction	1 (3.7%)	1 (5.9%)	0	0.6
Anterior wall STEMI	11 (40.7%)	9 (52.9%)	2 (20%)	0.1
Inferior wall STEMI	15 (55.5%)	7 (41.2%)	8 (80%)	0.1
Other infarct location	1 (3.7%)	1 (5.9%)	0	0.6
Systolic blood pressure (mmHg)	130.6 ± 14.9	132.2 ± 14.4	128 ± 16.1	0.5
Diastolic blood pressure (mmHg)	76.2 ± 13.8	75.8 ± 12.3	76.9 ± 16.8	0.8
Heart rate (beats/min)	72.4 ± 14.2	73.2 ± 13.5	71.1 ± 16	0.7
Time points (*min*)				
Pain to first medical contact	122.3 ± 69.5	108.5 ± 64	145 ± 75.7	0.2
Pain to fibrinolysis	158.5 ± 79.6	143.1 ± 65	187.7 ± 97	0.16
Fibrinolysis to PCI	1026.5 ± 476.7	1001.1 ± 464.1	1069.8 ± 520	0.7
Biological characteristics				
Creatinine (µmol/L)	79.8 ± 15.9	80.1 ± 18.4	79.3 ± 11.2	0.9
Hemoglobin (g/dL)	14.2 ± 1.4	14	14.4	0.6
Adjunctive therapy				
Clopidogrel loading dose 75 mg	3 (11.1%)	3 (17.6%)	0	0.3
Clopidogrel loading dose 300 mg	24 (88.9%)	14 (82%)	10 (100%)	0.2
Angiographic characteristics				
Culprit coronary artery				
Left anterior descending artery	12 (44.4%)	10 (58.8%)	2 (20%)	0.1
Left circumflex artery	3 (11.1%)	0	3 (30%)	0.04
Right coronary artery	12 (44.4%)	7 (41.2%)	5 (50%)	0.3
Angiographic thrombus grade				0.7
Grade 0	4 (14.8%)	3 (17.6%)	1 (10%)	1
Grade 1	11 (40.7%)	6 (35.3%)	5 (50%)	0.7
Grade 2	4 (14.8%)	2 (11.8%)	2 (20%)	0.6
Grade 3	6 (22.2%)	5 (29.4%)	1 (10%)	0.4
Grade 4	2 (7.4%)	1 (5.9%)	1 (10%)	0.5
Basal TIMI flow grade				0.3
Grade 2	5 (18.5%)	4 (23.5%)	1 (10%)	0.3
Grade 3	22 (81.5%)	13 (76.5%)	9 (90%)	0.3
Final TIMI flow grade				0.3
Grade 2	3 (11.1%)	3 (17.6%)	0	0.23
Grade 3	24 (88.9%)	14 (82.3%)	10 (100%)	0.2
Basal corrected TIMI frame count	48.08 ± 21.7	52.3 ± 25.1	40.8 ± 11.9	0.2
Final corrected TIMI frame count	43.8 ± 22	47.4 ± 25.2	37.6 ± 13.9	0.3
Basal myocardial blush grade				0.3
Grade 2	5 (18.5%)	4 (23.5%)	1 (10%)	0.3
Grade 3	22 (81.5%)	13 (76.5%)	9 (90%)	0.5
Final myocardial blush grade 3	27 (100%)	17 (100%)	10 (100%)	1
Stent diameter (mm)	3.2 ± 0.6	3.2 ± 0.6	3.1 ± 0.6	0.6
Total stent length (mm)	30.6 ± 13.8	31.9 ± 14.8	28.4 ± 12.2	0.5

PCI percutaneous coronary intervention, *STEMI* ST-elevation myocardial infarction

Table 2 OFDI findings of culprit lesions at baseline and post-stenting

OFDI characteristics	All $n=27$ (100%)	Rupture $n=17$ (63%)	Erosion $n=10$ (37%)	p
Culprit plaque analysis				
Lipid-rich plaque	21 (77.8%)	16 (94.1%)	5 (50%)	0.01
Cap thickness (μm)	76.1 ± 33.2	73.2 ± 36.2	86 ± 19.5	0.5
Thin-cap fibroatheroma	12 (54.6%)	11 (64.7%)	1(10%)	0.1
Fibrous plaque	6 (22.2%)	1 (5.9%)	5 (50%)	0.01
Calcification	10 (37%)	6 (35.3%)	4 (40%)	0.3
Microchannels	11 (40.7%)	9 (52.9%)	2 (20%)	0.08
Thrombus	27 (100%)	17 (100%)	10 (100%)	1
Red thrombus	18 (66.7%)	16 (94.1%)	1 (10%)	< 0.0001
White thrombus	10 (37%)	1 (5.9%)	9 (90%)	< 0.0001
Baseline analysis				
Reference lumen area (mm^2)	7.4 ± 3.3	7.4 ± 3.6	7.2 ± 5.2	0.8
Reference narrowing	4 (14.8%)	2 (11.8%)	2 (20%)	0.6
Minimum lumen area (mm^2)	1.43 ± 1.03	1.62 ± 1.18	1.08 ± 0.65	0.18
Area stenosis (%)	78.6 ± 13.7	76.6 ± 15.7	81.9 ± 9.4	0.34
Lumen volume (mm^3)	110.6 ± 54.6	125.8 ± 60	84.9 ± 32.1	0.06
Thrombus length (mm)	5.8 ± 3.9	6.3 ± 4	5 ± 3	0.4
Thrombus volume (mm^3)	9.47 ± 15.4	13.4 ± 18.4	2.8 ± 2.3	0.03
Thrombus burden (%)	7.67 ± 6.9	9.5 ± 7.2	4.6 ± 5.4	0.07
Thrombus burden TA* (%)	27.8 ± 15.8	33.8 ± 17.5	17.5 ± 9.9	0.007
Thrombus score	15.3 ± 10.4	17.4 ± 11.1	11.7 ± 4.7	0.1
Post-stenting analysis				
Instent dissection	1 (3.7%)	1 (5.9%)	0	0.6
Edge dissection	2 (7.4%)	1 (5.9%)	1 (10%)	0.5
Stent malapposition	6 (22.2%)	4 (23.5%)	2 (20%)	0.3
Instent minimal lumen area (mm^2)	6.3 ± 2.7	6.6 ± 2.7	5.8 ± 2.7	0.5
Intrastent protrusion > 500 μm	12 (44.4%)	8 (47.1%)	4 (40%)	0.3
Irregular protrusion	15 (55.6%)	11 (64.7%)	4 (40%)	0.15
Stent volume	180.7 ± 80.3	199.5 ± 86.3	148.7 ± 60.1	0.1
Atherothrombotic length (mm)	20.5 ± 6.9	21.2 ± 7	19.3 ± 6.9	0.5
Atherothrombotic volume (mm^3)	13.2 ± 8.8	14.9 ± 9.9	10.3 ± 6.3	0.2
Atherothrombotic burden (%)	7.42 ± 3.9	7.44 ± 3.4	7.4 ± 4.8	1
Atherothrombotic burden TA* (%)	8.1 ± 3.5	8.3 ± 2.9	7.8 ± 4.5	0.7
Thrombus score	33.8 ± 13.6	35.7 ± 16	30.5 ± 8	0.3

*TA** thrombotic area (calculation of the thrombus burden was restricted to the area containing thrombus)

area. After stenting, the atherothrombotic volume, burden and its distribution (Fig. 2b) were similar between groups.

There was a significant but weak correlation between baseline thrombus volume and post- stenting atherothrombotic volume ($r=0.39$; $p=0.045$). In the rupture group, the distance between the minimal lumen area and site of rupture was 3 ± 2.3 mm.

During hospitalization, one patient presented major bleeding in the rupture group, related to intracranial hemorrhage.

Discussion

Our study showed that pre-stenting thrombus architecture, distribution and amount after successful FL are mainly linked to the underlying mechanism of myocardial infarction. Compared to PE, PR was associated with dominantly red—versus white—thrombus, evenly distributed across and afar the minimal lumen area—versus focal—and greater thrombus burden. After stenting, the

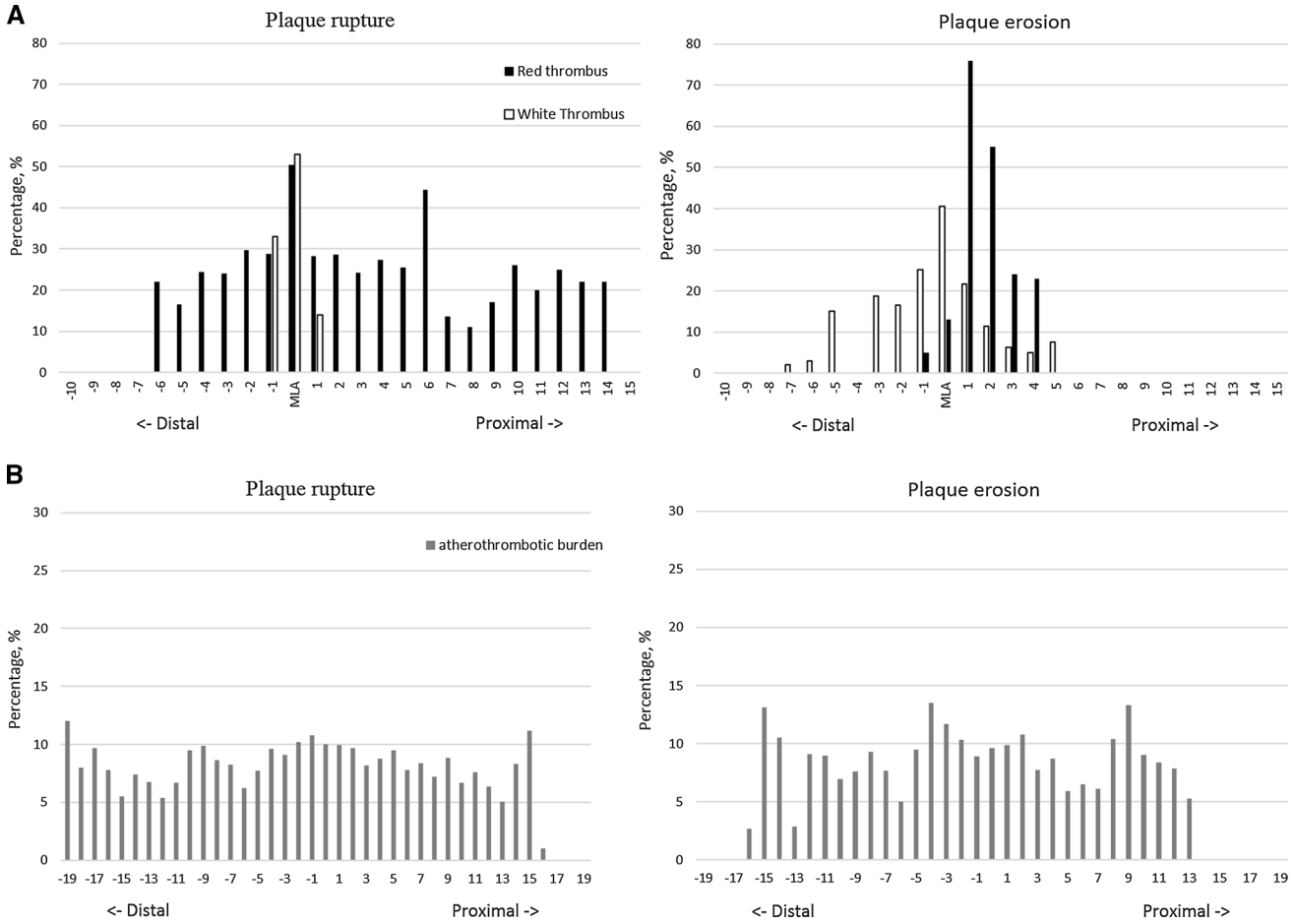

Fig. 2 Distribution of pre-stenting thrombus burden (**a**) and post-stenting atherothrombotic burden (**b**) in patients with plaque rupture vs plaque erosion, from either side of the minimal lumen area (MLA) in frames containing thrombus

atherothrombotic burden features became, however, similar between PR and PE.

Plaque rupture remains the most common substrate for coronary thrombosis and was identified in 63% of our patients concordantly with prior reports [7, 9, 26]. Although the amount of thrombus was variable between patients, it was identified on all assessed lesions regardless of the underlying mechanism. Two previous studies assessed thrombus by OCT after successful thrombolysis and identified thrombus in 65 and 100% of patients [27, 28]. PR was identified in half of their patients.

Our study outlined that PR was associated with greater thrombus burden, compared to PE. Post-thrombectomy OCT studies in STEMI patients treated with primary PCI did not all retrieve greater thrombus in the setting of rupture, based on the semi-quantitative thrombus score [7, 9]. Using the same score, we only found a trend toward higher thrombus burden in PR. However, using a more accurate volumetric quantification, in agreement with the only previous post-thrombolysis OCT study [27], we found a significantly higher thrombus volume and burden in the setting of PR compared to PE. PR and PE present different atherosclerotic patterns that could explain thrombus differences. While eroded plaques are often rich in smooth muscle cells and proteoglycans, PR usually complicates lipid-rich plaques [29], as shown in our study. PR occurs most frequently where the fibrous cap is the thinnest and heavily infiltrated by macrophage foam cells. Such conditions are associated with high tissue factor activity [30]. The necrotic core which is exposed to the circulation by PR is known to be the most thrombogenic component of atherosclerotic plaque [31]. The release of potent pro-thrombotic substances such as tissue factor and microparticles left behind after apoptotic cell death [32] may be one mechanistic explanation to higher thrombus volume associated with PR. Thrombi were also richer in erythrocyte in the rupture group and more platelet-rich in the erosion group, as previously described [9, 27, 33]. It is usually admitted that "red" thrombi correspond to old thrombi as opposed to recent platelet-rich "white" thrombi but ischemic times did not differ between groups in

our cohort. Plaque hemorrhage and extravasation of erythrocytes play an important role in necrotic core expansion and plaque vulnerability [26]. This may participate into the higher proportion of red thrombi observed in PR. On the contrary in the erosion group, the less intense thrombotic stimuli and smaller area of endothelial injury may be associated with more limited coagulation leading to more platelet-rich thrombi. Finally, inflammation plays a key role in the setting of STEMI and could explain thrombus differences. A recent OCT study showed that matrix metalloproteinase 9 (MMP-9) within the culprit plaques could affect thrombus formation [34]. Autopsy studies have reported lower rates of inflammatory markers in PE, as outlined by sparse infiltration of macrophages [26]. On the other hand, PR is associated with higher concentrations of C-reactive protein [35] which may explain its increased thrombogenicity. A recent OCT study showed different intracoronary cytokine expression patterns between PR and PE [36]. While PE was associated with preferential expression of thrombospondin-1, implicated in platelet recruitment [37], the increased MMP-9 expression observed in PR may explain the predominance of red thrombus as MMP-9 indirectly enhances tissue factor activity [38]. Although numerically higher, the post-stent atherothrombotic burden was not significantly different between both groups. A recent retrospective OCT study [39] including both STEMI and NSTEMI reported a higher incidence of thrombus after stenting with PR, even if the thrombus score-based analysis was not significantly different. Their population was different and thrombus volumes were approximately 10 times lower than in our study.

Importantly, our study showed that pre-stenting thrombus' distribution differed according to the causal mechanism. The setting of successful FL allowed a precise evaluation of plaque and thrombus characteristics without the changes in the architecture and the distribution of the plaque and the thrombus generated by balloon predilation or thrombectomy. In case of PR, the thrombus was evenly distributed across and afar the plaque while it remained focal within the minimal lumen area in case of erosion. Eroded plaques expose the underlying collagen as one nidus for thrombus formation [40] leading to a more localized and mural thrombus. The strong pro-thrombotic stimuli associated with PR induce a rapid and important thrombus formation—usually floating into the lumen—which contributes to the thrombus mass spreading. Additionally, the exposure of the plaque content including local inflammatory markers to the blood flow may participate to the thrombus' extension. At the opposite, local thrombospondin-1 release in PE secures platelet tethering and thrombus adherence to injured endothelium [37]. Moreover, autopsy studies have shown that more than 88% of coronary thrombi overlying plaque erosions exhibited late stages of healing, compared to 50% in plaque rupture [41]. This suggests repetitive less intense thrombotic stimuli in erosion, which may have left time to thrombus dissolution caused by spontaneous FL or by pharmacological FL in our patients. Then again, the post-stent atherothrombotic burden distribution was uniformly scattered in both groups, likely as a consequence of similar thrombus–plaque crushing spreading uniformly the atherothrombotic substrate along the stent. Hence, pre-stenting but not post-stenting thrombus amount, typesetting and distribution seem to be mainly linked to the underlying mechanism of myocardial infarction. However, we found a significant but relatively weak correlation between the pre- and post-stent thrombotic volume in the overall population as previously reported [6, 21]. The precise OCT analysis of the composition of the instent atherothrombotic burden is unreliable as the distinction between atheromatous tissue prolapse and thrombus is practically unfeasible after stenting. The clinical significance and the factors affecting the post-stenting atherothrombotic burden remain to be investigated by further adequately designed studies.

We did not find a significant association between the type of plaque and angiographic reperfusion indices. These results should be tempered by the small sample size and by the fact that all our patients had basal TIMI flow ≥ 2. Indeed, PR has been reported to be associated with higher risk of distal embolization [39], higher rates of no-reflow phenomenon and impaired TIMI flow and, larger infarct size after FL. Impaired angiographic reperfusion indices have also been reported in patients with greater residual thrombus assessed by OCT after aspiration thrombectomy or after stenting [42, 43]. Although our study may have lacked power to detect a significant difference, the numerically greater CTFC in the rupture group—pre- and post-stenting—may be considered as consistent with these studies. The larger thrombus extent in PR may also be a potential risk factor for distal embolization after stenting. However, one may also speculate that the use of OFDI may have influenced the choice of the diameter and the length of the stents and led to a more optimal plaque covering leading to both similar post-stenting atherothrombotic burden and myocardial reperfusion estimators in our study.

Limits

The findings of our study were based on a small cohort of patients who had successful FL and it may have lacked power to detect differences between some studied parameters. It could be difficult to differentiate PE from severe stenosis using OFDI but thrombus presence highlights an underlying endothelial injury process in this setting. Finally, it is possible that FL may be more effective on white thrombus mostly caused by erosion. However, the difference of thrombus amount between PR and PE in our cohort is consistent with previous studies without FL.

Conclusion

In the setting of PCI after successful FL for STEMI, the pre-PCI thrombus amount, typesetting and distribution assessed by OFDI seem mainly linked to the underlying mechanism of myocardial infarction. Compared to PE, PR is associated with dominantly red thrombus, higher thrombus burden and a more important longitudinal extent of thrombus across and afar the stenosis. Despite such differences, angiographic estimators of reperfusion and post-stenting atherothrombotic burden were similar between the two complicated plaque types.

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Compliance with ethical standards

Conflict of interest The authors have no conflicts of interest to declare.

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