International Journal of Cardiology 166 (2013) 646–651

Contents lists available at SciVerse ScienceDirect

International Journal of Cardiology

journal homepage: www.elsevier.com/locate/ijcard

Impact of tissue prolapse after stent implantation on short- and long-term clinical outcomes in patients with acute myocardial infarction: An intravascular ultrasound analysis

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ARTICLE INFO

Article history:
Received 25 July 2011
Received in revised form 12 October 2011
Accepted 26 November 2011
Available online 15 December 2011

Keywords:
Myocardial infarction
Stent
Prolapse
Intravascular ultrasound

ABSTRACT

Background: We used intravascular ultrasound (IVUS) to evaluate the association of tissue prolapse (TP) with short- and long-term outcomes after stent implantation in 418 acute myocardial infarction (AMI) patients.
Methods: We evaluated the incidences of stent thrombosis, no-reflow, and long-term outcomes between patients with TP (n = 142) and those without TP (n = 276).
Results: twb.42w?>Acute and subacute stent thromboses occurred more frequently in patients with TP compared with those without TP (3.5% vs. 0.7%, $p = 0.035$, and 4.2% vs. 0.7%, $p = 0.013$, respectively). TP volumes in 14 patients with stent thrombosis were significantly greater than those in 128 patients without stent thrombosis (3.3 ± 1.6 mm^3 vs. 2.6 ± 1.9 mm^3, $p = 0.012$). No-reflow was developed more frequently in patients with TP compared with those without TP (25.4% vs. 9.8%, $p < 0.001$). Creatine kinase-MB and cardiac-specific troponin I were elevated more significantly after stenting in patients with TP compared with those without TP ($\Delta = +9.0 \pm 25.2$ U/l vs. -4.2 ± 41.6 U/l, $p = 0.001$ and $\Delta = +10.0 \pm 43.5$ ng/ml vs. -1.2 ± 35.6 ng/ml, $p = 0.005$, respectively). There were no significant differences in the incidences of cardiac death, MI, and target vessel revascularization at 1-year. Multivariate analysis showed that TP was the independent predictor of composite of acute and subacute stent thromboses [odds ratio (OR) = 4.211; 95% CI 1.198–14.805, $p = 0.025$] and composite of acute stent thrombosis and no-reflow (OR = 2.551; 95% CI 1.315–4.952, $p = 0.006$).
Conclusions: TP was associated with poor short-term outcomes (more acute and subacute thromboses and no-reflow phenomenon), however it was not associated with worse long-term outcomes after stent implantation for infarct-related arteries in patients with AMI.

1. Introduction

Tissue prolapse (TP) is an intraluminal tissue (plaque and/or thrombus) extrusion through the stent struts and this can be easily and frequently detected by intravascular ultrasound (IVUS). Several studies have reported a 17% to 70% incidence of TP after stent implantation in patients with acute myocardial infarction (AMI) [1–4]. It is controversial whether TP can affect clinical outcomes. Although some study has reported that minor TP within stents might not be associated with long-term clinical outcome [2], another studies have reported that TP was associated with stent thrombosis [5–7] and post-stenting myonecrosis which was indicated by creatine kinase-MB or cardiac-specific troponin elevations [1–3].

Our hypothesis was that TP may not be a minor problem in terms of the impact on clinical outcomes after stent implantation. Therefore, we used IVUS to evaluate the association of TP and short- and long-term clinical outcomes after stent implantation for infarct-related arteries in patients with AMI.

2. Methods

2.1. Patient population

A total of 2123 patients with a first AMI were admitted to our institute from January 2007 to December 2009. Of these patients, we identified 418 consecutive patients with a first AMI who underwent pre-intervention IVUS within 24 h from symptom onset, were stented successfully, and had post-intervention IVUS imaging. All 418 infarct lesions were treated with stent implantation after balloon inflation: 105 with paclitaxel-eluting stents (Taxus® stent, Boston Scientific, Boston, Massachusetts), 74 with sirolimus-eluting stents (Cypher® stent, Cordis, Johnson and Johnson, Miami Lakes, Florida), 81 with zotarolimus-eluting stent (Endeavor® Sprint stent, Medtronic

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doi:10.1016/j.ijcard.2011.11.092

Inc., Minneapolis, Minnesota) and 158 with bare-metal stents. We excluded patients with severe heart failure or cardiogenic shock, hemodynamically unstable condition during or after the index procedure, ventricular tachyarrhythmia during hospitalization, important systemic disease, serum creatinine >2.5 mg/dl, prior MI, prior stent thrombosis, restenosis after stenting, coronary artery bypass graft failure, patients studied with IVUS more than 24 h after symptom onset at the time of admission, and patients in whom adequate IVUS images could not be obtained.

The diagnosis of AMI was according to a consensus document of the Joint European Society of Cardiology/American College of Cardiology Committee for the Redefinition of Myocardial Infarction [8]. Infarct-related arteries were identified using a combination of electrocardiographic findings, left ventricular wall motion abnormalities on left ventricular angiogram or echocardiogram, and coronary angiographic findings. All 418 infarct lesions were treated with stent implantation: 260 with drug-eluting stents and 158 with bare metal stents.

2.2. Laboratory analysis

Venous blood samples were obtained within 24 h after stenting. The blood samples were centrifuged, and serum was removed and stored at −70 °C until the assay could be performed. Absolute creatine kinase-MB levels were determined by radioimmunoassay (Dade Behring Inc., Miami, Florida). Cardiac-specific troponin I levels were measured by a paramagnetic particle, chemiluminescent immunoenzymatic assay (Beckman, Coulter Inc., Fullerton, California).

2.3. Coronary angiographic findings

Coronary angiogram was analyzed with validated QCA system (Phillips H5000 or Allura DCI program, Philips Medical Systems, the Netherlands). With the outer diameter of the contrast-filled catheter as the calibration standard, the reference diameter and the minimal lumen diameter were measured in diastolic frames from orthogonal projections. Perfusion was evaluated according to TIMI criteria [9].

2.4. IVUS imaging and analysis

All IVUS examinations were performed before and after stenting after intracoronary administration of 300 μg nitroglycerin using a commercially available IVUS system (Volcano Corp, Rancho Cordova, CA, USA). The IVUS catheter was advanced distal to the target lesion, and imaging was performed retrograde to the aorto-ostial junction at an automatic pullback speed of 0.5 mm/s.

IVUS analysis was performed according to the American College of Cardiology Clinical Expert Consensus Document on Standards for Acquisition, Measurement and Reporting of Intravascular Ultrasound Studies [10]. At pre-intervention, we measured external elastic membrane (EEM) and lumen cross-sectional area (CSA). Plaque plus media (P&M) CSA was calculated as EEM CSA minus lumen CSA, and plaque burden was calculated as P&M CSA divided by EEM CSA. Coronary artery remodeling was assessed by comparing the lesion site to the reference EEM CSA. Remodeling index was the lesion site EEM CSA divided by the average of the proximal and distal reference EEM CSA. Positive remodeling was defined as a remodeling index >1.05, intermediate remodeling as a remodeling index between 0.95 and 1.05, and negative remodeling as a remodeling index <0.95 [11]. Hypoechoic plaque was less bright compared with the reference adventitia. Hyperechoic, noncalcified plaque was as bright as or brighter than the reference adventitia without acoustic shadowing. Calcium plaque was hyperechoic with shadowing. A calcified lesion contained >90° of circumferential lesion calcium. A ruptured plaque contained a cavity that communicated with the lumen with an overlying residual fibrous cap fragment (Fig. 1A). A fragmented and loosely adherent plaque without a distinct cavity and without a fibrous cap fragment was not considered a plaque rupture. Rupture sites separated by a length of artery containing smooth lumen contours without cavities were considered to represent different plaque ruptures [12,13]. Plaque cavity was measured and extrapolated to the ruptured capsule area. Thrombus was an intraluminal mass having a layered or lobulated appearance, evidence of blood flow (microchannels) within the mass, and speckling or scintillation (Fig. 1B) [13,14]. A lipid-pool like image was defined as a pooling of hypoechoic or echolucent material covered with a hyperechoic layer.

At post-intervention, we measured the minimum stent CSA. Percent stent expansion was calculated as minimum stent CSA divided by mean reference lumen CSA. TP was defined as tissue extrusion through the stent strut at post-intervention (Fig. 1C) and it was intraluminal tissue including thrombus or plaque prolapse because we could not differentiate between thrombus and plaque prolapse using IVUS.

2.5. Short- and long-term clinical outcomes

The primary endpoint was a composite of acute and subacute (definite and probable) stent thromboses, as defined by the Academic Research Consortium [15]. The secondary endpoints included a) no-reflow after stenting, b) a composite of acute stent complications including acute stent thrombosis and no-reflow, c) incidence of post-hospital discharge cardiac death, MI, and target vessel revascularization at 1-year.

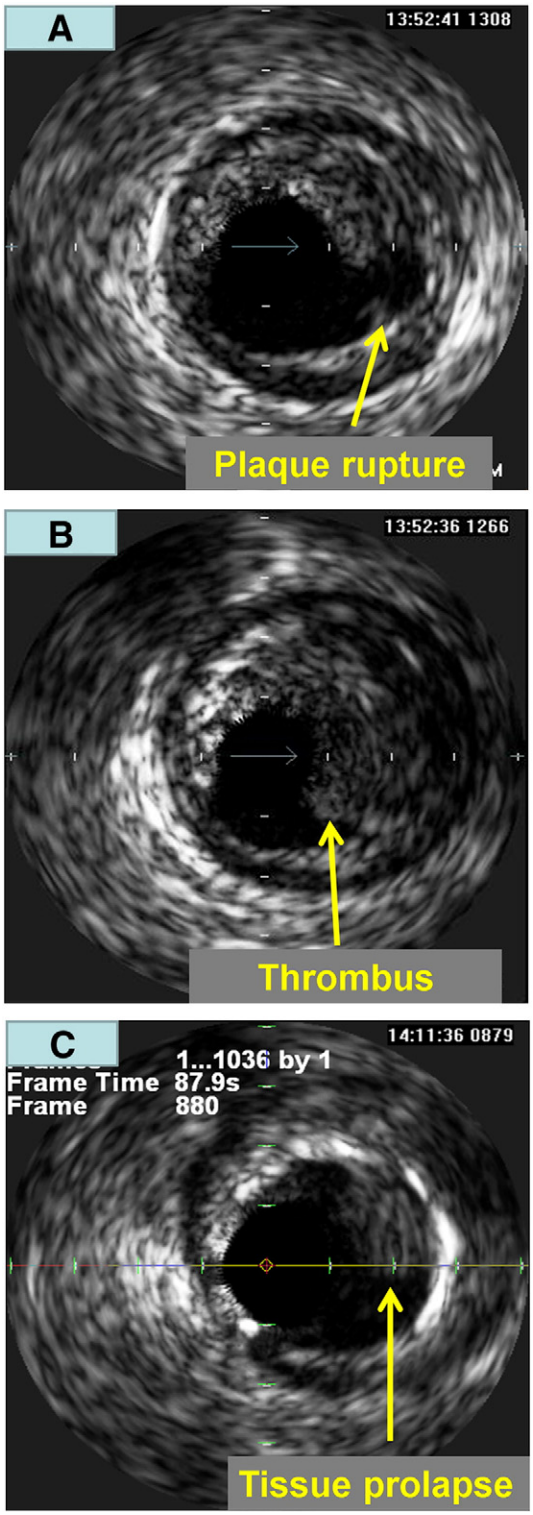

Fig. 1. The examples of plaque rupture (A) and intravascular ultrasound-detected thrombus (B) at pre-intervention and tissue prolapse (C) at post-intervention.

2.6. Statistical analysis

The statistical Package for Social Sciences (SPSS) for Windows, version 15.0 (Chicago, Illinois) was used for all analyses. Continuous variables were presented as the mean value ± 1SD; comparisons were conducted by Student's *t*-test or nonparametric Wilcoxon test if normality assumption was violated. Discrete variables were presented as percentages and relative frequencies; comparisons were conducted by chi-square statistics or Fisher's exact test as appropriate. Multivariate analysis was used to identify the independent predictors of composite of acute and subacute stent thromboses and