Impact of Distal Protection with Filter-Type Device on Long-term Outcome after Percutaneous Coronary Intervention for Acute Myocardial Infarction: Clinical Results with Filtrap®.

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Original Article

Impact of Distal Protection with Filter-Type Device on Long-term Outcome after Percutaneous Coronary Intervention for Acute Myocardial Infarction: Clinical Results with Filtrap®

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***Aim*: Although distal embolization during percutaneous coronary intervention (PCI) for acute myocardial infarction (AMI) deteriorates cardiac function, whether distal protection (DP) can improve prognosis is still controversial. We investigated whether a filter-type DP device, Filtrap®, could improve long-term outcomes after PCI for AMI.**
***Method*: We studied 164 patients (130 men, mean age: 65.7 years) who underwent PCI. Patients were divided into two groups based on the use of Filtrap®. The occurrence of congestive heart failure (CHF) and major adverse cardiac events (MACE) defined as cardiac death, recurrent AMI, and target vessel revascularization were compared.**
***Result*: Between DP (n=53, 41 men, mean age: 65.5 years) and non-DP (n=111, 89 men, mean age: 65.8 years) groups, although there was significantly greater plaque area in the DP group than in the non-DP group, there were no significant differences in coronary reperfusion flow after PCI. Interestingly, patients with CHF in the non-DP group exhibited a higher CK level than those in the DP group. During a 2-year follow-up period, cumulative CHF was significantly lower in the DP group than in the non-DP group (log-rank p=0.018), and there was no significant difference in the MACE rate (log-rank p=0.238). The use of DP device could not predict MACE, but could predict CHF by multivariate analysis (odds ratio=0.099, 95% CI: 0.02−0.42, p=0.005).**
***Conclusion*: These results demonstrate that favorable clinical outcomes could be achieved by the filter-type DP device in AMI, particularly in patients with CHF.**

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Key words: Distal protection, Acute myocardial infarction, Percutaneous coronary intervention

Introduction

It is evident that the occurrence of distal embolization and the associated slow-flow/no-reflow phenomenon after percutaneous coronary intervention (PCI) for acute myocardial infarction (AMI) result in poor prognoses[1-3]. However, under these conditions, some randomized clinical trials failed to show the superiority of the use of distal protection (DP) devices[4-6], although DP devices were considered to be effective in preventing distal embolization, particularly in experimental studies[7]. One of the reasons for these controversies may be explained by the fact that previous DP devices, such as balloon occlusion type, needed to interrupt coronary blood flow and, even under these conditions, resulted in incomplete protection of distal embolization[4, 8].

During the collection of materials that provoke distal embolization, filter-type DP devices[7, 9, 10] such as Filtrap® (Nipro Corporation, Tokyo, Japan) did not interrupt coronary flow in infarct-related vessels and

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needed less complicated procedures than previous DP devices. Indeed, experimental studies demonstrated that Filtrap® effectively protects distal coronary flow, preserving myocardial function after ischemia[7]. However, limited clinical data exists regarding filter-type DP devices, which are considered to be gentle to the coronary artery[11]. Therefore, the aim of this study was to investigate whether Filtrap® could improve reperfusion in primary PCI and to reveal long term outcomes.

Methods

Study Population

The study protocol was approved by the Bioethical Committee on Medical Research, Ishikawa Prefectural Central Hospital. Written informed consent for procedures was obtained from all patients before PCI. Between January 2007 and August 2010, 182 consecutive AMI patients (142 men, mean age: 66.1 years) who underwent emergent coronary interventions at our institutes were enrolled in this study. Of these, we examined 164 AMI patients (130 men, mean age: 65.7 years) who underwent intravascular ultrasound (IVUS) guided primary PCI. The remaining 18 patients had delivery failure of the Filtrap® device ($n=12$), a lesion at the bypass graft ($n=2$), and stent thrombosis after PCI procedures ($n=4$) and were thus excluded.

Procedures

All patients were pre-medicated with 200 mg of aspirin and received 8000 IU of heparin in a bolus injection. In addition, all patients were prescribed a loading dose of clopidogrel or ticlopidine after intervention. PCI was performed according to the standard clinical techniques with radial or femoral artery approaches using a 6Fr guiding catheter after intracoronary administration of isosorbide dinitrate. Culprit lesions were classified according to the AHA/ACC classification[12]. Intra-arterial flow was graded as 0−3 according to the Thrombolysis in Myocardial Infarction trial (TIMI) classification[13-15]. Collateral flow was graded according to Rentrop's score[16]. After passage of a 0.014-inch guidewire across the lesion, we performed a thrombus aspiration procedure to remove intracoronary thrombus by the aspiration device as much as possible. Then, we evaluated IVUS findings. If the IVUS catheter failed to deliver because of severe organic stenosis or calcification, dilatation by a small balloon (<2.5 mm) was added. According to the IVUS findings and angiographic information, the operator decided whether to deploy Filtrap® or not[15]. Then, we implanted a drug-eluting or bare metal stent. In the corrected TIMI frame count (CTFC) method, the number of frames required for the dye to reach a standardized distal landmark was counted[17]. Frames were counted by using images acquired at 30 frames per second with the 6Fr size of the catheter.

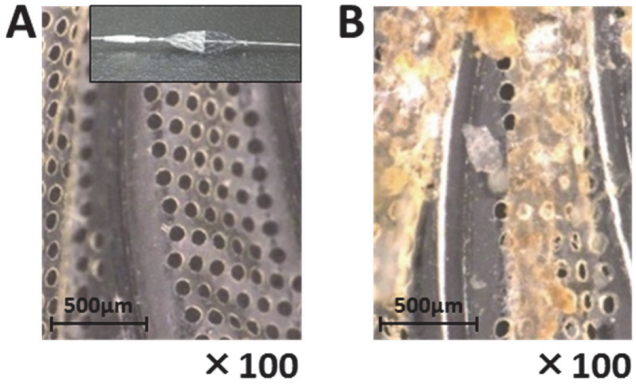

Fig. 1. View of the filter basket and internal side of the filter membrane of Filtrap® (NIPRO, Japan). A: Unused device, B: Membrane with the case of transient filter no-reflow. Note: a large amount of debris was observed.

The Basic Principle of Filtrap® (NIPRO, Japan)

Details of Filtrap® have already been described elsewhere[9, 15]. In brief, the filter is attached to the distal end of a 0.014-inch guidewire. The filter membrane is attached to half of the filter basket. The filter membrane is made of polyurethane and has approximately 1800 holes. Each hole size is approximately 100 μm, and the membrane is 40 μm thick (**Fig. 1A**).

Intravascular Ultrasound

IVUS (Eagle Eye Gold 2.9-F 20 MHz, Volcano Corporation, Rancho Cordova, USA) was performed before and after each procedure. IVUS images were recorded using a motorized transducer pull back system (0.5 m/s). IVUS studies were recorded and measured using computerized planimetry[18, 19]. We evaluated the sections with the smallest luminal cross-sectional area, distal reference, and proximal reference. The reference site was selected as the most visually normal section with the largest lumen and smallest plaque cross-sectional area within 10 mm proximal to the target lesion. The total vessel area was measured by tracing the outer border of the external elastic membrane (EEM), and the lumen area was measured by tracing the interface between the vessel lumen and leading edge of the initial echogenic layer. The plaque area was calculated as the difference between the vessel area and lumen area.

Plaque burden was defined as plaque area divided by the EEM cross-sectional area[20]. The remodeling index was the ratio of lesion site EEM cross-sectional area divided by the average of the proximal and distal

Table 1. Baseline patient characteristics

Variable	Total (n=164)	DP group (n=53)	Non-DP group (n=111)	p value
Age (years)	65.7±12.4	65.5±13.6	65.8±11.8	0.89
Males	130 (79%)	41 (77%)	89 (80%)	0.68
BMI (kg/m^2)	24.1±3.5	23.9±3.7	24.3±3.4	0.57
Diabetes mellitus	63 (38%)	23 (43%)	40 (36%)	0.37
Hypertension	97 (59%)	30 (57%)	67 (60%)	0.65
Dyslipidemia	107 (65%)	32 (60%)	75 (67%)	0.45
Current smoker	73 (44%)	26 (49%)	47 (42%)	0.42
Previous MI	11 (7%)	3 (6%)	8 (7%)	0.71
Serum creatinine (mg/dl)	0.89±0.69	0.82±0.25	0.92±0.81	0.39
eGFR (ml/min./1.73 m^2)	74.0±25.4	75.5±25.7	73.2±25.3	0.6
Systolic blood pressure (mmHg)	131.5±29.7	123.8±29.0	135.2±29.5	<0.01*
Heart rate (/min.)	73.3±21.2	68.9±17.4	75.5±22.5	0.04*
QRS score (mV)	27.4±36.4	36.8±34.6	22.8±36.6	0.02*
LVEF (%)	49.2±10.5	49.9±9.7	48.9±10.8	0.57
Killip class Ⅲ or Ⅳ	19 (12%)	1 (2%)	18 (16%)	<0.01*
Diagnosis				
STEMI	147 (90%)	49 (92%)	98 (88%)	0.42
NSTEMI	17 (10%)	4 (8%)	13 (12%)	0.42

Continuous data are presented as mean±standard deviation; categorical data are presented as a number (%)
*$p<0.05$ indicates that the DP group is significantly different from the non-DP group.
DP distal protection, *BMI* body mass index, *MI* myocardial infarction, *eGFR* estimated glemerular filtration rate, *LVEF* left ventricular ejection fraction, *STEMI* ST elevation myocardial infarction, *NSTEMI* non-ST elevation myocardial infarction

reference EEM cross-sectional areas[21, 22].

Study Outcomes

We divided 164 patients into two groups based on the use of Filtrap®. Angiogram, IVUS findings, CTFC, ST resolution, and peak CK levels were compared between both the groups. We evaluated the incidence of major adverse cardiac events (MACE) defined as cardiac death, recurrent acute myocardial infarction, and ischemia-driven target vessel revascularization (TVR) during a 2-year follow-up. We also evaluated the incidence of congestive heart failure (CHF) and malignant arrhythmia during a 2-year follow-up. CHF was defined as New York Heart Association Class Ⅲ or Ⅳ requiring intra-venous medication (including diuretics, vasodilators, or inotropic agents) for objective evidence of congestion or left ventricular dysfunction. In addition, we evaluated the incidence of hospitalization due to a worsening of CHF during a 2-year follow-up. Malignant arrhythmia included sustained ventricular tachycardia and ventricular fibrillation.

ECG was performed when patients were admitted to the hospital and at 60 min after reperfusion. The QRS score was defined as the sum of QRS voltages in all 12 leads on the basis of a previous study[23]. ST-segment elevation was summed from all the infarct-related leads on the baseline ECGs and from the same leads on the post-PCI ECGs to analyze ST resolution in the ST elevation myocardial infarction cases. The percentage of ST resolution from the baseline to 60 min after the procedure was calculated. We defined an ST resolution >70% as complete ST resolution[10]. Follow-up data were obtained from hospital charts and information from patients' general physicians as well as by a direct interview or a telephone interview with patients or one of their close relatives.

Statistical Analysis

SPSS version 21.0 (SPSS Inc., Chicago, IL) was used for all analyses. Categorical data were expressed as absolute frequencies and percentages and were compared using a Chi square test or Fisher's exact test, as appropriate. Continuous variables, which were expressed as the mean±standard deviation (SD), were compared using either an unpaired Student's t-test or the Mann−Whitney U test. Cumulative incidences were estimated using the Kaplan−Meier method, and differences were assessed with the log-rank test. Multiple logistic regression analysis was used to determine the independent predictors for outcomes. A p value of <0.05 was considered statistically significant.

Table 2. Pre-procedural morphologic characteristics

Variable	Total ($n=164$)	DP group ($n=53$)	Non-DP group ($n=111$)	p value
Lesion location				
LMT	6 (3.7%)	1 (1.9%)	5 (4.5%)	0.41
LAD	59 (36.0%)	13 (24.5%)	46 (41.4%)	0.04*
LCX	20 (12.2%)	2 (3.8%)	18 (16.2%)	0.02*
RCA	80 (48.8%)	37 (69.8%)	43 (38.7%)	<0.01*
Lesion AHA/ACC types				
A	0 (0%)	0 (0%)	0 (0%)	NS
B1	16 (9.8%)	2 (3.7%)	14 (12.6%)	0.03*
B2	139 (84.7%)	48 (90.6%)	91 (82.0%)	0.13
C	9 (5.5%)	3 (5.7%)	6 (5.4%)	0.41
Collateral (≥grade 2) received	42 (26%)	19 (36%)	23 (21%)	0.04*
Multivessel disease	94 (57%)	26 (49%)	68 (61%)	0.14
IVUS findings				
Proximal reference				
EEM-CSA (mm^2)	17.7±5.0	19.2±5.3	17.0±4.6	0.01*
Lumen diameter (mm)	3.2±0.6	3.3±0.5	3.0±0.6	<0.01*
Distal reference				
EEM-CSA (mm^2)	14.2±5.2	16.5±4.6	13.1±5.1	<0.01*
Lumen diameter (mm)	2.9±0.6	3.2±0.6	2.7±0.5	<0.01*
Reference diameter (mm)	3.0±0.5	3.3±0.5	2.9±0.5	<0.01*
Lesion site				
EEM-CSA (mm^2)	17.2±5.3	19.8±5.7	16.1±4.7	<0.01*
Plaque area (mm^2)	14.4±5.2	16.7±5.7	13.2±4.6	<0.01*
Plaque burden (%)	82.1±6.9	83.1±8.2	81.5±6.1	0.21
Remodeling index	1.10±0.22	1.12±0.18	1.09±0.24	0.5

Continuous data are presented as mean±standard deviation; categorical data are presented as a number (%)
*$p<0.05$ indicates that the DP group is significantly different from the non-DP group.
LMT left main trunk, *LAD* left anterior descending artery, *LCX* left circumflex artery, *RCA* right coronary artery, *NS* not significant, *EEM* external elastic membrane, *CSA* cross-sectional area

Results

Baseline Demographics and Lesion Characteristics

DP was performed in 53 patients, but not in 111 patients (**Table 1**). Although there were no significant differences in patients' basic background between both the groups, patients in the DP group showed lower systolic blood pressure and lower heart rate at admission than those in the non-DP group. In addition, the DP group had a higher incidence of Killip class Ⅲ or Ⅳ. The QRS score was lower in the DP group than in the non-DP group. As for pre-procedural angiographic characteristics, Filtrap® was more frequently used for RCA lesions than for LAD or LCX lesions. The DP group received more sufficient collateral artery than the non-DP group (**Table 2**).

Pre-procedural IVUS findings are shown in **Table 2**. The EEM cross-sectional area in the proximal reference site was significantly greater in the DP group than in the non-DP group (19.2±5.3 mm^2 vs. 17.0±4.6 mm^2, $p=0.01$). The relations were similar in the EEM cross-sectional area of the distal reference site (16.5±4.6 mm^2 vs. 13.1±5.1 mm^2, $p<0.01$) and of the lesion site (19.8±5.7 mm^2 vs. 16.1±4.7 mm^2, $p<0.01$). Although pre-procedural IVUS imaging demonstrated significantly greater plaque area in the DP group than in the non-DP group (16.7±5.7 mm^2 vs. 13.2±4.6 mm^2, $p<0.01$), plaque burden was equivalent in both the groups (83.1±6.9% vs. 81.5±6.1%, $p=0.21$).

Outcomes

Table 3 shows the TIMI flow grade before and after PCI. Overall, TIMI flow grade clearly improved after PCI. Although there were no significant differences in TIMI flow grade 3 and CTFC after PCI between both the groups, the incidence of TIMI flow grade 0-1 after PCI was significantly lower in the DP

Table 3. Procedural results

Variable	Total (n=164)	DP group (n=53)	Non-DP group (n=111)	p value
Pre-procedure				
TIMI flow grade				
0-1	128 (78.0%)	41 (77.4%)	87 (78.4%)	0.88
2	30 (18.3%)	9 (17.0%)	21 (18.9%)	0.76
3	6 (3.7%)	3 (5.6%)	3 (2.7%)	0.41
Post-procedure				
TIMI flow grade				
0-1	4 (2.4%)	0 (0%)	4 (3.6%)	0.04*
2	27 (16.5%)	10 (18.9%)	17 (15.3%)	0.58
3	133 (81.1%)	43 (81.1%)	90 (81.1%)	0.99
Corrected TIMI frame count	26.2 ± 17.3	25.0 ± 14.2	26.8 ± 18.6	0.52

Continuous data are presented as mean ± standard deviation; categorical data are presented as a number (%)
*p < 0.05 indicates that the DP group is significantly different from the non-DP group.
TIMI thrombolysis in myocardial infarction

Table 4. Multivariate logistic regression analysis of baseline variables, procedural findings in predicting CHF and MACE

Variable	Odds ratio	95% CI	p value
For CHF			
Age	1.129	1.06-1.21	< 0.01
LVEF	0.946	0.9-0.99	0.04
Distal protection device usage	0.099	0.02-0.42	< 0.01
Peak CK	1.003	1.0001-1.0006	< 0.01
For MACE			
DM	3.688	1.57-9.16	< 0.01
Killip Ⅲ or Ⅳ	4.497	1.36-15.99	0.02

CHF congestive heart failure, *MACE* major adverse cardiac events, *CI* confidence interval, *DM* diabetes mellitus, *LVEF* left ventricular ejection fraction

group than in the non-DP group. ST resolution and peak CK level were not different in both the groups (**Table 5**).

In multivariate logistic regression analysis, diabetes mellitus and Killip class Ⅲ or Ⅳ predicted MACE independently (**Table 4**). The use of DP device did not predict MACE independently. Meanwhile, age, LVEF, DP device usage, and peak CK were independent predictors of CHF following multivariate analysis (**Table 4**).

There was no significant difference in the incidence of MACE up to a 2-year follow-up between the two groups (p=0.21). Interestingly, the incidence of CHF was significantly higher in the non-DP group than in the DP group (21.6% vs. 5.7%, p=0.01) (**Table 5**). Importantly, the cumulative incidence of CHF over 2 years was significantly lower in the DP group than in the non-DP group (log-rank p=0.018) (**Fig. 2**).

Fig. 3 shows the results of the detailed sub-analysis of the relationships between peak CK level in the acute phase and CHF during the 2-year follow-up. On the whole, patients with CHF (n=27) showed higher CK levels than those without CHF (n=137) (5154 ± 4516 IU/L vs. 2786 ± 1925 IU/L, p=0.012). In the DP group, the CK level was not different in patients with or without CHF (2298 ± 1672 IU/L vs. 3281 ± 2187 IU/L, p=0.449). However, in the non-DP group, the CK level was significantly higher in patients with CHF than in those without CHF (5511 ± 4649 IU/L vs. 2502 ± 1707 IU/L, p=0.005) with a background of lower LVEF in patients with CHF than in those without CHF (41.7% vs. 50.8%, p<0.01). Despite this underlying bias in cardiac function, DP usage and LVEF predicted CHF independently (**Table 4**). Interestingly, patients with CHF after the non-DP procedure exhibited a higher CK level than that in patients with CHF after the DP procedure (p=0.049),

Table 5. Clinical results in a hospital and during a two-year follow-up

Variable	Total ($n=164$)	DP group ($n=53$)	Non-DP group ($n=111$)	p value
Complete ST resolution	51 (34.7%)	17 (32.1%)	34 (30.6%)	1.00
Peak CK (IU/L)	3176 ± 2728	3225 ± 2240	3152 ± 2942	0.86
MACE up to two years	34 (20.7%)	14 (26.4%)	20 (18.0%)	0.21
Cardiac death	9 (5.5%)	2 (3.8%)	7 (6.3%)	0.51
Myocardial infarction	11 (6.7%)	5 (9.4%)	6 (5.4%)	0.33
Target vessel revascularization	17 (10.4%)	7 (13.2%)	10 (9.0%)	0.39
VF/VT	13 (8.0%)	2 (3.8%)	11 (9.9%)	0.17
CHF	27 (16.5%)	3 (5.7%)	24 (21.6%)	0.01*

Continuous data are presented as mean ± standard deviation; categorical data are presented as a number (%)
*$p<0.05$ indicates that the DP group is significantly different from the non-DP group.
MACE major adverse cardiac events, *VF* ventricular fibrillation, *VT* ventricular tachycardia, *CHF* congestive heart failure

even with a background of similar LVEF between the two groups (39.4% vs. 41.7%, $p=0.721$).

Discussion

The two main findings of this study are summarized further. Firstly, Filtrap® usage was an independent predictor for the CHF-free status of patients, and the long-term CHF rate was significantly lower in the DP group than in the non-DP group during the 2-year follow-up. Secondly, although there were no differences in CK levels between the DP and non-DP groups, a DP procedure may induce lower CK levels than a non-DP procedure in particular cases of CHF.

Several studies have suggested that pre-stenting IVUS findings are associated with the occurrence of the angiographic slow-flow/no-reflow phenomenon during PCI in patients with AMI[19, 24-27]. Tanaka *et al* reported that a lesion EEM cross-sectional area ($p<0.05$; odds ratio: 1.55; 95% CI: 1.01−2.38) served as an independent predictive factor of the no-reflow phenomenon after reperfusion for AMI[26]. Another study noted that the vessel area and plaque area in the culprit lesion in patients in which transient no-reflow occurred during PCI were significantly greater than in patients without no-reflow[27]. Thus, in this study, the risk of slow-flow/no-reflow was higher in the DP group than in the non-DP group because the DP group had greater EEM and plaque area than the non-DP group.

Although CTFC in the final angiogram was not different in both the groups, Filtrap® usage successfully and significantly reduced the incidence of the most serious TIMI 0-1 (**Table 3**). In this study, there was no significant difference in the MACE rate up until the 2-year follow-up between both the groups. Although there was no difference in plaque burden at the lesion site between both the groups, the absolute quantity of plaque at the lesion was greater in the DP group than in the non-DP group. Therefore, it was possible that the DP group could have a worse prognosis. From this point of view, use of Filtrap® preserved myocardial perfusion and resulted in less CHF compared with the non-DP group.

Causes of the no-reflow phenomenon are considered to be multifactorial. There are four considerable pathogenic mechanisms of the no-reflow phenomenon: 1) distal atherothrombotic embolization[28], 2) ischemic injury, 3) reperfusion injury, and 4) susceptibility of coronary microcirculation to injury[29]. Large emboli (>200 μm in diameter) can obstruct pre-arterioles, and thrombus formation may be associated with inadequate antiplatelet effects of clopidogrel, which is related to cytochrome P450 2C19 polymorphisms[30]. Ischemic injury, which is caused by reduced regional myocardial blood flow, causes endothelial protrusions, interstitial edema, and myocardial cell swelling. Filtrap® cannot prevent all these factors, but one positive effect is thought to be its ability to trap most embolization (**Fig. 1B**). Indeed, filter slow-flow/no-reflow, which suggests the capture of sufficient debris, was found in the DP group, as was also observed in our present study[15].

In the association between outcomes and Filtrap® usage, the frequency of CHF was significantly lower in the DP group than in the non-DP group (5.7% vs. 21.6%, $p=0.01$). This difference was found at an early stage after PCI. Interestingly, multivariate regression analysis showed that Filtrap® usage was one of the independent predictors of CHF (**Table 4**). It is possible that there could be considerable myocardial necrosis in patients with CHF because they exhibited higher peak CK levels than patients without CHF. In addition, the CK level of patients with CHF in the non-DP group was significantly higher than those with

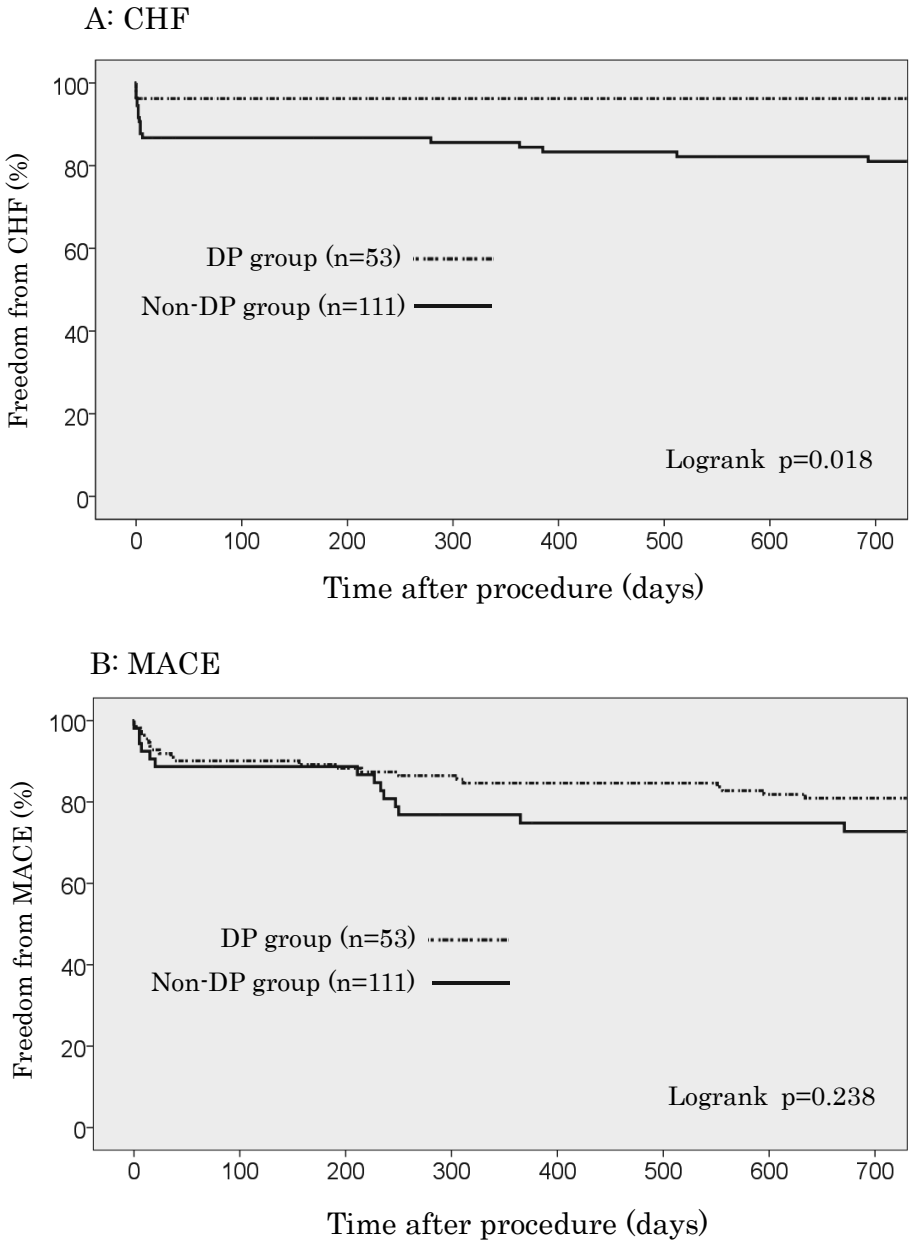

Fig. 2. Kaplan-Meier curves of the clinical outcomes during a two-year follow-up in patients treated with or without Filtrap®. A: The cumulative occurrence of CHF was significantly lower in the DP group than in the non-DP group (3.8% vs. 17.1%, log-rank $p=0.018$). B: The cumulative occurrence of MACE was similar in 2 groups (26.4% vs. 18.9%, log-rank $p=0.238$).

CHF congestive heart failure, *MACE* major adverse cardiac events

CHF in the DP group, whereas the CK level was similar among patients without CHF regardless of DP (**Fig. 3**). There could be a considerable mechanism for difference in the protective effect of the DP device between patients with CHF and those without CHF, as follow: patients without CHF in the non-DP group ($n=87$) had smaller vessels and less plaque areas in the infarct-related coronary artery than patients without CHF in the DP group ($n=50$) (reference diameter: 2.85 mm vs. 3.35 mm, $p<0.01$; plaque area: 13.0 mm^2 vs. 16.5 mm^2, $p<0.01$). We have inferred that small vessels and plaques could have contributed to a numerically low CK level in patients without CHF, even without usage of the DP device.

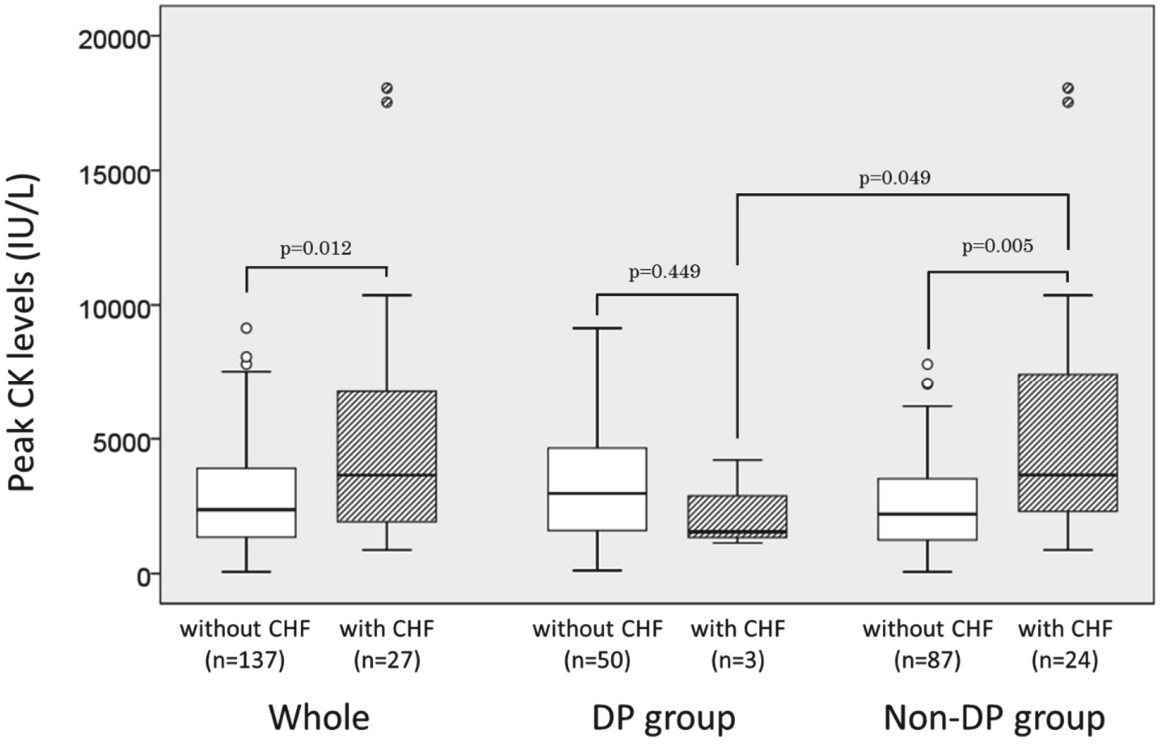

Fig. 3. Comparison of peak CK level after PCI in patients with or without congestive heart failure (CHF) during a two-year follow-up. On the whole, patients with CHF ($n=27$) showed higher CK levels than those without CHF ($n=137$) (5154 ± 4516 IU/L vs. 2786 ± 1925 IU/L, $p=0.012$). In the DP group, the CK level was not different in patients with or without CHF (2298 ± 1672 IU/L vs. 3281 ± 2187 IU/L, $p=0.449$). In the non-DP group, the CK level was significantly higher in the patients with CHF than those without CHF (5511 ± 4649 IU/L vs. 2502 ± 1707 IU/L, $p=0.005$). Note that the patients with CHF in the non-DP group exhibited higher CK levels than in patients with CHF after the DP procedure (5511 ± 4649 IU/L vs. 2298 ± 1672 IU/L, $p=0.049$).

CHF congestive heart failure

Several studies describing the use of DP devices during primary PCI have shown heterogeneous results probably because of different population subsets, a variety of surrogate endpoints, and differences in the protection device technology used[31-34]. Recent clinical trials that examined distal filter-type devices, which were structurally different from Filtrap®, failed to demonstrate an improved clinical outcome in patients with AMI[35, 36]. Filtrap® has a spindle-shaped spiral Ni−Ti wire basket. This unique self-expandable structure, which gently attaches to the vessel wall, may efficiently retrieve thrombosis and is less invasive than previous products. There were no complications when Filtrap® was used in our study. The procedure is relatively easy and can be performed in a short period of time, supported by Filtrap®, which is useful for primary PCI with AMI patients. Further investigation is warranted to elucidate the efficacy of the filter-based DP device Filtrap® on selected high risk patients with distal embolization.

Limitations

This study has several potential limitations. First, this study was a retrospective single-center experience in a limited number of patients. However, even under these conditions, the use of Filtrap® exhibited a preferable outcome, particularly in patients with CHF. Second, the clinical backgrounds, such as the artery involved, the hemodynamic state before PCI, Killip class, and the QRS score, were different between both the groups, thus indicating that several undefined biases may have influenced the outcome. Meanwhile, DP usage was still an independent predictor for CHF after adjustment of these factors. Third, incomplete embolic protection may have occurred because of the limited size of Filtrap®, although all patients in the DP group were angiographically considered to be adapted.

Conclusion

This study demonstrated the possible ability of Filtrap® to prevent CHF in AMI patients.

Acknowledgments

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Conflicts of Interest

The authors report no financial relationships or conflicts of interest regarding the content herein.

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