Cilostazol Reduces Angiographic Restenosis After Endovascular Therapy for Femoropopliteal Lesions in the Sufficient Treatment of Peripheral Intervention by Cilostazol Study

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Background—It remains unclear whether cilostazol, which has been shown to improve the clinical outcomes of endovascular therapy for femoropopliteal lesions, also reduces angiographic restenosis.

Methods and Results—The Sufficient Treatment of Peripheral Intervention by Cilostazol (STOP-IC) study investigated whether cilostazol reduces the 12-month angiographic restenosis rate after percutaneous transluminal angioplasty with provisional nitinol stenting for femoropopliteal lesions. Two hundred patients with femoropopliteal lesions treated from March 2009 to April 2011 at 13 cardiovascular centers were randomly assigned 1:1 to receive oral aspirin with or without cilostazol. The primary end point was 12-month angiographic restenosis rate. Secondary end points were the restenosis rate on duplex ultrasound, the rate of major adverse cardiac events, and target lesion event-free survival. Researchers evaluated all follow-up data and assessed the end points in a blinded fashion. The mean lesion length and reference vessel diameter at the treated segment were 128±86 mm and 5.4±1.4 mm, respectively. The frequency of stent used was similar between groups (88% versus 90% in the cilostazol and noncilostazol group, respectively, P=0.82). During the 12-month follow-up period, 11 patients died and 152 patients (80%) had evaluable angiographic data at 12 months. The angiographic restenosis rate at 12 months was 20% (15/75) in the cilostazol group versus 49% (38/77) in the noncilostazol group (P=0.0001) by intention-to-treat analysis. The cilostazol group also had a significantly higher event-free survival at 12 months (83% versus 71%, P=0.02), although cardiovascular event rates were similar in both groups.

Conclusions—Cilostazol reduced angiographic restenosis after percutaneous transluminal angioplasty with provisional nitinol stenting for femoropopliteal lesions.


Key Words: angioplasty ■ peripheral vascular disease ■ restenosis

F emoropopliteal (FP) lesions are found in 60% to 70% of patients with symptomatic peripheral artery disease (PAD).1–3 Restenosis occurs in 40% to 60% of patients with FP disease by 12 months after percutaneous balloon angioplasty, which has been the standard and traditional endovascular revascularization procedure.4–6 Recently, implantation of nitinol stents has improved the long-term outcome of endovascular therapy (EVT) for FP lesions in comparison with balloon angioplasty.7,8 and guidelines have been revised to extend the indications of EVT because of these improvements.9,10

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However, despite the use of stents, there remains a 20% to 50% incidence of restenosis at 1 year as an important challenge for endovascular intervention.1-6

Current guidelines do not recommend any specific medical intervention to reduce the incidence of restenosis after EVT for FP disease. Several studies have shown that medical intervention with statins and cilostazol decreases the risk of restenosis after EVT for FP disease.11-13 However, all had limitations such as a small sample size or use of inappropriate methods and criteria to determine the incidence of restenosis. The present study therefore was designed to determine by angiographic follow-up whether treatment with cilostazol reduces restenosis at 12 months after percutaneous transluminal angioplasty (PTA) with provisional nitinol stenting for FP disease.

Methods

Study Design

This was a prospective, randomized, open-label, multicenter study. During the period from March 2009 to March 2011, 200 patients with symptomatic PAD attributable to de novo FP lesions were enrolled and randomly assigned. Analysis of angiographic data and diagnosis of restenosis at 12 months after EVT was routinely done at a core laboratory in an end point–blinded manner.

Randomization

Patients with symptomatic PAD who were classified as greater than Rutherford 1 were screened for noninvasive tests to detect limb ischemia and the presence of de novo FP lesions. Patients were excluded if (1) they had been treated with drug-eluting coronary stents; (2) they had heart failure, defined as a history of hospital admission owing to heart failure or the presence of heart failure symptoms with systolic or diastolic dysfunction evaluated by cardiac echocardiography; (3) they had an FP lesion with inflow aortoiliac lesions; (4) they had FP lesions with severe calcification that preclude adequate balloon dilatation; and (5) they had poor below-the-knee runoff defined as number of below-the-knee runoff <1. After informed consent was obtained, eligible patients were randomly assigned to groups with or without cilostazol treatment. Randomization was done centrally by using a computer algorithm. Within 1 week after randomization, each patient was admitted and underwent PTA with provisional nitinol stenting. Patients in the noncilostazol group received aspirin at 100 mg/d for 12 months, whereas those in the cilostazol group received the same dose of aspirin plus cilostazol (200 mg/d) for 12 months. Patients who received stents in both groups were also treated with a thienopyridine for 1 month after the procedure to prevent acute stent occlusion.

Endovascular Therapy

The lesion was dilated by using a balloon with a diameter equal to the reference vessel diameter, as determined by quantitative angiography. After balloon inflation for at least 1 minute, stenting was done if there was flow-limiting dissection, a pressure gradient ≥10 mm Hg, or >30% residual stenosis. Patients received S.M.A.R.T stents (Cordis Corp, Miami Lakes, FL) with a diameter 1 mm larger than the reference vessel diameter.

Follow-Up Protocol

At 1, 3, 6, and 12 months after EVT, each patient was assessed for symptoms, the ankle-brachial index (ABI) was measured, and duplex ultrasound was performed to assess the presence of restenosis. At 12 months after EVT, patients who had not died without any reintervention were routinely scheduled for readmission at the study institution and underwent follow-up angiography to determine whether there was restenosis at the treated lesion. Quantitative angiographic data obtained before EVT, immediately after EVT, and 12 months after EVT were analyzed at a core laboratory with the use of CAASV software ver. 5.7 (Pie Medical Imaging).

End Points

The primary end point of this study was the angiographic restenosis rate at 12 months after EVT. Secondary end points were as follows: (1) the restenosis rate assessed by duplex ultrasound at 1, 3, 6, and 12 months after EVT; (2) angiographic findings including minimum lumen diameter, late lumen loss (percentage), the percentage of diameter stenosis, and the frequency of stent fracture at 12 months; (3) event-free survival; (4) temporal changes in ABI and Rutherford category after EVT; and (5) major adverse cardiovascular events, which were defined as all-cause death, myocardial infarction, and stroke.

Definitions

Event-free survival was defined as the freedom from death, major amputation, clinically driven target lesion revascularization, and target limb ischemia requiring surgical intervention. Clinically driven target lesion revascularization was defined as reintervention performed for >50% diameter stenosis identified by angiography within 5 mm of the target lesion after documentation of recurrent clinical symptoms of PAD. Angiographic restenosis was defined as recurrence of ≥50% diameter stenosis as determined by quantitative vascular angiography. A peak systolic velocity ratio of >2.0 on Duplex ultrasound was interpreted as indicating restenosis according to the objectives, performance goals of the Vascular Interventional Advances group.14 Initial success was defined as ≤30% residual stenosis and a pressure gradient ≤10 mm Hg.

Statistical Analysis

The sample size was estimated based on the binary restenosis rates from 2 previous trials.12,13 An overall sample size of 200 subjects was expected to have 82% power to detect a difference in 12-month angiographic restenosis rate for cilostazol in comparison with noncilostazol at a 2-sided α of 0.05, assuming a binary restenosis rate of 30% in the cilostazol group and 50% in the noncilostazol group. All analyses were based on intention-to-treat (ITT) analyses. We compared the primary outcome of the 12-month angiographic restenosis rate and other dichotomous outcomes between the groups by using the χ² test with an odds ratio and its 95% confidence interval. Per protocol set (PPS) analyses were also done to evaluate the robustness of the conclusion from the ITT analyses for the primary outcome. To account for the correlation among repeated measures from the same patient, the secondary end points of restenosis rates, ABI, and Rutherford classification were analyzed with the use of generalized estimating equations, and a Bonferroni adjusted probability value was calculated for the group comparisons at multiple time points, where baseline was included as a covariate for ABI, and Rutherford classification. Event-free survival was estimated with the use of the Kaplan-Meier method and was compared between the groups by the log-rank test. In addition, we performed subgroup analyses to investigate the effects of sex, diabetes mellitus, end-stage renal disease on dialysis, Rutherford classification, TransAtlantic Inter-Society Consensus II classification, length of target lesion, reference vessel diameter, chronic total occlusion, poor below-the-knee runoff, number of stents, and stent implantation on the effect of cilostazol. The treatment-by-subgroup interaction was assessed for all subgroups; we either calculated the odds ratios between subgroups or used interaction terms in logistic regression models. All probability values are 2 sided. Statistical analyses were done with SAS version 9.3 for Windows. This study was registered at ClinicalTrials.gov (NCT00912756).

Study Participants

Figure 1 shows the disposition of the 200 patients who were enrolled and randomly assigned to receive cilostazol (n=100; cilostazol group) or not to receive cilostazol (n=100;
noncilostazol group). After randomization, 9 patients (7 in the cilostazol group and 2 in the noncilostazol group; P=0.17) were withdrawn from the study without undergoing EVT. Of the 7 patients withdrawn from the cilostazol group, 1 took the drug and achieved symptomatic improvement, 2 had no FP disease on baseline angiography, and 4 withdrew their consent to the study after randomization. Of the 2 patients withdrawn from the noncilostazol group, 1 had no FP disease on baseline angiography, and the other withdrew consent after randomization. During the 12-month follow-up period after EVT, 11 patients died (7 versus 4 patients in the cilostazol versus the noncilostazol group) and 28 patients (11 versus 7 patients in the cilostazol versus the noncilostazol group) were lost to follow-up angiography. Thus, 80% of the enrolled patients (152/191) were evaluable at 12 months angiography after EVT.

Baseline Characteristics and Angiographic Data

The baseline clinical characteristics of the ITT population, which are summarized in Table 1, were similar for the 2 groups, with the exception of preprocedural ABI. Baseline angiographic data for the target lesions are shown in Table 2. The mean length of the treated segment was 132±90 mm in the cilostazol group versus 125±82 mm in the noncilostazol group, whereas the proximal reference vessel diameter was 5.4±1.4 mm and 5.3±1.4 mm, respectively. There was no significant difference between the 2 groups with regard to the TransAtlantic Inter-Society Consensus classification of the lesions or the frequency of complete occlusion. The frequency of stent used also was similar between groups (88% [82/93] versus 90% [88/98], for the cilostazol and noncilostazol group, respectively; P=0.82).

Primary End Point

The angiographic restenosis rate (primary end point) is compared between the 2 groups in Figure 2. Of the 191 patients undergoing PTA with provisional stenting, 152 patients (80%) had evaluable angiographic data at 12 months. By ITT analysis, the angiographic restenosis rate at 12 months was 20% (15/75) in the cilostazol group in comparison with 49% (38/77) in the noncilostazol group (P=0.001; odds ratio, 0.26; 95% confidence interval, 0.13–0.53). Four patients in the cilostazol group and 1 patient in the noncilostazol group were excluded from the PPS. In the cilostazol group, 1 patient developed a headache and another had a skin rash. For the patient who developed a headache, the dose of cilostazol was reduced from 200 mg/d to 100 mg/d, whereas the doses of both cilostazol and ticlopidine were reduced for the patient who had a skin rash. The remaining 2 patients underwent implantation of drug-eluting stents (DES) for new coronary lesions, which led to a change of antiplatelet therapy from cilostazol to clopidogrel. In the noncilostazol group, a patient was excluded because of acute renal failure. Thus, analysis of the PPS revealed that the angiographic restenosis rate at 12 months was 21% (15/71) in the cilostazol group versus 50% (38/76) in the noncilostazol group (P=0.003; odds ratio, 0.27; 95% confidence interval, 0.13–0.55; Figure I in the online-only Data Supplement).
Secondary End Points

At 12 months after the index procedure, the assessment of restenosis by duplex ultrasound was done in 87% (167/191) of patients.

Restenosis Rate at 1, 3, 6, and 12 Months After EVT

Figure 3 show the restenosis rates assessed by duplex ultrasound at different times up to 12 months. Both ITT and PPS analyses showed no significant differences between the 2 groups at 3 or 6 months. However, both analyses revealed that the restenosis rate at 12 months was significantly lower in the cilostazol group than in the noncilostazol group (ITT, 22% versus 45%; \(P=0.006\) [Figure 3]; PPS, 22% versus 46%; \(P=0.005\) [Figure II in the online-only Data Supplement]).

Angiographic Findings

Table 3 shows angiographic outcomes at 12 months. The cilostazol group had a larger minimum lumen diameter (3.1±1.5 mm versus 2.2±1.1 mm; \(P<0.001\)), a smaller late lumen loss (1.1±0.6 mm versus 1.4±0.7 mm; \(P=0.03\)) and percentage of diameter stenosis (39±23% versus 52±22%; \(P<0.001\)), leading to a lower rate of target lesion revascularization (17% versus 40%) by ITT analysis. The frequency of stent fracture in the overall cohort was 18%, without a significant difference between the groups (\(P=0.64\)).

Event-Free Survival

Figure 4 shows event-free survival. At 12 months, event-free survival was significantly higher in the cilostazol group than in the noncilostazol group (83% versus 71%; \(P=0.02\)). During the study period, there were no major amputations in either group.
Changes in ABI and Rutherford Classification

Figure 5 shows the changes in ABI over time. Although preprocedural ABI was significantly higher in the cilostazol group than in the noncilostazol group, there were no other significant differences between the groups. At 12 months, ABI was not statistically different between groups ($P=0.20$). Figure 6 shows the changes in Rutherford classification. There were no other significant differences between the groups preprocedure, and at 1, 3, and 6 months; at 12 months, it was significantly lower in the cilostazol group than in the noncilostazol group ($P=0.03$).

Major Adverse Cardiovascular Events

The frequency of major adverse cardiovascular events was similar during follow-up between the 2 groups (12% [11/93] in the cilostazol group versus 9% [9/98] in the noncilostazol group; $P=0.64$).

Finally, subgroup analysis of the influence of cilostazol on 12-month angiographic restenosis is shown in Figure 7. There was no significant treatment-by-subgroup interaction for all subgroups, and the greater benefit of cilostazol versus noncilostazol was consistent across all subgroups.

Discussion

Main Findings of This Study

This prospective, randomized, multicenter study was conducted to determine whether cilostazol treatment reduces the incidence of angiographic restenosis rate after PTA with provisional nitinol stenting for FP disease. The angiographic patency rate at 1 year after PTA with provisional nitinol stenting was 76% in patients receiving cilostazol treatment in comparison with 51% in patients not receiving it, and cilostazol significantly reduced angiographic restenosis after EVT for FP disease. The cilostazol group also had a significantly higher event-free survival rate at 12 months, although the rate of cardiovascular events was similar between groups.

Comparisons With Previous Studies – What Are the Differences?

Comparison With Conventional EVT and Stent Placement

EVT with nitinol stenting is associated with a lower incidence of restenosis after treatment of PAD, and this has led to a dramatic
revision of the TransAtlantic Inter-Society Consensus guidelines for the management of PAD. In the Vienna Absolute Trial: Balloon Angioplasty Versus Stenting in the Superficial Femoral Artery (ABSOLUTE), patients who underwent stenting for target lesions with a mean length of 101±75 mm had a stent fracture rate of 2% (1/49) and a 12-month restenosis rate of 37%.7 

As is the case for coronary intervention, DES for the superficial femoral artery has recently been introduced, and this has further improved the outcome. The Zilver-PTX stent is a DES that has been reported to achieve 1-year patency of 83% for lesions with a mean length of 6.6 cm.15 Nevertheless, the risk of restenosis when peripheral lesions are treated with DES remains higher than after coronary intervention and should be reduced further.

Optimal Antiplatelet Therapy After EVT for PAD

It is recommended that patients who undergo EVT for PAD should receive antiplatelet therapy. However, there are no specific recommendations regarding the optimum drugs or duration of treatment. Administration of statins and cilostazol has been reported to reduce the risk of restenosis; however, previous studies had various limitations with regard to the method of detecting restenosis and sample size.

Detection of Restenosis

Duplex ultrasound is the standard method for the detection of restenosis after EVT for FP disease. However, the diagnostic threshold of the peak systolic velocity ratio for restenosis has not been standardized, being variously reported as 2.0 to 2.8.14,16-18

The present study provides evidence that cilostazol reduces the incidence of angiographic restenosis at 12 months after PTA with provisional nitinol stenting. In patients treated with cilostazol, the restenosis rate was 20% for FP disease with a mean lesion length of 124±88 mm despite a stent fracture rate of 16%. This long-term outcome was better than that previously reported after EVT with nitinol stenting. The lower incidence of restenosis in patients receiving cilostazol probably resulted from the improvement of outflow because of its vasodilatory effect and the suppression of neointimal hyperplasia attributable to its anti-inflammatory effect together with the improvement of vascular endothelial function and the inhibition of vascular smooth muscle cell proliferation.

The restenosis rate evaluated by duplex ultrasound was similar for the 2 groups at 3 and 6 months after EVT, but was significantly different at 12 months. A similar trend was observed in recent randomized, controlled trials of EVT with stenting for PAD. In a study that evaluated the efficacy of EVT with everolimus-eluting stents (Superficial Femoral Artery Treatment with Drug-eluting Stents [STRIDES] trial),19 the 12-month restenosis rate was a high 68%, which was in sharp contrast to a patency rate of 94% at 6 months. In patients treated with Zilver-PTX stents, however, the lesion patency rate was 95% at 6 months and 83% at 12 months in comparison with respective rates of 88% and 73% in patients receiving bare metal stents. The smaller reduction of the patency rate with this stent over time indicates that its effect persists for up to 12 months. Similar to the effect of DES, treatment with cilostazol for 12 months after EVT for PAD prevented restenosis from 6 to 12 months, a period with a high incidence of restenosis, thereby producing a significant reduction of restenosis at 12 months.

Figure 4. Event-free survival (ITT analysis). At 12 months, event-free survival was significantly higher in the cilostazol group than in the noncilostazol group (83% versus 71%; \( P=0.02 \)). ITT indicates intention-to-treat.

Figure 5. Changes in ABI (ITT analysis). ABI was assessed at 6 times: pre- and postprocedure, and at 1, 3, 6, and 12 months. Although preprocedural ABI was significantly higher in the cilostazol group than in the noncilostazol group, there were no other significant differences between the groups. At 12 months, ABI was not statistically different between groups (\( P=0.20 \)). ABI indicates ankle-brachial index; and ITT, intention-to-treat.
Clinical Implications – What Does This Study Teach Us?

The results of this study suggest that cilostazol can be used as first-line antiplatelet therapy for reducing the incidence of restenosis in patients undergoing EVT with stenting for FP disease. In our ITT population, the 12-month restenosis rate was 20% in the cilostazol group in comparison with 49% in the noncilostazol group.

Limitations

1. This study was not double blinded and was not placebo controlled. However, the angiographic data were analyzed in an end point–blinded manner.

<table>
<thead>
<tr>
<th>Overall</th>
<th>Cilostazol # of Restenosis / Total # (%)</th>
<th>Non-Cilostazol</th>
<th>P</th>
<th>Odds ratio (95% CI)</th>
<th>P for interaction</th>
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<td>18/82(22) / 38/85(45)</td>
<td>38/54(15) / 22/56(39)</td>
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<td>0.52 (0.13-2.05)</td>
<td>0.35</td>
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<td>16/29(55) / 15/36(42)</td>
<td>34/54(28) / 23/49(47)</td>
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<td>42/12(25) / 51/38(47)</td>
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<td>0.65 (0.25-1.65)</td>
<td>0.65</td>
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<td>Rutherford classification</td>
<td>2:5</td>
<td>3:4</td>
<td>6:10</td>
<td>0.007</td>
<td>0.65 (0.25-1.65)</td>
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<td>TASC II classification</td>
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<td>3:4</td>
<td>6:10</td>
<td>0.007</td>
<td>0.65 (0.25-1.65)</td>
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<td>Length of target lesion</td>
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<td>3:4</td>
<td>6:10</td>
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<td>Reference vessel diameter</td>
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<td>6:10</td>
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<td>6:10</td>
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<td>3:4</td>
<td>6:10</td>
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<td>0.007</td>
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Subgroup analysis for efficacy of cilostazol on 12 months angiographic restenosis

Figure 7. Subgroup analysis of the influence of cilostazol on 12-month angiographic restenosis (ITT analysis). Odds ratios for 12-month restenosis in selected patient subgroups are shown. The probability values for interaction indicate that none of the variables had a significant interaction with treatment. BTK indicates below-the-knee; CI, confidence interval; ITT, intention-to-treat; and TASC II, TransAtlantic Inter-Society Consensus II.
2. EVT was performed by using first-generation bare metal stents. At the start of this study, the S.M.A.R.T stent was the only one available for peripheral lesions in Japan. However, newer-generation stents such as nitinol stents or DES have not sufficiently reduced the incidence of restenosis in comparison with the effect of such stents on percutaneous coronary intervention. Cilostazol may be the most effective option to reduce the incidence of restenosis in patients treated for PAD with bare metal stents, with maximum risk of occurrence at 6 to 12 months after intervention. 

3. Rutherford classification at 12 months was significantly lower in the cilostazol group than in the noncilostazol group, whereas ABI at 12 months was not significantly different between groups. The discrepancy between changes in symptom and objective testing results also has been documented elsewhere.

To this end, cilostazol has been reported to have additional beneficial effects on arterial compliance, symptomatology, and quality of life without associated significant improvement in objective testing parameters including ABI. Although ABI is an effective and practical method for diagnosis of PAD with 95% sensitivity, 5% of false negatives remain. In this study, the discrepancy potentially might be secondary to a high incidence of noncompliant vessel calcification leading to overestimation. ABI is helpful in the clinical setting; however, its use for the assessment of treatment response is limited because patients can frequently improve walking distances without any significant change in ABI. Duplex or angiogram, instead of ABI, therefore is mandatory for assessing outcomes after endovascular reconstruction.

4. Finally, this study population is Asian and results should be confirmed in other ethnic groups.

**Conclusion**

Cilostazol reduces the risk of restenosis in patients undergoing PTA with provisional stenting for FP disease.

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**Disclosures**

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**References**


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Femoropopliteal (FP) lesions are found in 60% to 70% of patients with symptomatic peripheral artery disease. Although implantation of nitinol stents has improved the long-term outcome of endovascular therapy for FP lesions, there remains a 20% to 50% incidence of restenosis at 1 year as an important challenge for endovascular intervention. Current guidelines do not recommend any specific medical intervention to reduce the incidence of restenosis after endovascular therapy for FP disease. The Sufficient Treatment of Peripheral Intervention by Cilostazol (STOP-IC) study investigated whether cilostazol reduces the 12-month angiographic restenosis rate after percutaneous transluminal angioplasty with provisional nitinol stenting for FP lesions. The angiographic patency rate at 1 year after endovascular therapy was 80% in patients receiving cilostazol treatment in comparison with 51% in patients not receiving it, and cilostazol significantly reduced angiographic restenosis after endovascular therapy for FP disease. The cilostazol group also had a significantly higher event-free survival rate at 12 months, although the rate of cardiovascular events was similar in the 2 groups (83% versus 71%; P=0.02). Cilostazol reduced angiographic restenosis after percutaneous transluminal angioplasty with provisional nitinol stenting for FP lesions.
Cilostazol Reduces Angiographic Restenosis After Endovascular Therapy for Femoropopliteal Lesions in the Sufficient Treatment of Peripheral Intervention by Cilostazol Study
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Supplemental Material
Supplemental Figure 1

Restenosis rate (%)

OR: 0.27
(95% CI: 0.13, 0.55)
P = 0.0003

Non-Cilostazol
50%
(38/76)

Cilostazol
21%
(15/71)
Supplemental Figure 2

![Bar chart showing restenosis rate (%) at 3, 6, and 12 months for Non-Cilostazol and Cilostazol groups.](image)

- **3Mo:** 7% (6/83) for Non-Cilostazol, 10% (8/79) for Cilostazol, P=0.87
- **6Mo:** 18% (15/83) for Non-Cilostazol, 17% (13/77) for Cilostazol, P=1.00
- **12Mo:** 46% (38/83) for Non-Cilostazol, 22% (17/77) for Cilostazol, P=0.005

Legend:
- □ Non-Cilostazol
- ■ Cilostazol
Supplemental Figure Legends

Supplemental figure 1.

Twelve-month angiographic restenosis rates following PTA with provisional nitinol stenting for symptomatic de novo femoropopliteal lesions in the cilostazol group and the non-cilostazol group. (Primary endpoint, PPS analysis)

The angiographic restenosis rate was significantly lower in the cilostazol group than in the non-cilostazol group. By PPS analysis, the patency rate was 21% and 50%, respectively (p=0.0003).

Supplemental figure 2.

The 12-month restenosis rate evaluated by duplex ultrasound following PTA with provisional nitinol stenting for symptomatic de novo femoropopliteal lesions in the cilostazol group and the non-cilostazol group. (Secondary endpoint, PPS analysis)

Restenosis was defined as a peak systolic velocity ratio>2.0. According to PPS analysis, restenosis rates were similar between the cilostazol and non-cilostazol groups at 3 and 6 months, whereas the restenosis rate was
significantly lower in the cilostazol group at 12 months.
STOP-IC participants: interventional cardiologist, participating

JPN centers (overall number of endovascular intervention for femoropopliteal lesions and patients enrolled into this trial); asterisk denotes centers taking part in the STOP-IC)

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