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# Poor 1-year outcomes following percutaneous coronary interventions in systemic lupus erythematosus: Report from the National Heart, Lung and Blood Institute Dynamic Registry

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## Abstract

**Background—**Women with systemic lupus erythematosus (SLE) have premature and accelerated atherosclerosis. Although percutaneous coronary intervention (PCI) is utilized frequently to treat coronary artery disease (CAD) in SLE, little is known regarding PCI outcomes immediately post-PCI and after discharge.

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Dr. Williams receives grant support from Cordis, Boston Scientific, and Abbott Vascular.
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Subject Codes: [3] acute coronary syndromes; [4] acute myocardial infarction; [116] restenosis; [24] catheter-based coronary interventions: stents

**Methods and Results—**Baseline demographic, procedure-related and adverse outcome data on consecutive patients undergoing PCI during 5 recruitment "waves" of the National Heart, Lung, and Blood Institute Dynamic Registry across 23 clinical centers were collected. SLE patients (n= 28) were compared to nonSLE patients (n=3385). SLE patients were younger and more often female in comparison to nonSLE patients undergoing PCI. SLE patients were less likely than nonSLE patients to have hyperlipidemia, but had a similar prevalence of hypertension, diabetes mellitus, and tobacco use. The prevalence of multi-vessel disease was similar between groups. Initial intervention success (by angiographic definition) was not significantly different between groups. At one year, SLE patients were more likely to suffer a myocardial infarction (MI) (15.6% vs. 4.8%, p=0.01), and more often required repeat PCI (31.3% vs. 11.8%, p=0.009) than nonSLE patients, even following adjustment for important covariates.

**Conclusions—**SLE patients had significantly worse CV outcomes at one year than nonSLE patients. Even considering the small number of SLE patients, these differences were striking. Further study is warranted to explore other factors potentially accounting for this disparity, including SLE disease activity and duration, presence of hypercoagulable state, and immunosuppressive therapy.

## Keywords

angioplasty; catheterization; restenosis; revascularization; systemic lupus erythematosus

The advent of glucocorticoid therapy in the 1950s was pivotal in improving survival in patients with SLE. However, despite this advance, the actuarial 5-year survival for all SLE patients in the 15 years following was only 50% (1). This finding was further investigated in a study by Urowitz *et al,* in which a bimodal mortality pattern in SLE was identified. In this series, patients who died within a few years of diagnosis often succumbed to the effects of disease or infection associated with immunosuppressive therapy, whereas those dying later following disease onset frequently had clinically quiescent SLE with autopsy evidence of acute myocardial infarction (MI) (2). Both autopsy and epidemiologic studies have provided further evidence for premature subclinical atherosclerosis and cardiovascular events in SLE patients. Traditional risk factors alone are insufficient to account for these findings (3-5).

Little is known about outcomes of coronary revascularization in SLE. Although Ward *et al* found that in-hospital outcomes for MI in SLE were similar between SLE and nonSLE patients, the only systematic study examining outcomes of coronary artery bypass grafting (CABG) in patients with connective tissue diseases (including a subset of patients with SLE) found lower survival and a higher frequency of re-intervention (mean follow-up: 35 months) when compared to the general population (6,7). Population-based studies have demonstrated subgroups of patients in whom coronary revascularization outcomes are poorer. Given the high prevalence of the metabolic syndrome, insulin resistance, and other comorbidities that characterize SLE patients, their course might be anticipated to parallel that of patients with DM and thus portend a poorer outcome.

The National Heart, Lung, and Blood Institute (NHLBI) Dynamic Registry is a multicenter, prospective observational study designed to characterize the practice of PCI and to report clinical outcomes. This comprehensive database provides a unique opportunity to examine cardiovascular risk factors, pharmacologic therapy, and both short and mid-term outcomes following PCI. We compared outcomes of PCI between patients with SLE and nonSLE patients.

## Patients and Methods

### Study population

The Dynamic Registry consists of 23 medical centers in North America, with a coordinating center at the University of Pittsburgh. Baseline demographic and procedure-related data on consecutive patients undergoing PCI during 5 recruitment "waves" were collected. No information about admission medications was recorded. A total of 10,962 patients undergoing PCI (wave 1: July 1997-February 1998, n = 2524; wave 2: February 1999-June 1999, n = 2105; wave 3: October 2001-March 2002, n = 2047; wave 4: February 2004-May 2004, n=2112, and wave 5: February 2006-August 2006, n=2174) comprised the parent sample.

Patients enrolled in Waves 1-3 were contacted by telephone to obtain follow up data at their one year anniversary, while patients in waves 4 and 5 were contacted at one 1 month, 6 months, and annually. Follow-up data for discharged patients who consented to be contacted were available in 100% of SLE patients and 95.5% of nonSLE patients. Medical records were reviewed whenever possible for patients requiring repeat hospitalizations.

Patients with SLE (n=28) were identified by review of entry forms, where noncardiac diagnoses were entered as a write-in item. We selected patients whose diagnosis was entered as "systemic lupus erythematosus" or "SLE". To reduce potential misclassification bias, non-SLE patients were drawn from the same centers (n=14) reporting SLE.

Death was defined as all cause mortality. Myocardial infarction was defined as the presence of electrocardiographic or biochemical evidence of myocardial necrosis. The primary endpoint major adverse cardiac event (MACE) included death, MI, and any repeat revascularization (repeat PCI or CABG) procedure. Repeat PCI included both target and non-target vessel interventions.

Each center's Institutional Review Board approved the protocol, and informed consent to collect information during and/or after hospital discharge was obtained from all patients.

### Statistical analysis

Patient characteristics pertaining to the index PCI, including demographics, medical history, cardiac presentation, peri-procedural medications, procedural characteristics, and in-hospital outcomes were compared by the non-parametric Wilcoxon rank sum test for continuous variables and chi-square test for categorical variables. Similar methods were used for lesion-level analyses. Associations between recruitment wave and medications were compared for trend using the Cochran-Mantel-Haenszel test for both patients with and without SLE. One-year event rates were calculated using the Kaplan-Meier approach, and comparisons of survival curves were performed using the log-rank test. Patients who did not experience the outcome of interest were censored at the last known date of contact or at one year if contact extended beyond one year.

Cox proportional hazards modeling was used to assess the independent effect of SLE on select one-year adverse outcomes. Unadjusted hazard ratios for 1-year adverse cardiac events were calculated initially followed by the calculation of adjusted hazard ratios after the inclusion of (1) pre-specified variables (age, sex, vessel disease, presence of diabetes mellitus, history of myocardial infarction, reason for revascularization, acuity of PCI, and year of the procedure) and (2) only important covariates identified by forward stepwise selection (entry p value criterion of $\leq 0.25$, retain criteria of $\leq 0.05$). Proportional hazards assumptions were evaluated and met for all Cox proportional hazards models.

The authors had full access to and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

## Results

### *Baseline clinical characteristics* (Table 1)

Combining the 5 waves of recruitment from the 14 centers in which SLE patients were identified, a total of 3,385 patients were studied. In the entire cohort, just under half of patients were over the age of 65, 64.3% were male, and 72.6% were Caucasian. Twenty-eight patients with SLE were identified, with a significantly younger mean age (55.0 years) and fewer males (10.7%) but similar ethnicity (64.3% Caucasian).

Mean and median body mass index were similar between SLE and nonSLE patients. The frequency of prior PCI and CABG procedures was not significantly different between SLE and nonSLE patients. SLE patients were less likely to have hyperlipidemia (50.0% vs. 71.5%, p=0.01) than nonSLE patients. The prevalence of hypertension, diabetes mellitus, and tobacco use were similar between groups.

### Indications for intervention and associated therapies (Table 2)

There were no significant differences between SLE and nonSLE patients in reasons for revascularization (asymptomatic CAD, stable angina, unstable angina, and MI). The need for urgent and emergent procedures was similar between groups. Periprocedural use of aspirin (ASA), thienopyradines, or heparin did not significantly different between groups.

### Baseline cardiac function and anatomic features

The prevalence of abnormal LV function and mean and median ejection fractions was similar in SLE and nonSLE patients. There were no significant differences in coronary artery dominance or the prevalence of multivessel disease between groups. SLE patients were more likely to have isolated LAD lesions (42.3% vs. 19.3%, p=0.003) than nonSLE patients.

Characteristics of attempted lesions were similar between groups pertaining to tortuosity, mean lesion length, mean percent stenosis, total occlusion, collateral supply, Class-C type, or pre-procedural TIMI flow.

### Procedure details

Lesions technically amenable to PCI, and mean and median numbers of lesions treated were similar between groups. Over three-quarters of lesions attempted in both groups were in native vessels. The use of balloons and stents was similar among groups. The percentage of patients receiving drug-eluting stents was also similar when restricting the analysis to include the periods after the approval of such stents.

### *Adverse events and in-hospital outcomes*

The overall angiographic procedural success rate and attainment of TIMI 3 flow was > 95%, and not significantly different between groups. The incidence of major dissection, embolization, side branch occlusion, and abrupt closure occurred infrequently (<5%), and these rates were not significantly different between groups. There were no significant differences in peri-procedural MI, CABG, ventricular fibrillation, stroke, thrombus, major entry site complications, mean length of hospital stay, or in-hospital deaths.

### Discharge medications

Discharge medications varied considerably across groups, as demonstrated in Table 3. Patients with SLE were less likely to be discharged on aspirin therapy (78.6% vs. 96.2%, $p<0.0001$) and more likely to be discharged on warfarin (28.6% vs. 7.3%, $p<0.0001$) than nonSLE patients. The use of cholesterol-lowering agents and antiplatelet agents as a class was similar between groups. There was no significant difference between groups in the use of medication administered as part of a research study.

We also examined the trends of medication use in subsequent waves, realizing that standard of care practice often changes over time. As shown in Table 4, in earlier waves, SLE patients were less likely to receive medications considered standard of care for secondary prevention, but by the last recruitment wave, this discrepancy had nearly disappeared.

### Adverse outcomes

At four months, the need for repeat PCI in SLE patients was significantly higher compared to nonSLE (Figure 1). This difference remained statistically significant at one year. The majority of SLE patients requiring repeat PCI required reintervention in the same vessel as the index procedure. SLE patients were also more likely to have an MI in the year following PCI (Table 5, Figure 2). Even after adjustment for important covariates, at one year the risk of MI was 3-fold greater in SLE patients (HR 3.58), and they were nearly twice as likely to require repeat PCI (HR 1.98).

All SLE patients with MI required repeat PCI. Of the 4 SLE patients who underwent more than 1 repeat PCI, 2 subsequently required CABG. Overall risk for CABG between SLE and nonSLE patients at one year was not significant different, and patients with SLE were not more likely to die by one year. Although overall MACE were more frequent in SLE patients (Figure 3), this difference was not statistically significant after adjusting for important covariates.

## Discussion

Despite a lower prevalence of traditional risk factors associated with CV events in comparison to nonSLE patients, SLE patients were more likely to require repeat PCI and to have an MI during one year follow-up after PCI in the NHLBI Dynamic Registry. These adverse outcomes are most apparent in the first four months post-PCI, when thrombosis is most likely. Aspirin therapy was less likely to be utilized in patients with SLE at the time of hospital discharge, especially in earlier recruitment waves. The differences in outcomes between SLE and nonSLE patients at four months post-PCI suggest that restenosis or plaque destabilization in SLE may happen more aggressively, or that SLE patients receive different post-procedural management that adversely impacts outcomes of PCI. As yet, no SLE-specific mechanisms have been identified. However, considering the role of inflammation and other novel CV risk factors in atherogenesis and prediction of events, in a disease characterized by increased, sustained immune activation, several potential factors can be considered.

The association between markers of a procoagulant state with vessel restenosis and recurrent CV events following PCI has been documented. Even when controlling for traditional risk factors, patients with anticardiolipin antibodies but no underlying autoimmune disease who undergo PCI had significantly higher rates of restenosis at one year (40% vs. 14%, $p< 0.01$) than anticardiolipin-negative patients (8). Additionally, Marcucci *et al* demonstrated that levels of plasminogen activator inhibitor-1 (PAI-1) and homocysteine levels are independent risk factors for MACE following PCI for ACS (9). Furthermore, it has been noted that the prevalence of anticardiolipin antibodies is higher in SLE patients (44%) when compared to nonSLE controls (9%) (10,11). SLE patients also have been found to have higher levels of

PAI-1, homocysteine, and tissue factor when compared with controls (12,13). The frequency of warfarin administration in SLE patients in this study suggests an increased prevalence of a hypercoagulable state or prior thrombotic event, and may account for the low frequency of ASA therapy at hospital discharge in early recruitment waves. The Dynamic Registry did not include information on anticoagulants nor other indicators of hypercoagulability at PCI admission; therefore we cannot ascertain if aspirin and warfarin usage at hospital discharge were influenced by such pre-existing conditions. Event numbers were too small to determine associations between events and increased ASA utilization across waves in the SLE patients.

Patients receiving warfarin therapy often are not given ASA because of increased risk of hemorrhage. However, it has been demonstrated that combined antiplatelet therapy is associated with fewer CV events than warfarin + ASA therapy following PCI, with the role of warfarin + an antiplatelet agent other than ASA less clearly defined (14,15). Given this data, the use of triple therapy (warfarin + 2 antiplatelet agents) following PCI for the first 4-12 weeks in patients requiring chronic anticoagulation has been suggested (16). However, studies examining bleeding complications associated with this regimen have found conflicting results (17,18). Without a better understanding of the precipitants of post-PCI events in SLE, it remains difficult to weigh risk vs. benefit of a more aggressive approach to antiplatelet therapy in SLE patients requiring chronic anticoagulation therapy.

In considering the data linking inflammation to CVD and CV risk, one might assume that this would be an important factor in SLE patients, who have increased, sustained immune activation and systemic inflammation. The data examining acute phase reactants and their association with surrogate markers of CAD in SLE have been inconclusive. However, it may be that these markers better reflect conditions that promote plaque destabilization or thrombosis rather than indicating the presence of plaque. Studies from the general population demonstrate association between markers of inflammation and increased levels of matrix metalloproteinases, oxidant stress, and tissue factor, all of which are purported to have a role in plaque destabilization and/or thrombosis (19-23). Inflammation has also been linked to other novel markers of CV risk, including lipoprotein particle size, acute phase HDL, protein nitration, and protein glycoxidation (24-27). The utility of these markers in the prediction of CVD and CV events in SLE has not been determined.

Complement activation and associated immunologic responses are a key aspect of SLE pathogenesis. Complement also has a purported role in vascular injury, atherogenesis, and plaque destabilization in the general population (28-30). Multiple cohort studies examining the association between disease-associated markers and surrogate markers of CAD in SLE found a positive association between CAD and elevated levels of C3. This is reminiscent of the association between elevated levels of C3 with the presence of CAD and CV events found in the general population (31,32). However, the significance of this relationship in SLE and the general population remains unclear.

Previous studies have demonstrated that CV risk factor assessment and management is often suboptimal in SLE patients. In 2004, Costenbader *et al* found that 23% of SLE patients receiving care at an academic center had never had cholesterol screening, and in those with hypercholesterolemia, only 21% were receiving medical therapy (33). Additionally, only 58% of all modifiable risk factors received any intervention. Although ASA and HMG-CoA reductase inhibitors are considered standard of care for secondary prevention of CAD, in our SLE group, only 70% were discharged on ASA, and just 50% on statin therapy, although usage patterns increased over the five recruitment waves. Patients from earlier recruitment waves, especially those with normal cholesterol levels, were less likely to be placed on statin therapy, which is true for the entire cohort. As a referral center, we continue to see SLE patients who have never had CV risk factor assessment, or have known risk factors that were suboptimally

managed, suggesting that this remains a problem with an undetermined impact on CV outcomes.

Our study has several limitations. Our SLE population was small, and defined by a write-in diagnosis. Although our chart survey of a subgroup of SLE patients to assess the accuracy of this means of identifying patients did not identify any misdiagnoses, in future studies the diagnosis of SLE can be better substantiated by prospectively collected supportive data. Furthermore, prospectively gathered information regarding disease duration and activity, medical therapy, and coexisting hypercoagulable state is essential to further examine the role of disease-related factors in the poor PCI outcomes noted in this population in future studies. Despite these limitations, we believe that this work highlights a previously unrecognized vulnerability in SLE patients that mandates vigilant post-PCI attention.

The dismal outcomes in SLE patients following PCI in this study are striking. These results highlight the potential importance of post-interventional secondary preventative measures in SLE, and provide an impetus to dedicate further study directed at identifying modifiable risk factors to improve CV outcomes in SLE patients undergoing PCI.

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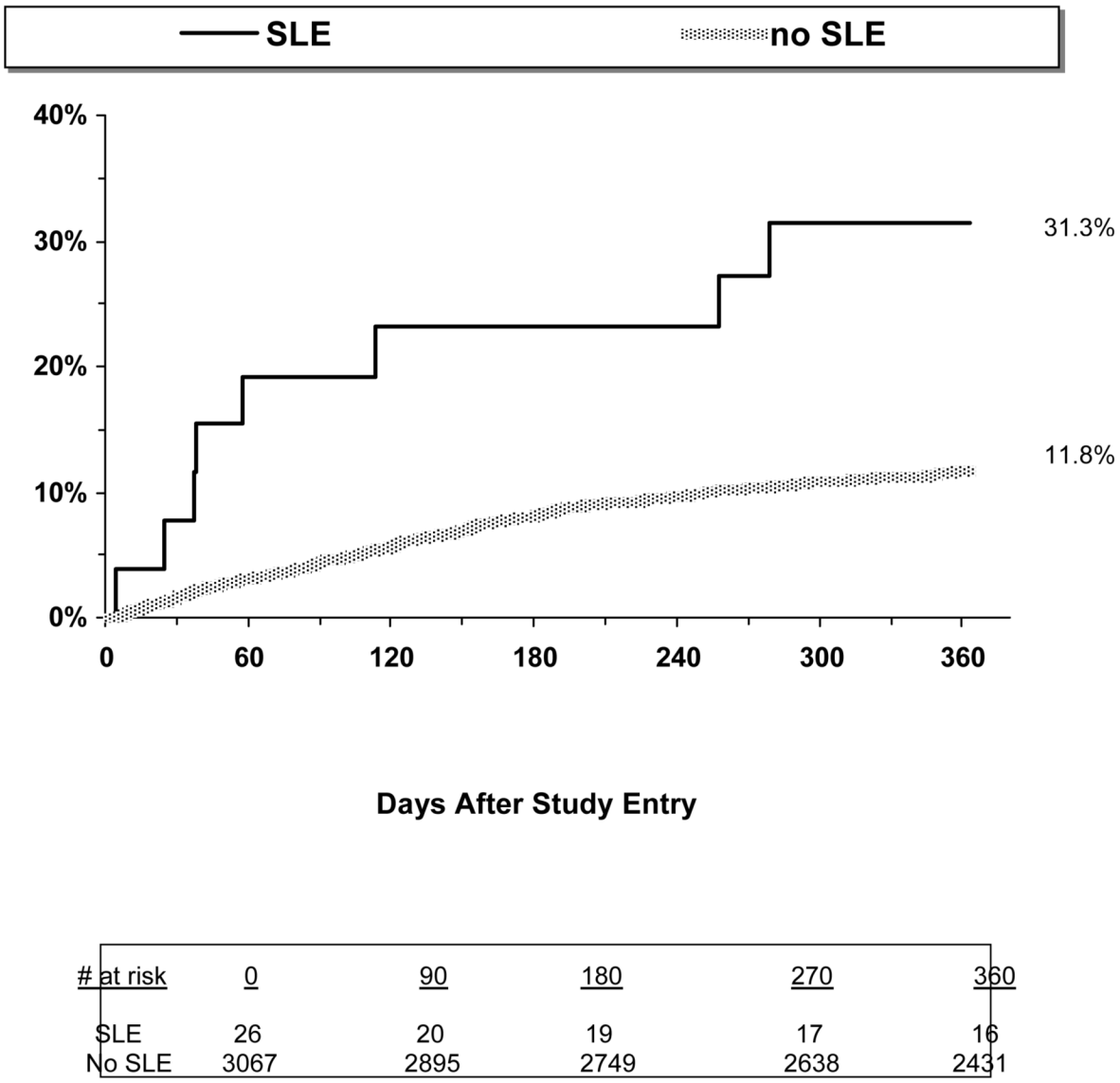
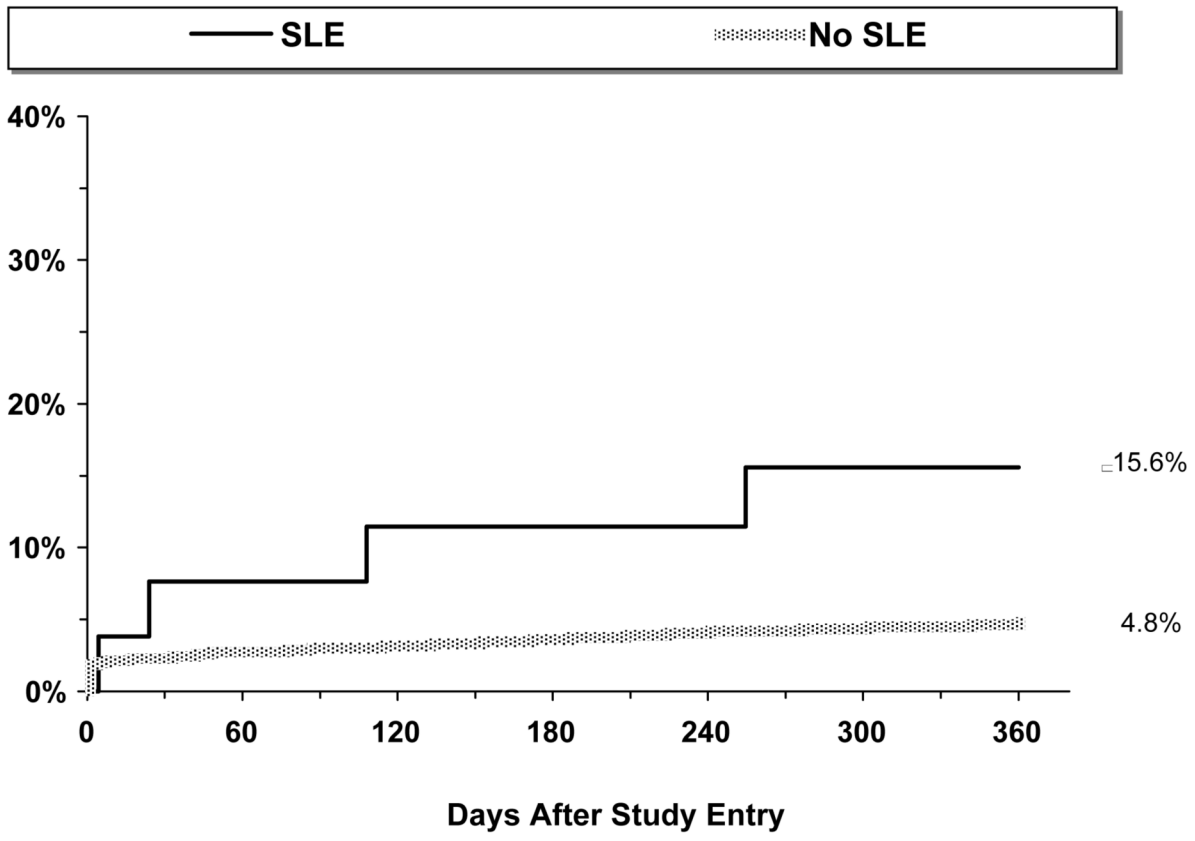
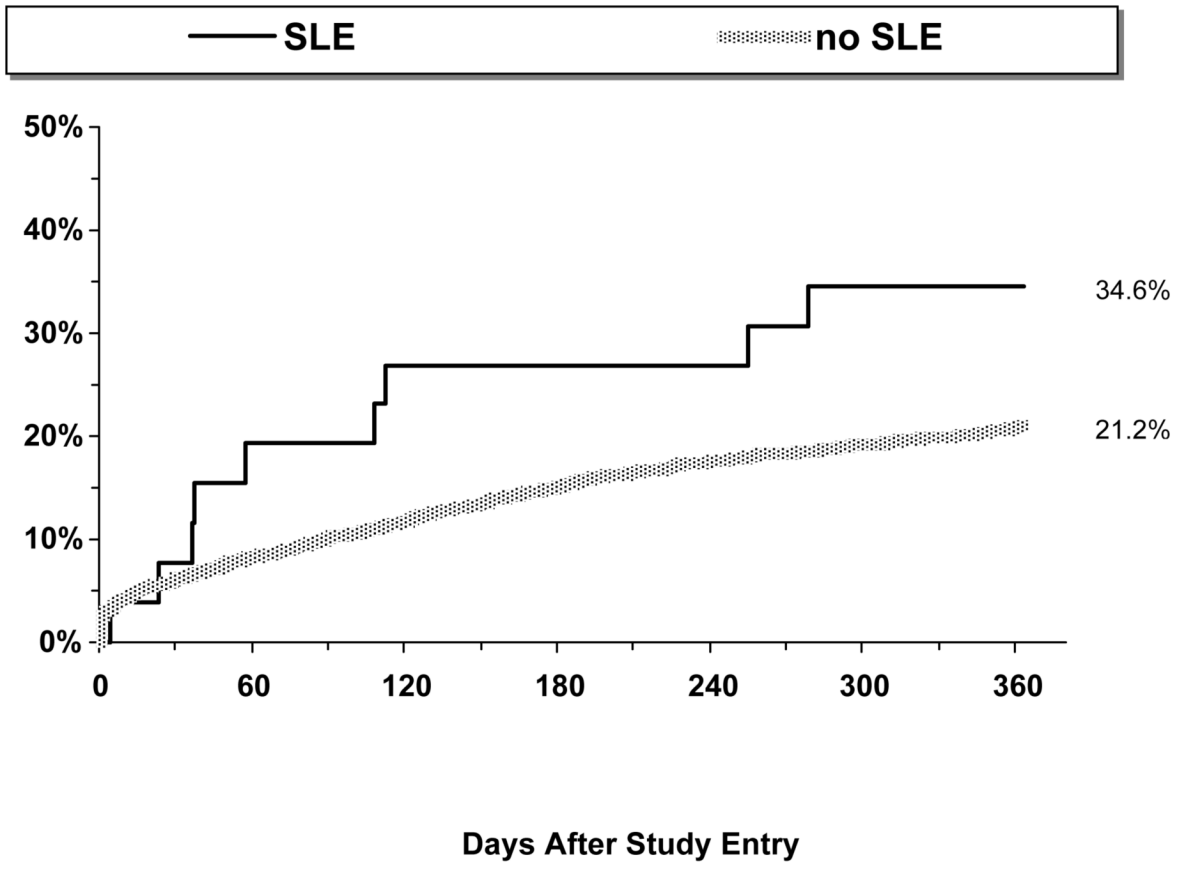



**Figure 1. One-year need for repeat PCI after hospital discharge**




| # at risk | 0 | 90 | 180 | 270 | 360 |
|---|---|---|---|---|---|
| SLE | 28 | 23 | 21 | 20 | 19 |
| No SLE | 3344 | 2938 | 2884 | 2817 | 2616 |

**Figure 2. One-year myocardial infarction rate**




| # at risk | 0 | 90 | 180 | 270 | 360 |
|---|---|---|---|---|---|
| SLE | 28 | 20 | 18 | 18 | 15 |
| No SLE | 3344 | 2790 | 2627 | 2508 | 2292 |

**Figure 3. One-year major adverse coronary events (death, MI, PCI or CABG)**

## Table 1

### Baseline characteristics

| Characteristic | Total (N=3385) | No SLE (N=3357) | SLE (N=28) | p_value |
|---|---|---|---|---|
| Age, mean, median | 63.4, 64 | 63.5, 64 | 55.0, 55 | 0.0044 |
| Age over 65, % | 46.1 | 46.3 | 25.0 | 0.0244 |
| Female, % | 35.7 | 35.3 | 89.3 | <0.0001 |
| Race, % | | | | |
| White | 72.6 | 72.7 | 64.3 | 0.5718 |
| Black | 17.5 | 17.4 | 28.6 | |
| Asian | 2.7 | 2.7 | 3.6 | |
| Hispanic | 6.8 | 6.8 | 3.6 | |
| Other | 0.5 | 0.5 | 0.0 | |
| Body mass index (kg/m2), mean, median | 29.3, 28 | 29.3, 28 | 27.5, 27 | 0.0834 |
| Prior Percutaneous Procedure(s), % | | | | |
| None | 66.2 | 66.2 | 67.9 | 0.8947 |
| One | 21.2 | 21.2 | 17.9 | |
| More than one | 12.6 | 12.6 | 14.3 | |
| Prior CABG, % | | | | |
| None | 80.7 | 80.6 | 88.9 | 0.2830 |
| One | 17.6 | 17.7 | 7.4 | |
| More than one | 1.7 | 1.6 | 3.7 | |
| Prior MI, % | 27.4 | 27.4 | 21.4 | 0.4789 |
| Severe non-cardiac concomitant disease, % | 39.7 | 39.2 | 100.0 | <.0001 |
| Cerebrovascular, % | 8.8 | 8.8 | 14.3 | 0.3080 |
| Renal, % | 7.2 | 7.2 | 7.1 | 0.9902 |
| PVD, % | 9.5 | 9.5 | 7.1 | 0.6700 |
| Pulmonary, % | 9.5 | 9.5 | 7.1 | 0.6700 |
| Cancer, % | 7.9 | 7.9 | 10.7 | 0.5773 |
| Other, % | 12.4 | 11.7 | 100.0 | <.0001 |
| History of diabetes, % | 33.6 | 33.7 | 17.9 | 0.0766 |
| Current treatment--mutually exclusive | | | | |
| None, % | 67.9 | 67.8 | 82.1 | 0.1046 |
| Diet (no medical treatment), % | 3.8 | 3.8 | 3.6 | 0.9566 |
| Oral medication (no insulin), % | 17.1 | 17.3 | 3.6 | 0.0557 |
| Insulin, % | 11.2 | 11.2 | 10.7 | 0.9335 |
| History of CHF, % | 11.2 | 11.1 | 18.5 | 0.2261 |
| History of hypertension, % | 74.9 | 74.9 | 67.9 | 0.3895 |
| History of hypercholesterolemia, % | 71.3 | 71.5 | 50.0 | 0.0160 |
| Smoking, % | | | | |
| Never | 34.5 | 34.4 | 48.1 | 0.3189 |
| Current | 25.5 | 25.5 | 18.5 | |
| Former | 40.0 | 40.1 | 33.3 | |

## Table 2
## Indications for intervention and associated therapy

| Characteristic | Total (N=3385) | No SLE (N=3357) | SLE (N=28) | p-value |
|---|---|---|---|---|
| Revascularization reason | | | | |
| Asymptomatic coronary artery disease, % | 11.1 | 11.1 | 3.6 | 0.2036 |
| Stable angina, % | 18.0 | 18.1 | 10.7 | 0.3138 |
| Unstable angina, % | 39.4 | 39.3 | 53.6 | 0.1239 |
| Acute MI, % | 25.2 | 25.2 | 28.6 | 0.6805 |
| Other, % | 4.0 | 4.0 | 3.6 | 0.9036 |
| Cardiogenic shock, % | 1.9 | 1.8 | 3.6 | 0.5013 |
| Thrombolytic therapy, % | 4.5 | 4.5 | 7.1 | 0.4964 |
| Circumstances of procedure, % | | | | |
| Elective | 57.2 | 57.2 | 57.1 | 0.9918 |
| Urgent | 31.4 | 31.4 | 32.1 | |
| Emergent | 11.4 | 11.4 | 10.7 | |
| Device access site, % | | | | |
| Femoral | 97.7 | 97.7 | 96.4 | 0.8473 |
| Brachial | 0.2 | 0.2 | 0.0 | |
| Radial | 2.1 | 2.1 | 3.6 | |
| Meds <24h prior or during procedure | | | | |
| Aspirin, % | 91.1 | 91.1 | 96.4 | 0.3206 |
| Ticlopidine, % | 9.4 | 9.5 | 0.0 | 0.0865 |
| Clopidogril and/or ticlopidine % | 69.7 | 69.8 | 57.1 | 0.1469 |
| IIb/IIIa receptor antagonist, % | 36.5 | 36.5 | 39.3 | 0.7597 |
| Other, % | 45.3 | 45.4 | 35.7 | 0.4682 |
| Investigational drugs, % | 0.4 | 0.4 | 3.6 | 0.0089 |
| Heparin % | 18.9 | 18.9 | 22.2 | 0.6599 |

## Table 3

### Medications at hospital discharge

| Medications | Total (N=3385) | No SLE (N=3357) | SLE (N=28) | p-value |
|---|---|---|---|---|
| None, % | 0.0 | 0.0 | 0.0 | 0.9267 |
| ACE inhibitor, % | 47.2 | 47.1 | 53.6 | 0.4975 |
| Aspirin, % | 96.0 | 96.2 | 78.6 | <.0001 |
| Beta blocker, % | 75.9 | 75.9 | 75.0 | 0.9103 |
| Calcium channel blocker, % | 25.0 | 25.0 | 21.4 | 0.6644 |
| Cholesterol lowering agent - statins, % | 71.6 | 71.7 | 57.1 | 0.0894 |
| other than statins, % | 9.3 | 9.2 | 10.7 | 0.7898 |
| Digitalis, % | 5.3 | 5.3 | 3.6 | 0.6856 |
| Diuretics, % | 22.6 | 22.6 | 21.4 | 0.8792 |
| Long-acting nitrates, % | 20.3 | 20.3 | 17.9 | 0.7481 |
| Low-molecular-weight heparin, % | 0.7 | 0.7 | 3.6 | 0.0729 |
| Warfarin, % | 7.5 | 7.3 | 28.6 | <.0001 |
| Clopidogrel and/or ticlopidine, % | 88.6 | 88.6 | 89.3 | 0.9101 |
| Study drugs, % | 0.8 | 0.8 | 3.6 | 0.1114 |

**Table 4**

**Medications at discharge by wave**

| | Recruitment Wave (years) | | | | | Trend |
|---|---|---|---|---|---|---|
| **Discharge medication (%)** | **1 7/97- 2/98** | **2 2/99 - 6/99** | **3 10/01 - 3/02** | **4 2/04 - 5/04** | **5 2/06 - 8/06** | **p-value** |
| **Aspirin** | | | | | | |
| SLE | 50.0 | 66.7 | 85.7 | 66.7 | 100.0 | 0.08 |
| No SLE | 94.7 | 93.2 | 95.3 | 96.7 | 98.2 | <0.001 |
| **Statin** | | | | | | |
| SLE | 25.0 | 33.3 | 71.4 | 50.0 | 75.0 | 0.12 |
| No SLE | 39.2 | 70.3 | 76.6 | 82.0 | 82.0 | <0.001 |
| **Beta Blocker** | | | | | | |
| SLE | 25.0 | 66.7 | 71.4 | 83.3 | 100.0 | 0.006 |
| No SLE | 71.4 | 66.5 | 72.1 | 79.5 | 81.0 | <0.001 |
| **Thienopyradine** | | | | | | |
| SLE | 25.0 | 100.0 | 100.0 | 100.0 | 100.0 | 0.002 |
| No SLE | 64.7 | 82.8 | 95.5 | 96.6 | 97.7 | <0.001 |

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**Table 5**

**Cumulative event rates, crude and adjusted hazard ratios (HR) and 95% confidence intervals (CI) for one-year adverse outcomes**

| Adverse outcome (%) | Event Rates | | Unadjusted Model | | | Adjusted Model[1] | | | Adjusted Model[2] | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | No SLE | SLE | HR | 95% CI | p-value | HR | 95% CI | p-value | HR | 95% CI | p-value |
| **Death** | 5.0 | 3.8 | 0.76 | 0.11-5.42 | 0.78 | 1.10 | 0.15-8.01 | 0.93 | 1.53 | 0.21-11.17 | 0.67 |
| **MI** * | 4.8 | 15.6 | 3.28 | 1.21-8.85 | 0.02 | 3.62 | 1.30-10.04 | 0.01 | 3.58 | 1.28-10.02 | 0.01 |
| **CABG** | 4.0 | 7.8 | 2.00 | 0.50-8.10 | 0.33 | 0.99 | 0.24-4.14 | 0.99 | 1.19 | 0.29-4.93 | 0.81 |
| **Death or MI** | 9.4 | 19.2 | 2.12 | 0.88-5.12 | 0.09 | 2.59 | 1.05-6.38 | 0.04 | 2.47 | 1.00-6.10 | 0.05 |
| **Death or MI or CABG** | 12.5 | 19.2 | 1.57 | 0.65-3.80 | 0.31 | 1.48 | 0.60-3.62 | 0.39 | 1.50 | 0.61-3.67 | 0.37 |
| **Repeat PCI** † | 11.8 | 31.3 | 3.13 | 1.55-6.31 | 0.001 | 2.40 | 1.17-4.91 | 0.02 | 2.65 | 1.28-5.49 | 0.009 |
| **Repeat revascularization** * | 15.0 | 31.3 | 2.39 | 1.19-4.80 | 0.01 | 1.64 | 0.80-3.35 | 0.17 | 1.98 | 0.97-4.06 | 0.06 |
| **MACE** | 21.2 | 34.6 | 1.78 | 0.92-3.44 | 0.08 | 1.48 | 0.76-2.88 | 0.25 | 1.55 | 0.79-3.03 | 0.20 |

MI = myocardial infarction, CABG = coronary artery bypass graft surgery, PCI = percutaneous coronary intervention, MACE = major adverse coronary event - includes death, MI, CABG or need for repeat PCI Repeat revascularization includes need for CABG or repeat PCI

Event rate comparisons:

*p=0.01

†p<0.001

[1]All models adjusted for age, sex, primary reason for revascularization, history of myocardial infarction, acuity of procedure, vessel disease, and recruitment wave (drug eluting stent era vs bare metal stent era).

[2]Models adjusted for age, sex, recruitment wave (drug eluting stent era vs bare metal stent era), and outcome-specific risk factors.