Poor 1-Year Outcomes After Percutaneous Coronary Interventions in Systemic Lupus Erythematosus

Report From the National Heart, Lung, and Blood Institute Dynamic Registry

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

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 with non-SLE patients (n=3385). SLE patients were younger and more often female in comparison with non-SLE patients undergoing PCI. SLE patients were less likely than non-SLE patients to have hyperlipidemia but had a similar prevalence of hypertension, diabetes mellitus, and tobacco use. The prevalence of multivessel disease was similar between groups. Initial intervention success (by angiographic definition) was not significantly different between groups. At 1 year, SLE patients were more likely to experience a myocardial infarction (15.6% versus 4.8%, \( P = 0.01 \)) and more often required repeat PCI (31.3% versus 11.8%, \( P = 0.009 \)) than non-SLE patients, even after adjustment for important covariates.

Conclusions—SLE patients had significantly worse cardiovascular outcomes at 1 year than non-SLE 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. (Circ Cardiovasc Intervent. 2008;1:201-208.)

Key Words: angioplasty ■ catheterization ■ restenosis ■ revascularization ■ systemic lupus erythematosus

The advent of glucocorticoid therapy in the 1950s was pivotal in improving survival in patients with systemic lupus erythematosus (SLE). However, despite this advance, the actuarial 5-year survival for all SLE patients in the following 15 years 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 their study, patients who died within a few years of diagnosis often succumbed to the effects of the disease or infection associated with immunosuppressive therapy, whereas those who died later after disease onset frequently had clinically quiescent SLE with autopsy evidence of acute myocardial infarction (MI).2 Both autopsy and epidemiological 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 non-SLE 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 reintervention (mean follow-up, 35 months) when compared with 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 diabetes mellitus 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 percutaneous coronary intervention (PCI) and to report clinical outcomes. This comprehensive database provides a unique opportunity to examine cardiovascular risk factors, pharmacological therapy, and both short- and midterm outcomes after PCI. We compared the outcomes of PCI between patients with SLE and non-SLE patients.

Methods

Study Population

Patients enrolled in waves 1 to 3 were contacted by telephone at 1 year after the PCI to obtain follow-up data, whereas patients in waves 4 and 5 were contacted at 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 non-SLE 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 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 end point major adverse cardiac event included death, MI, and any repeat revascularization (repeat PCI or CABG) procedure. Repeat PCI included both target and nontarget vessel interventions.

The institutional review board of each center approved the protocol, and informed consent to collect information during and after hospital discharge was obtained from all patients.

Statistical Analysis
Patient characteristics pertaining to the index PCI, including demographics, medical history, cardiac presentation, periprocedural medications, and in-hospital outcomes were compared by using the nonparametric Wilcoxon rank sum test for continuous variables and χ² test for categorical variables. Similar methods were used for lesion-level analyses. Associations between the 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 1 year if contact extended beyond 1 year.

Cox proportional hazards modeling was used to assess the independent effect of SLE on select 1-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) prespecified variables (age, sex, vessel disease, presence of diabetes mellitus, history of MI, reason for revascularization, acuity of PCI, and year of the procedure) and (2) only important covariates identified by forward stepwise selection (entry probability value criterion of ≤0.25, retain criteria of ≤0.05). Proportional hazards assumptions were evaluated and met for all Cox proportional hazards models.

Results

Baseline Clinical Characteristics
Combining the 5 waves of recruitment from the 14 centers in which SLE patients were identified, a total of 3,385 patients were included in the study (Table 1). In the entire cohort, just fewer than half of the patients were older than 65 years, 64.3% were male, and 72.6% were white. 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% white).

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

Indications for Intervention and Associated Therapies
There were no significant differences between SLE and non-SLE patients in reasons for revascularization (asymptomatic coronary artery disease [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 differ between groups (Table 2).

Baseline Cardiac Function and Anatomic Features
The prevalence of abnormal left ventricular function and mean and median ejection fractions was similar in SLE and non-SLE 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% versus 19.3%, P=0.003) than non-SLE 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 preprocedural thrombolysis in myocardial infarction flow.

Procedure Details
Lesions technically amenable to PCI and mean and median numbers of lesions treated were similar between groups. More than 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 thrombolysis in myocardial infarction 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 periprocedural 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 ASA therapy (78.6% versus 96.2%, \(P<0.0001\)) and more likely to be discharged on warfarin (28.6% versus 7.3%, \(P<0.0001\)) than non-SLE 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 4 months, the need for repeat PCI in SLE patients was significantly higher compared with non-SLE patients (Figure 1). This difference remained statistically significant at 1 year. The majority of SLE patients requiring repeat PCI were more likely to require repeat PCI and to have an MI during 1-year follow-up after PCI in the NHLBI Dynamic Registry. These adverse outcomes are most apparent in the first 4 months post-PCI, when thrombosis is most likely. Aspirin therapy was less likely to be used in patients with SLE at the time of hospital discharge, especially in earlier recruitment waves. The differences in outcomes between SLE and non-SLE patients at 4 months post-PCI suggest that restenosis or plaque destabilization in SLE may happen more aggressively, or that SLE patients receive different postprocedural 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 cardiovascular 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 cardiovascular events after 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 1 year (40% versus 14%, P<0.01) than anticardiolipin-negative patients. Additionally, Marcucci et al demonstrated that levels of plasminogen activator inhibitor (PAI)-1 and homocysteine levels are independent risk factors for major adverse cardiac event after PCI for ACS. Furthermore, it has been noted that the prevalence of anticardiolipin antibodies is higher in SLE patients (44%) when compared with non-SLE controls (9%).

Despite a lower prevalence of traditional risk factors associated with cardiovascular events in comparison with non-SLE patients, SLE patients were more likely to require repeat PCI and to have an MI during 1-year follow-up after PCI in the NHLBI Dynamic Registry. These adverse outcomes are most apparent in the first 4 months post-PCI, when thrombosis is most likely. Aspirin therapy was less likely to be used in patients with SLE at the time of hospital discharge, especially in earlier recruitment waves. The differences in outcomes between SLE and non-SLE patients at 4 months post-PCI suggest that restenosis or plaque destabilization in SLE may happen more aggressively, or that SLE patients receive different postprocedural 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 cardiovascular risk factors in atherogenesis and prediction of events, in a disease characterized by increased sustained immune activation, several potential factors can be considered.

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### Discussion

Table 3. Medications at Hospital Discharge

<table>
<thead>
<tr>
<th>Medications</th>
<th>Total (n=3385)</th>
<th>No SLE (n=3357)</th>
<th>SLE (n=28)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Statins, %</td>
<td>71.6</td>
<td>71.7</td>
<td>57.1</td>
<td>0.0894</td>
</tr>
<tr>
<td>Other than statins, %</td>
<td>9.3</td>
<td>9.2</td>
<td>10.7</td>
<td>0.7898</td>
</tr>
<tr>
<td>Digitalis, %</td>
<td>5.3</td>
<td>5.3</td>
<td>3.6</td>
<td>0.6856</td>
</tr>
<tr>
<td>Diuretics, %</td>
<td>22.6</td>
<td>22.6</td>
<td>21.4</td>
<td>0.8792</td>
</tr>
<tr>
<td>Long-acting nitrates, %</td>
<td>20.3</td>
<td>20.3</td>
<td>17.9</td>
<td>0.7481</td>
</tr>
<tr>
<td>Low-molecular weight heparin, %</td>
<td>0.7</td>
<td>0.7</td>
<td>3.6</td>
<td>0.0729</td>
</tr>
<tr>
<td>Warfarin, %</td>
<td>7.5</td>
<td>7.3</td>
<td>28.6</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Clopidogrel and/or ticlopidine, %</td>
<td>88.6</td>
<td>88.6</td>
<td>88.3</td>
<td>0.9101</td>
</tr>
<tr>
<td>Study drugs, %</td>
<td>0.8</td>
<td>0.8</td>
<td>3.6</td>
<td>0.1114</td>
</tr>
</tbody>
</table>

Despite a lower prevalence of traditional risk factors associated with cardiovascular events in comparison with non-SLE patients, SLE patients were more likely to require repeat PCI and to have an MI during 1-year follow-up after PCI in the NHLBI Dynamic Registry. These adverse outcomes are most apparent in the first 4 months post-PCI, when thrombosis is most likely. Aspirin therapy was less likely to be used in patients with SLE at the time of hospital discharge, especially in earlier recruitment waves. The differences in outcomes between SLE and non-SLE patients at 4 months post-PCI suggest that restenosis or plaque destabilization in SLE may happen more aggressively, or that SLE patients receive different postprocedural 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 cardiovascular risk factors in atherogenesis and prediction of events, in a disease characterized by increased sustained immune activation, several potential factors can be considered.
tration 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 NHLBI Dynamic Registry did not include information on anticoagulants nor other indicators of hypercoagulability at PCI admission; therefore, we cannot ascertain whether ASA and warfarin usage at hospital discharge were influenced by such preexisting conditions. Event numbers were too small to determine associations between events and increased ASA use 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 cardiovascular events than warfarin plus ASA therapy after PCI, with the role of warfarin plus an antiplatelet agent other than ASA less clearly defined.\textsuperscript{14,15} Given this data, the use of triple therapy (warfarin plus 2 antiplatelet agents) after PCI for the first 4 to 12 weeks in patients requiring chronic anticoagulation has been suggested.\textsuperscript{16} However, studies examining bleeding complications associated with this regimen have found conflicting results.\textsuperscript{17,18} Without a better understanding of the precipitants of post-PCI events in SLE, it remains difficult to weigh risk versus benefit of a more aggressive approach to antiplatelet therapy in SLE patients requiring chronic anticoagulation therapy.

In considering the data linking inflammation to cardiovascular disease and cardiovascular 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, markers of oxidant stress, and tissue factor, all of which are purported to have a role in

Table 4. Medications at Discharge by Wave

<table>
<thead>
<tr>
<th>Recruitment Wave (Years)</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin SLE</td>
<td>50.0</td>
<td>66.7</td>
<td>85.7</td>
<td>66.7</td>
<td>100.0</td>
<td>0.08</td>
</tr>
<tr>
<td>Aspirin No SLE</td>
<td>94.7</td>
<td>93.2</td>
<td>95.3</td>
<td>96.7</td>
<td>98.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Statin SLE</td>
<td>25.0</td>
<td>33.3</td>
<td>71.4</td>
<td>50.0</td>
<td>75.0</td>
<td>0.12</td>
</tr>
<tr>
<td>Statin No SLE</td>
<td>39.2</td>
<td>70.3</td>
<td>76.6</td>
<td>82.0</td>
<td>82.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Beta blocker SLE</td>
<td>25.0</td>
<td>66.7</td>
<td>71.4</td>
<td>83.3</td>
<td>100.0</td>
<td>0.006</td>
</tr>
<tr>
<td>Beta blocker No SLE</td>
<td>71.4</td>
<td>66.5</td>
<td>72.1</td>
<td>79.5</td>
<td>81.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Thienopyradine SLE</td>
<td>25.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>0.002</td>
</tr>
<tr>
<td>Thienopyradine No SLE</td>
<td>64.7</td>
<td>82.8</td>
<td>95.5</td>
<td>96.6</td>
<td>97.7</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Figure 1. One-year need for repeat PCI after hospital discharge. — indicates SLE; = no SLE.
plaque destabilization and thrombosis.\textsuperscript{19–23} Inflammation has also been linked to other novel markers of cardiovascular risk, including lipoprotein particle size, acute phase high-density lipoprotein, protein nitration, and protein glycoxidation.\textsuperscript{24–27} The utility of these markers in the prediction of cardiovascular disease and cardiovascular 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.\textsuperscript{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 cardiovascular events found in the general population.\textsuperscript{31,32} However, the significance of this relationship in SLE and the general population remains unclear.

Previous studies have demonstrated that cardiovascular 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.\textsuperscript{33} Additionally, only 58% of all modifiable risk factors received any intervention. Although ASA and 3-hydroxy-3-methylglutaryl-coenzyme A reductase inhibitors are considered standard of care for secondary prevention of CAD, in our SLE group, only 70% were discharged on ASA therapy, and just 50% were on statin therapy, although usage patterns increased over the 5 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 cardiovascular risk factor assessment or have known risk factors that were suboptimally managed, suggesting that this remains a problem with an undetermined impact on cardiovascular 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

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### Table 5. Cumulative Event Rates, Crude and Adjusted Hazard Ratios (HRs), and 95% CIs for 1-Year Adverse Outcomes

<table>
<thead>
<tr>
<th>Adverse Outcome (%)</th>
<th>Event Rates</th>
<th>Unadjusted Model</th>
<th>Adjusted Model‡</th>
<th>Adjusted Model§</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No SLE</td>
<td>SLE</td>
<td>HR</td>
<td>95% CI</td>
</tr>
<tr>
<td>Death</td>
<td>5.0</td>
<td>3.8</td>
<td>0.76</td>
<td>0.11–5.42</td>
</tr>
<tr>
<td>MI*</td>
<td>4.8</td>
<td>15.6</td>
<td>3.28</td>
<td>1.21–8.85</td>
</tr>
<tr>
<td>CABG</td>
<td>4.0</td>
<td>7.8</td>
<td>2.00</td>
<td>0.50–8.10</td>
</tr>
<tr>
<td>Death or MI</td>
<td>9.4</td>
<td>19.2</td>
<td>2.12</td>
<td>0.88–5.12</td>
</tr>
<tr>
<td>Death or MI or CABG</td>
<td>12.5</td>
<td>19.2</td>
<td>1.57</td>
<td>0.65–3.80</td>
</tr>
<tr>
<td>Repeat PCI†</td>
<td>11.8</td>
<td>31.3</td>
<td>3.13</td>
<td>1.55–6.31</td>
</tr>
<tr>
<td>Repeat revascularization*</td>
<td>15.0</td>
<td>31.3</td>
<td>2.39</td>
<td>1.19–4.80</td>
</tr>
<tr>
<td>MACE</td>
<td>21.2</td>
<td>34.6</td>
<td>1.78</td>
<td>0.92–3.44</td>
</tr>
</tbody>
</table>

MACE indicates 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: *\( P < 0.01; \) †\( P < 0.001.\)

‡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 versus bare metal stent era).

§Models adjusted for age, sex, recruitment wave (drug-eluting stent era versus bare metal stent era), and outcome-specific risk factors.

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![Figure 2. One-year MI rate. — indicates SLE; —— no SLE.](http://circinterventions.ahajournals.org/)
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 after PCI in this study are striking. These results highlight the potential importance of postinterventional secondary preventative measures in SLE and provide an impetus to dedicate further study directed at identifying modifiable risk factors to improve cardiovascular outcomes in SLE patients undergoing PCI.

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Disclosures
Dr Williams receives grant support from Cordis, Boston Scientific, and Abbott Vascular. Dr Wasko reported being a consultant to Centocor in cardiovascular outcomes in rheumatoid arthritis clinical trials and has been a coinvestigator in a Merck-sponsored study of thromboembolic markers in rheumatoid arthritis and osteoarthritis and site principal investigator in clinical trials sponsored by Centocor and Roche.

References


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http://circinterventions.ahajournals.org/content/1/3/201