Acute Myocardial Infarction Outcomes in Systemic Lupus Erythematosus (from the Nationwide Inpatient Sample)

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One of the major causes of mortality in systemic lupus erythematosus (SLE) is acute myocardial infarction. Whether in-hospital outcomes and management of ST-segment elevation myocardial infarction (STEMI) and non-STEMI (NSTEMI) are different in SLE patients compared with those without SLE from large, recent dataset is unclear. We queried the Nationwide Inpatient Database from 2005 to 2014 and identified STEMI and NSTEMI admissions with and without SLE. The primary outcome was in-hospital mortality. Secondary outcomes were revascularization strategy (percutaneous coronary intervention, coronary artery bypass surgery, or thrombolytics), medical therapy rates (no reperfusion), and major adverse clinical events. A propensity-matched cohort was created to compare these outcomes. Odds ratio (OR) was calculated from the propensity-matched cohort. A total of 321,048 STEMI admissions, of which 1,001 (0.31%) and 572,971 NSTEMI admissions, of which 2,134 (0.37%) were SLE, were identified. In those with STEMI, 882 SLE and non-SLE admissions were propensity-matched. In-hospital mortality (9.1% vs 11.8%, OR 0.75, p = 0.07), revascularization strategy, medical therapy rates, and major adverse events were similar. Similarly, in those with NSTEMI, 1,770 SLE and 1,775 non-SLE were matched. In-hospital mortality (4.1% vs 4.50%, OR 0.90, p = 0.51), coronary artery bypass surgery, medical therapy rates, and major adverse events were mostly similar but the rate of percutaneous coronary intervention was higher in SLE (32.9% vs 29.6%, OR 1.16, p = 0.04). For both STEMI and NSTEMI, hospital cost and length of stay were similar between SLE and non-SLE cohorts. From a large administrative database in the United States, revascularization strategies and in-hospital outcomes of acute coronary syndrome were mostly similar between SLE and non-SLE. **(Am J Cardiol 2019;123:227−232)**

One of the major causes of mortality in systemic lupus erythematosus (SLE) patients is cardiovascular disease from accelerated atherosclerosis caused by both traditional and disease-specific risk factors.[1,2,3] A population-based study showed that acute myocardial infarction (AMI) hospitalizations increased from 1996 to 2012 in SLE while they decreased in the general population.[4] The incidence of AMI in SLE was higher compared with non-SLE.[4] Outcomes of AMI in SLE cohorts have been reported in the past[5,6] but several clinically important questions remain not fully addressed. First, a previous study that reported SLE presenting with AMI had worse in-hospital outcomes[6] was not reflective of the recent advances in the management of AMI and did not differentiate ST-segment elevation myocardial infarction (STEMI) and non-STEMI (NSTEMI). Second, whether those with SLE presenting as AMI received different revascularization strategies due to more advanced coronary artery disease from additional disease-specific atherosclerosis risk factors (i.e., steroid use, chronic inflammation, lupus nephritis) is not clear.[7−10] We queried Nationwide Inpatient Sample (NIS) database to address these clinically relevant issues.

Methods

Data were obtained from the Agency for Healthcare Research and Quality Healthcare Cost and Utilization Project−NIS files between 2005 and 2014. The data were queried to identify patients ($\geq$ 18 years) who underwent either percutaneous coronary intervention (PCI), coronary artery bypass graft (CABG), thrombolytic therapy, or medical therapy (no reperfusion) using the International Classification of Diseases, Ninth Revision, Clinical Modification. The NIS is the largest all-payer inpatient database in the United States, and it includes a 20% sample of US community hospitals from up to 45 states and approximates 20% of all US community hospitals. Hospitals are short-term, nonfederal general and specialty hospitals selected based on 5 sampling strata, and once selected, include 100% of their hospitalizations.[11]

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Table 1
Baseline characteristics of participants with STEMI with and without SLE

Variable	STEMI (unmatched)			STEMI (matched)		
	SLE	No SLE	p value	SLE	No SLE	p value
No. of observation, unweighted	1,001 (0.31%)	320,047 (99.69%)		822	822	
No. of observation, weighted	4,810	1,538,256		3,953	3,948	
Age, mean (standard deviation) (y)	58.68 (14.16)	64.24 (14.48)	<.0001	58.88 (14.16)	58.72 (15.59)	0.825
Female	77.19%	33.73%	<.0001	76.86%	79.15%	0.271
White	66.80%	77.84%	<.0001	66.61%	66.82%	0.715
Black	20.07%	8.07%		20.22%	18.29%	
Hispanic	7.62%	7.80%		7.72%	8.97%	
Asia	2.27%	2.35%		2.11%	1.90%	
Dyslipidemia	40.14%	54.00%	<.0001	41.16%	41.93%	0.754
Prior myocardial infarction	8.64%	7.79%	0.336	8.37%	7.10%	0.338
Prior percutaneous coronary intervention	10.41%	9.74%	0.477	10.82%	8.56%	0.119
Prior coronary bypass	2.80%	3.94%	0.071	3.18%	3.56%	0.661
Prior pacemaker	0.88%	1.39%	0.162	0.94%	0.84%	0.835
Atrial fibrillation	12.14%	7.86%	<.0001	8.36%	8.32%	0.974
Chronic obstructive pulmonary disease	21.09%	15.76%	<.0001	20.48%	19.99%	0.805
Carotid artery disease	1.32%	0.86%	0.127	0.89%	1.33%	0.413
Prior cerebrovascular disease	5.07%	3.61%	0.014	5.21%	6.06%	0.466
Hypertension	61.46%	59.83%	0.302	62.98%	64.96%	0.402
Peripheral vascular disease	6.30%	7.40%	0.200	6.73%	6.47%	0.829
Hypothyroidism	14.02%	7.26%	<.0001	13.75%	11.42%	0.158
Diabetes mellitus	19.90%	27.11%	<.0001	20.09%	22.34%	0.262
Obesity	9.08%	10.44%	0.161	9.42%	9.59%	0.904
Anemia deficiency	19.27%	9.80%	<.0001	19.11%	19.74%	0.739
Congestive heart failure	0.69%	0.62%	0.812	0.84%	0.69%	0.751
Renal failure	13.84%	9.34%	<.0001	14.91%	14.05%	0.633
Liver disease	1.74%	0.94%	0.010	1.86%	2.12%	0.712
Electrolyte derangement	19.14%	15.92%	0.005	18.86%	16.06%	0.129
Smoker	36.30%	39.40%	0.052	36.24%	34.87%	0.565
Maintenance dialysis	2.13%	0.68%	<.0001	2.34%	3.28%	0.224
Elixhauser score			<.0001			0.771
0 - 3	61.26%	86.18%		59.97%	60.67%	
≥ 4	38.74%	13.82%		40.03%	39.33%	
Hospital bed size			0.740			0.639
Small	9.72%	9.53%		10.23%	10.19%	
Medium	22.57%	23.64%		21.74%	23.63%	
Large	67.71%	66.82%		68.04%	66.18%	
Hospital teaching status			0.0002			0.527
Rural	8.05%	9.90%		7.39%	6.02%	
Urban, nonteaching	39.34%	44.31%		39.52%	40.42%	
Urban, teaching	52.61%	45.78%		53.09%	53.56%	
Expected primary payer			<.0001			0.568
Medicare	47.50%	45.76%		48.00%	48.52%	
Medicaid	10.04%	6.39%		9.80%	11.62%	
Private	34.58%	35.41%		34.61%	32.21%	
Others	7.88%	12.44%		7.59%	7.66%	
Median household income (quartile)			0.158			0.676
1st	29.53%	26.65%		29.51%	29.07%	
2nd	26.40%	26.65%		26.04%	23.94%	
3nd	24.66%	25.07%		24.82%	26.96%	
4th	19.40%	21.63%		19.63%	20.03%	
Hospital region			0.013			0.378
Northeast	16.61%	16.80%		19.09%	20.76%	
Midwest	22.73%	23.31%		17.13%	14.43%	
South	44.21%	39.73%		47.56%	46.78%	
West	16.46%	20.16%		16.32%	18.03%	

SLE = systemic lupus erythematosus; STEMI = ST-segment elevation myocardial infarction.
Obesity- ICD-9-CM codes: 278.0, 278.00, 278.01.
Dyslipidemia: Clinical classification code 53.
Anemia deficiency: 280.1 to 281.9, 285.21 to 285.29, 285.9.
Electrolyte derangement: 276.0 to 276.9.

Our study population included patients (≥ 18 years) who were admitted for AMI between 2005 and 2014 with or without SLE. These patients were identified using appropriate ICD-9 diagnostic codes in the discharge records (Supplement 1). Those who were discharged on the same day alive, transferred between facilities, elective admission, and who had both PCI and CABG were excluded from the analysis. The NIS discharge records were queried to identify demographics and hospitalization outcomes variables. Baseline characteristics were captured using the International Classification of Diseases, Ninth Revision, Clinical Modification codes. Clinical outcomes were compared in patients who were admitted for AMI with and without SLE. The primary outcome of interest was in-hospital mortality. Assessment of healthcare resources was performed by comparing nonroutine home discharge rate, cost, and length of stay. Hospital cost information was obtained from the hospital accounting reports collected by the Centers for Medicare and Medicaid Services.[12] We estimated the cost of each inpatient stay by multiplying the total hospital charge with cost-to-charge ratios. Costs were inflation-adjusted using the Consumer Price Index provided by the US Bureau of Labor Statistics, with 2017 as the index base.[13] This method allowed us to standardize costs over the study period. The co-morbidities were identified using the Agency for Healthcare Research and Quality co-morbidity measures. The severity of co-morbidities was identified by using the Elixhauser co-morbidity index.[14]

A propensity score matching model was developed to derive 2 matched groups for comparative outcome analysis to account for potential confounding factors and reduce the effect of selection bias. We used a multivariable logistic regression model with STEMI/NSTEMI with SLE as the outcome variable, and all co-morbidities in Table 1 and patient-level NIS weights were used as covariates.[15,16] A 1-to-1 greedy matching protocol and a caliper width of 0.1 multiplied by the standard deviation of the logit of the propensity score was used to create a matched cohort between SLE and non-SLE in in STEMI and NSTEMI.

Data extraction and analyses were performed with Statistical Analysis System (SAS V.9.4). All statistical tests were 2-sided and a p value of < 0.05 was determined a priori to be statistically significant. We first conducted bivariate analyses to compare demographic. Clinical characteristics and hospital characteristics in AMI admissions with or without SLE. Chi-square tests were used for categorical variables whereas *t* test for continuous variables with normal distribution. We reported the mean and standard deviation for continuous variables, and percentages for categorical variables for the matched variables. The baseline characteristics were computed with a paired *t* test for continuous variables with normal distribution and Mc-Nemar's test for categorical variables. Binary outcomes were modeled with binomial logistic regressions. The morality trend in SLE was accessed by fitting a poisson regression model with a robust error variance to evaluate for changes in the number of mortality in SLE per year and adjusting for demographic, co-morbidities, and hospital characteristic and keeping the "year" as a continuous variable in the model. Discrete numeric variables were modeled with generalized linear model regressions. Analyses were performed in SAS with appropriate statements to account for the complex clustered sampling methods.[16,17]

Results

We identified a total of 321,048 STEMI admissions of which 1,001 (0.31%) were SLE. In unmatched population, SLE admissions were more female gender and younger. SLE admissions had more atrial fibrillation, previous stroke, liver disease, chronic obstructive pulmonary disease, anemia, hypothyroid, electrolyte abnormality, and renal failure. After propensity-matching, a total of 1,644 STEMI admissions with SLE were well matched with non-SLE STEMI admissions (822 admissions each; Table 1). There were no differences in terms of revascularization strategy between SLE and non-SLE for STEMI (PCI 64.8% vs 63.7%, p = 0.71, CABG 4.2% vs 3.7%, p = 0.52%, thrombolytics 3.5% vs 2.9%, p = 0.49, and medical therapy 29.6% vs 32.2%, p = 0.33, respectively). In-hospital mortality (9.1% vs 11.8%, p = 0.07) and major clinical events were all similar in both groups as summarized in Table 2. Average cost and length of stay were also similar between the 2 groups (Table 2).

Table 2
Clinical outcomes of STEMI with and without SLE

Variable	SLE	Non-SLE	OR/MR (95% CI)	p value
In-patient mortality	9.13%	11.83%	0.75 (0.55, 1.03)	0.073
Percutaneous coronary intervention	64.83%	63.74%	1.04 (0.85, 1.27)	0.711
Coronary bypass	4.21%	3.67%	1.17 (0.72, 1.92)	0.522
Thrombolytics	3.50%	2.94%	1.22 (070, 2.13)	0.493
Medical therapy	29.57%	32.33%	0.90 (0.72, 1.12)	0.329
Bleeding requiring transfusion	2.10%	1.61%	1.39 (0.67, 2.88)	0.374
Acute kidney injury	11.21%	9.05%	1.27 (0.93, 1.75)	0.137
Acute kidney injury requiring dialysis	1.06%	0.84%	1.29 (0.47, 3.51)	0.618
Stroke	1.43%	1.21%	1.20 (0.51, 2.81)	0.670
Sepsis	2.59%	2.77%	0.91 (0.49, 1.67)	0.755
DVT/PE	1.19%	1.54%	0.70 (0.31, 1.60)	0.399
Cost, average (US dollars)	25,073	25,858	0.97 (0.88, 1.07)*	0.536
Length of stay average (d)	4.72	4.93	0.96 (0.86, 1.07)*	0.427

CI = confidence interval; DVT = deep vein thrombosis; MR = mean ratio; OR = odds ratio; PE = pulmonary embolism; SLE = systemic lupus erythematosus.

* Mean ratios.

Table 3
Baseline characteristics of participants with NSTEMI with and without SLE

Variable	NSTEMI (unmatched)			NSTEMI (matched)		
	SLE	No SLE	p value	SLE	No SLE	p value
No. of observation, unweighted	2,134 (0.37%)	570,837 (99.63%)		1,770	1,775	
No. of observation, weighted	10,290	2,755,876		8,556	8,588	
Age, mean (standard deviation) (y)	61.16 (14.12)	68.85 (14.27)	<.0001	61.49 (14.14)	61.38 (15.71)	0.824
Female	81.99%	42.17%	<.0001	81.77%	82.08%	0.812
White	62.52%	74.64%	<.0001	63.08%	62.39%	0.234
Black	24.69%	11.40%		24.43%	23.46%	
Hispanic	8.30%	8.10%		8.39%	0.56%	
Asia	1.40%	2.28%		1.09%	1.87%	
Dyslipidemia	43.45%	55.90%	<.0001	44.13%	44.85%	0.677
Prior myocardial infarction	11.81%	11.92%	0.875	12.26%	11.60%	0.551
Prior percutaneous coronary intervention	13.09%	12.82%	0.713	13.63%	12.87%	0.518
Prior coronary bypass	5.31%	9.67%	<.0001	5.19%	5.91%	0.332
Prior pacemaker	2.18%	3.18%	0.009	2.34%	1.44%	0.053
Atrial fibrillation	12.40%	17.84%	<.0001	13.13%	13.78%	0.577
Chronic obstructive pulmonary disease	26.79%	22.81%	<.0001	26.77%	27.33%	0.703
Carotid artery disease	1.63%	2.05%	0.172	1.49%	1.49%	0.994
Prior cerebrovascular disease	8.23%	6.43%	0.001	8.66%	9.46%	0.399
Hypertension	70.15%	70.16%	0.994	70.02%	69.68%	0.824
Peripheral vascular disease	10.85%	12.18%	0.071	11.26%	10.95%	0.774
Hypothyroidism	16.54%	10.70%	<.0001	17.81%	18.44%	0.625
Diabetes mellitus	26.03%	37.25%	<.0001	26.78%	27.08%	0.842
Obesity	12.20%	12.18%	0.978	12.38%	10.98%	0.195
Anemia deficiency	24.31%	16.76%	<.0001	24.70%	23.95%	0.601
Congestive heart failure	0.84%	0.77%	0.718	0.89%	1.47%	0.096
Renal failure	26.52%	20.49%	<.0001	25.87%	25.88%	0.993
Liver disease	1.99%	1.34%	0.017	2.17%	1.98%	0.708
Electrolyte derangement	24.19%	20.51%	<.0001	23.70%	22.99%	0.612
Smoker	31.80%	32.08%	0.791	33.41%	33.87%	0.771
Maintenance dialysis	5.87%	2.47%	<.0001	5.48%	6.62%	0.150
Elixhauser score			<.0001			0.729
0 - 3	45.45%	73.54%		45.66%	46.24%	
≥ 4	54.55%	26.46%		54.34%	53.77%	
Hospital bed size			0.820			0.809
Small	9.25%	9.69%		9.15%	8.52%	
Medium	24.59%	24.58%		25.31%	25.32%	
Large	66.16%	65.74%		65.54%	66.16%	
Hospital teaching status			0.0001			0.970
Rural	6.79%	9.42%		6.57%	6.77%	
Urban, nonteaching	42.33%	43.00%		43.28%	43.16%	
Urban, teaching	50.88%	47.59%		50.15%	50.07%	
Expected primary payer			<.0001			0.690
Medicare	58.06%	61.30%		59.29%	59.24%	
Medicaid	11.24%	6.08%		10.46%	11.63%	
Private	25.97%	24.47%		25.37%	24.36%	
Others	4.73%	8.14%		4.88%	4.77%	
Median household income (quartile)			0.003			0.747
1st	31.55%	28.50%		31.62%	30.17%	
2nd	27.65%	26.75%		27.39%	28.12%	
3nd	23.07%	24.34%		22.96%	24.01%	
4th	17.73%	20.42%		18.02%	17.70%	
Hospital region			0.0003			0.929
Northeast	16.12%	19.63%		17.85%	17.71%	
Midwest	22.26%	22.10%		18.54%	17.76%	
South	45.31%	41.15%		46.41%	46.81%	
West	16.32%	17.12%		17.20%	17.72%	

NSTEMI = Non-ST-segment elevation myocardial infarction; SLE = systemic lupus erythematosus.

There were a total of 572,971 NSTEMI admissions of which 2,134 (0.37%) were SLE. In unmatched admissions, SLE admissions had a higher prevalence of chronic obstructive pulmonary disease, previous stroke, hypothyroidism, anemia, renal failure, liver disease, and electrolyte disturbance. A propensity-matched cohort of 3,545 (1,770 SLE and 1,775 non-SLE) was created (Table 3). SLE admissions had a higher rate of PCI compared with non-SLE (32.9% vs

Table 4
Clinical outcomes of NSTEMI by SLE

Variable	SLE	Non-SLE	OR/MR (95% CI)	p value
In-patient mortality	4.07%	4.50%	0.90 (0.65, 1.24)	0.505
Percutaneous coronary intervention	32.91%	29.61%	1.16 (1.01, 1.34)	0.041
Coronary bypass	5.09%	6.03%	0.83 (0.63, 1.11)	0.214
Medical therapy	62.05%	64.18%	0.91 (0.80, 1.05)	0.192
Bleeding requiring transfusion	1.63%	1.80%	0.90 (0.55, 1.50)	0.696
Acute kidney Injury	14.18%	14.43%	0.99 (0.82, 1.19)	0.885
Acute kidney Injury requiring dialysis	0.89%	1.40%	0.66 (0.35, 1.26)	0.207
Stroke	0.75%	1.94%	0.41 (0.22, 0.76)	0.005
Sepsis	3.21%	3.31%	0.98 (0.68, 1.42)	0.921
DVT /PE	1.65%	1.99%	0.85 (0.52, 1.39)	0.527
Cost, average (US dollars)	21,123	20,975	1.01 (0.93, 1.09)*	0.854
Length of stay average (d)	5.12	5.35	0.96 (0.88, 1.04)*	0.282

CI = confidence interval; DVT = deep vein thrombosis; MR = mean ratio; OR = odds ratio; PE = pulmonary embolism; SLE = systemic lupus erythematosus.
* Mean ratio.

29.6%, p = 0.041) but a similar rate of CABG (5.1% vs 6.0%, p = 0.21) and medical therapy (62.1% vs 64.2%, p = 0.19). However, in-patient mortality (4.1% vs 4.5%, p = 0.51), as well as most of the major clinical events were similar between the 2 groups (Table 4). Average in-hospital cost ($21,123 vs $20,975, p = 0.85) and length of stay (5.1 days vs 5.4 days, p = 0.28) were comparable between the 2 groups.

Discussion

The salient findings of this study could be summarized as follows: (1) Based on NIS data from 2005 to 2014, the in-hospital mortality, as well as major clinical events, were mostly similar in both STEMI and NSTEMI between SLE and non-SLE admissions. (2) NSTEMI admissions with SLE had a higher rate of PCI but a similar rate of CABG compared with non-SLE. (3) STEMI admissions had similar PCI, CABG, and thrombolytic rates between SLE and non-SLE.

The risk of cardiovascular disease in SLE is substantially higher in the first year after the diagnosis, with a sixfold increase compared with patients without SLE[1,18,19] and is twofold higher atherosclerosis burden compared with diabetics.[2] Traditional risk factors for coronary artery disease do not fully explain the increased risk and cardiovascular disease risk prediction models underestimate the risk in this population.[20] Autoimmune vascular damage may contribute to accelerated atherosclerosis, coronary vasculitis, and arterial thrombosis. Therefore, the etiology of acute coronary syndromes may vary from nonocclusive coronary artery or thrombotic occlusion with angiographically normal or mildly diseased coronary vasculature to vascular wall inflammation in young patients and coronary atherosclerosis in older patients.[21] Based on the results of our analysis, AMI in SLE did not necessarily translate into a higher in-hospital mortality rate. We found similar in-hospital mortality and major adverse event rates in both STEMI and NSTEMI admissions with SLE compared with non-SLE. These findings could be attributed to the improved management of STEMI and NSTEMI with prompt revascularization, utilization of newer antiplatelets and anticoagulants, use of high-intensity statin therapy, control of risk factors, and adoption of quality measures that have led to the improvement of in-hospital outcomes after AMI in the general population.[22] Moreover, increased awareness of the association between SLE and cardiovascular disease as well as recognition of the underestimated risk in SLE based on traditional risk factors alone, may have led to early identification and treatment of patients with SLE at higher risk or with cardiovascular disease.

Anti-inflammatory approaches, such as statins and anti-interleukin-1β antibodies, have recently been shown to decrease the rate of cardiovascular events in those with elevated high-sensitivity C-reactive protein. These medications may be also highly effective in prevention of AMI in patients with autoimmune diseases including SLE.[23,24] The use of routine screening in the absence of symptoms with exercise or pharmacologic stress testing may identify asymptomatic patients at high risk for acute coronary syndromes and its role should be evaluated better in SLE patients with risk factors and longer duration of active disease.[25] Finally, the use of aspirin and hydroxychloroquine may prevent cardiovascular events in SLE patients without clinically evident cardiovascular disease.[26] These advances could explain the reasons for worse outcomes in SLE compared with non-SLE reported in the previous cohorts from 1993 to 2004.[6,27]

In a traditional risk-matched cohort, our analysis showed a higher rate of PCI but similar CABG rate in NSTEMI. The developmental risk of atherosclerosis in SLE patients can be broadly divided into 2 categories, traditional (i.e., hypertension, diabetes, smoking) and disease-specific (i.e., lupus nephritis, chronic inflammation, steroid use, and autoimmunity).[6,8–10,28] Because we matched for traditional risk factors of atherosclerosis, the higher rate of PCI in NSTEMI may suggest advanced coronary artery disease at the time of NSTEMI presentation in SLE patients from additional disease-specific risks contributing to atherosclerosis. Indeed, Kaul et al reported an approximately twofold higher incidence of angiographically significant (≥70% stenosis in a major epicardial coronary artery) coronary artery disease in SLE.[29]

There are several limitations to our study. First, the analysis of the NIS database is subject to all the biases of retrospective studies. However, the large sample size of the NIS allows valuable insights into rare disease such as SLE. Second, there is a possibility of coding error as the NIS uses ICD-9 code to identify diseases and clinical events. The exact accuracy of ICD-9 code for SLE is unclear. Third, the NIS data do not contain variables to quantitatively assess the severity of coronary artery disease. The difference in revascularization strategy may not fully represent reflect the severity of coronary artery disease in SLE and non-SLE cohorts. Fourth, the data on immunosuppressive therapy or level of antiphospholipid antibody were not available. Last, variables not included in the NIS were not matched and could work as a confounder. However, we have matched for most of the traditional risk factors and such an effect may be minimal.

In conclusion, revascularization strategies and in-hospital outcomes of AMI, both STEMI and NSTEMI, in SLE were mostly similar compared without SLE in the contemporary era. This suggests that improved outcomes of acute coronary syndrome extend to the subgroup of patients with SLE.

Disclosures

The authors have no conflicts of interest to disclose.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.1016/j.amjcard.2018.09.043.

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