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Coronary Artery Perforations After Contemporary Percutaneous Coronary Interventions: Evaluation of Incidence, Risk Factors, Outcomes, and Predictors of Mortality

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Objectives: We sought to evaluate the incidence, risk factors, in-hospital, and long-term outcomes and predictors of mortality of coronary artery perforations (CAP) in the contemporary percutaneous coronary intervention (PCI) era. Background: CAP is a rare but serious complication of PCI associated with increased risk of morbidity and mortality. Methods: We included 181,590 procedures performed across 47 hospitals in Michigan from January 1, 2010 to December 31, 2015. Endpoints evaluated included the incidence of CAP and its association with in-hospital outcomes. Logistic regression analysis was utilized to determine independent risk factors for CAP and to examine whether the effect of CAP on mortality varied by gender. Results: CAP occurred in 625 (0.34%) patients. Independent predictors for CAP included older age, peripheral arterial disease, presence of left ventricular dysfunction or cardiomyopathy, lower body mass index, pre-PCI insertion of a mechanical ventricular support device, treatment of complex lesions (Type C), and treatment of chronic total occlusions, the latter of which was the strongest predictor of perforation (adjusted odds ratio (OR) 7.01, $P < 0.001$). After adjusting for baseline risk, the incidence of adverse outcomes remained substantially greater in patients with a perforation, with an adjusted OR estimate of 5.00 for mortality (95% CI 3.42–7.31), 3.25 for acute kidney injury (95% CI 2.30–4.58), and 5.26 for transfusion (95% CI 4.03–6.87) (all $P < 0.001$). Perforation was associated with a higher mortality in women than men (interaction P value = 0.01). Conclusions: CAP is a rare complication but is associated with high morbidity and mortality especially in women. Further investigation is warranted to determine why women fare worse after CAP.

Key words: coronary aneurysm/dissection/perforation; gender; outcomes/studies; health care outcomes

Additional Supporting Information may be found in the online version of this article.

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INTRODUCTION

The incidence of coronary artery perforation (CAP) during percutaneous coronary interventions (PCI) is estimated at 0.1–0.84% [1]. Although rare, CAP is associated with an increased risk of adverse outcomes including tamponade, myocardial infarction, need for emergency coronary artery bypass grafting (CABG), and death [2–4]. The majority of studies thus far have included a modest number of patients and it is unclear which preprocedural patient factors are associated independently with an increased risk of CAP as well as which factors increase the risk of adverse outcomes after CAP. In addition, given improvement of equipment and therapies, advancing age of the population and changing guidelines, PCI utilization in certain higher risk subgroups—including the elderly or those with chronic total occlusions (CTO)—has been increasing [5–8]. This study represents a large cohort of patients developing CAP after PCI and provides an updated analysis of the incidence, risk factors, and in-patient and long-term outcomes associated with CAP in contemporary practice.

METHODS

We included data from patients undergoing PCI at 47 hospitals participating in the Blue Cross Blue Shield of Michigan Cardiovascular Consortium (BMC2). The details of the BMC2 registry and its data collection and auditing process have been described previously [9]. Briefly, procedural data on all patients undergoing PCI at participating hospitals are collected using standardized data collection forms. Baseline data include clinical, demographic, procedural, and angiographic characteristics as well as medications used before, during, and after the procedure, and in-hospital outcomes. All data elements have been prospectively defined, and the protocol is approved by local institutional review boards at each hospital. In addition to a random audit of 2% of all cases, medical records of all patients undergoing multiple procedures or CABG and of patients who died in the hospital are reviewed routinely to ensure data accuracy.

The study population for this analysis included all consecutive patients who underwent PCI between January 2010 and December 2015. If a patient underwent more than one PCI procedure during a hospital stay, only procedural data reflecting the earliest procedure is included in this analysis. For a subset of Medicare beneficiaries in the dataset, 90-day readmission and long-term survival data were available through indirect matching on admission, discharge, and procedure dates for the index hospitalization, patient gender and date of birth, and hospital and operator NPI numbers with Medicare data for PCI episodes in collaboration with the Michigan Value Collaborative (MVC) [10,11].

Study Endpoints

In-patient mortality was defined as mortality from any cause during the initial hospitalization following PCI. Contrast-induced acute kidney injury (CI-AKI) was defined as an increase in preprocedural to postprocedural serum creatinine ≥ 0.5 mg/dl, as this definition has been strongly associated with in-patient mortality and new requirement for dialysis [12]. Preprocedural serum creatinine values were measured within 30 days prior to PCI, with the value closest to time of PCI chosen as the baseline value. Peak postprocedural serum creatinine was defined as the highest value after the PCI and before the next procedure or discharge.

Statistical Analysis

Univariate comparisons were performed using student *t* tests for continuous measures, and Fisher exact tests for dichotomous measures. The Cochrane-Armitage trend test was used to assess whether the rate of CAP and the rate of treatment of CTO lesions changed over time. Multivariate logistic regression with stepwise variable selection using Akaike Information Criteria was utilized to identify preprocedural clinical and demographic patient characteristics associated with the development of CAP. Patient characteristics included as candidate predictors were chosen based on both significant univariate differences as well as associations with CAP observed in previous studies [1–3].

Preprocedural patient risk of in-hospital outcomes including mortality, transfusion, and CI-AKI were estimated using the current BMC2 random Forest risk models from baseline patient clinical and demographic characteristics. The models are implemented for patient preprocedural risk prediction, and model inputs are described at the SCAI/BMC2 PCI online risk prediction tool available at both the BMC2 https://bmc2.org/calculators/multi and SCAI http://www.scai.org/PCIRiskAssessmentTools/default.aspx websites [13–16]. Risk-adjusted mortality, transfusion, and CI-AKI rates were estimated for subgroups by the overall collaborative outcome incidence multiplied by the ratio of observed-to-expected outcome rates for the subgroup (overall rate $\times$ O/E ratio for subgroup).

The association of CAP with adverse outcomes of in-patient mortality, need for transfusion, and development of CI-AKI was assessed using multivariate logistic regression models adjusting for baseline patient risk. To assess whether the effect of CAP on outcomes potentially varied by gender, likelihood ratio tests were

TABLE I. Characteristics of Patients With and Without Coronary Artery Perforation

Characteristic	Coronary perforation: no. (%)		*P* value
	No $N = 180{,}965$	Yes $N = 625$	
Demographic			
Age (years) ± SD	65.08 ± 12.02	67.35 ± 12.04	<0.001
Female gender	60,349 (33.3%)	263 (42.1%)	<0.001
Current or recent smoker	52,677 (29.1%)	177 (28.4%)	0.696
Height (cm)	171.15 ± 10.59	168.95 ± 10.86	<0.001
Weight (kg)	89.70 ± 21.43	84.21 ± 19.84	<0.001
Historical			
Hypertension	154,589 (85.5%)	553 (88.8%)	0.019
Dyslipidemia	148,315 (82.0%)	516 (82.7%)	0.668
Diabetes mellitus	69,556 (38.4%)	221 (35.4%)	0.113
Peripheral arterial disease	29,069 (16.1%)	126 (20.2%)	0.005
Prior MI	63,507 (35.1%)	244 (39.0%)	0.039
Prior PCI	82,465 (45.6%)	294 (47.0%)	0.464
Prior CABG	33,415 (18.5%)	136 (21.8%)	0.034
Cardiomyopathy or left ventricular systolic dysfunction	19,177 (10.6%)	91 (14.6%)	0.001
Procedural			
PCI status: elective	66,218 (36.6%)	237 (38.0%)	0.478
PCI status: urgent	83,790 (46.3%)	287 (46.0%)	0.868
PCI status: emergency	30,498 (16.9%)	96 (15.4%)	0.325
PCI status: salvage	364 (0.2%)	4 (0.6%)	0.015
Arterial access site: radial	37,473 (20.7%)	123 (19.7%)	0.538
Pre-PCI insertion of IABP or other mechanical ventricular support device	2,467 (1.4%)	25 (4.0%)	<0.001
Pre-PCI left ventricular ejection fraction ± SD	51.90 ± 12.84	49.89 ± 14.42	0.003
Cardiogenic shock at start of PCI	3,765 (2.1%)	24 (3.8%)	0.002

SD: standard deviation; MI: myocardial infarction; PCI: percutaneous coronary intervention; CABG: coronary artery bypass grafting; IABP: intra-aortic balloon pump.

utilized to determine whether inclusion of a gender by CAP interaction term significantly improved model fit.

In the subset of Medicare patients for which long-term survival data were available, propensity score matching was employed to account for baseline patient clinical and demographic variables that could confound the analysis of the impact of CAP on postdischarge survival. Logistic regression was utilized to construct the propensity score, and each CAP patient was matched to 10 similar non-CAP cases without replacement using a greedy algorithm. Variables included in the propensity score model and comparison of the cohorts are included in Supporting Information, Tables I and II. Kaplan–Meier incidence curves were used to visualize postdischarge survival by group, and Cox proportional hazard regression was utilized to assess differences in survival between groups.

RESULTS

Baseline Demographics and Clinical Characteristics

Between January 2010 and December 2015, 181,590 patients underwent PCI in the state of Michigan and were included in this analysis. Of these patients, 625 (0.34%) suffered CAP during the PCI procedure and 41 (6.56%) patients with perforation died before discharge. The rate of perforation overall was relatively stable over the 6 years included in the analysis (Supporting Information, Fig. 1A). The proportion of PCI cases where a CTO lesion was treated increased in statistically significant fashion over the same 6-year period, from 1.6% to 2.8% of all cases (Cochran Armitage trend test $P < 0.001$), while the incidence of perforation among the subgroup of CTO cases did not demonstrate a statistically significant trend (Cochrane Armitage trend test $P = 0.06$) (Supporting Information, Fig. 1B,C).

Baseline clinical, demographic, and procedural characteristics for patients with and without perforation are provided in Table I. Patients with CAP were older, more likely to be female, have peripheral arterial disease or heart failure, require intra-aortic balloon pump (IABP) or other mechanical ventricular support prior to PCI, and to be in cardiogenic shock at the start of the procedure compared to those without CAP. Conversely, patients with perforations were less likely to have diabetes.

Of the 625 patients who developed CAP, 22 (3.52%) underwent postprocedural CABG, of which 2 (9.09%) died before discharge.

TABLE II. Perforations by Device Type and Type of Coronary Lesion

	Percentage of cases where device was used or lesion variable present (%)	Perforation rate in cases with device or lesion variable (%)	Perforation rate in cases without device or lesion variable (%)	Odds ratio	*P* value (Fisher exact test)
Intracoronary device type					
Thrombectomy	5.67	0.31	0.33	0.940	0.850
Cutting balloon	4.92	0.42	0.32	1.301	0.155
Atherectomy	1.03	0.98	0.32	3.051	<0.001
Bare metal stent	17.69	0.32	0.33	0.952	0.731
Drug-eluting stent	72.13	0.26	0.50	0.524	<0.001
Extraction catheter	3.51	0.18	0.33	0.535	0.043
Embolic protection	2.10	0.42	0.33	1.281	0.357
Laser	0.13	2.82	0.33	8.876	<0.001
Type of coronary lesion					
Chronic total occlusion	1.91	1.91	0.30	6.514	<0.001
Type C lesion	56.62	0.36	0.29	1.242	0.017
Thrombus present	15.91	0.26	0.34	0.761	0.036
Bifurcation lesion	5.67	0.31	0.33	0.940	0.850

Association of CAP with Intracoronary Devices and Coronary Lesion Variables

Among the 625 cases of CAP, 524 (83.8%) involved only one treated vessel and these cases were utilized to examine the association of CAP with specific devices or coronary lesion variables (Table II). Lesion length was significantly longer in vessels that developed CAP (29.24 ± 19.66 mm) compared to those without CAP (23.74 ± 13.46 mm) ($P < 0.001$). The use of atherectomy and laser were each associated with significantly higher rates of CAP with odds ratio of 3.05 and 8.88, respectively (both $P < 0.001$). When analyzing the treatment of specific coronary lesion variables, CAP occurred in 1.91% of patients with a treated CTO lesion compared to only 0.3% of patients without CTO, and a CTO PCI was the strongest univariate predictor of increased risk of CAP (odds ratio 6.51, Fisher Exact test $P < 0.001$).

Predictors of CAP

Multivariate stepwise logistic regression identified older age, peripheral arterial disease, presence of left ventricular dysfunction or cardiomyopathy, treatment of high complexity (type C) lesions, treatment of CTO, and use of a mechanical ventricular support prior to PCI as predictors of CAP (Table III). Treatment of CTO remained associated with the greatest estimated risk of CAP after adjusting for other covariates, with patients treated for CTO having a sevenfold greater odds of developing CAP, OR 7.01 (95% CI 5.48–8.98; $P < 0.001$). Greater height, higher body mass index, and presence of diabetes were associated with a lower risk for the development of CAP.

TABLE III. Independent Adjusted Risk Factors for the Development of Coronary Artery Perforations

	Odds ratio	*P* value	95% confidence interval
Age (per 5 year increase)	1.05	0.005	1.015–1.089
Height (per 5 cm increase)	0.91	<0.001	0.872–0.941
BMI (per 1 unit increase)	0.98	0.001	0.965–0.991
PAD	1.21	0.064	0.989–1.483
Diabetes	0.86	0.090	0.723–1.024
Cardiomyopathy or LV dysfunction	1.30	0.026	1.032–1.633
IABP or MV support device implanted prior to PCI	2.04	0.002	1.302–3.207
Pre-PCI creatinine	0.90	0.061	0.803–1.005
Type C/high complexity lesion	1.15	0.100	0.973–1.364
CTO lesion	7.01	<0.001	5.478–8.980
Cardiogenic shock at start of PCI	1.41	0.138	0.896–2.211

BMI: body mass index; PAD: peripheral arterial disease; LV: left ventricle; MV: mechanical ventricular; CTO: chronic total occlusions.

In-Hospital Outcomes

Mean baseline risk estimates and outcomes of patients with and without perforations are provided in Supporting Information, Table III. Mean predicted risks were significantly higher in patients with perforation, reflecting a greater burden of comorbidites in these patients. After adjusting for baseline predicted risk in a logistic regression model, patients with perforations remained at a substantially higher risk of adverse outcomes, with an adjusted OR estimate of 5.00 (95% CI 3.42–7.31) for mortality, 3.25 (95% CI 2.30–4.58) for CI-AKI, and 5.26 (95% CI 4.03–6.87) for transfusion (all $P < 0.001$).

TABLE IV. Characteristics of Patients with Coronary Artery Perforations who were Discharged Alive Versus Deceased

Characteristic	Discharged alive $N = 584$	In-hospital death $N = 41$	P value	Absolute standard difference
Demographic				
Age ± SD	66.98 ± 11.94	72.66 ± 12.28	0.006	46.87
Female gender	234 (40.1%)	29 (70.7%)	<0.001	64.85
Current or recent smoker	162 (27.8%)	15 (36.6%)	0.230	18.81
Historical				
Hypertension	518 (89.0%)	35 (85.4%)	0.476	10.90
Dyslipidemia	487 (83.5%)	29 (70.7%)	0.036	30.84
Diabetes mellitus	207 (35.4%)	14 (34.1%)	0.867	2.73
Prior MI	226 (38.7%)	18 (43.9%)	0.509	10.58
Prior PCI	279 (47.8%)	15 (36.6%)	0.165	22.80
Prior CABG	134 (22.9%)	2 (4.9%)	0.007	54.08
CAD Presentation				
STEMI or equivalent	77 (13.2%)	13 (31.7%)	0.001	45.53
Heart failure within 2 weeks	87 (14.9%)	14 (34.1%)	0.001	45.91
Cardiogenic shock within 24 hr	10 (1.7%)	8 (19.5%)	<0.001	60.37
Cardiac arrest within 24 hr	10 (1.7%)	4 (9.8%)	<0.001	35.11
Pre-PCI insertion of IABP or other mechanical ventricular support device	16 (2.7%)	9 (22.0%)	<0.001	61.04
PCI status: elective	231 (39.6%)	6 (14.6%)	0.001	58.56
PCI status: urgent	267 (45.8%)	20 (48.8%)	0.711	5.98
PCI status: emergency	85 (14.6%)	11 (26.8%)	0.036	30.58
PCI status: salvage	0 (0.0%)	4 (9.8%)	<0.001	46.50
Postprocedural outcomes				
Cardiogenic shock	58 (9.9%)	30 (73.2%)	<0.001	167.30
Heart failure	36 (6.2%)	8 (19.5%)	0.001	40.72

SD: standard deviation; CAD: coronary artery disease; MI: myocardial infarction; PCI: percutaneous coronary intervention; CABG: coronary artery bypass grafting; STEMI: ST-segment elevation myocardial infarction; IABP: intra-aortic balloon pump; CK-MB: creatinine kinase; CVA: cerebral vascular accident.

Predictors of In-Hospital Mortality

Table IV provides a comparison of baseline and procedural patient characteristics of patients with CAP who died during the hospitalization and those discharged alive. Among the 41 patients that died, multiple preprocedural patient characteristics, clinical presentations, procedural outcomes, and complications occurred more frequently including older age, female gender, ST-segment elevation myocardial infarction (STEMI) presentation, recent heart failure, cardiogenic shock or cardiac arrest, and pre-PCI insertion of IABP or other mechanical ventricular support. Of the 41 perforation patients who died, 29 (70.7%) were female, compared to only 40.1% of those discharged alive ($P < 0.001$). Thirteen of the 41 perforation deaths (31.7%) occurred in the catheterization lab, and 20 deaths occurred on the same calendar date as the index PCI procedure (48.8%).

Figure 1a–c provides risk-adjusted mortality, CI-AKI, and transfusion rates for subgroups defined by gender and perforation. The effect of CAP on mortality was significantly greater in women compared to men when assessed by multivariate logistic regression model (gender by perforation interaction $P = 0.01$). No significant CAP by gender interaction was observed for the CI-AKI ($P = 0.32$) or transfusion ($P = 0.50$).

Subsequent analysis stratified by gender demonstrated a statistically significant increase in mortality with CAP in both men with adjusted OR = 2.70 (95% CI: 1.37–5.30; $P = 0.004$) and women with an OR = 7.32 (95% CI: 4.60–11.65; $P < 0.001$). By contrast, no significant weight by CAP interaction was observed in the mortality model ($P = 0.80$), indicating no significant evidence that the relationship between CAP and mortality varied across the spectrum of patient weights.

Of the 625 cases of CAP, covered stents were successfully deployed in 70 (11.2%). Of the 70 CAP cases where covered stents were used, 12 (17.1%) patients died versus 29 deaths (5.23%) among 555 patients where covered stents were not utilized. After adjusting for predicted risk of death in logistic regression, the OR for in-patient mortality for cases of CAP treated with covered stents vs no covered stent was 4.46 ($P < 0.001$) likely related to the fact that covered stent use is reserved for perforations that are more likely to be hemodynamically significant.

Long-Term Outcomes

Using propensity score matching, 1,030 Medicare patients without CAP having similar baseline clinical and demographic characteristics to the 103 available

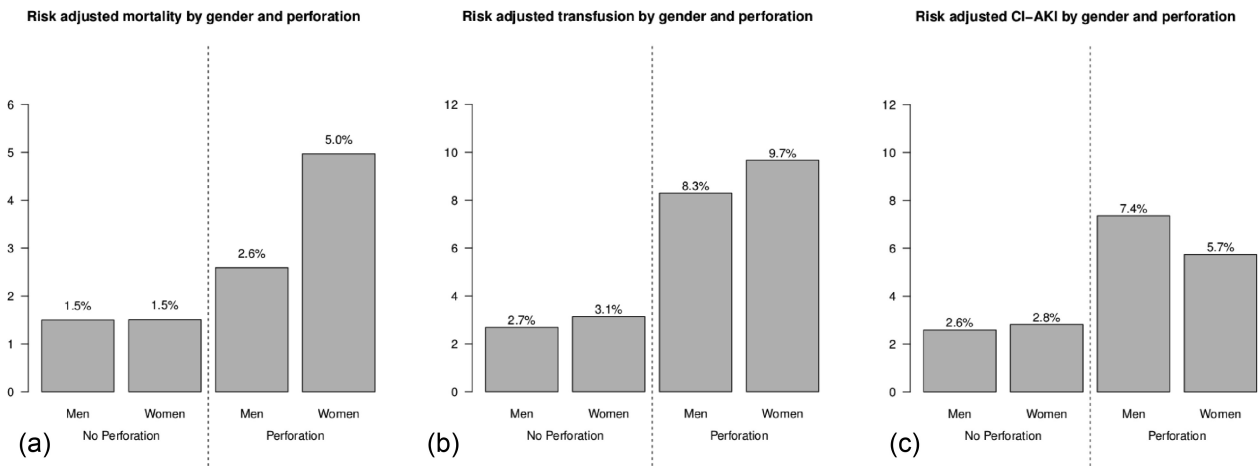

Fig. 1. Risk-adjusted mortality, transfusion, and contrast-induced acute kidney injury (CI-AKI) rates for subgroups defined by gender and perforation. (A) Risk-adjusted mortality associated with coronary perforation in men and women. (B) Risk-adjusted transfusion associated with coronary perforation in men and women. (C) Risk-adjusted CI-AKI associated with coronary perforation in men and women.

Medicare patients with CAP (Supporting Information, Tables I and II) were selected. Median postdischarge follow-up in this cohort was 23 months for both groups, with 34 (33.0%) deaths observed among CAP patients, and 205 (19.9%) deaths among non-CAP patients during follow up. Kaplan–Meier survival curves with confidence band shading are provided in Fig. 2. In Cox proportional hazards regression, CAP was associated with significantly greater mortality (HR = 1.63, $P = 0.008$). In contrast to in-hospital mortality, no significant gender by CAP interaction was observed in Cox regression ($P = 0.430$), and women and men with CAP had comparable estimated mortality at 2 years (men 31.1% (95% CI 14.4–44.6%), women 23.6% (95% CI 10.2–35.0%)). No significant difference was observed for 90-day readmission (30.1% for CAP and 23.8% no CAP, $P = 0.185$).

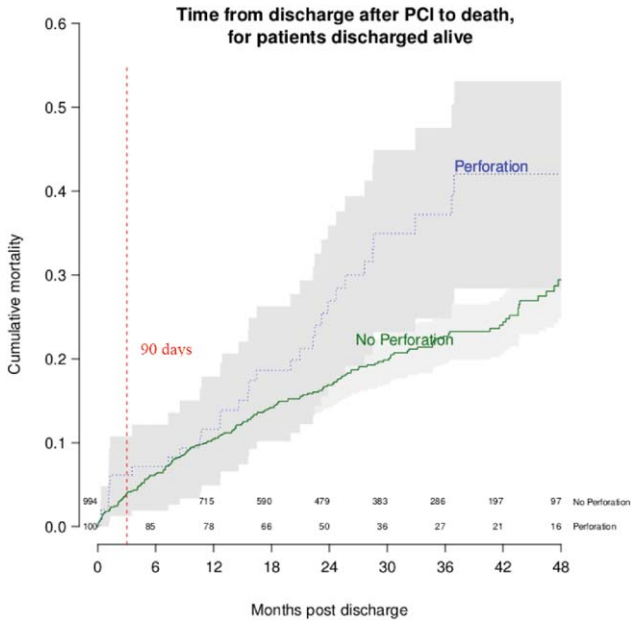

Fig. 2. Long-term mortality of propensity matched patients with and without coronary artery perforation among those discharged alive after PCI. [Color figure can be viewed at wileyonlinelibrary.com]

DISCUSSION

This study includes one of the largest patient cohorts describing the risk of CAP during PCI in contemporary practice. The incidence of CAP in our population was 0.34%, confirming that CAP remains an uncommon event. However, the high in-hospital mortality rate of 6.56% confirms the associated high mortality and highlights the need for further investigation into understanding risk factors, outcomes, and potential therapies for this serious complication.

Small retrospective studies have identified various patient and angiographic characteristics as potential risk factors for CAP including older age, female gender, presence of chronic kidney disease, hypertension, and previous PCI or CABG as well as angiographic characteristics such as type C lesions, chronic total occlusions, calcified lesions, and culprit lesions in the right coronary artery [1–4]. In addition, certain procedural characteristics including a higher balloon-to-artery ratio and the use of atheroablative devices were shown in these limited studies to increase the risk of this serious event [1,3,4].

Our study adds to the existing literature by identifying more than 600 patients with CAP. Some factors that have been suggested to be associated with CAP were

not found to be independently associated with CAP in this large study, including hypertension, chronic kidney disease, and prior CABG [3,4]. However, consistent with prior studies, our analysis identified several patient and procedural characteristics as independent risk factors for CAP, including older age and treatment of chronic total occlusions [2–4]. In fact, treatment of CTO was the strongest risk factor for CAP development, which is an important consideration as recently there has been an increasing interest in treatment of these higher risk lesions as techniques and technology have improved.

Consistent with prior work, we found CAP to be associated with an increased risk of in-hospital mortality [1,2]. However, we also identified that CAP was associated with increased risk of long-term mortality and with other adverse in-hospital outcomes, including development of CI-AKI and the need for transfusion. Possible mechanisms to explain the increased risk of CI-AKI and need for transfusion include hemodynamic compromise related to development of complications of tamponade or myocardial infarction as well as need for additional procedures or surgical repair.

Our study also demonstrated that the effect of CAP on mortality may vary by gender with our results indicating that perforation is significantly more deleterious in women than in men. The etiology of the increased risk of death in women with CAP is unknown but potentially relates to anatomical or hormonal differences between sexes. One hypothesis relates to a presumed smaller vessel diameter or differences in vessel wall thickness size in women that could increase the risk of development of CAP and/or higher grades of CAP [17]. The influence of estrogen on coagulation factors and inflammatory markers has been proposed as a mechanism to explain the increased susceptibility for vascular injury. Our study did not examine the influence of smaller vessel size on the development of CAP, as the BMC2 database does not include information on vessel diameter.

The manner in which CAP is treated is dependent on the severity of the CAP, often graded by Ellis type classification, and whether certain complications associated with CAP, such as tamponade, are present [18]. Potential therapies include the use of prolonged balloon occlusion, deployment of covered stents, thrombin or gelfoam embolization, coils, or CABG. We found that covered stents were utilized in the minority of patients with CAP; however, these patients experienced higher rates of inpatient mortality. We hypothesize that this is related to the use of these therapies in more severe cases of CAP and that the worse outcomes in these patients are related to this underlying higher grade perforation than the use of the covered stent itself. However, there is also evidence that covered stents are at higher risk for restenosis and thrombosis [19]. We do not have data on the exact reason why these patients died and further investigation into optimal treatment of CAP is needed.

There are several limitations to our study. First, our study was a retrospective analysis utilizing data from a large database which does not include details on the severity of CAP (such as Ellis type classification) or vessel size, which would be important to examine when attempting to identify the etiology of the potentially worse prognosis of CAP in women than men. In addition, various outcomes that may develop after CAP—including tamponade, need for emergency surgery or subsequent development of MI—could not be evaluated in our population based on database restrictions. Evaluating incidence and efficacy of various treatments for CAP was limited to assessing the use of covered stents in our population with CAP.

In conclusion, CAP remains an uncommon but serious complication of PCI, associated with an increased incidence of inpatient mortality, CI-AKI, and need for transfusion as well as long-term mortality. Treatment of CTO lesions was the strongest independent predictor of CAP. CAP was more harmful in women than in men in our study. Further investigation into the etiology of the poorer prognosis of CAP in women as well as efficacy of various therapies to treat CAP is needed.

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Editorial Comment

Preventing and Treating Coronary Perforations: Lessons From Disaster Management

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Key Points

- Coronary artery perforations (CAP) are rare in patients undergoing routine PCI with recent contemporary databases reporting an incidence ~0.3%.
- Older age, cardiomyopathy or left ventricular dysfunction, mechanical circulatory support prior to PCI, and CTO lesions have the strongest association with CAP.
- Prevention, early detection, preparedness and familiarity with the equipment used to treat perforations (such as efficient use of covered stents, and fat or coil embolization) and regrouping to examine and educate about these experiences are key for improving our response to cath lab disasters.

Parsh et al. analyzed coronary artery perforations from a large contemporary PCI United States (US) registry reporting three key findings [1]. Coronary perforations: (a) are rare (0.3%); (b) occur more frequently in more complex lesions, especially chronic total occlusions (CTOs); and (c) are associated with high rates of death, acute kidney injury, and transfusion. The low incidence of coronary perforations (or any procedural complication) in the cardiac catheterization laboratory is both good and bad news. On one hand, the low complication rate is certainly excellent news for patients (and operators) who are infrequently subjected to this complication. On the other hand the very low incidence of procedural

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