Outcomes of Percutaneous Coronary Intervention in Patients With Rheumatoid Arthritis

Luke P Dawson, MBBS, MPH[a], Diem Dinh, BAppSci, PhD[b], Jessica O'Brien, MBBS[c], Stephen J. Duffy, MBBS, PhD[b,c,d], Emma Guymer, MBBS[e,f], Angela Brennan, RN[b], David Clark, MBBS, DMedSci[g], Ernesto Oqueli, MD[h,i], Chin Hiew, BSc(Hons), MBBS[j], Melanie Freeman, MBBS[k], Christopher M. Reid, PhD[b], and Andrew E. Ajani, MBBS, MD[a,b,l,*], on behalf of the Melbourne Interventional Group (MIG) Investigators

Rheumatoid arthritis (RA) is the most common inflammatory arthritis and is associated with increased risk of cardiovascular events and mortality. Evidence regarding outcomes following PCI is limited. This study aimed to assess differences in outcomes following percutaneous coronary intervention (PCI) between patients with and without RA. The Melbourne Interventional Group PCI registry (2005 to 2018) was used to identify 756 patients with RA. Outcomes were compared with the remaining cohort (n = 38,579). Patients with RA were older, more often female, with higher rates of hypertension, previous stroke, peripheral vascular disease, obstructive sleep apnea, chronic lung disease, myocardial infarction, and renal impairment, whereas rates of dyslipidemia and current smoking were lower, all p <0.05. Lesions in patients with RA were more frequently complex (ACC/AHA type B2/C), requiring longer stents, with higher rates of no reflow, all p <0.05. Risk of long-term mortality, adjusted for potential confounders, was higher for patients with RA (hazard ratio 1.53, 95% confidence interval 1.30 to 1.80; median follow-up 5.0 years), whereas 30-day outcomes including mortality, major adverse cardiovascular events, bleeding, stroke, myocardial infarction, coronary artery bypass surgery, and target vessel revascularization were similar. In subgroup analysis, patients with RA and lower BMI ($P_{for\ interaction} < 0.001$) and/or acute coronary syndromes ($P_{for\ interaction} = 0.05$) had disproportionately higher risk of long-term mortality compared with patients without RA. In conclusion, patients with RA who underwent PCI had more co-morbidities and longer, complex coronary lesions. Risk of short-term adverse outcomes was similar, whereas risk of long-term mortality was higher, especially among patients with acute coronary syndromes and lower body mass index. **(Am J Cardiol 2021;140:39−46)**

Rheumatoid arthritis (RA) is the most common inflammatory arthritis and is associated with increased risk of cardiovascular disease (CVD) and cardiovascular mortality, with a similar magnitude to that seen in diabetes mellitus.[1,2] Potential mechanisms for this association include systemic inflammation,[3,4] a higher prevalence of traditional cardiovascular risk factors in RA cohorts,[3,5] medications that may have a detrimental effect on plaque stability (such as glucocorticoids and NSAIDs),[6,7] and shared genetic risk factors.[8] Percutaneous coronary intervention (PCI) is the most common method of revascularization in ischemic heart disease. However, data assessing differences in patient characteristics, procedural details and outcomes following PCI among patients with RA are limited to relatively small cohorts with conflicting results.[5,9−12] Data regarding long-term outcomes are especially scant. The current study aimed to determine differences in characteristics and outcomes between patients with and without RA in a large registry-based PCI cohort.

Methods

We analyzed data from prospectively collected consecutive PCI procedures included in the Melbourne Interventional Group (MIG) registry between January 2005 and December 2018. The cohort was divided into patients with

[a]Department of Cardiology, Royal Melbourne Hospital, Melbourne, Victoria, Australia; [b]Centre of Cardiovascular Research and Education in Therapeutics, Monash University, Melbourne, Victoria, Australia; [c]Department of Cardiology, The Alfred Hospital, Melbourne, Victoria, Australia; [d]Baker Heart and Diabetes Institute, Melbourne, Victoria, Australia; [e]Department of Rheumatology, Monash Medical Centre, Melbourne, Victoria, Australia; [f]Department of Medicine, Monash University, Melbourne, Victoria, Australia; [g]Department of Cardiology, Austin Health, Melbourne, Victoria, Australia; [h]Department of Cardiology, Ballarat Health Services, Ballarat, Victoria, Australia; [i]School of Medicine, Faculty of Health, Deakin University, Geelong, Victoria, Australia; [j]Department of Cardiology, University Hospital, Geelong, Victoria, Australia; [k]Department of Cardiology, Box Hill Hospital, Melbourne, Victoria, Australia; and [l]Department of Medicine, Melbourne University, Victoria, Australia. Manuscript received August 3, 2020; revised manuscript received and accepted October 22, 2020.

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*Corresponding author: Tel: +61 9342 7000.

E-mail address: andrew.ajani@mh.org.au (A.E. Ajani).

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RA and patients without RA to determine differences in procedural characteristics and outcomes.

The MIG registry, which has previously been described in detail,[13] is a voluntary PCI registry that prospectively collects data on all PCI procedures at 6 tertiary referral hospitals in Victoria, Australia. Baseline, clinical and procedural characteristics are recorded on standardized case-report forms at the time of index PCI, with 30-day outcome data collected by site nurse coordinators by telephone follow-up. An independent audit of a number of verifiable fields from 5% of enrolled patients is periodically conducted at all enrolling sites by an investigator not affiliated with the registry.[14] In the most recent audit, overall accuracy was 98%, which compares favorably to other large registries. Long-term mortality is derived through periodic linkage with the Australian National Death Index (NDI). Approval was gained from each individual hospital's ethics committee before commencement of the registry and "opt-out" informed consent obtained in all patients. Ethics approval for this specific analysis was also obtained from the Alfred Hospital ethics committee.

A diagnosis of RA was determined from medical records or patient self-report at time of PCI by the clinician completing the standardized MIG case-report form. Indication for PCI was classified as ST-elevation myocardial infarction (STEMI), non-ST elevation acute coronary syndrome (NSTEACS), and nonacute coronary syndrome (non-ACS) presentations according to standard definitions.[15] The MIG registry does not collect data regarding RA duration, disease activity or treatments, and therefore these data are not presented in this study.

Outcomes collected included in-hospital PCI complications, 30-day outcomes, and long-term mortality. In-hospital complications were recorded by clinicians on standardized case reports during index PCI admission. Medication status and outcomes at 30 days, including cardiac and noncardiac mortality, stroke, myocardial infarction (MI), coronary artery bypass surgery (CABG), target vessel revascularization (TVR), major adverse cardiovascular events (MACE, including death, MI, or TVR), and readmission details were determined by patient telephone follow-up performed by hospital site nurse coordinators. All in-hospital and 30-day outcomes were defined according to standard Academic Research Consortium definitions.[16] Mortality beyond 30 days was determined through NDI-linked data.

Continuous data are expressed as mean ± standard deviation for parametric data or median (interquartile range) for nonparametric data and are compared using the Student's *t* test or Mann-Whitney U test as appropriate. Categorical variables are presented as number (%) and are compared using the Pearson's chi-square test. Multivariable analysis was performed to determine whether RA status was independently associated with risk of long-term mortality or 30-day adverse outcomes. Cox regression proportional hazards analysis was used to identify unadjusted and adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) for long-term mortality and 30-day cardiac and noncardiac mortality, while logistic regression was used to determine odds ratio's (ORs) and 95% CIs for 30-day MACE, stroke, MI, CABG, TVR, and readmission. Potential confounders included in the adjusted analyses were age, gender, body mass index (BMI), diabetes, hypertension, dyslipidemia, peripheral vascular disease, smoking status, previous stroke, previous PCI, previous CABG, STEMI, out-of-hospital cardiac arrest, cardiogenic shock, renal function, and left ventricular ejection fraction. To test the consistency of the observed association with long-term mortality and identify any effect modification, we performed stratified analysis across subgroups that have been found to affect risk of cardiovascular events in patients with RA in previous studies. Subgroups included were age,[1,2,17] gender,[18] diabetes,[3] hypertension,[17] dyslipidemia,[3,4] indication for PCI (whereby ACS may reflect inflammatory state),[19] BMI,[3] and current smoking.[20] Finally, the unadjusted association between RA status and long-term mortality, overall and separated by indication type, was graphically displayed with Kaplan-Meier curves. Statistical analysis was conducted using Stata version 14.2 for Windows (College Station, TX). All calculated p values were 2-sided, and a p value <0.05 was considered statistically significant.

Results

A total of 41,146 patient procedures were entered into the MIG registry between January 2005 and December 2018. Of these, 756 patients were recorded as having RA, 38,579 patients did not have RA, and 1,811 (4.4%) had incomplete RA field data and were excluded (Supplemental Figure 1).

Patients with RA were older, more frequently female, had lower BMI and were less frequently active smokers compared with patients without RA (Table 1). Co-morbidities were generally more common among patients with RA, with higher rates of hypertension, previous stroke, peripheral vascular disease, obstructive sleep apnea, chronic lung disease, renal impairment, previous MI, and previous valve surgery. Rates of diabetes, either diet controlled or insulin-requiring, and heart failure were similar between patients with and without RA, while rates of dyslipidemia were lower.

Indications for PCI, including rates of ACS, out-of-hospital cardiac arrest, and cardiogenic shock, were similar between groups. Patients with RA had lower diastolic blood pressure, higher pulse pressure, but similar systolic blood pressure at time of PCI.

Lesions among patients with RA were more frequently complex (ACC/AHA type B2/C) and long (stent ≥20 mm in length) compared with patients without RA (Supplemental Table 1). However, rates of stent thrombosis, in-stent restenosis, bifurcation location, multivessel disease, and chronic total occlusion PCI were similar.

Rates of transient or persistent no reflow during PCI were higher among the RA group. However, rates of other complications were similar including coronary perforation, blood transfusion requirement, vascular complications, coronary dissection, and major and minor bleeding.

Among patients with RA, rates of angiotensin converting enzyme inhibitors (ACEI) or angiotensin receptor II blocker (ARB) use were lower, whereas rates of aldosterone antagonist and nitrate use were higher (Table 2). A trend toward lower rates of statin use was observed, but this did not reach significance. Patients with RA more frequently used 2 or 3 antianginal agents at 30 days compared with patients without RA. Other medications, including antiplatelets,

Table 1
Baseline characteristics according to rheumatoid arthritis status

Variable	Rheumatoid arthritis: Yes n = 756	Rheumatoid arthritis: No n = 38,579	p
Age, mean ± SD (years)	68.9 ± 10.0	64.6 ± 12.0	<0.001
<50	24 (3%)	4335 (11%)	<0.001
50−59	114 (15%)	8,828 (23%)	
60−69	242 (32%)	11,372 (30%)	
70−79	258 (34%)	9,500 (25%)	
≥80	118 (16%)	4544 (12%)	
Women	302 (40%)	8,824 (23%)	<0.001
Body mass index, mean ± SD (kg/m^2)	28.0 ± 5.7	28.6 ± 5.4	<0.01
<25	208 (30%)	271 (39%)	0.03
25−29	271 (39%)	14,353 (41%)	
≥30	214 (31%)	11,597 (33%)	
Ever smoker	472 (66%)	25,262 (67%)	0.40
Current smoker	143 (20%)	9,662 (26%)	<0.001
Hypertension	530 (70%)	25,661 (67%)	0.04
Dyslipidemia	456 (61%)	25,303 (66%)	<0.01
Diabetes mellitus	178 (24%)	9,863 (26%)	0.21
Insulin requiring	58 (8%)	2,751 (7%)	0.57
Prior stroke	71 (9%)	2,242 (6%)	<0.001
Peripheral vascular disease	65 (9%)	2,294 (6%)	<0.01
Obstructive sleep apnea	77 (10%)	1,798 (5%)	<0.001
Chronic lung disease	164 (22%)	4,422 (12%)	<0.001
Congestive heart failure	40 (5%)	1,593 (4%)	0.11
eGFR, mean ± SD (ml/min/1.73m^2)	80.0 ± 34.1	90.0 ± 38.9	<0.001
>60	500 (69%)	28,149 (76%)	<0.001
30−59	200 (28%)	7,667 (21%)	
<30	24 (3%)	1,236 (3%)	
Coronary artery disease			
Prior myocardial infarction	240 (32%)	10,238 (27%)	<0.01
Prior PCI	200 (27%)	9,996 (26%)	0.74
Prior bypass surgery	58 (8%)	3,124 (8%)	0.67
Prior valve surgery	13 (2%)	341 (1%)	0.02
Indication			0.18
Nonacute coronary syndrome	231 (31%)	12,976 (34%)	
Non-ST elevation acute coronary syndrome	284 (38%)	13,591 (35%)	
ST-elevation myocardial infarction	241 (32%)	11,990 (31%)	
Out-of-hospital cardiac arrest	22 (3%)	1,278 (3%)	0.54
Cardiogenic shock	36 (5%)	1,504 (4%)	0.23
Left ventricular ejection fraction ≤45%	163 (24%)	7,985 (24%)	0.60
Systolic blood pressure, mean ± SD (mm Hg)	129.6 ± 25.1	128.6 ± 25.5	0.35
Diastolic blood pressure, mean ± SD (mm Hg)	69.7 ± 15.3	71.7 ± 14.7	<0.01
Pulse pressure, mean ± SD (mm Hg)	59.8 ± 21.8	56.9 ± 21.2	<0.01

Data are presented as number (%) unless otherwise indicated.

anticoagulants, heart failure medications, and other lipid lowering agents were used by similar proportions of both groups.

Patients with RA had increased risk of 30-day noncardiac mortality in unadjusted analysis (HR 2.07, 95% CI 1.16 to 3.69, p = 0.01), but not after adjustment for potential confounders (HR 1.66, 95% CI 0.84 to 3.29, p = 0.15; Table 3). Similarly, risk of readmission within 30 days of PCI was higher in unadjusted (HR 1.34, 95% CI 1.10 to 1.63, p <0.01), but not adjusted analysis (HR 1.22, 95% CI 0.97 to 1.54, p = 0.08). No significant differences in risk for other 30-day outcomes (including all-cause mortality, MACE, stroke, MI, CABG, and TVR) were observed in either unadjusted or adjusted models.

Readmission rates within 30 days of PCI were higher among patients with RA (Table 3). Among patients with RA, rates of readmission for "other" (ie, noncardiovascular) reasons were higher, rates of readmissions for planned PCI were lower, and rates of readmission for heart failure, MI, recurrent angina, arrhythmia, CABG, stroke, and elective angiography were similar (Table 4). In the RA group, the most common reasons for readmission classified as "other" included cardiac reasons not captured by other fields (such as chest pain, dyspnea, and syncope—28%), infective presentations (25%), and anemia, bleeding or vascular complications of PCI (23%). Readmissions specifically relating to RA were infrequent (5%).

Table 2
Medication use at 30 days according to rheumatoid arthritis status

Medication	Rheumatoid arthritis		p
	Yes	No	
Antiplatelet			
Aspirin	669 (96%)	34,399 (97%)	0.19
P2Y12 inhibitor	666 (96%)	34,170 (97%)	0.49
Anticoagulant			
Novel oral anticoagulant	21 (5%)	736 (3%)	0.19
Warfarin	38 (6%)	2,164 (6%)	0.50
Lipid lowering			
Statin	643 (93%)	33,285 (95%)	0.06
Ezetimibe	48 (7%)	1,974 (6%)	0.16
Fibrates	12 (2%)	673 (2%)	0.70
Beta-blocker	538 (78%)	27,698 (79%)	0.59
ACE-inhibitor or angiotensin receptor blocker	517 (75%)	27,686 (79%)	0.02
Aldosterone antagonist	30 (5%)	1,017 (3%)	0.03
Ca^{2+} channel blockers	122 (19%)	5,496 (17%)	0.21
Nitrates	95 (14%)	3,346 (10%)	<0.001
No. of antianginals			0.001
0	109 (16%)	5,183 (15%)	
1	428 (62%)	24,106 (69%)	
2	132 (19%)	5,164 (15%)	
3	21 (3%)	702 (2%)	

Data are presented as number (%) unless otherwise indicated.

The risk of long-term mortality was higher for patients with RA in both unadjusted analysis (HR 1.70, 95% CI 1.49 to 1.95, p <0.001) and after adjustment for potential confounders (HR 1.53, 95% CI 1.30 to 1.80, p <0.001; Table 3). Median NDI-linked follow-up was 5.0 years (interquartile range 2.0 to 8.9 years). Kaplan-Meier survival curves by indication subtype are depicted in Figure 1.

The association between RA and long-term mortality was further assessed through stratified analysis among subgroups with selection based on previous studies identifying effect modification of the subgroup on cardiovascular risk or mortality among patients with RA (Figure 2). Patients with RA and ACS as indication for PCI had disproportionately higher risk of long-term mortality compared with patients with RA who underwent PCI for non-ACS indications (HR 1.61, 95% CI 1.35 to 1.93 vs HR 1.02, 95% CI 0.69 to 1.52, $P_{for\ interaction} = 0.05$). Similarly, patients with RA of low or normal BMI (BMI <25.0 kg/m^2) had markedly higher risk of long-term mortality compared with patients without RA (HR 2.08, 95% CI 1.62 to 2.68), whereas overweight (BMI 25.0 to 29.9 kg/m^2) patients with RA had a moderate elevation in risk (HR 1.54, 95% CI 1.17 to 2.03), and obese patients with RA (BMI >30.0 kg/m^2) had no increase in risk (HR 0.93, 95% CI 0.63 to 1.37; $P_{for\ interaction} < 0.001$). While there were some variations in magnitude of risk across the other subgroups, none reached significance.

Table 4
Reasons for readmission within 30 days of percutaneous coronary intervention according to rheumatoid arthritis status

Reason	Rheumatoid arthritis		p
	Yes	No	
Heart failure	4 (4%)	220 (5%)	0.48
Myocardial infarction	3 (3%)	182 (4%)	0.44
Recurrent angina	21 (19%)	985 (23%)	0.34
Arrhythmia	6 (6%)	123 (3%)	0.11
PCI—planned	15 (14%)	1053 (25%)	<0.01
Bypass surgery	0 (0%)	97 (2%)	0.11
Stroke	1 (0.9%)	21 (0.5%)	0.54
Elective angiogram	0 (0%)	26 (0.6%)	0.41
Other	60 (55%)	1559 (36%)	<0.001
Days to readmission	11.5 (3-22)	12 (5-20)	0.35

Data are presented as number (%), while days to readmission are presented as median (interquartile range).

Discussion

The main findings of the current study regarding outcomes following PCI among patients with RA can be summarized

Table 3
Risk of adverse outcomes according to rheumatoid arthritis status

	Rheumatoid arthritis		Unadjusted		Adjusted	
	Yes	No	Odds/hazards ratio (95% CI)	p	Odds/hazards ratio (95% CI)	p
Long-term outcomes						
National Death Index-linked mortality*	213 (28%)	7,423 (19%)	**1.70 (1.49−1.95)**	**<0.001**	**1.53 (1.30−1.80)**	**<0.001**
30-day outcomes						
All-cause mortality*	33 (4%)	1,270 (3%)	1.31 (0.93−1.85)	0.12	1.11 (0.70−1.77)	0.65
Noncardiac mortality*	12 (2%)	296 (1%)	**2.07 (1.16−3.69)**	**0.01**	1.66 (0.84−3.29)	0.15
Major adverse cardiac events†	50 (7%)	2,427 (6%)	1.05 (0.79−1.41)	0.72	0.83 (0.56−1.22)	0.34
Stroke†	3 (0.4%)	189 (0.5%)	0.81 (0.26−2.54)	0.72	0.61 (0.15−2.47)	0.49
Myocardial infarction†	14 (2%)	735 (2%)	0.97 (0.57−1.66)	0.92	0.61 (0.30−1.24)	0.17
Bypass surgery†	5 (0.7%)	402 (1%)	0.63 (0.26−1.53)	0.31	0.46 (0.15−1.45)	0.19
Target vessel revascularization†	12 (2%)	860 (2%)	0.71 (0.40−1.26)	0.24	0.58 (0.29−1.17)	0.13
Readmission†	118 (16%)	4642 (13%)	**1.34 (1.10−1.63)**	**<0.01**	1.22 (0.97−1.54)	0.08

Statistically significant results are highlighted in bold.

Adjusted model includes age, gender, body mass index, diabetes mellitus, hypertension, dyslipidemia, peripheral vascular disease, smoking status, previous stroke, previous percutaneous coronary intervention, previous bypass surgery, ST-elevation myocardial infarction, out-of-hospital cardiac arrest, cardiogenic shock, renal function, left ventricular ejection fraction.

* Cox proportional hazards regression analysis.

† Multivariable logistic regression analysis.

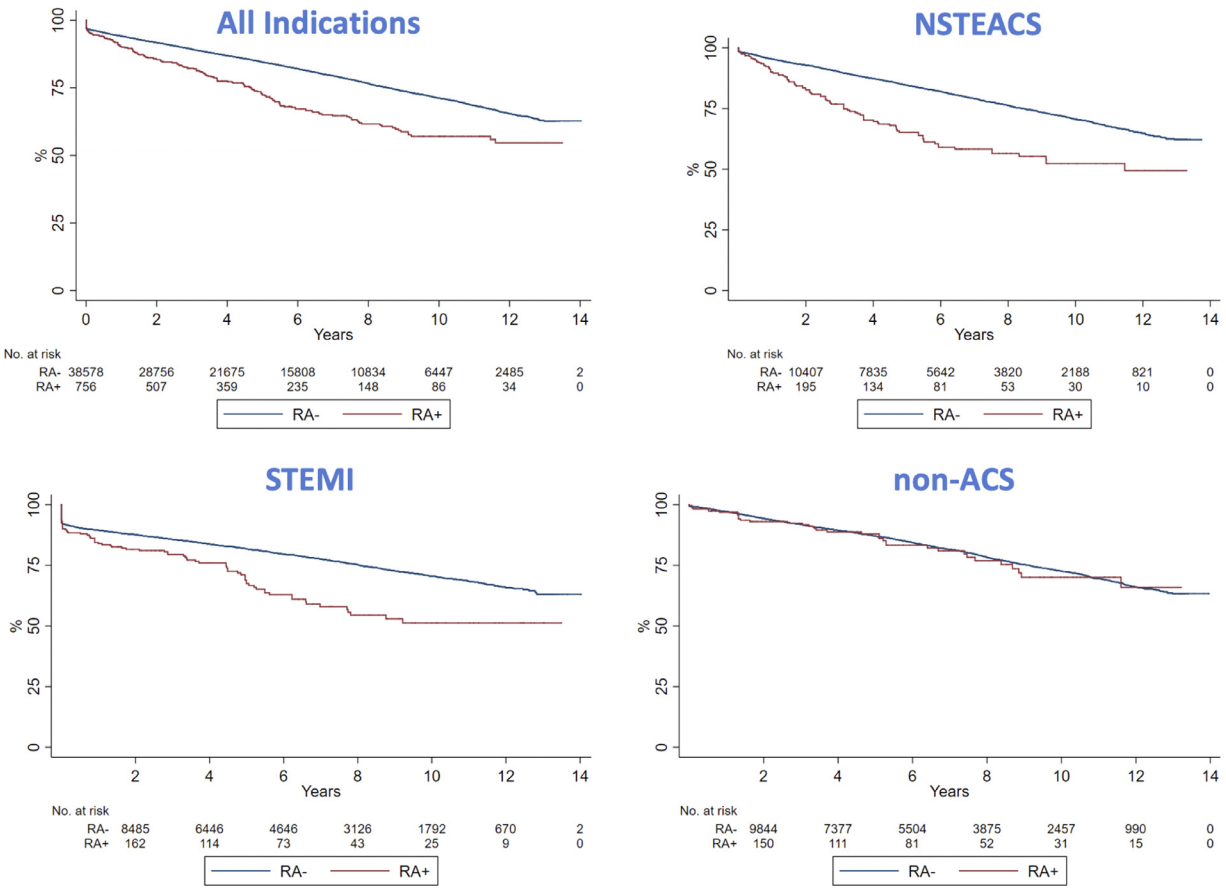

Figure 1. All-cause mortality through 14 years. Kaplan-Meier curves for unadjusted long-term survival out to 14 years following PCI procedures separated by rheumatoid arthritis status among the entire cohort (top left panel), and by indication subgroups—ST-elevation myocardial infarction (bottom left panel), Non-ST elevation myocardial infarction (top right panel), and nonacute coronary syndromes (bottom right panel).

as follows: (1) patients with RA are older, more frequently female, and have more comorbidities; (2) patients with RA may be more symptomatic following PCI (reflected by 30-day antianginal use); (3) coronary lesions among patients with RA are more frequently long and complex with higher rates of transient or persistent no reflow; and (4) although short-term outcomes are similar (aside from readmission rates), the risk of long-term mortality is higher, especially among patients with ACS and lower BMI.

An abundance of data have identified an increased risk of incident MI and cardiovascular mortality in RA cohorts, with a magnitude suggested to be similar to that seen among patients with diabetes mellitus (HRs 1.38 to 1.70).[1,2] Risk increases with RA disease activity and duration, and coronary plaque prevalence, extent and severity are all higher in comparison to controls.[21,22] The reasons for these observations are thought to be fourfold. First, RA cohorts have a higher prevalence of classical cardiovascular risk factors compared with the general population (an observation confirmed in our study).[3,5] Second, measures of inflammation and disease activity in RA are associated with MI risk.[3,4] Third, glucocorticoids and nonsteroidal anti-inflammatory agents (both commonly used in RA) are associated with detrimental plaque remodeling and increased MI risk.[6,7] Finally, shared genetic risk factors have been identified common to both RA and CVD.[8]

Our study identifies a number of new observations that further the idea of a more aggressive coronary disease process in patients with RA. Their coronary lesions more frequently required longer stents and were more complex in nature. Transient or persistent no reflow more frequently complicated PCI procedures, which may reflect lesion complexity or possibly greater plaque disruption and thrombotic tendencies in the setting of an underlying inflammatory state. Following PCI, antianginal use was higher—a finding possibly explained by higher rates of microvascular abnormalities as the cause of myocardial ischemia in RA cohorts (rather than obstructive coronary disease where symptoms may improve with PCI).[19] Among limited previous literature regarding RA in PCI cohorts, one smaller study also identified higher rates of longer stent use (consistent with our study), in addition to higher rates of multivessel disease and bifurcation lesions (not observed in our study).[5] The other observations in our study have not previously been reported.

In our subgroup analysis, we found that long-term mortality risk was disproportionately increased among patients with RA with lower BMI or with ACS as the indication for PCI compared with patients without RA. Both of these observations make mechanistic sense. Lower BMI may reflect rheumatoid cachexia—a state of low or normal weight in which pro-inflammatory cytokines mediate involuntary loss of muscle, which is associated with increased

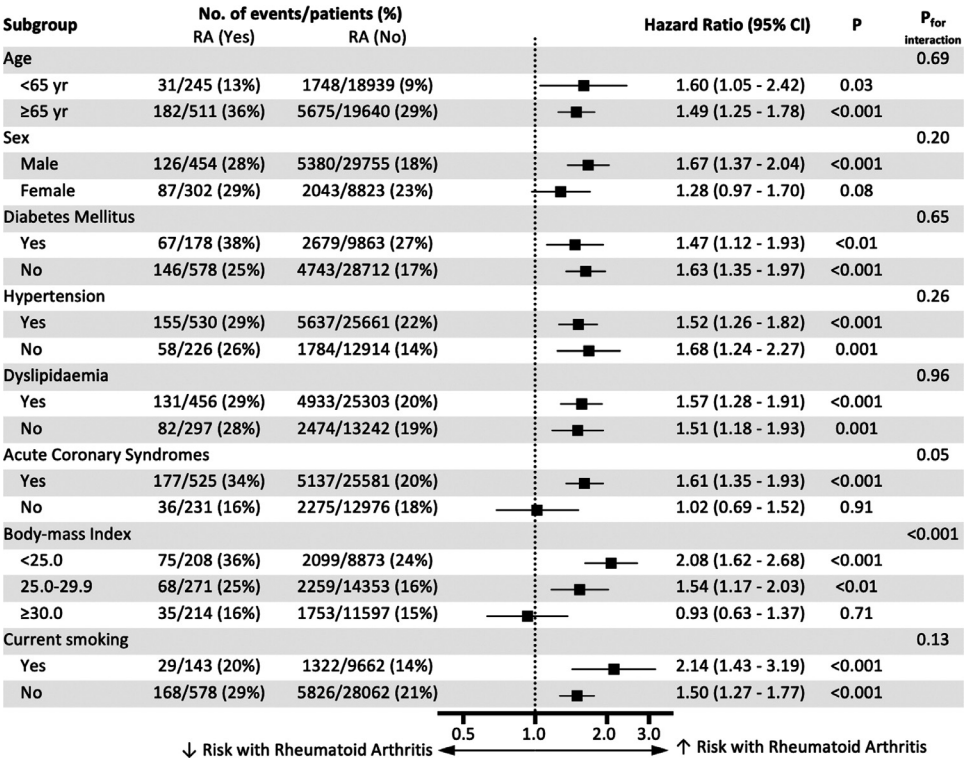

Figure 2. Hazard ratios for long-term mortality in prespecified subgroups. Forest plot summarizing the association between rheumatoid arthritis and the risk of long-term mortality following percutaneous coronary intervention (PCI) among subgroups. Hazard ratios were determined using Cox proportional hazards regression analysis adjusted for age, gender, body mass index, diabetes, hypertension, dyslipidemia, peripheral vascular disease, smoking status, previous stroke, previous PCI, previous bypass surgery, ST-elevation myocardial infarction, out-of-hospital cardiac arrest, cardiogenic shock, renal function, and left ventricular ejection fraction. RA = rheumatoid arthritis, CI = confidence interval.

risk of cardiovascular mortality.[23] In ACS, systemic inflammation plays a significant role in plaque instability, rupture and thrombosis.[24] Levels of systemic inflammation in patients with RA and ACS are likely higher than the non-ACS group, increasing risk of both RA disease progression and cardiovascular events, and thus explaining the disproportionate increase in risk of mortality. Other variables that have demonstrated effect modification in previous studies include: a nonlinear relationship between lipid levels and CVD risk (however, our lipid assessment was limited to dichotomous data),[3] higher age-specific incident MI risk in younger patients with RA (not observed in our cohort),[17] and an interaction between MI risk and smoking in RA (a nonsignificantly higher HR was observed in our cohort).[20]

In our study, short-term outcomes following PCI were similar to the non-RA group. Noncardiac mortality at 30 days was higher in unadjusted analysis, but this relationship was no longer present after adjustment. Conversely, the risk of long-term mortality was 53% higher in the adjusted model. Previous data on revascularization outcomes are generally limited to smaller studies, many predating widespread use of drug-eluting stent, with variable adjustment for confounders, and overall conflicting results. Previous reports have noted worsened short- and long-term outcomes following PCI (although without adjustment for cardiogenic shock or cardiac arrest),[9] worsened long-, but not short-term outcomes following PCI,[5,10] no difference in short- or long-term outcomes following CABG,[11] and improved short-term outcomes.[12,25] The magnitude of long-term mortality risk identified in our study is similar to previous estimates in 3 smaller studies from Taiwan (525 patients, HR 1.55, 95% CI 1.35 to 1.79, median follow-up ~4 years),[9] Switzerland (197 patients, HR 2.05, 95% CI 1.14 to 3.70, follow-up 2 years),[5] and the United States (77 patients, HR 1.47, 95% CI 1.04 to 2.08, median follow-up 2.6 years).[10] The largest study assessing 69,354 patients with RA paradoxically demonstrated lower risk of in-hospital mortality, which the authors attributed to possible residual confounding despite adjustment (long-term mortality was not assessed).[25] Readmission rates within 30 days of PCI were higher among patients with RA in our cohort, driven by "other" reasons for readmission—a similar finding to higher readmission rates observed in RA populations following heart failure diagnosis (also driven by noncardiac reasons).[26]

Increased cardiovascular risk and worsened long-term outcomes among patients with RA are concerning. Optimal care should be multidisciplinary, involving both cardiologists and rheumatologists, to target the mechanisms responsible for the increase in risk—specifically by modifying classic cardiovascular risk factors, improving RA disease control (and thus reducing systemic inflammation), and avoiding medications that can lead to adverse outcomes (glucocorticoids and NSAIDs). In recent years, the idea of the "Heart Team" has become widely accepted to facilitate the process of patient-centered evidence-based decisions for coronary revascularization and disease management.[27]

For patients with RA, the inclusion of a rheumatologist in this team when appropriate is likely to be beneficial.

The lower rates of ACEI/ARB use and the trend to lower statin use identified in our study are consistent with previous data.[28] ACEI/ARB use (while perhaps partially explained by slightly higher renal impairment rates in our study) has been associated with lower risk of subsequent MI among patients with RA.[28] Similarly, the signal toward lower statin use in patients with RA is of concern. Patients with RA have lower baseline cholesterol levels compared with controls, a well-described phenomenon whereby lipid levels are inversely associated with inflammation.[29] Paradoxically, patients with RA with lower lipid levels have higher CVD risk, and statins have a similar impact on reducing cardiovascular events.[30] Clinicians should carefully consider the value of these medications when determining medication regimens following PCI in patients with RA.

The major limitation of this study is the lack of data regarding RA disease activity, duration and treatments, which are known to affect cardiovascular risk and mortality. Second, RA disease prevalence and cardiovascular risk varies considerably by country and presented data may not necessarily be generalizable to populations outside of Australia. Outcomes beyond 30 days are limited to all-cause mortality, with data relating to longer term cardiac-specific outcomes not available in the dataset. Finally, while we aimed to include a broad array of potential confounders in the multivariable models, residual confounding by variables not available in the MIG dataset cannot be excluded.

In conclusion, the present study highlights important differences in demographics, coronary lesion complexity, and presentation among patients with RA treated with PCI compared with patients without RA. Overall, short-term outcomes are similar. Risk of long-term mortality is significantly higher, especially among patients with RA presenting with ACS or with lower BMI.

Author Contribution

Luke P Dawson: Conceptualization, Methodology, Validation, Formal analysis, Writing - original draft; Diem Dinh: Methodology, Validation, Formal analysis, Writing - review & editing; Jessica O'Brien: Conceptualization, Methodology, Writing - review & editing; Stephen J. Duffy: Conceptualization, Methodology, Writing - review & editing; Emma Guymer: Methodology, Writing - review & editing; Angela Brennan: Data curation, Resources, Methodology, Validation, Writing - review & editing; David Clark: Methodology, Writing - review & editing; Ernesto Oqueli: Methodology, Writing - review & editing; Chin Hiew: Methodology, Writing - review & editing; Melanie Freeman: Methodology, Writing - review & editing; Christopher M. Reid: Methodology, Writing - review & editing; Andrew E. Ajani: Supervision, Methodology, Writing - review & editing.

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.2020.10.048.

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