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Comparison of Symptoms, Treatment and Outcomes of Coronary Artery Disease among Rheumatoid Arthritis and Matched Subjects Undergoing Percutaneous Coronary Intervention

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Abstract

Objective—Rheumatoid arthritis (RA) is associated with an increased prevalence of coronary artery disease (CAD). We investigated the presenting symptoms of CAD, coronary anatomy (single vs. multivessel CAD), and treatment among a group of subjects undergoing percutaneous coronary intervention (PCI) with angioplasty and/or stenting.

Methods—We evaluated a retrospective cohort of 43 RA subjects and 43 matched non-RA subjects undergoing PCI at 2 academic referral centers. RA subjects were matched to non-RA subjects on age, gender, history of coronary artery bypass grafting (CABG), date of PCI and Interventional Cardiologist. We compared cardiac risk factors, presentation, treatment and outcomes.

Results—The mean age of the study cohort was 71 ± 10 years, and the distribution of traditional cardiac risk factors was similar in the subjects with RA compared to the matched non-RA subjects (all *P* values > 0.05). Seventy-four percent of subjects with RA compared to 67% of those without RA presented with an acute coronary syndrome prior to PCI ($P = 0.48$). All subjects in this cohort undergoing PCI had at least one stenosis in a major epicardial vessel and similar percentages of subjects with RA (44%) and without RA (40%) had multivessel CAD ($P = 0.66$). The administration of cardiac medications both at PCI and at hospital discharge was not different among subjects with RA compared to matched non-RA subjects.

Conclusions—Among this cohort with significant CAD undergoing PCI, clinical characteristics, presentation, severity of CAD, treatment modalities and outcomes were similar in subjects with RA and well-matched non-RA subjects.

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Introduction

Patients with Rheumatoid Arthritis (RA) have a higher prevalence of coronary artery disease (CAD) and increased likelihood of experiencing a cardiovascular event.[1] A higher incidence of CAD exists in the few years prior to the diagnosis of RA with RA patients less likely to report angina.[2] The relative risk of a myocardial infarction is three-fold higher among women with RA compared to controls.[3] There is an increased incidence of cardiovascular events (cardiovascular related hospitalizations, procedures or deaths) in patients with RA (3.43 per 100 patient-years) compared to patients without RA (0.59 per 100 patient-years) and cardiovascular disease mortality is increased by about 50% in RA patients compared to the general population.[4] [5] Healthcare costs associated with RA and concomitant CVD exceed $14,000 annually. [6]

Despite these data, information regarding associations between the extent of CAD in patients with and without RA is generally lacking. An autopsy study of RA patients demonstrated increased inflammation in coronary artery walls and increased frequency of vulnerable plaques, but overall less severe CAD compared to controls.[7] In contrast, a case control study of RA patients with new onset CAD demonstrated more multi-vessel disease at coronary angiography and a trend towards more cardiovascular death.[8] A separate cohort study also found a higher prevalence of three-vessel CAD and coronary revascularization among RA patients compared to controls.[9] In addition, there are likely other factors beyond coronary obstruction that can lead to adverse cardiac outcomes, such as elevations in the c-reactive protein and coronary spasm.[10] [11]

Previous studies are limited by: a lack of information on some important covariates (family history of CAD, smoking, age); differences in control groups utilized for comparison; and the remote study periods (1950s- early 1990s). In addition, there is a paucity of data in patients with established cardiovascular disease and RA who undergo percutaneous coronary intervention (PCI). Given the significant advances in the diagnosis, management and treatment of both CAD and RA over the past 20 years, it is important to examine a population of patients undergoing PCI and to assess for differences in clinical presentation or care that may exist among RA subjects and non-RA subjects.

With this in mind, we investigated a cohort of RA and non-RA subjects undergoing PCI with angioplasty and/or stenting. We assessed the following features: the distribution of traditional cardiovascular risk factors, the presenting symptoms prompting PCI, the prevalence of single versus multi-vessel CAD, the use of cardiac medications, the utilization of drug-eluting versus bare metal stents, and short-term outcomes. In distinction to prior studies, we evaluated a subgroup of patients with established CAD and thus did not focus on rates or relative risks of CAD among RA compared with controls.

Methods

Study cohort

We utilized an electronic database comprising all diagnoses and procedure codes to identify subjects with RA at two major academic tertiary care referral hospitals. From 1990–2007, each hospital performed over 20,000 PCIs. All subjects from the time period of 1990–2007 with at least one diagnosis of RA (ICD: 714.0) and at least one visit to a Rheumatology clinic at either of the two hospitals and at least one diagnosis of removal of coronary obstruction (ICD: 36.0) were included. The diagnosis of RA was established through detailed medical record review and was based on the evaluation of a board-certified Rheumatologist. The diagnosis of removal of coronary obstruction (cardiac catheterization with angioplasty and/or stenting of at least one coronary artery) was corroborated with

interventional cardiology databases and medical record review of coronary catheterization reports to define subjects as having undergone PCI. Subjects were included only if they met the above criteria.

Subjects without RA were identified through the use of interventional cardiology databases at both hospitals. Matching was performed on age (± 5 years), gender, prior history of coronary artery bypass grafting (CABG), Interventional Cardiologist who performed the PCI and date of coronary catheterization (± 3 years unless procedure performed pre-2001). However, there were 4 subjects with RA who underwent PCI during 1998–2000, for whom matches during that time period could not be found. The matching parameters were widened to ± 6 years with an assumption that secular trends in PCI (angioplasty and/or stenting of coronary vessels) were not significantly different from 1998–2000 compared with 2001–2004.

Data collection

Information on covariates was obtained through detailed electronic medical record review and abstraction from interventional cardiology databases at both hospitals. Demographic information and medical history was collected. Data on cardiac risk factors, cardiology testing reports, cardiac medication use at the time of PCI or at hospital discharge was gathered. We assembled information on rheumatologic medications, laboratory parameters and radiographic data. Height (cm) and weight (kg) were recorded on the day of PCI for all subjects to calculate body mass index (BMI). Three subjects had this data collected from the most recent hospital visit.

Single vessel disease was defined as involvement of a major epicardial vessel with ≥ 70% stenosis (left anterior descending, left circumflex, right coronary artery, posterior descending artery, posterior left ventricular, diagonal, obtuse marginal, ramus) or ≥ 50% stenosis (left main). Since our cohort was limited to subjects who underwent PCI, all subjects in our sample had significant CAD with involvement of at least one major epicardial vessel. Multi vessel disease was classified as ≥ 2 major epicardial vessels.

The indications for PCI were characterized as subacute if the patient presented electively, asymptomatically or with stable angina. Acute coronary syndrome was defined as unstable angina, non-ST elevation myocardial infarction or ST-elevation myocardial infarction.

Ischemic electrocardiogram findings were considered positive if ST segment elevation or ST segment depression, T wave inversions, Q waves or new left bundle branch block was present and reported consistent with ischemia. Cardiac stress testing was considered positive based on Cardiologist report interpretation.

Cardiomyopathy was defined as a left ventricular ejection fraction of ≤ 45% either by echocardiography or by cardiac stress testing with nuclear imaging performed prior to the date of PCI or during hospital admission for PCI.

Complications occurring post-PCI were defined according to the American College of Cardiology National Cardiovascular Data Registry guidelines. [12] [13] Complications included, but were not limited to, retroperitoneal bleeding, gastrointestinal bleeding and myocardial infarction. Data was extracted on in-hospital death during admission for PCI and death after hospital discharge when this information was available through the electronic medical record.

Statistical Analysis

Means and standard deviations were calculated for continuous variables and RA subjects and matched non-RA subjects were compared using t-tests. Frequencies were calculated for categorical variables and RA subjects and matched non-RA subjects were compared using Chi-Square and Fisher's exact test when appropriate. Analysis was first performed among the RA and matched non-RA subjects using McNemar's chi-square test for matched pair analysis. All statistical analysis was performed using SAS (version 9.1, Cary, NC).

Results

The mean age of the cohort was 71 ± 10 years. About 90% (N=78) of the cohort was Caucasian and 56% (N=48) were female. Cardiovascular risk factors were equally distributed among those with and without RA. Subjects with RA had mean disease duration of 14 years and 42% (N=18) were seropositive. Sixty-four percent (N=16) had erosions, 79% (N=33) were on disease-modifying anti-rheumatic drugs (DMARDs), 29% (N=12) were on TNF α inhibitors or other biologics and 42% (N=18) were on corticosteroids at the time of PCI (Table 1).

In this cohort of subjects undergoing PCI with angioplasty and/or stenting of a coronary vessel, all subjects had significant CAD defined as involvement of a major epicardial vessel with ≥ 70% stenosis or ≥ 50% stenosis of the left main artery. The distribution of significant lesions was the same among subjects with and without RA (Table 2).

The symptoms leading to PCI exhibited striking similarity between the RA subjects (74%, N=32) and matched non-RA subjects (67%, N=29), with acute coronary syndrome prompting urgent PCI in the majority of subjects ($P = 0.48$). Twenty-five percent (N=11) of RA subjects presented subacutely (either asymptomatic or stable angina) versus 33% (N=14) among matched controls ($P = 0.48$). Ischemic changes on electrocardiogram (ECG) were seen in similar proportions of RA (52%, N=17) and non-RA (56%, N=19) subjects ($P = 0.72$). In those with available data (N=21), cardiac stress testing was positive for ischemia in the same frequency among RA subjects (73%, N=8) and matched non-RA subjects (80%, N=8) ($P > 0.99$). Data on ejection fraction, either measured by echocardiography or stress testing, was available in 50% of the cohort. Of these subjects, a trend towards higher prevalence of cardiomyopathy existed among RA subjects (48%, N=10) versus non-RA subjects (23%, N=5; $P = 0.09$).

At the time of PCI, the use of heparin or alternative agents (enoxaparin, argatroban and bivalirudin), glycoprotein IIb/IIIa inhibitors and clopidrogrel was consistent in the RA and non-RA cohort. The use of drug-eluting stents was alike in the RA (35%, N=15) and non-RA patients (44%, N=19; $P = 0.38$). During hospital discharge, 100% of subjects were prescribed aspirin, over 90% received statins and over 80% were administered beta blockers. There were slight differences in the use of angiotensin converting enzyme inhibitors (ACEIs) and angiotensin receptor blockers (ARBs) between the RA (88%, N=37) and matched non-RA cohort (70%, N=30; $P = 0.04$) (Table 2).

Hematocrit, blood urea nitrogen, serum creatinine, total cholesterol, low density lipoprotein, high density lipoprotein and creatinine phosphatase kinase were available in some of the subjects. RA and matched non-RA subjects did not have differences in these laboratory parameters (Table 3).

There were a total of five complications in the cohort of 86 subjects (5.8%), 3 among the RA subjects and 2 among the matched non-RA subjects. The complications experienced were three retroperitoneal bleeds (defined as requiring blood transfusion and corroboration by

abdominal CT scan), one post-procedure myocardial infarction (defined as elevation of CK-MB > 3x upper limit of normal) and one gastrointestinal bleed (defined as requiring blood transfusion, corroborated by colonoscopy demonstrating active bleeding and leading to longer hospital stay). The mean follow-up for the cohort was 3.8 ± 2 years (22% missing data). Four subjects died during admission for PCI: 1 with RA and 3 without RA.

Discussion

We studied a cohort of RA subjects and matched non-RA subjects undergoing PCI for established significant CAD, with a focus on symptoms, treatment and outcomes. The study design allowed us to focus on subjects with significant established CAD, thereby limiting our ability to comment on the rate or relative risk of CAD in RA versus non-RA subjects. All subjects presented with similar symptoms (subacute versus acute coronary syndrome) leading to PCI. Prior work has demonstrated a lower likelihood of angina symptoms in RA subjects.[2] However, our cohort represented those with established CAD undergoing PCI, a subgroup of RA patients with recognized CAD. Thus, it is difficult to compare the symptoms of our cohort with prior studies of RA subjects who did not have known CAD. [14]

Atherosclerosis and RA are characterized by inflammatory underpinnings. It is not clear whether inflammation from CAD precedes RA or whether RA serves as an inflammatory nidus for the development of CAD. In our cohort, we excluded 13 of our original 56 subjects with RA as their diagnosis of RA was made subsequent to the index date of PCI. These 13 of 56 cases (23%) had significant CAD warranting PCI with a mean time of 4 years (SD 1.9 years) to development of RA. This may be consistent with the finding of a higher incidence of hospitalized MI in the 2 years preceding the diagnosis of RA, adding to the concept of inflammation as a common denominator in the pathogenesis of both RA and CVD. [2]

All subjects in the cohort had significant CAD as this was a study of those who underwent PCI with either angioplasty and/or stenting; thus multi-vessel disease was distributed similarly. There was a trend toward an increased frequency of stenosis of the right coronary artery among RA subjects; the significance of this finding is unclear. Cardiomyopathy (ejection fraction ≤ 45 percent) was seen in 48% of RA subjects but only 29% of matched non-RA subjects (P=0.09). With 50% missing data for this variable, the importance of this finding is limited but consistent with prior data suggesting an increase in heart failure in patients with RA,[15] [16] while patients without clinical cardiovascular disease have been shown to have preserved systolic function. [17]

The use of cardiac medications was remarkably consistent between RA subjects and matched non-RA subjects. Cardiology guidelines for the management of cardiovascular disease care have been implemented successfully into clinical practice and thus our results reflect these treatment algorithms. [18] Over 90% of subjects received aspirin, clopidrogrel and statins at the time of hospital discharge. These rates of therapy for secondary prevention following PCI are consistent with previously reported registries of subjects undergoing PCI. [19] Although there was a statistically significant difference between the rates of ACEI and ARB usage among RA and non-RA subjects, the reason for this finding remains unclear.

The overall post-PCI complication rate was 5.8% with short term outcomes (inhospital death) the same between RA and non-RA subjects. The 2 academic referral centers studied had high-volume interventional cardiac catheterization laboratories. Although there was 22% missing data on mortality, there were no differences noted post-PCI between the two groups. The majority of our population underwent PCI for ACS, not just MI. There are data

that RA subjects with acute myocardial infarction have a higher case fatality and decreased survival time compared with controls. [20] [21]

Our study provides new information on the presentation, care and outcomes of RA and non-RA subjects undergoing PCI and differs from prior work. A histology study reviewed autopsy specimens from RA subjects with CAD and controls between 1985 and 2003. It found that multivessel CAD was less prevalent among RA subjects compared with controls (32% versus 61%, $P = 0.018$), although inflammation was increased. [7] Two previous studies have focused on multivessel CAD among patients with RA as demonstrated by angiography. A retrospective case-control analysis of new-onset CAD during the time period 1985–1998 comparing patients with RA and age- and sex-matched controls, observed that multivessel CAD was more likely among RA patients compared to matched controls (24 versus 17% for 3-vessel disease, $P = 0.002$). [8] Our study investigated a cohort of RA subjects with established significant CAD undergoing PCI and a well-matched group of non-RA subjects, reported more current trends reflective of the time period 1998 to 2000 and included information on family history of CAD and BMI, two relevant covariates.

A second retrospective analysis also reported an excess of multivessel CAD among RA subjects compared to age- and sex-matched controls who underwent coronary angiography. Some limitations were that controls were not matched on date of angiography, controls were less likely to have diabetes mellitus, family history of CAD and BMI were not reported and obstructive CAD was defined as > 50% obstruction in any coronary artery. [9] Our study examines only RA subjects with established significant CAD (≥ 70% stenosis of major epicardial vessel or ≥ 50% stenosis of the left main artery) undergoing PCI to a well-matched cohort of non-RA subjects with detailed information on traditional cardiovascular risk factors, symptoms, treatment and outcomes. Our data demonstrate that in this cohort of subjects undergoing PCI for established significant CAD, there is no difference in the distribution of traditional cardiovascular risk factors, treatment or outcomes.

Our study adds new information to the literature. Although many papers report on the epidemiology of cardiovascular disease and subclinical atherosclerosis measures in RA, an important area of exploration is symptoms, treatment and outcomes of subjects with established CAD who undergo PCI. We were able to define a control group which was well matched on several important factors, including age, gender, history of CABG, date of PCI and Interventional Cardiologist. This allows for a fair comparison of subjects who have established significant CAD undergoing PCI while focusing on whether RA is associated with differences in presentation, care or outcome. Since the average disease duration of RA in our sample was 14 ± 13 years and subjects underwent PCI between 1998 and 2007 with a mean age of 70 years, data from our cohort represent current trends in PCI for established CAD.

There are important limitations of this study which warrant consideration. First, due to the small sample size (N=86), it was possible that we did not have sufficient statistical power to detect differences in some of the outcomes examined. Second, by study design, we focused only on those RA subjects with established CAD undergoing PCI and did not examine the entire cohort of RA patients at our 2 institutions with CAD. Thus, we are unable to draw inferences about the severity of CAD among the entire cohort of RA subjects at our 2 institutions. Third, this is a cohort of RA subjects and matched non-RA subjects treated at 2 tertiary care referral centers, limiting the generalizability of our results. Fourth, this was a retrospective analysis based on detailed medical record review of available data; disease activity scores were not routinely collected in clinical practice and were not available for analysis. RA classification criteria were not applied for RA diagnosis and rather a Rheumatologist's assessment was used to determine whether a patient had RA. Thus,

information such as laboratory testing and radiology was based on the discretion of the treating physicians, leading to missing data on some relevant covariates. We did however conduct chart reviews to determine how frequently the 42 RA patients met 1987 ACR classification criteria and found that: 32/42 (76%) patients met the criteria and in the remaining 10 patients, 6 were on DMARD therapy, 2 patients were in clinical remission and 2 did not have sufficient information in the electronic health record from 1990 until present to determine whether they would have met the criteria. Fifth, the reviewer extracting information from the electronic medical record was not blinded to RA disease status. Sixth, our classification of CAD was based on cardiac catheterization reports of subjects undergoing PCI, not a review of actual angiograms. There may be some inter-reader variability in the degree of stenosis visualized and reported. Since all cases were interventional and not diagnostic, we did not have complete information on non-critical coronary artery stenosis as they were not uniformly measured or reported.

In summary, among a cohort of subjects with established CAD who underwent PCI with either angioplasty and/or coronary stenting, we found no major differences among RA and non-RA subjects with respect to cardiac risk factors, presenting symptoms prompting PCI, single versus multivessel disease, use of drug-eluting stents, medications at PCI or hospital discharge and short term outcomes. Although prior studies have evaluated cardiovascular risk factors, surrogate measures of CAD and extent of atherosclerosis, our study is novel in that we report on subjects with established CAD undergoing PCI. Future studies in larger cohorts of subjects with and without RA and with varying severity of CAD may be useful to examine these issues further.

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Table 1

Patient Characteristics

Variables	RA (N=43)	Non-RA (N=43)	p-value †
	N(%) or Mean ±SD		
Age	70.6 ± 10.0	71.5 ± 10.0	0.68
Race/ethnicity (Caucasian)	41 (95)	37 (86)	0.27
Gender (Female)	24 (56)	24 (56)	>0.99
Hypertension	38 (88)	39 (91)	>0.99
Hypercholesterolemia	35 (81)	33 (77)	0.60
Diabetes mellitus	16 (37)	17 (40)	0.82
Smoking status *	30 (71)	33 (79)	0.45
Family history of CAD	14 (38)	19 (46)	0.45
Body mass index	28.2 ± 4.4	28.8 ± 6.2	0.62
History of CABG	6 (14)	5 (12)	0.75
Duration of RA	14.3 ± 13.2	-	-
Seropositive (RF or CCP)	18 (42)	-	-
Erosions **	16 (64)	-	-
DMARD use	33 (79)	-	-
Corticosteroid use	18 (42)	-	-
TNF or other biologic use	12 (29)	-	-

Abbreviations: RA = rheumatoid arthritis, CAD = coronary artery disease, CABG = coronary artery bypass grafting, RF = rheumatoid factor, CCP = anti-cyclic citrullinated peptide antibody, DMARD = disease-modifying anti-rheumatic drug, TNF = tumor necrosis factor α inhibitors

* Smoking status defined as current or prior history of tobacco use

** Erosion data was missing in 50% of the RA cohort

† p-values from t-test for continuous variables and Chi-square and Fisher's exact test for categorical variables

Non-RA cohort matched on age (± 5 years), gender, hx of CABG, date of coronary intervention (± 3 years unless PCI performed pre-2000 ± 6 years) and Interventional Cardiologist

Table 2

Coronary Anatomy and Medications at Percutaneous Coronary Intervention and Hospital Discharge

	RA (N=43)	Non-RA (N=43)	P value *
Left main stenosis ≥ 50%	1 (2)	2 (5)	> 0.99
Left anterior descending stenosis ≥ 70%	28 (65)	27 (63)	0.82
Left circumflex stenosis ≥ 70%	16 (37)	17 (40)	0.82
Right coronary artery stenosis ≥ 70%	25 (58)	17 (40)	0.08
Bypass graft stenosis ≥ 70%	4 (9)	5 (12)	> 0.99
Single vessel disease	24 (56)	25 (58)	0.83
Multi vessel disease	19 (44)	17 (40)	0.66
Heparin or heparin-alternatives (enoxaparin, argatroban and bivalirudin) at PCI	40 (95)	38 (90)	0.68
Glycoprotein IIb/IIIa inhibitors at PCI	16 (38)	23 (53)	0.15
Aspirin at discharge	41 (100)	42 (100)	> 0.99
Clopidogrel or ticlopidine at discharge	39 (93)	41 (95)	0.68
Statin at discharge	38 (90)	39 (91)	> 0.99
Beta blocker at discharge	37 (88)	36 (84)	0.56
ACEI and/or ARB at discharge	37 (88)	30 (70)	0.04

Abbreviations: ACEI = angiotensin converting enzyme inhibitor, ARB = angiotensin receptor blocker

Multi vessel disease = ≥ 2 major epicardial vessels

* p-values from McNemar's Chi-square test

Table 3

Laboratory Parameters at the time of Percutaneous Coronary Intervention

	RA (n=43)	Non-RA (n=43)	P value
Hematocrit (mean admission or within 24 hrs of PCI) *	35.1 % ± 4.0	36.2 ± 4.5	0.21
Blood urea nitrogen (mg/dL mean) *	22.8 ± 11.0	21.5 ± 9.2	0.61
Serum creatinine (mg/dL mean admission or day of PCI)*	1.19 ± 0.58	1.14 ± 0.52	0.67
Total cholesterol (mg/dL)	154.0 ± 41.7	146.6 ± 32.5	0.41
LDL (mg/dL)	84. 0 ± 36.6	80.6 ± 30.9	0.67
HDL (mg/dL)	44.9 ± 14.7	41.4 ± 14.0	0.30
CPK (U/L median with min/max) (peak during admission for PCI)	145 (19, 4539)	162 (36, 3533)	0.30

* Missing data n (%): hematocrit 3(3), blood urea nitrogen 13 (15), serum creatinine 13(15)

All values are shown as mean ± SD except for CPK which is represented with median (min, max).