Gaps in Addressing Cardiovascular Risk in Rheumatoid Arthritis: Assessing Performance Using Cardiovascular Quality Indicators

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ABSTRACT. Objective. Cardiovascular disease (CVD) is a major comorbidity for patients with rheumatoid arthritis (RA). This study sought to determine the performance of 11 recently developed CVD quality indicators (QI) for RA in clinical practice.

Methods. Medical charts for patients with RA (early disease or biologic-treated) followed at 1 center were retrospectively reviewed. A systematic assessment of adherence to 11 QI over a 2-year period was completed. Performance on the QI was reported as a percentage pass rate.

Results. There were 170 charts reviewed (107 early disease and 63 biologic-treated). The most frequent CVD risk factors present at diagnosis (early disease) and biologic start (biologic-treated) included hypertension (26%), obesity (25%), smoking (21%), and dyslipidemia (15%). Performance on the CVD QI was highly variable. Areas of low performance (< 10% pass rates) included documentation of a formal CVD risk assessment, communication to the primary care physician (PCP) that patients with RA were at increased risk of CVD, body mass index documentation and counseling if overweight, communication to a PCP about an elevated blood pressure, and discussion of risks and benefits of antiinflammatories in patients at CVD risk. Rates of diabetes screening and lipid screening were 67% and 69%, respectively. The area of highest performance was observed for documentation of intent to taper corticosteroids (98%–100% for yrs 1 and 2, respectively).

Conclusion. Gaps in CVD risk management were found and highlight the need for quality improvements. Key targets for improvement include coordination of CVD care between rheumatology and primary care, and communication of increased CVD risk in RA. (J Rheumatol First Release August 1 2016; doi:10.3899/jrheum.160241)

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Cardiovascular disease (CVD), including myocardial infarction (MI) and stroke, is more common in patients with rheumatoid arthritis (RA) compared with the general population, with an estimated 48% increased risk of incident CVD[1]. The reasons for this are complex, ultimately reflecting the consequences of inflammation predisposing to endothelial dysfunction[2,3,4,5,6] and premature atherosclerosis[7,8,9]. Despite improvements in the treatment of the

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inflammatory burden in RA, there continues to be evidence of a widened "mortality gap"[10]. One potential contributor is the underidentification and undertreatment of traditional CVD risk factors in patients with RA, including smoking, hypertension (HTN), obesity, and dyslipidemia[11,12,13,14], treatment for which results in improved CVD outcomes in the general population.

To guide improvement in the quality of CVD care delivered to patients with RA, a set of 11 CVD quality indicators (QI) was developed to assess risk factor screening and management[15]. Our study reports on the adherence to the CVD QI for 2 cohorts of patients with RA followed at the University of Calgary Rheumatology Clinics: an early RA (ERA) cohort and a biologics-treated RA cohort. The objective was to determine QI performance over a 2-year period, while concurrently assessing the feasibility of identifying the QI from subspecialty medical records.

MATERIALS AND METHODS

Patient population. Two established patient cohorts were used in our study to represent a spectrum of both disease and treatment.

(1) ERA cohort: The cohort was established in 2004 for patients with inflammatory arthritis of recent onset (< 12 weeks)[16,17]. Standardized data were collected at baseline including demographic information (age, sex, ethnicity, duration of symptoms, comorbidities) and medication history. Disease activity, medications, and functional status were recorded at each clinic visit. Patients were typically followed for 1 year in this clinic and then discharged to a general rheumatology clinic for followup (none were transferred to the biologics cohort described below). Followup intervals and treatment decisions were at the discretion of the treating rheumatologist (n = 7).

(2) The Alberta Biologics Pharmacosurveillance Program (ABioPharm): This provincial registry was initiated in 2000 to evaluate the efficacy, safety, and cost-effectiveness of biologic therapies for patients with RA[18,19]. Patients were assessed at baseline initiation or switching of a biologic, 3 months after therapy initiation, and then yearly or more frequently if required. Data elements collected were identical to the ERA cohort. For our study, only data for Calgary patients (n = 10 rheumatologists) with a new therapy initiated during the study period were reviewed (because this was the point at which they were entered into the cohort).

Inclusion criteria. Charts were selected using a computer-generated random sample of half of the patients from each physician in each cohort and included if the patient met the 2010 criteria for RA[20], had no preexisting diagnosis of CVD including prior MI, stroke, or peripheral vascular disease, and was enrolled in one of the cohorts between January 1, 2010, and August 1, 2014. This start date was selected because it was after the publication of the international recommendations for CVD risk management for patients with RA[21], bringing heightened attention to the need for the management of this comorbidity.

Data sources and abstraction. Rheumatology charts (electronic and paper) of patients enrolled in either cohort were randomly selected for retrospective review. Three reviewers with medical training abstracted the data independently (1 rheumatologist, 1 cardiologist, and 1 medical student). Standardized data abstraction forms were developed to identify the data elements listed in the next section. The first clinical visit after January 1, 2010, was used as the baseline visit and records were reviewed for 2 years.

Sample size calculations. Because routine CVD screening was not a mandate for either of these clinics, a worst-case scenario of 50% ± 10% adherence to the QI was assumed when conducting sample-size calculations. Sample size adjustments were also made to account for physician practice variation using an ICC of 0.05[22], as well as the population sizes of eligible patients in the 2 cohorts (217 in ERA and 148 in the biologics cohort). Based on these calculations, a minimum of 59 biologic charts and 98 ERA would need to be included. To ensure that performance rates were estimated with the desired precision, some additional charts were included. Further estimation of CI was not done for QI performance rates because the sample size ensures a precision ± 10% or narrower.

Clinical variables. Comorbidities including HTN, diabetes, and dyslipidemia were deemed present at baseline if there was a documented history in the medical record or the patient was receiving treatment for the condition. Obesity was defined as a body mass index (BMI) ≥ 30 kg/m^2 and overweight as a BMI 25–29.99 kg/m^2. HTN was defined as ≥ 140/90 mmHg and patients with a lower recommended threshold because of diabetes or chronic kidney disease were excluded from the denominator of the HTN QI because other published quality measures better applied to these populations. Medications were identified from each clinic visit.

Cardiac risk assessment. A Stata module (Framingham)[23] was used to calculate a baseline 10-year Framingham Risk Score (FRS)[24] in eligible patients (ages 30–74 yrs and not receiving a statin at baseline). The FRS was chosen because it is recommended by the Canadian Cardiovascular Society guidelines[25]. Lipid values closest to the baseline visit were used in FRS calculation within a window of up to 6 months prior to the baseline visit or 1-year post-visit, which is more stringent than previous retrospective applications of the score[26]. Patients for whom a 10-year FRS score could be calculated were classified into the following levels of risk: < 10% (low risk), 10%–19% (intermediate risk), and ≥ 20% (high risk)[25].

Disease activity was calculated using the 28-joint Disease Activity Score (DAS28) with erythrocyte sedimentation rate[27,28], and patients were classified according to disease activity score: remission (≤ 2.6), low disease activity (2.7 to ≤ 3.2), moderate disease activity (3.3 to ≤ 5.1), and high disease activity (> 5.1). The Health Assessment Questionnaire measured baseline functional status[29].

Analysis. Descriptive statistics were used to summarize baseline clinical features and CV comorbidities using proportions, means and SD, or medians (interquartile range), depending on data normality. The chi-square test or Fisher's exact test and the Student t tests or Wilcoxon-Mann-Whitney tests as appropriate for the data were used to investigate baseline characteristics for patients where an FRS could be computed compared with those in whom it could not.

Adherence to each QI was reported as a percentage and calculated based on the predefined criteria for the numerator, denominator, and for exclusion[15] (case record form available upon request). Patients were not eligible for inclusion in the denominator of measures reported at Year 2 if the patient was lost to followup, died, was followed for less than 2 years based on their baseline date of study entry, or had a CVD event during the course of followup.

Interrater reliability of extraction of data was assessed on 48 randomly selected charts reviewed in duplicate. The percent agreement and Cohen κ[30] were calculated for each QI. K scores were interpreted according to suggested guidelines[31]: almost perfect agreement (0.81–0.99), substantial agreement (0.61–0.80), moderate agreement (0.41–0.60), fair agreement (0.21–0.40), and slight agreement (0.01–0.20). Where disagreement occurred, the chart was re-reviewed by an expert reviewer (a rheumatologist who helped develop the QI) to determine whether the QI was met. Stata IC version 13.1 (StataCorp) was used for all analyses.

Ethics approval. The University of Calgary Health Research Ethics Board approved this project (REB13-1314). All patients consented to inclusion in the cohorts. The study was deemed exempt by our ethics board from obtaining additional consent from patients for this specific study.

RESULTS

Baseline characteristics. Demographic and clinical characteristics of the included patients are shown in Table 1. There were 170 patients included (63 in the biologics cohort and

Table 1. Baseline clinical characteristics for patients in the biologics and ERA cohorts included in the quality indicator review. Values are n (%) unless otherwise specified.

Characteristics	Overall, n = 170*	Biologics, n = 63*	ERA, n = 107*
Age, yrs, mean (SD), range	54.6 (13.6), 22–89	57.5 (15.1), 25–89	52.9 (12.4), 22–89
Female	119 (70)	51 (81)	68 (64)
Ethnicity	82 (48) white, 69 (41) not stated	42 (67) white, 13 (21) not stated	40 (37) white, 56 (52) not stated
Disease duration since diagnosis at baseline visit, yrs	Could not be calculated because N/A for ERA	Median 5.5, IQR 2–15, n = 62	N/A
Duration between symptom onset and diagnosis, mos	Median 6.1, IQR 3.3–12.2, n = 159	Median 6.7, IQR 3.0–21.8, n = 52	Median 6.1, IQR 3.5–11.6, n = 107
Duration of followup, days**	Median 607, IQR 482–678	Median 616, IQR 524–685	Median 606, IQR 472–674
RF-positive	125 (74)	45 (71)	80 (75)
Anti-CCP–positive	139 (82), n = 166	47 (75), n = 61	92 (86), n = 105
Nodules	20 (12)	14 (22)	6 (6)
Erosions on baseline radiographs	59 (35), n = 165	40 (63), n = 62	19 (18), n = 103
Extraarticular RA manifestations***	11 (7)	8 (13)	3 (3)
Baseline HAQ score, mean (SD)	1.35 (0.7), n = 165	1.7 (0.6), n = 63	1.13 (0.7), n = 102
Baseline DAS28, mean (SD)	5.46 (1.37), n = 156	5.76 (1.04), n = 59	5.28 (1.51), n = 97
Moderate disease activity, DAS28 > 3.2 to ≤ 5.1	49 (35)	17 (29)	32 (33)
High disease activity, DAS28 > 5.1	97 (62)	42 (71)	55 (57)
Baseline RA treatment at end of the first visit			
Any baseline DMARD	153 (90)	53 (84)	100 (93)
Plaquenil	94 (55)	21 (33)	73 (68)
MTX	124 (73)	37 (59)	87 (81)
Leflunomide	10 (6)	10 (16)	0
Sulfasalazine	12 (7)	8 (13)	4 (13)
Any combination DMARD therapy	80 (47)	18 (29)	62 (58)
MTX + plaquenil	66 (39)	8 (13)	58 (54)
Biologics	62 (37)	62 (98)	0
NSAID, other than ASA at first visit	64 (38)	30 (48)	34 (32)
Prednisone	37 (22)	21 (33)	16 (15)
Intramuscular GC	62 (37)	7 (11)	55 (51)
Intraarticular GC	12 (7)	3 (5)	9 (8)
No. followups over study and disease activity at end of followup			
No. followup visits over 2 yrs, mean (SD)	6.1 (1.9)	5.9 (1.8)	6.3 (2.0)
DAS28 at end of followup, mean (SD)	2.3 (1.2), n = 108	2.9 (1.3), n = 38	2.1 (1.1), n = 70
Remission or low disease activity, DAS ≤ 3.2	82 (76)	23 (61)	59 (84)
CVD events over the course of followup	2 patients (2 MI, 1 aneurysm repair)	0	2 (2 MI, 1 aneurysm repair)

* Unless otherwise specified. ** Duration of followup between baseline visit and last followup date within 2 years (in ERA clinic, calculated from date of diagnosis of RA, and in biologics from biologics clinic date where a switch or new start to a biologic was made). *** Other extraarticular manifestations included interstitial lung disease, pleural disease, and RA vasculitis (rheumatoid nodules not counted here). ERA: early rheumatoid arthritis; RF: rheumatoid factor; anti-CCP: anticyclic citrullinated peptide antibodies; RA: rheumatoid arthritis; HAQ: Health Assessment Questionnaire; DAS28: 28-joint Disease Activity Score; DMARD: disease-modifying antirheumatic drug; MTX: methotrexate; NSAID: nonsteroidal antiinflammatory drugs; ASA: acetylsalicylic acid; GC: glucocorticoids; CVD: cardiovascular disease; N/A: not applicable; IQR: interquartile range; MI: myocardial infarction.

107 in the ERA cohort), with a mean age of 55 years, and 70% were women. The median total available followup between the baseline date and last clinic visit within the 1-year window of evaluation was 607 days and the median number of visits was 6.

The patients with RA from the biologics cohort had higher disease activity, worse functional status, more erosions, and extraarticular manifestations compared with the ERA cohort (Table 1). Baseline treatments are shown in Table 1. Thirty-eight percent of patients were receiving nonsteroidal antiinflammatory drugs (NSAID), while 22% received oral prednisone. Over the course of followup, the disease activity declined to a mean DAS28 of 2.3 ± 1.2, and 76% of patients were in clinical remission.

There were 7 patients lost to followup, 2 could not be assessed for 2-year outcomes based on their baseline date of entry into the cohorts, and 2 had CV events. For these reasons, 11 patients in total were not included in the denominators of Year 2 QI reporting or in QI where measurement occurred over a 2-year period.

CV risk profile. The CV risk profile of patients is shown in Table 2. A quarter of patients had a history of HTN (26%), and 90% these patients were receiving treatment. A further 14% had elevated blood pressures (≥ 140/90) at their first

Table 2. CV risk profile and treatment characteristics of patients with rheumatoid arthritis in the biologics and ERA cohorts. Values are n (%) unless otherwise specified.

Characteristics	Overall, n = 170	Biologics, n = 63	ERA, n = 107
HTN	44 (26)	23 (37)	21 (20)
Blood pressure ≥ 140/90 at first visit and no prior diagnosis of HTN	23 (14)	4 (6)	19 (18)
Diabetes	11 (7)	4 (6)	7 (7)
Dyslipidemia	26 (15)	10 (16)	16 (15)
Obesity, BMI ≥ 30	43 (25)	14 (22)	29 (27)
BMI, mean (SD)	27.3 (4.9)	26.7 (5.1)	27.5 (4.9)
Family history of CVD	4 (2)	1 (2)	3 (3)
Missing information on family history	58 (34)	41 (65)	17 (16)
Current smoker	35 (21)	8 (13)	27 (25)
Ex-smoker	56 (33)	26 (41)	30 (28)
Non-smoker	69 (41)	25 (40)	44 (41)
Missing smoking data	10 (6)	4 (6)	6 (6)
Baseline FRS, n = 59 FRS calculated*			
FRS low	39 (66)	6 (75)	33 (65)
FRS intermediate	13 (22)	2 (25)	11 (22)
FRS high	7 (11)	0 (0)	7 (14)
FRS could not be calculated, but patients were eligible for FRS estimation, n = 134			
FRS could not be calculated	75 (56)	39 (83)	36 (41)
Baseline CV risk factor treatment at end of first visit			
Aspirin	19 (11)	9 (14)	10 (10)
Statin**	19 (11), n = 168	7 (11), n = 62	12 (11), n = 12
Antihypertensive agents, any	40 (24)	21 (33)	19 (18)

* Baseline FRS was calculated with baseline variables at first visit using lipid values from up to 6 months prior to first visit and 1 year after. ** Two patients had major missing and/or conflicting information in the chart and statin use and dyslipidemia history could not be determined. CV: cardiovascular; ERA: early rheumatoid arthritis; HTN: hypertension; BMI: body mass index; CVD: CV disease; FRS: Framingham Risk Score.

visit, but had no history of HTN. A quarter of patients were obese. Eleven patients had diabetes (7%) and 26 (15%) had dyslipidemia. Twenty-one percent of patients were current smokers. A baseline FRS could be calculated for only 44% of eligible patients (n = 59) because the remainder were missing documentation of key variables required for the calculation of the score. Where an FRS could be calculated, 34% were at intermediate or high risk of CVD events. Clinical characteristics including age, sex, number of visits, and baseline CV comorbidities were compared between patients for whom an FRS could be calculated with those who did not have enough information to calculate the score, and there were no statistically significant differences (data not shown).

QI reporting. The results of the QI reporting are shown in Table 3. The lowest performance rates were on documentation of a formal CV risk assessment (QI #2), which was not present on any of the charts during the period of review. Low performance rates (2%) were also observed for communication to the primary care physician (PCP) that RA was associated with an increased risk of CVD disease (QI #1).

High performance rates (94%) were noted with baseline documentation of smoking status (QI #3A); however, by Year 2, re-documentation of smoking status in known smokers was lower (42%). Documentation of smoking cessation advice to current smokers (QI #3B) was low in both measurement years (17% and 24% in Yr 1 and Yr 2, respectively).

QI #4 assessed whether a blood pressure had been measured at 80% or more of clinic visits during each measurement period. For this QI, performance rates for Year 1 and Year 2 were 58% and 66%, respectively. However, when an elevated blood pressure was recorded, recommendations for addressing this were rarely sent back to the PCP (5% and 7% in Yr 1 and Yr 2, respectively; QI #5).

Lipid (QI #6) and glucose screening (QI #7A) were measured over the 2-year period of followup in 69% and 67% of charts, respectively. However, QI #7B, which identified whether there had been yearly measurement of a fasting glucose or a hemoglobin A1C in individuals with risk factors for diabetes as defined by specific criteria published in the original QI specifications and outlined in Table 3, was slightly lower (54% in Yr 1 and 48% in Yr 2).

Yearly discussion of physical activity recommendations (QI #8) had poor performance in Year 1 (33%) that declined in Year 2 (15%). Similarly, BMI screening rates were low (QI #9A; 4–6%). Although the rates of BMI calculation were low, height and weight were available for the majority of patients and were used to estimate the denominator for QI #9B, which identifies lifestyle counseling to overweight and obese patients. Performance on this part of the indicator was also low (5%–9%).

Table 3. Adherence to 11 CV quality indicators in 2 cohorts.* Values are n (%).

Quality Indicator	Total		Biologics		ERA	
1. Communication of increased CV risk in RA: IF a patient has RA, THEN the treating rheumatologist should communicate to the PCP, at least once within the last 2 yrs that patients with RA have an increased CV risk.	3/158 (2)		0/62 (0)		3/96 (3)	
2A. CV risk assessment: IF a patient has RA, THEN a formal CV risk assessment according to national guidelines should be done at least once in the first 2 yrs after evaluation by a rheumatologist.	0/150 (0)		0/58 (0)		0/92 (0)	
2B. IF initial assessment suggests intermediate or high risk, THEN treatment of risk factors according to national guidelines should be recommended.	There were no patients with a risk assessment, therefore both the numerator and denominator for this QI was 0.					
	Y1	Y2	Y1	Y2	Y1	Y2
3A. Smoking status and cessation counseling: IF a patient has RA, THEN their smoking and tobacco use status should be documented at least once in the last yr.	160/170 (94)	16/38** (42)	59/63 (94)	5/10** (50)	101/107 (94)	11/28** (39)
3B. IF they are current smokers or tobacco users, THEN they should be counseled to stop smoking.	6/35 (17)	4/17 (24)	3/8 (38)	1/4 (25)	3/27 (11)	3/13 (23)
4. Screening for HTN: IF a patient has RA, THEN their blood pressure should be measured and documented in the medical record at ≥ 80% of clinic visits.	98/170 (58)	105/159 (66)	21/63 (33)	45/62 (73)	77/107 (72)	60/97 (62)
5. Communication to PCP about a documented high blood pressure: IF a patient has RA AND has a blood pressure measured during a rheumatology clinic visit that is elevated (systolic blood pressure ≥ 140 mmHg and/or diastolic blood pressure ≥ 90 mmHg), THEN the rheumatologist should recommend that it be repeated and treatment initiated or adjusted if indicated.	5/76 (7)	3/59 (5)	3/24 (4)	1/19 (5)	2/52 (4)	2/40 (5)
6. Measurement of a lipid profile: IF a patient has RA, THEN a lipid profile should be done at least once in the first 2 yrs after evaluation by a rheumatologist.	110/159 (69)		37/62 (59)		73/97 (75)	
7A. Screening for diabetes: IF a patient has RA, THEN diabetes should be screened for as part of a CV risk assessment at least once within the first 2 yrs of evaluation by a rheumatologist.†	100/149 (67)		34/58 (59)		66/91 (73)	
	Y1	Y2	Y1	Y2	Y1	Y2
7B. Yearly in intermediate or high risk patients.‡	72/132 (54)	57/119 (48)	21/50 (42)	20/44 (45)	51/82 (62)	37/75 (49)
8. Exercise: IF a patient has RA, THEN physical activity goals should be discussed with their rheumatologist at least once yearly.	55/168 (33)	24/158 (15)	12/63 (19)	12/62 (19)	43/105 (41)	12/96 (13)
9A. BMI screening and lifestyle counseling: IF a patient has RA, THEN their BMI should be documented at least once every yr.	11/170 (6)	6/159 (4)	6/63 (10)	6/62 (10)	5/107 (5)	0/97 (0)
9B. IF patient is overweight or obese according to national guidelines, THEN they should be counseled to modify their lifestyle.	10/111 (9)	5/103 (5)	1/40 (3)	2/39 (5)	9/71 (13)	3/64 (5)
10. Minimizing corticosteroid usage: IF a patient with RA is receiving oral corticosteroids, THEN there should be evidence of intent to taper off the corticosteroids or reduce to the lowest possible dose.	56/57 (98)	28/28 (100)	25/26 (96)	18/18 (100)	31/31 (100)	10/10 (100)
11. Communication about risks/benefits of antiinflammatories in patients at high risk of CV events: IF a patient has RA AND has established CVD OR is at intermediate or high CV risk AND is receiving an NSAID (or COX-2 inhibitor), THEN a discussion about the potential CV risks should occur and be documented.	2/23 (9)	0/17 (0)	0/4 (0)	0/6 (0)	2/19 (11)	0/11 (0)

* QI are reported either over a 1- or 2-year measurement basis (as indicated). The denominators vary for each indicator as shown depending on the eligibility criteria for each denominator criterion as published in Gabriel and Crowson[9] and rationale for exclusion from the denominators is available upon request. Overall, there were 11 patients who were not eligible for inclusion in any of the denominators for indicators in Year 2 because of lack of followup or new incident CVD after Year 1. ** Denominator for this indicator in Year 2 does not include patients who were documented to be nonsmokers in Year 1. † The final part of this indicator "AND if screening is abnormal, this information should be communicated to the PCP for appropriate followup and management if indicated" was not reported on because of very small sample sizes in the denominator. ‡ Patients at high or intermediate risk for diabetes include patients with the following risk factors: family history of type 2 diabetes in a first-degree relative, history of metabolic syndrome, obesity or overweight (BMI ≥ 25 kg/m^2), steroid use, history of gestational diabetes or a macrosomic infant, history of impaired fasting glucose (≥ 6.1 mmol/l) or HbA1C ≥ 6.0%, history of HTN (blood pressure ≥ 140/90 mmHg), member of a high-risk population (e.g., Aboriginal, Asian, Hispanic, South Asian, African, Pacific Islanders), or high risk based on validated diabetes risk calculators or high or intermediate CV risk based on CV risk calculators (e.g., Framingham Risk Score). CV: cardiovascular; ERA: early rheumatoid arthritis; RA: rheumatoid arthritis; PCP: primary care physician; HTN: hypertension; BMI: body mass index; CVD: CV disease; NSAID: nonsteroidal antiinflammatory drugs; Y1: Year 1; Y2: Year 2; QI: quality indicator; COX-2: cyclooxygenase-2.

Finally, with regard to the 2 treatment QI, performance on QI #10 (minimizing corticosteroid usage) was high (98%–100%, depending on the measurement year). In contrast, communication regarding risks of NSAID in patients at intermediate or high risk of CVD events was low, although the denominator was small because many patients were excluded owing to an uncertain level of risk, or the patients were not clearly receiving an NSAID during the measurement year.

While the mandate of our study was not to compare the QI performance rates between cohorts, the majority of performance rates were similar between the cohorts with 2 exceptions. The rate of blood pressure screening was significantly lower in Year 1 in the biologics cohort compared with the ERA cohort (33% vs 72%, $p < 0.001$) and the rates of exercise counseling were higher in the ERA cohort in Year 1 compared with the biologics cohort (41% vs 19%, $p = 0.003$).

Interrater reliability of the QI. Interrater reliability was calculated using Cohen κ for each QI in 48 charts (Table 4). Overall, there was moderate to perfect agreement in 11 out of 13 measures where a κ score could be calculated (85%) with some variation in scores by year noted. Because of the known properties of κ, whereby QI with a high or low performance can be accompanied by a high agreement but a low κ score[32], 2 κ scores were misleading (QI #1 and QI #2B; Table 4).

DISCUSSION

Our study systematically addresses performance on recently developed CVD QI for RA[15]. Consistent with other reports[13], our patients have a high burden of CV risk factors and would benefit from improvements in the screening and management of CVD risk to address the performance gaps identified.

There is substantial debate in the rheumatology community about the role rheumatologists should play in evaluating and treating CVD risk factors[33]. Proponents of the rheumatologists taking a role in management of CVD risk cite deficits in primary care screening management of CVD risk factors in RA[12,34,35,36]. Indeed, prospective and systematic evaluation of patients with RA in a number of studies has uncovered previously unidentified and untreated risk factors including HTN, dyslipidemia, and hyperglycemia[13,14]. Conversely, PCP are expert in CVD screening in the general population and some argue that screening should occur in primary care. Although, to accomplish better primary care screening for CVD risk in RA, primary care education and improved coordination of care with the rheumatologist are likely necessary to achieve optimal processes and outcomes[33,37].

Unfortunately, a major critique of systematic assessment for CVD risk factors is that it can be time consuming[14]. The CVD QI evaluated in our study were developed and worded to enhance CVD care in RA without placing the entire burden of care on the rheumatologist[15]. For example, many of the QI are framed in such a way that identifies whether another physician, e.g., the PCP, was reminded to monitor the patients' CV risk factors, a situation that shares the burden of care and removes it somewhat from the rheumatologist, who may be less familiar with CVD treatment guidelines. Unfortunately, many of the communication QI had poor adherence. Also, CVD preventive care such as smoking cessation advice, exercise review, and lifestyle counseling may occur, but be poorly documented. Failure to document such discussions is an opportunity for quality improvement not only in CVD risk screening, but also in coordination of care.

Although the main objective of our study was not to compare the performance of the QI between the cohorts, 2 significant differences were observed. Performance on QI #4 (blood pressure measurement) was higher in the ERA cohort compared with the biologics cohort and this may reflect differences in who was collecting baseline data in the cohort charts (i.e., there were more nursing assessments in the ABioPharm clinics where the biologics patients were seen and it is possible that routine blood pressure estimation was done less frequently for these visits than a typical rheumatologist visit). Exercise (QI #8) was more frequently discussed in the ERA cohort in Year 1 and this likely reflected the practice of referring new patients for a physiotherapy assessment and exercise counseling in this cohort.

Our study demonstrated that although CV risk estimation was not documented in our study (QI #2A), 69% of the time there was a lipid profile done within 2 years of baseline (QI #6) and often there was available information for the calculation of a risk score, a level substantially higher than in other reports[11]. Also of note for QI #9A, although BMI was actually calculated on very few charts, both height and weight were identified on all charts at baseline, but this did not meet criteria for the QI numerator. Failure to document BMI and CVD risk assessments may be lost opportunities for identification of risk and further work will need to be done to examine barriers to risk estimation and CVD screening in the rheumatology clinic. Alternatively, an evaluation of primary care practices of CVD risk estimation and treatment in RA could be done to better understand this suspected gap in care because our study did not have access to primary care records and it is unclear to what degree risk assessment and treatment is occurring at this level.

Limitations of our study are recognized. First, although the QI could be measured using information extracted from patient charts, it should be highlighted that the process was time consuming and resource intensive, and may not be a practical approach in routine practice. Second, our study included patients from research cohorts, and it is anticipated that QI adherence could be higher in this setting owing to more standardized delivery of care and/or collection of data. Additionally, in the original specification of some QI, the interval for evaluation was up to 5 years. Unfortunately, there

Table 4. Assessment of interrater reliability for each CV quality indicator in 48 randomly selected charts. The interrater reliability for 2 of 3 chart reviewers was reported for each QI by calculating a κ score. The interpretation of the score is as follows: below 0.0 = poor, 0.00–0.20 = slight agreement, 0.21–0.40 = fair agreement, 0.41–0.60 = moderate agreement, 0.61–0.80 = substantial agreement, and 0.81–0.99 = almost perfect agreement between reviewers. Values are n (%) unless otherwise specified.

Quality Indicator	κ		Percent Agreement, n = 48		Interpretation
1. Communication of increased CV risk in RA	0*		47 (98)		Falsely low because of low performance on the indicator
2A. CV risk assessment	1.0		48 (100)		Perfect agreement
2B. IF intermediate or high risk according to guidelines, THEN recommended that treatment of risk factors be initiated	0*		41 (85)		Falsely low due to low performance on the indicator
	Y1	Y2	Y1	Y2	
3A. Smoking status and cessation counseling	0.28	0.59	31 (65)	39 (81)	Fair agreement Y1 and moderate agreement Y2
3B. IF smoker counseled to quit smoking	0.88	0.57	46 (96)	44 (92)	Almost perfect agreement Y1 and moderate agreement Y2
4. Screening for HTN	0.91	0.50	46 (96)	38 (79)	Almost perfect agreement Y1 and moderate agreement Y2
5. Communication to PCP about a documented high blood pressure	0.70	0.70	41 (85)	41 (85)	Substantial agreement
6. Measurement of a lipid profile	0.91		46/48 (96)		Almost perfect agreement
7A. Screening for diabetes	0.84		44/48 (92)		Almost perfect agreement
	Y1	Y2	Y1	Y2	
7B. Yearly screening for diabetes in patients at intermediate or high risk**	0.75	0.61	40 (83)	38 (79)	Substantial agreement
8. Exercise	0.49	0.33	37 (77)	37 (77)	Moderate agreement Y1 and fair agreement Y2
9A. BMI screening and lifestyle counseling	0.85	0.63	47 (98)	44 (92)	Almost perfect agreement Y1 and substantial agreement Y2
9B. IF they are overweight or obese according to national guidelines, THEN they should be counseled to modify their lifestyle	0.71	0.75	41 (85)	42 (88)	Substantial agreement
10. Minimizing corticosteroid usage	0.79	0.57	43 (90)	40 (83)	Substantial agreement Y1 and moderate agreement Y2
11. Communication about risks/benefits of antiinflammatories in patients at high risk of CV events	0.51	0.69	42 (88)	45 (94)	Moderate agreement Y1 and substantial agreement Y2

* Because of the way κ scores are calculated, these indicators have a high agreement but a low κ score and are falsely 0. ** Patients at high or intermediate risk for diabetes include patients with the following risk factors: family history of type 2 diabetes in a first-degree relative, history of metabolic syndrome, obesity or overweight (BMI ≥ 25 kg/m^2), steroid use, history of gestational diabetes or a macrosomic infant, history of impaired fasting glucose (≥ 6.1 mmol/l) or HbA1C ≥ 6.0%, history of HTN (blood pressure ≥ 140/90 mmHg), member of a high-risk population (e.g., Aboriginal, Asian, Hispanic, South Asian, African, Pacific Islanders), or high risk based on validated diabetes risk calculators or high or intermediate CV risk based on CV risk calculators (e.g., Framingham Risk Score). CV: cardiovascular; QI: quality indicator; RA: rheumatoid arthritis; HTN: hypertension; PCP: primary care physician; BMI: body mass index; Y1: Year 1; Y2: Year 2.

was not enough followup available on the patients from the time when CV guidelines were first recommended for RA[21]. It should also be noted that the estimate of patients with dyslipidemia included individuals with a diagnosis of dyslipidemia, abnormal lipid tests, or those taking statin therapy. This estimate, therefore, may have included patients with diabetes being treated with statin therapies prophylactically, without a diagnosis of dyslipidemia. Given that the number of patients with concomitant diabetes and dyslipidemia was low (n = 6), this likely did not substantially affect the result. Finally, there was no control population of patients without RA given that the QI were specifically designed for this population, and it is possible that similar results could be found in patients with other chronic diseases.

Because this was the first time the QI have been studied, the interrater reliability was assessed; it is a key facet of QI testing. Although 85% of all QI had moderate to perfect agreement based on κ score, not all QI met this threshold. This did not substantially affect the final results because any disagreements between reviewers were verified in the chart to obtain the most accurate data identification.

During the assessment of interrater reliability, there were 3 types of potential challenges with applying the QI. The challenges and potential solutions are identified below:

(1) Issues with correctly determining eligibility for a QI. Accurate calculation of dates is required to ascertain whether a patient was eligible for a QI. Errors may also have occurred where criteria for denominator inclusion were complex. One

example is QI #11, where performance was based on 2 or more factors, i.e., where patients in the denominator had to be receiving NSAID and had to have intermediate or high risk for CVD during the period of evaluation. Electronic identification of the QI with automatic calculation and checking of dates and eligibility criteria would rectify this problem.

(2) Difficulty interpreting QI wording to ascertain inclusion in the numerator. This was most problematic for QI #8 (exercise). Although specific criteria for meeting this were listed in the case record form, there were different interpretations of eligibility by the reviewers. A modification to the inclusion criteria for the QI is therefore suggested, which requires provider documentation that the patient meets defined exercise targets. According to Canadian guidelines[38], this would be 150 min of moderate-intensity activity per week in bouts of ≥ 10 min.

(3) Complexity of chart review because of multiple sources of data. Some pieces of information, e.g., smoking status for biologic patients, were more reliably identified in the study chart than the clinical chart. This led to poor inter-rater reliability in some cases, depending on the thoroughness and/or interpretation of data for review in these multiple sources. Again, a single data source, perhaps an electronic medical record with better medication identification, would be helpful in documenting adherence to many of the QI.

Our study demonstrates that even in a highly controlled setting where standardized data collection is completed for reporting on clinical cohorts, there is room for improvement in CVD screening and care in RA. Gaps in CVD risk management were found and highlight the need for quality improvements. Key targets for improvement include coordination of CVD care between rheumatology and primary care, and communication of increased CVD risk in RA.

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