Patients with Rheumatoid Arthritis have Similar Excellent Outcomes after Total Knee Replacement Compared with Patients with Osteoarthritis

Susan M. Goodman, Beverly Johnson, Meng Zhang, Wei-Ti Huang, Rebecca Zhu, Mark Figgie, Michael Alexiades, and Lisa A. Mandl

ABSTRACT. ***Objective.*** Although new treatments for rheumatoid arthritis (RA) are extremely effective in preventing disease progression, rates of total knee replacement (TKR) continue to rise. The ongoing need for TKR is problematic, especially as functional outcomes in patients with RA have been reported to be worse than in patients with osteoarthritis (OA). The purpose of this study is to assess pain, function, and quality of life 2 years after TKR in contemporary patients with RA compared with patients with OA.

Methods. Primary TKR cases enrolled between May 1, 2007 and July 1, 2010 in a single institution TKR registry were eligible for this study. Validated RA cases were compared with OA at baseline and at 2 years.

Results. We identified 4456 eligible TKR, including 136 RA. Compared with OA, RA TKR had significantly worse preoperative Western Ontario and McMaster Universities Osteoarthritis Index pain (55.9 vs 46.6, $p < 0.0001$) and function (58.7 vs 47.3, $p < 0.0001$); however, there were no differences at 2 years. Within RA, there was no difference for patients who were treated with biologic disease-modifying antirheumatic drugs versus those who did not in pain ($p = 0.41$) or function ($p = 0.39$) at 2 years. In a multivariate regression, controlling for multiple potential confounders, there was no independent association of RA with 2-year pain ($p = 0.18$) or function ($p = 0.71$). Satisfaction was high for both RA and OA.

Conclusion. Patients with RA undergoing primary TKR have excellent 2-year outcomes, comparable with OA, in spite of worse preoperative pain and function. In this contemporary cohort, RA is not an independent risk factor for poor outcomes. (First Release December 1 2015; J Rheumatol 2016;43:46–53; doi:10.3899/jrheum.150525)

Key Indexing Terms:
RHEUMATOID ARTHRITIS
DISEASE-MODIFYING ANTIRHEUMATIC DRUGS
ARTHROPLASTY
OSTEOARTHRITIS

Rheumatoid arthritis (RA) is a form of inflammatory arthritis that can destroy cartilage and erode joints, leading to significant pain and functional impairment. Historically, over 50% of patients with RA have undergone orthopedic surgery, most commonly arthroplasty, over the course of their illness[1,2,3]. Although most reports describe significant pain relief for patients with RA undertaking total knee replacement (TKR)[1,4], others have reported less successful outcomes[5]. Moreover, demonstration of improvement in function and other quality of life measures has not been consistent[1,2,3] and, importantly, improvement in function has not been equivalent to osteoarthritis (OA)[4]. For patients with OA, endstage knee damage is often a localized problem, which can be effectively treated with TKR. However, for RA, knee destruction is only 1 component of a systemic disease, which may explain why replacement of a single joint may not lead to the same degree of functional improvement compared with OA.

The increased use of disease-modifying antirheumatic

From the Division of Rheumatology, Weill Cornell Medical School, and Department of Medicine, and Department of Orthopedic Surgery, and Department of Biostatistics Core, Hospital for Special Surgery; Albert Einstein College of Medicine; Jacobi Medical Center, New York; North Central Bronx Hospital, Bronx, New York, USA.

Funding for this project was obtained by the Weill Cornell Clinical Translational Science Center (UL1-TR000457-06), AHRQ CERT GRANT: U18 HS016075, and the Block Family Foundation.

S.M. Goodman, MD, Associate Professor of Clinical Medicine, Weill Cornell Medicine College, and Associate Attending Physician, Department of Rheumatology, Hospital for Special Surgery; B.K. Johnson, MD, MS, FACR, Assistant Professor of Medicine, Albert Einstein College of Medicine, and Director of Rheumatology, Jacobi Medical Center, and North Central Bronx Hospital; M. Zhang, PhD, Biostatistician, Hospital for Special Surgery; W.T. Huang, MS, Biostatistician, Hospital for Special Surgery; R. Zhu, BA, Research Assistant, Research and Rheumatology, Hospital for Special Surgery; M.P. Figgie, MD, Professor of Orthopedic Surgery, Weill Cornell College of Medicine, and Attending Orthopedic Surgeon, Chief of Surgical Arthritis Service, Hospital for Special Surgery; M.M. Alexiades, MD, Associate Professor of Orthopedic Surgery, Weill Cornell Medicine College, and Associate Attending Physician, Hospital for Special Surgery; L.A. Mandl, MD, MPH, Assistant Professor of Research Medicine, Assistant Professor of Public Health, Weill Cornell Medicine College, and Assistant Attending Physician, Department of Rheumatology, Hospital for Special Surgery.

Address correspondence to Dr. S.M. Goodman, Hospital for Special Surgery, 535 East 70th St., New York, New York 10021, USA. E-mail: goodmans@hss.edu

Accepted for publication September 16, 2015.

drugs (DMARD) and biologics has resulted in tremendous improvements in function and quality of life, while decreasing articular destruction in contemporary patients with RA[6]. Not surprisingly, rates of many types of orthopedic surgery in RA, such as soft tissue procedures, have significantly decreased[7,8]. However, while the proportion of arthroplasty performed for RA has decreased compared with OA, TKR rates among patients with RA continue to increase[9,10,11]. It appears that TKR will remain an important treatment option for patients with RA with advanced knee damage.

The purpose of our study was to evaluate the pain, function, and quality of life after primary TKR in a contemporary cohort of patients with RA compared with OA. Our hypothesis was that among patients with RA TKR with high DMARD and biologic use, outcomes after TKR will be comparable with OA.

MATERIALS AND METHODS

Our study was performed in a high-volume center that performs over 4300 TKR annually. Most surgeons were experienced in performing arthroplasty in patients with RA; only 10% of the RA cases were performed by surgeons with fewer than 10 RA cases during the study period. All primary TKR patients enrolled in a prospective joint replacement registry between May 1, 2007 and July 1, 2010, who were alive 2 years after surgery, and had baseline data were eligible for our study (Supplementary Figure 1 available online at jrheum.org).

We initially identified 1131 patients using the International Classification of Diseases, 9th ed (ICD-9) code or self-report as RA who were entered into the arthroplasty registry for all knee procedures (primary, revision, bilateral) between May 1, 2007 and February 29, 2011. Of the 267 cases identified by the ICD-9 code and self-report, 200 met the predetermined criteria for RA. Of the 280 identified by ICD-9 code only, 54 met our criteria, and of the 584 cases identified by self-report only, 14 cases met criteria, yielding a total of 268 cases. From this cohort of 268 cases identified and validated between May 1, 2007 and February 29, 2011, we then excluded the cases enrolled outside of our study cohort dates of May 1, 2007 and July 1, 2010, selected to permit 2-year followup, leaving 209 cases. We then excluded revision and bilateral cases, and cases with second procedures during the study period, leaving our study cohort of 136. There were no deaths among the RA cases during this time period. Patients with the ICD-9 codes for fracture, avascular necrosis, or other inflammatory diseases besides RA, as well as patients undergoing a revision or bilateral primary TKR, were excluded. Patients who had 2 eligible procedures only contributed data from the second procedure. About 80% of patients undergoing TKR consented to enroll in the registry.

RA cases were identified by self-report or the ICD-9 code 714.0, and the diagnosis was validated by physician (BKJ) review of medical records[12]. Because investigators did not have access to rheumatology-specific clinical records to ascertain the American College of Rheumatology (ACR) criteria for RA, the diagnosis was validated by meeting predetermined criteria: when the preoperative evaluation by a rheumatologist confirmed the diagnosis of RA, or a preoperative evaluation by an internist confirmed the diagnosis of RA and the patient was receiving a DMARD or biologic agent (excluding steroids). The addition of a rheumatologist's diagnosis of RA and documented use of DMARD significantly increased the accuracy of RA diagnosis for cases identified by the ICD-9 code[13,14].

Additional RA-specific information about medication use was obtained by a questionnaire sent 6 months to 3.5 years after the TKR. Information regarding RA medication use was also obtained from the admission history. Self-reported outcome measures were gathered systematically preoperatively and at 2 years, including the Knee Osteoarthritis Outcome Score, from which the Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) is derived[15], and the Medical Outcomes Study Short Form-12 (SF-12)[16]. We additionally used our hospital administrative database to obtain the American Society of Anesthesiologists (ASA) scores and the ICD-9–based Deyo comorbidities (excluding RA)[17].

Pain, function, and quality of life were assessed using the WOMAC and SF-12 questionnaires. The WOMAC is a widely used self-report instrument that is specific for the lower extremity. Lower extremity pain, stiffness, and function are assessed using 3 subscales, on which a higher score indicates worse status. A difference of 10–15 points is clinically significant, and a score > 40 indicates significant pain and poor function[18,19].

The SF-12 is a generic measure of general health and well-being. The 12-item scale contains 2 subscales, the physical component summary (PCS) and the mental component summary (MCS) scored 1–100. Higher scores on the SF-12 indicate better status. A change of 5 points is clinically significant[16]. Satisfaction was assessed at 2 years. Patients were asked about their satisfaction with the surgery in 4 specific areas: (1) relief of pain, (2) improving ability to do recreational activities, (3) improving ability to do housework or yardwork, and (4) overall satisfaction with the results of the surgery. Satisfaction scores were assessed in each area using a 5-point Likert scale. A global satisfactions question asked, "How much did the surgery improve the quality of your life?" with answers ranging from "more improvement than I ever dreamed possible" to "the quality of my life is worse". Expectations were assessed using the validated Hospital for Special Surgery (HSS) Knee Expectations Survey, which covers areas specific to recovery from knee surgery[20].

Administrative data included the Deyo Comorbidity Index, which is based on the ICD-9 codes and is used to assess comorbid conditions that contribute to overall health. For the patients at our institution undergoing almost exclusively elective TKR, the scores rarely exceeded 3, although the total score possible is 26[17]. Because of this lack of variability, we evaluated the number of Deyo-Charlson comorbidities rather than calculating the index. The ASA score is a ranking used to quantify surgical risk and ranges from 0–6, with a score of 0 indicating excellent health and a score of 6 indicating an organ transplant donor[21].

Descriptive statistics were performed using the Student t test, chi-square test, or Fisher's exact test as appropriate. Significant characteristics of interest were identified in the univariate analysis and included in the multivariable model. The primary outcome measures, WOMAC pain and function, were analyzed as both continuous and dichotomized variables. In the dichotomized analysis, WOMAC pain and function > 40 was defined as a poor outcome. Multivariate linear and logistic regression analyses were then performed controlling for potentially significant confounding variables together with variables of clinical interest, even if not statistically significant, to evaluate the independent association of RA with 2-year pain or function. Collinearity was tested and was not observed during the model-building process.

Our study was approved by our Institutional Review Board.

RESULTS

We identified 9830 primary TKR cases between May 1, 2007 and December 31, 2010. After exclusions, 4456 cases remained eligible for this analysis: 4320 OA cases and 136 validated RA cases.

For the OA cases, 2-year data were available on 94.7% of patients (Table 1). For OA, there were minimal differences between those who completed the 2-year followup survey (n = 4220) and those who did not (n = 236). For RA, 108 (79%) patients had 2-year data. There was no significant difference between patients who responded to the 2-year questionnaire and those who did not for age (63.0 vs 65.4, p = 0.24), body mass index (BMI; 28.4 vs 29.0, p = 0.75), sex (female 90% vs 93%, p = 1.00), or race (white 74% vs

Table 1. Patient characteristics for patients with and without 2-year outcomes. Values are mean (SD) unless otherwise specified.

Characteristic	With 2-yr Outcome, n = 4220 (94.7%)	Without 2-yr Outcome, n = 236 (5.3%)	p
All patients, n = 4456			
Age, yrs	67.1 (10.3)	66.9 (8.9)	0.7143
Female, n (%)	2454 (59)	150 (64)	0.1008
BMI, kg/m^2	30.6 (6.2)	31.8 (7.1)	**0.0140**
Education status at baseline, n (%)			**0.0045**
College or up	2522 (60)	119 (50)	
No college	1698 (40)	117 (50)	
Race, n (%)			0.6579
White	3672 (87)	203 (86)	
Non-white	548 (13)	33 (14)	
RA, n (%)	108 (3)	28 (12)	
Baseline Deyo-Charlson comorbidities, n (%)			0.3892
0 comorbidities	2952 (71)	159 (68)	
1+ comorbidities	1230 (29)	75 (32)	
ASA Class, n (%)			0.2396
Class 1	154 (4)	5 (2)	
Class 2	3054 (72)	162 (69)	
Class 3+	1009 (24)	68 (29)	
Characteristic	With 2-yr Outcome, n = 108 (79%)	Without 2-yr Outcome, n = 28 (21%)	p
Patients with RA, n = 136			
Age, yrs	63.0 (12.1)	65.4 (8.6)	0.2376
Female, n (%)	96 (90)	26 (93)	1.0000
BMI, kg/m^2	28.4 (6.7)	29.0 (9.3)	0.7455
Education status at baseline, n (%)			**< 0.0001**
College or up	47 (44)	1 (4)	
No college	61 (56)	27 (96)	
Race, n (%)			0.6243
White	80 (74)	22 (79)	
Non-white	28 (26)	6 (21)	
Baseline Deyo-Charlson comorbidities, n (%)			0.4354
0 comorbidities	41 (38)	13 (46)	
1+ comorbidities	66 (62)	15 (54)	
ASA Class, n (%)			0.0815
Class 1	0 (0)	0 (0)	
Class 2	69 (64)	13 (46)	
Class 3+	38 (36)	15 (54)	

Significant data are in bold face. BMI: body mass index; RA: rheumatoid arthritis; ASA: American Society of Anesthesia.

79%, p = 0.62). However, those without 2-year data had less educational achievement: 96% of those without 2-year data had no college education compared with 56% of those with 2-year data (p < 0.0001). There was no significant difference in baseline WOMAC pain (with 2-yr response 44.6, SD 18.1 vs without 39.5, SD 14.6, p = 0.3473) or WOMAC function (with 2-yr response 41.1, SD 19.7 vs without 43.0, SD 14.0, p = 0.8244). We included all patients with RA, regardless of 2-year data, to maximize the size of the RA cohort.

Baseline characteristics. Patients with RA were younger (63.5 yrs vs 67.2 yrs, p = 0.0002) and more likely to be women (90% vs 58%, p < 0.0001; Table 2). BMI was significantly lower for RA (28.5 vs 30.7, p < 0.0001). There was a significant difference between RA and OA in terms of educational achievement; 65% of RA had some college education or above compared with 78% of OA (p = 0.015). Fewer RA were white (77% vs 86%) and more RA were African American (12% vs 6%, p = 0.002). Patients with RA had significantly more comorbidities: 40% of RA had no Deyo comorbidities, while 71% of OA had no Deyo comorbidities (p < 0.0001). RA cases also had worse ASA scores (ASA 1 to 2: RA 61% vs OA 76%, p < 0.0001). Length of stay was significantly longer for RA (5.5 days vs 5.1 days, p = 0.0002). Thirteen percent of patients with RA were not treated with DMARD, 35.6% were treated with nonbiologic DMARD only, 38.5% were treated with tumor necrosis factor inhibitors

Table 2. Patient characteristics. Values are mean (SD) unless otherwise specified.

Characteristic	OA, n = 4320	RA, n = 136	p
Age, yrs	67.2 (10.1)	63.5 (11.4)	**0.0002**
Female, n (%)	2482 (58)	122 (90)	**< 0.0001**
BMI, kg/m^2	30.7 (6.2)	28.5 (7.3)	**< 0.0001**
Length of stay, days	5.1 (1.6)	5.5 (1.6)	**0.0002**
Education status at baseline, n (%)			**0.0147**
No college	820(19)	32 (21)	
Some college or above	3292 (78)	68 (65)	
Other	130 (3)	5 (5)	
Race, n (%)			**0.0017**
White	3672 (86)	104 (77)	
Asian	25 (0.6)	3 (2)	
African American	240 (6)	16 (12)	
Hispanic	139 (3)	9 (7)	
Other/mixed	186 (4)	3 (2)	
Baseline Deyo-Charlson comorbidities, n (%)			**< 0.0001**
0 comorbidities	3057 (71)	54 (40)	
1–2 comorbidities	1108 (26)	75 (56)	
3+ comorbidities	116 (3)	6 (4)	
ASA Class, n (%)			**< 0.0001**
Class 1–2	3293 (76)	82 (61)	
Class 3–4	1024 (24)	53 (39)	
Presence of back pain at baseline, n (%)	1606 (42)	41 (42)	0.9403
HSS expectation score at baseline	78.6 (17.9)	68.9 (23.2)	**0.0006**
Underwent prior knee replacement, n (%)	793 (21)	31 (32)	**0.0083**

Significant data are in bold face. OA: osteoarthritis; RA: rheumatoid arthritis; BMI: body mass index; ASA: American Society of Anesthesia; HSS: Hospital for Special Surgery.

(TNFi), 7.4% were treated with non-TNFi biologics, and 5.2% were treated with corticosteroids alone (Table 3). Patients with RA had significantly lower expectations of outcome than patients with OA, with a total HSS Expectations score of 68.9 versus 78.6 (p = 0.0006, with 100 = highest expectations), and a significantly higher proportion of patients with RA had undergone a prior contralateral TKR (32% vs 21%, p = 0.008). There was no significant difference in the presence of back pain reported at the time of TKR, 42% for both patients with RA and OA (p = 0.94).

WOMAC pain was significantly worse at baseline for patients with RA undergoing TKR (55.9 vs 46.6, p < 0.0001) compared with OA (Table 4). However, WOMAC scores at 2 years were equivalent, with excellent pain scores (13.3 vs 12.7, p = 0.65) for both groups. Almost all RA and OA cases achieved a clinically meaningful (ΔWOMAC > 10) improvement in pain (89% in both, p = 0.83). There was no difference in the percent of patients with RA or OA who had poor outcomes (WOMAC > 40) for pain (10% vs 7%; p = 0.44). Among RA, there was no association between use of biologic DMARD and a poor outcome for pain (WOMAC > 40, p = 0.85).

Patients with RA undergoing TKR had clinically and statistically significantly worse baseline WOMAC function (58.7 vs 47.3, p < 0.0001) compared with OA. However, WOMAC scores at 2 years were equivalent, with excellent function scores (17.4 vs 14.7, p = 0.60) for both groups. Almost all RA and OA cases achieved a clinically meaningful change (ΔWOMAC > 10) in function (93% vs 87%, p = 0.21). In the dichotomized analysis, the difference in the percent of patients with RA or OA who had poor outcomes (WOMAC > 40) for function was on the borderline of statis-

Table 3. RA medications. Patients could be taking more than 1 medication.

Medicine	Frequency	Percent	Cumulative Frequency	Cumulative Percent
DMARD	48	35.56	48	35.56
Non-TNF biologics	10	7.41	58	42.96
Steroid	7	5.19	65	48.15
TNF	52	38.52	117	86.67
None	18	13.33	135	100.00

RA: rheumatoid arthritis; DMARD: disease-modifying antirheumatic drugs; TNF: tumor necrosis factor.

Table 4. Preoperative and 2-year pain and function. Values are mean (SD) unless otherwise specified.

Variable	OA, n = 4320	RA, n = 136	p
WOMAC baseline Pain	46.6 (18.0)	55.9 (17.8)	**< 0.0001**
WOMAC 2-yr Pain	12.7 (16.1)	13.3 (15.7)	0.6506
WOMAC baseline Function	47.3 (18.3)	58.7 (19.1)	**< 0.0001**
WOMAC 2-yr Function	14.7 (14.1)	17.4 (13.0)	0.6038
ΔWOMAC > 10, Pain, n (%)	2470 (89)	59 (89)	0.8276
ΔWOMAC > 10, Function, n (%)	2030 (87)	50 (93)	0.2088
Poor outcome at 2 yrs, WOMAC Pain > 40, n (%)	215 (7)	7 (10)	0.4374
Poor outcome at 2 yrs, WOMAC Function > 40, n (%)	276 (9)	12 (16)	0.0527
SF-12 PCS baseline	33.9 (8.2)	28.7 (7.9)	**< 0.0001**
SF-12 PCS at 2 yrs	45.7 (10.1)	40.5 (10.5)	**< 0.0001**
SF-12 MCS baseline	50.8 (12.2)	46.4 (13.2)	**0.0009**
SF-12 MCS at 2 yrs	53.8 (9.4)	48.9 (11.8)	**0.0003**

Significant data are in bold face. OA: osteoarthritis; RA: rheumatoid arthritis; WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index; PCS: physical component summary; MCS: mental component summary.

tical significance (16% vs 9%, p = 0.053). Among RA, there was no difference in outcome for poor function (WOMAC > 40) for patients receiving biologic and synthetic DMARD (p = 0.25).

Quality of life and satisfaction. Preoperative SF-12 PCS was clinically and statistically significantly worse for RA (28.7 vs 33.9, p < 0.0001) and remained so at 2 years (40.5 vs 45.7, p < 0.0001). SF-12 MCS was statistically, but not clinically significantly worse for RA both preoperatively (46.4 vs 50.8, p = 0.0009) and at 2 years (48.9 vs 53.8, p = 0.0003). Satisfaction was high for both RA and OA for TKR (Supplementary Table 1 available online at jrheum.org). Both RA and OA reported that they were very satisfied with pain relief (81% vs 77%, p = 0.89). There was no significant difference in satisfaction reported for RA or OA in the ability to perform recreational activities (very satisfied 54% vs 58%, p = 0.44) or overall satisfaction (very satisfied 72% vs 74%, p = 0.78). There was no significant difference in satisfaction with the improved quality of life, with 74% of patients with RA reporting "more improvement than I ever dreamed possible" and "great improvement" compared with 75% of patients with OA (p = 0.24).

Predictors of poor postoperative pain (Table 5 and Table 6). A multivariate linear regression analysis was performed to identify predictors of poor pain (WOMAC > 40) at 2 years (Table 6). This analysis, controlling for age, sex, diagnosis, education, race, preoperative WOMAC pain, preoperative WOMAC function, preoperative MCS and PCS, and back pain showed that RA was not an independent risk factor for poor postoperative pain (estimated coefficient –3.32, 95% CI –8.2 to 1.56, p = 0.18). Age, sex, education, race, preoperative function, and preoperative PCS were not associated with pain at 2 years. Higher preoperative WOMAC pain scores significantly increased the likelihood of a poor pain outcome (estimated coefficient 0.09, 95% CI 0.02–0.16). Higher preoperative MCS strongly decreased the likelihood of a poor pain outcome (estimated coefficient –0.16 (95% CI –0.23 to –0.10). Similar results were obtained when WOMAC pain was analyzed as a dichotomous variable in a logistic regression controlling for age, sex, education, race, baseline WOMAC pain, baseline WOMAC function, SF-12 PCS, SF-12 MCS, and presence of back pain (Supplementary Table 1 available online at jrheum.org). RA was not a significant risk factor for a poor outcome (OR 1.11, 95% CI 0.40–3.06).

Predictors of poor postoperative function (Table 5 and Table 6). A multivariate linear regression was performed to identify predictors of function at 2 years (Table 6). This analysis, controlling for age, sex, diagnosis, education, race, preoperative WOMAC pain and function, preoperative MCS and PCS, and back pain showed that RA was not an independent risk factor for poor function (estimated coefficient –0.61, 95% CI –5.18 to 3.96, p = 0.79). Higher (worse) preoperative WOMAC function score was a significant predictor of poor functional outcome (estimated coefficient 0.18, 95% CI 0.11–0.26). Higher preoperative PCS (estimated coefficient –0.20, 95% CI –0.32 to –0.08), higher MCS (estimated coefficient –0.21, 95% CI –0.28 to –0.14), and being white (estimated coefficient –2.73, 95% CI –5.23 to –0.24) were protective against poor function. When WOMAC function was analyzed as a dichotomous variable in a logistic regression controlling for age, sex, education, race, baseline WOMAC pain, baseline WOMAC function, SF-12 PCS, SF-12 MCS, and presence of back pain, RA was not a significant risk factor for poor outcome (WOMAC > 40) at 2 years (OR 1.08, 95% CI 0.43–2.70; Supplementary Table 2 available online at jrheum.org).

DISCUSSION

Patients with RA in a contemporary cohort achieved excellent pain and function outcomes after primary TKR, and no longer lag behind patients with OA, despite having significantly

Table 5. Univariate analysis for pain or function 2 years after surgery*.

Characteristic	WOMAC Pain at 2 Yrs, Estimated Coefficient (Standard Error)	WOMAC Function at 2 Yrs, Estimated Coefficient (Standard Error)
Age, yrs	**–0.12 (–0.19 to –0.05)*** **	0.03 (–0.04 to 0.10)***
Female vs male	**2.98 (1.63–4.33)****	**3.29 (1.92–4.67)****
RA vs OA	–0.22 (–4.59 to 4.14)***	1.85 (–2.57 to 6.26)***
ASA Class 2 vs ASA Class 1	2.07 (–1.31 to 5.45)	**3.51 (0.06–6.96)**
ASA Class 3+ vs ASA Class 1	3.32 (–0.26 to 6.90)	**6.85 (3.19–10.52)**
≥ 1 Deyo comorbidities vs 0 Deyo comorbidities	0.64 (–0.85 to 2.13)	**1.76 (0.23–3.28)**
BMI, kg/m^2	0.09 (–0.02 to 0.20)	**0.19 (0.08–0.30)**
≥ College education vs < college	**–3.70 (–5.09 to –2.31)****	**–3.94 (–5.36 to –2.52)****
While vs non-white	**–4.68 (–6.91 to –2.45)****	**–4.93 (–7.20 to –2.66)****
Pre-op WOMAC Pain	**0.21 (0.17–0.24)****	**0.18 (0.15–0.22)****
Pre-op WOMAC Function	**0.21 (0.17–0.25)****	**0.26 (0.22–0.30)****
Pre-op PCS	**–0.32 (–0.41 to –0.24)****	**–0.43 (–0.52 to –0.35)****
Pre-op MCS	**–0.27 (–0.33 to –0.21)****	**–0.34 (–0.39 to –0.28)****
Previous replacement vs no previous replacement	–0.41 (–2.03 to 1.22)	0.82 (–0.85 to 2.48)
Back pain vs no back pain	**4.55 (3.20–5.89)****	**4.41 (3.04–5.79)****
Expectation score	–0.03 (–0.07 to 0.01)	**–0.05 (–0.09 to –0.002)**

Significant data are in bold face. * Univariate linear regression controlling for each individual predictor as listed in the table. ** Variables with p value < 0.05 in the univariate analysis and included in the multivariate regression. *** Variables with p value > 0.05 in the univariate analysis; included in the multivariate regression based on the research of interest. WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index; OA: osteoarthritis; RA: rheumatoid arthritis; ASA: American Society of Anesthesia; BMI: body mass index; Pre-op: preoperative; PCS: physical component summary; MCS: mental component summary.

Table 6. Multivariate analysis for pain or function 2 years after surgery*.

Characteristic	WOMAC Pain at 2 Yrs, Estimated Coefficient (95% CI)	WOMAC Function at 2 Yrs, Estimated Coefficient (95% CI)
Age, yrs	**–0.10 (–0.17 to –0.02)*** **	0.04 (–0.03 to 0.12)***
Female vs male	1.47 (–0.02 to 2.96)	0.83 (–0.60 to 2.26)**
RA vs OA	–3.32 (–8.20 to 1.56)***	–0.61 (–5.18 to 3.96)***
≥ College education vs < college	**–1.56 (–3.09 to –0.04)**	–1.36 (–2.81 to 0.08)**
White vs non-white	**–3.49 (–5.90 to –1.07)**	**–3.93 (–6.21 to –1.65)****
Pre-op WOMAC Pain	**0.09 (0.02–0.16)****	–0.04 (–0.11 to 0.02)
Pre-op WOMAC Function	0.04 (–0.04 to 0.11)	**0.18 (0.11–0.26)****
Pre-op PCS	**–0.13 (–0.24 to –0.01)**	**–0.24 (–0.35 to –0.14)****
Pre-op MCS	**–0.16 (–0.23 to –0.10)****	**–0.24 (–0.31 to –0.18)****
Back pain vs no back pain	**2.83 (1.35–4.31)**	**1.78 (0.38–3.19)**

Significant data are in bold face. * Multivariate linear regression controlling for age, sex, diagnosis, education, race, preoperative WOMAC Pain score, preoperative WOMAC Function score, preoperative PCS, preoperative MCS, and previous back pain. ** Variables with p value < 0.05 in the multivariate regression. *** Variables with p value > 0.05 in the univariate analysis; included in the multivariate regression based on the research interest. WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index; RA: rheumatoid arthritis; OA: osteoarthritis; Pre-op: preoperative; PCS: physical component summary; MCS: mental component summary.

worse preoperative pain and function. While equivalent outcomes for pain have been described for patients with RA[4], equivalent outcomes in function have not been described[5]. Poor baseline pain and function were significant risk factors for poor outcomes for patients with RA, similar to descriptions for patients with OA[22,23]. In addition, the excellent outcomes for patients with RA after TKR occurred in spite of having more comorbidities, another known risk factor for poor outcomes in patients with OA after TKR[24]. It is tempting to speculate that patients with RA, a chronic painful musculoskeletal disease, are better able to cope with the demands of a painful postoperative physical therapy regimen, which is particularly important for achieving good outcomes after TKR.

Contemporary RA have better overall status when compared with patients with RA several decades ago[6]; however, pain and function at the time our subjects elected TKR was significantly worse in RA compared with OA. Therefore, improved TKR outcomes do not simply reflect this overall improved status. We recently demonstrated that patients with RA undergoing total hip replacement (THR) during the same time period in the same institution were significantly more likely to have a poor outcome for WOMAC function than patients with OA[25]. However, there were differences between the patients with RA undergoing TKR described here compared with the patients with RA undergoing THR. Compared with the TKR group, the THR group had a higher comorbidity burden, and fewer had a college education. Prior TKR series have demonstrated that education is strongly predictive of a good outcome for the patients undergoing TKR[26]. Other TKR series also demonstrated an improvement in function and quality of life for RA[27] while older TKR series described worse functional outcomes and similar pain after TKR for RA compared with OA. In this older study, all primary TKR cases were eligible, whereas only the second TKR was included in our study if 2 procedures were performed[4]. Since RA was associated with poor 2-year pain scores only in those undergoing their first THR[25], that might also apply to the TKR patients. High RA-specific surgical volume has been associated with less likelihood of complications after arthroplasty, and might contribute to improved functional outcomes in our cohort as well[28]. Additionally, the inclusion of older cases during a time of significant change in medical, anesthetic, and orthopedic care may explain the difference in these results.

The improved overall quality of life for RA has been attributed to the widespread use of potent DMARD and biologic agents such as the TNFi among patients with RA[29]. In our cohort, 86.7% of patients with RA undergoing primary TKR were receiving DMARD, biologics, and corticosteroids with no difference in outcomes compared with those not receiving synthetic DMARD or biologics. Although better function is reported after knee arthroplasty in high-volume centers[19], this would not affect the strength of our comparison of patients with RA to patients with OA TKR performed in the same high-volume center.

To our knowledge, this is the first large study of TKR in contemporary patients with RA using prospectively gathered data with carefully validated diagnosis, demonstrating equivalent outcomes for patients with RA and OA undergoing TKR. The diagnosis of RA in our study was validated by chart review with an algorithm using both DMARD use and a rheumatologist's diagnosis. While administrative databases alone are well validated for accurate identification of total hip or total knee arthroplasty cases[30,31], the diagnosis of RA by administrative data alone may not be accurate[14]. However, the addition of DMARD therapy increases the accuracy from a positive predictive value (PPV) of 30–60%, and addition of a rheumatologist's diagnosis further increases the PPV to 88–91%[13,14]. Importantly, we used patient-reported outcomes such as the WOMAC to assess pain and function because this lower-extremity–specific survey has been found to be more responsive to change after TKR than generic patient-reported outcomes such as the SF-36 or the Health Assessment Questionnaire[32,33].

A weakness of our study is that all surgery was performed at a specialized high-volume tertiary referral center, with TKR performed by surgeons with high RA-specific volume, so our results may not be generalizable. While the difference between patients with OA and patients with RA in the proportion with poor postoperative function approached statistical significance, we may be underpowered to detect a significant difference. However, as there was no difference in baseline WOMAC function between those with and without 2-year responses, it is unlikely that we systematically excluded those at higher risk of doing poorly. In addition, we did not have direct access to treating rheumatologist's medical records to validate RA cases using the ACR criteria. We had no information about RA disease activity at the time of the surgery, which might have an effect on postoperative course. Although there was little difference in the 79% of patients with 2-year data compared with those without in multiple variables including baseline pain and function, bias could be introduced if there was selective nonresponse by those with poorer outcomes.

Patients with RA undergoing TKR in a contemporary cohort with high prevalence of DMARD and biologic therapy have excellent outcomes and report improvements which are as good as the outcomes of patients with OA for both pain and function after undergoing primary TKR. Our study demonstrates that RA is no longer an independent risk factor for poor TKR outcomes for either pain or function. As patients with RA continue to undergo TKR at increasing rates[34], it is important to have an accurate assessment of TKR outcomes so patients can be given appropriate expectations of TKR.

ONLINE SUPPLEMENT

Supplementary data for this article are available online at jrheum.org.

REFERENCES

1. Osnes-Ringen H, Kvien TK, Henriksen JE, Mowinckel P, Dagfinrud H. Orthopaedic surgery in 255 patients with inflammatory arthropathies: longitudinal effects on pain, physical function and health-related quality of life. Ann Rheum Dis 2009;68:1596-601.
2. Kapetanovic MC, Lindqvist E, Saxne T, Eberhardt K. Orthopaedic surgery in patients with rheumatoid arthritis over 20 years: prevalence and predictive factors of large joint replacement. Ann Rheum Dis 2008;67:1412-6.
3. Massardo L, Gabriel SE, Crowson CS, O'Fallon WM, Matteson EL. A population based assessment of the use of orthopedic surgery in patients with rheumatoid arthritis. J Rheumatol 2002;29:52-6.
4. Singh JA, Lewallen DG. Better functional and similar pain outcomes in osteoarthritis compared to rheumatoid arthritis after primary total knee arthroplasty: a cohort study. Arthritis Care Res 2013;65:1936-41.

5. Kirwan JR, Currey HL, Freeman MA, Snow S, Young PJ. Overall long-term impact of total hip and knee joint replacement surgery on patients with osteoarthritis and rheumatoid arthritis. Br J Rheumatol 1994;33:357-60.
6. Pincus T, Sokka T, Kautiainen H. Patients seen for standard rheumatoid arthritis care have significantly better articular, radiographic, laboratory, and functional status in 2000 than in 1985. Arthritis Rheum 2005;52:1009-19.
7. Hekmat K, Jacobsson L, Nilsson JA, Petersson IF, Robertsson O, Garellick G, et al. Decrease in the incidence of total hip arthroplasties in patients with rheumatoid arthritis—results from a well defined population in south Sweden. Arthritis Res Ther 2011;13:R67.
8. Weiss RJ, Stark A, Wick MC, Ehlin A, Palmblad K, Wretenberg P. Orthopaedic surgery of the lower limbs in 49,802 rheumatoid arthritis patients: results from the Swedish National Inpatient Registry during 1987 to 2001. Ann Rheum Dis 2006;65:335-41.
9. Mertelsmann-Voss C, Lyman S, Pan TJ, Goodman SM, Figgie MP, Mandl LA. US trends in rates of arthroplasty for inflammatory arthritis including rheumatoid arthritis, juvenile idiopathic arthritis, and spondyloarthritis. Arthritis Rheumatol 2014;66:1432-9.
10. Mont MA, Jacobs JJ. AAOS clinical practice guideline: preventing venous thromboembolic disease in patients undergoing elective hip and knee arthroplasty. J Am Acad Orthop Surg 2011;19:777-8.
11. Kurtz S, Mowat F, Ong K, Chan N, Lau E, Halpern M. Prevalence of primary and revision total hip and knee arthroplasty in the United States from 1990 through 2002. J Bone Joint Surg Am 2005;87:1487-97.
12. Johnson BK, Goodman SM, Alexiades MM, Figgie MP, Demmer RT, Mandl LA. Patterns and associated risk of perioperative use of anti-tumor necrosis factor in patients with rheumatoid arthritis undergoing total knee replacement. J Rheumatol 2013;40:617-23.
13. Kim SY, Servi A, Polinski JM, Mogun H, Weinblatt ME, Katz JN, et al. Validation of rheumatoid arthritis diagnoses in health care utilization data. Arthritis Res Ther 2011;13:R32.
14. Ng B, Aslam F, Petersen NJ, Yu HJ, Suarez-Almazor ME. Identification of rheumatoid arthritis patients using an administrative database: a Veterans Affairs study. Arthritis Care Res 2012;64:1490-6.
15. Bellamy N. Instruments to assess osteoarthritis—current status and future needs. Ann Rheum Dis 1995;54:692-3.
16. Busija L, Osborne RH, Nilsdotter A, Buchbinder R, Roos EM. Magnitude and meaningfulness of change in SF-36 scores in four types of orthopedic surgery. Health Qual Life Outcomes 2008;6:55.
17. Deyo RA, Cherkin DC, Ciol MA. Adapting a clinical comorbidity index for use with ICD-9-CM administrative databases. J Clin Epidemiol 1992;45:613-9.
18. Escobar A, García Pérez L, Herrera-Espiñeira C, Aizpuru F, Sarasqueta C, Gonzalez Sáenz de Tejada M, et al. Total knee replacement; minimal clinically important differences and responders. Osteoarthritis Cartilage 2013;21:2006-12.
19. Katz JN, Mahomed NN, Baron JA, Barrett JA, Fossel AH, Creel AH, et al. Association of hospital and surgeon procedure volume with patient-centered outcomes of total knee replacement in a population-based cohort of patients age 65 years and older. Arthritis Rheum 2007;56:568-74.
20. Mancuso CA, Sculco TP, Wickiewicz TL, Jones EC, Robbins L, Warren RF, et al. Patients' expectations of knee surgery. J Bone Joint Surg Am 2001;83-A:1005-12.
21. American Society of Anesthesiologists. ASA physical status classification system. [Internet. Accessed October 14, 2015.] Available from: www.asahq.org/resources/clinical-information/asa-physical-status-classification-system
22. Fortin PR, Clarke AE, Joseph L, Liang MH, Tanzer M, Ferland D, et al. Outcomes of total hip and knee replacement: preoperative functional status predicts outcomes at six months after surgery. Arthritis Rheum 1999;42:1722-8.
23. Dempsey KE, Collins JE, Ghazinouri R, Alcantara L, Thornhill TS, Katz JN. Associations between preoperative functional status and functional outcomes of total joint replacement in the Dominican Republic. Rheumatology 2013;52:1802-8.
24. Singh JA, Lewallen DG. Medical and psychological comorbidity predicts poor pain outcomes after total knee arthroplasty. Rheumatology 2013;52:916-23.
25. Goodman SM, Ramsden-Stein DN, Huang WT, Zhu R, Figgie MP, Alexiades MM, et al. Patients with rheumatoid arthritis are more likely to have pain and poor function after total hip replacements than patients with osteoarthritis. J Rheumatol 2014;41:1774-80.
26. Greene ME, Rolfson O, Nemes S, Gordon M, Malchau H, Garellick G. Education attainment is associated with patient-reported outcomes: findings from the Swedish Hip Arthroplasty Register. Clin Orthop Relat Res 2014;472:1868-76.
27. Benoni AC, Bremander A, Nilsdotter A. Patient-reported outcome after rheumatoid arthritis-related surgery in the lower extremities: a report from the Swedish National Register of Rheuma Surgery (RAKIR). Acta Orthop 2012;83:179-84.
28. Ravi B, Croxford R, Austin PC, Hollands S, Paterson JM, Bogoch E, et al. Increased surgeon experience with rheumatoid arthritis reduces the risk of complications following total joint arthroplasty. Arthritis Rheumatol 2014;66:488-96.
29. Strand V, Sharp V, Koenig AS, Park G, Shi Y, Wang B, et al. Comparison of health-related quality of life in rheumatoid arthritis, psoriatic arthritis and psoriasis and effects of etanercept treatment. Ann Rheum Dis 2012;71:1143-50.
30. Bozic KJ, Chiu VW, Takemoto SK, Greenbaum JN, Smith TM, Jerabek SA, et al. The validity of using administrative claims data in total joint arthroplasty outcomes research. J Arthroplasty 2010;25 Suppl:58-61.
31. Daneshvar P, Forster AJ, Dervin GF. Accuracy of administrative coding in identifying hip and knee primary replacements and revisions. J Eval Clin Pract 2012;18:555-9.
32. Bachmeier CJ, March LM, Cross MJ, Lapsley HM, Tribe KL, Courtenay BG, et al. A comparison of outcomes in osteoarthritis patients undergoing total hip and knee replacement surgery. Osteoarthritis Cartilage 2001;9:137-46.
33. March LM, Cross MJ, Lapsley H, Brnabic AJ, Tribe KL, Bachmeier CJ, et al. Outcomes after hip or knee replacement surgery for osteoarthritis. A prospective cohort study comparing patients' quality of life before and after surgery with age-related population norms. Med J Aust 1999;171:235-8.
34. Mertelsmann-Voss C, Pan TJ, Lyman S, Figgie MP, Mandl LA. Trends in US arthroplasty rates 1991-2005: patients with inflammatory arthritis continue to require joint replacement. Arthritis Rheum 2012;64:S1122.