# Patients with Rheumatoid Arthritis Are More Likely to Have Pain and Poor Function After Total Hip Replacements than Patients with Osteoarthritis

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ABSTRACT. Objective. Total hip replacement (THR) outcomes have been worse for patients with rheumatoid arthritis (RA) compared with those who have osteoarthritis (OA). Whether this remains true in contemporary patients with RA with a high use of disease-modifying and biologic therapy is unknown. The purpose of our study is to assess pain, function, and quality of life 2 years after primary THR, comparing patients with RA and patients with OA.

Methods. Baseline and 2-year data were compared between validated patients with RA and patients with OA who were enrolled in a single-center THR registry between May 1, 2007, and February 25, 2011. Results. There were 5666 eligible primary THR identified, of which 193 were for RA. RA THR had worse baseline Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) pain (44.8 vs 53.2, p < 0.001) and function (38.7 vs 49.9, p < 0.001) compared with OA. These differences remained after surgery: pain (88.4 vs 94.0, p < 0.001) and function (82.9 vs 91.8, p < 0.001). Patients with RA were as likely to have a significant improvement as patients with OA ( $\Delta$  WOMAC > 10) in pain (94% vs 96%, p = 0.35) and function (95% vs 94%, p = 0.69), but were 4 times as likely to have worse function (WOMAC  $\leq$  60; 19% vs 4%, p < 0.001) and pain (12% vs 3%, p < 0.001). In multivariate logistic regression controlling for multiple potential confounders, RA increased the odds of poor postoperative function (OR 4.32, 95% CI 1.57-11.9), and in patients without a previous primary THR, worse postoperative pain (OR 3.17, 95% CI 1.06–9.53). Conclusion. Contemporary patients with RA have significant improvements in pain and function after THR, but higher proportions have worse 2-year pain and function. In addition, RA is an independent predictor of 2-year pain and poor function after THR, despite high use of diseasemodifying therapy. (J Rheumatol First Release Aug 1 2014; doi:10.3899/jrheum.140011)

*Key Indexing Terms:* ARTHROPLASTY OSTEOARTHRITIS

RHEUMATOID ARTHRITIS OUTCOME MEASURES

Total hip replacement (THR) is one of the most successful surgical interventions, consistently relieving pain and restoring function for patients with endstage osteoarthritis (OA) of the hip<sup>1</sup>. Historically, over 50% of patients with

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rheumatoid arthritis (RA) have reported use of orthopedic procedures, predominantly large-joint arthroplasty<sup>2</sup>. Quality of life and health status have improved dramatically for patients with RA over the past 30 years, coincident with the increased use of synthetic disease-modifying antirheumatic drugs (DMARD) such as methotrexate<sup>3</sup>, as well as biologic therapy, such as tumor necrosis factor- $\alpha$  inhibitors (TNFi)<sup>4</sup>. However, despite significant improvements associated with the widespread use of these agents, rates of THR for patients with RA have not significantly decreased, and therefore THR maintains an important role in RA management<sup>5,6</sup>.

THR outcomes in contemporary patients with RA are not well described. Studies in older cohorts have suggested that while THR leads to significant pain relief in the operated joint, it is less effective in improving health-related quality of life (HRQOL) and overall function<sup>7,8,9</sup>. This may be less important now, because health status in RA has increased dramatically with conventional and biologic DMARD use. The purpose of our study is to assess pain, function, and

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quality of life 2 years after THR in a contemporary cohort of patients with RA with a high level of DMARD and biologic use, compared to patients with OA. We additionally compared satisfaction and expectations of THR.

#### MATERIALS AND METHODS

Our study took place at a high-volume musculoskeletal specialty hospital. We included all patients undergoing primary THR between May 1, 2007, and February 25, 2011, who were enrolled in a single institutional THR registry, had preoperative data, and were alive at 2 years after THR. Patients with International Classification of Diseases, 9th ed (ICD-9) codes for fracture, avascular necrosis, or other inflammatory diseases besides RA, as well as patients undergoing a revision or bilateral primary THR, were excluded. Patients who had 2 eligible procedures only contributed information from the second procedure. Data collected included basic demographic information, Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC), and the SF-12v2 Short Form Health Survey (SF-12)<sup>10,11</sup>. We additionally used our hospital administrative database to obtain the Deyo list of ICD-9 morbidities, with RA being excluded as a comorbidity for the purposes of this analysis<sup>12</sup>, and American Society of Anesthesia (ASA) score<sup>13</sup>.

RA was identified by self-report or ICD-9 code 714.0, and the diagnosis was validated by medical record review. As a tertiary referral center, patients seen at our institution often receive rheumatologic care elsewhere, so rheumatology-specific medical records were not available. Therefore, a diagnosis of RA was validated if a preoperative evaluation by a rheumatologist confirmed the diagnosis of RA, or if a preoperative evaluation by an internist confirmed the diagnosis of RA and the patient was receiving a DMARD or biologic agent, excluding corticosteroids. Information on medication use was obtained from the hospital chart.

Pain, function, and health status were assessed using the WOMAC and SF-12 questionnaires. The WOMAC, a well-validated lower extremity specific scale, evaluates pain, stiffness, and function on a 0-100 scale; a high score indicates better status in our study. An improvement of 10 points in WOMAC is considered clinically significant<sup>14,15</sup>. A poor outcome for WOMAC pain or function is defined as a score  $\leq 60^{14}$ . The SF-12 is a generic measure of general health and well-being. The physical component scale (PCS) and the mental component scale (MCS) are 2 subscales that consist of 12 items and are scored 1-100. Higher scores on the SF-12 indicate better status. A change of 5 points is considered clinically significant<sup>11</sup>. Expectations were measured on the Hospital for Special Surgery (HSS) Total Hip Replacement Expectations Survey, a validated instrument that specifically questions a patient's expectations prior to THR in areas specific to THR. These include pain relief, and expected resumption of specific activities including sports and recreation on a 1-100 scale<sup>16</sup>. A difference of 7 is clinically meaningful<sup>17</sup>. Satisfaction was assessed at 2 years. Patients were asked about their satisfaction with the surgery in 4 areas using a 5-point Likert scale: 1 = relief of pain, 2 = improving ability to do housework or yard work, 3 = improving ability to do recreational activities, and 4 = overall satisfaction with the results of the surgery. A final, fifth question asks, "How much did the surgery improve the quality of your life?" Answers range from "more improvement than I ever dreamed possible" to "the quality of my life is worse"18. The ASA score is a ranking used to quantify surgical risk and ranges from 0-6. A score of 0 indicates excellent health, and a score of 6 indicates an organ donor<sup>13</sup>.

Chi-square, Fisher's exact test, and t tests were used to compare baseline characteristics, as appropriate. Multivariate logistic regressions were performed, controlling for potentially significant confounding variables, to evaluate the independent association of RA with poor 2-year pain or function. After we observed a significant difference between patients with RA and patients with OA among our baseline risk factors, we used them to build multivariate logistic regressions. Backward selection was used based on smaller Akaike Information Criterion value. Some variables had to be excluded from the final models attributable to nonconvergence. Collinearity was tested for and was not observed during the model-building process.

Our study was approved by the HSS Institutional Review Board.

## RESULTS

There were 847 potential RA THR identified by ICD-9 codes or self-report, and 258 (30.4%) were validated after chart review. After excluding cases not meeting all entry criteria, 5473 patients with OA and 193 patients with RA remained eligible for this analysis (Table 1). For eligible patients, the 2-year followup response rate was 69% for RA and 98% for OA. However, there were no statistically significant differences between patients with RA with and without responses to 2-year followup data with regards to age (62.9 vs 64.3 yrs), body mass index (BMI; 27.5 vs 28.4), female sex (76% vs 80%), ASA Class (1 + 2: 67% vs 56%), and race (white: 79% vs 69%; Table 2). There was no significant difference in WOMAC pain (44.7 vs 55.0, p = 0.49) or WOMAC function (38.4 vs 55.9, p = 0.23). The only statistically significant difference between responders and nonresponders was the length of hospital stay in days (4.9 vs 5.7; p = 0.01).

There was no significant difference in age (OA 62.8 yrs vs RA 63.3, p = 0.51) or BMI (OA 28.2 vs RA 27.7, p =

Characteristics	OA, n = 5473	RA, n = 193	р
Age, mean (SD)	62.8 (11.6)	63.3 (13.6)	0.51
Female, n (%)	2613 (48)	147 (77)	< 0.001
BMI, mean (SD)	28.2 (5.4)	27.7 (6.6)	0.33
Length of stay, days, mean (S	D) 4.6 (1.3)	5.1 (2.1)	< 0.001
Expectation score, mean (SD)	83.6 (16.2)	80.0 (18.3)	< 0.001
College degree or higher,			
n (%)	3834 (70)	71 (37)	< 0.001
Race, n (%)			< 0.001
White	5031 (92)	147 (76)	
Asian	37 (1)	3 (2)	
African American	194 (4)	20 (10)	
Other/Mixed	39 (1)	8 (4)	
Deyo-Charlson comorbidities	< 0.001		
0	4296 (79)	60 (31)	
1+	1131 (21)	131 (68)	
ASA class, n (%)			< 0.001
Class 1 or 2	4575 (83)	124 (64)	
Class 3 or 4	895 (17)	69 (36)	
Previous hip replacement,			
n (%)	953 (20)	31 (27)	0.06
Back pain at baseline, n (%)	2294 (45)	41 (35)	0.04
DMARD		41.5%	
TNFi		28.5%	
Non-TNF biologics		5.2%	
Corticosteroid		8.3%	
None		16.6%	

Values in bold face are statistically significant. OA: osteoarthritis; RA: rheumatoid arthritis; BMI: body mass index; ASA: American Society of Anesthesia; DMARD: disease-modifying antirheumatic drugs; TNFi: tumor necrosis- $\alpha$  inhibitors.

Table 2. RA responders versus nonresponders.

	Responders, n = 134	Nonresponders, n = 59	р
Age, yrs, mean (SD)	62.9 (13.5)	64.3 (13.8)	0.52
BMI, kg/m <sup>2</sup> , mean (SD)	27.5 (6.4)	28.4 (7.0)	0.42
Length of stay, days, mean (SD	<b>) 4.9</b> ( <b>1.7</b> )	5.7 (2.7)	0.01
Female, n (%)	100 (76)	47 (80)	0.55
ASA class 1 and class 2, n (%)	91 (67)	33 (56)	0.20
White, n (%)	106 (79)	41 (69)	0.44
Baseline WOMAC pain	44.7 (20.4)	55.0 (63.6)	0.49
Baseline WOMAC function	38.4 (19.4)	55.9 (62.4)	0.23

BMI: body mass index; ASA: American Society of Anesthesia; WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index.

0.33) between groups (Table 1). Patients with OA were less likely to be women (48% vs 77%, p < 0.001). Of the patients with RA, 37% were at least college graduates, compared with 70% with OA (p < 0.001). Fewer patients with RA were white (76% vs 92%), and more were African American (10% vs 4%) or Hispanic (7% vs 3%; p < 0.001 for trend). Length of hospital stay was significantly longer for RA (5.1 days vs 4.6 days, p < 0.001). For RA, 41.5% were receiving DMARD, 28.5% were receiving TNFi therapy, 5.2% were receiving non-TNFi biologics, 16.6% were taking no DMARD, and 8.3% were taking corticosteroids. RA had statistically significantly lower overall expectations of THR outcomes than OA (summary score of the HSS Expectations Survey, 80.0 vs 83.6, p = 0.03), a difference that was statistically significant but not clinically meaningful.

RA had significantly more comorbidities than OA (0 Deyo comorbidities: RA 31% vs OA 79%, p < 0.001). ASA class was also worse for RA. Only 16% of OA were in ASA class 3 or 4, compared with 36% of RA (p < 0.001). Function. RA THR (Table 3) had worse baseline WOMAC function compared to OA (38.7 vs 49.9, p < 0.001), a difference that was clinically and statistically significant. Similar proportions of patients with RA and patients with OA had clinically significant improvements in function (defined as a WOMAC > 10; 95% vs 94%, respectively; p = 0.69). However, RA had clinically and significantly worse WOMAC function at 2 years (82.9 vs 91.8, p < 0.001). In addition, patients with RA were 4 times more likely to have a poor 2-year functional outcome than OA (defined as WOMAC  $\leq 60, 19\%$  vs 4%, p < 0.001). Within RA, patients treated with biologics or synthetic DMARD were as likely to have a poor functional outcome (WOMAC  $\leq 60$ ) as those not treated with biologics or synthetic DMARD (p = 0.98). Pain. RA had statistically significantly worse preoperative WOMAC pain (44.8 vs 53.2, p < 0.001) and had worse pain at 2 years (88.4 vs 94.0, p < 0.001). However, neither of these differences was clinically meaningful. Although both RA and OA were equally likely to show a clinically meaningful improvement in pain (WOMAC > 10, RA 94%

Table 3. Baseline and 2-year outcomes. Data are mean (SD) unless otherwise indicated.

	OA, n = 5473	RA, n = 193	р
WOMAC baseline pain (SD)	53.2 (17.9)	44.8 (21.1)	< 0.001
WOMAC 2-yr pain (SD)	94.0 (11.2)	88.4 (17.1)	< 0.001
WOMAC > 10, pain, n (%)	3549 (96)	72 (94)	0.35
WOMAC baseline function (SD)	49.9 (18.6)	38.7 (20.3)	< 0.001
WOMAC 2-yr function (SD)	91.8 (12.5)	82.9 (19.6)	< 0.001
WOMAC > 10, function, n (%)	3071 (94)	63 (95)	0.69
Poor pain at 2 yrs, WOMAC			
pain ≤ 60, n (%)	122 (3)	10 (12)	< 0.001
Poor function at 2 yrs,			
WOMAC function $\leq 60$ , n (%)	152 (4)	16 (19)	< 0.001
SF-12 PCS baseline (SD)	33.9 (8.4)	29.3 (7.9)	< 0.001
SF-12 PCS at 2 yrs (SD)	50.1 (9.3)	40.8 (13.0)	< 0.001
SF-12 MCS baseline (SD)	50.1 (12.5)	46.4 (13.6)	< 0.001
SF-12 MCS at 2 yrs (SD)	54.1 (8.8)	52.1 (10.9)	0.05

OA: osteoarthritis; RA: rheumatoid arthritis; WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index; SF-12: SF-12 Short Form Health Survey; PCS: physical component scale; MCS: mental component scale.

vs OA 96%, p = 0.35), 4 times more RA had poor pain score at 2 years (WOMAC pain  $\leq$  60, RA 12% vs OA 3%, p < 0.001). Within RA, patients treated with biologics or synthetic DMARD were as likely to have a poor pain outcome (WOMAC  $\leq$  60) than those not treated with biologics or synthetic DMARD (p = 0.98).

RA as an independent predictor of poor pain or function. Multivariate logistic regression was performed to determine predictors of poor function (WOMAC  $\leq 60$ ), controlling for age, sex, diagnosis, education, race, expectations score, number of Deyo comorbidities, previous hip replacement (by definition this was a contralateral THR, as only primary THR were included in this analysis), BMI, as well as preoperative WOMAC pain, WOMAC function, MCS, and presence of back pain self-reported preoperatively (Table 4). RA was found to be a significant independent risk factor for poor function (WOMAC  $\leq$  60) at 2 years (OR 4.32, 95% CI 1.57-11.9) compared with OA. Additional significant predictors included higher expectations, which decreased the odds of poor WOMAC function (OR 0.97, 95% CI 0.96-0.99, p = 0.0005). Measured on a scale of 1-100, a 10-point higher score would decrease the odds of a poor outcome by about 80%. Better MCS and WOMAC function also significantly decreased the odds of poor function, with a similar magnitude of effect. Prior THR on the other side did not change the odds of a poor functional outcome. However, because we were concerned that the experience of a previous THR may influence outcomes, an analysis stratified on those with [RA: n = 31 (27%), OA: n = 953 (20%), p = 0.06] and those without a previous THR on the other side was also performed, and no difference was observed.

In a second multivariable logistic regression to determine predictors of poor pain (WOMAC  $\leq$  60) controlling for age,

	Poor WOMAC Pain at 2 Yrs, OR (95% CI)	р	Poor WOMAC Function at 2 Yrs, OR (95% CI)	р
Age	0.99 (0.97, 1.01)	0.14	1.01 (0.99, 1.00)	0.34
Female vs male	1.06 (0.61, 1.83)	0.36	1.25 (0.69, 2.26)	0.46
RA vs OA	3.22 (1.29, 8.07)	0.01	4.32 (1.57, 11.89)	0.005
Back pain vs no back pain	1.25 (0.75, 2.08)	0.30	1.62 (0.93, 2.82)	0.08
$25 \leq \mathrm{BMI} < 30$ vs $18.5 < \mathrm{BMI} < 25$	0.91 (0.48, 1.73)	0.78	1.36 (0.65, 2.84)	0.42
$30 \leq \mathrm{BMI} < 35$ vs $18.5 < \mathrm{BMI} < 25$	0.94 (0.46, 1.93)	0.87	1.64 (0.75, 3.56)	0.22
$35 \leq \mathrm{BMI} < 40$ vs $18.5 < \mathrm{BMI} < 25$	0.42 (0.13, 1.31)	0.13	0.78 (0.23, 2.66)	0.70
BMI > 40 vs 18.5 < BMI < 25	1.30 (0.44, 3.85)	0.63	2.93 (0.95, 8.99)	0.06
Preoperative WOMAC pain	0.99 (0.97, 1.01)	0.17	1.01 (0.98, 1.03)	0.71
Preoperative PCS	0.95 (0.91, 0.99)	0.01	**	**
Preoperative WOMAC function	**	**	0.96 (0.94, 0.99)	0.03
Preoperative MCS	0.96 (0.94, 0.98)	0.0002	0.98 (0.96, 0.999)	0.04
Education: college vs no college	0.83 (0.49, 1.41)	0.56	0.74, (0.43, 1.30)	0.30
White vs other	0.46 (0.23, 0.92)	0.02	0.66 (0.30, 1.47)	0.31
Expectation score	**	**	0.97 (0.96, 0.99)	0.0005
Deyo: 0 comorbidities vs 1–3+	**	**	0.80 (0.44, 1.47)	0.47
ASA class 2 vs ASA class 1	1.01 (0.37, 2.74)	0.99	**	**
ASA class 3+ vs ASA class 1	2.11 (0.67, 6.59)	0.20	**	**
Previous hip replacement vs no				
previous replacement	1.37 (0.75, 2.50)	0.30	1.35 (0.72, 2.53)	0.35

*Table 4*. Predictors of having poor pain or function (WOMAC < 60) 2 years after hip replacement\* (logistic regression results).

\* Multivariate regression controlling for age, sex, diagnosis, BMI, education, race, back pain, previous hip replacement, preoperative WOMAC pain and function, preoperative MCS and PCS, expectations score, ASA class, and number of Deyo comorbidities. \*\* Variable excluded because of nonconvergence of logistic model. WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index; RA: rheumatoid arthritis; OA: osteoarthritis; BMI: body mass index; PCS: physical component scale; MCS: mental component scale; ASA: American Society of Anesthesia.

sex, diagnosis, education, race, ASA class, previous hip replacement, BMI, as well as preoperative WOMAC pain, PCS, MCS, and presence of preoperative back pain, RA was significantly associated with poor pain (OR 3.22, 95% CI 1.29-8.07, p = 0.01). Better MCS and PCS also reduced the odds of poor pain (Table 4), with a similar magnitude of effect as for function. However, when patients were stratified into those with and without a previous contralateral THR, having a previous THR appeared to be an effect modifier. RA remained associated with poor pain in those without a previous THR (Table 6, OR 3.17, 95% CI 1.06-9.53, p = 0.04). However, in patients with a previous contralateral THR, RA was not associated with poor postoperative pain (OR 4.00, 95% CI 0.72-22.33, p = 0.11). In fact, only race remained a significant predictor of a poor pain outcome in those with a previous contralateral THR (OR 0.20, 95% CI 0.05–0.85, p = 0.03).

Although patients with RA were as satisfied with pain relief as those with OA (94% vs 97%, p = 0.43), they were less likely to describe themselves as "very/somewhat satisfied" with the overall THR outcome (90% vs 96%, p < 0.001) and were less satisfied with the improvement in their quality of life (more improvement than I ever dreamed possible/great improvement: 79% vs 89% for OA, p < 0.001). RA were also significantly less likely to be very/somewhat satisfied with improved ability to do recreational activities (79% vs 92%, p = 0.002; Table 5).

## DISCUSSION

Our study shows that even in a high-volume orthopedic center of excellence, contemporary patients with RA with high use of biologic and synthetic DMARD continue to have worse pain and function 2 years after primary THR compared to patients with OA. This suggests that replacing a single joint may be less helpful in RA compared with OA. RA is a symmetric, polyarticular disease, in contrast to OA, which may only affect a single joint. Worse outcomes in RA may be attributable to "other troublesome joints," which we were not able to record<sup>7</sup>. However, there is also a high prevalence of other affected joints in patients undergoing arthroplasty for OA16. In fact, worse functional outcomes have been associated with both ipsilateral and contralateral joint involvement for patients with OA undergoing arthroplasty<sup>19</sup>. Therefore, for RA THR, other unmeasured confounders such as RA disease activity or overall frailty may contribute to the worse outcome. This should be assessed in future studies. Additionally, there was no difference in the proportion of patients with RA with poor outcomes who were treated with biologics and traditional DMARD compared to those not treated, which may have

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	OA, n = 5473	RA, n = 193	р
Very/Somewhat satisfied with pain relief, n (%) Very/Somewhat satisfied with improved ability to do	3710 (97)	72 (94)	0.43
recreational activities, n (%)	3470 (92)	60 (79)	0.002
Very/Somewhat satisfied overall, n (%)	3676 (96)	<b>69 (90)</b>	< 0.001
Great/moderate improvement in quality of life, n (%)	3436 (89)	63 (79)	< 0.001

OA: osteoarthritis; RA: rheumatoid arthritis.

*Table 6*. Predictors of having poor pain (WOMAC < 60) 2 ys after hip replacement\* for patients with and without previous hip replacement (logistic regression results).

	Poor WOMAC Pain at 2 Yrs Without Previous Hip Replacement, OR (95% CI)	р	Poor WOMAC Pain at 2 Yrs With Previous Hip Replacement, OR (95% CI)	р
Age	0.99 (0.96, 1.02)	0.46	0.98 (0.93, 1.04)	0.54
Female vs male	1.02 (0.54, 1.91)	0.96	1.06 (0.33, 3.37)	0.93
RA vs OA	3.17 (1.06, 9.53)	0.04	4.00 (0.72, 22.33)	0.11
Back pain vs no back pain	1.17 (0.65, 2.11)	0.59	1.63 (0.54, 4.89)	0.38
$25 \le BMI < 30 \text{ vs } 18.5 < BMI < 25$	0.87 (0.42, 1.78)	0.70	1.23 (0.27, 5.54)	0.79
$30 \le BMI < 35 \text{ vs } 18.5 < BMI < 25$	0.93 (0.42, 2.07)	0.85	1.07 (0.19, 6.16)	0.94
$35 \le BMI < 40 \text{ vs } 18.5 < BMI < 25$	0.35 (0.09, 1.32)	0.12	0.74 (0.07, 8.00)	0.81
BMI > 40 vs 18.5 < BMI < 25	0.65 (0.16, 2.72)	0.55	5.61 (0.78, 40.27)	0.09
Preoperative WOMAC pain	0.98 (0.97, 1.00)	0.19	1.00 (0.96, 1.04)	0.86
Preoperative PCS	0.94 (0.89, 0.99)	0.01	0.96 (0.88, 1.05)	0.39
Preoperative MCS	0.96 (0.94, 0.98)	0.0009	0.96 (0.92, 1.01)	0.10
Education: college vs no college	0.83 (0.46, 1.51)	0.55	0.74 (0.24, 2.30)	0.61
White vs other	0.59 (0.26, 1.32)	0.20	0.20 (0.05, 0.85)	0.03
ASA class 2 vs ASA class 1	0.84 (0.30, 2.36)	0.74	**	0.96
ASA class 3+ vs ASA class 1	1.89 (0.56, 6.34)	0.30	**	0.95

\* Multivariate regression controlling for age, sex, diagnosis, back pain, BMI, preoperative WOMAC pain and function, preoperative MCS and PCS, education, race, expectations score, ASA class, number of Deyo comorbidities, and previous hip replacement. \*\* Variable excluded because of nonconvergence of logistic model. WOMAC: Western Ontario and McMaster Universities Osteoarthritis Index; RA: rheumatoid arthritis; OA: osteoarthritis; BMI: body mass index; PCS: physical component scale; MCS: mental component scale; ASA: American Society of Anesthesia.

been attributable to the high prevalence of use in this cohort, where all but 16.6% of patients with RA were so treated.

RA was found to be an independent predictor of poor postoperative function after controlling for multiple potential confounding variables. Interestingly, RA was also associated with poor postoperative pain only in those having their first primary THR. Whether having had a previous contralateral hip THR is a true effect modifier for poor postoperative pain is intriguing. Patients electing a second primary THR may do so only if the first went well, and therefore those with a higher risk of poor outcomes may not choose surgery. However, because the number of patients with RA having a second THR was small, we may be underpowered to show a true significant association in this group. Studies in other cohorts need to replicate this finding.

Our findings that patients with RA have worse outcomes after THR contrasts with our previous work, which demonstrated that contemporary patients with RA undergoing primary total knee replacement (TKR) have similar excellent outcomes compared with OA<sup>20</sup>. Although others have reported that patients with RA have worse function or no significant improvement in function after THR<sup>21</sup>, these were older cohorts<sup>9</sup>, and used the Medical Outcomes Study Short Form-36 and Health Assessment Questionnaire, which are less sensitive to change in function after THR than is the WOMAC<sup>22,23</sup>. We hypothesized that the significant changes in RA therapy, as well as better surgical techniques, would have led to improvements in THR similar to those we found in TKR. Our results were also unexpected because THR typically have more predictable improvements in pain and function than do TKR<sup>24,25,26</sup>. Others have also found significant improvements after THR<sup>23,27</sup>, but the small numbers of THR in those studies and lack of an arthroplasty comparator group limit their conclusions. Further work needs to be done in other cohorts to confirm our findings.

Despite having worse baseline pain and function, and more comorbidities, patients with RA had similarly high

expectations for pain relief from THR as had patients with OA, and were as satisfied as patients with OA in terms of pain relief, although fewer of our patients with RA described high levels of overall satisfaction. Having more comorbidities has been associated with less improvement in HRQOL after THR for patients with OA<sup>28,29</sup>, but this was not an independent risk factor for poor pain or function in our patients. This may reflect improved contemporary surgical or anesthetic practice, because all our cases were gathered after 2007.

A strength of our study is the large number of recent patients with THR for whom we used prospectively acquired data. RA cases were carefully validated using a proven methodology<sup>33</sup>. We assessed patient-reported outcomes using well-validated instruments, including the WOMAC<sup>24</sup>.

Limitations include the fact that all THR were performed at a specialized high-volume tertiary referral hospital, so our results may not be generalizable, because most THR are performed at low-volume hospitals<sup>30</sup>. However, high surgical volume has not been shown to be associated with better functional outcomes after THR<sup>31</sup>. Although the RA diagnosis was not based on American College of Rheumatology criteria<sup>32</sup>, we used an algorithm that included ICD-9 code, and/or self-report, as well as DMARD use, and diagnosis of RA by a rheumatologist. This approach has been shown to significantly increase diagnostic validity compared to the use of ICD-9 code alone<sup>33</sup>. In addition, because our data is taken from a surgical registry, we did not have RA-specific information such as duration of disease and activity of disease, which would add significantly to our study.

Patients with RA had lower proportions of followup data compared with patients with OA. This could lead to bias if there were systematic nonresponse. However, there were no major differences between responders and nonresponders in baseline demographics or self-report outcomes. In addition, if patients with RA with worse outcomes were less likely to follow up, this would have resulted in a conservative bias and would underestimate our finding of poorer function for RA.

Although patients with RA demonstrate significant improvements in pain and function after primary THR, RA appears to be an independent risk factor for poor pain and function 2 years after THR. This is important information for patients and their physicians to know as they consider treating RA with THR.

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