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Risk of Osteoarthritis in an Incident Cohort of People With Psoriatic Arthritis: A Population-based Cohort Study

Rachel A. Charlton[1], Amelia Green[1], Gavin Shaddick[2], Julia Snowball[1], Alison Nightingale[1], William Tillett[3], Catherine Smith[4], and Neil J. McHugh[1], on behalf of the PROMPT Study Group

ABSTRACT. ***Objective.*** To determine the risk of a diagnosis of osteoarthritis (OA) in patients with psoriatic arthritis (PsA) compared to patients with psoriasis and a general population cohort.

Methods. Incident PsA patients aged 18–89 years at diagnosis were identified from the United Kingdom Clinical Practice Research Datalink between 1998 and 2014. All patients with PsA were matched to 2 cohorts of patients, both at a 1:4 ratio. The first cohort included patients with psoriasis (and no PsA) and the second was a general population cohort (with no psoriasis or PsA). The baseline prevalence of OA was calculated for each study cohort. The incidence of OA was calculated, and adjusted relative risks (RR_{adj}) were calculated using conditional Poisson regression.

Results. We identified 6783 incident PsA patients. The baseline prevalence of OA ranged from 22.1% (95% CI 21.1–23.1) in the PsA cohort to 12.6% (95% CI 12.2–13.0) and 11.0% (95% CI 10.6–11.3) in the psoriasis and general population cohorts, respectively. The incidence of OA was significantly higher in the PsA cohort compared to the psoriasis and general population cohorts after adjusting for BMI (RR_{adj} 1.68, 95% CI 1.46–1.93, and RR_{adj} 1.86, 95% CI 1.62–2.14, respectively).

Conclusion. An increased risk of OA was observed in patients with PsA compared to patients with psoriasis alone and those in the general population. Further work is needed to determine whether this reflects a true increase in OA risk or misdiagnosed PsA, and the extent to which it can be explained by differences in the opportunity for OA diagnosis between cohorts.

Key Indexing Terms: cohort study, osteoarthritis, psoriasis, psoriatic arthritis

Psoriatic arthritis (PsA) is a chronic inflammatory arthritis that causes pain, stiffness, and swelling around the joints. PsA is well recognized to be progressive, resulting in reduced quality of life and work disability, that can be improved with early diagnosis and effective treatment.[1,2] PsA is reported to affect between 10 and 40% of individuals with psoriasis[3]; in the majority of patients, PsA presents after, or synchronously with, psoriasis onset.[4] Osteoarthritis (OA) is a common form of arthritis and typically commences late in the fifth decade.[5] OA is a disabling condition that can affect any joint; one of the most commonly affected sites is the knee, followed by the hand and hip. PsA and OA have long been considered 2 distinct arthropathies; however, they do have some overlapping features and symptoms. In certain circumstances, it can be difficult to differentiate between them, particularly in the small joints of the hands or spine.[5] It has been demonstrated that obesity is a risk factor for both OA and PsA.[6,7] The present study aimed to determine the risk of OA in patients with PsA in the United Kingdom, and compare this with the risk in a matched cohort of psoriasis patients without PsA and a matched general population cohort, in order to determine whether there is any evidence of an increased risk of OA in patients with PsA and/or psoriasis, and whether there is any evidence of PsA being misdiagnosed as OA.

METHODS

This study used data from the Clinical Practice Research Datalink (CPRD), an electronic healthcare database containing anonymized longitudinal medical records for ~15 million individuals collected within UK primary care, which has been shown to be generally representative of the UK population.[8] The protocol was reviewed by the Independent Scientific Advisory Committee for MHRA Database Research (approved protocol 15_154R).

Study population. A cohort of incident PsA patients was identified in the

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[1]R.A. Charlton, Research Fellow, PhD, A. Green, Research Officer, MSc, J. Snowball, Research Fellow, MSc, A. Nightingale, Research Fellow, PhD, N.J. McHugh, Professor of Pharmacoepidemiology, MBChB, MD, Department of Pharmacy and Pharmacology, University of Bath, Bath; [2]G. Shaddick, Professor of Statistics, PhD, Department of Mathematics, University of Exeter; [3]W. Tillett, Consultant Rheumatologist and Senior Lecturer, MBChB, PhD, Department of Pharmacy and Pharmacology, University of Bath, Bath, and Royal National Hospital for Rheumatic Diseases, Bath; [4]C. Smith, Professor of Dermatology and Therapeutics, MD, FRCP, Guys and St Thomas' NHS Foundation Trust, London, UK.

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Address correspondence to Prof. N.J. McHugh, Department of Pharmacy and Pharmacology, University of Bath, Claverton Down, Bath, BA2 7AY, UK. Email: n.j.mchugh@bath.ac.uk.

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CPRD who were diagnosed between January 1, 1998, and December 31, 2014, and aged 18–89 years at diagnosis. Patients were required to have ≥ 1 year of data contribution considered to be up to the standard required for research before their PsA diagnosis date (index date). Cases of PsA were matched at a 1:4 ratio to 2 randomly selected cohorts based on their index date, year of birth, sex, and general practice. The first matched cohort (general population cohort) included patients with no psoriasis, no PsA, and no other inflammatory arthritis (which did not include OA) at baseline; the second cohort (psoriasis cohort) included patients with psoriasis but no diagnosis of PsA or other inflammatory arthritis (which did not include OA) at baseline. The index date of the matched case was assigned to patients in the comparator cohorts, and they were required to have ≥ 1 year of research standard data contribution prior to the index date. Patients were followed from the index date until the date they were no longer eligible for the cohort or were diagnosed with the outcome of interest. Patients in the general population, and psoriasis cohorts who developed psoriasis or PsA after the index date, had their person-time contribution to that cohort censored the day before the diagnosis date, after which they contributed to the corresponding psoriasis or PsA cohort.

Identification of PsA and psoriasis patients. Patients with PsA were identified based on the presence of a Read code, which previous studies in a similar UK database have found to have a high positive predictive value (85%, 95% CI 75.8–91.7).[9] An algorithm was developed to exclude patients where there was evidence to suggest that the PsA diagnosis may have been a misdiagnosis of another condition. This involved identifying (1) patients with other diagnoses (such as rheumatoid arthritis, reactive arthritis, enteropathic arthritis, gout, fibromyalgia) and looking at the number of codes for each of the different diagnoses, (2) the order in which the different diagnosis codes were recorded, and (3) potential supporting evidence, including prescribing, of the alternative diagnosis. The PsA index date was taken as the date of the first PsA Read code. For patients with prescriptions for conventional synthetic disease-modifying antirheumatic drugs (csDMARDs) before their first PsA code, where there was no evidence of an alternative indication for the prescribing (including methotrexate prescribed on the same date as a psoriasis or dermatology code), the PsA index date was backdated to the date of the first csDMARD prescription; this applied to 17.0% of PsA patients. The index date was also backdated for patients who had received a code for psoriasis and a general nonspecific arthritis code on the same date, before their first PsA code.

Patients with psoriasis were identified based on psoriasis Read codes, which have been demonstrated to have a high validity with a questionnaire survey to general practitioners finding that 90% of psoriasis diagnoses within the study sample (n = 4634) were confirmed.[10] Patients were also identified as having psoriasis if they had received ≥ 2 prescriptions for a vitamin D analog in the absence of a psoriasis code and in the absence of an alternative indication (e.g., vitiligo); this method of identification accounted for approximately 4% of patients identified with psoriasis. The date of psoriasis diagnosis was taken as the date of the first psoriasis Read code. For patients receiving psoriasis-specific treatment (e.g., vitamin D analog, dithranol, coal tar, phototherapy) prior to the date of the first psoriasis Read code, the diagnosis date was backdated to the date of the first psoriasis-specific prescription; this applied to 12.5% of psoriasis patients.

Outcome identification. The primary outcome of interest was a diagnosis of OA of any site, identified based on Read codes. To our knowledge, no study has yet validated the recording of general OA of any site within the CPRD. However, 1 study looking at OA of the hip in the CPRD, in patients aged > 65 years, found a positive predictive value (PPV) of 88.2% (95% CI 82.4–94.0) if clinical or radiographic criteria could be used to confirm the diagnosis.[11] As our work formed part of a wider study and these cohorts have been used to study a range of other outcomes reported elsewhere,[12,13] initially all cases of OA were identified within the study cohorts, including prevalent cases where the OA diagnosis was recorded on or before the date of PsA diagnosis or the corresponding index date. Cases of OA for the incidence analyses were identified from the start of contribution of person-time to the study (i.e., after the date of PsA diagnosis or corresponding index date) following the exclusion of all prevalent cases of OA from the study population. For the secondary analyses, the outcomes of interest were site-specific OA diagnoses of the hand/wrist, hip/knee, and/or spine. For these, the first code for OA of the specific site of interest was taken as the diagnosis date, ignoring any previous general/nonsite-specific OA codes. As with the primary outcome, incident cases were identified following the exclusion of prevalent cases.

Statistical analyses. The patient characteristics at baseline for each of the study cohorts were described. The baseline prevalence of OA in each of the cohorts was calculated as a percentage, and then those prevalent cases were excluded from the numerators and denominators of the incidence calculations. The incidence of any OA per 10,000 person-years (PY) was calculated for each study cohort. Conditional Poisson regression was used to calculate crude and adjusted relative risks (RRs), comparing the risk in the PsA cohort with the risk in the general population and psoriasis cohorts. BMI as a continuous variable, based on the closest record within 3 years of the index date, was adjusted for in the model where available, as this is a known risk factor for both OA and PsA. Patients with no BMI record within 3 years of the index date were excluded from the RR calculations. No other variables were considered as potential confounders for adjustment. The same baseline prevalence and incidence analyses were repeated for the 3 secondary site-specific OA outcomes. The analysis of each site was carried out separately, and patients with records of OA of multiple sites were eligible for inclusion in all relevant sites. Two sensitivity analyses were carried out. The first excluded PsA and psoriasis patients who had their index dates backdated based on the presence of a relevant prescription, to evaluate the presence of immortal time bias. The second sensitivity analysis aimed to evaluate the effect of diagnostic uncertainty and looked at OA rates starting 1 year after the index date. Analyses were performed using R 3.3.0 (R Core Team).

RESULTS

In total, 6783 eligible incident cases of PsA were identified and matched to 27,132 patients from the general population and 27,132 psoriasis patients. Table 1 shows the baseline patient characteristics for each cohort. The median age at PsA diagnosis was 49 years (IQR 39–59). The mean duration of follow-up after the index date was approximately 5.5 years in all 3 cohorts. Data on BMI were missing for around 50% of patients in all cohorts, but where available, patients in the PsA cohort had a higher BMI than those in the psoriasis and general population cohorts. The baseline prevalence of OA ranged from 22.1% (95% CI 21.1–23.1) in the PsA cohort to 12.6% (95% CI 12.2–13.0) and 11.0% (95% CI 10.6–11.3) in the psoriasis and general population cohorts, respectively. Of the 1497 PsA patients with a prevalent OA diagnosis, 259 (17.4%) received the OA diagnosis within the 12 months before the PsA diagnosis, and 420 (28.1%) within the 2 years before. Approximately 50% of PsA patients received the OA diagnosis more than 5 years prior to their PsA diagnosis. The extent of the increased OA prevalence in the PsA cohort, compared to the general population cohort, was greater for those who received their PsA diagnosis < 40 years vs the age of 40 (Table 1).

Table 2 shows the incidence of OA in each of the 3 cohorts, which ranged from 162.4 per 10,000 PY in the PsA cohort, to 117.1 in the psoriasis cohort, and 109.2 per 10,000 PY in the general population cohort. The sensitivity analysis found that backdating the index date had little effect, with the revised

Table 1. Baseline characteristics of the PsA, psoriasis, and general population cohorts, including prevalence of OA codes at index date.

	PsA		Psoriasis		General Population	
	N	(%)	N	(%)	N	(%)
N	6783		27,132		27,132	
Sex (% male)	3327	(49.05)	13,308	(49.05)	13,308	(49.05)
Age, age, yrs, median (IQR) [a]	49	(39–59)	49	(39–59)	49	(39–59)
Follow-up postindex, yrs, mean (SD)	5.8	(4.1)	5.5	(4.1)	5.5	(4.1)
Duration of psoriasis, yrs, mean (SD)[a]	11.3	(10.9)	11.8	(10.6)	–	–
BMI[b]						
< 25.0	869	(12.8)	4190	(15.4)	4149	(15.3)
25.0–29.9	1226	(18.1)	4511	(16.6)	4257	(15.7)
30.0–34.9	844	(12.4)	2602	(9.6)	2254	(8.3)
≥ 35.0	641	(9.5)	1746	(6.4)	1252	(4.6)
Unknown	3203	(47.2)	14,083	(51.9)	15,220	(56.1)
Smoking status[b]						
Nonsmoker	3059	(45.1)	11,001	(40.6)	13,533	(49.9)
Current smoker	1605	(23.7)	8199	(30.2)	6509	(24.0)
Ex-smoker	2084	(30.7)	7480	(27.6)	6067	(22.4)
Unknown	35	(0.5)	452	(1.7)	1023	(3.8)
Baseline OA prevalence[c]	1497	(22.1)	3408	(12.6)	2974	(11.0)
< 40 yrs at index	56	(3.2)	60	(0.9)	49	(0.7)
≥ 40 yrs at index	1441	(28.6)	3348	(16.6)	2925	(14.5)

[a] On the index date. [b] Closest to and within 3 years prior to the index date. [c] ≥ 1 diagnosis code on or before the index date. OA: osteoarthritis; PsA: psoriatic arthritis.

Table 2. Incidence of OA in the PsA, psoriasis, and general population cohorts.

Any OA[a]	Cases	PY	Incidence Rate Per 10,000 PY	(95% CI)
General population	1374	125,798	109.2	(103.5–115.0)
Psoriasis	1432	122,279	117.1	(111.0–123.2)
PsA	464	28,574	162.4	(147.6–177.2)

[a] Including spondylosis. OA: osteoarthritis; PsA: psoriatic arthritis; PY: person-years.

incidence rates being 158.3, 116.6, and 110.2 per 10,000 PY for the PsA, psoriasis, and general population cohorts, respectively. The sensitivity analysis looking at OA rates starting 1 year after the index date reduced the incidence of OA in the PsA population from 162.4 (95% CI 147.6–177.2) to 140.6 (95% CI 125.5–155.7) per 10,000 PY but did not substantially change the OA incidence in the psoriasis and general population cohorts (116.9, 95% CI 110.1–123.8; and 111.6, 95% CI 125.5–155.7 per 10,000 PY, respectively).

The incidence of OA was significantly higher in the PsA cohort when compared to the general population after adjusting for BMI (RR_{adj} 1.86, 95% CI 1.62–2.14) and when compared to the psoriasis cohort (RR_{adj} 1.68, 95% CI 1.46–1.93; Table 3). When looking at OA rates starting 1 year following the index date, these reduced to RR_{adj} 1.55 (95% CI 1.31–1.83) and RR_{adj} 1.39 (95% CI 1.18–1.63; Table 3).

Table 4 shows the prevalence and incidence of site-specific OA in each of the study cohorts. Only approximately 50% of OA patients had a code stating the specific site, so these will be an underestimation; however, of those who did, it could be seen that patients in the PsA cohort were around 3 times more likely to have a diagnosis of OA of the hand/wrist before their PsA index date than those in the psoriasis or general population comparator groups were before their matched index date. Increases were seen for hip/knee OA and OA of the spine, but to a lesser extent. For site-specific OA diagnosed and recorded after the index date, a small increase was observed in OA of the hand/wrist and OA of the spine in the PsA cohort compared with the general population cohort. The prevalence and incidence rates in the general population and psoriasis cohorts were found to be similar for all sites. The sensitivity analysis looking at OA rates starting 1 year after the index date had the greatest effect on OA of the spine, but overall, the incidence rates did not substantially change (data not shown).

DISCUSSION

This large population-based study has demonstrated an 86% and 68% increased risk of an OA diagnosis, after adjusting for BMI, among patients with PsA compared to patients in the general population and patients with psoriasis, respectively. Following sensitivity analyses, the risk reduced to a 55% and 39% increased risk when looking at OA diagnoses starting at least 1 year after the index date. The study also found a higher baseline prevalence of OA diagnoses, particularly of hand/wrist OA, in patients prior to their PsA diagnosis when compared to the matched psoriasis and general population cohorts.

There is considerable variation in the literature in terms of the prevalence and incidence of OA depending on the age group reported and the definition used, whether it is radiographic, symptomatic, or self-reported.[6] When using electronic

Table 3. Risk of incident OA in patients with PsA compared with patients in the general population and patients with psoriasis.

	PsA Compared with a General Population Cohort						PsA Compared with a Psoriasis Cohort					
	Unadjusted			Adjusted[a]			Unadjusted			Adjusted[a]		
	RR	95% CI	*P*	RR	95% CI	*P*	RR	95% CI	*P*	RR	95% CI	*P*
OA	1.87	1.67–2.10	< 0.0001	1.86	1.62–2.14	< 0.0001	1.68	1.50–1.88	< 0.0001	1.68	1.46–1.93	< 0.0001
Sensitivity analysis[b]	1.51	1.33–1.73	< 0.0001	1.55	1.31–1.83	< 0.0001	1.40	1.23–1.60	< 0.0001	1.39	1.18–1.63	< 0.0001

RR: relative risk. [a]Adjusted for BMI taken as the closest entry within 3 years of the index date. [b]OA diagnoses starting 1 year after the index date. OA: osteoarthritis; PsA: psoriatic arthritis.

Table 4. Risk of site-specific OA in patients with PsA compared with patients in the general population and patients with psoriasis.

	Cases Before Index Date, N	Prevalence, %	Cases After Index Date, n	PY	Incidence Rate Per 10,000 PY	(95% CI)
Hand or wrist OA						
General population	162	0.6	133	146568	9.1	(7.5–10.6)
Psoriasis	184	0.7	143	145353	9.8	(8.2–11.5)
PsA	132	1.9	53	38506	13.8	(10.1–17.5)
Hip or knee OA						
General population	733	2.7	602	141767	42.5	(39.1–45.9)
Psoriasis	862	3.2	583	140086	41.6	(38.2–45.0)
PsA	318	4.7	184	37082	49.6	(42.5–56.8)
Spine OA[a]						
General population	1115	4.1	277	140303	19.7	(17.4–22.1)
Psoriasis	1277	4.7	299	138163	21.6	(19.2–24.1)
PsA	410	6.0	99	36382	27.2	(21.9–32.6)

[a]Including spondylosis. OA: osteoarthritis; PsA: psoriatic arthritis; PY: person-years.

healthcare data, variations in the number of OA cases identified can also result from the use of surgical or diagnostic proxies for OA diagnoses.[14,15] Our incidence figures for any OA in the general population are, however, in line with those from a UK-based study looking at the consultation incidence of OA using data from the Consultations in Primary Care Archive for a region of England.[16] Our prevalence figures in the general population are in line with a recent study using CPRD data by Swain, *et al*, but our incidence figures were higher than those reported in the same paper.[17] There are a number of possible explanations for the difference; it could in part be the result of differences in inclusion criteria or definitions, and the fact that our study included Read codes for spondylosis within the list of codes for OA. In addition, our study period ran from 2000 to 2015, while the Swain, *et al* study reported a decline in OA incidence between 1997 and 2017, with the earlier years being more in line with our overall incidence.

Only around 50% of patients with an OA diagnosis had a code that specified the actual site. In addition, our identification of site-specific OA did not take into account any prior codes for OA in general that did not specify the site. It is therefore possible that some patients identified based on their first site-specific OA code after their index date may have had a general OA code recorded prior to the index date and have been prevalent rather than incident. Given these limitations, it does not seem appropriate to make comparisons between our site-specific OA figures and those in the literature. The only comparisons that have been made are between the 3 study cohorts, and given that the methods of OA identification were the same for all cohorts, the effect should have been similar across cohorts and have a limited effect on the internal comparisons. To our knowledge, no previous study has reported on the incidence of OA in a cohort of patients newly diagnosed with PsA. A study in France has looked at the prevalence of self-reported psoriasis in a cohort of patients with hip OA aged 50–75 years and found the frequency of psoriasis to be almost twice that of the general population.[18]

The strengths of our study include its population-based nature, the large number of PsA patients, the inclusion of both a psoriasis and general population comparator, and the use of validated codes to identify PsA and psoriasis. To our knowledge, Read codes for OA in general have not been validated, and this study did not require any supporting evidence to confirm the diagnosis. It is therefore possible that in some patients, OA may have been recorded as a working diagnosis and was later ruled out. One study that aimed to determine the diagnostic accuracy of coding of OA of the hip in the CPRD in patients aged over 65 years, found a PPV of 88.2% (95% CI 82.4–94.0) if clinical or radiographic criteria could be used to confirm the diagnosis.[11] PsA and OA have long been considered 2 distinct arthropathies; however, they do have some overlapping features and symptoms,

and in certain circumstances it can be difficult to differentiate between them, particularly in the small joints of the hands or spine.[5] For example, the distribution of joints affected can be similar between OA and PsA (both arthropathies can affect the distal interphalangeal [DIP] joints of the hands. Clinically and radiologically, the osteoproliferation (bone formation) seen in PsA can be very difficult to distinguish from osteophytes (chunky bone formation) in OA. In addition, not all patients with PsA have obvious skin psoriasis at the time of PsA presentation and early joint symptoms.[19] The higher baseline prevalence of an OA diagnosis in patients with PsA, and particularly of hand/wrist OA, may in part reflect the fact that some patients were initially misdiagnosed as having OA before further investigations led to the PsA diagnosis. This could be supported by the fact that 17.4% and 28.1% of PsA patients with prevalent OA received their OA diagnosis within 1 and 2 years before the PsA diagnosis, respectively. The fact that the increase in prevalence prior to PsA diagnosis was greater in those who were diagnosed with PsA before the age of 40 years may also be evidence to support this, as OA in individuals younger than 40 years of age is uncommon.[20,21] A recent study from the United States looking at the pathway to PsA diagnosis reported that OA was the second most common misdiagnosis, occurring in 26.6% of the 203 respondents.[22] However, in our study, over 50% of PsA patients received their OA diagnosis more than 5 years before their PsA diagnosis; this suggests that misdiagnosed PsA is unlikely to account for all prevalent OA cases, although it is acknowledged that there can be a long delay in the time to PsA diagnosis.

The higher incidence of an OA diagnosis following a PsA diagnosis may suggest an increased risk of OA in patients with PsA compared to those without. This could possibly be secondary to damage from PsA or compounded by obesity. In addition to this, there may be some degree of detection and referral bias, as patients undergoing imaging and further investigations for their PsA may be more likely to have their OA identified and diagnosed than those in the general population. This is partly supported by the sensitivity analysis, where the increase in OA risk was reduced in the patients with PsA when looking more than 1 year following the index date; this suggests that the increased clinical interaction at and around the time of PsA diagnosis may provide an increased opportunity to diagnose OA. It is therefore possible that the increase in OA incidence in the PsA cohort to some extent reflects a level of undiagnosed OA within the psoriasis and general population cohorts. It is also possible, however, that in some patients, the effect of PsA on the joints is actually misdiagnosed as OA.

Within our study cohorts, there did appear to be differential levels of missing information on BMI, with 47% missing in the PsA cohort and 56% in the general population cohort. High BMI is known to be associated with an increased risk of both PsA and OA.[6,23] If the BMI data are not missing at random, it is possible that those with complete data may not be representative of the wider population and also that restricting to those who had a BMI record could have biased the results. In the UK, biologic therapy is almost entirely prescribed in secondary care and is unfortunately not captured within the CPRD. It was therefore not possible to know which patients were exposed to biologic therapy, and these exposures could have had an effect on OA incidence within these groups.

This study has demonstrated an increased risk of OA in patients with PsA compared to patients with psoriasis and those in the general population, after adjusting for BMI. Further work is required to determine whether these results reflect a true increase in the risk of symptomatic OA in patients with PsA, or whether it can be explained by differences in the opportunity for OA diagnosis between the different cohorts, an initial misdiagnosis of PsA, or a combination of both.

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