Baseline patient characteristics associated with response to biologic therapy in patients with psoriatic arthritis enrolled in the Corona Psoriatic Arthritis/Spondyloarthritis Registry

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ABSTRACT

Objectives To compare baseline characteristics between patients with psoriatic arthritis (PsA) who achieved and did not achieve minimal disease activity (MDA) with biologic therapy in the US-based Corona Psoriatic Arthritis/Spondyloarthritis Registry.

Methods Patients with PsA aged ≥18 years enrolled between March 2013 and March 2016 who were receiving biologics at enrollment, not in MDA and had ≥2 follow-up visits were included. Patients were classified as those who remained on their index biologic and achieved MDA at the second follow-up visit (MDA-achievers (MDA-A)) and those who did not (MDA non-achievers (MDA-NA)). Demographics, clinical characteristics, patient-reported outcomes and medication history were compared between groups.

Results Of 148 patients with PsA who met the inclusion criteria, 34 (23.0%) and 114 (77.0%) were classified as MDA-A and MDA-NA, respectively. At baseline, most patients (96.6%) were receiving tumour necrosis factor inhibitors, and both groups were similar in age, sex, race, medication history, enthesitis and dactylitis counts, disease duration and comorbidities. Compared with MDA-A, MDA-NA had significantly worse mean tender joint count (7.2 vs 3.4), patient-reported pain (51.2 vs 35.7), patient-reported fatigue (54.1 vs 42.4), physical function (Health Assessment Questionnaire, 1.0 vs 0.6), Bath Ankylosing Spondylitis Functional Index (5.0 vs 3.4) and Bath Ankylosing Spondylitis Disease Activity Index (4.0 vs 2.0) scores (all p<0.05). Conclusions Approximately one in four patients achieved MDA with their index biologic at the time of the second follow-up visit. Both groups were similar in several baseline demographic and clinical features; however, patients who did not achieve MDA generally had worse tender joint counts and patient-reported outcomes.

INTRODUCTION

Psoriatic arthritis (PsA) is a chronic inflammatory disease of the skin and musculoskeletal system with an estimated prevalence of 0.1%–1.0%. PsA is closely associated with psoriasis; estimates indicate that up to one-third of patients with psoriasis develop PsA. A heterogeneous condition, PsA is often characterised by axial skeleton disorders, nail and skin changes, peripheral joint inflammation, enthesitis and dactylitis,
present either in isolation or in combination with each other. In addition, patients with PsA may have other comorbidities that contribute to psychological and physical function impairment and increase their clinical and quality-of-life burden. The heterogeneity of disease can complicate treatment choices. Deeper insight into the immunopathogenesis of PsA has resulted in the introduction of novel therapeutic agents and strategies that have improved patient outcomes.

Selecting the most appropriate treatment for PsA can be challenging. International guidelines have been developed by the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis and the European League Against Rheumatism. Both groups indicate the goal of treatment should be to achieve the lowest possible levels of disease activity across all domains, with a target of remission, if possible, or minimal/low disease activity. As the first biologic therapies approved for the treatment of PsA, tumour necrosis factor inhibitors (TNFi) showed superior efficacy versus placebo in treating all manifestations of PsA, including arthritis, enthesitis, dactylitis, spondylitis, skin and nail disease, inflammatory bowel disease and uveitis. However, not all patients with PsA respond to TNFis, and some patients may have contraindications to the use of these agents; therefore, some patients may switch to agents with a different mode of action. Several non-TNF biologics and targeted synthetic disease-modifying antirheumatic drugs (DMARD) have been approved by the US Food and Drug Administration for the treatment of PsA, including monoclonal antibodies targeting interleukin (IL)-17A and IL-12/23, a T-cell costimulation modulator, an oral phosphodiesterase 4 inhibitor and an oral Janus kinase inhibitor.

Although several biologic therapies have been studied in clinical trials of PsA, results in patients enrolled in clinical trials are not necessarily generalisable to real-world clinical settings, where patients may present with a wider spectrum of disease, have multiple comorbidities and may have very different treatment histories. Investigation of the reasons behind varying responses to biologic therapy is an unmet need in the field. In this study, we sought to characterise patients with PsA based on response to their index biologic using data from the Corrona Psoriatic Arthritis/Spondyloarthritis (PsA/SpA) Registry, a large, national prospective cohort of patients with PsA and SpA in the USA.

**METHODS**

**Study design**

The Corrona PsA/SpA Registry is a large, independent, prospective observational cohort of patients with PsA or SpA. The Corrona PsA/SpA Registry database includes information about 10174 patient visits, with a mean duration of patient follow-up of 2.8 years (median, 3.1 years). As of November 2017, data on 2445 patients with PsA/SpA had been collected from 32 private and academic practice sites across 21 states in the USA, with 42 participating rheumatologists. This study included all patients with PsA enrolled in the Corrona PsA/SpA Registry between March 2013 and March 2016 who initiated or were already receiving biologics and/or targeted synthetic DMARDs (index biologic), including a monoclonal antibody targeting IL-12/23 and a phosphodiesterase 4 inhibitor, at enrolment (baseline) and had ≥2 follow-up visits after enrolment at =6-month intervals.

Patients were stratified based on their response to their index biologic and were classified based on achievement of minimal disease activity (MDA) while persisting on their index biologic therapy. Achievement of MDA occurred if a patient met ≥5 of the seven following criteria: tender joint count ≤1, swollen joint count ≤1, affected body surface area (BSA) ≤3%, patient pain on a visual analogue scale (VAS) ≤15, patient global activity VAS ≤20, Health Assessment Questionnaire (HAQ) score ≤0.5 and tender enthesal points ≤1. For the purpose of this study, patients who achieved MDA and who were still on their index biologic therapy at the time of the second follow-up visit were classified as MDA achievers (MDA-A). Patients who discontinued their index biologic at any time prior to their second follow-up visit or did not achieve MDA at their second follow-up visit were classified as MDA non-achievers (MDA-NA).

**Study assessments and outcomes**

Data were collected using provider and patient questionnaires from treating rheumatologists and patients at routine visits occurring at 6-month intervals, not to exceed two visits in any 12-month period. All assessments, including demographics, current and prior medication use, clinical characteristics and patient-reported outcomes, were collected at baseline. Specifically, information was collected for patient demographics (eg, age, sex, race, body mass index (BMI)); current and prior medication use (eg, biologics, conventional synthetic DMARDs, targeted synthetic DMARDs and prednisone); clinical characteristics including clinical features (eg, disease duration, human leukocyte antigen (HLA)-B27 test results, history of comorbidities, enthesitis, dactylitis, affected BSA and tender and swollen joint counts), laboratory measurements (eg, C-reactive protein (CRP) and erythrocyte sedimentation rate), disease activity measures (eg, physician global skin assessment, Clinical Disease Activity Index (CDAI), Bath Ankylosing Disease Activity Index (BASDAI), Disease Activity Score in 28 joints using CRP (DAS28-CRP) and Ankylosing Spondylitis Disease Activity Score using CRP (ASDAS-CRP)), physical
function measures (eg, Bath Ankylosing Spondylitis Functional Index (BASFI) and HAQ), spinal mobility measures (eg, occiput-to-wall distance, lateral lumbar flexion); and patient-reported outcomes (eg, patient-reported pain and fatigue as measured on a VAS, morning stiffness measured in minutes, quality of life using the EQ-5D-3L and Work Productivity and Activity Impairment questionnaire).

Physician-reported reasons for discontinuation or switch of the index biologic therapy (side effects, social reasons, lack of effect, doing well and other) by the second follow-up visit were described for MDA-NA. Side effects included both serious and minor as well as a fear of side effects; social reasons included cost of, preference for and frequency of treatment administration; lack of effects included inadequate response and failure to maintain initial response; doing well included achievement of remission and other similar events; and other was inclusive of all other reasons that could not otherwise be categorised. Up to three reasons for treatment discontinuation or switch could have been captured on follow-up physician questionnaires.

Data analysis
Descriptive analyses of patient demographics, clinical characteristics, patient-reported outcomes and medication history were conducted for all patients with PsA who had been enrolled in Corrona at baseline and stratified by their response to index biologic therapy. Response to a biologic was measured at the second follow-up visit from baseline, as described above.

Categorical variables (eg, sex, race, BMI, prevalence of comorbidities) were summarised using frequency counts and percentages. Continuous variables (eg, age, clinical measures and disease measures) were summarised by the counts and mean (SD). Statistical comparisons between response groups were evaluated using t-tests for continuous variables and χ² tests or Fisher’s exact tests for categorical variables. All analyses were performed using STATA V.13.

RESULTS
Patient population and baseline patient characteristics
Of the 1729 patients with PsA in the Corrona PsA/SpA Registry, 969 received a biologic at baseline (ie, patients who initiated a biologic or were already receiving a biologic at the baseline visit). Of these patients, 489 had ≥2 follow-up visits, with 322 patients who had enough information to determine MDA status both at baseline and at the second follow-up visit. The 174 patients who were in MDA at baseline were excluded, resulting in 148 patients who met the criteria for achievement/non-achievement of MDA and were included in this analysis (figure 1). At the time of the second follow-up visit (mean, 15.7 months), 34 patients (23.0%) remained on their index biologic, had achieved MDA and were categorised as MDA-A; the remaining 114 patients (77.0%) were labelled as MDA-NA. Among the 114 patients who were classified as MDA-NA, 44 had discontinued their index therapy or been switched to another biologic by the second follow-up visit. Of these 44 patients, 15 had physician-reported reasons for discontinuation/switch, which were categorised as lack of effect (n=9), side effects (n=3), social reasons (n=1), doing well (n=1) and other (n=1) (figure 2).

At baseline, MDA-A and MDA-NA were similar across all demographic and patient characteristics, including age, sex, BMI, disease duration and history of comorbidities (eg, cardiovascular disease, diabetes, cancer and serious infection) (table 1). Past and current use of biologics was similar between MDA-A and MDA-NA, with a mean number of 1.4 prior biologics used; nearly all patients (96.6%) received a TNFi as their index biologic therapy while the remaining 3.4% received an anti-IL-12/23 monoclonal antibody or an oral phosphodiesterase 4 inhibitor. Among those that initiated TNFis as their index biologic therapy, MDA-NA had a higher proportion of initiations prior to baseline compared with MDA-A (96.3% vs 84.9%; p<0.05).

Clinical characteristics
Overall, MDA-A and MDA-NA were mostly similar with regard to most clinical characteristics and measures of disease activity (table 2). There were no significant differences between MDA-NA and MDA-A for enthesitis counts (mean, 4.4 vs 2.4), dactylitis counts (mean, 2.4 vs 2.4) or swollen joint counts (mean, 2.8 vs 2.5); however, MDA-NA had a significantly higher tender joint count at baseline than MDA-A (mean, 7.2 vs 3.4; p<0.05). Despite the differences in tender joint counts, there was no between-group difference for CDAI, which incorporates tender/swollen joint counts and patient/physician global disease activity (mean, 14.0 vs 12.8 for MDA-NA and MDA-A, respectively). There were no significant differences between groups in acute-phase reactants, which is also reflected in disease activity scores that include a CRP component (mean DAS28-CRP, 3.2 vs 3.1 and mean ASDAS-CRP, 2.2 vs 1.9 for MDA-NA and MDA-A, respectively). However, MDA-NA had significantly worse spondyloarthritides-related disease activity and function at baseline than MDA-A, as assessed by BASDAI (mean, 5.0 vs 3.4) and BASFI (mean, 4.1 vs 2.0; both p<0.05).

Patient-reported outcomes
Although clinical characteristics were mostly similar in MDA-A and MDA-NA, patient-reported outcomes were significantly worse at baseline in patients who did not achieve MDA with their index biologic (table 3). There were no differences between groups in the proportion of patients who experienced ≥30 min of morning stiffness; however, compared with MDA-A, MDA-NA reported significantly worse pain (mean, 51.2 vs 35.7; p<0.05), physical function (mean HAQ, 1.0 vs 0.6) and quality of life (mean EQ-5D-3L, 0.7 vs 0.8; all p<0.05) at baseline. Work productivity and activity were reduced in MDA-NA; at baseline, MDA-NA reported significantly higher
percentages than MDA-A in impairment while working (27.4% vs 15.8%), overall work impairment (32.1% vs 17.5%) and overall activity impairment (32.9% vs 18.8%; all p<0.05). However, although MDA-NA had a higher percentage of work time missed compared with MDA-A (6.1% vs 1.4%), this was not statistically significant.

DISCUSSION
In this study, 23.0% of patients achieved MDA with their index biologic therapy at the time of their second follow-up visit. Other observational studies and clinical trials have investigated achievement of MDA with biologic therapy over similar durations of follow-up. Unlike many of these other studies, in which most patients’ prior treatments were conventional DMARDs and/or non-steroidal anti-inflammatory drugs, in our study, nearly all the patients were previously treated with biologics. Similar to our study, patients included in these studies were not in MDA at baseline. An observational study in the UK found that 60% of their patient population achieved MDA at ≥1 visit, and 34% of these achieved sustained MDA over consecutive visits for ≥12 months; 2% of their total patient population had prior TNFi therapy. An analysis of 226 TNFi-naive patients with PsA from the University of Toronto Psoriatic Arthritis Cohort found that 64% of patients achieved MDA within 1.3 years of follow-up. A prospective observational study showed that 61% of patients treated with etanercept, adalimumab or golimumab achieved MDA at 12 months, and a retrospective analysis conducted by the same study group found that 43% of patients treated with subcutaneous etanercept, adalimumab or golimumab achieved sustained MDA for at least 12 months. A subanalysis of the Adalimumab Effectiveness in Psoriatic Arthritis Trial (ADEPT) showed...
that 39% of adalimumab-treated patients achieved MDA at week 24. In RAPID-PsA, 38.8% of certolizumab pegol-treated patients achieved MDA at week 48 regardless of prior TNFi exposure; only 20% of patients in this study were previously treated with TNFis. Lastly, 42% of golimumab-treated patients achieved MDA by week 52 in the GO-REVEAL study. Other studies have looked at achievement of MDA with TNFis; however, they have either focused on early PsA cohorts, the impact of obesity or they had much shorter follow-up than Corrona.

Compared with MDA-NA, MDA-A exhibited significantly reduced mean tender joint count, patient-reported pain, patient-reported fatigue, physical function impairment, BASDAI score and BASFI score at baseline, suggesting that there may be detectable differences in our study population that contributed to failure to achieve MDA. We found that patients with oligoarthritis (≤4 swollen joints) at baseline were approximately twice as likely to achieve MDA at the second follow-up visit compared with patients who had polyarthritis, which is not surprising, as a swollen joint count of ≤1 is one of the criteria for achievement of MDA. It is also possible that worse scores of subjective components of MDA (eg, pain, patient global activity) could be driven by central sensitisation. In our study, we found that 11 patients classified as MDA-NA had fibromyalgia at baseline compared with 0 patient classified as MDA-A; however, the numbers were too small to detect any significant differences between response groups, and the binary of having fibromyalgia may not capture the relativity or degree of central sensitisation, such as pain levels or tender joint counts. Additionally, since the majority of the patients in our study were previously treated with biologics, which may have included other TNFis, it is possible that some patients were already receiving their second, third or more TNFi at baseline and may have been more refractory to their index biologic than other patients who had received fewer biologics (and/or for a shorter duration) or who were biologic naive. Prolonged exposure to TNFis has been linked to attenuated response rates with each successive TNFi used. Although MDA-A and MDA-NA were similar with regard to the mean number of prior biologics used, we did not conduct subgroup analyses based on the number and type of previous biologics used in MDA-A versus MDA-NA. We also did not have information on reasons for discontinuation of biologics prior to enrolment in the Corrona Registry, and therefore were unable to assess whether achievement of a response was influenced by intolerance to or adverse events with previous treatment. Despite these potential differences, MDA-A and MDA-NA were similar with regard to age, sex, race, disease duration, prevalence of comorbidities, multiple clinical characteristics (eg, enthesitis and dactylitis counts) and prior/current medications at baseline.

Within the last few years, studies have examined predictors of response to biologic therapies in patients with PsA, with much of the emphasis on identification of genetic or protein biomarkers. A meta-analysis of clinical trials and observational studies concluded that although numerous factors were associated with better response to TNFis in patients with ankylosing spondylitis (eg, younger age, male sex, high baseline BASDAI and CRP, low baseline BASFI and HLA-B27 positivity), no predictors of response were identified for patients with PsA. In contrast, an analysis of the infliximab and golimumab clinical trials demonstrated that increased CRP levels at baseline were predictive of improved therapeutic response at the time point specified by the primary end point; in our analysis, MDA-A had a higher CRP level at baseline than MDA-NA, but this difference was not significant. In a separate study of 97 patients with PsA who received treatment with etanercept for ≥3 months, two different polymorphisms were identified from genomic DNA extracted from buccal epithelial cells that were predictive of poor response to treatment (improvement in Psoriatic Arthritis and Severity Index ≥75% after 12 months)
Table 1  Patient characteristics and medication history of MDA-A versus MDA-NA at baseline

| Characteristic* | Overall n=148 | MDA-A† n=34 | MDA-NA n=114 | P values |
|-----------------|--------------|-------------|--------------|----------|
| Age (years)     | 54.7 (11)    | 54.8 (12.8) | 54.7 (10.5)  | 0.99     |
| Female, n (%)   | 80 (54.4)    | 15 (44.1)   | 65 (57.5)    | 0.17     |
| Race, n (%)     |              |             |              | 0.44     |
| White           | 136 (95.1)   | 32 (94.1)   | 104 (95.4)   |          |
| Asian           | 3 (2.1)      | 1 (2.9)     | 2 (1.8)      |          |
| Pacific Islander| 1 (0.7)      | 1 (2.9)     | 0 (0.0)      |          |
| Mixed race      | 2 (1.4)      | 0 (0.0)     | 2 (1.8)      |          |
| Other           | 1 (0.7)      | 0 (0.0)     | 1 (0.9)      |          |
| BMI (kg/m²)     | 33.1 (7.6)   | 32.5 (7.1)  | 33.2 (7.8)   | 0.60     |
| BMI (in kg/m²) classifications, n (%) |          |             |              | 0.76     |
| Normal/underweight (<25.0) | 13 (9.0) | 2 (6.1) | 11 (9.8) |          |
| Overweight (25.0 to <30.0) | 50 (34.5) | 13 (39.4) | 37 (33.0) |          |
| Obese (≥30.0)   | 82 (56.6)    | 18 (54.6)   | 64 (57.1)    |          |
| Disease duration (years) | 11.8 (10.1) | 11.6 (8.2) | 11.9 (10.6) | 0.48     |
| HLA-B27 test result on record, n (%) | 34 (23.0) | 8 (23.5) | 26 (22.8) | 0.08     |
| Positive test result (in patients with test results on record), n (%) | 9 (26.5) | 0 (0.0) | 9 (34.6) |          |
| History of comorbidities, n (%) |          |             |              |          |
| Cardiovascular disease‡ | 92 (62.2) | 20 (58.8) | 72 (63.2) | 0.65     |
| Diabetes mellitus | 19 (12.8) | 5 (14.7) | 14 (12.3) | 0.71     |
| Any cancer§      | 15 (10.1)    | 2 (5.9)     | 13 (11.4)    | 0.52     |
| Fibromyalgia     | 11 (7.4)     | 0 (0.0)     | 11 (9.7)     | 0.07     |
| Serious infections¶ | 7 (4.7) | 1 (2.9) | 6 (5.3) | 1.00     |
| Prior medication use, n (%) |          |             |              |          |
| Biologic therapy | 140 (94.6) | 30 (88.2) | 110 (96.5) | 0.08     |
| Mean (SD) number of prior biologics | 1.4 (0.8) | 1.4 (1.0) | 1.4 (0.7) | 0.74     |
| Prior biologics, n (%) |          |             |              | 0.16     |
| 0               | 8 (5.4)      | 4 (11.8)    | 4 (3.5)      |          |
| 1               | 94 (63.5)    | 19 (55.9)   | 75 (65.8)    |          |
| 2+              | 46 (31.1)    | 11 (32.4)   | 35 (30.7)    |          |
| csDMARD therapy | 113 (76.4) | 26 (76.5) | 87 (76.3) | 0.99     |
| Prednisone      | 17 (11.5)    | 3 (8.8)     | 14 (12.3)    | 0.76     |
| Current biologic/tsDMARD use, n (%)** |          |             |              | 1.00     |
| TNFis           | 142 (96.6)  | 33 (97.1)   | 109 (96.5)   |          |
| Initiations at baseline | 9 (6.3) | 5 (15.1) | 4 (3.7) | 0.03     |
| Initiations prior to baseline | 133 (93.7) | 28 (84.9) | 105 (96.3) |          |
| Other biologics/tsDMARD | 6 (3.4) | 1 (2.9) | 5 (3.5) |          |
| Current prednisone use, n (%) | 8 (5.4) | 1 (2.9) | 7 (6.1) | 0.68     |

*All values were calculated based on available data and are presented as ‘mean (SD)’ unless otherwise stated. All variables had <20% missing data.
†MDA-A were classified as those patients who remained on their index biologic and achieved MDA at the second follow-up visit. MDA was determined to have been achieved if a patient met ≥5 of the seven following categories [25]: tender joint count ≤1, swollen joint count ≤1, BSA ≤3%, patient pain VAS ≤15, patient global activity VAS ≤20, HAQ score ≤0.5 and tender entheseal points ≤1.
‡Combined histories of myocardial infarction, acute coronary syndrome, coronary artery disease, congestive heart failure, hypertension, hyperlipidaemia, peripheral artery disease, cardiac revascularisation procedure, ventricular arrhythmia, cardiac arrest, unstable angina, stroke, transient ischaemic attack, pulmonary embolism, carotid artery disease, deep vein thrombosis or other cardiovascular event.
§Excludes non-melanoma of the skin.
¶Includes those infections that lead to hospitalisation or intravenous antibiotics: joint/bursa, cellulitis, sinuses, diverticulitis, sepsis, pneumonia bronchitis, gastroenteritis, meningitis, urinary tract infection, upper respiratory infection or infection of other specified site.
**Index biologic and tsDMARD therapies included adalimumab, apremilast, certolizumab pegol, etanercept, golimumab, infliximab and ustekinumab.
BMI, body mass index; BSA, body surface area; csDMARD, conventional synthetic disease-modifying antirheumatic drug; HAQ, Health Assessment Questionnaire; HLA, human leukocyte antigen; MDA, minimal disease activity; MDA-A, minimal disease activity achievers; MDA-NA, minimal disease activity non-achievers; TNFi, tumour necrosis factor inhibitor; tsDMARD, targeted synthetic disease-modifying antirheumatic drug; VAS, visual analogue scale.
As with any observational study, there are limitations to our analysis. This was a descriptive analysis of the patients who subsequently achieved or did not achieve MDA with their index biologic therapy, using stringent criteria for a target of response (persistence on index biologic therapy for ≥2 follow-up visits and achievement of MDA).

We chose MDA as a conservative estimate of response, with the understanding that there are likely patients who did not achieve MDA in this analysis but who are still satisfied with their treatment. The criteria selected for inclusion meant that only one-half of patients who received a biologic at baseline had ≥2 follow-up visits, and not everyone had enough clinical information to determine MDA status at baseline and the time of the second follow-up visit, limiting the sample size of the study. We did consider including all patients with ≥1 follow-up visit instead of ≥2 follow-up visits, but this did not increase the number of patients appreciably. The overall sample size was too small to only include those patients who initiated a biologic at the time of the enrolment visit. To compensate, the study population consisted of patients who either initiated a biologic at the enrolment visit or were already receiving a biologic therapy at time of enrolment. Among patients who initiated TNFis, a significantly higher proportion of MDA-NA had initiated their index TNFi prior to baseline compared with MDA-A; therefore,

### Table 2  Disease measures of MDA-A versus MDA-NA at baseline

| Characteristic                      | Overall n=148 | MDA-A n=34 | MDA-NA n=114 | P values  |
|------------------------------------|--------------|-----------|-------------|---------|
| Enthesitis, n (%)                  | 47 (31.8)    | 7 (20.6)  | 40 (35.1)   | 0.11    |
| SPARCC Enthesitis Index (1–16)     | 4.2 (3.1)    | 2.4 (0.5) | 4.4 (3.3)   | 0.20    |
| Dactylitis, n (%)                  | 20 (13.5)    | 6 (17.6)  | 14 (12.3)   | 0.42    |
| Dactylitis count (1–20)            | 2.4 (1.7)    | 2.4 (2.6) | 2.4 (1.3)   | 0.21    |
| BSA, % affected                    | 7.7 (13.2)   | 5.6 (8.1) | 8.3 (14.4)  | 0.31    |
| Physician global skin assessment, n (%) | 36 (24.5)    | 10 (29.4) | 26 (23.0)   | 0.80    |
| CDAI                               | 13.7 (8.2)   | 12.8 (6.8) | 14.0 (8.5) | 0.44    |
| Tender joint count (68)            | 6.3 (10.1)   | 3.4 (5.3)  | 7.2 (11)    | 0.02    |
| Swollen joint count (66)           | 2.7 (3.7)    | 2.5 (3.5)  | 2.8 (3.7)   | 0.71    |
| Oligoarthritis, n (%)†             | 114 (78.1)   | 29 (87.9)  | 85 (75.2)   | 0.12    |
| BASDAI (0–10)                      | 4.6 (2.3)    | 3.4 (2.1)  | 5.0 (2.3)   | 0.001   |
| BASFI (0–10)                       | 3.6 (2.5)    | 2.0 (1.9)  | 4.1 (2.4)   | <0.001  |
| Spinal mobility measures           |              |           |             |         |
| Occiput-to-wall distance (cm)      | 1.2 (2.1)    | 0.9 (1.8)  | 1.3 (2.3)   | 0.80    |
| Lateral lumbar flexion (cm)        |              |           |             |         |
| Left                               | 16.5 (8.9)   | 15.5 (8.2) | 16.9 (9.3)  | 0.97    |
| Right                              | 16.4 (8.5)   | 17.2 (5.8) | 16.1 (9.4)  | 0.20    |
| DAS28-CRP                          | 3.2 (0.9)    | 3.1 (0.9)  | 3.2 (0.9)   | 0.73    |
| ASDAS-CRP                          | 2.2 (0.9)    | 1.9 (0.8)  | 2.2 (1.0)   | 0.14    |
| CRP (mg/L)                         | 4.3 (10.9)   | 4.9 (10.3) | 4.1 (11.1)  | 0.60    |
| ESR (mm/hour)                      | 17.1 (15.8)  | 16.0 (14.8)| 17.5 (16.1)| 0.88    |

*All values were calculated based on available data and are presented as ‘mean (SD)’ unless otherwise stated. All variables had <20% missing data except for SPARCC Enthesitis Index (n=37/47), ASDAS-CRP (n=85), CRP (n=94) and ESR (n=92).
†Oligoarthritis was defined as ≤4 swollen joints.

ASDAS-CRP, Ankylosing Spondylitis Disease Activity Score using C-reactive protein; BASDAI, Bath Ankylosing Spondylitis Disease Activity Index; BASFI, Bath Ankylosing Spondylitis Functional Index; BSA, body surface area; CDAI, Clinical Disease Activity Index; CRP, C-reactive protein; DAS28-CRP, Disease Activity Score in 28 joints using C-reactive protein; ESR, erythrocyte sedimentation rate; MDA, minimal disease activity; MDA-A, minimal disease activity achievers; MDA-NA, minimal disease activity non-achievers; SPARCC, Spondyloarthritis Research Consortium of Canada; VAS, visual analogue scale.
some of the baseline measurements and assessments in this analysis may be affected by the duration of biologic use prior to enrolment (eg, clinical and disease activity measures and patient-reported outcomes). We also do not have information on the reasons for why biologic therapy was initiated, so there may be subgroups of patients in the study population who may or may not be more likely to achieve MDA with their index biologic. More data on the prescribing physicians, as well as the patient population, are needed to help address this question. Any differences between MDA-A and MDA-NA that were observed at registry enrolment should not be interpreted as predictive of any specific outcome. Future longitudinal analyses with larger sample sizes are needed to apply more rigorous statistical models that adjust for confounding variables (eg, fibromyalgia, the presence of oligoarthritis vs polyarthritis and prior biologic use at baseline) and determine which covariates contribute to achievement of MDA with biologic therapies.

This analysis of patients with PsA in the Corrona Registry is the first to describe baseline characteristics of patients who do not achieve they are receiving optimal care and management of their disease. Early recognition of patients who do not achieve a treatment target, such as MDA, may prevent delays in clinical response and unnecessary treatment costs. Additional studies with larger sample sizes are needed to examine predictors of biologic response in patients with PsA.

Table 3  Patient-reported outcomes in MDA-A versus MDA-NA at baseline

| Characteristic* | Overall n=148 | MDA-A n=34 | MDA-NA n=114 | P values |
|-----------------|---------------|------------|-------------|---------|
| Patient pain (VAS 0–100) | 47.6 (25.3)  | 35.7 (23.4) | 51.2 (24.8) | 0.002  |
| Patient-reported fatigue (VAS 0–100) | 51.5 (25.7)  | 42.4 (29.4) | 54.1 (24.1) | 0.05   |
| Morning stiffness, n (%) | | | | |
| Yes | 134 (92.4) | 27 (84.4) | 107 (94.7) |  
| <30min | 21 (15.7) | 5 (18.5) | 16 (15.0) | 0.65   |
| ≥30min | 113 (84.3) | 22 (81.5) | 91 (85.0) |  
| HAQ-DI (0–3) | 0.9 (0.6) | 0.6 (0.5) | 1.0 (0.6) | 0.001  |
| HAQ-S (0–3) | 0.9 (0.6) | 0.6 (0.5) | 1.0 (0.6) | <0.001 |
| EQ-5D-3L (0–1) | 0.7 (0.2) | 0.8 (0.1) | 0.7 (0.2) | <0.001 |
| WPAI | | | | |
| % Work time missed | 4.7 (11.6) | 1.4 (5.9) | 6.1 (13.1) | 0.08   |
| % Impairment while working | 24 (21) | 15.8 (20.8) | 27.4 (20.4) | 0.01   |
| % Overall work impairment | 27.5 (24) | 17.5 (22.5) | 32.1 (23.5) | 0.01   |
| % Activity impairment | 28.8 (23.9) | 18.8 (22.7) | 32.9 (23.4) | 0.01   |

*All values were calculated based on available data and are presented as “mean (SD)” unless otherwise stated. All variables had <20% missing data except for WPAI (n (range)=54–68).

HAQ-DI, Health Assessment Questionnaire Disability Index; HAQ-S, Health Assessment Questionnaire for Spondyloarthropathies; MDA-A, minimal disease activity achievers; MDA-NA, minimal disease activity non-achievers; VAS, visual analogue scale; WPAI, Work Productivity and Activity Impairment questionnaire.

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