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ORIGINAL RESEARCH

Impact of psoriatic arthritis and comorbidities on ustekinumab outcomes in psoriasis: a retrospective, observational BADBIR cohort study

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ABSTRACT

Objectives Psoriasis and psoriatic arthritis (PsA) are independently associated with comorbidities, including obesity and metabolic syndrome, which may impact treatment outcomes. This study aimed to assess baseline differences between patients with plaque psoriasis alone and those with concomitant PsA, and to investigate the impact of these characteristics on ustekinumab (UST) persistence and outcomes.

Methods 9057 patients receiving UST or conventional systemic disease-modifying antirheumatic drugs were selected from the British Association of Dermatologists Biologic and Immunomodulators Register. The psoriasis and PsA cohorts were compared at baseline. Time to discontinuation during 10-year follow-up was assessed using multivariable Cox regression and Kaplan–Meier analyses, stratifying for interacting covariates and PsA status. Generalised linear mixed models assessed the impact of baseline characteristics on Psoriasis Area and Severity Index (PASI) and Dermatology Life Quality Index over time.

Results Greater comorbidity burden, including hypertension, diabetes, obesity and depression, and greater inability to work were observed in the PsA cohort than in the psoriasis cohort. PsA (HR 1.98), female sex (HR for male sex 0.72) and depression (HR 1.21) were associated with shorter UST persistence. PsA showed a differential association with UST persistence by PASI strata and prior biologic exposure. Quality of life was negatively impacted by depression and PsA.

Conclusions The negative impact of comorbidities on treatment persistence identified in this study emphasises the need for patient-centric, multidisciplinary care in screening for and managing comorbidities in psoriasis and PsA treatment. Psychological support and lifestyle management of modifiable risk factors, including obesity, should be considered.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Patients with psoriasis and psoriatic arthritis (PsA) experience comorbidities that may impact treatment outcomes.

WHAT THIS STUDY ADDS

⇒ Use of the British Association of Dermatologists Biologic and Immunomodulators Register allows for comparison of patients with psoriasis and concomitant PsA with those who have psoriasis alone in a single UK population.

⇒ A greater comorbidity burden was observed in patients with concomitant PsA compared with psoriasis alone, and this was also associated with shorter ustekinumab (UST) persistence, with the greatest negative impact of PsA on persistence observed in patients with a low burden of skin disease.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study highlights the need for patient-centric care in psoriatic disease; lifestyle and weight advice should form part of careful comorbidity management.

⇒ The clear impact of psoriatic disease on quality of life and the higher likelihood of depression in patients treated with UST suggests psychological support should also be considered in these patients.

INTRODUCTION

Psoriatic arthritis (PsA) is a chronic inflammatory form of arthritis associated with psoriasis. Up to 42% of patients with psoriasis develop PsA during the course of the disease,[1] typically within 10 years of psoriasis onset.[2] Both skin and joint symptoms contribute to the burden of psoriatic disease, and more severe skin symptoms have been associated with poorer quality of life (QoL), more severe disability and lower work productivity than less severe skin symptoms.[3 4]

Comorbidities, particularly cardiovascular, metabolic and mental health disorders, have been found to be more common in patients with PsA than controls.[5 6] In a meta-analysis of observational studies, both cardiovascular and cerebrovascular risks were increased in patients with PsA compared with the general population, including risk of myocardial

infarction, cerebrovascular events and heart failure.[7] PsA disease activity has also been associated with cardiovascular risk as measured by Systematic Coronary Risk Evaluation following carotid ultrasound assessment.[8] Obesity, another such comorbidity commonly associated with PsA, is a modifiable risk factor for the development of PsA and affects the persistence of biologics, a core treatment option for patients with moderate-to-severe disease.[9–13] Several emerging reports consider the impact of sex on PsA outcomes. Although PsA affects men and women equally, women are more likely to develop polyarticular disease and less likely to respond favourably to antitumour necrosis factor (TNF) biologics, and experience greater inability to work and limitations in daily functioning than men.[14 15] Owing to the heterogeneity in manifestations of psoriatic disease, which can include joint and axial involvement, skin disease, enthesitis and dactylitis, as well as the burden of comorbidities, a multidisciplinary approach to care is often required. Shared decision making with the patient is also important for agreeing on and achieving treatment goals.[16–18]

Registry data can complement clinical trial results, as they are often more generalisable to clinical practice scenarios with regard to patient characteristics and disease outcomes.[19 20] The use of real-world evidence is emphasised by the US Food and Drug Administration as part of regulatory decision making as well as ongoing safety monitoring.[21]

One such registry for PsA is the British Society for Rheumatology Psoriatic Arthritis Register. Although recruitment has started, there is currently a lack of analysable registry data specific to patients with PsA in the UK. An alternative database for exploring outcomes in PsA is the British Association of Dermatologists Biologic and Immunomodulators Register (BADBIR). This was established to evaluate the long-term safety of biologics for the treatment of psoriasis. Owing to the large number of patients with psoriasis who have PsA concomitantly, use of this database allows for comparison of patients with psoriasis and concomitant PsA with those who have psoriasis alone in a UK population.[22]

This real-world study of patients with psoriasis aimed to evaluate whether there is an association between the presence of PsA and comorbidity burden, inability to work and treatment outcomes, including ustekinumab (UST) persistence and patient-reported outcomes.

METHODS

Data source and population

This was a retrospective, observational cohort study using data from the BADBIR. Dermatologists are encouraged to register all patients who fulfil the BADBIR inclusion criteria and are receiving treatment for psoriasis in the UK. Patients included in this study were enrolled into the BADBIR as part of two cohorts: those receiving biologics and those receiving conventional synthetic disease-modifying antirheumatic drugs (csDMARDs), including methotrexate or ciclosporin.[22] In the UK, csDMARDs are frequently used to treat patients with psoriasis who do not respond to topical therapies, and lack of response to csDMARDs is usually required before biologic treatment initiation.[23] At the time of BADBIR enrolment, information on the type of psoriasis; involvement of nails, flexures or scalp; year of psoriasis onset; family history; and presence of PsA are recorded.[22] Data included in the registry are collected by a trained healthcare professional, with PsA recorded by response to the question 'Has the patient a diagnosis by a rheumatologist of inflammatory arthritis?'.

The BADBIR was identified as a suitable database to meet the objectives of this study due to the reporting of PsA as a comorbidity, which allows assessment of patients with psoriasis and concomitant PsA versus patients with psoriasis alone.[22] Patients were first enrolled in July 2009, and the cut-off date for this analysis was October 2020. Patients included in this study had a diagnosis of plaque psoriasis and were receiving either UST as their biologic treatment (commencing or switching from another biologic in the previous 6 months) or csDMARDs (commencing or switching from another csDMARD in the previous 6 months). Psoriasis Area and Severity Index (PASI) and Dermatology Life Quality Index (DLQI) scores are collected at baseline and every 6 months until 36 months. PASI score is then collected yearly throughout time in the BADBIR.

Comparison of baseline characteristics

Comparisons were made between patients with psoriasis alone (psoriasis cohort) versus those with psoriasis and concomitant PsA (PsA cohort). To account for differences in disease severity and stage of treatment in the UST and csDMARD groups at baseline, comparisons were made within each treatment group. Time of BADBIR enrolment aligned with time of treatment initiation for most patients receiving UST (99%). Baseline characteristics, including obesity, smoking history, employment status, comorbidities (diabetes, hypertension, myocardial infarction and depression (the year and date of these pre-existing conditions are recorded at BADBIR enrolment)) and PASI score, were evaluated. All pre-existing comorbidities were captured at the time of enrolment into the BADBIR. The strength of association between variables at baseline was assessed using ORs, and 95% CIs were generated; two-sided p values were obtained using Fisher's exact test in the case of categorical variables and the Mann–Whitney U test in the case of continuous variables.

Analysis of time to discontinuation (TTD) of UST

TTD was defined as the time from treatment start date until treatment stop date and was censored at the last follow-up date in the registry without a recorded treatment stop date. Kaplan–Meier analysis was performed to estimate the probability of discontinuation over time, and log-rank test was used to compare persistence among groups. TTD was assessed using multivariable and univariate analyses.

Table 1 Baseline characteristics of patients in the BADBIR treated with UST versus csDMARD

Characteristic*	UST, psoriasis	UST, PsA	csDMARD, psoriasis	csDMARD, PsA
Patients	2720 (82)	593 (18)	5208 (91)	536 (9)
Mean age (years) (SD)	45.3 (14.1)	49.0 (12.2)	43.8 (14.6)	46.8 (13.3)
Sex, female	43.8	41.6	37.9	52.4
Psoriasis duration (years)				
<5	165 (6.1)	20 (3.4)	919 (17.6)	62 (11.6)
5–20	321 (11.8)	41 (6.9)	874 (16.8)	75 (14.0)
>20	1681 (61.8)	348 (58.7)	3401 (65.3)	397 (74.1)
Smoking status, no/yes	1881 (69.2)/687 (25.3)	413 (69.6)/141 (23.8)	3106 (59.6)/1631 (31.3)	356 (66.4)/135 (25.2)
Prior biologic exposure, no/yes	2169 (79.7)/551 (20.3)	312 (52.6)/281 (47.4)	NA	NA
PASI score at baseline, mean (SD)	13.14 (7.71)	14.82 (8.26)	14.83 (7.61)	15.67 (7.97)

Missing data were reported for psoriasis duration, smoking status and PASI score at baseline. Psoriasis duration: 20% for UST, psoriasis; 31% for UST, PsA; <1% for csDMARD, psoriasis; and <1% for csDMARD, PsA. Smoking status: 6% for UST, psoriasis; 6% for UST, PsA; 9% for csDMARD, psoriasis; 8% for csDMARD, PsA. PASI score at baseline: 3% for UST, psoriasis; 4% for UST, PsA; 4% for csDMARD, psoriasis; and 1% for csDMARD, PsA.
*Data are n (%) unless otherwise stated.
BADBIR, British Association of Dermatologists Biologic and Immunomodulators Register; csDMARD, conventional synthetic disease-modifying antirheumatic drug; NA, not available; PASI, Psoriasis Area and Severity Index; PsA, psoriatic arthritis; UST, ustekinumab.

HRs, CIs and p values (Wald test) were generated using multivariable Cox proportional hazards regression for associations between PsA status, depression and TTD, adjusting for obesity (defined as a body mass index (BMI) of ≥30 kg/m^2), sex and PASI strata. This was also the case for interactions between TTD, PsA status and prior biologic exposure, as well as TTD, PsA status and PASI strata. Subsequently, a Kaplan–Meier analysis was performed in which patients were stratified by PsA diagnosis and PASI score at baseline to demonstrate the statistical interaction between PsA status and PASI strata, PsA status and prior biologic exposure, and TTD.

Longitudinal analysis of PASI and DLQI scores

Longitudinal associations between baseline variables were generated using generalised linear mixed models using third-term polynomial models to evaluate the relationship between time and outcome. Random effect terms were used to account for within-subject variability. P values were generated for fixed effects by the Walk score test; random effect variances and continuous predictors were tested using the likelihood ratio test. This analysis included time on UST or csDMARD treatment, the latter of which included switching between csDMARDs.

Patient and public involvement

There was no involvement of patients or the public in the design of this study.

RESULTS

Comparison of disease burden in patients with psoriasis alone and those with concomitant PsA

Baseline characteristics are listed in table 1. csDMARDs prescribed included methotrexate, ciclosporin, acitretin, psoralen and ultraviolet A (PUVA), hydroxycarbamide and fumaric acid esters. The cohort of patients with psoriasis receiving csDMARDs had a slightly higher proportion of men (62.1%) and lower mean age (43.8 years) than the other groups. Of those receiving UST, a greater proportion of patients in the PsA cohort (281/593, 47.4%) had prior biologic experience than those with psoriasis alone (551/2720, 20.3%).

Comparison of baseline characteristics showed that patients in the PsA cohort had a greater comorbidity burden than patients with psoriasis alone. Patients in the PsA cohort initiating either UST or a csDMARD had a higher prevalence of diabetes, obesity and hypertension than those with psoriasis alone (figure 1).

Ability to work was also notably lower in the PsA cohort compared with the psoriasis cohort (figure 1); a greater proportion of patients in the PsA cohort in both treatment groups were unable to work than in the psoriasis cohort (online supplemental figure S1).

Patients in the PsA cohort receiving UST were more likely to have a diagnosis of depression than those in the psoriasis cohort; this was not observed in the csDMARDs cohort (figure 1).

Association between PsA and TTD of UST

Median follow-up time for the UST persistence cohort was 2.2 years (mean 2.6, SD 1.97).

In a multivariable analysis using Cox proportional hazards regression, treatment persistence with UST was assessed for all patients with psoriasis with the primary aim of determining the effect of PsA status on treatment discontinuation. The effects of prior biologic experience, baseline PASI scores and comorbidities of interest (including depression and obesity) were also evaluated. The model also assessed differential effects of the presence/absence of PsA on TTD with regard to other clinical covariates, including strata of PASI score at baseline and prior biologic experience. The median TTD was

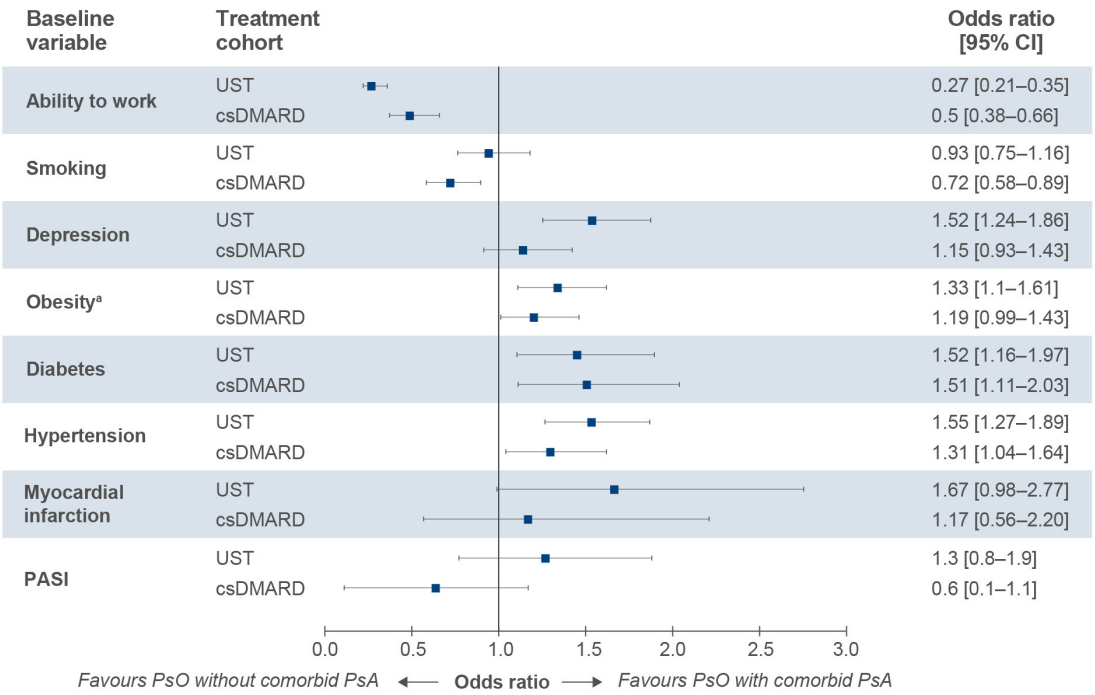

Figure 1 Unadjusted ORs of baseline variables by treatment cohort and presence of concomitant PsA. [a]Obesity is defined as a body mass index of ≥30 kg/m^2. csDMARD, conventional synthetic disease-modifying antirheumatic drug; PASI, Psoriasis Area and Severity Index; PsA, psoriatic arthritis; PsO, psoriasis; UST, ustekinumab.

shorter in patients with PsA than in those with psoriasis alone and in patients with depression versus no depression (figure 2). Men were also shown to have a lower risk of discontinuation than women (figure 2). Prior biologic experience increased the risk of discontinuation in comparison with biologic-naïve patients in the PsA cohort. The effect size of prior biologics was higher in those with psoriasis and PsA than in those with psoriasis alone (figure 2).

Regarding the effect of baseline skin severity, a PASI score of >3 at baseline was associated with an increased probability of discontinuation versus PASI score of ≤3. When considering both PASI score and the presence of PsA, more severe skin symptoms at baseline were

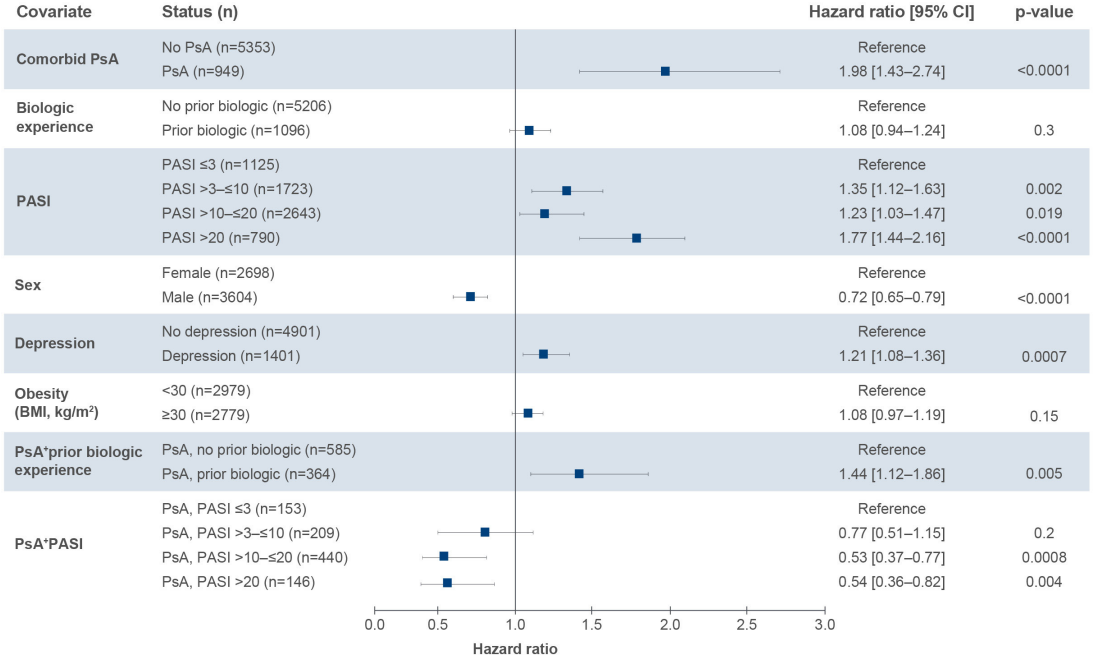

Figure 2 Multivariable Cox regression model for TTD in patients treated with UST. BMI, body mass index; PASI, Psoriasis Area and Severity Index; PsA, psoriatic arthritis; TTD, time to discontinuation; UST, ustekinumab.

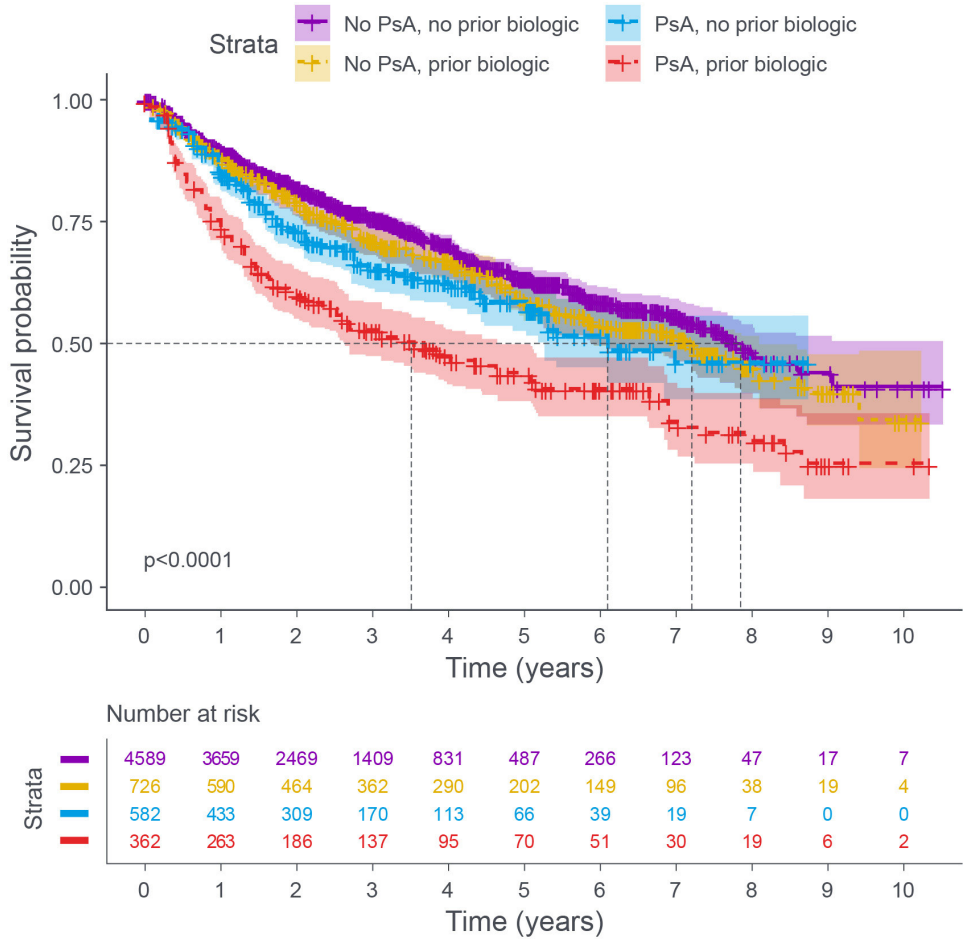

Figure 3 Drug survival in the UST cohort by comorbid PsA and prior biologic experience. Patients with plaque psoriasis treated with UST were stratified by comorbid PsA at baseline and prior biologic experience. P value for drug survival probability across all groups. The shaded area indicates 95% CI. PsA, psoriatic arthritis; UST, ustekinumab.

associated with a greater TTD, suggesting PsA has a greater negative impact on UST persistence in patients with less severe skin symptoms at baseline. The effect size of PsA was higher in patients with a low PASI score than in those with a high PASI score (figure 2).

In a Kaplan–Meier analysis stratified by PsA status at baseline and prior biologic exposure status, biologic-experienced patients in the PsA cohort had a significantly higher likelihood of discontinuation than any other group, with a median TTD of 3.51 years vs 6.09 years for biologic-naïve patients in the PsA cohort, 7.20 years for biologic-experienced patients with psoriasis alone and 7.85 years for biologic-naïve patients in the psoriasis alone cohort (p<0.0001) (figure 3).

In a further Kaplan–Meier analysis, the median TTD in patients with psoriasis alone and PASI scores of ≤3 was 8.11 years vs 5.27 years for patients with psoriasis and PsA. In patients with PASI scores of >20, the median TTD for patients with psoriasis alone was 5.33 years vs 3.89 years for patients with psoriasis and PsA (figure 4). This effect is illustrated in a Kaplan–Meier curve with bivariate stratification by PASI and PsA status.

Association between baseline characteristics and PASI and DLQI scores

Longitudinal endpoints were assessed in both patient cohorts. Using generalised linear mixed modelling of PASI over time, male sex, PsA and obesity showed small but significant fixed effects on increased PASI score regardless of the assessed time point in both the csDMARD and UST cohorts (figure 5).

Depression at baseline was associated with increased DLQI score irrespective of the time point assessed in both the csDMARD and UST treatment groups. Additionally, in the UST cohort, PsA and female sex were associated with increased DLQI scores, regardless of the assessed time point (figure 5).

Irrespective of other factors, such as depression, PsA status, obesity and gender, patients treated with UST had consistently lower PASI scores and DLQI scores than patients receiving csDMARDs, regardless of the assessed time point (figure 5).

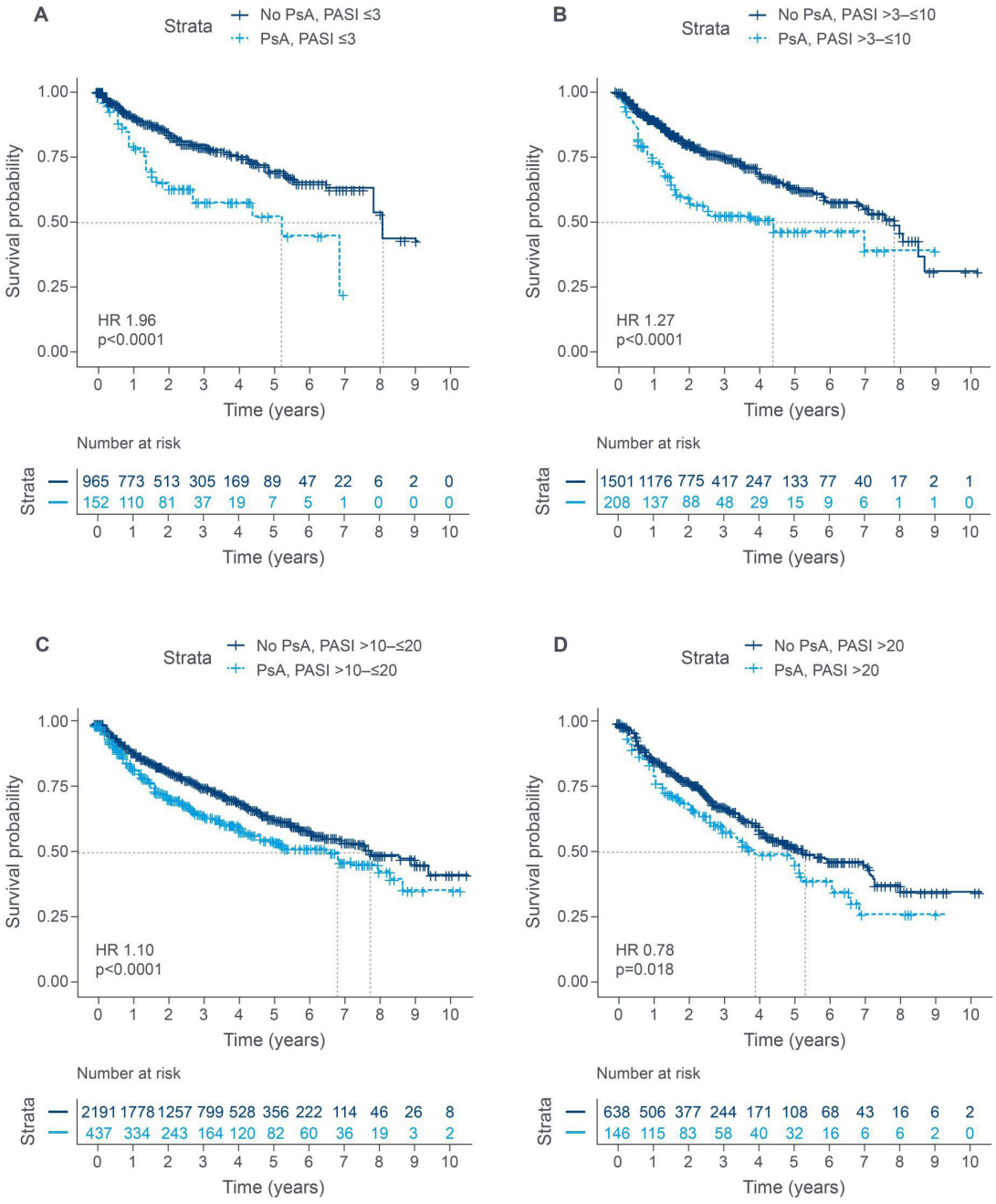

Figure 4 Drug survival in the UST cohort by comorbid PsA and PASI score. Patients with plaque psoriasis treated with UST were stratified by the presence of comorbid PsA and PASI scores of (A) ≤3, (B) >3 to ≤10, (C) >10 to ≤20 and (D) >20. PASI, Psoriasis Area and Severity Index; PsA, psoriatic arthritis; UST, ustekinumab.

DISCUSSION

In this retrospective cohort study, patients with psoriasis and concomitant PsA were compared with those with psoriasis alone. At baseline, a greater degree of comorbidity burden, including hypertension, diabetes, obesity and depression, as well as greater inability to work, was observed in the PsA cohort than in the psoriasis cohort. Additionally, psoriasis with PsA was associated with a shorter TTD on UST than psoriasis alone.

In other studies, results demonstrating the impact of PsA on persistence have been mixed. In the Psoriasis Longitudinal Assessment and Registry, UST demonstrated better persistence than anti-TNF agents; however, the presence of PsA as a comorbidity was not associated with discontinuation.[9] By contrast, in an earlier analysis of data from the BADBIR, longer UST survival was seen in the cohort of patients without PsA compared with those with PsA.[24] However, a systematic meta-analysis of psoriasis studies found that PsA positively predicted persistence in a combined biologics group.[10]

In this analysis, female sex and depression were associated with a reduced TTD. This aligns with two real-world studies of patients with PsA in Denmark and Germany

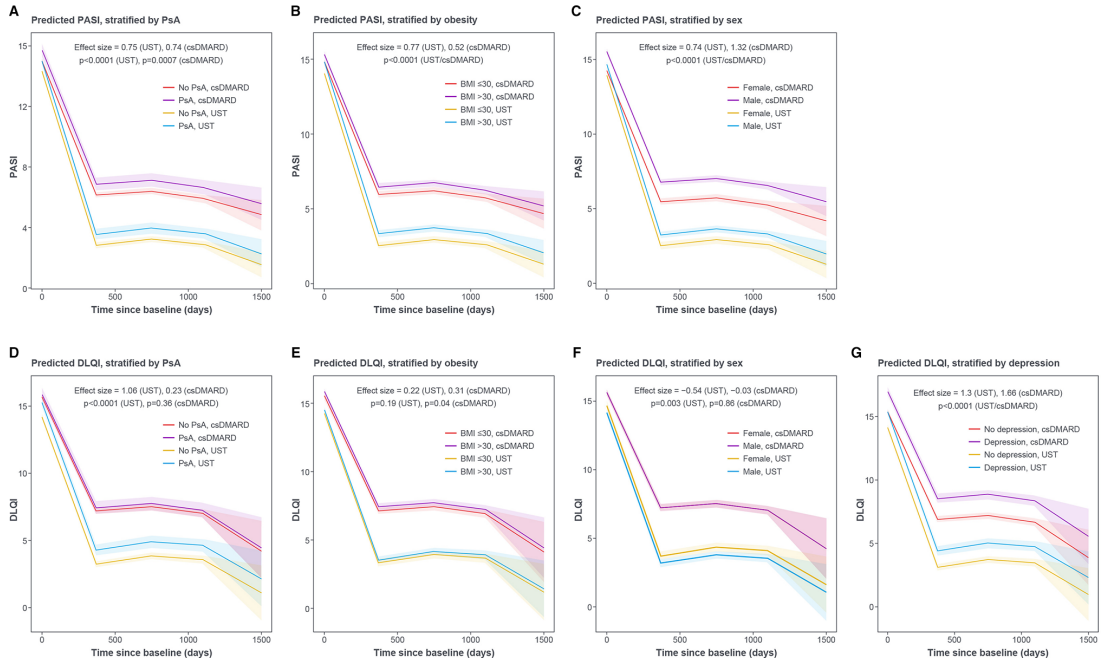

Figure 5 PASI and DLQI over time modelled in the UST and csDMARD cohorts. Patients stratified by PsA (A,D), obesity* (B,E), sex (C,F) and depression (G). *Obesity is defined as a BMI of ≥30 kg/m^2. BMI, body mass index; csDMARD, conventional synthetic disease-modifying antirheumatic drug; DLQI, Dermatology Life Quality Index; PASI, Psoriasis Area and Severity Index; PsA, psoriatic arthritis; UST, ustekinumab.

demonstrating higher persistence with biologics in men than in women.[15 25]

It should be noted that the HRs for the estimated effects of PASI score, biologic experience (both as proxies for disease severity and duration), sex and obesity (the inflammatory burden of which is linked to PsA occurrence) are likely to underestimate the true direct controlled effect. This is because these covariates were included as potential confounders for the effect of PsA on UST persistence. Therefore, any effects mediated via PsA from these variables are included in the estimate for PsA.

As depression is not a true confounder for the effect of PsA on TTD, but rather more likely to act as a possible mediator of the effect, the HR for the effect of depression on TTD (assuming no unmeasured confounding) can likely be assumed to be the full estimate of the controlled direct effect. The HR for the effect of PsA on TTD, although significant and notable, may therefore be an underestimate of the true effect size. This is because any effect caused by a diagnosis of PsA and mediated via depression (ie, for patients who experience depression due to or influenced by their PsA diagnosis resulting in shorter treatment time) would not be included in the effect estimate.[26]

Patients with severe skin disease were more likely to discontinue treatment than patients with less severe skin disease, when removing any effect mediated via PsA status; PsA had a greater association with discontinuation in patients with low PASI scores than in those with high PASI scores. The negative effect of increased skin disease severity on treatment persistence has been previously described,[27] though the relationship with PsA adds further to this observation. This underscores the need for thorough understanding of the importance of skin involvement to individual patients in order to optimise treatment outcomes.

During follow-up, UST treatment was associated with lower PASI and DLQI scores than csDMARD treatment irrespective of the time point. PsA, obesity and male sex were associated with higher PASI scores during the course of UST treatment. No comorbidities were shown to affect the improvement in PASI score over time with a successful treatment in this model. Depression was associated with a small increase in DLQI score irrespective of the time point at which it was measured, whereas a lower DLQI score was seen in men than in women in the UST cohort. Evaluation of the psychosocial impact of psoriatic disease may be needed to ensure adequate support and advice is given to patients. The addition of psychological support to disease management can improve patient satisfaction, with the potential for better clinical outcomes.[18]

Depression is also linked to central sensitisation[28]; the associated chronic pain is often difficult to distinguish from PsA disease activity and may result in unnecessary intensification or switching of therapy.[29] Management of depression through educational programmes, cognitive behavioural therapy or exercise therapy can improve patients' perception of pain.[30]

With the higher prevalence of obesity in the PsA group, as well as the association between obesity and numerically higher PASI scores in modelling, consideration should also be given to weight and lifestyle management for the treatment of patients with PsA. Weight loss interventions

are recommended to reduce disease severity and have been shown to be effective in both PsA and psoriasis.[31–33] Furthermore, BMI has been shown to be a modifiable risk factor in the development of PsA in patients with psoriasis, with reduction in BMI over a 10-year period linked to a reduction in the risk of PsA development.[13] Although guidelines encourage assessment of comorbidities and multidisciplinary working with different medical specialisms for patients with PsA,[17] there is a lack of evidence in clinical settings. These data emphasise the importance of comorbidity assessment and treatment.

This study has limitations. The BADBIR is a database intended to assess the treatment of psoriasis and does not capture PsA disease activity or involvement of specific disease domains, such as enthesitis and dactylitis.[22] Similarly to other analyses of this type, causal relationships can be difficult to determine, as although we have attempted to account for confounding bias via multivariable modelling, the possibility of unmeasured confounding cannot be ruled out and model mis-specification is a possibility when using regression modelling to account for confounding bias. The possible impact of factors like socioeconomic status and availability of alternative biologics for PsA could not be accounted for as this analysis compares UST with csDMARDs and does not include a biologic comparator arm. Another potential limitation is the use of TTD as a surrogate measure of efficacy, as clinical decisions to discontinue treatment can be based on factors other than loss of efficacy, such as skin disease severity or intolerance.

CONCLUSIONS

These results emphasise that patient-centric, multidisciplinary care needs to be considered to achieve the best possible outcomes in psoriatic disease. Psychological support should be considered as part of patient care because of the impact of psoriatic disease on QoL and the higher likelihood of depression observed in those treated with biologics than in those receiving csDMARDs. Lifestyle advice and weight management should also be considered for patients with psoriatic disease to optimise treatment outcomes.

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Contributors All named authors meet the International Committee of Medical Journal Editors criteria for authorship for this article and contributed substantially to the conception and design (WT, AO, AP and PG), acquisition of data (AP and PG), analysis of the data (WT, AO, AP and PG), interpretation of data and drafting of the manuscript (WT, AO, AP and PG). All authors revised the manuscript critically for important intellectual content, approved the version to be published and agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. WT and AO contributed equally to this manuscript. On behalf of the other authors, WT acts as the corresponding author. PG is the guarantor for this manuscript.

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Competing interests WT reports fees from AbbVie, Amgen, Celgene, Eli Lilly, Janssen, MSD, Novartis, Pfizer and UCB, and grants/research support from AbbVie, Celgene, Eli Lilly, GlaxoSmithKline, Janssen and UCB. AO reports fees from AbbVie, Amgen, Bristol Myers Squibb, Celgene, CorEvitas, Eli Lilly, Janssen, Novartis and Pfizer; grants/research support to the University of Pennsylvania from AbbVie, Novartis and Pfizer; and grants/research support to Forward/NDB from Amgen. AP and PG are employees of Janssen-Cilag Ltd.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants and no separate ethical approval was sought. The British Association of Dermatologists Biologic and Immunomodulators Register is approved by NHS Research Ethics Committee North West England (reference 07/MRE08/9). Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

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