EXTENDED REPORT

Predictors for radiological damage in psoriatic arthritis: results from a single centre

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Background: The predictors for the development of clinical damage in psoriatic arthritis (PsA) have been reported previously.
Aim: To identify predictors for radiological damage in PsA.
Methods: Patients followed-up prospectively according to a standard protocol at The University of Toronto between 1978 and 2004 were included. The principal outcome was the change in the number of damaged joints between visits, both clinically and radiologically. Explanatory variables considered included: sex, age, duration of arthritis at first visit, time in clinic, number of tender swollen joints, functional class, erythrocyte sedimentation rate (ESR), concentration of drugs and, to adjust for within-patient correlation, the number of clinically damaged joints at the first of the two visits over which change was observed.
Results: At the time of this analysis, 625 patients were recorded in the database. Multivariate analyses of predictors for both clinical and radiological damage show that age, time in clinic, initial ESR, number of tender and swollen joints at previous visit, and number of deformed joints at previous visit were related to both clinical and radiological damage.
Conclusions: The number of actively inflamed joints, particularly the number of swollen joints, was associated with the progression of radiological damage. The higher the number of previously damaged joints, the higher the risk for progression of damage. Thus, patients with PsA need to be treated, even in the presence of damage as long as there is evidence of inflammation, to prevent the progression of damage.

Psoriatic arthritis (PsA) is an inflammatory arthritis associated with psoriasis that may affect 0.3–1% of the population.[1] In the past, PsA was considered a mild form of arthritis. However, over the past two decades, it has become apparent that PsA is associated with considerable morbidity.[2–5] Moreover, patients with PsA are at increased risk of death compared with the general population.[6] Disease severity at presentation to clinic was found to be a predictor of mortality among patients with PsA.[7]

We have reported previously that patients with PsA had progression of clinical joint damage in that when patients were followed-up for 10 years, >55% had clinical damage (defined as clinical deformity, reduction in range of movement of >20% of the range, flail joints and ankylosed joints) in ⩾5 joints.[8] Predictors for the progression of clinical damage included the number of swollen joints at presentation to clinic and the level of treatment (non-steroidal anti-inflammatory drugs, malaria drugs or sulfasalazine, gold or methotrexate, ciclosporine or psoralen and ultraviolet A, corticosteroids), whereas a low erythrocyte sedimentation rate (ESR) was protective.[9] The number of actively inflamed joints at each visit, and the degree of clinical damage, predict subsequent clinical damage.[10] Other investigators have also found that polyarticular disease at presentation predicted the progression of damage, both clinically and radiologically.[4 5]

Radiological damage has been identified early in the course of PsA. At the first visit to the clinic, 67% of the patients were found to have at least one joint with erosion.[2 11] Within 2 years of onset of symptoms, 47% of the patients with PsA developed erosive disease.[3] In a previous study, we found that radiological damage in the hands was detected before clinical damage in most of the cases.[12] The aim of the current investigation was to identify predictors for radiological damage in a large cohort of patients with PsA followed-up prospectively.

METHODS

Patient selection

The University of Toronto PsA Clinic, Toronto, Ontario, Canada, is a longitudinal observational cohort of patients, with PsA followed-up prospectively since 1978.[2] All patients registered in the PsA Clinic database between 1978 and 2004 were included in this study. Patients were followed-up at 6–12 month intervals according to a standard protocol, which included a clinical evaluation and routine laboratory assessments. Clinical evaluation included a complete history, including a history of drugs, general physical examination and rheumatological assessment comprising an assessment of actively inflamed joints (stress pain, joint line tenderness and/or joint swelling). Radiological evaluations of the hands, feet and spine were carried out at 2-year intervals. All information collected was tracked in an ORACLE database. The study was approved by the institutional ethics board. All patients provided informed consent for their data to be included in the database for future analyses.

Assessment of clinical damage (deformities)

Clinical damage is determined by the presence of a limitation of range of movement of >20% of the range not related to the presence of joint effusion, the presence of joint deformities, subluxation, loosening or ankylosis. This measure has been proved reliable both in our clinic and by Canadian rheumatologist members of the SpondyloArthritis Research Consortium Canada.[13 14] Clinical damage can be assessed on all 68 joints examined clinically, including wrists, all metacarpophalangeals, proximal interplalangeals (PIPs) and distal interphalangeals of the hands, MTPs and PIPs of the feet, ankles, midtasal

Abbreviations: ESR, erythrocyte sedimentation rate; PIP, proximal interplalangeals; PsA, psoriatic arthritis

joints, knees, hips, elbows, shoulders, sterno-clavicular and temporomandibular joints.

Assessment of radiological damage

Radiological damage is assessed according to a modification of the Steinbrocker method.[15] Each joint is scored as 1, normal (with possible soft tissue swelling); 2, surface or pocket erosions; 3, erosion and joint space narrowing; and 4, disorganisation (including ankylosis, pencil-in-cup change or total joint destruction) or as requiring surgery. This method has also proved reliable in our clinic, both in terms of interobserver and intraobserver agreement and in terms of sensitivity to change over time.[15] Radiological damage is assessed only in the joints of the hands (wrists, all metacarpophalangeals, PIPs and distal interphalangeals) and feet (MTPs and interphalangeal fist toes), which are 42 joints in total.

Outcome variable

The principal outcome was the change in the number of permanently damaged joints between visits. There were two possible means of identifying damaged joints: assessment of clinically damaged joints and assessment of radiographically damaged joints (radiological score >1). The first method was applied at every visit to the clinic and formed the basis of the model selection procedure. The radiographs were recorded only about every 2 years. Therefore, in addition to having longer gaps between observations, it was also necessary to update time-dependent covariates in any analysis of radiographic damage only at the time of visits at which radiographs were taken. Furthermore, the radiographs were only available for joints in the hands and feet, hence an additional analysis, for comparison purposes, was that of clinical damage restricted to the hands and feet.

Thus, analyses undertaken initially were to update earlier publications on clinical damage,[9 10] to examine clinical damage only in the hands and feet, and finally to achieve the objective of this study and identify predictors for radiographic damage progression in this cohort.

Predictors

Explanatory variables considered included sex, age, arthritis duration at first visit, active and swollen joints, functional class, ESR, drugs and, to adjust for within-patient correlation, the number of clinically or radiographically damaged joints at the first of the two visits over which change was observed. For the latter purpose, previously used categories of 0, 1–4, 5–9 and >10 damaged joints were used for all analyses, although the actual counts were those of the relevant outcome variable. To allow for any remaining, unadjusted, within-patient correlation, a generalised estimating equation analysis was used to provide robust estimated standard errors (SE) when fitting our primary multivariate models. The actively inflamed and swollen joint counts were used to produce two variables for examination: the swollen joint count and a count of the ''tender'' joints, defined as the difference between the active joint count and the swollen joint. For analyses of damage in the hands and feet, the counts of tender and swollen joints were also restricted to the hand and feet joints, thus assuming that the disease progression in the hands and feet proceeds independently of the other joints in the body.

An attempt to summarise treatment concisely was made by defining variables to code the most severe form of drugs ever taken before a clinic visit. The order of increasing severity was no drug, non-steroidal anti-inflammatory drugs, disease-modifying anti-rheumatic drugs and steroids. None of the patients were taking biologicals.

Variables that changed over time and were recorded at each clinic visit were swollen joints, tender joints, duration of attendance at clinic, ESR and the drug history.

Statistical modelling

As in our earlier publication on time-varying predictors, the outcome variable was assumed to follow a negative binomial distribution, where the expected number of newly damaged joints accrued between visits over a period of time, 1 year on the time-scale used here, is modified by a multiplicative factor exp(R), where R takes the standard regression form,

$$R = B_1X_1+B_2X_2+ \ldots +B_pX_p$$

Each patient contributes observations from all pairs of consecutive clinic visits. The model adjusts for the differing lengths of time between clinic visits. The B_i are the logarithms of the relative damage rates and are estimated using maximum likelihood estimation procedures. When a covariate X_i can take only two values, 0 or 1, then the relative rate of damage, comparing an individual with an X_i coded to 1 with an individual with the covariate coded to 0, is $\exp(B_i)$, if the individuals have the same values for the remaining covariates. When the covariate can take arbitrary values, then $\exp(B_i)$ compares two individuals who differ by one unit in X_i.

Initially, time-varying factors were added individually to a baseline model that only used covariates recorded at the first clinic visit and the current count of damaged joints. The baseline covariates, chosen on the basis of our previous publication,[9] were sex, age, arthritis duration, functional class, ESR, tender joint count, swollen joint count and drugs. This allows the identification of time-varying covariates, in addition to the current count of damaged joints, which provides information over and above the information provided at the first clinic visit. Initially, all the covariates were stratified into levels and coded through binary indicator variables, as table 1 indicates. Significance levels were obtained through likelihood-ratio tests. A second model was fitted that included all the time-varying covariates, and the effect of removing the individual covariates from the model was assessed using the likelihood-ratio test.

On the basis of the information from these single-factor analyses, a multifactor model was developed. For this model, the ESR and the tender and swollen joint counts were represented using a linear function of the raw data, rather than the categorisation previously used, to cope with the loss of statistical power in a multifactor model; to test for non-linearities, quadratic terms were added to the model to check for significance.

An interaction between the duration of the illness (both at first clinic visit and the cumulative time at subsequent visits) and the extent of damage was also considered because of a trend in the damage duration index (the crude number of damaged joints divided by the duration of the illness) decreasing over time that was observed in previous work.

RESULTS

Table 1 shows the distributions of the covariates recorded at the first clinic visit. At the time of this analysis, 625 patients were recorded in the database. The median number of clinic visits is 7 per person, with a range of 2–76. For the data analysed in the previous work,[10] the median and range of the number of clinic visits was 6 (2–31), whereas the newer patients had 7 (2–42). This indicates a substantial increase in the amount of information available compared with our previous publication.

As described in the Methods section, we estimated a multivariate model based on information at baseline to which

Table 1 Characteristics of patient population at clinic entry

Number of patients	625
Female/male	272/353
Median (range) age (years)	34 (9–86)
Median (range) duration of arthritis (years)	4.5 (0–47.7)
Median (range) number of tender joints (all joints)	4 (0–43)
Median (range) number of tender joints (hands and feet)	3 (0–35)
Median (range) number of swollen joints (all joints)	2 (0–33)
Median (range) number of swollen joints (hands and feet)	1 (0–28)
Median (range) ESR rate	22.5 (0–105)
Functional class	
Good (I)	29.3% (183)
Medium (II)	59.2% (370)
Poor (III, IV)	11.5% (72)
Damaged joints (all joints)	
None	62.2% (389)
1–4	20.8% (130)
5–9	5.9% (37)
>9	11.1% (69)
Damaged joints (hands and feet)	
None	68.3% (427)
1–4	17.3% (108)
5–9	5% (31)
>9	9.4% (59)
Drugs	
None	24.3% (152)
NSAIDs	30.6% (191)
DMARDs	40.5% (253)
Steroids	4.6% (29)

DMARDs, disease-modifying antirheumatic drugs; ESR, erythrocyte sedimentation rate; NSAIDs, non-steroidal anti-inflammatory drugs.

Table 2 Single-factor analyses of time-varying factors for progression of clinical damage

	Single-factor analyses		All factors included	
Factor	**Relative damage rate (95% CI)**	**p Value**	**Relative damage rate (95% CI)**	**p Value**
Functional class		<0.001		0.1
Good (I)	1		1	
Medium (II)	1.56 (1.24 to 1.96)		1.16 (0.89 to 1.5)	
Poor (III, IV)	1.37 (0.96 to 1.91)		0.87 (0.59 to 1.28)	
Tender joints		<0.001		0.2
None (0)	1		1	
Low (1–4)	1.45 (1.13 to 1.86)		1.15 (0.89 to 1.51)	
Medium (5–9)	1.63 (1.19 to 2.24)		1.27 (0.91 to 1.78)	
High (>9)	2.09 (1.54 to 2.85)		1.37 (0.97 to 1.95)	
Effusions		<0.001		<0.001
None (0)	1		1	
Low (1–4)	1.32 (1.07 to 1.63)		1.12 (0.89 to 1.42)	
Medium (5–9)	1.84 (1.33 to 2.55)		1.48 (1.02 to 2.13)	
High (>9)	2.95 (1.82 to 4.78)		2.6 (1.56 to 4.36)	
ESR		0.17		0.75
Low (<15)	1		1	
Medium (15–30)	1.05 (0.82 to 1.39)		0.99 (0.77 to 1.28)	
High (>30)	1.27 (0.94 to 1.73)		1.09 (0.8 to 1.48)	
Arthritis duration	0.67 (0.55 to 0.8) per extra decade in clinic	<0.001	0.73 (0.6 to 0.89)	<0.001
Drugs		0.143		0.044
None	1		1	
NSAIDs	0.72 (0.44 to 1.18)		1.11 (0.65 to 1.91)	
DMARDs	0.89 (0.6 to 1.32)		1.32 (0.84 to 2.07)	
Steroids	1.04 (0.68 to 1.6)		1.64 (1.02 to 2.68)	

DMARD, disease-modifying antirheumatic drug; ESR, erythrocyte sedimentation rate; NSAID, non-steroidal anti-inflammatory drug.

the time-varying variables were added. This baseline multivariate model was qualitatively similar to those in our previous investigation of clinical damage using only variables present in the first visit.[9] Strong relationships were identified between clinical damage development and swollen joints, ESR and arthritis duration, with borderline significance found for age and tender joints. No relationship was identified for functional class or drugs.

The first column of results in table 2 show the single-factor analyses of the time-dependent covariates when added to the model of baseline covariates measured at the first clinic visit (as outlined in the Methods section). Recall that the outcome variable is the change in the clinically damaged joint count for all joints. The second column of results derives from simultaneously adding all the time-dependent covariates to the baseline model and considering the significance of removing, in turn, each time-dependent covariate. Except for ESR, all the variables show some relationship in the single-factor analyses, but only joint swelling and arthritis duration are significant in the multifactor analysis.

Table 3 displays a multifactor model for the clinically damaged joint count. This provides the update of our previous work.[10] From the results shown in table 2, functional class and drugs at first clinic visit were excluded as non-significant. Only the initial value of ESR was found to be predictive, whereas just using the time-varying tender and swollen joint counts was sufficient. Sex and drugs were found to be non-significant, but have been retained to show that the results are adjusted for these factors. Note that before entry to the clinic, the longer the duration, the more damage caused by arthritis, but during duration the more damage in the clinic the effect is the opposite: the longer the follow-up, the lesser the damage. Notably, however, arthritis duration at first visit is a predictor for progression in patients who do not have damage at first visit, but once a patient has a damaged joint, the predictive power of the arthritis duration evaporates. Current damage, included to adjust for within-patient correlation, was also a significant predictor. All the quadratic terms in ESR, tender and swollen joint counts were non-significant, thus justifying the use of continuous representation of these variables and linear effects; by contrast, the current damage did show significant quadratic terms, hence it is represented in the original categorical form.

In the development of this multivariate model for clinical damage in all joints, and subsequently fitting a model with the same set of predictors for the other outcome measures, we checked for consistency in the excluded variables as well. Some variation was observed for initial swollen count, which was predictive for some but not all outcome measures. However, for simplicity of interpretation and for consistency of presentation across the different outcome measures, this factor was omitted from our summary tables 3–5. The p values for adding initial swollen count to the models given in tables 3–5 were 0.07 for the clinical damage, 0.4 for clinical damage in the hands and feet, and 0.01 for the radiological damage.

The absence of functional class (time dependent) from the model and the role of arthritis duration are the differences from previous analyses.[10] Also, the stronger effect of swollen joints (RR 1.08, 95% CI 1.05 to 1.10) compared with tender joints (RR 1.02, 95% CI 1.01 to 1.03) gives a significant difference in effects with a ratio of RR of 1.06 (95% CI 1.03 to 1.09). The estimated RR of 1.08 associated with each swollen joint means that the progression rate is increased by 8% for each joint in a cumulative or compound fashion. Thus, for example, the RR is $(1.07)^5 = 1.4$ when comparing two patients whose swollen joint count differs by 5.

Table 4 simply fits the same model (in terms of variables) to the change in the clinically damaged joint count for hands and feet only. This was necessary as a prelude to the next analysis looking at radiological damage, for which we only have hand and feet radiographs for all patients. The results are roughly comparable to those in table 3 with a lesser effect for initial arthritis duration in without damaged joints patients. Further analyses were therefore performed whereby the same predictor variables were used as in table 3, but the outcome variables were the increment in the number of damaged hand and feet joints, and the increment in the number of damaged joints in the remainder of the body. This indicated that the risk for initial

Table 3 Clinical damage model

Factor	Relative damage rate	Lower 95% CI	Upper 95% CI	p Value
Sex				
Female	1			
Male	1.08	0.88	1.32	0.45
Age	1.08 per decade	1	1.16	0.05
Time in clinic	0.66 per decade	0.56	0.78	<0.001
Initial ESR	1.01	1	1.01	<0.001
Tender	1.02 per joint	1.01	1.03	0.002
Swollen	1.08 per joint	1.05	1.1	<0.001
Deformed joints				
None (0)	1			
Low (1–4)	2.84	2.1	3.85	<0.001
Medium (5–9)	5.6	3.89	8.08	<0.001
High (>9)	5.88	4.18	8.28	<0.001
Drugs	1			
None				
NSAIDs	1.18	0.86	1.61	0.31
DMARDs	1	0.67	1.48	0.99
Steroids	1.43	0.99	2.06	0.06
Initial arthritis duration				
Damaged	1.06 per decade	0.92	1.22	0.39
Undamaged	1.54 per decade	1.22	1.96	<0.001

DMARDs, disease-modifying antirheumatic drugs; ESR, erythrocyte sedimentation rate; NSAIDs, non-steroidal anti-inflammatory drugs.

Table 4 Hands and feet clinical damage model

Factor	Relative damage rate	Lower 95% CI	Upper 95% CI	p Value
Sex				
Female	1			
Male	0.95	0.75	1.2	0.66
Age	1.09 per decade	1	1.19	0.05
Time in clinic	0.67 per decade	0.52	0.86	0.001
Initial ESR	1.01	1	1.01	0.003
Tender	1.03 per joint	1.01	1.04	<0.001
Swollen	1.07 per joint	1.04	1.1	<0.001
Deformed joints				
None (0)	1			
Low (1–4)	2.8	1.87	4.21	<0.001
Medium (5–9)	5.41	3.53	8.31	<0.001
High (>9)	5.19	3.16	8.52	<0.001
Drugs				
None	1			
NSAIDs	1.39	0.96	2.02	0.09
DMARDs	0.96	0.61	1.52	0.87
Steroids	1.86	1.21	2.85	0.004
Initial arthritis duration				
Damaged	1.08 per decade	0.9	1.29	0.44
Undamaged	1.3 per decade	1.06	1.6	0.01

DMARDs, disease-modifying antirheumatic drugs; ESR, erythrocyte sedimentation rate; NSAIDs, non-steroidal anti-inflammatory drugs.

arthritis duration in patients without damage affected the hand and feet (RR 1.45, 95% CI 1.07 to 1.99 p = 0.02) joints less than the remainder of the body (RR 1.74, 95% CI 1.24 to 2.43, p = 0.001). In table 4, there was more evidence of increased damage for patients receiving steroids (this obviously not being a randomised comparison).

Table 5 represents the same analysis again, but this time based on radiological damage. The effect of initial arthritis duration decreases to the level of giving no evidence to support a relationship, and the effect of steroids becomes non-significant. Note also that the effects associated with the damaged joint count at the first of the two visits are less, but this is probably to be expected as the gap between visits is so much longer for this radiological damage analysis. Also, as a consequence of the increased duration between observations, the tests for quadratic effects in tender and swollen joints (but not initial ESR) were significant. In a model not shown, the hand and feet data were modified to ignore the observations at clinic visits where radiological measurements were not taken; these quadratic terms were also significant, thus providing evidence to support the

Table 5 Radiological damage model

Factor	Relative damage rate	Lower 95% CI	Upper 95% CI	p Value
Sex				
Female	1			
Male	1.09	0.86	1.37	0.47
Age	1.1 per decade	1	1.21	0.05
Time in clinic	0.68 per decade	0.53	0.87	0.002
Initial ESR	1.01	1	1.01	0.004
Tender	1.03 per joint	1.01	1.04	<0.001
Swollen	1.07 per joint	1.04	1.09	<0.001
Deformed joints				
None (0)	1			
Low (1–4)	1.84	1.29	2.61	0.001
Medium (5–9)	2.77	1.9	4.03	<0.001
High (>9)	2.98	2.1	4.24	<0.001
Drugs				
None	1			
NSAIDs	0.97	0.71	1.33	0.86
DMARDs	0.87	0.6	1.25	0.44
Steroids	0.85	0.59	1.24	0.4
Initial arthritis duration				
Damaged	0.99 per decade	0.81	1.19	0.88
Undamaged	0.84 per decade	0.63	1.12	0.23

DMARDs, disease-modifying antirheumatic drugs; ESR, erythrocyte sedimentation rate; NSAIDs, non-steroidal anti-inflammatory drugs.

claim that the difference is due to the longer time intervals rather than the different measure of joint damage.

Table 6A provides an assessment of the fitted multivariate models in terms of the observed and estimated intervisit increases in joint counts. Table 6B provides, for the radiological damage model, the estimated probabilities of new damaged joints over a 2-year period for various centiles of a prediction score defined by the linear predictor function, R, in the negative binomial model. It can be seen, for example, that a patient in the 90th centile of the prediction score has a 20% chance of developing damage in ≥ 5 previously undamaged joints over a 2-year period whereas for a patient in the 10th centile, that risk is only 1.4%. It can thus be seen that the models do reflect the observed data fairly well and that the variation in risk represented in the models is of clinical relevance.

DISCUSSION

Damage is an important outcome in patients with any form of arthritis, PsA included. The original studies by Wright and Moll[16] looked at a subset of patients with PsA who had an extremely destructive form of arthritis, which they labelled ''arthritis mutilans''. The authors recognised this form as presenting clinically with ''telescoping'' fingers or toes, and radiologically as the ''pencil-in-cup'' change.[1] In patients with PsA, damage may be noted clinically as joint deformities, fusion, flail joints and total joint ankylosis. These changes are noted clinically, whereas radiologically one may identify subluxed joints, joints with pencil-in-cup change or ankylosed joints. Some patients with PsA develop damage early and progress rapidly, whereas others do not develop damage even after prolonged disease duration. Identifying predictors for progression of damage may lead to better management of patients with PsA. Although clinical damage may be assessed at each patient–physician encounter, radiographs are not usually performed, often because of the radiation effect. Thus, recognising predictors for both clinical and radiological damage may be useful for clinicians managing patients with PsA. This type of analysis can only be performed in centres where prospective evaluation of patients according to standard protocols that include both clinical and radiological assessments have been done. The University of Toronto PsA Clinic provides a unique opportunity to perform such analyses as it has accrued a large number of patients with PsA followed-up prospectively with a computerised database. This investigation aimed specifically at identifying predictors for radiological damage in a time-dependent analysis. We first analysed the predictors for progression of clinical damage, then identified predictors for clinical damage in hands and feet only, and finally analysed predictors for radiological progression in the hands and feet.

In our previous studies, we showed that the number of actively inflamed joints both at presentation and on subsequent evaluations predicted progression of clinical damage.[9 10] We also reported that low sedimentation rate was associated with reduced damage, whereas a high drug level was associated with progression of clinical damage, as was the current level of damage. Those studies included 365 patients and were based on the clinically damaged joint count, which included 68 joints and a smaller number of patients.[9 10] Reports from other centres have confirmed our results, showing that patients who present with polyarthritis are more likely to develop both clinical and radiological damage.[4 5] However, the numbers of patients included were much smaller than those in our study: 71 in one study[4] and 100 patients in the other.[5]

Our analysis of 625 patients followed-up prospectively in our psoriatic arthritis clinic over $>$ 27 years is broadly consistent with our earlier work. Our analysis shows the following predictors for progression of clinical damage: the number of tender and swollen joints, age, current level of damage, disease duration at presentation for patients with no damage at clinic entry and ESR level at presentation.

Before performing an analysis of the prognostic factors relevant to the development of radiological damage, which was the aim of this study, we repeated a similar analysis restricted to the joints of the hands and feet. This analysis was undertaken as these are the joints that are evaluated radiologically in a systematic way. This analysis of clinical damage in the hands and feet identified initial ESR, number of tender and swollen joints, and number of deformed joints in the previous visit as predictors of progression of clinical damage, whereas time in the clinic was ''protective''. Thus, essentially the same relationships as noted in the initial analysis of clinical damage when all joints are included was seen in the hands and feet only The effect of initial disease duration was most marked in joints other than hands and feet. As this is a new finding in this analysis, it should be treated with some caution.

The analysis of predictive factors for progression in radiological damage showed that the initial ESR, the number of tender and swollen joints, and the number of deformed joints in the previous visit were predictive of the progression of radiological damage, whereas shorter time in the clinic was associated with less damage. This is an important new observation relating the progression of radiological damage to the status of joint inflammatory activity in a time-dependent manner. Importantly, the same predictors also apply to the

Table 6 Model assessment

Increase in count	Clinical		Clinical damage in hands and feet		Radiological	
	Observed	Predicted	Observed	Predicted	Observed	Predicted
(A) Observed increases in damaged joint counts						
0	4436	4432.3	4726	4721.5	627	644.7
1–4	5293	5305.9	5381	53393.6	1218	1193.3
5–10	152	135.9	107	90.6	132	130.7
>10	61	64.3	37	40.8	31	57

Increase in joint count	Centile of the prediction score				
(B) Estimated probabilities of new damage over a 2-year period					
	10%	30%	50%	70%	90%
0	0.821	0.760	0.693	0.637	0.583
1–4	0.165	0.201	0.222	0.226	0.221
>4	0.014	0.039	0.085	0.137	0.195

progression of clinical damage, suggesting that regardless of how damage is detected, clinically or radiologically, it is predicted by the same factors. We would like to associate the apparent benefit of time followed in the clinic with our patient management, but this would be speculative. It is sufficient to consider the variable primarily as an adjustment variable for consideration of other factors in the model, although we believe there are advantages, for a variety of reasons, that patients be seen early by a disease specialist.

This study confirms that the number of actively inflamed joints, particularly swollen joints, predicts the progression of radiological damage. This is an important observation as it supports the hypothesis that active inflammation leads to joint damage. This observation leads credence to the notion that if we control inflammation in patients with PsA, we may be able to prevent the progression of both clinical and radiological damage.

Our study shows that the higher the number of inflamed joints, the higher the risk for progression of joint damage. The study also shows that the higher the number of previously damaged joints, the higher the risk for progression of damage. This suggests that the management of patients with PsA should aim to minimise the number of inflamed joints and that control of inflammation should continue even in the presence of damage. Ideally, inflammation should be treated before there is any resultant joint damage.

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