

Comparison of Radiological Severity in Psoriatic Arthritis and Rheumatoid Arthritis

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ABSTRACT. Objective. To compare the radiological severity of patients with psoriatic arthritis (PsA) and rheumatoid arthritis (RA).

Methods. Patients were identified from the University of Toronto PsA and RA databases. Using the earliest available radiographs, each RA patient was matched to a single PsA patient on the basis of sex, age, and disease duration. Two rheumatologists blinded to the patient's diagnosis scored the radiographs using the modified Steinbrocker method.

Results. PsA and RA groups were similar with respect to demographics as well as the use of disease modifying antirheumatic medications. No significant difference in Steinbrocker score for the hands and feet or the hands only was noted. Patients with RA had a higher radiological score in the feet. The 2 groups were similar in the number of joints with significant radiological damage (Steinbrocker 3 and 4).

Conclusion. Overall the radiological severity in the hands and feet of patients with PsA was comparable to that of patients with RA. (J Rheumatol 2001;28:1041-4)

Key Indexing Terms:

PSORIATIC ARTHRITIS
RADIOLOGICAL SEVERITY

RHEUMATOID ARTHRITIS
STEINBROCKER METHOD

Psoriatic arthritis (PsA) is a distinct clinical entity separate from rheumatoid arthritis (RA) on the basis of its unique epidemiological, clinical, and radiological features¹. The pathogenesis of both these disorders has yet to be elucidated. Both PsA and RA are characterized by chronic polyarticular synovial inflammation that is thought to result from infiltration and activation of lymphocytes, polymorphonuclear leukocytes, and dendritic cells^{2,3}. However, differences exist with respect to the distribution and extent of infiltration of the various cell populations as well as cytokine profiles^{4,5}. Based on our current understanding, radiological damage is presumed to be a consequence of the same pathological processes that cause inflammation. As a result radiological assessment is a useful measure to gauge

disease severity as well as progression in rheumatic diseases.

Although many feasible radiographic scoring methods exist for the assessment of joint damage, most methods were developed to capture changes in RA⁶. The most widely used radiological methods include those developed by Sharp, Larsen, and Steinbrocker⁷. These methods and their modifications have been validated in RA⁸. We recently validated the modified Larsen and Steinbrocker radiological methods in PsA⁹. Therefore, these methods can now be used to reliably assess radiological severity and progression in PsA.

While earlier studies suggested that PsA was not a severe disease, our studies and those of others have noted that PsA is not as benign as previously thought^{1,10}. We therefore tested the hypothesis that radiological severity in RA is greater than in PsA. We compared the radiological severity in patients with PsA versus RA.

MATERIALS AND METHODS

Patient Selection

Rheumatoid arthritis. Patients were identified from the University of Toronto RA database, which includes demographics, actively inflamed and clinically damaged joints, extraarticular features, and medications for all clinic visits to the Centre for Prognosis Studies in the Rheumatic Diseases. A record of radiographs taken is also kept. All patients satisfied the 1987 ACR diagnostic criteria for RA¹¹. Only patients with plain radiographs available of the hands or feet were included in this study. The earliest available radiographs were used. When radiographs were available of both the hands and feet for a patient, these had to have been taken on the same day. When available, cervical radiographs taken on the same day as the peripheral joint radiographs were also reviewed.

Psoriatic arthritis. For each RA patient, a single PsA match was found from the University of Toronto Psoriatic Arthritis Clinic database at the Centre for Prognosis Studies in Rheumatic Diseases. Patients were matched for sex,

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and within ± 10 years for disease duration (from the time of diagnosis) and age at the time of the earliest radiographs available for RA patients. The PsA database contains all information resulting from a comprehensive standardized assessment protocol that is done on patients every 6 to 12 months. Radiographs are taken every 2 years, or when clinically indicated. All patients included in the database satisfy the accepted definition of PsA (an unequivocal evidence of past or present psoriatic skin lesion and inflammatory arthritis, which has been confirmed by a physician)¹². Those with other diseases such as nodular RA, systemic lupus erythematosus, gout, inflammatory bowel disease, and osteoarthritis are excluded. Rheumatoid factor (RF) alone is not an exclusion criterion because 13% of patients with psoriasis uncomplicated by arthritis have positive RF¹³. Patients with isolated psoriatic spondyloarthropathy (SpA) were not included in this study.

Radiographic assessment. The anterior-posterior radiographs of the hands/wrists and feet were presented randomly to 2 rheumatologists (DDG and PR), who were blinded to the patient's diagnosis. They scored the films together and any discrepancy between them was settled by consensus. Forty-four joints were examined. These included the wrists, all metacarpal phalangeal joints, proximal interphalangeal and distal interphalangeal joints, all metatarsophalangeal joints and the interphalangeal joints of the first toes. The modified Steinbrocker method was used to score radiological severity^{9,14}. We have shown that this method provides information identical to that of the Larsen scores⁹. According to this method, each of the 44 joints is scored on a scale of 0 to 4. Joints are scored 0 if they are normal, 1 if there is osteopenia or soft tissue swelling, 2 if there is erosion, 3 if there is erosion associated with joint space narrowing, and 4 if there is total joint destruction. Joint space narrowing without erosion is not recorded. All individual joint scores are added to form a total score. The maximum score is 176 (128 for the hands/wrists, 48 for the feet).

Radiographic features of the hands, feet, and cervical spine (see Table 3) relevant to RA and PsA but not captured by the Steinbrocker method were also recorded. This was done using antero-posterior radiographs of the hands and feet (including heels), and lateral cervical films and odontoid views. All the radiographs for a patient used in this study were taken on the same day.

Statistical analysis. Descriptive statistics, the Wilcoxon matched pairs signed rank-sum test, and McNemar's test were used to compare the matched patient populations when appropriate. A paired analysis of the data was performed in accordance with the one-to-one matching of patients¹⁵.

RESULTS

Forty-two pairs of RA and PsA patients were identified (33 women, 9 men). There were 39 matched pairs of radiographs available of the hands, 31 of the feet, and 25 of the cervical spine. Table 1 shows the demographic and clinical characteristics of the 2 patient groups. The matched pairs of patients were similar in the number of actively inflamed and clinically damaged joints. By definition there is a difference in seropositivity for RF. Ninety-three percent of the patients with RA were seropositive, compared to only 7% of patients with PsA. The 3 patients with PsA who had a positive RF were all female and had typical features of PsA: one had concomitant sacroiliitis, another had an asymmetric arthritis with nail changes, and the third had oligoarthritis. Eighteen patients with PsA (43%) had radiographic changes in the spine consistent with SpA. Six of the 42 patients with PsA (18.9%) had oligoarthritis.

Using McNemar's test for paired comparisons there were significantly more RA than PsA patients with a positive RF ($p < 0.001$). There were 37 matched pairs in which the RA patient had positive RF and the PsA patient did not, and there was one pair when the PsA patient had positive RF and the RA patient did not. Similarly, patients with RA were more likely to have an elevated erythrocyte sedimentation rate (ESR) ($p = 0.005$). There were 15 matched pairs in which the RA patient had elevated ESR and the PsA patient did not, and 3 cases when the PsA patient had an elevated ESR when the RA patient did not. Overall, there was no statistically significant difference in the therapy between the 2 groups. However, more patients with PsA were treated with nonsteroidal antiinflammatory drugs (NSAID) alone. Although patients with RA were more likely to have received treatment with disease modifying medications than

Table 1. Demographics of patients with psoriatic (PsA) and rheumatoid arthritis (RA).

	RA, n = 42	PsA, n = 42	Paired Difference
Female:male	33:9	33:9	—
Age, yrs			
Mean (SD)	53.0 (15.0)	53.1 (26)	-0.1 (1.9)
Median (range)	54.2 (25-77)	54.0 (26-79)	-0.4 (-3.5-8.5)
Disease duration, yrs			
Mean (SD)	6.1 (7.3)	6.9 (5.8)	-0.8 (2.8)
Median (range)	3.9 (0-36)	5.2 (0.2-26)	-0.6 (-6.6-10.0)
No. of actively inflamed joints			
Mean (SD)	11.2 (10.8)	8.0 (7.9)	3.3 (12.3)
Median (range)	8.5 (0-42)	5.0 (0-38)	1.0 (-25-38)
No. of clinically damaged joints			
Mean (SD)	4.6 (7.7)	4.7 (8.5)	-0.1 (9.9)
Median (range)	0 (0-28)	1.5 (0-41)	0 (-30-28)
Positive RF	39 (92.8)	3 (7.1)	—
Elevated ESR	37 (88.1)	25 (59.5)	—
Medications, n (%)			
None	6 (14.3)	3 (7.1)	—
NSAID	10 (23.8)	20 (47.6)	—
DMARD	21 (50.0)	14 (35.3)	—
Corticosteroids	5 (11.9)	5 (11.9)	—

patients with PsA the difference was not statistically significant.

The modified Steinbrocker scores for each patient group and the means and medians of the paired differences are shown in Table 2. There was no difference between matched RA and PsA patients in the modified Steinbrocker score for hands and feet combined ($p = 0.14$) or for the hands only ($p = 0.75$). Patients with RA, however, had a significantly higher radiological score in the feet ($p = 0.03$), but the difference was not significant when correction for multiple testing was performed (corrected $p = 0.18$). The 2 groups were also compared with respect to the number of joints with significant radiological damage, defined as those with Steinbrocker grades 3 or 4. The differences were not statistically significant for both the hands and feet combined ($p = 0.53$), the hands only ($p = 0.78$), and the feet only ($p = 0.24$).

Table 3 shows the other radiographic features examined. The frequency of these features was small. While the McNemar test did not reveal any statistically significant differences there was an excess of features representing new bone formation in the hands of patients with PsA, as evidenced by a higher frequency of fluffy periostitis and bony proliferation.

DISCUSSION

Radiographs are useful endpoints in rheumatic disorders. Radiological assessment of the small joints of the hands and feet is particularly helpful as radiographic changes can often be detected at an early stage of the disease at these sites. Further, radiographic assessment plays a key role in the present classification for assessing outcome measures in clinical trials with a disease duration of one year or more, as determined by the OMERACT/ILAR/WHO core set¹⁶.

Several recent studies documenting disease characteristics in PsA have shown that patients are more severely affected than previously thought^{10,12,17-19}. Indeed, it has been shown that with time, the majority of patients with PsA have polyarthritis, which is symmetric in half the cases, and that

Table 3. Characteristic radiographic features of patients with psoriatic (PsA) and rheumatoid arthritis (RA).

Feature	RA, %	PsA, %
Hands	n = 39	n = 42
Fluffy periostitis	2.6	9.5
Tuft resorption	2.6	0
Bony proliferation	2.6	4.8
Dactylitis	0	7.1
Feet	n = 33	n = 39
Fluffy periostitis	3.0	2.6
Tuft resorption	27.3	25.6
Bony proliferation	3.0	0
Dactylitis	0	0
Calcaneal spur	27.3	35.9
Achilles spur	15.2	12.8
Cervical spine	n = 29	n = 37
Atlantoaxial subluxation	10.3	5.4
Subaxial subluxation	20.7	16.2
Odontoid erosion	3.4	0
Discovertebral erosion	3.4	0
Intervertebral disc narrowing	37.9	27.0

erosive disease is common. This is the first study to compare the radiological severity of RA and PsA in patients matched for age, sex, and disease duration. Patients with PsA were matched to the patients with RA to control for sex and age differences unrelated to the disease process. We have described a gender effect in the expression of both psoriatic SpA and in ankylosing spondylitis²⁰. We also matched the patients by the disease duration at the time the radiographs were taken so that both patient populations were subject to the same time effect of their disease process. We noted that there was no difference in overall radiographic severity of the small joints in RA compared to PsA. While the mean radiographic score for the small joints of the feet was higher in RA patients than in PsA, this finding was not statistically significant when adjusted for multiple comparisons. The majority of our patients with PsA had polyarthritis, as noted from the number of actively inflamed, clinically damaged and radiologically damaged joints. Only 6 of the 42 patients

Table 2. Radiological severity of patients with psoriatic (PsA) and rheumatoid arthritis (RA).

	RA		PsA		Paired Difference (RA-PsA)	
	Mean (SD)	Median (range)	Mean (SD)	Median (range)	Mean (SD)	Median (range)
Overall damage[†]						
Total (0-176)	27.8 (26.8)	19.5 (0-93)	20.7 (27.2)	10.0 (0-126)	6.5 (35.7)	8.0 (-90-91)
Hands (0-128)	14.1 (16.8)	8.0 (0-68)	13.6 (21.9)	5.5 (0-104)	-0.5 (25.9)	2.0 (-76-54)
Feet (0-48)	12.0 (12.0)	10.0 (0-40)	6.5 (7.0)	4.0 (0-24)	5.5 (12.9)	2.0 (-18-40)*
Severe damage[‡]						
Total (0-44)	4.8 (7.0)	2 (0-22)	3.5 (7.0)	0 (0-33)	0.8 (10.7)	0 (-30-22)
Hands (0-32)	2.5 (4.3)	1 (0-15)	2.5 (5.8)	0 (0-29)	-0.2 (1.3)	0 (-2-8)
Feet (0-12)	1.9 (3.1)	0 (0-10)	0.8 (1.5)	0 (0-6)	0.9 (3.5)	0 (-5-10)

[†]modified Steinbrocker score; [‡]No. of joints scored 3 or 4.

* $p = 0.03$ based on the Wilcoxon matched pairs signed-rank sum test, corrected $p = 0.18$.

with PsA had oligoarthritis. We have documented that by 6–10 years of disease 46% of the patients develop more than 5 clinically damaged joints¹. Thus it is not surprising that the majority of the patients in this group had polyarthritis. Whether patients with PsA who remain oligoarticular have the same degree of damage as patients with RA cannot be inferred from the current study. The groups of patients included in this study differed with regard to seropositivity for RF, which by definition distinguishes patients with RA from patients with PsA. An elevated ESR was more likely to be present in patients with RA than patients with PsA. Nonetheless, similar levels of radiological changes were noted. As we had reported, patients with PsA do not have the same degree of ESR elevation as patients with RA¹². This may result from differences in the pathogenetic processes leading to joint inflammation in the 2 conditions. Characteristic radiographic features for RA and PsA previously reported^{1,6,10} occurred infrequently in our study. Furthermore, there was a significant overlap in features between PsA and RA. Therefore the utility of these features in differentiating the 2 disorders based solely on these classic radiographic manifestations may be limited. The frequency of new bone formation was higher in patients with PsA, although this did not reach statistical significance. At present none of the commonly used radiographic methods attempts to capture this change. As the deposition of new bone may lead to damage of the surrounding articular and periarticular structures, a new radiographic classification system for PsA that accounts for this manifestation may need to be developed.

There are a few limitations in this study. The number of patients compared was small, thus it is possible that more subtle differences were overlooked due to a lack of power. A larger, multicenter study may be necessary to address this issue. However, it should be noted that this still represents the largest study to compare radiographic features of RA and PsA. Although more patients with PsA received only NSAID as their therapy, and more patients with RA received disease modifying antiinflammatory drug therapy, these differences did not reach statistical significance. We have reported¹⁷ that despite active treatment and a reduction in joint inflammation, PsA may lead to a deforming arthritis. Similar studies in RA reveal that disease modifying agents do not necessarily halt progression of damage²¹. However, the rate of damage may have been reduced with disease modifying agents. It is conceivable that the rate of damage accrued by PsA and RA differs. As this was a cross-sectional study we are unable to address issues concerning disease progression. This issue would be better addressed by a larger multicenter study.

We demonstrate that the radiological changes in PsA are comparable to RA in patients matched for age, sex, and disease duration, supporting the notion that PsA is as severe a disease.

REFERENCES

- Gladman DD. Natural history of psoriatic arthritis. In: Wright V, Helliwell P, editors. *Bailliere's clinical rheumatology. Psoriatic arthritis*. London: Bailliere Tindall; 1994:379-94.
- Abu Shakra M, Gladman DD. Aetiopathogenesis of psoriatic arthritis. *Rheumatol Rev* 1994;3:1-7.
- Bresnihan B. Pathogenesis of joint damage in rheumatoid arthritis. *J Rheumatol* 1999;26:717-9.
- Veale D, Yanni G, Rogers S, Barnes L, Bresnihan B, Fitzgerald O. Reduced synovial membrane macrophages numbers, ELAM 1 expression, and lining layer hyperplasia in psoriatic arthritis as compared with rheumatoid arthritis. *Arthritis Rheum* 1993;3:893-90.
- Ritchlin C, Haas-Smith SA, Hicks D, Cappuccio J, Osterland CK, Looney RJ. Patterns of cytokine production in psoriatic synovium. *J Rheumatol* 1998;25:1544-52.
- van der Heijde DMFM. Plain X-rays in rheumatoid arthritis: overview of scoring methods, their reliability and applicability. *Clin Rheumatol* 1996;10:425-53.
- Kaye JJ. Radiographic assessment of rheumatoid arthritis. *Rheum Dis Clin North Am* 1995;21:395-406.
- van der Heijde D, Boer M, Lassere M. Methodological issues in radiographic scoring methods in rheumatoid arthritis. *J Rheumatol* 1999;26:726-30.
- Rahman P, Gladman DD, Cook RJ, Zhou Y, Young G, Salonen D. Radiological assessment in psoriatic arthritis. *Br J Rheumatol* 1998;37:760-5.
- Torre-Alonso JC, Rodrigues-Perez A, Arribas-Castrillon JM, et al. Psoriatic arthritis: a clinical, immunological, and radiological study of 180 patients. *Br J Rheumatol* 1991;30:245-50.
- Arnett FC, Edworthy SM, Bloch DA, et al. The American Rheumatism Association 1987 revised criteria for the classification of rheumatoid arthritis. *Arthritis Rheum* 1988;31:315-24.
- Gladman DD, Shuckett R, Russell ML, Thorne JC, Schachter RK. Psoriatic arthritis — clinical and laboratory analysis of 220 patients. *Q J Med* 1987;62:127-41.
- Gladman DD, Anhorn KA, Schachter RK, Mervart H. HLA antigens in psoriatic arthritis. *J Rheumatol* 1986;13:586-92.
- Steinbrocker O, Traeger CH, Batterman RC. Therapeutic criteria in rheumatoid arthritis. *JAMA* 1949;140:659-62.
- Altman Douglas G. *Practical statistics for medical research*. London: Chapman and Hall; 1991:189.
- Boers M, Tugwell P, Felson DT, et al. World Health Organization and International League of Associations for Rheumatology core endpoints for symptom modifying antirheumatic drugs in rheumatoid arthritis clinical trials. *J Rheumatol* 1994;21 Suppl 41:86-9.
- Gladman DD, Stafford-Brady F, Chang CH, et al. Longitudinal study of clinical and radiological progression in psoriatic arthritis. *J Rheumatol* 1990;17:809-12.
- Jones SM, Armas JB, Cohen MG, et al. Psoriatic arthritis: outcome of disease subsets and relationship of joint disease to nail and skin disease. *Br J Rheumatol* 1994;33:834-9.
- Marsal S, Armadans-Gil L, Martínez M, Gallardo D, Ribera A, Lience E. Clinical, radiographic and HLA associations as markers for different patterns of psoriatic arthritis. *Rheumatology* 1999;38:332-7.
- Gladman DD, Brubacher B, Buskila D, Langevitz P, Farewell VT. Differences in the expression of spondyloarthropathy: a comparison between ankylosing spondylitis and psoriatic arthritis. Genetic and gender effects. *Clin Invest Med* 1993;16:1-7.
- Wolfe F, Sharp JT. Radiographic outcome of recent-onset rheumatoid arthritis. *Arthritis Rheum* 1998;41:1571-82.