Spectrum of psoriatic spondyloarthropathy in a cohort of 100 Spanish patients

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In their initial work, Moll and Wright recognised five patterns of psoriatic arthritis (PsA)—namely, distal joint disease, oligoarthritis, polyarthritis, arthritis mutilans, and spondylitis.[1] Although psoriatic spondyloarthropathy was recognised as a specific pattern of PsA, it has become clear that the presence of isolated spondyloarthropathy (SpA) in PsA is unusual, and in most cases, it occurs with peripheral arthritis.[2 3] We undertook the present work to analyse the clinical features of our patients with psoriatic SpA and to compare the different subgroups included within the psoriatic SpA spectrum.

One hundred patients with psoriatic SpA, defined in accordance with the European Spondyloarthropathy Study Group (ESSG) criteria,[4] were consecutively recruited and their clinical records were analysed in this retrospective cross sectional study. All patients were evaluated according to a standard protocol, and their functional ability was assessed using the Health Assessment Questionnaire-Specific for SpA (HAQ-S).[5] We included patients with isolated axial involvement, as well as those with axial plus peripheral disease. The study group was divided according to the articular patterns seen during the past five years of follow up. The presence of mutilans forms and distal interphalangeal (DIP) disease was also recorded.

We performed HLA-B27 typing by serological methods and HLA-Cw typing by molecular biology techniques (SSOP-PCR) in our 100 patients and in 177 blood donors of the same racial origin.

Sacroiliac radiographic changes were graded as follows: 0, normal; 1, possible; 2, minimal; 3, moderate; and 4, ankylosis. All symptomatic joints were radiographed. At least two senior rheumatologists read all *x* ray films, and there was a complete agreement, except for three cases, where consensus was achieved.

Categorical data were analysed by Pearson's χ^2 analysis and Fisher's exact test, whereas continuous data were compared by analysis of variance. Relative risk (RR) was calculated by Woolf's method.[6]

According to the clinical patterns seen during the past five years of follow up, 23 patients developed isolated axial disease only (M:F ratio 3.6:1), 36 showed a mixed pattern of axial disease and polyarthritis (M:F ratio 1:1), and 41 had oligoaxial disease (M:F ratio 1.7:1).

Thirty four patients carried the HLA-B27 antigen compared with a normal distribution of this allele of 7% in the control group (RR 7, $p<0.0004$), whereas HLA-Cw*0602 was seen in 58 of the 100 patients compared with a frequency in the control group of 18% (RR 6.2, $p<0.0001$). This last allele was equally distributed among the three articular categories.

Table 1 Comparison of psoriatic spondyloarthropathy subgroups. Continuous data are expressed as mean (standard deviation)

Variables	Pure axial pattern (n=23)	Polyaxial pattern (n=36)	Oligoaxial pattern (n=41)	p Values
Age (years)	43 (11)	54 (12)	46 (15)	0.039
Psoriasis duration (years)	16 (9)	19 (8)	17 (11)	NS
Arthritis duration (years)	10 (6)	13 (7)	10 (5)	NS
Psoriasis onset age (years)	27 (10)	35 (13)	29 (14)	NS
Arthritis onset age (years)	33 (8)	40 (12)	37 (15)	NS
Psoriasis-arthritis latency (years)	8 (4)	8 (7)	14 (10)	0.07
Family history (%)	17	19	22	NS
IBP (%)	83	36	37	0.009
Neck pain (%)	22	25	27	NS
Radiological neck involvement (%)	35	56	39	NS
Marginal syndesmophytes (%)	26	19	19	NS
Para-marginal syndesmophytes (%)	13	28	24	NS
Bilateral SI (%)	56	31	37	NS
Unilateral SI (%)	44	44	48	NS
Onychopathy (%)	30	75	51	0.015
ESR (mm/1st h)	27 (20)	47 (26)	20 (21)	0.0001
CRP (mg/l)	160 (250)	190 (160)	100 (130)	NS
C3 (g/l)	1.3 (0.3)	1.2 (0.3)	1.2 (0.3)	0.037
C4 (g/l)	0.3 (0.1)	0.2 (0.1)	0.3 (0.1)	0.045
IgG (g/l)	13.70 (2.60)	16.05 (5.00)	12.17 (2.80)	0.029
IgM (g/l)	1.93 (1.02)	1.71 (0.73)	1.55 (0.89)	NS
IgA (g/l)	3.12 (1.42)	2.85 (1.29)	2.86 (1.86)	NS
Erosive disease (%)	4*	75	37	0.0001
DIP disease (%)	17	64	51	0.015
HLA-B27 (%)	57	19	29	0.016
Uveitis (%)	30	11	10	0.022
Schober's test (cm)	2.5 (0.3)	2.7 (0.5)	2.8 (0.4)	NS
HAQ-S (mean)	1.4 (0.4)	1.8 (0.4)	1.5 (0.3)	0.03

IBP, inflammatory back pain; SI, sacroiliitis; ESR, erythrocyte sedimentation rate; CRP, C reactive protein; DIP, distal interphalangeal joint; HAQ-S, Health Assessment Questionnaire-Specific for spondylarthropathy.
*Erosive coxofemoral joint disease.

Table 1 compares the articular categories found in this study.

A satisfactory definition is still lacking for PsA, in particular for psoriatic SpA, and therefore we reported only those cases fulfilling the criteria given by the ESSG,[4] in order to standardise our observations and to enable comparisons with other past or future reports.

After a minimum of five years of observation, we have found that three clear patterns of psoriatic SpA are easy to document—namely, isolated axial disease, polyaxial disease, and oligoaxial pattern. Moreover, DIP disease and mutilans forms did not remain as independent patterns over time.

Patients with isolated axial disease were more often men, and had more clinical (inflammatory back pain), radiological (bilateral sacroiliitis and marginal syndesmophytes), immunogenetic (HLA-B27), and extra-articular (uveitis) features which strongly correlate this group with idiopathic ankylosing spondylitis. We found that the spondylitic process was similar between groups, though patients with polyaxial disease had more neck radiological damage than the other patients, probably because this group had more peripheral erosions and a longer duration of arthritis (table 1). Involvement of the neck was associated with arthritis duration ($p=0.043$) and peripheral erosions ($p=0.037$), confirming previous reports.[7]

Unquestionably, more than 50% of patients with PsA have DIP joint disease, but the presence of isolated DIP disease is still controversial, as is its role as a prognostic marker.[8] Our observations suggest that DIP disease is a typical feature of PsA rather than an additional articular category, and that it is mostly associated with peripheral forms of the disease; indeed, the highest percentages of onychopathy and DIP disease were seen among patients with the polyaxial pattern, whereas the lowest percentages were found in the group with isolated axial disease (table 1). This latter observation is at odds with the supposed association between DIP disease and sacroiliitis or the reported association between HLA-B27 and DIP disease.[8] We have obtained data which point not only to a different genetic basis in psoriatic SpA models but also to different pathogenic mechanisms. As a whole, in our study HLA-B27 was a stronger risk factor for isolated and oligoaxial variants of psoriatic SpA than for the polyaxial one. In this last group we found higher rates of complement consumption, more peripheral erosions, poorer functional performance, and higher immunoglobulin (IgG) values, supporting the view that immunocomplexes may be an important mechanism of tissue injury in psoriatic polyarthritis.[9] On the other hand, the ileocolonoscopic studies of Schatteman *et al* found gut lesions resembling those of inflammatory bowel disease in patients with psoriatic oligoarthritis and spondylitis but not in patients with psoriatic polyarthritis.[10]

In summary, some HLA, clinical and pathogenic data support the subdivision of psoriatic SpA as proposed in the present report.

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REFERENCES

1 **Moll JMH**, Wright V. Psoriatic arthritis. Semin Arthritis Rheum 1973;3:55–78.
2 **Torre Alonso JC**, Rodriguez Perez A, Arribas Castrillo JM, Ballina Garcia J, Riestra Noriega JL, Lopez Larrea C. Psoriatic arthritis (PA): a clinical, immunological and radiological study of 180 patients. Br J Rheumatol 1991;30:245–50.
3 **Marsal S**, Armadans Gil L, Martinez M, Gallardo D, Ribera A, Lience E. Clinical, radiographic and HLA associations as markers for the different patterns of psoriatic arthritis. Rheumatology (Oxford) 1999;38:332–7.
4 **Dougados M**, van der Linden SM, Jhulin R. The European Spondyloarthropathy Study Group preliminary criteria for the classification of spondyloarthropathy. Arthritis Rheum 1991;34:1218–27.
5 **Daltroy LH**, Larson MG, Roberts WN, Liang MH. A modification of the Health Assessment Questionnaire for the spondyloarthopathies. J Rheumatol 1990;17:946–50.
6 **Woolf B**. On estimating the relation between blood groups and disease. Ann Hum Genet 1955;19:251–3.
7 **Salvarani C**, Macchioni P, Cremonesi T, Mantovani W, Battistel B, Rossi F, *et al*. The cervical spine in patients with psoriatic arthritis: a clinical, radiological and immunogenetic study. Ann Rheum Dis 1992;51:73–7.
8 **Gladman DD**. Psoriatic arthritis. Rheum Dis Clin North Am 1998;24:829–43.
9 **Rivas D**, Riestra JL, Torre Alonso JC, Rodriguez A, Gutierrez Martin C. Decrease in detectable complement receptor type I from erythrocytes from patients with psoriatic polyarthritis. Br J Rheumatol 1994;33:626–30.
10 **Schatteman L**, Mielants H, Veys EM, Cuvelier C, De Vos M, Gyselbrecht L, *et al*. Gut inflammation in psoriatic arthritis: a prospective ileocolonoscopic study. J Rheumatol 1995;22:680–3.