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Psoriatic Arthritis: A Clinical, Radiological and Genetic Study of 58 Italian Patients

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It is well known that genetic heterogeneity and/or the complex interaction of several MCH-linked risk factors can explain the onset and the broad spectrum of Psoriatic Arthritis (PsA) from the clinical point of view. Fifty-eight patients with PsA (Moll and Wright criteria), 35 men and 23 women, mean age of 45, 14, were studied; all the patients were assessed by both clinical and radiological examination, with particular attention to the sacro-iliac joints. HLA typing of the patients confirmed the association between PsA and HLA-B39 ($p = 0.0008$) and Cw6 ($p = 0.0011$). In addition a significant increase in DQ2 antigen ($p = 0.004$) has been found. No correlation of any particular HLA antigen with clinical subsets (oligo-polyarticular peripheral PsA, axial PsA and axial with peripheral PsA) or erosive incidence of joint involvement – generally related to the duration of the disease – was found. ***Key words: psoriatic arthritis; HLA antigens; disease severity.***

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Increasing numbers of clinical, radiographic, serologic and epidemiological studies support the nosographic classification of psoriatic arthritis (PsA) as a true entity (1). Despite regarding Moll and Wright criteria as useful for diagnosis, a global definition of PsA may be "a syndrome in which psoriasis is associated with inflammatory arthritis, usually having a negative sheep cell agglutination test" (2, 3, 4).

The pathogenesis of PsA is still to be elucidated, but it is certain that genetic, environmental and immunologic factors are thought to be prominent in the development and perpetuation of the disease (5, 6). The opportunity to observe a clinical subset of rheumatoid arthritis-like PsA, in any case seronegative for rheumatoid factor, has stimulated the study of the genetic differences between PsA and rheumatoid arthritis (RA): psoriasis and PsA are associated with both Class I (B13, B17, B39, B27, Cw6) and Class II (DR4, DR7) HLA antigens, while RA is associated only with Class II antigens (DR4, DQ3) (1, 7).

While for other rheumatic diseases the role of the MHC in autoimmunity is hypothesized, the mechanism of the MHC association with PsA is still unknown (1). Contradictory is the association of PsA with DR and DQ antigens; DR4 in association with severity of the disease (8) and rheumatoid arthritis-like disease (8, 9); DR4 and DQ53 with earlier age of onset of arthritis (3). A9 and B5 antigens are associated with a higher radiological lesion (erosions) (3), A9 antigen with a later onset of psoriasis. Finally, there is a significant increase in A1, B38 in patients with asymmetric peripheral disease and B39 with symmetric peripheral disease (3). From the clinical and radiological points of view it has been noted that, in PsA, joint damage tends to occur early in the course of the disease (4); recent clinical studies suggest that PsA may be as destructive as RA (10, 11), but with the occurrence of both joint lysis and ankylosis in various joints in the same patient (1). Our aim is to contribute to the clinical and radiological physiognomy of PsA and to report the HLA antigens distribution in our patients and the possible association between clinical subgroups and HLA antigens.

MATERIALS AND METHODS

Fifty-eight italian outpatients with PsA were studied. The diagnosis was established according to the Moll and Wright criteria (2). All patients had cutaneous and/or nail psoriasis and underwent X-ray examination of hands and wrists, feet, spine and sacro-iliac joints. Modified ARA criteria of RA were used to document the radiological stage of each joint (4). Axial and sacro-iliac radiological involvement was evidenced

Table I.

	PsA (total)	Peripheral disease		Axial disease	
		Oligo-arthr.<5	Poly-arthr. ≥5	With periph. involv.	Without periph. involv.
No. of patients	58	29	23	4	2
Female/male	23/35	15/14	8/15	0/4	0/2
Mean age (years)	45.17	41.79	48.56	50.00	45.50
Mean age of onset PsA (years)	42.46	40.24	44.69	46.50	41.00
Mean duration of P. (months)	96.90	65.10	125.74	201.00	18.00
Mean duration of arthr. (months)	33.52	19.45	48.00	42.00	54.00
Family history of P. (%)	34.48	34.48	34.78	33.33	
Nail lesions	63.79%	16 pts	18 pts	2 pts	1 pt

Table II. *Radiological features of the 58 patients*

	PsA (total)	Peripheral disease		Axial disease	
		Oligo-arthr.	Poly-arthr.	With periph. involv.	Without periph. involv.
No. of patients	58	29	23	4	2
* X-ray stage:					
I		6/29	3/23	1/4	–
II		8/29	5/23	2/4	–
III		15/29	13/23	0/4	–
IV		–	2/23	1/4	–
Enthesitis	22.41%	3	8	2	–
Sacro-iliac joint inv.	43.10%	11	8	4	2

* Modified ARA criteria were used to document the radiological stage of each joint (radiographs of hands and feet only).

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Table III.

	Controls		Patients		*p*-value	RR
	n	%	*n*	%		
Locus B	(116)		(58)			
B13	7	6	6	10.34	ns	–
B17	14	12.1	12	20.69	ns	–
B27	7	6	5	8.6	ns	–
B38*	6	5.2	7	12.6	ns	–
B39*	1	0.8	8	13.79	$p=0.0008$	18.1
Locus C	(114)		(44)			
Cw6	20	17.5	19	43.18	$p=0.0011$	3.6
Locus DR	(116)		(56)			
DR2	32	27.6	8	14.28	$p=0.038$	0.4
DR3	12	10.3	12	21.43	$p=0.049$	2.2
DR7	33	28.4	21	37.50	ns	–
Locus DQ	(116)		(54)			
DQ2	36	31	29	53.7	$p=0.004$	3.2

* $n=114$

through the New York criteria and/or syndesmophyte presence. Clinical subsets of PsA were: oligo-polyarticular peripheral; axial; axial with peripheral joint involvement. HLA A, B and C antigens were typed by the standard NIH microlymphocytotoxicity technique (12); HLA DR and DQ antigens were typed by a prolonged cytotoxicity technique (13) on B lymphocyte purified preparation (14). Typing sera were selected from the 9th and 10th International Histocompatibility Workshop, and correlated sera were also used.

Data analysis was carried out using Fisher's exact test to compare the frequencies of HLA antigens. Relative risks (RR) were calculated using the Woolf method.

RESULTS

Clinical features of 58 patients with PsA are reported in Table I.

Radiologic features of our 58 patients are given in Table II.

The distribution of HLA antigens in our 58 patients of PsA, compared with normal controls, is shown in Table III.

DISCUSSION

Our data confirm the prevalence of psoriatic onset in the natural history of PsA and the importance of a family history of psoriatic disease. Psoriatic onychopathy was observed in 63.79% of total PsA, peripheral and/or axial not necessarily associated with distal interphalangeal articular involvement. From the radiological point of view, sacro-iliac involvement in our patients was 43.10%, with evidence of this location not only in the axial subgroup; enthesopathy was present in 22.41% of our patients. X-ray joint lesions, stages III and IV of modified ARA criteria, were present in 55.35% of our patients with an association with major duration of the disease (mean duration of 43.74 months in the patients with erosions versus a mean of 19.20 months in those without erosions). HLA typing of our patients confirms, according to other authors (1, 7), the increased association between PsA and HLA-B39 ($p=0.0008$) and Cw6 ($p=0.0011$) and for the II Class of antigens a significant increase in DQ2 antigen ($p=0.004$), the latter in partial contradiction of other reports (3). Despite the experience of other authors (1, 3), there was not significant statistical correlation between PsA and HLA-DR antigens either. The presence of HLA-B27 antigen was limited and not only associated with the 'axial' patients. No correlation of any particular HLA antigen with our clinical subgroups (oligo-polyarticular peripheral PsA, axial PsA and axial with peripheral PsA) was found, presumably due to the small number of patients in each subgroup.

We can conclude by underlining the importance of psoriatic familiarity and the presence of HLA-B39, Cw6 and DQ2 antigens, in order to define a 'risk' of disease, while the spreading of the radiologic lesions is particularly associated with the duration of PsA.

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