Systemic onset juvenile chronic arthritis, polyarticular pattern and hip involvement as markers for a bad prognosis

C. Modesto[1], P. Woo[2], J. García-Consuegra[3], R. Merino[3], M. García-Granero[4], C. Arnal[1], A.-M. Prieur[5]

[1]Rheumatology Department, Hospital Vall d'Hebron, Barcelona, Spain;
[2]College of London, Pediatric Rheumatology Department, Great-Ormond Street Hospital, London, UK;
[3]Department of Pediatric Rheumatology, Hospital Infantil La Paz, Madrid, Spain;
[4]University of Navarre, Navarre, Spain;
[5]Departement d'Hématologie et Immunologie Pédiatrique, Hôpital Necker-Enfants Malades, Paris, France

Abstract

Objective

To explore all the common clinical and biological variables that are characteristic of Systemic onset Juvenile Chronic Arthritis (SoJCA) in order to determine which of them are suitable as predictors of a bad articular outcome (persistence of inflammatory symptoms and/or established limitation of the range of motion (ROM)).

Material and methods

Clinical charts for 124 SoJCA patients were retrospectively reviewed. From them, 91 were finally included in the study because they had all of the clinical and biological data at disease onset properly recorded. All have been followed for at least 3 years since the beginning of the disease. Data collected at onset, and after 3 and 6 months of the disease included: 1) systemic symptoms; 2) joint involvement, using both the usual articular count and the value of an articular index (Helsinki Index = HI) which intentionally excludes those joints that are not uniformly recorded in clinical charts; and 3) biological data. HI was used to separate the patients into two groups. When applied 3 years after the disease onset, HI ≥ 10 represented a bad articular outcome while HI < 10 meant a good prognosis. SPSS for Windows 6.1 was used for both the univariate and multivariate analyses.

Results

From the multivariate logistic regression analysis, two different "clusters" of clinical data were found to be the best predictors of a bad articular outcome. A bad prognosis was linked at onset with the presence of generalized lymphadenopathies, age < 8 years and an HI > 6; at six months a bad outcome was linked with the presence of a polyarticular pattern plus hip involvement.

Conclusion

Clinical parameters at the beginning of the disease were shown to be extremely useful in predicting the articular outcome of SoJCA. Therefore, they could constitute a good instrument to help clinicians tailor the best therapy for their patients.

Key words

Juvenile chronic arthritis, childhood, clinical findings, articular outcome, treatment.

C. Modesto, MD, PhD, Pediatric Rheumatologist, formerly Associate Professor of Pediatrics at the University of Navarre;
P. Woo, MD, PhD, Professor of Pediatric Rheumatology, London, UK;
J. García-Consuegra, MD, Director of the Dept. of Pediatric Rheumatology, and R. Merino, MD, Hospital Infantil La Paz;
M. García-Granero, PhD, Statistician attached to the University of Navarre;
C. Arnal, MD, Chief of Rheumatology Department, Hospital Vall d'Hebron;
A.-M. Prieur, MD, Staff member, Hôpital Necker-Enfants Malades.

Please address correspondance and reprint requests to: Consuelo Modesto, MD, Departamento de Reumatología, Hospital General Vall d'Hebron (tercer piso), Paseo de la Vall d'Hebron 119-229, Barcelona 08035, Spain.
E-mail: cex3pi18.hmi@cs.vhebron.es.

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Introduction

Systemic onset Juvenile Chronic Arthritis (SoJCA) accounts for only 10 to 30% of the patients with JCA (JRA in North America). However, although half of these children will be free of symptoms, another 50% will continue to suffer progressive, destructive arthritis, becoming moderately to severely handicapped by the end of their childhood (1-3).

The majority of pediatric rheumatologists would agree, on the basis of their clinical practice, that the number of joints involved at the beginning of the disease is an important factor to be taken into account when predicting the articular outcome for a particular patient. Since clinical and biological data can now be analyzed and predictions can be made more accurately than before using computerized single and multiple variable regression analyses, we decided to test this assertion by studying in a large population the prognosis indicators of bad outcome already found by other groups (4-9), and also try to establish distinct patterns of presentation linked to a good or bad articular outcome in order to facilitate decisions regarding therapy.

Recently different therapeutical approaches have been tailored for the initial treatment of SoJCA (10). Some ignore the classic recommendations (11) for an increasingly aggressive therapy using immunomodulating drugs (alone or in combination) from the beginning of the disease. In addition, autologous bone marrow transplantation or stem cell transplantation have been included in the list of therapeutical tools to be applied in the most severe cases (12, 13). We share the opinion of other authors (14) of the importance of developing methods whereby the assessment of a poor articular prognosis can be accomplished early in the disease course. The goal of our study was to find a method of predicting a bad articular outcome in SoJCA, in order to gain a better understanding of the appropriateness of using aggressive treatments.

Patients and methods

Patients

Data was collected on patients from three different hospitals: Hôpital Necker-Enfants Malades (Paris), Great Ormond Street Hospital (London) and Hospital Infantil La Paz (Madrid). A total of 124 charts were reviewed, all from children who met the EULAR criteria for SoJCA (similar to the ACR criteria for JRA) (15, 16).

Thirty-three charts that did not contain all of the clinical and laboratory data at onset had to be excluded. These patients did not differ in their clinical presentation from those finally included in the study. They constituted quite a randomized sample (different outcomes, different onset, different treatments) and therefore, from a statistical point of view, their exclusion would not introduce any sample bias in the study.

Ninety-one patients could be included because: 1) they fulfilled the classification criteria of the EULAR for SoJCA; 2) they have been followed for at least three years after the initial diagnosis; and 3) all of the clinical and laboratory data at the time of diagnosis had been properly recorded. When a diagnosis was made outside the three participating hospitals, original records were kindly sent to us by the pediatricians who initially followed those patients. Among the 91 children, 43 were boys and 48 girls. The mean age at onset was 5.65 ± 3.84 years (m ± SD) (range: 4 months - 15.58 yr.). The mean follow-up period was 8.57 ± 4.87 years (range: 3.00 - 23.9 yr.). Fifty-six had received steroid treatment during the first six months of the disease (see below).

Clinical and biological data

All data was recorded retrospectively by two of us (CM; RM). The first author directly reviewed all charts from the French and English children; the second those from the Spanish group. The following clinical data were recorded at diagnosis and after 3 and 6 months: 1) presence of fever; 2) rash with"rheumatoid" characteristics (pinkish, evanescent rash); 3) hepatomegaly and/or splenomegaly; 4) generalized lymphadenopathy (more than two lymphadenopathy groups); 5) arthritis; 6) serositis; 7) other symptoms (abdominal discomfort, diarrhea, vomiting, malaise, weight loss, myalgia, lethargia,

Table I. The Helsinky Index, a retrospective articular index for the classification of patients with systemic onset juvenile chronic arthritis.

Joint	Swelling	ROM	Max. score
Neck		1	1
Shoulder (R/L)	1/1	1/1	4
Elbow (R/L)	1/1	1/1	4
MCPs (R/L)	1/1	1/1	4
PIPs (R/L)	1/1	1/1	4
Hip		2/2	4
Knee (R/L)	1/1	1/1	4
Ankle (R/L)	1/1	1/1	4

Maximum index = 33 (those joints that were not uniformly mentioned in the clinical charts were excluded). R = right; L = left.

anorexia, cough, sore throat).

Joint involvement was recorded in two different ways based on the number of active joints: 1) following the established subdivision into the oligo/pauciarticular and polyarticular patterns (involvement of < 4 joints, or ≥ 5 joints); and 2) using an articular index, created for us and named the Helsinki Index because it was presented for the first time at the Fourth European Conference on Pediatric Rheumatology in Helsinki in 1996. This index (Table I) constitutes an arbitrary tool for performing a quantification of the number of affected joints (swelling, limitation of motion). The Helsinki Index purposely excludes those joints that are not uniformly mentioned in medical records and those which cause the most debate among observers (small joints of the feet, subtalar). For the same reason, MCPs and PIPs (hands) have been counted as a single joint, regardless of the exact number of affected fingers. Also, the degree of involvement (mild, moderate or severe) had to be obviated for the sake of simplification. The correlation of the HI with other standard measures of disease activity (number of swollen joints, number of tender joints, number of joints with limitation of motion, physician's global assessment, patient/parent's global assessment) is being studied at present in a different population of JCA patients.

All patients were classified as having a good (group A) or a bad articular outcome (group B) according to the following criteria based on the HI scores: a) HI after 3 years of follow-up < 8 was interpreted as a good articular outcome (group A); b) HI after 3 years of follow-up ≥ 10 signified bad or poor articular outcome (group B); and c) HI after 3 years equal to 8 or 9. These patients were classified according to the disease behavior during the years up to the last visit. If the HI had a tendency to increase they were classified as having a "bad" articular prognosis; if they improved they were classified as having a "good" articular prognosis. This group included only 7 of the 91 patients.

The HI usually reflects the pattern of articular involvement, with patients having HI ≥ 6 being classified in the polyarticular group. However, the two classifications do not always overlap. For example, if both hips are involved, we can find an HI = 6 with only 3 joints affected (i.e., both hips and a knee) and therefore an oligoarticular pattern. It is for this reason that the HI and the articular pattern were used separately in the multivariate analysis.

We collected the following laboratory data at the time of diagnosis, and after 3 and 6 months: hemoglobin level, leukocyte count, platelet count, polymorph count, ESR, CRP, immunoglobulins, ANA, and rheumatoid factor. Unfortunately, CRP and immunoglobulin values were not always available at 3 and 6 months after onset of the disease.

Statistical analysis

All data were entered in a database for computerized analyses using SPSS 6.1 for Windows. Independent t-tests for normally distributed variables, the Mann-Whitney U test for non-normally distributed variables, the chi-square test and Fisher's exact test were used to compare the groups with a good or bad prognosis. To identify the predictors of a bad articular outcome, single logistic regression analysis (univariate) was applied at the first step; independent variables that showed significant results using univariate tests underwent multiple logistic regression analysis (multivariate) (stepwise, unconditional, model computed) (17). Cut-off points were automatically selected by the SPSS program throughout the study. All models obtained from the multiple logistic regression analysis, if clinically applicable, were evaluated as possible predictors of a bad articular outcome (18). Their sensitivity and specificity were also automatically calculated by the statistical program, using a cut-off point of 0.5.

Results

Differences at onset

There were no differences between the two groups regarding sex, presence of fever (which was mandatory for the diagnosis), rash, hepatomegaly or splenomegaly. However, the age at onset differentiated the group with a bad prognosis. The younger children had a higher risk of a bad outcome ($m \pm SD$ 4.73 ± 3.49 vs 6.94 ± 3.98; $p < 0.003$) (Table II). Generalized lymphadenopathy was present in 51% (25) of the patients in group B, and in 24% (9) in group A ($p = 0.01$). The presence of lymphadenopathy at onset increased the risk of a bad articular prognosis by more than three-fold (OR = 3.35).

HI was statistically higher in the group with a bad prognosis (5.65 ± 4.51 vs. 3.13 ± 2.66; $p = 0.009$). Although differences in the articular pattern did not reach statistical significance, the polyarticular presentation showed a tendency to be associated with a bad outcome, with 79% (23/29) of the polyarticular patients in the group with a bad articular outcome. Hemoglobin values were significantly lower in group B (93.3 ± 15.5 vs. 105.2 ± 14.8; $p = 0.001$). The other biological data recorded showed no differences between groups.

Logistic regression-single element analysis established a significant relative risk of poor outcome for the following variables: Onset under the age of 8 was associated with a 2.8 times higher risk for each year under this age ($p = 0.028$); HI > 6 had a risk 2.97 times higher for each point above this figure ($p = 0.029$); A polyarticular pattern multiplied by four the risk of a bad outcome compared with an oligoarticular pattern ($p = 0.004$); The presence of generalized lymphadenopathy increased the risk

Table II. Clinical and biological data at onset (Group A = good prognosis, group B = bad prognosis).

Variable	Group A (m ± SD)	Group B (m ± SD)	P	OR (95% CI)
Age at onset	6.94 ± 3.98	4.73 ± 3.49	**0.003**	0.855 per year (0.76 - 0.96)
Sex (F/M)	23/15	25/28	NS	
Fever (inclusion criteria)	38 (100%)	53 (100%)	NS	
Rash	34 (89%)	41 (79%)	NS	
Hepatomegaly	4 (11%)	9 (18%)	NS	
Splenomegaly	7 (19%)	11 (22%)	NS	
Lymphadenopathy	9 (24%)	25 (51%)	**0.01**	3.35 (1.32 - 8.54)
Serositis	5 (14%)	8 (16%)	NS	
Helsinki Index (HI)	3.13 ± 2.66	5.65 ± 4.51	**0.009**	1.21 per unit (1.06 - 1.40)
Articular pattern Oligoarticular Polyarticular	 32 6	 30 23	**0.06**	 4.1 (1.46 - 11.42)
Hemoglobin	105.2 ± 14.8	93.3 ± 15.5	0.001	0.60 per 10 units (1.43 - 0.83)
Leucocytes	18.3 ± 17.4	19.4 ± 12.0	NS	
Polymorphs	13.9 ± 6.9	14.4 ± 11.8	NS	
Platelets	507.2 ± 163.3	579.3 ± 243.9	NS	
ESR	81.44 ± 32.03	84.39 ± 30.42	NS	
CRP	8.3 ± 5.8	10.4 ± 6.3	NS	
IgG	11.74 ± 3.79	15.67 ± 7.29	NS	

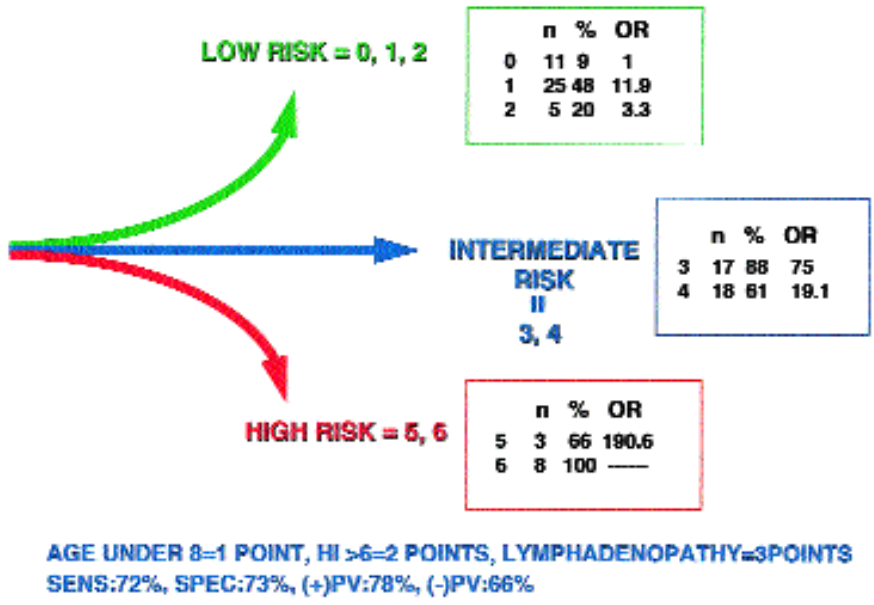

Fig. 1. Simplified model of predictive factors in systemic onset juvenile chronic arthritis *(see text)*.

3.35 times (p = 0.008); finally, hemoglobin values < 100 gr/L made the risk 3.4 times higher for every 10 units under this limit (p = 0.009).

Multiple logistic regression analysis can find which combination of risk factors,if any, is more predictive than each of those factors individually. Applied to the variables at onset, this analysis indicated that the best combination to predict a bad outcome was: the presence of generalized lymphadenopathy + age at onset + HI (Helsinki Index) (sensitivity: 79%, specificity: 60.5%; positive predictive value: 71%; negative predictive value: 70%). No other combination, with or without laboratory data, added any predictive power to the individual variables. In this model, those patients older than 8 years of age at disease onset, without lymphadenopathies and having an HI ≤ 6 were at lower risk and were considered as having an OR = 1. The highest risk was represented by those children younger than 8, with generalized lymphadenopathies and an HI > 6 (100% of the patients with a bad outcome; the OR could not be calculated) and those more than 8 years of age, with generalized lymphadenopathies and HI > 6 (OR = 190.62).

All possible combinations of the three variables included in the model at onset are presented in simplified form in Figure 1, assigning different values to the presence of each risk factor: a) age under 8 = 1 point; b) HI > 6 = 2 points; c) generalized lymphadenopathy = 3 points. Adding the different values, sums equal to 0, 1 or 2 would represent a low risk; sums equal to 3 or 4 an intermediate risk and sums equal to 5 or 6 a high risk for the development of bad articular disease. For example, a 9-year-old child without lymph nodes and HI > 6 will have a total value of 2, and would therefore be in the low risk group. In contrast, a 4-year-old boy with HI > 6 and lymphadenopathies will have a total value of 6 and should be included in the high risk group. This simplified model has a sensitivity of 72%, a specificity of 73%, a positive predictive value of 78% and a negative predictive value of 66%.

Fifty-six patients received steroid treat-

ment during the first six months of the disease. Most of them initiated steroids at 2 mg/Kg/day of prednisone (m ± SD 35.04 ± 15.61) with a total dose that varied from 10 to 60 mg per day, followed by a conventional schedule of tapering. Single and multiple logistic regression analysis showed similar results before and after the introduction of the variable "steroid treatment".

Differences at three months

73% of the patients with a bad outcome continued to have fever after 3 months of disease compared with 32% in the group with a good outcome (p = 0.008). Generalized lymphadenopathy differentiated both groups, being present in 33% of the patients in group B and only in 6% of those in group A (p = 0.005). HI was clearly higher in group B: 6.56 ± 5.84 vs. 2.84 ± 3.77 (p = 0.001), and the articular pattern achieved statistical significance when a bad articular outcome was associated with a polyarticular presentation (p = 0.01). Hepatomegaly was present in 48% of the patients in group B and only in 1 patient in group A (9%) (p = 0.04).

Of the biological data, only lower hemoglobin values were associated with a bad prognosis (96.8 ± 21.7 vs. 107.8 ± 17.3; p = 0.04). The leukocyte count, platelets and ESR were only slightly different between groups.

Logistic regression-single element analysis established a significant relative risk for the following variables: presence of fever (OR = 3.46) (p = 0.008); hepatomegaly (OR = 8.10) (p = 0.01); generalized lymphadenopathy (OR = 7.24) (p = 0.003); HI > 6 (OR = 4.86) (p = 0.001); polyarticular pattern (OR = 3.7) (p = 0.007); and hemoglobin values < 110 (OR = 3.6) (p = 0.04).

Multiple logistic regression analysis was not performed at this time.

Differences after 6 months of onset

After six months of disease, the variables directly related with joint involvement proved to be the most clearly associated with poor prognosis. HI was three times higher in group B (9.21 ± 7.11 vs. 3.42 ± 3.68; p < 0.001) and 92% of the patients in this group were already showing a polyarticular pattern (p < 0.001). Fever, rash, and hepatomegaly/splenomegaly did not differ between groups (Table III). Generalized lymphadenopathy continued to be present more often in group B (34% vs. 9%; p = 0.005). At this time we studied the impact of some specific joints on the prognosis. Hip and neck involvement present at any time during the six first months of disease were associated with bad prognosis (hip p = 0.002; neck p = 0.01).

All of the biological data recorded, except for the leukocyte count, showed significant differences. Hemoglobin continued to show lower values in the group with a bad prognosis, (93.7 ± 20.2 vs. 104.5 ± 13.2; p = 0.007), while the platelet count, ESR and CRP values were higher in this group.

Logistic regression-single element analysis established some factors of risk: Generalized lymphadenopathy (OR = 5.65) (p = 0.003); HI > 6 (OR = 8.43) (p < 0.0001); Polyarticular pattern (OR = 26.24) (p < 0.0001); Hip involvement (OR = 5.57) (p = 0.001); Neck involvement (OR = 3.13) (p = 0.01); Platelets > 450.000 (OR = 4.21) (0.01); ESR > 30 (OR = 3.91) (p = 0.01); CRP > 30 (OR = 6.42) (p = 0.01); and hemoglobin values <100 (OR= 2.75) (p = 0.03). Multiple logistic regression analysis found that the best predictive combination after 6 months of disease was: articular pattern+hip involvement (sensitivity:81%, specificity:81%, positive predictive value: 86%, negative predictive value: 76%). This model was more predictive than the model including HI. Low risk is represented by an oligoarticular pattern without involvement of the hip (OR = 1). As in the model at onset, even the low risk group had a

Table III. Clinical and biological data at six months.

Variable	Group A	Group B	P	OR (95% CI)
Fever	15 (40%)	28 (53%)	NS	
Rash	15 (40%)	18 (34%)	NS	
Hepatomegaly	7 (19%)	9 (17%)	NS	
Splenomegaly	4 (11%)	10 (19%)	NS	
Lymphadenopathy	3 (9%)	18 (34%)	**0.005**	5.65 (1.52 - 20.98)
Helsinki Index	3.42 ± 3.68	9.21 ± 7.11	**< 0.0001**	1.24 per unit (1.11 - 1.38)
Articular pattern				
Oligoarticular	35	3	**< 0.0001**	26.24
Polyarticular	16	36		(7.03-98.05)
Hemoglobin	104.5 ± 13.2	93.7 ± 20.2	**0.007**	0.70 per 10 units (0.53 - 0.94)
Leucocytes	13.5 ± 6.1	16.2 ±7.4	NS	
Polymorphs	9.0 ± 5.8	11.2 i 7.2	NS	
Platelets	518.5 ± 190.6	668.2 ± 288.6	**0.04**	1.28 per 10^5 (1.04 - 1.59)
ESR	48.81 ± 37.85	65.66 ± 30.52	**0.04**	1.01 per unit (1.001-1.07)
Hip involvement (6 mos.)	4 (10%)	21 (39%)	**0.002**	(1.73 - 18 02)
Neck involvement (6 mos.)	10 (26%)	28 (53%)	**0.01**	3.13 (1.27 - 8.29)
CRP	5.5 ± 5.8	10.4 ± 6.0	**0.01**	1.01 per unit (1.002 - 1.03)

number of patients (24% at this time) that suffered significant joint damage later in the disease. All of the patients in the high risk group (polyarticular pattern + hip involvement) had a poor articular outcome (statistically, the OR cannot be calculated). Intermediate risk is represented by those patients with an oligoarticular pattern, but with hip involvement (OR = 5.5) and those with a polyarticular pattern without hip involvement (OR = 26.6).

Differences at last visit

Data available at the last visit suggests that the grouping made after three years of follow up using the HI corresponds well with the later outcome. HI was clearly higher in group B (16.56 ± 5.27 vs. 2.54 ± 3.29) ($p < 0.0001$). Almost all of the patients in group B were still on steroids (88% vs. 40% in group A), 60% vs. 46% were under immunosuppressive treatment ($p = 0.006$) and 96% vs. 52% continued taking NSAIDs ($p < 0.0001$). This difference in treatment, despite the differences in therapeutic approach among rheumatologists, reflects a more severe disease in group B. The differences in ESR (47.42 ± 32.19 vs. 20.97 ± 24.64; $p < 0.0001$) and hemoglobin values (111.6 ± 17.9 vs. 123.0 ± 16.0; $p = 0.002$) between the two groups continued to be statistically significant.

Discussion

SoJCA is the less common form of JRA, but the one that leads to a higher number of handicapped patients (1, 2). The natural history of the disease does not seem to be affected by steroid treatment, although steroids can ameliorate the symptoms (4). Recently some authors reported a few cases treated almost from the disease onset with immunosuppressors, thus casting doubt on the classical pyramid of treatment (10). Their rationale was to stop the inflammatory process as soon as possible by using more efficacious drugs for a relatively short period of time, even if this involved the risk of some potentially serious side effects. On the other hand, we already know that if immunosuppressors are used late in the disease, the clinical response is usually very poor (19, 20).

Our main goal was to elaborate a guide, simple enough to be used in daily clinics to differentiate between patients with a good or bad articular outcome. For this reason, although differences in individual variables were also recorded, the most important point was the creation of two alternative models (onset, six months) to predict articular outcome in SoJCA.

Knowing all of the limitations of a retrospective study, due to the low incidence of SoJCA (only 10 to 30% of the total number of JCA patients), even gathering data from three different hospitals, it was not possible in practice to conduct a prospective study. Also, it may be argued that the population included in this project was not selected in a completely unbiased way. Certainly, our pool of patients only included those who had been sent by their pediatricians to specialized children's hospitals equipped with a pediatric rheumatology department. However, we do not believe that this problem in the recruitment of patients invalidates our results for two reasons: a) most patients with SoJCA, anywhere in the world, will be sent to one of those hospitals at some point, even if not at the disease onset; b) we were attempting to create a diagnostic tool appropriate for this type of patient, i.e. the child with potentially severe JCA who is referred to a children's hospital. It may be pointed out that most of the studies of prognostic factors carried out so far have exhibited the same limitations (9, 21, 23).

Because we were aware that having many observers could result in diversification, we created a "simplified" articular index, or joint count index to minimize inter-observer variations. We knew that at the physical examination of a child with JCA the exact number of MCP or PIP joints involved at one specific moment may differ enormously among observers. However, these differences are less significant if we evaluate, for example, the range of motion of the hip. The HI was created initially to be used retrospectively on the charts of patients followed for long periods of time. As mentioned before, we are currently validating the HI on a different population of JCA patients, and hopefully it will be found suitable for use in prospective studies, as many of our colleagues have proposed at international conferences.

In our opinion, the simplified model at disease onset seems to be very useful. Age under 8 years, HI over 6 and the presence of generalized lymphadenopathy are the factors of risk. All children under 8 years of age with one of the two other factors are at moderate or high risk of a bad articular outcome. For children over 8 years of age, the presence of lymph nodes, a common finding in younger children but quite rare above this age, is always related to a bad articular outcome. It must be mentioned that our multiple logistic regression analysis found a negative association between age and the presence of lymph nodes. For this reason, the presence of generalized lymphadenopathies in children less than 8 years of age modifies only moderately their risk of a bad outcome, and they should be placed in the intermediate risk group. On the basis of this alternative model, for those children belonging to the high risk group an aggressive treatment, including immunossupressive therapy, should be considered.

Schneider *et al*. (9) proposed the application of their prognostic factors to larger populations to test their reproducibility. All of the variables found to be predictive in their study at six months, with the exception of the persistence of systemic symptoms and the leukocyte count, were also predictive in our study. Single element logistic regression analysis showed the articular pattern to be the best predictor at this time. This was also the case in Schneider's study, but here the platelet count $\geq 600 \times 10^9$/L and persistent systemic symptoms were the most predictive variables. In our series, the cut-off point for the platelet count was set automatically by the program at 450×10^9/L. We did not find the persistence of systemic symptoms to be predictive, probably because we used a different definition for persistent fever than Schneider did. We always considered a patient to be positive for this variable if it was present clinically, regardless of

the treatment at that moment. For Schneider, a patient had the persistent systemic symptom of fever if he/she needed steroids to control it. In our series, data at 3 months showed that 35 children in group B (73%) had persistent fever, but only 28 were still symptomatic at 6 months. Most of the children that become afebrile at 6 months had been placed on steroid treatment between 3 and 6 months of the disease. Therefore, if we had used exactly the same criteria as Schneider *et al*. did, our results probably would not have been so different.

More recently, the same authors (24) validated their prognostic criteria by applying them and the Childhood Health Assessment Questionnaire to a larger population. We focused our study on the articular outcome. We agree with others (9, 22) that a bad articular outcome does not necessarily mean a bad functional outcome. However, at the time that our study was performed, validated functional scales were not available for the French and Spanish populations. Differences with the results obtained by other authors (3, 14, 22, 25) are not easy to establish, especially because they studied different JCA subgroups, not specifically So-JCA.

Multiple logistic regression analysis at six months found the combination of articular pattern and hip involvement to be more predictive than either of these variables alone. Other combinations, including biological data, did not add any particular advantage to the articular pattern. Both the sensitivity and specificity were around 80% in this model, with a positive predictive value of 86%, which gives enough strength for the test to be reliable. We found this model easy to apply, with no added difficulty than the performance of a thorough physical examination. Statistical analysis could have drawn completely different conclusions; however, it has proved what experienced cliniciansshave known for a long time. These results are preliminary and will have to be tested in a different population in order to achieve validation.

It seems to be evident to the majority of pediatric rheumatologists that current approaches to the treatment of arthritis in children are still insufficient to stop the course of this destructive disease (13). We need new strategies and probably a more aggressive approach. To achieve this, the possibility of predicting the articular outcome at the beginning of the disease will be essential.

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