A Retrospective Study on Possible Predictive Factors for Long-term Temporomandibular Joint Degeneration and Impaired Mobility in Juvenile Arthritis Patients

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Aims: To determine possible predictive factors for long-term temporomandibular joint (TMJ) degeneration and dysfunction in juvenile idiopathic arthritis (JIA) patients. **Methods:** A total of 94 patients (77% female) who had received a JIA diagnosis in an outpatient rheumatology clinic from 1993 to 1994 at a mean ± standard deviation age of 8.3 ± 4.53 years were included in the study. At inclusion, TMJ status regarding condylar degeneration was assessed orthopantomographically and given a Rohlin and Petersson score of 0 or ≥ 1. The maximal mouth opening (MMO) was also measured. Data on possible predictors were gathered retrospectively from the consultation at intake: gender, age at JIA onset, JIA subtype, physical limitations (ie, a Steinbrocker classification score of 0 or ≥ 1), human leukocyte antigen-B27, and antinuclear and rheumatoid factors. Disease duration and medication type were also considered. Associations between all of these factors and long-term condylar degeneration and MMO were assessed by using single and multiple regression analyses. **Results:** Long-term TMJ degeneration and smaller MMO were both associated with younger age at JIA onset ($P = .01$; $P = .03$) and longer disease duration ($P = .05$; $P = .002$). Moreover, MMO was negatively associated with physical limitations at intake ($P = .04$). **Conclusion:** Within the limitations of this retrospective study design, these results suggest that young JIA patients with early physical limitations and prolonged disease are at risk of long-term TMJ degeneration and impaired mobility. *J Oral Facial Pain Headache 2017;31:165–171. doi: 10.11607/ofph.1656*

Keywords: *juvenile idiopathic arthritis, long-term, mandibular function, predictive factors, temporomandibular joint*

Juvenile idiopathic arthritis (JIA) is persisting arthritis of unknown etiology that begins before the patient's sixteenth birthday. JIA manifestations include chronic synovitis, joint degeneration, decreased joint mobility, and growth disturbances, and can lead to reduced quality of life.

In 55% to 78% of JIA patients, inflammation also occurs in the temporomandibular joint (TMJ),[1,2] a joint that is important for mandibular growth and essential jaw tasks (eg, chewing and talking). TMJ arthritis can result in mandibular underdevelopment, facial asymmetry, and limited mouth opening.[2] Unfortunately, these consequences often become apparent late, as TMJ degeneration usually occurs silently and without pain.[2] In time, condylar regeneration may occur,[3,4] but TMJ abnormalities usually remain, with consequences into adulthood.[4,5]

To prevent lifelong orofacial complications, it is important to identify JIA patients at risk of long-term TMJ degeneration and jaw dysfunction. Current data on predictive factors are scarce and contradictory. Bakke et al[5] reported that long-term radiographic signs of TMJ degeneration and limited mouth opening in JIA patients were positively associated with disease duration; however, a study by Arvidsson et al[4] found no association of radiographic TMJ degeneration with disease duration, but rather with the presence of general physical limitations at intake into the study.[6]

Therefore, the aim of the present study was to determine predictive factors for the presence of long-term TMJ degeneration and mandibular dysfunction in JIA patients.

Materials and Methods

This retrospective study was conducted according to the Declaration of Helsinki and approved by the review board of Academic Centre for Dentistry Amsterdam.

Participants

The database of an outpatient rheumatology clinic was searched for patients who had been diagnosed with JIA from 1993 to 1994 (intake time, T0) and were under 30 years of age at the beginning of the present study (inclusion time, T1). The search retrieved 94 subjects (mean ± standard deviation [SD] age 17.65 ± 6.69 years at inclusion; 72 [77%] female). Informed consent was obtained for the clinical and radiographic examination of the masticatory system and the use of the available data in the patient file for retrospective analysis. All subjects consented for participation in the study.

Procedures

Previously, at T0 in the outpatient rheumatology clinic, the patients underwent a rheumatologic examination. No clinical or radiographic examination of the masticatory system was performed at that point in time. Among other factors, general physical limitations were assessed using the Steinbrocker classification, where 0 = normal function; 1 = limitations with adequate capacity to conduct normal activities; 2 = little or no capacity to conduct daily activities; and 3 = largely incapacitated, bedridden, or confined to a wheelchair.[6] Blood was tested for, among others, antinuclear factor (ANF), rheumatoid factor (RF), and human leukocyte antigen (HLA)-B27. Juvenile arthritis and its subtypes (ie, oligoarticular, polyarticular, systemic) were diagnosed according to Brewer et al.[7] Further, an individually tailored treatment was applied: nonsteroidal anti-inflammatory drugs (NSAID), disease-modifying anti-rheumatic drugs (DMARD), and/or immunosuppressive drugs (ISD). None of the patients were treated with TMJ intra-articular corticosteroid injections. The presence of inflammatory disease activity was monitored over time.

Patients were re-examined by a rheumatologist on average 8.4 ± 5.8 years after initial diagnosis. At this time (T1), a dental specialist examined the masticatory system by, among other methods, measuring the active maximal mouth opening (MMO) between the incisal edges in mm using a ruler. The overbite (ie, the overlap of the upper over the lower incisors when occluding the jaws) was added to this measurement. TMJ radiographic examination was performed by using an orthopantomogram (OPG). Condylar images were assessed by one examiner using the Rohlin and Petersson score, where 0 = normal morphology; 1 = slight abnormality; 2 = early destructive abnormality; 3 = moderate abnormality; 4 = severe abnormality; and 5 = mutilating abnormality.[8] The intra-observer reliability was assessed by scoring all OPGs twice, 2 weeks apart.

Statistical Analyses

In order to estimate the prevalence of mouth opening limitation in the study sample, the MMO measurements were compared to proposed cut-off values. For subjects below 18 years of age, normal values for Caucasian children and adolescents from the study by Müller et al[9] were used as the reference standard. MMO values smaller than the 10th percentile for MMO of the respective age and gender group were considered limited; for adult subjects, MMO values smaller than 40 mm were considered limited.[10]

For the intra-observer reliability of the OPG scoring, percent agreement and Cohen's kappa were calculated.[11] For this and further analyses, OPG scores were dichotomized: 0 = normal, ≥ 1 = condylar degeneration.

For the statistical analyses, the Steinbrocker et al values[6] were dichotomized because the scores had a skewed distribution, with a disproportional weight on the 0 scores (64 scores of 0 versus 30 scores of ≥ 1). In cases of such skewed distributions, dichotomization is called for. The Rohlin and Petersson scores[8] were also dichotomized because the study aimed only to identify the presence or absence of TMJ degeneration and not its severity.

To determine possible predictors for the presence or absence of condylar degeneration and for the mouth opening capacity at T1, logistic and linear regression methods were used (single and stepwise backward multiple analyses). Independent variables, measured at T0, were: gender, age at JIA onset, JIA subtype at onset, physical limitations (dichotomized Steinbrocker classification of 0 or ≥ 1), RF, ANF, and HLA-B27. To correct for their possible influences, disease duration and type of medication used (NSAID; NSAID and DMARD; NSAID and ISD; NSAID, DMARDS and ISD) were also analyzed. Categorical variables (viz, JIA subtype, physical limitations, RF, ANF, HLA-B27, and medication type) were analyzed using dummy coding. Variables with *P* values of ≤ .10 after single testing were included in multiple testing. The *P* value to exclude during stepwise backward multiple modeling was > .05.

Results

The average age at JIA onset was 8.3 ± 4.53 years (Table 1). At T0, 12 patients had systemic JIA, 27 polyarticular JIA, and 55 oligoarticular JIA. The average disease duration was 8.14 ± 6.07 years.

Table 1 Descriptive Statistics of the Sample of Patients Diagnosed with Juvenile Idiopathic Arthritis (JIA) from 1993 to 1994

Variable	Total group	Subjects without TMJ degeneration (Rohlin and Petersson score = 0)	Subjects with TMJ degeneration (Rohlin and Petersson score ≥ 1)	*P* value
Total participants (n)	94	17	77	
Gender				
Male	22	5	17	.54[a]
Female	72	12	60	
Age at inclusion in the study (mean ± SD) (y)	17.65 ± 6.69	18.5 ± 5.13	17.46 ± 7	.56[b]
Duration of follow-up (mean ± SD) (y)	8.36 ± 5.83	6.41 ± 4.24	8.79 ± 6.06	.13[b]
Age at JIA onset (mean ± SD) (y)	8.3 ± 4.53	11.06 ± 3.07	7.7 ± 4.58	**.001[b]**
Duration of disease (mean ± SD) (y)	8.14 ± 6.07	5.41 ± 4.55	8.74 ± 6.22	**.04[b]**
JIA subtype at onset (n)				
Systemic	12	3	9	.78[a]
Polyarticular	27	5	22	
Oligoarticular	55	9	46	
General functional limitation (Steinbrocker et al classification[6]) (n)				
No	64	13	51	.57[a]
Yes	30	4	26	
Human leukocyte antigen-B27 (n)				
Positive	10	2	8	.98[c]
Negative	49	10	39	
Antinuclear factor (n)				
Positive	19	2	17	.53[a]
Negative	59	11	48	
Uncertain	16	4	12	
Rheumatoid factor (n)				
Positive	6	0	6	.47[a]
Negative	34	7	27	
Uncertain	6	1	5	
Type of medication (n)				
NSAID only	40	9	31	.65[a]
NSAID & DMARD	32	6	26	
NSAID & ISD	9	2	7	
NSAID & DMARD & ISD	13	1	12	
Maximum mouth opening (mean ± SD) (mm)	48.27 ± 10	54.12 ± 6.08	46.97 ± 10.26	**.001[b]**

Data at intake (T0) is shown (gender, age at JIA onset, JIA subtype at onset, general functional limitations, HLA-B27, ANF, and RF), as well as data gathered at inclusion (T1) (number of participants, age at inclusion in the study, duration of disease, type of medication, maximum mouth opening).
[a]Chi-square test.
[b]Independent *t* test.
[c]Fisher's exact test.
Bolded *P* values are statistically significant. SD = standard deviation; NSAID = nonsteroidal anti-inflammatory drug; DMARD = disease-modifying anti-rheumatic drug; ISD = immunosuppressive drug.

The intra-observer agreement for the presence of radiographic degeneration per TMJ was 84%, and per subject was 85%. In both cases, the reliability was fair to good (k = 0.62 and k = 0.47, respectively).[9]

At T1, radiographic signs of TMJ degeneration were observed in 82% of the subjects. Unilateral and bilateral degeneration were seen, respectively, in 28% and 54% of all subjects. MMO smaller than the proposed cut-off values for limited mouth opening[9,10] was seen in 17% of the subjects, and all of these subjects showed condylar degeneration.

Both the single and multiple logistic regression analyses for condylar degeneration indicated that independent predictors were age at JIA onset (inverse association, odds ratio [OR] = 0.83, *P* = .01) and disease duration (weak positive association, OR = 1.15, *P* = .05) (Table 2). The explained variance of the multiple logistic regression model was 21.1% (Table 2).

In the linear regression analysis, mouth opening capacity, age at JIA onset, disease duration, physical limitations at T0, treatment with NSAID and DMARD, and treatment with all medication types were significantly associated at the single level (Table 3). In the

Table 2 Single and Multiple Logistic Regression Analyses of Predictive Factors for the Presence of Temporomandibular Joint (TMJ) Condylar Degeneration in the Long Term in Patients Previously Diagnosed with Juvenile Idiopathic Arthritis (JIA)

	Count	Single analysis			Multiple analysis		
Variable	n (valid %)	OR	95% CI	*P* value	OR	95% CI	*P* value
Gender	94 (100)						
Male	22 (19.15)	Reference	–	–	–	–	–
Female	72 (80.85)	1.47	0.46 to 4.76	.52			
Age at JIA onset[a] (y)	94 (100)	0.83	0.72 to 0.95	**.01**	0.83	0.72–0.96	**.01**
Duration of JIA[a] (y)	94 (100)	1.14	1.00 to 1.29	**.05**	1.15	1.00–1.32	**.05**
JIA subtype at onset	94 (100)						
Oligoarticular	55 (58.5)	Reference	–	–	–	–	–
Polyarticular	27 (28.7)	0.59	0.13 to 2.6	.48			
Systemic	12 (12.8)	0.87	0.26 to 2.87	.81			
General functional limitation (Steinbrocker et al classification[6])	94 (100)						
No	64 (68.09)	Reference	–	–	–	–	–
Yes	30 (31.91)	0.60	0.18 to 2.04	.42			
Human leukocyte antigen-B27	59 (100)						
Positive	10 (16.9)	Reference	–	–	–	–	–
Negative	49 (83.1)	1.03	0.19 to 5.60	.98			
Antinuclear factor	94 (100)						
Positive	19 (20.2)	Reference	–	–	–	–	–
Negative	59 (62.8)	0.51	0.1 to 2.56	.42			
Uncertain	16 (17)	0.35	0.06 to 2.25	.27			
Rheumatoid factor	46 (100)						
Positive	6 (13)	–	–	–	–	–	–
Negative	34 (73.9)						
Uncertain	6 (13)						
Type of medication	94 (100)						
NSAID only	41 (43)	Reference	–	–	–	–	–
NSAID and DMARD	32 (34.4)	1.26	0.40 to 4.00	.70			
NSAID and ISD	8 (8.6)	2.03	0.22 to 18.76	.53			
NSAID and DMARD and ISD	13 (14)	3.48	0.40 to 30.54	.26			

Explained variance of the multiple logistic regression model: 21.1%.

[a]Continuous variable. Bold *P* values are statistically significant. OR = odds ratio; CI = confidence interval; NSAID = nonsteroidal anti-inflammatory drug; DMARD = disease-modifying anti-rheumatic drug; ISD = immunosuppressive drug.

multiple model, the degree of mouth opening was associated with age at JIA onset (estimated regression coefficient [B] and standard error [SE] = 0.46 [0.22], *P* = .03), disease duration (inverse association, B [SE] = −0.51 [0.16], *P* = .002), and physical limitations at T0 (inverse association, B [SE] = −4.31 [2.1], *P* = .04). The explained variance of the multiple linear regression model was 21% (Table 3).

Discussion

The results of the present study indicate that young age at JIA onset and longer disease duration are predictive for long-term TMJ degeneration. Young JIA patients may have a more aggressive disease leading to more tissue destruction and/or may have less chance of recovery compared to JIA patients with older age at disease onset; additionally, the longer the disease duration, the more progressive the degeneration.

Young age at JIA onset and prolonged disease are predictive of unfavorable long-term outcomes in other joints as well.[12] Similarly, Bakke et al[5] found that disease duration is predictive of long-term TMJ degeneration. Arvidsson et al[4] found no such correlation, but at follow-up correlations were determined with the presence of physical limitations at baseline, the number of joints with limited motion, and the patients' subjective well-being. Both studies concluded that a more severe disease led to more frequent long-term TMJ involvement.

In the present study, early physical limitations, together with younger age at JIA onset and prolonged disease, were predictive of smaller long-term MMO. In all subjects with MMO below proposed cut-off values for limited mouth opening,[9,10] TMJ degeneration was present. Altogether, this indicates that young patients with a more severe disease are at risk of TMJ degeneration and consequently decreased mobility. TMJ degeneration in JIA leads not only to joint stiff-

Table 3 Single and Multiple Linear Regression Analyses of Predictive Factors for the Degree of Active Maximal Mouth Opening (MMO; in mm) in the Long Term in Patients Previously Diagnosed with Juvenile Idiopathic Arthritis (JIA)

	Count	Single analysis			Multiple analysis		
Variable	n (valid %)	B (SE)	95% CI	*P* value	B (SE)	95% CI	*P* value
Gender	94 (100)						
Male	22 (19.15)	Reference			–	–	–
Female	72 (80.85)	–4.04 (2.41)	–8.84 to 0.75	.10			
Age at JIA onset[a] (continuous) (y)	94 (100)	0.49 (0.23)	0.04 to 0.93	**.03**	0.46 (0.22)	0.04 to 0.89	**.03**
Duration of JIA[a] (continuous) (y)	94 (100)	–0.63 (0.16)	–0.95 to –0.32	**.00**	–0.51 (0.16)	–0.83 to –1.19	**.00**
JIA subtype at onset	94 (100)						
Oligoarticular	55 (58.5)	Reference			–	–	–
Polyarticular	27 (28.7)	–3.06 (3.17)	–7.70 to 1.58	.19			
Systemic	12 (12.8)	2.70 (2.34)	–3.59 to 8.99	.40			
General functional limitation (Steinbrocker et al classification[6])	94 (100)						
No	64 (68.09)	Reference					
Yes	30 (31.91)	–4.75 (2.17)	–9.06 to –0.44	**.03**	–4.31 (2.1)	–8.46 to –0.15	**.04**
Human leukocyte antigen-B27	59 (100)						
Positive	10 (16.9)	Reference			–	–	–
Negative	49 (83.1)	0.48 (3.48)	–6.48 to 7.43	.89			
Antinuclear factor	94 (100)						
Positive	19 (20.2)	Reference			–	–	–
Negative	59 (62.8)	0.44 (2.67)	–4.85 to 5.74	.87			
Uncertain	16 (17)	0.54 (3.43)	–6.27 to 7.36	.86			
Rheumatoid factor	46 (100)						
Positive	6 (13)	Reference			–	–	–
Negative	34 (73.9)	–0.59 (3.65)	–7.90 to 6.80	.88			
Uncertain	6 (13)	–3.67 (4.75)	–13.25 to 5.92	.45			
Type of medication	94 (100)						
NSAID only	41 (43)	Reference			–	–	–
NSAID and DMARD	32 (34.4)	–4.68 (2.24)	–9.14 to –0.23	**.04**			
NSAID and ISD	8 (8.6)	–3.76 (3.66)	–11.05 to 3.50	.31			
NSAID and DMARD and ISD	13 (14)	–10.70 (2.94)	–16.54 to 4.87	**.00**			

Explained variance of the multiple linear regression model: 21%.
Bold *P* values are statistically significant. B = estimated regression coefficient; SE = standard error; CI = confidence interval;
NSAID = nonsteroidal anti-inflammatory drug; DMARD = disease-modifying anti-rheumatic drug; ISD = immunosuppressive drug.

ness but also to a decreased mandibular size, as mandibular growth is impaired in patients of a young age at JIA onset, and mandibular ramus height decreases with ongoing disease.[13] Mandibular size, especially condyle-incisor distance, greatly influences the MMO;[14] hence, a smaller jaw could lead to reduced MMO. Furthermore, jaw pain, which is more frequent in former JIA patients than in healthy subjects,[5] could also account for decreased mobility.

In the current study sample, 82% of the subjects showed long-term TMJ degeneration, similar to most previous reports.[4,5] Twilt et al[3] found a lower prevalence (40%) and reported a high recovery rate in time. This could be related to their use of newer, possibly more effective medication (eg, viz, biologic drugs). Biologic drugs were not used for the patients in the present study, nor were the patients treated with TMJ intra-articular corticosteroid injections. The use of such interventions would likely have resulted in a smaller proportion of subjects with long-term TMJ degeneration.

Of the patients in the present study, 17% had a limitation in MMO, while previous reports showed higher figures (eg, 28% and 38%).[5,15] This may be due to the longer follow-up and disease duration in those studies. Another explanation for this discrepancy may be found in the approach of establishing a limitation in MMO, which was different for children/adolescents and for adults. Based on the study by Müller et al,[9] the 10th percentile for MMO in 18-year-old boys is 44 mm and in 18-year-old girls is 42 mm, while the cut-off for adults was set at 40 mm according to the Diagnostic Criteria for Temporomandibular Disorders.[10] Hence, it is likely that the rate of adult patients with limited mouth opening in this setting will be lower than the respective rate in non-adult patients.

OPG is currently the standard screening tool for TMJ degeneration because of its availability, low cost, and minimal radiation. Other less available techniques (eg, cone beam computed tomography [CBCT] and

magnetic resonance imaging) are sometimes used, especially for early arthritis diagnostics.[2] OPG has several limitations such as a low sensitivity of detecting TMJ degenerative changes such as flattening and erosion, but its specificity corresponds to target values (ie, ≥ 90%).[16] Since the present study was merely focused on evident late consequences of JIA, OPG use was considered sufficient. In order to minimize the chance of non-JIA, age-related degeneration detected with OPG, subjects older than 30 years were excluded from this study. Condylar degeneration was assessed on OPG with the Rohlin and Petersson score,[3,8] which is widely used and comparable to the few other existing scoring systems.[1]

In this study, the presence of possible MMO limitation was estimated on the basis of a set of age-specific cut-off values instead of a single cut-off, since in the sample not only adults but also children and adolescents were included. Application of the largely used single cut-off value of 40 mm in children would result in a large number of false-positive subjects with limited MMO,[10] since MMO of 40 mm or less is not an unusual finding in children, especially below the age of 12 years.[9] The MMO in children increases with growth and seems to stabilize in adolescence,[9] indicating that in studies with children, the use of age-specific cut-off values is necessary.

This study had several limitations, mainly related to the retrospective character of the study. The analysis of association between RF and TMJ degeneration could not be processed because of missing data causing low RF-positive counts (ie, zero RF-positive subjects without TMJ degeneration; Table 1); however, RF count distribution over the groups of participants with and without TMJ degeneration suggested no association. Furthermore, no significant associations were found between TMJ degeneration or MMO and other types of blood markers or JIA subtype, one reason for which might have been the small number of participants per subgroup (eg, small number of systemic JIA and HLA-B27–positive patients) (Table 1). The current patient sample included patients diagnosed in 1993 to 1994, when the medical treatment of JIA patients was very limited in comparison with the line of drugs administered nowadays. Treatment of JIA has changed from a gradual add-on approach into an early and more aggressive approach with biologic drugs, thereby improving treatment outcomes. The conclusions of this study are therefore limited from a contemporary point of view.

In the present study, long-term jaw function was assessed using MMO measurements. In the future, other more objective functional parameters such as chewing efficiency could be studied. Moreover, subjective function should be addressed using the mandibular function impairment questionnaire[17] adjusted for use in children.

In the present study, none of the considered blood markers (HLA-B27, ANF, RF) were predictive of long-term TMJ degeneration or dysfunction. Also, the explained variance of both multiple models was low (21.1%). Altogether, this indicates that additional predictive markers may exist, such as local (ie, in TMJ synovial fluid) or systemic cytokine profiles, bone tissue factors, or other genetic and/or hormonal factors. Moreover, muscle involvement (eg, myositis) and patient-specific factors (eg, oral habits) should be considered.

Conclusions

Young JIA patients with early physical limitations and prolonged disease are at risk of long-term TMJ degeneration and dysfunction.

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