

Oral Health of Children with Juvenile Idiopathic Arthritis

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ABSTRACT. Objectives. To estimate dental disease indices and temporomandibular joint (TMJ) dysfunction in children with juvenile idiopathic arthritis (JIA).

Methods. Indices were recorded for dental caries, bacterial dental plaque, gingival inflammation, and TMJ dysfunction in children with JIA and matched controls.

Results. There was no significant difference in dental caries experience or the mean plaque score between children with JIA and controls. The mean gingivitis score for the permanent teeth only was significantly greater in the JIA children compared with the controls ($p = 0.02$). There was a significantly greater proportion of children with JIA with signs of both left and right TMJ dysfunction ($p = 0.05$, $p = 0.02$) and symptoms ($p = 0.0001$, $p = 0.0001$) compared with controls.

Conclusion. The low caries rate was attributed to the fact that children with JIA had received preventive dental care from an early age combined with sugar free medication. (J Rheumatol 2004; 31:1639-43)

Key Indexing Terms:

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Juvenile idiopathic arthritis (JIA), previously known as juvenile chronic arthritis (JCA) and juvenile rheumatoid arthritis (JRA), is a heterogeneous group of diseases of uncertain etiology. The incidence of JIA is 5 to 19 per 100,000 and the prevalence is 1 in 1,000 children¹. JIA begins in childhood and involves persistent inflammation of one or more joints¹. Boys are affected less frequently than girls² except for the systemic form. Juvenile arthritis was historically classified into 3 subgroups, pauciarticular, polyarticular, and systemic-onset disease on the basis of number of joints affected at disease onset and a variety of extra-articular clinical features³. Additional classifications were proposed for spondyloarthritis⁴ and psoriatic arthritis^{5,6}. More recently, the Classification Task Force in Childhood Rheumatic Disease of the International League

of Associations for Rheumatology (ILAR) has proposed a new, all-embracing classification that includes 7 JIA subtypes together with an unclassifiable group^{7,8}. This classification is now being used almost universally.

Approximately one-third of affected children have either persistent disease as adults or sequelae from inflammation. These can include joint damage, visual impairment as a result of uveitis, and osteoporosis from systemic steroid therapy. There have been major changes in the management of JIA during the previous 10 years, in particular more effective use of disease modifying antirheumatic drugs (DMARD)⁹, which should, by virtue of their steroid-sparing activity, minimize growth failure and the later effects of osteoporosis. Intraarticular corticosteroids are commonly used in children with oligoarticular JIA and may also benefit children with other types of disease¹⁰, although their use has rarely been reported in the treatment of TMJ problems. The introduction of biologic treatments like anti-tumor necrosis factor (TNF)^{11,12} and autologous stem cell transplantation^{13,14} have had promising early results in the management of JIA. These treatments have further increased the overall degree of disease control and have even induced remission.

The TMJ is often involved in JIA¹⁵⁻¹⁷, leading to disturbance of growth, facial deformity, and difficulties with mouth opening. TMJ dysfunction can be unilateral, bilateral, painful or completely asymptomatic, and may not be clinically apparent for many years^{18,19}. Although there are few data for dental disease in either adults or children with JIA, increased caries experience has been reported²⁰⁻²⁵. However, the methods of dental disease assessment were different in these investigations, and matched controls were not always

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included. In addition, altered sialochemistry including lower mean concentrations of calcium, phosphorus, potassium, lysozyme, and IgA were detected in the saliva of JIA children with a greater level of decayed, missing, and filled teeth (DMFT) than controls²¹. Later studies found both a reduced resting salivary flow together with a reduced response to stimulation in patients with JIA compared to controls. The levels of both calcium and phosphate ions were also lower in the JIA group, although not significantly²⁶. This may contribute to earlier reported higher caries prevalence in children with JIA.

The gingival health of children with JIA has been reported to be significantly worse than that of controls²⁷, with more gingival inflammation, dental plaque, and poorer oral cleanliness²⁵. Involvement of the upper limb in JIA can result in functional disability so that there may be considerable difficulty with oral hygiene procedures thus allowing plaque accumulation^{23,24}.

In a retrospective investigation of enamel defects in children with JIA, there was no significant difference in the overall prevalence of or specific type of enamel defects compared to controls²⁸.

We assessed oral health and TMJ dysfunction in children with JIA.

MATERIALS AND METHODS

Ethics approval was obtained from Great Ormond Street Hospital for Children NHS Trust, and the Eastman Dental Institute & Hospital Joint Research Ethics Committee. Written consent was obtained from the parent(s) and verbal consent from the children.

Subjects. Consecutive children aged between 4 and 16 years attending the Rheumatology Clinic and children on the Rheumatology Ward at Great Ormond Street Hospital for Children were included in the study.

Controls. Children attending the Trauma Clinic in the Department of Paediatric Dentistry were matched for age, gender, and ethnicity with the subjects.

Reproducibility. Studies were completed to assess the reproducibility of recording indices for caries and the kappa value calculated²⁹. Ten full arch tooth blocks (blocks of plaster set with extracted teeth that have a variety of different cavities) were examined by the main examiner and another dental surgeon to assess interexaminer agreement using World Health Organization (WHO) criteria³⁰. The same tooth blocks were examined again after one week by the main examiner to assess intraexaminer agreement. Indices for bacterial dental plaque and gingivitis were recorded for 10 children by the main examiner and another dentist.

Dental caries. All the primary teeth have erupted by the age of 2.5 years. The first permanent molar teeth start to erupt at 6 years and for the period between 6 and 12 years, both primary and permanent teeth are present in the mouth. By the age of 12 or 13 years, all the primary teeth have been lost. Teeth were visually examined for caries with a mirror using the WHO criteria³¹. The indices were recorded as the decayed, missing, and filled surfaces of the primary teeth/decayed, missing, and filled primary teeth (dmsf/dmft) and the permanent surfaces and teeth (DMFS/DMFT).

Bacterial dental plaque. Four gingivally related quadrisections of each tooth (mesiobuccal, distobuccal, mesiolingual, distolingual) were visually examined for bacterial dental plaque deposits to give the plaque score, using a modification of the O'Leary index³². This has been shown to be reproducible in small children.

Gingival inflammation. The gingivae were visually examined for inflammation using a simplified gingival index based on the number of tooth quadrisections associated with gingival inflammation to give the gingivitis score³². Spontaneous gingival bleeding was also recorded.

TMJ dysfunction. Signs and symptoms of TMJ disorders were recorded using WHO criteria³³. These included limited opening of the mouth, clicking of the joint(s), and pain.

Enamel defects. Enamel defects were recorded using the Federation Dentaire Internationale (FDI) notation³⁴.

Fluoride intake. Each parent was asked if their child was taking or had been taking fluoride supplements.

Statistical analyses. Data were tested for normality using the Shapiro-Wilk test²⁹ and found to be non-normal in distribution. Nonparametric statistical tests were used. The chi-square test was used for categorical data and the Mann-Whitney test for comparisons between the JIA and control groups.

RESULTS

Fifty-five subjects were included in the study, 21 boys and 34 girls. Of these, 47 were outpatients and 8 were inpatients. There was no significant difference in the mean age of the children with JIA, 8.9 years (SD 3.2) compared with 9.2 years (SD 3.2) for controls. The diagnoses and medication for JIA are shown in Table 1.

Reproducibility. Studies were completed to assess the reproducibility of recording indices of caries, bacterial dental plaque, and gingival inflammation, and the kappa value calculated. For caries the kappa value for inter-examiner agreement was 0.65 and 0.89 for the intra-examiner agreement. For bacterial dental plaque the kappa value was 0.86 and for gingivitis 0.88. For dental caries, there was substantial intra-examiner agreement and good inter-examiner agreement. There was substantial inter-examiner agreement for bacterial dental plaque and gingivitis²⁹.

Dental caries. The proportion of children with JIA with no caries was 27.3%, not significantly different from the matched controls (20%). There were no significant differences in the dmfs/dmft or DMFS/DMFT between the JIA and control children (Table 2). A significantly greater proportion of children with JIA (32.1%) had untreated caries ($p = 0.05$) compared with controls (16%).

Bacterial dental plaque. There was no significant difference in the mean plaque score for either the primary or permanent teeth between the JIA and control groups (Table 3).

Gingival inflammation. There was both a significantly greater mean gingivitis score in the JIA group [25.5 (SD 24.8) and index 39.9 (SD 30.3)] compared to controls [17.1 (SD 29.9) and index 23.5 (SD 27.1)] ($p = 0.002$ and $p = 0.003$) (Table 3). There was no spontaneous gingival bleeding in either the JIA or control groups.

Developmental enamel defects. Developmental enamel defects were observed in the permanent dentition of 18 (33%) children with JIA and 13 (24%) controls. These were mainly white/cream opacities.

TMJ dysfunction. There was a significantly greater proportion of children with JIA with signs of both left (16.4%, chi-

Table 1. Diagnoses and medication for the children with JIA.

Medication	Systemic Onset JIA (n = 10)	Oligoarthritis (n = 12)	Extended Oligoarthritis (n = 14)	Polyarticular Arthritis (n = 14)	Enthesitis Arthritis (n = 5)
Methotrexate	10	3	12	10	3
Steroids					
Oral	5	3	7	4	2
IV	–	–	1	–	–
Intraarticular	5	–	5	1	1
NSAID	10	9	14	13	4
Cyclosporine	2	–	2	2	–
Anti-TNF	1	–	1	–	–
DMARD	2	–	2	2	3

NSAID: nonsteroidal antiinflammatory drugs; Anti-TNF: anti-tumor necrosis factor; DMARD: disease modifying antirheumatic drug.

Table 2. Decayed, missing and filled surfaces and teeth and developmental enamel defects: JIA and control groups. There were no significant differences between the 2 groups.

	JIA (n = 55)					Controls (n = 55)				
	Mean	SD	Median	Min	Max	Mean	SD	Median	Min	Max
dmfs	2.9	4.4	1	0	16	4.3	4.4	4	0	16
dmft	2.0	2.6	1	0	10	3.2	3.2	3	0	11
DMFS	2.0	2.7	0.5	0	11	2.5	2.8	2	0	11
DMFT	1.8	2.4	1	0	11	2.1	2	2	0	7

Table 3. Bacterial dental plaque and gingival inflammation score and index: JIA and control groups.

	JIA (n = 55)					Controls (n = 55)					
	Mean	SD	Median	Min	Max	Mean	SD	Median	Min	Max	p
Primary teeth											
Plaque score	22.2	16.4	20	0	56	26.8	23.1	24	0	80	NS
Plaque index	42.8	30.6	37.5	0	100	44.6	33.7	40	0	100	NS
Gingivitis score	10.8	15	5	0	56	9.6	16.1	2	0	72	NS
Gingivitis index	21.4	30.6	10	0	100	15.2	22.9	3.6	0	100	NS
Permanent teeth											
Plaque score	39.5	29.1	36.5	0	112	33.6	29.7	24	0	112	NS
Plaque index	58.1	29.9	57.5	0	100	48.7	29.8	48.2	0	100	NS
Gingivitis score	25.5	24.8	16	0	112	17.1	29.9	10	0	112	0.02
Gingivitis index	39.9	30.3	31.7	0	100	23.5	27.1	16.7	0	100	0.003

NS: not significant.

square 4.4949, df 1, $p = 0.05$) and right (20%, chi-square 7.066, df 1, $p = 0.02$) TMJ dysfunction, compared to controls (Table 4). There was also a significantly greater proportion of children with JIA with reduced mobility of both the left (35%, chi-square 16.494, df 1, $p = 0.0001$) and right TMJ (35%, chi-square 17.786, df 1, $p = 0.0001$) compared to controls (Table 4). Children in all subgroups had signs and/or symptoms of TMJ dysfunction: systemic onset JIA $n = 6$, oligoarthritis $n = 3$, extended oligoarthritis $n = 7$, polyarticular arthritis $n = 7$, and enthesitis related arthritis $n = 4$.

Fluoride intake. Fourteen children (25%) with JIA had been taking fluoride supplements. Of these, 1 child used a mouth

rinse, 8 other children drops, and a further 5 used tablets. Nineteen (35%) controls had been taking fluoride supplements. There was no significant difference in the proportions of the 2 groups who had used fluoride supplements.

DISCUSSION

There were no significant differences in the dmfs/dmft or DMFS/DMFT between the children with JIA and controls. Although an increased incidence and prevalence of dental caries has been reported in other groups of children with JIA^{22,23,25}, authors concluded that further controlled studies were needed. The proportion of decayed tooth surfaces was significantly greater in children with JIA compared to

Table 4. Signs and symptoms of TMJ dysfunction: JIA and control groups.

	JIA (n = 55)		Controls (n = 55)		p
	Yes	No	Yes	No	
Left TMJ	9	46	2	53	0.05
Right TMJ	11	44	2	53	0.02
Symptoms					
	Left TMJ	Right TMJ	Left TMJ	Right TMJ	p
No symptoms	33	32	51	51	NS
Clicking	3	4	1	1	NS
Tenderness on palpation	0	0	0	0	NS
Reduced mobility	19	19	3	3	0.0001

NS: not significant.

controls; although our results indicate that children with JIA are less likely to have had restorative treatment, approximately 20% of both the JIA group and controls had received restorative treatment in the previous 6 months. A greater proportion of children with JIA had had a dental checkup within the preceding 6 months compared to controls and a similar proportion of parents of both the children with JIA and controls claimed to have given their children fluoride supplements.

The DMFT was similar to that of another group of chronically sick children undergoing heart and heart/lung transplantation, (1.9)³⁵ and a different group with chronic renal failure (2.7)³⁶, but greater than that recorded for children with HIV (0.8)³⁷ and glycogen storage disease (0.6)³⁸. All these groups of children are cared for at the same tertiary referral center where preventive dental health is part of the program of care. Most of the parents of the children with JIA were aware of the relationship between the frequency of sugar ingestion and dental caries and thus limited the children's sugar intake. There are a number of factors that may explain a greater DMFT compared with some groups of chronically sick children. Children with JIA are often treated with medications containing sugar, and longterm use may increase the prevalence of caries^{39,40}. There is evidence that children with JIA have small frequent meals thus increasing their exposure to sugar⁴¹.

Although there was no difference in the plaque score, both the mean gingivitis score and index for the permanent teeth was greater in the children with JIA. This may be attributed to several factors. Although 30% of the JIA group had TMJ dysfunction consistent with other groups^{24,42}, there was no difference in the plaque score between the children with JIA and controls. Signs and symptoms of TMJ dysfunction were recorded in all disease subgroups, and it is likely that this may be missed by pediatricians, particularly if mild, or if not entirely expected in a particular subgroup, for example oligoarticular JIA. In addition, approximately 50% of these children also had upper limb disability and limited

manual dexterity. Although there was no significant difference in DMFT and DMFS between the children with upper limb and hand disability and those with relatively normal movement, there was a trend towards fewer caries in the children with limb disability. It could be conjectured that parents are aware of the difficulties with tooth brushing and are more careful with diet as a consequence and do the tooth brushing themselves.

The gingivitis score was significantly greater in the children with JIA. The possible reasons for this are an altered local immune response due to certain drugs, for example methotrexate, and perhaps the disease process itself. An increased gingivitis score together with an increased plaque score was detected in other groups of chronically sick children with limited manual dexterity: for example, children with craniosynostosis⁴³ and with dystrophic epidermolysis bullosa⁴⁴.

In conclusion, the proportion of children with JIA with dental caries was similar to matched controls although the proportion of unrestored permanent tooth surfaces was greater. This may reflect difficulties associated with limited mouth opening or a reluctance to accept restorative treatment. The increased gingivitis score in the permanent dentition may be associated with lowered local salivary and mucosal immunity. It would be useful and informative to assess the salivary immunoglobulins in children with JIA and introduce mouth care regimens aimed at controlling the gingival inflammation, for example twice daily tooth-brushing together with twice daily chlorhexidine mouth rinses. It would also be appropriate to encourage more restorative treatment.

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