Oral health and juvenile idiopathic arthritis: a review

A. G. Walton, R. R. Welbury, J. M. Thomason\(^1\) and H. E. Foster\(^2\)

Department of Child Dental Health, The Dental Hospital and School, Richardson Road, Newcastle upon Tyne, \(^1\)Department of Restorative Dentistry, The Dental Hospital and School, Richardson Road, Newcastle upon Tyne and \(^2\)Department of Child Health, Royal Victoria Infirmary and Rheumatology, Musculoskeletal Unit, Freeman Hospital, Newcastle upon Tyne, UK

Abstract

Juvenile idiopathic arthritis (JIA) results in significant morbidity that includes an adverse impact on oral health that is generally not well recognized. This review describes current literature which demonstrates poor oral health in children with JIA. The impact of JIA on oral health is probably multifactorial and these factors are discussed. This review emphasizes the role of paediatric dentistry in the multidisciplinary management of JIA and highlights the need for further research.

**Key words:** Juvenile idiopathic arthritis, Oral health, Dental caries, Periodontal disease, Saliva, Temporomandibular joint, Medication, Sugar-free medicines, Therapy.

Juvenile idiopathic arthritis (JIA) is the commonest chronic arthritis of childhood with a prevalence of 1 in 1000 in the UK [1, 2]. The morbidity from JIA is extensive and includes uveitis, joint damage, growth abnormalities, osteoporosis and adverse psychosocial impact. However, the effect of JIA on oral health is less well documented. There are several areas where JIA may impact on oral health, namely dental caries, periodontal disease, saliva abnormalities, involvement of the temporomandibular joint (TMJ) and the effect of facial growth. In addition, the systemic effect of chronic disease may impact on oral health. The purpose of this article is to review the current literature on oral health in JIA, highlight areas for future research and emphasize the role of a paediatric dentist in the multidisciplinary management of JIA.

There are three main areas where JIA can impact on oral health which are described in detail below.

**Dental caries, periodontal disease and saliva**

**Dental caries**

Dental caries (decay) is a sugar-dependent bacterial disease of the calcified tissues of the teeth. It is caused by acid demineralization of the tooth substance and requires the presence of a bacterial plaque (the firmly adherent film of bacteria in a mucopolysaccharide matrix formed on the surface of the teeth a few hours after toothbrushing) and dietary fermentable carbohydrate (sugar). The acid production causes demineralization of the tooth surface with the liberation of calcium and phosphate ions. It is a multifactorial disease, the major aetiological factor being the frequent consumption of sugar. Caries is a dynamic process which in the early stages of demineralization can be reversed if the plaque pH rises again. However, if there is a net loss of tooth substance then irreversible cavitation may ensue. An often used reliable and epidemiologically validated index of decay experience is the DMFT (decayed, missing, filled teeth) index. This is a measure of the current decay experience (decayed element) and any previous decay experience (missing and filled elements) and excludes tooth removal for other reasons. It is a purely clinical index not relying upon radiographic evidence. It can be applied to both the adult (permanent) dentition and milk (deciduous) dentition, the latter being called the dmft index.

Table 1 summarizes the studies of dental caries and JIA to date. The number of studies and patients included is often small, there is a paucity of controls, and there are different measures of assessment of oral health. However, all studies reported increased amounts of caries in patients with JIA [3–7].

The involvement of the upper limb in JIA can cause considerable functional disability and the patient may
have increased difficulty with oral hygiene measures (toothbrushing and flossing) and hence plaque removal [6–9]. The TMJ is commonly involved in JIA and may result in a restriction in mouth opening which may also impede plaque removal. The studies to date (summarized in Table 1) did not include detailed clinical and functional documentation of JIA and the potential role of medication on oral disease was not addressed.

**Periodontal disease**

There is a direct correlation between the amount of plaque at the cervical (gum) margin of the teeth and the severity of gingivitis (gum inflammation), and a causal relationship is likely.

Periodontal disease broadly consists of two elements, gingivitis and periodontitis. Gingivitis is a reversible inflammatory response of gums (gingivae) to plaque and is present in most mouths and is characterized by redness, swelling and bleeding (on probing) of the gingivae. Although gingivitis is readily reversible with effective plaque control (toothbrushing, flossing, mouthwashes), it may be a precursor of chronic periodontitis, which is the irreversible progression of infection and inflammation of gingivitis into the gingivae, periodontal ligament (anchors tooth in bone) and bone causing destruction of the periodontium. The destruction occurs in bursts of activity between resting phases, the duration and frequency of the activity bursts being dependent on changes in the plaque microflora, the host response to systemic disease, local factors, and as yet undetermined variations in the inflammatory response to plaque.

In a small study of adults with rheumatoid arthritis, poor periodontal health was reported, but was not significantly different from that of controls despite limitation of upper limb movement [10].

**Saliva**

Dry mouth (a feature of Sjögren’s syndrome) commonly associates with poor oral health in adults but is rarely reported in children. Low levels of salivary calcium and phosphorus are associated with an increased risk of dental caries, since saliva acts as a reservoir of calcium and phosphate ions which favours remineralization of teeth previously demineralized through dental caries. In a small study of 16 JIA patients with a mean age of 9.3 yr, there was no statistical difference in parotid flow rate between subjects and healthy controls [4]. Nevertheless the mean salivary concentrations of calcium, phosphorus, potassium, lysozyme and IgA were significantly lower in the JIA patients and the mean DMFT in the 16 children with JIA was 6 which was almost twice that of the control group (mean 3.2). Gingival inflammation was greater in patients than in controls [4].

The increased prevalence of dental caries in JIA is probably multifactorial [7], although the relative effect of different factors has not been elucidated. Other factors relevant to the risk of dental caries include diet, fluoride intake and other barriers to health care (access to dental care, patient knowledge regarding diet and oral hygiene). JIA can have an impact on diet through poor intake (secondary to anorexia with active disease), difficulty chewing (TMJ disease) or sore mouth due to medication. There is some evidence that children with JIA often have small, frequent meals and hence the exposure to sugar is more frequent (Moynihan, unpublished data). Furthermore, children are often consoled with sweets from well-meaning parents and grandparents [7].

**The effects of medication used to treat JIA**

Medications used in the management of JIA aim to control symptoms of pain, stiffness and swelling, minimize functional disability and prevent joint damage. Most children with JIA will receive analgesics and non-steroidal anti-inflammatory drugs (NSAIDs). Increasingly there is a trend to use intra-articular steroid injections and disease-modifying anti-rheumatic drugs (DMARDs) earlier in the disease course, to minimize functional disability and minimize the use of oral steroids. Consequently children often require several medicines concurrently.

**NSAIDs**

The most commonly prescribed NSAIDs are ibuprofen and naproxen (Walton et al., unpublished data). Salicylates are now rarely used in children but have been associated with mucosal ulceration and enamel erosion [6]. NSAIDs are generally well tolerated by children; mild gastrointestinal disturbances (abdominal pain, nausea) are the most common side-effects, albeit rare. However, young patients may be unable to swallow...
of the drugs used to treat JIA, but rarely are severe also that facial growth disturbance may be present
the dental practitioner with gingival enlargement, ulceration (mimicking aphthous stomatitis) [7].

The pathological changes include hypertrophic mucosal pigmentation [7, 22]. It is noteworthy that symptomatic TMJs may
oral ulceration (mimicking aphthous stomatitis) [7]. JIA. Of those children with symptoms, the most fre-
fi

fevers including those of dental origin [6, 7]. Cyclosporin although this is disputed [33]. Bony erosion seems to
ect may be greater if they are taken

comitant use of folic acid [17]. Methotrexate may inter-

acidity and high with early onset of JIA, long disease duration, bilateral

occur later in JIA than in adult rheumatoid arthritis, presumably reflecting the predominance of cartilage and therefore explaining the reported delay in radiographic changes [34]. It is generally agreed that the severest radiographic bony destruction is significantly associated with early onset of JIA, long disease duration, bilateral lesions, and pauci-articular onset with polyarticular disease course [35]. Signs of TMJ damage may be seen in children without clinical symptoms or a diagnosis of JIA. Of those children with symptoms, the most fre-

uct may be greater if they are taken

Gold, penicillamine and anti-malarial agents are now rarely used in view of their relative inefficacy and high toxicity. A metallic taste (agneusia) as well as stomatitis, glossitis, and gingivitis are well documented with gold treatment [21]. Penicillamine may induce chelosis and oral ulceration (mimicking aphthous stomatitis) [7]. Anti-malarials such as hydroxychloroquine may cause mucosal pigmentation [7, 22].

Blood dyscrasias are a possible complication of many of the drugs used to treat JIA, but rarely are severe enough to be clinically significant. They may present to the dental practitioner with gingival enlargement, ulceration, or bleeding (especially post-extraction haemorrhage) and obviously some dental treatment may be contraindicated due to the risk of haemorrhage or infection.

Corticosteroids
For patients who are taking long-term corticosteroids, delayed wound healing and increased risk of infection may impact on the role of the dental practitioner, who must be alert to the risk of adrenal cortical insufficiency when any surgery or stressful treatment is planned [23].

The TMJ and facial growth
The TMJ is commonly involved in JIA [24–27] and can lead to growth disturbance, facial deformity and restricted mouth opening. TMJ disease can be asymmetrical, asymptomatic and may not be evident clinically [24, 28, 29]. The reported prevalence of radiographic changes in TMJs varies between 17 [30] and 63% [31], which may represent differences in radiographic techniques as well as differences in the threshold of reporting and patient selection criteria.

The degree of condylar abnormality may vary from minor erosion and flattening of the articular surface to severe destruction of the condylar head, and studies describe the changes as possible, definite or extreme [31]. The reported prevalence of unilateral lesions of the mandibular condyle varies between 17 and 40% [32] with bilateral lesions accounting for the remainder. It is suggested that TMJ lesions start asymmetrically in the early stages and then become symmetrical [28, 31] although this is disputed [33]. Bony erosion seems to occur later in JIA than in adult rheumatoid arthritis, presumably reflecting the predominance of cartilage and therefore explaining the reported delay in radiographic changes [34]. It is generally agreed that the severest radiographic bony destruction is significantly associated with early onset of JIA, long disease duration, bilateral lesions, and pauci-articular onset with polyarticular disease course [35]. Signs of TMJ damage may be seen in children without clinical symptoms or a diagnosis of JIA. Of those children with symptoms, the most frequently reported are TMJ sounds (26% of patients) and pain [26]. It is noteworthy that symptomatic TMJs may not be associated with facial growth disturbance but also that facial growth disturbance may be present without TMJ symptoms [9].

The pathological changes include hypertrophic inflammatory synovitis characterized by cellular infiltration, with proliferation and congestion of the blood vessels. The lining cells elongate and the synovial villi become hypertrophied; these changes may resolve with remission of the inflammatory process. However, in chronic disease, infiltrating pannus gradually extends over the surface of the articular cartilage, leading to articular destruction and joint deformity. Occasionally adhesions between opposing layers of pannus lead to fibrous ankylosis of the joint. Tendons and muscles become oedematous and infiltrated with cells and any

<table>
<thead>
<tr>
<th>Medication</th>
<th>Sugar content g/5 ml</th>
<th>Typical daily sugar content (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Naproxen (Naprosyn)</td>
<td>1.275</td>
<td>5.1</td>
</tr>
<tr>
<td>Ibuprofen (Brufen)</td>
<td>3.3</td>
<td>19.8</td>
</tr>
<tr>
<td>Paracetamol (Calpol 6 + )</td>
<td>1.8</td>
<td>7.2</td>
</tr>
<tr>
<td>Codeine phosphate</td>
<td>2.73</td>
<td>10.9</td>
</tr>
<tr>
<td>BP 25 mg/5 ml</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Oral health and JIA

effect on the chondrogenic zone of the TMJ may lead to retarded growth [9]. The effects of JIA on facial growth are well documented [25, 35–37] and may be predominantly due to condylar abnormalities [29]. Larheim and Haanes [25] and Stabrun [27] concluded that the interference with mandibular growth is brought about by a combination of the direct effect on the TMJs and the restricted function. Pedersen et al. [38] stated that joint damage led to a change in joint function and hence mandibular position, which in turn led to a change

![Image of teeth](image1)

**Fig. 1.** Anterior open bite (space between upper and lower incisors), and lower incisor crowding affecting child with JIA. Reprinted from [40] with the permission of Macmillan Press.

![Image of child](image2)

**Fig. 2.** 'Bird face deformity' affecting child with JIA. Reprinted with the permission of Gower Medical Publishing.
of muscular function, causing developmental changes in mandibular morphology and an unstable occlusion. This then resulted in a decreased total mandibular function causing further joint damage, and a vicious circle was established. Muscle weakness developing in association with microscopic tissue changes has been reported [37] and maximal molar bite forces and endurance times were significantly reduced in children with JIA [39].

The facial appearance of growth disturbance in JIA patients is well described and includes characteristic features of small mandible, Angle class II malocclusion (mandibular retrognathia) (unilateral or bilateral), anterior open bite (Fig. 1), lower incisor crowding, and incisal protrusion [9]. The facial disturbance is largely due to a backward rotational growth pattern of the mandible. The characteristic description of ‘Bird face’ deformity [41] (Fig. 2) is often used to describe this facial form, but although commonly seen in JIA (10–30% of patients) it is not pathognomonic.

The role of corticosteroids on the growth of the mandible is controversial. Long-term steroids may cause erosion, decreased bone density, and condylar collapse similar to that seen in JIA itself [34], but short-term steroids of less than 3 months do not appear to be significant in terms of ramal height and mandibular length [35]. JIA may be associated with varying degrees of mandibular hypofunction due to restricted movement of the jaws brought about by pain, tenderness, and a malocclusion [25]. Imaging of the TMJ is important for diagnosis and the planning of treatment. Radiographic examination includes the dental panoramic tomogram (DPT), although in recent years it has been shown that magnetic resonance imaging (MRI), ultrasound and computed tomography (CT) may be more sensitive to detecting early joint changes [42–44]. Ultrasound and MRI incur no radiation risk and are hence advantageous over CT. Ultrasound is increasingly used in rheumatology and is usually more accessible.

Discussion

This review demonstrates that there is a detrimental effect of JIA on oral health. This is multifactorial and includes mechanical factors, functional disability, biochemical abnormalities, diet and the medications used to treat JIA. There is no research into the relative importance and interplay between these potential factors and, furthermore, ‘barriers to dental care’ (e.g. diet, knowledge, access to dental care) have not been explored. Not all children with JIA have oral health problems and research is needed to identify children who are at risk, propose interventional strategies that may be effective, and consider their cost-effectiveness. Poor oral health in children with JIA adds significantly to their global morbidity and any interventions to improve oral health are likely to be well received by patients and their families.

Despite the paucity of research so far, a clear role for an experienced paediatric dentist in close liaison with the paediatric rheumatology team does none the less exist. Regular dental examination and early intervention, together with orthodontic monitoring of growth, is advised.

Prevention is the cornerstone of success of this dental management with emphasis on patient education, dietary advice, plaque control, fissure sealants, and the use of topical fluorides. Sugar-free medicines should be used wherever possible. The use of regular daily exercises has been proposed to improve the range of movement of the TMJs and hence facilitate oral hygiene maintenance [24], and the use of electric toothbrushes has also been advocated [45]. The conventional treatment strategy for TMJ disease has been to postpone orthodontic therapy and any orthognathic surgical treatment until growth had ceased. Current opinion however favours early treatment often with orthodontic functional appliances during the pubertal growth spurt to try and maintain occlusal stability, increase function of the joint, with the aim of allowing continuous uninhibited growth of the mandible [33, 38].

Minimal operative intervention is the aim, but where dental treatment is required there may be difficulty with access to the oral cavity due to limited jaw opening, and both steroid and antibiotic cover may be needed for those at risk of adrenal crisis and infective endocarditis. General anaesthesia may be problematic due to anaemia (mostly anaemia of chronic disease) and intubation and airway maintenance may be difficult because of micrognathia or cervical spine disease.

Poor oral health is a risk factor for systemic infection, and especially when the patient is immunosuppressed through the use of corticosteroids or DMARDs. Good oral health is therefore important to minimize complications of JIA and its treatment as well as reducing the morbidity of caries and periodontal disease.

Acknowledgement

This work was supported by an Arthritis Research Campaign (ARC) project grant.

References


