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CLINICAL reports

A soft tissue lesion related to salicylate treatment of juvenile rheumatoid arthritis: clinical report

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Abstract

A clinical report of a soft tissue lesion related to salicylate treatment of juvenile rheumatoid arthritis (JRA) is presented. JRA affects 250,000 children in the United States. The literature on JRA is reviewed with emphasis on treatment and the adverse effects of treatment manifested in the oral cavity.

Juvenile rheumatoid arthritis (JRA) is a disease first described by the English pediatrician George Still in 1897.[1] Today, an estimated 250,000 U.S. children are affected with the disease.[2] Females are reported to be affected two times more often than males.[3] The disease usually begins before age 16 and has a peak onset in the one- to three-year age group and a lesser peak in the 8- to 12-year group.[3] Five per cent of all cases of rheumatoid arthritis are reported to begin in childhood.[2]

JRA is an extremely variable disease which encompasses three broad clinical groups: systemic, polyarticular, and pauciarticular. Each clinical group is characterized by a specific mode of onset. *Systemic* onset accounts for 20% of all JRA cases and is characterized by arthralgia, spiking fever, rheumatoid rash, and a generalized lymphadenopathy. *Polyarticular* onset makes up 30–40% of the cases and is defined by an arthritis of four or more joints. There are fewer systemic signs and a low-grade fever. The remaining 50% of the cases are of the *pauciarticular* form. By definition, fewer than four joints are involved.

The disease etiology is obscure although two hypotheses have been suggested. One hypothesis theorizes that the disease results from an infection with as yet unidentified microorganisms. The second hypothesis suggests that the disease is a hypersensitivity or autoimmune reaction to unknown stimuli.[2] There may be a genetic component to JRA, but the extent is uncertain.

There is no specific cure for JRA although the prognosis is good for most patients. With appropriate care when the disease is active more than 75% of children will remain free of significant disability.[4] Management goals consist of preserving joint function and treating any extra-articular manifestations. Acetylsalicylic acid (aspirin) is the drug of choice for treating all types of JRA;[5] it suppresses the inflammatory reaction in the most satisfactory and safest manner. Dose varies depending on the type of disease and the presence or absence of an acute febrile episode. Clinical toxicity to aspirin is of relatively low incidence (16%).[6] In cases where the arthritis does not respond to a trial of salicylates, a slower-acting drug such as intramuscular gold, hydroxychloroquine sulfate, or penicillamine has been employed.[3] Physical therapy always is indicated for the JRA patient.

There are few references to JRA in the dental literature. Temporomandibular joint problems are reported most commonly (Figure 1). Typically patients present with symmetrically underdeveloped mandibles and unaffected maxillas.[7] Serial records have shown that the deformity may become progressively worse as the patients mature.[8,9] Recently, a relationship was reported between JRA patients who chewed aspirin and tooth erosion.[10] That report postulated that tooth erosion was caused by the acidic nature of aspirin particles lodged in the occlusal surfaces of the teeth. This article presents a patient with tooth erosion and a soft tissue lesion related to salicylate treatment of JRA.

Clinical Report

L.H., a three-year, ten-month-old white female was the seven-pound, four-ounce product of an uncomplicated pregnancy and delivery. She obtained milestones at appropriate times and was developing normally until she was two years, seven months of age. At that time she was seen by her physician for

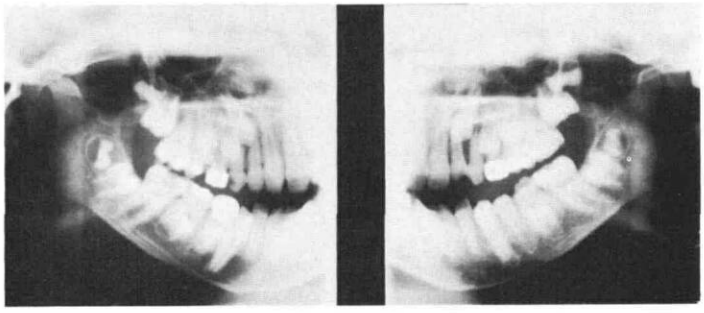

FIGURE 1. A panoramic radiograph of a 10-year-old child with JRA. The right condylar head shows significant erosion due to the disease.

an examination related to rash development following penicillin administration for a sore throat. She had a fever of 103°F and swelling of both ankles. The patient's sedimentation rate was elevated. Assays for autoimmune antigens were negative. JRA was suspected but not confirmed. An undocumented aspirin prescription resulted in severe tinnitus and visual hallucinations, but subjectively there was decreased swelling of the ankles. Discontinuation of the aspirin resulted in a decreased range of motion. A number of visits to different hospitals in the following weeks yielded similar results. The patient was treated with Tolectin®, Gemmisyn®, and Decadron® at separate times with little improvement.

At three years of age the patient was admitted to Duke University Hospital for further evaluation. A diagnosis of systemic polyarticular JRA was made during the admission based on the patient's history of erythematous rash, fever, arthritis, hepatomegaly, and adenopathy. Further studies were initiated because the patient exhibited a severe anemia and a questionable myositis. A diagnosis of anemia due to chronic disease was made because iron levels were found to be normal in bone marrow studies. The myositis was ruled out because no evidence of muscle wasting could be found. The patient was prescribed Tolectin® 100 mg QID and aspirin 80 mg/kg/day for the JRA.

The patient was admitted to the Lenox Baker Children's Hospital (LBCH) for intramuscular gold therapy and physical therapy at three years, ten months of age. During hospital admission the patient presented to the LBCH dental clinic for a routine oral examination. The patient had no known allergies and immunizations were up-to-date. Medication consisted of 20 chewable aspirin — five aspirin four times per day, Tolectin® 100 mg QID, and an antacid as needed. The patient had been started on gold therapy four days prior to the initial dental examintion. An intraoral examination revealed soft tissue structures of normal contour, texture, and color; however, a white surface lesion was noted in the mandibular buccal vestibule. The lesion could be detected extending from right to left around the anterior vestibular area and it was especially prominent in the posterior regions (Figure 2). Neither the patient nor the patient's mother was aware of the lesion and could not give information as to its origin or duration. The lesion surface could be rubbed off leaving a slightly inflamed, minimally uncomfortable, ulcerated area. A hard tissue examination revealed a primary dentition with flush terminal plane molar and Class I canine relationships. The patient had no carious lesions, yet the posterior teeth were eroded. The erosion was characterized by exposed, yellow brown dentin in the major occlusal pits (Figure 3). After application of disclosing solution, plaque was found to cover less than 15% of the teeth.

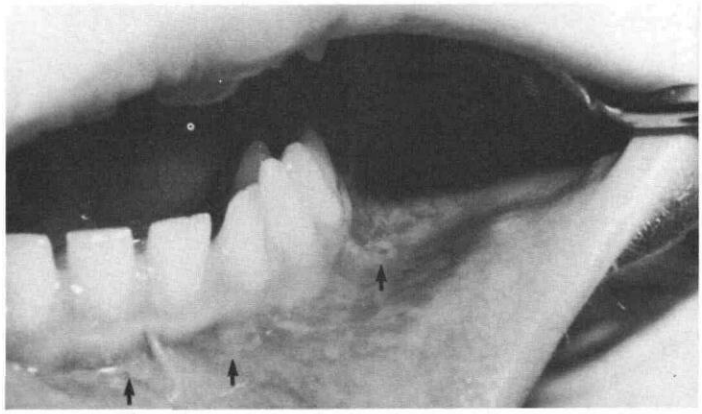

FIGURE 2. The white surface lesion (arrows) is apparent in the mandibular buccal vestibule. The lesion is especially prominent in the posterior region.

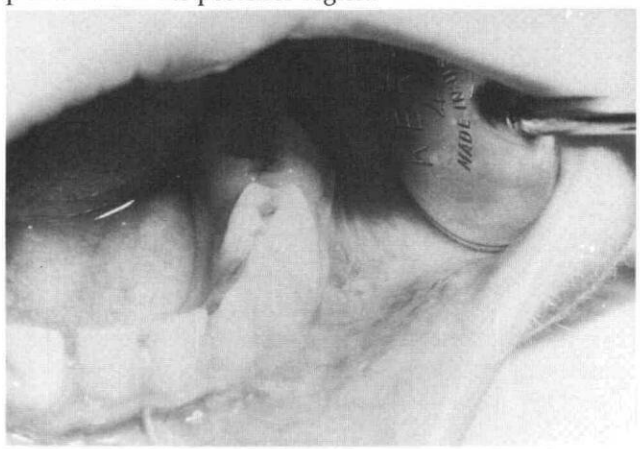

FIGURE 3. The first primary molar shows erosion characteristic of the patient's posterior teeth. There is exposed, yellow brown dentin in the major occlusal pits. Erosion is not as pronounced in the second primary molar.

The differential diagnosis of the lesion in the buccal vestibule included: (1) an epithelial reaction to large, continuous doses of acetylsalicylic acid allowed to dissolve in the mouth; or (2) shallow ulcerations manifesting a toxic reaction to the gold therapy.

The patient's physicians were notified about the problem. After conferences, a decision was made to continue the gold therapy until a definitive diagnosis

could be made due to the child's poor response to aspirin and Tolectin® therapy. The patient was rescheduled three days later to determine if any changes in the lesion could be detected that might aid in establishing the cause of the oral lesion.

At the second appointment remnants of the chewable aspirin the patient had received one-half hour earlier were detected in the mandibular buccal vestibule. After questioning, the mother disclosed that her daughter preferred to suck on the flavored children's aspirin as if it were candy. The mother further revealed that this habit had developed during initial treatment of the JRA. Given this information, it was concluded that the lesion was an epithelial reaction to large, continuous doses of asirin over a 12-month period.

Discussion

The presence of an unusual soft tissue lesion in this case was significant. A common adverse reaction to gold therapy is development of shallow ulcers on the buccal mucosa, border of the tongue, and palate. Any clinical sign of gold toxicity is an immediate indication to cease treatment. Unfortunately, the patient had not been appointed for an oral examination until after gold therapy had been initiated. This case reinforces the importance of obtaining a dental consultation prior to beginning therapy with a drug known to exhibit oral manifestations of toxicity. A baseline examination would have simplified lesion diagnosis in this case.

Oral pathology textbooks have documented the effect of aspirin on the oral mucosa; however, the presentation of the lesion in this case was uncharacteristic. The use of aspirin topically or locally in the oral cavity causes a mucosal reaction known as oral aspirin burn. The caustic action of the drug causes separation and sloughing of the epithelium and frequently bleeding in the immediate area of tablet placement.[11] In this case the child's habit of allowing the flavored, chewable aspirin to dissolve slowly in the mouth stimulated an epithelial change in the mandibular buccal vestibule which may be termed a "chronic aspirin burn." Repeated doses of dissolved aspirin apparently irritated the epithelium enough to create a white surface lesion; however, irritation was insufficient to cause bleeding, patient discomfort, or other signs or symptoms.

Sullivan and Kramer felt that erosion of teeth in JRA patients was due to the acidic nature of aspirin particles packed into occlusal grooves and crevices.[10] In this clinical report the patient did not chew the aspirin but let it dissolve in her mouth. Because the teeth were eroded it may be deduced that aspirin need not lodge in the teeth to cause erosion. Rather, it need only be present in the oral cavity for a length of time sufficient to lower salivary pH to levels sufficient for enamel dissolution to begin. A similar case has been described wherein the lingual and incisal surfaces of the maxillary incisors were eroded in a patient who allowed aspirin, phenacetin, and caffeine (APC) powder to dissolve slowly in the saliva before swallowing.[12]

In order to identify aspirin as the absolute causative agent of the lesion, it would have been necessary to discontinue the aspirin therapy until the lesion resolved. In this case, discontinuation of treatment could not be considered because the physician felt that the disease was too severe to stop therapy. Consequently, the lesion was identified as an epithelial reaction to aspirin based on maternal information and evidence of other tissue alterations related to salicylate therapy, namely erosion of teeth and low levels of plaque colonization.

Conclusion

Chewable aspirin therapy in patients with JRA previously has been noted to have caused tooth erosion due to the acidic nature of aspirin particles. This report describes a case in which chewable aspirin caused a change in the epithelium as well as tooth erosion when tablets were allowed to dissolve in the oral cavity. The epithelial change, similar to an aspirin burn, has been described as a chronic aspirin burn.

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