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The American Academy of Pediatric Dentistry
Volume 9 Number 4

Case Reports

Tuberculous osteomyelitis in the mandible of a child

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Abstract

Tuberculous osteomyelitis is a specific infection caused by the acid-fast bacillus Mycobacterium tuberculosis. Involvement of the oral region is rare, especially in children. Almost all cases arise secondary to known pulmonary disease and involve the oral tissues via three routes: direct inoculation; extension from other infected sites; or hematogenous seeding. The clinical, radiologic, histologic, and therapeutic aspects of this condition are reviewed.

Literature Review

Tuberculosis is an infectious condition caused by *Mycobacterium tuberculosis*, although *M. bovis* and *M. avium* have also been implicated (Harris and McClement 1983). Transmission is via air-suspended droplets and is more likely to occur in areas where hygiene practices and nutritional status are less than ideal because the disease is not particularly infectious in healthy individuals (Harris and McClement 1983). Initial lesions are almost exclusively pulmonary; however, once infected, redirection throughout the body may occur via direct inoculation, extension of adjacent tuberculous lesions, or hematogenous seeding (Cohen 1959).

In the most recent review, the World Health Organization (WHO) estimated that 7 million cases of infectious tuberculosis existed in the world, with higher concentrations reported in Asia, Africa, and the southern and eastern Pacific rim (WHO, 1974). Kassner (1978) stated that tuberculosis is South Africa's number one health problem and even in the United States, 27,000 new cases of tuberculosis were reported in 1980 according to Serota (1983).

The increased prevalence of acquired immunodeficiency syndrome (AIDS) may result in a heightened susceptibility to tuberculosis lesions (Doble et al. 1985), especially from atypical Mycobacteria, (Ma and Armstrong 1984) leading to the possibility of an increased incidence of tuberculosis in the future. Furthermore, even though the number of cases of pulmonary tuberculosis has declined, the incidence of extrapulmonary tuberculosis has remained relatively stable (Laurence 1983).

Skeletal lesions, although not a common accompaniment of tuberculosis, (Davidson and Horowitz 1970), accounted for 6.6% of extrapulmonary lesions in a series of patients reported by Weir and Thornton (1985).

Despite this rather grim picture, tuberculous osteomyelitis of the mandible is comparatively rare, accounting for less than 2% of osseous lesions (Jones and Miller 1963). A review of the reported cases (Table 1) reveals

TABLE 1. Cases of Oral Tuberculosis Involving Bone

Study	*Age*	*Sex*	*Site*
Darlington and Salnan (1937)	30	♂	tooth apex
	58	♂	apex tooth 46
	35	♀	apex tooth 38
	20	♀	apex tooth 36
	27	♂	apex tooth 46
	50	♀	apex tooth 26
	50	♂	apex tooth 47
Asbell and Humphries (1939)	6	♂	mandible
Meng (1940)	8	♂	all in mandible
	6	♀	
Brodsky (1942)	33	♂	alveolus
Thoma (1945)	39	♂	posterior mandible
Stuteville and Hulswit (1948)	30	♀	mandible
Oppenheim et al. (1951)	41	♂	tooth apex
	33	♂	tooth apex
Shengold and Sheingold (1951)	—	—	mandibular angle (1), apical osteitis (9)
Thilander and Wennström (1954)	39	♀	mandibular angle
Pekarsky (1954)	10	♂	mandibular angle
Spilka (1955)	20	♂	posterior mandible
Boyes et al. (1956)	8	♀	posterior mandible
	8	♀	tooth socket
Allan (1957)	26	♂	posterior mandible
Cohen (1959)	48	♂	posterior mandible
Bradnum (1961)	32	♂	posterior mandible
Taylor and Booth (1964)	6	♂	right ramus
Weidmann and MacGregor (1969)	22	♂	apex of tooth 47
Juniper (1973)	60	♂	bilateral maxilla
Ratliff (1973)	42	♀	tooth 43 region
McAndrew et al. (1976)	45	♂	posterior maxilla
Sachs and Eisenbud (1977)	2	♀	multiple skeletal
Garber and Harrigan (1978)	39	♂	posterior mandible
Sepheriadou-Mavoropoulo and Yannoulopoulous (1986)	35	♂	posterior mandible
	43	♀	apices teeth 11, 12

that osseous involvement in oral tuberculosis is not uncommon, with the most frequent presentation being localized apical osteitis. Horizontal, periodontal bone loss secondary to tuberculous gingivitis was less common, with fulminant tuberculous osteomyelitis of the jaw being relatively rare.

The purpose of this paper was to present a case of tuberculous osteomyelitis of the mandible in a nine-year-old male and to review the clinical, radiologic, and therapeutic aspects of this disease.

FIG 1. Panoramic radiograph of patient taken at the time of presentation reveals bone destruction and production with sequestrum formation and periosteal new bone (arrow).

Case Report

M.F., a nine-year-old male patient was referred to the Faculty of Dentistry, University of Stellenbosch, for consultation because of persistent infection in an extraction site. The patient was known to have tuberculosis confirmed by a chest radiograph and lymph node biopsy. The mandibular left first molar had been extracted two weeks previously at another institution because it had become loose. A panoramic radiograph at the time of presentation revealed a 3.5-cm diameter mixed radiolucent/radiopaque lesion in the left mandibular molar region (Fig 1). There was a central area of bone destruction with several small sequestra present. Surrounding the central radiolucent zone was a band of sclerosing osteitis. Examination of the inferior border of the mandible revealed a layer of periosteal new bone. Residual sclerosing osteitis existed around the apices of the extracted molar teeth. The developing mandibular left third molar had been displaced superiorly a substantial distance. A provisional diagnosis of osteomyelitis was made and the patient was subjected to sequestrectomy and biopsy of the left mandible. During surgery, several fragments of bone and soft tissue were recovered. These fragments showed zones of caseating necrosis with Langhans' giant cells and epithelioid cells (Fig 2). Surrounding this area were collections of chronic inflammatory cells. The final diagnosis was tuberculous osteomyelitis.

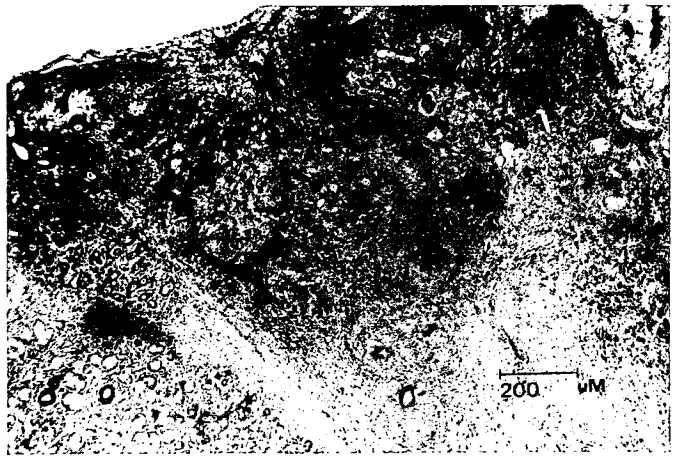

FIG 2. Histologic specimen (200x) reveals typical tuberculous granuloma with caseation necrosis, epithelioid cells, and Langhans' giant cells.

The patient was treated with a combination of isoniazid (15 mg/kg/24 hr) and rifampin (20 mg/kg/24 hr) and postoperative follow-up revealed that the surgical site had healed well (Fig 3).

Discussion

This case illustrates several important points in the management of patients with suspected tuberculous osteomyelitis. In a patient with known pulmonary tuberculosis and a destructive osseous jaw lesion, tuberculous osteomyelitis must be considered. The most common cause of sequestrum and laminar periosteal new bone formation, however, is suppurative osteomyelitis (Nortjé et al. 1987). Even though the patient had no evidence of caries or periodontal pathosis, the initial therapy was extraction. If teeth are loose, there is always

FIG 3. Panoramic radiograph of patient exposed three months following sequestrectomy showing regeneration of bone at the margin of the lesion (arrows).

a reason which must be ascertained before treatment is instituted. The infection in this patient was active at the time of the initial extraction and therefore probably arose via a hematogenous route. It is unlikely that the granuloma was caused following extraction by settling of expectorated bacillus, but this cannot be discounted.

Radiologic signs such as bone destruction, sclerosing osteitis, sequestration, and periosteal new bone formation are common in chronic osteomyelitis (Worth 1963; Adekeye and Cornah 1985) and were also present in this case. An unusual sign, present in this patient but not reported in other series of osteomyelitis patients, was the bodily movement of the third molar tooth follicle. It is possible that the granulomatous nature of this infection displaced the follicle in the same manner as a benign tumor could.

It is unclear why the mouth should be so infrequently affected by tuberculosis given the number of cases in some geographic locales. Meng (1940) postulated that the oral cavity has a natural high resistance to infection that may prevent this rather weak pathogen from establishing itself; however, there have been no controlled studies of the incidence of osseous oral lesions in tuberculosis patients.

The histologic pattern was that of a tuberculous granuloma and was characterized by caseous necrosis surrounded by epithelioid cells, lymphocytes, and occasional Langhans-type giant cells (Shafer et al. 1983).

Successful treatment of oral lesions is predicated upon ablation of the pulmonary disease (Margileth 1980). With respect to osseous involvement, it is important that all abscesses be drained and chemotherapy continued until radiologic evidence of healing has occurred (Margileth 1980). Because of the high frequency of secondary infection in the jaws (Adekeye and Cornah 1985), it is wise to perform sequestrectomy and provide drainage of abscesses concomitant with chemotherapy. Current therapy for childhood skeletal tuberculosis consists of a triad of antibiotics. Isoniazid and rifampin are used initially for a 12-month period at doses of 15-20 mg/kg/24 hr and 20 mg/kg/24 hr, respectively. Following this, para-aminosalicylate or ethambutol may be substituted for rifampin (Margileth 1980).

Summary

A case of a nine-year-old black male patient with tuberculosis of the left posterior mandible is reported. The radiologic features, although not typical of tuberculosis, did include sequestrum formation and laminar periosteal new bone. The lesion was treated by a combination of sequestrectomy and chemotherapy and responded well.

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Pioneer in Pediatric Dentistry: Kenneth Evermont Lawrence

Dr. Kenneth Evermont Lawrence was born in Stansberry, Missouri. He died on Monday, November 27, 1978.

Dr. Lawrence received the DDS degree from the University of Missouri School of Dentistry in 1940 and from 1940 to 1941 he interned at the Forsyth Dental Infirmary for Children in Boston. When he returned to Kansas City, prior to entering private practice in 1942, he was appointed director of the Lowry Clinic for Children. He returned to the Lowry Clinic in 1946-49 as its director when he returned from a tour of duty in the Navy at the end of World War II.

Dr. Lawrence, affectionately known as "Kenny" to his friends and colleagues, was in the private practice of pediatric dentistry throughout his professional career. He also was associated with the University of Missouri School of Dentistry as a special lecturer.

In 1973 Dr. Lawrence joined the faculty of Northwestern University School of Dentistry as an associate professor of clinical pediatric dentistry. He conducted a seminar with the graduate students in pediatric dentistry and also assisted the undergraduate students in the pediatric dental clinic.

Dr. Lawrence served the American Academy of Pediatric Dentistry as a member of the Board of Directors and as president in 1969-70. He was general chairman of the Academy's Annual Session in Kansas City in May, 1974. He was president of the Kansas City and Missouri units of the American Society of Dentistry for Children, Fellow of the American College of Dentists, and a member of Omicron Kappa Upsilon and the Rotary Club.

An excellent golfer, Dr. Lawrence was a member of the Missouri Hills Country Club, serving as its secretary at the time of his death. He was a member of the Par Club, the Western Golf Association, the Evans Scholarship Fund, and the Missouri Seniors Golf Association.

Dr. Lawrence is survived by his wife Geraldene, son Kenneth, daughter Mrs. Jerry Craig, mother Mrs. Mable Lawrence, brother Charles, sister Mrs. Jean Arnold, and three grandchildren.

Ralph L. Ireland, Historian