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Garre's osteomyelitis: a case report

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Abstract

One case of Garre's osteomyelitis involving the mandible was seen, treated and documented. It was treated by extraction of the causal infected tooth with no supplement of antibiotics. The patient experienced a complete regression of the lesion with six months.

Introduction

Garre's osteomyelitis was first described by Carl Garre in 1893 as "a focal gross thickening of periosteum with peripheral reactive bone formation resulting from infection."[1] It was reported only in long bones, particularly in the tibia, until 1948 when Berger described a case involving the mandible.[2] In 1973, Batcheldor et al.[3] claimed that only six reports of proliferative osteomyelitis of the jaw had been reported, and added two cases of their own. Since then, a few more[4,5] were presented and described in the English literature. Authors agree that there are many more cases, but they are not recognized and therefore not reported.

Several connotations have been adopted for this entity, but today the most commonly used is "Chronic osteomyelitis with proliferative periostitis."[6]

Signs and Symptoms

Garre's osteomyelitis occurs most commonly before the age of 20, though Thoma[7] described such a lesion in a 53-year-old patient. In the young, there is still considerable activity of osteoblastic cells in the periosteum, causing, therefore, a condensation of cortical bone rather than an osteolytic process. It usually affects the mandible and results in a hard swelling over the jaw, producing facial asymmetry with little or no pain.

Approximately 55% of the patients described had no pain at all, even to palpation. The others experienced little or moderate pain, with or without temperature elevation. The overlying skin was normal, but could occasionally be inflammed, mostly when pain was present.

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Palpation revealed a usually smooth, bone-hard lesion which felt like an inherent part of the mandible. The size of the bone lesion could vary from a few centimeters to the whole length of the mandible, and could expand as much as 2 cm laterally. All cases caused a noticeable asymmetry of the face.

Unlike other forms of osteomyelitis, there is no marked increase in fever, white blood cell count, sedimentation rate or alkaline phosphatase values.

Radiographic Findings

Panoramic and occlusal views would typically show a localized overgrowth of bone on the outer surface of the cortex. This mass of bone, which is supracortical but subperiosteal, is smooth, fairly calcified, and is often described as a duplication of the cortical layer of the mandible.

Since panoramic and occlusal radiographs can only demonstrate a vertical and a lateral apposition of bone respectively, it can be helpful to take a lateral oblique view of the jaw in order to visualize the expansion of the lesion which tends to be both inferior and lateral to the lower border of the mandible.

Smith and Farman,[5] and Rowe and Heslop[8] described on their radiographs the "onion peel" appearance of the subperiosteal bone formation.

Intraoral radiographs would show a carious tooth, a radicular cyst, or a chronic infectious process in approximation to the bony mass.

Histologic Findings

The main characteristic is formation of new bone, or osteoid tissue, with bordering osteoblasts and some areas of bone resorption. Lymphocytes are commonly seen in marrow spaces.

All histologic examinations revealed young reactive bone formation, arranged as trabeculae of lamellated bone separated by connective tissue. The trabeculae were more or less close together, depending upon the cases. Ellis et al.[4] mentioned the trabeculae radially arranged to the cortical bone. Thoma[7] described them as being at a right angle to the cortex. All findings included the presence of diffuse chronic inflammation

with infiltration of lymphocytes and plasma cells.

Authors agree that the reaction is destructive in the early stage when osteoporosis can be observed in the adjacent medullary bone. However, as the layers of new bone arrange themselves around the lesion, the lytic lesions become more sclerotic.[8]

Etiology and Evolution

Microorganisms which are isolated in most cases are *Staphylococci pyogenes,* variety *aureus* or *albus,* although various *Streptococci* and some mixed organisms can be associated.[10]

Typical evolution of this lesion can be attributed to the fact that the high osteogenetic potential in young patients allows an osteoblastic process which is superior to the osteolytic one. This pattern is identical to that of condensing osteitis, which is frequently seen in the periapical areas of carious teeth, except that the proliferation of bone is of periosteal origin rather than endosteal.

Case Report

A 10-year-old white female was seen at the Montreal Children's Hospital in July, 1973, with a mass at the right side of the mandible; the patient complained of pain only in the lower left first molar (Figure 1).

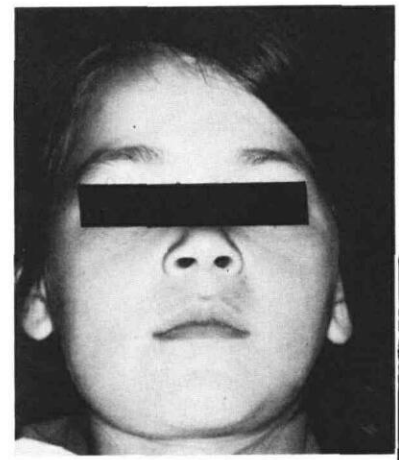


Figure 1. Ten-year-old white female patient with mass at the right side of the mandible.

Clinical examination revealed a non-tender, bone-hard mass extending from the second premolar to the second molar along the lower border of the right side of the mandible. The right lower first molar was cariously involved. The child had not sought treatment for that lesion, but for the painful carious left molar. She had no temperature elevation. Periapical radiographs showed caries on the lower first molars extending into the pulp chambers. The panoramic view revealed a smooth regular apposition of bone extending along the lower border of the right mandible and exhibiting a definite cortical outline (Figures 2 and 3). An occlusal radiograph showed an enlargement of bone, extending 1.5 cm buccally to the first lower right molar and stretching the periosteum. A clinical diagnosis of le Garre's osteomyelitis was made.

Both lower first molars were extracted. No other therapy was instituted, except for follow-ups. The patient failed to return until January, 1974, at which time a panoramic radiograph showed a nearly completed resolution of the lesion. Clinically, the mandible had remodeled itself, and the child's face was symmetrical. Another panorex in 1978 showed complete and permanent healing (Figure 4).

Discussion and Conclusion

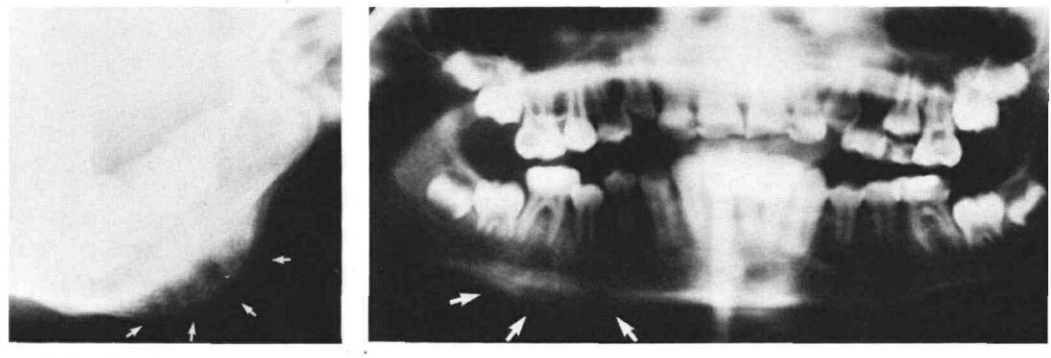
This case exhibited the same characteristic features as those reviewed in the literature:

— A long-standing carious lesion or other odontogenic infectious process associated with a bony hard swelling lateral to the inferior border of the mandible producing facial asymmetry, which brought the patient to seek treatment rather than the pain.

— The regression of the lesion with subsequent bone remodeling occurred within the same six to eight month period as seen in the literature.[4,6,9-11]

Because this case was associated with obvious dental causes and exhibited typical clinical and radiographic features of le Garre's osteomyelitis, it was not deemed necessary to perform any bone biopsies. However, in atypical cases with a negative history of deep carious lesions, chronic abscesses, or trauma to the area, bone biopsies are recommended in order to rule out several disease entities. These include the following:

1. Infantile Cortical Hyperostosis or Caffey's disease, which is a syndrome of unknown etiology arising during the first six months of life and affecting mostly the



Figures 2 and 3. Lateral and Panoramic radiographs revealing the mass.

mandible with the manifestation of a peripheral bony tumor. This disease runs a benign course and subsides without treatment in several months.[12]

2. Ewing's sarcoma, a rare malignant neoplasm occurring predominantly in children, which produces a bony tumor showing layers of new subperiosteal bone on radiographs when affecting the mandible. Radical surgery coupled with radiotherapy is recommended, but the prognosis is very poor.[12]

3. Osteogenic sarcoma, which mostly affects males between 10 and 25 years of age, and produces facial asymmetry when the mandible is involved. The sclerosing form exhibits the typical sun-rays appearance of osteosarcoma on X-Rays. Although radical surgery is the recommended treatment, this highly malignant disease carries a poor prognosis.[12]

4. Cherubism, a familial disease showing a slow, painless, symmetric swelling of the jaws which regresses as the patient approaches puberty.[12]

5. Histiocytosis X. Oral manifestations of the disease, if present, may include loss of alveolar bone, local pain, swelling and tenderness of the jaw. McKelvy et al.[13] describe a patient, diagnosed and treated for "osteomyelitis and sinus tract with a proliferative reaction in the buccal vestibule." After a few weeks of unsuccessful treatment, past medical history and a biopsy were taken and the final diagnosis of Histiocytosis was made.

A review of the literature has shown that this supposedly rare form of bone infection is becoming more and more common. Two reasons could account for this growing incidence:

1. As a result of the increase of health and living standards, people are responding in a "anabolic rather than catabolic manner."

2. The increased use of antibiotics has affected the virulence of microorganisms, turning an osteolytic process into a osteoblastic one. However, the abuse of antibiotics could be harmful to the patient. In many cases, the evolution into le Garre's osteomyelitis could be prevented if the dentist had thought about eliminating the causal factor rather than just instituting some antibiotic therapy.

We all agree that, in the presence of an infected tooth, the microorganisms are responsible for the irritation causing the host's proliferative response. However, Thoma[14] recalls that cultures from the bone of the tibia or femur are always sterile. Smith and Farman[5] offer a diagram showing a sinus formation with pus from the periapical area tracking toward the surface of the bone. Rowe and Heslop[8] took a culture of such a sinus which proved to be sterile as well. It seems that the culprit of the tissue irritation is not the bacteria per se, but the product of their presence and degradation. The different toxins and endotoxins, spread to surrounding tissues can account for the

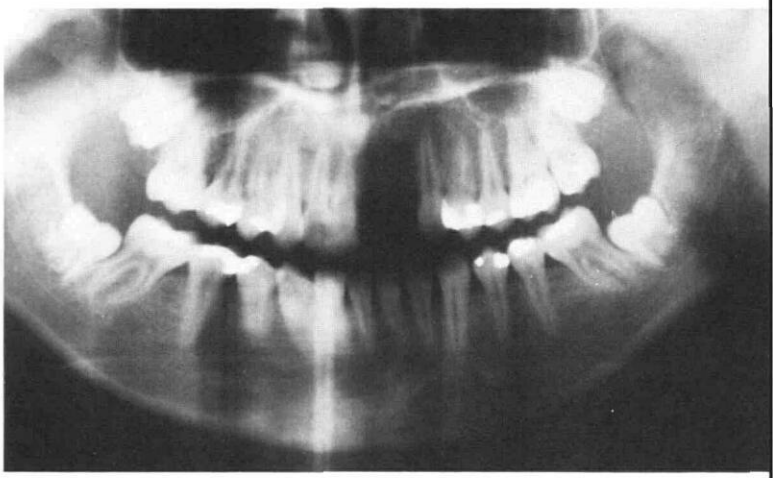


Figure 4. Panorex taken in 1978 showing absence of mass and normal symmetry.

maintenance of the chronic inflammation sites seen in microscopic examinations. The young host cannot entirely dispose of those byproducts, but can stimulate new bone formation in an attempt to encapsulate the lesion.

Because in many cases, there is no complaint of a "tooth ache," many physicians are inclined to perform bone biopsies (through an extraoral approach) without even thinking about a possible dental etiological factor. We feel, therefore, that patients presenting with any maxillofacial tumefaction, with or without oral symptoms, should be sent to a dentist for an evaluation.

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Quotable Quotes

The relationships of nutrition, diet, and cancer can be viewed from three perspectives: (1) diet as a factor in cancer causation; (2) the effect of cancer and its treatment on nutritional status; and (3) nutritional management of the cancer patient.

Various types of studies (epidemiologic, animal, case control) have described a number of highly suggestive associations between diet and cancer in humans, but there is as yet no absolute proof of a direct cause/effect relationship. The role that ingestion of food-borne carcinogens or carcinogen precursors has in causing major human cancers remains to be determined. It is likely that diet has an indirect role, modifying carcinogenesis. Several mechanisms are advanced to explain this effect. For example, it is theorized that excess dietary fat may promote carcinogenesis via its influence on altering bile acid production and/or gut microflora development in colon cancer, and secretion of endocrine glands in breast cancer.

Although there is probably no specific "preventive" diet for cancer, it may be advisable to eat a variety of foods, adjust energy intake to energy expenditure, and avoid moldy food, a deficiency of certain nutrients (e.g., vitamin A) and known dietary carcinogens such as alcohol. Cancer per se exerts both systemic effects (e.g., cachexia) and localized effects (e.g., malabsorption due to pancreatic insufficiency) which can lead to profound nutritional problems for the cancer patient. In addition, specific treatment modalities (e.g., surgery, radiotherapy, and chemotherapy), used singly or in combination, may compromise the patient's nutritional status.

Malnutrition need not be a necessary condition for the cancer patient. Advantages of nutritional intervention via oral, enteral, or intravenous hyperalimentation include improved well-being, enhanced weight gain, improvement in immunocompetence, and potentially a better response of the tumor to oncologic treatment. The effect of nutritional support on the overall outcome for the cancer patient is unknown. A concern is the possibility that nutritional support may harm the host by promoting tumor growth. Consequently, it is recommended that nutritional intervention be accompanied by adequate antitumor treatment. To date, there is insufficient evidence to support the suggestion that megadoses or reduced amounts of any essential nutrient, or removal of any normal dietary component, prevents cancer or has a useful role in its treatment in human beings.

From: *Diary Council Digest,* Vol. 51, No. 5, p 25, September-October 1980.

Diabetes mellitus and its complications are now thought to be the third leading cause of death in disease and cancer. According to a report issued by the National Commision on Diabetes in 1976, as many as 10 million Americans, or close to 5% of the population, may have diabetes, and the incidence is increasing yearly. The direct and indirect effects of diabetes on the U.S. economy are enormous, exceeding $5 billion per year. If current trends continue, the average American born today will have better than one chance in five of ultimately developing the disease. The likelihood of becoming diabetic appears to double with each decade of life and with every 20% of excess body weight . . .

Many aspects of diabetes remain a mystery, but recent work in three seemingly unrelated fields — genetics, immunology, and virology — has supported the contention that diabetes is a heterogeneous group of diseases rather than a single one. This work has also indicated that diabetes arises from a complex interaction between the genetic constitution of the individual and specific environmental factors.

From: Notkins, A. L.: "The Cause of Diabetes," *Scientific American,* Vol. 241, #5, pp 62-73, 1978.