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RESEARCH ARTICLE

ORAL OSTEOMYELITIS

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ABSTRACT

Introduction: Oral osteomyelitis is a bacterial infection transmitted through periodontal bone lesions, the incidence and prevalence decreased significantly due to early diagnosis and appropriate use of antibiotic therapy. 1 The clinical picture and course depend on the causative organism, and the strength of the patient's history and physical examination are essential for diagnosis and treatment. 2,3 Treatment is based on the surgical approach and the use of antimicrobials that can terminate the infection early in the course of the disease, so it is important to establish the etiology and specific antimicrobial therapy and to avoid unnecessary use of broad-spectrum antibiotics. It is necessary to understand the pathogenesis of the disease, because if not diagnosed or treated properly, the infection can be fatal or tend to become chronic. ***Objective:*** To inform the dental profession of the knowledge that makes up the state of the art on the diagnosis and timely treatment of osteomyelitis, in order to expedite the comprehensive management of this pathology that, without timely care, can be fatal**.** ***Material and methods:*** Taking as a reference recentreports of the scientific literature on the subject, this bibliographic review of the state of the art on oral osteomyelitis is carried out, using different sources such as: journals, scientific articles, books, archived material and other academic works with scientific basis. This documentary review provides a clear basis on osteomyelitis at present, information that allows clinicians to have a detailed overview of thispathology. ***Conclusions:*** osteomyelitis is an infection whose main risk is chronicity with impaired function. Early diagnosis and correct treatment is the basis for ensuring recovery and reducing the numberof recurrences. In spite of the fact that its diagnosis may raise doubts, due to the similarity with other entities, it is possible to detect it by gathering all the necessary data.5,6.

INTRODUCTION

Oral osteomyelitis is the infection of maxillary bones related to bacterial infection, transmitted to the bonethrough a devitalized tooth, periodontal lesions or traumatic bone lesions. The clinical picture and evolution of osteomyelitis depend, on the one hand, on the patient's resistance, immunological status, anatomical site of the lesion, nutritional status, patient's age and presence of pre-existing systemic factorsand, on the other hand, on the virulence of the causal microorganism. The incidence of oral osteomyelitisis not high, due to the fact that normal bone is very resistant to infection and, fundamentally, because of the development of antibiotic therapy.[7] The objective of this article is to inform the guild of endodontic and dental specialists in general, the knowledge that makes up the state of the art on the diagnosis and timely treatment of osteomyelitis, in order to expedite the comprehensive management of this pathology that, without timely care, can be fatal.

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This information is intended to summarize the most important aspects to be taken into account in the careof oral osteomyelitis at the first level of health care.

BACKGROUND: Etymologically, the term osteomyelitis derives from *osteo* , from the Greek “bone” *myelós* , “marrow”, and from *itis* “inflammation ". It is a rare disease nowadays. [1] Before the antibiotic era, it was a serious risk to life, but nowadays it is one of the health problems solved by modern medicine. One of the earliest references to this entity seems to be the one attributed to Sir Benjamin Brodie (1783- 1862), an English physician, who first described what would be known to the present day as Brodie's abscess, one of the chronic forms of osteomyelitis, in an article entitled "Results of some chronic cases ofabscesses of the tibia". [23]

EPIDEMIOLOGY: Osteomyelitis, by definition is the inflammation of the bone that includes the spongy portion, medullary, cortical, periosteum, blood vessels, nerves and epiphysis (Table 1); usually the term is related to the picture of suppurative infection. In the case of the jaws, odontogenic infection is considered the most frequent cause of osteomyelitis, through a devitalized tooth, periodontal lesions

or trauma. The infection interacts with the patient's immunological resistance factors and thus defines the clinical picture, the extent of the inflammatory process and the speed of development of the infection (Table 2). Osteomyelitis occurs at any age, with a greater predominance among men. It mainly affects thelower jaw, while it is rare in the upper jaw, due to its greater vascularization.[8]

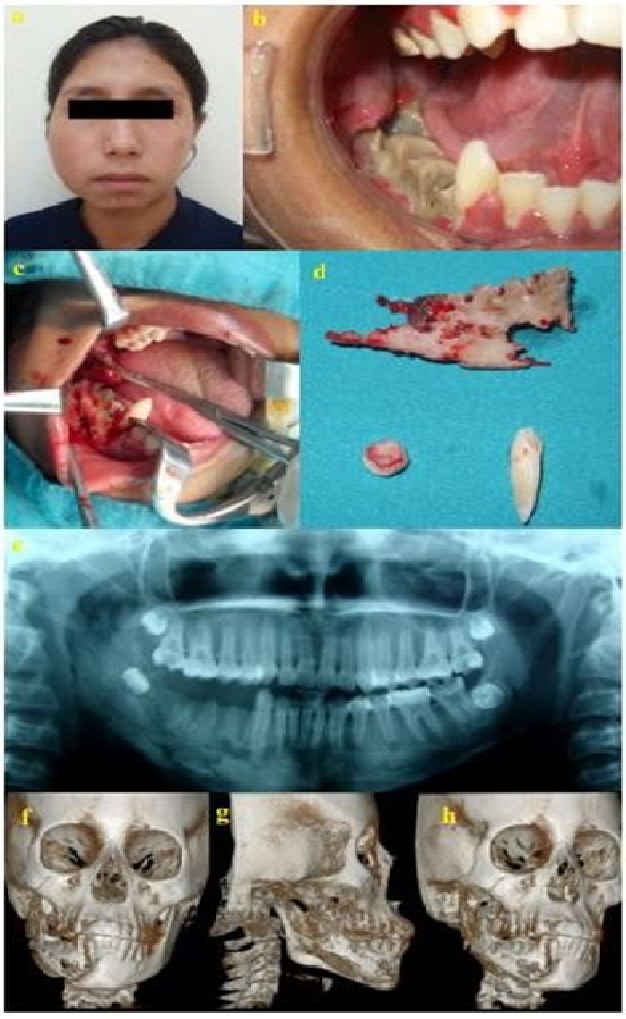
Box 1.0 Osteomyelitis, a suppurative infection of the bone, includes inflammation of:
Spongy, medullary and cortical portion.
Periosteum.
Blood vessels
Nerves
Epiphysis

Table 2.0 Common Causes of Osteomyelitis of the Jaws:
odontogenic infection .
Devitalized tooth.
periodontal lesions.
Open fractures.
Low immune resistance of the patient

ETIOPATHOGENY: The incidence of osteomyelitis has decreased and the prognosis has improved notably due to current antibiotic therapy.[9] In many cases it resolves spontaneously and in others it is aborted by antibiotic treatment. If not diagnosed or treated correctly, the infection becomes chronic. [10] It is important to know the etiopathogenesis of the disease, since delay in diagnosis and/or treatment can cause death. Usually the microorganisms are carried through the blood, from the primary focus; sometimesthe infectious source cannot be found, but it is assumed that there is a clinically minor lesion that is not apparent or has healed. Bacteremia may occur in circumstances such as: minor oral mucosal injury; vigorous chewing of hard foods, with or without obvious dental infection; frequent skin wounds and scratches. These infections are more frequent and severe in debilitated individuals. [11,12]

ETIOLOGY: Pyogenic osteomyelitis is usually caused by bacteria, rarely by fungi. Microorganisms can reach the bone by the hematic route; by direct contamination to the jaw from an adjacent infection, for example, from a dental abscess; by direct traumatic introduction, including surgery, especially when a metallic implant is used. [14] Almost all pathogens can be responsible, but the most frequent are Staphylococcus aureus (80-90%), often resistant to penicillin. Other less frequently encountered microorganisms are Escherichia coli, Pseudomona and Klebsiella; the causative microorganism of the most serious infections is coagulase- positive Staphylococcus, fortunately also rare (See figure one). [15]

In infections caused by extension or direct introduction of microorganisms, as occurs in post-surgical and post-fracture cases, mixed and anaerobic infections are frequent. In a minority of cases no germs are identified; this may be because they have been eradicated by the previous antibiotic or because the isolation method was not adequate. Chronic osteomyelitis may be a sequel to untreated or inadequately treated acute osteomyelitis or a prolonged low-grade inflammatory reaction that never progressed to a clinically significant acute phase. The clinical picture and course depend on the causative microorganism and the patient's resistance. The anatomical location, immune status, nutritional status, age of the subject and presence of systemic factors, such as osteoporosis and diabetes, affect the clinical picture and natural history of the disease.[16]



Patient with increased volume of the right hemiface. b) Clinical examination shows exposure of the alveolar ridge. c and d) Surgery: removal of bone abduction with dental pieces from the affected area and curettage of residual tissue. e) Panoramic radiography: Compromise of the mandibular body, angle and right coronoid with loss of the mandibular basal line. f, g and h) Tomographic reconstruction: The affectedregions are better evaluated and compromise of the mandibular condyle is also seen. Tomographic reconstruction: The affected regions are better evaluated and the involvement of the mandibular condyle is also appreciated.[7] Source: Juan Francisco Oré Acevedo and Martín La Torre Caballero, Osteomyelitis maxillae and mandible in pediatric patients. 2015, pp. 86-90. Not all cases of acute osteomyelitis follow this destructive course. The initial infection may be limited toa small area and remain isolated forming a localized abscess, or it may be a chronic nidus of infection.

Typical triggering or precipitating events include periapical inflammation as a result of a devitalized tooth, extractions, periodontal disease and fractures communicating with skin and mucosa. It is difficult to identify the specific infectious agent causing chronic osteomyelitis, either by microscopic or microbiological studies. Significant errors in the specimen are due to a small bacterial focus that is difficult to reach or contamination of the lesion by resident flora. Previously administered antibiotics also reduce the likelihood of culturing the causative microorganism. Although an etiologic agent is often not confirmed, most investigators believe that bacteria such as staphylococci or streptococci cause most cases of chronic osteomyelitis.[17]

DIAGNOSIS: The clinical history is important to determine host-dependent factors, both local and systemic. In the physical examination it is essential to establish which bone is involved, what the wound looks like, how long it is draining; and, if the time is long, if there is evidence of the existence of a

fistuloustract and any other morphofunctional abnormality that could be a warning sign. Laboratory diagnosis is not very sensitive and excessively variable; it is the clinical picture that should alert the dentist. For example, the mandible, especially the molar region, is affected much more frequently than the maxilla andpain is usually present, but varies in intensity and is not necessarily related to the extent of the disease. The duration of symptoms is proportional to the extent of the disorder. Swelling of the jaw is a frequently found sign; less often tooth loss and fistulous tracts are observed. Anesthesia of the affected region is veryrare.[18] Intense throbbing pain in the affected region is the main characteristic of this inflammatory process. It is accompanied by high hyperthermia, hypersensitivity aggravated by any movement, painful lymphadenopathy, leukocytosis; other frequently observed signs and symptoms of infection are redness, swelling and pain on palpation of adjacent soft tissues.

a) Patient with increase of right facial volume, b) Presents exposure of the upper maxillary rim, remnantsof dental pieces, c) retraction of the palatal and vestibular mucosa. d, e and f) Tomographic reconstructionwhere the compromise and bone destruction at the level of the upper maxilla up to the midline is visualized, but that reaches the lower orbital rim and nasal lateral wall. [7] Source: Juan Francisco Oré Acevedo and Martín La Torre Caballero, Osteomyelitis maxillae and mandible in pediatric patients. 2015, pp. 86-90. Sometimes, after spreading in a localized area of the bone, it is limited by the natural resistance of the hostor controlled by treatment. In some cases, the torpid nature of the infection stimulates osteoblastic activity,especially in the periosteum, forming new subperiosteal bone that surrounds and encloses the inflammatory focus (see figure 01). New bone formation also occurs within the marrow cavity, further localizing the infection. Histological changes depend on the stage of osteomyelitis and its duration. Basically, two elements can be identified: necrosis, destructive, suppurative and ischemic, and repair, fibrous and bone (see figure 02). [13]

However, the presentation may be much less striking, with unexplained fever, especially in children, or conversely, in the adult, only localized pain without fever. Occasionally, when the jaw is affected, paresthesia of the lower lip occurs. In the clinical differential diagnosis, the presence of this symptom should also suggest malignant mandibular neoplasia.Unless the process lasts more than a week, radiographic evidence of acute osteomyelitis is usually absent; with time, diffuse radiolucent foci begin to appear. [16] Diagnosis is confirmed by radiological demonstration of bone destruction. However, in the first days of osteomyelitis, bone necrosis may not be sufficient to produce radiological changes, so it is not useful in early diagnosis since changes often do not appear until after seven days. In these cases isotopic studies demonstrating tracer accumulation in foci of increased cellularity and vascularization are useful. On radiography, chronic osteomyelitis presents primarily as a radiolucent lesion that sometimes evidences focal areas of opacification. The pattern of transparency is often described as "termite gnawed" " due to its mottled radiographic appearance. The lesions can be very extensive and it is not uncommon for the borders to be indistinguishable. In the laboratory, the erythrocyte sedimentation rate is elevated and the number of leukocytes and neutrophils is increased. If acute osteomyelitis is suspected, blood cultures should be taken and large doses of antibiotics should be administered immediately to prevent septicemia.[15,]

RADIOGRAPHIC CHARACTERISTICS: Bone resorption is significant and the radiograph shows an osteolytic pattern. Sclerotic areas may be seen around the osteolytic area in long-standing lesions or in cases with pre-existing focal sclerosing osteomyelitis. [17] On plain radiographs, the margin of bone resorption is ill defined, but may be well defined at the site of cortical bone resorption (perforation). The periosteal reaction is usually laminated and appears as a thin, faint, radiopaque line adjacent and nearly parallel or slightly convex to the bone surface. A radiolucent band separates the new periosteal bone from the bone surface. If the process occurs repeatedly, an "onionskin" appearance is observed, caused by the presence of multiple lamellae. [17] On CT images, the pattern of osteolytic change in bone is continuous, not scattered, and extends into the periosteum through sites of cortical bone perforation (cloaca).

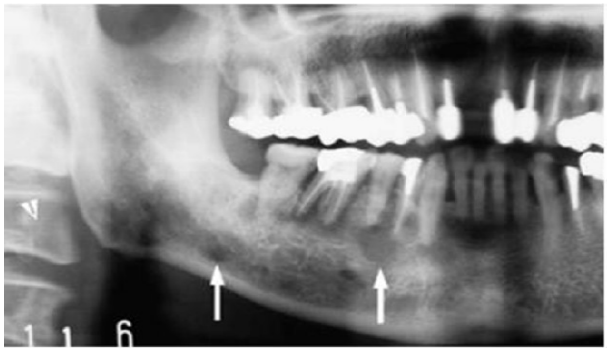


Figure 03. Panoramic X-ray

The margin of the cortical bone resorptionzone is moderately well defined and the remaining cortical bone appears to have near normal density. Periosteal reaction may be seen around the perforation site. [17] Bacterial osteomyelitis of the right mandible. The margin of the lesion is poorly defined, but sites of cortical bone resorption are seen with moderately well-defined margins (arrows). 19 Source: Y. Suei; A. Taguchi; K.

Tanimoto; 2003 axial CT image of a case of bacterial osteomyelitis shows cortical bone perforation (arrow). The remainingcortical bone is of near normal density. [19] Source: Y. Suei; A. Taguchi; K.Tanimoto; _ 2003. An axial CT image of a case of bacterial osteomyelitis shows cortical bone perforation (arrow). The remaining cortical bone has near normal density. 19 Source: Y. Suei; A. Taguchi; K. Tanimoto; 2003.

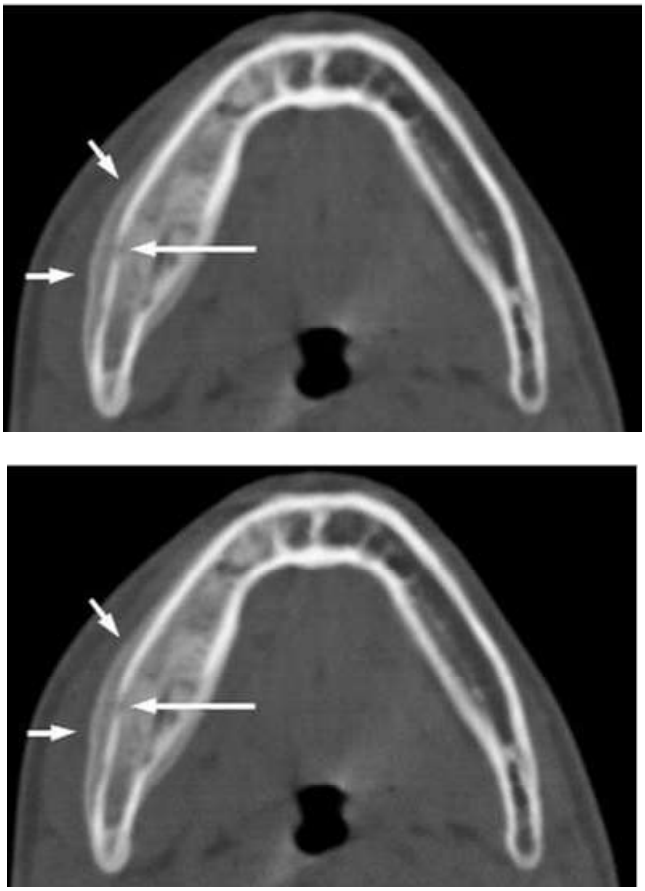
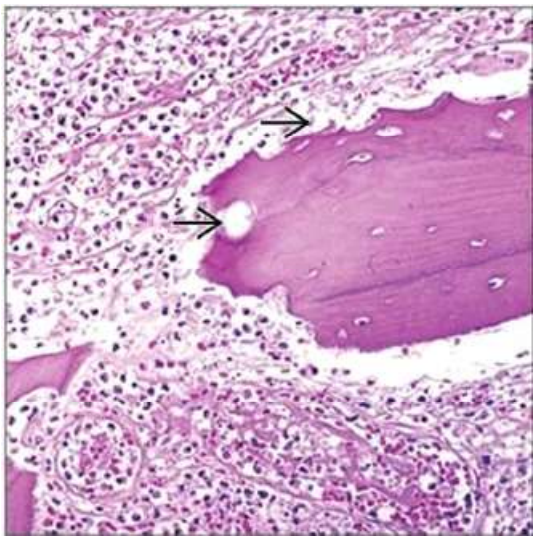


Figure 05. Axial CT 1

Histological characteristics: Among the general characteristics of osteomyelitis, fibrosis or edema of themedulla is a hallmark. The changes produced will depend on the phase (acute, subacute, chronic) in whichit is found. [20, 21]

Acute / Subacute Osteomyelitis: The biopsy of patients with acute osteomyelitis is not a common specimen, due to the predominance of the fluid component and the lack of a soft tissue component, because it mostly consists of necrotic bone. Histologically the bone shows loss of osteocytes from its lacunae, peripheral resorption and bacterial colonization. The periphery of the bone and the haversian canals contain necrotic debris and an acute inflammatory infiltrate consisting of polymorphonuclear leukocytes. The term "subacute osteomyelitis" is not clearly defined in the literature. Many authors use the term interchangeably with acute osteomyelitis, and some use it to describe cases of chronic osteomyelitis with more prominent features (subacute). We can observe inflammatory infiltrate of neutrophils, chronic inflammatory cells, bone marrow fibrosis, bone death and resorption. [23]

Chronic Osteomyelitis: Biopsy material from patients with chronic osteomyelitis demonstrates a significant soft tissue component consisting of chronically or subacutely inflamed fibrous connective tissue filling the intertrabecular areas of bone. Bone death with foci of new bone formation are common.[23]



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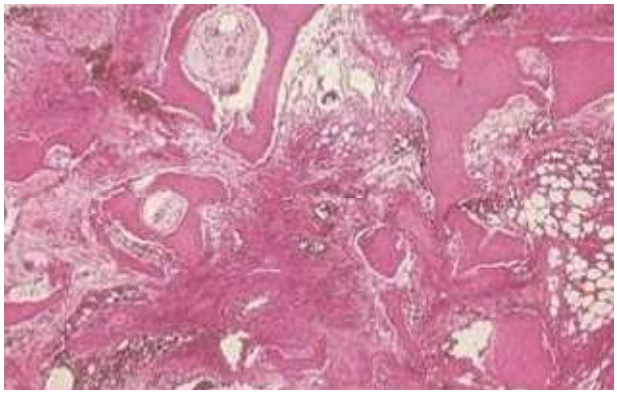

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TREATMENT: Some infections self-limit spontaneously and others are aborted by appropriate antibiotic treatment. Prompt treatment with antimicrobials can abort the infection at any early stage, before radiographic changes appear. Acute osteomyelitis is usually treated with antibiotics and drainage; ideally the causative agent should be identified and an appropriate antibiotic selected by laboratory sensitivity testing. Surgical approach may be part of the treatment and this is variable, as each case must be judged on an individual basis due to the degree of severity of the disease, causative microorganism and general condition of the patient. In most cases, long-term intravenous antimicrobial therapy is needed. It is therefore very important to obtain a biopsy of the involved bone to establish the etiology and antimicrobial treatment of the disease and thus use the appropriate antibiotic, avoiding the unnecessary use of broad-spectrum antibiotics. Blood cultures may be positive, especially in acute cases, but in chronic osteomyelitis they are usually negative.Undoubtedly, antibiotic selection should be guided by the corresponding antibiograms (see figure two).[10-16]

DISCUSSION

Oral osteomyelitis is transmitted through periodontal lesions that infect the maxillary bones; its clinical evolution depends on the microorganism and the patient's resistance and general condition. It is a conditionthat if not diagnosed and treated in a timely manner can be fatal. The infection can be self-limited or maintained as a chronic infection. It is caused by bacteria, transmitted by blood, by traumatic introduction or by direct contamination from an adjacent infection. The clinical history is fundamental to diagnose it in early stages. Diagnosis can be confirmed by radiological demonstration and laboratory tests, particularly blood culture. Timely treatment with specific antimicrobials.[15] The differential diagnosis described in the literature includes odontogenic facial cellulitis, abscesses or adenophlegmons, inflammation of the salivary glands, masseteric hypertrophy, infantile cortical hyperostosis or Caffey-Silverman syndrome, Ewing's sarcoma, fibrous dysplasia, hyperparathyroidism, Paget's disease, ossifying fibroma, reparative granuloma, chondroma and osteoclastoma, tuberculosis andsyphilis. Faced with a clinical picture like the one described above, an odontogenic infection or osteomyelitis was thought to be present, but it was only after performing the radiographic examination that the clinical diagnosis could be concluded. The definitive diagnosis was obtained with the anatomopathological examination.[16] With the advent of antibiotics, the prognosis of osteomyelitis is considered favorable, although it may be hindered in patients with debilitating systemic diseases, impaired responsiveness to infection and who areunder treatment with corticosteroids. [16] According to literature reports, osteomyelitis of the jaws can occur after infections of dental origin or oralsurgery treatment and is a life-threatening infection that most often affects the mandible. Osteomyelitis secondary to osteopetrosis occurs in 10% of these cases and tends to be refractory due to reduced blood supply and accompanying anemia and neutropenia. Its incidence has been dramatically reduced since theintroduction of antibiotics. [17]

CONCLUSIONS

After analyzing the state of the art, it is possible to identify that osteomyelitis is an infection whose mainrisk is chronicity with impairment of function. Early diagnosis and correct treatment is the basis for ensuring recovery and reducing the number of recurrences. Although its diagnosis may raise doubts, dueto the similarity with other entities, it is possible to detect it by gathering all the necessary data. The treatment period can be long, but there are several alternatives to achieve remission of the disease, based, in the first place, on antibiotic therapy.[18] Osteomyelitis is an acute or chronic inflammatory process that can involve cortical and cancellous bone tissue. It is an infectious inflammatory disease produced by pyogenic germs. Almost any microorganism can be an etiological factor, although staphylococci and streptococci are most often identified. In this casethe bone marrow space is occupied by a purulent exudate, the bone trabeculae show reduced osteoblastic activity and increased osteoclastic resorption. In areas of bone necrosis (sequestration), osteocytes are lost and the bone marrow undergoes liquefaction. [19] Clinical findings in osteomyelitis include pain, inflammation and suppuration, as in the case in question, which two years ago showed a clinical picture that had these three elements, and more recently only presented with intrabuccal and extrabuccal edema and suppuration. [20]

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