مجلة المنتدى الأكاديمي (العلوم التطبيقية)
المجلد (9) العدد (1) 2025
ISSN (Print): 2710-446x , ISSN (Online): 2710-4478
تاريخ التقديم: 2024/12/21 ، تاريخ إرسال التعديلات: 2025/01/12، تاريخ النشر: 2025/01/16

The concept of Osteomyelitis and Inflammatory Bone Disorders: Histopathology, Diagnosis and management

Laila Mustafa Abdelrahim

Collage of Dentistry and Oral Surgery, Alasmarya university, Zliten, Libya
Lailamustafa2014@gmail.com

Abstract

Inflammatory diseases of the jaw bone can encompass a variety of conditions, characterized by the presence of inflammation in the maxillary or mandibular bones. Osteomyelitis, a bacterial infection of the bone, is one of the most notable examples. The jawbone's complex anatomy, including its proximity to vital structures such as nerves and sinuses, means that inflammatory diseases can lead to significant complications. Early intervention is crucial for preventing irreversible damage and alleviating pain. As research continues, new horizons are emerging in understanding the mechanisms behind inflammatory diseases of the jaw bone. Advances in molecular biology and genetics are paving the way for targeted therapies and personalized medicine approaches.

This paper aimed to categorized bone infections into. Meanwhile, a brief histopathological feature, radiographic appearance and treatment modality for each entity was provided.

Keywords: Bone disease, Osteomyelitis, Histopathology, Treatment.

Introduction

Osteomyelitis is extensive inflammation of the bone, and typically spreading through the marrow spaces. Osteomyelitis of the jaw is a common complication of dental sepsis (Ueda N et al., 2021). Various clinical subtypes were recognized, leading to confusion in typing and classification, due to variation in the clinical and pathological features of osteomyelitis being acute, chronic, suppurative or sclerotic, this reflecting the balance between the nature and severity of the irritant, the host defense, local and systemic predisposing factors (Koorbusch, G. F et al., 2011)

The vast majorities of osteomyelitis cases are caused by bacterial infections and result in an expanding lytic destruction of the involved bone, with suppuration and sequestra formation. This condition (osteomyelitis) may appropriately be termed

suppurativeosteomyelitis, bacterial osteomyelitis or secondary osteomyelitis. Osteomyelitis may also result from bacteremia (Baltensperger M., Eyrich G 2009). Another ill defined group of an idiopathic inflammatory disorder of bone that do not responds consistently to antibacterial medications and typically demonstrate sclerosis of bone without suppuration or sequestra formation may be included under the term of diffuse sclerosing osteomyelitis. Other unique patterns of inflammatory bone diseases include focal sclerosing osteomyelitis, proliferative periostits, and alveolar osteitis.

Suppurative oseomyelitis of the jaw is uncommon in developed countries, but it is significant difficulty in developing nations. The most common cause is odontogenic infections and jaw fractures. In Africa an important cause is the presence of acute necrotizing gingivitis or NOMA (Suei Y et al., 2005).

Predisposing factors

Osteomyelitis has been associated with multiple systemic diseases including diabetes, autoimmune states, malignancies, malnutrition, and acquired immunodeficiency syndrome, and disorders associated with decreased vascularity of bone (Kesting M.R et al., 2008)). Tobacco use, alcohol abuse and intravenous drug abuse, exanthematous fever and malaria, sickle cell anemia, malnutrition, collagen vascular disease, AIDS, dysosteosclerosis, pagets disease, end-stage cemento-osseous, dysplasia, may result in hypovascularized bone that is predisposed to necrosis and inflammation (Baur, D. et al., 2015).

The medications linked to osteomyelitis are steroids, chemotherapeutic agents, and bisphosphonates. Local conditions that adversely affect the blood supply can also predispose the host to a bony infection. Radiation therapy, osteopetrosis, and bone pathology can alter the blood supply to the area and cause osteomyelitis (Kesting M.R et al., 2008).

The incidence of osteomyelitis is much higher in the mandible due to the dense poorly vascularized cortical plates and the blood supply primarily from the inferior alveolar neurovascular bundle. It is much less common in the maxilla due to the excellent blood supply from multiple nutrient feeder vessels. In addition, the maxillary bone is less dense than the mandible (Julien Saint Amand M et al ., 2017).

Pathogenesis

In the maxillofacial region, osteomyelitis primarily occurs as a result of contiguous spread of odontogenic infections or as a result of trauma. Bone can get infected via the hematogenous route of infection through bacteremic seeding of bone from a distant source of infection, contiguous spread from surrounding tissue and joints, or direct inoculation of bone from trauma or surgery Primary hematogenous osteomyelitis is rare in the maxillofacial region (Schmitt SK., 2017). generally occurring in the newly born and very young due to breast feeding in this way the infection spread from the infected breast to the maxillary bone causing infection and osteomyelitis (Schmitt SK., 2017). The adult process is initiated by an inoculation of bacteria into the jaw bones. This can occur with the extraction of teeth, root canal therapy, or fractures of the maxilla or mandible.

This initial insult results in a bacteria-induced inflammatory process, with inflammation there is hyperemia and increased blood flow to the affected area. Additional leukocytes are present in the area to fight off infection. Pus is formed, when the pus and subsequent inflammatory response occur in the bone marrow, an elevated intramedullary pressure is created which further decreases the blood supply to this region(Hatzenbuehler J, Pulling TJ .,2011).

The pus can travel via haversian and Volkmann's canals to spread throughout the medullary and cortical bones (Lew, D. P., & Waldvogel, F. A., 2004). Once the pus has perforated the cortical bone and collects under the periosteum, the periosteal blood supply is compromised and this further aggravates the local condition. The end point occurs when the pus exits the soft tissues either by intraoral or extraoral fistulas (Lew, D. P & Waldvogel, F. A., 2004).

Different Types of Bone Infections (Osteomyelitis)

1. **Acute osteomyelitis**.

Acute suppurative osteomyelitis the condition results when an acute inflammatory process spreads through the medullary spaces of the bone and insufficient time has passed for the body to react to the presence of the inflammatory infiltrate (Haeffs TH et al., 2018).

Patients with acute osteomyelitis have signs and symptoms of an acute inflammatory process that has typically been less than 1 month in duration, Fever, leukocytosis, lymphadenopathy, significant sensitivity and soft tissue swelling of the affected area and paresthesia of lower lip may be present (Koorbusch GF et al., 2011). A fragment of necrotic bone that has separated from the adjacent vital bone is termed a sequestrum.Sequestra often exhibit spontaneous exfoliation, On occasion; Fragments

of necrotic bone may become surrounded by vital bone and the mass of encased nonvital bone is called an involucrum (Andre CV et al., 2017).

Histopathologic Features

obtaining biopsy material from patients with acute osteomyelitis is not common because of the predominantly liquid content and lack of a soft-tissue component(Schuknecht B, Valavanis A 2003). When submitted, the material consists predominantly of necrotic bone. The bone shows a loss of the osteocytes from their lacunae. Peripheral resorption and bacterial colonization (Schuknecht B, Valavanis A., 2003). The periphery of the bone and the haversian canals contain necrotic debris and an acute inflammatory infiltrate consisting of polymorphonuclear leukocytes. The submitted material will be diagnosed as a sequestrum unless a good clinicopathologic correlation points to the appropriate diagnosis of acute osteomyelitis (Schuknecht B, Valavanis A., 2003).

Treatment and Prognosis

antibiotics and drainage. Microbiologic study of the infectious material typically reveals a polymicrobial infection of organisms normally present in the oral cavity. The antibiotics most frequently selected include penicillin, clindamycin, cephalexin, cefotaxime, tobramycin, and gentamicin. In most patients, a sufficient and appropriate antibiotic regimen aborts the infection and averts the need for surgical intervention (Paterson DL., 2000). Several investigators have suggested that antibiotic therapy can bring about sterilization of the sequestra; therefore, these non vital bone fragments should be allowed to remain in place as scaffolding for the future development of new bone (Koorbusch GF et al., 2011).

2. Chronic osteomyelitis

If acute osteomyelitis is not resolved expeditiously, the enhancement of chronic osteomyelitis occurs, or the process may arise primarily without a previous acute episode (Baur DA et al., 2015).the condition result when the defensive response leads to the production of granulation tissue, which subsequently forms dense scar tissue in an attempt to wall of the infected area (Haeffs TH et al., 2018). The encircled dead space acts as a reservoir for bacteria, and antibiotics are difficult to reach the site (Prasad KC et al., 2007).

There may be swelling, pain, sinus formation, purulent discharge, sequestrum formation, tooth loss, or pathologic fracture, Patients mayexperience acute exacerbation or periods of decreased pain associated with chronic smoldering progression (Baur DA et al., 2015) .Radiographs reveal a patchy, ragged and ill-defined radiolucency that often containscentral radiopaque sequestra, occasionally;

the surrounding bone may exhibit an increased radiodensity, and the cortical surface can demonstrate significant osteogenic periosteal hyperplasia (Uçkay I et al.,2012). Because of an anatomic peculiarity, large portions of each jawbone receive their blood supply through multiple arterial loops originating from a single vessel. Involvement of this single feeder vessel can lead to necrosis of a large portion of the affected bone. Sequestration that has involved an entire quadrant of the jaw has been reported in long-standing cases of chronic osteomyelitis (Julien Saint Amand M et al ., 2017).

Histopathologic Features

Biopsy material from patients with chronic osteomyelitis demonstrates a significant soft tissue component that consists of chronically or sub acutely inflamed fibrous connective tissue filling the lntertrabecular areas of the bone. Scattered sequestra and pockets of abscess formation are common (Schuknecht B, Valavanis A ., 2003).

Treatment and Prognosis

Chronic osteomyelitis is difficult to manage medically, presumably because pockets of dead bone and organisms are protected from antibiotics by the surrounding wall offibrous connective tissue. Surgical intervention is mandatory (Hatzenbuehler, J., & Pulling, T. J. 2011). The antibiotics are similar to those used in the acute form but must be given intravenously in high doses (Conterno, L. O., & Turchi, M. D 2013). The extent of the surgical intervention depends on the spread of the process; removal of all infected material down to good bleeding bone is mandatory in all cases. For small lesions, curettage, removal of necrotic bone, and saucerization are sufficient (Dym H, Zeidan J 2017). In patients with more extensive osteomyelitis decortications or saucerization often is combined with transplantation of cancellous bone chips. In cases of persisting osteomyelitis, resection of the diseased bone followed by immediate reconstruction withan autologous graft is required (Hatzenbuehler, J., & Pulling, T. J. 2011). Weakened jawbones must be immobilized. The goal of surgery is removal of all infected tissue. Persistence of chronic osteomyelitis is typically due to incomplete removal of diseased tissue ((Koorbusch GF et al., 2011).. Upon successful elimination of all infected material, resolution is expected. Adjunctive procedures (e.g. hyperbaric oxygen) are rarely necessary if thorough surgical curettage and sequestrectomy have been accomplished (Chen, L.,et al., 2013). Hyperbaric oxygen is primarily recommended for the rare patient whodoes not respond to standard therapy or for disease arising in hypovascularized bone e.g., osteoradionecrosis, osteopetrosis, Paget's disease and cemento-osseous dysplasia (Koorbusch GF et al., 2011).

Radiographical appearance

It is possible to see a patient with acute osteomyelitis that has a normal-appearing orthopantomogram. However, one can often see the appearance of "moth-eaten" bone or sequestrum of bone, which is the classic appearance of osteomyelitis (Omami G 2023). Computerized tomography (CT) scans have become the standard in evaluating maxillofacial pathology such as osteomyelitis. its sensitive in late stage when 30%-50% of the bone demineralized (Schuknecht, B., & Valavanis, A 2003).

Magnetic resonance imaging (MRI) is generally considered more valuable in the evaluation of soft tissue lesions of the maxillofacial region. However, MRI can assist in the early diagnosis of osteomyelitis by loss of the marrow signal before cortical erosion or sequestrum of the bone appears. Thus, MRI may benefit in identifying the earlier stages of osteomyelitis. Nuclear medicine has evolved to aid in the diagnosis of osteomyelitis. The technetium 99 bone scan is very sensitive in high lighting areas of increased bone turnover (Schuknecht, B., & Valavanis, A. 2003).

3. Focal Sclerosing Osteitis

Focal sclerosing osteitis is a relatively common phenomenon that is believed to represent a focal bony reaction to a low-grade inflammatory stimulus (Holly, D et al., 2009). It is usually seen at the apex of a tooth with long-standing pulpitis. This lesion may occasionally be adjacent to a sound, unrestored tooth, suggesting that other etiologic factors such as malocclusion may be operative (Yoshino, A. et al., 2011). Synonyms for focal sclerosing osteitis include focal sclerosing osteomyelitis, bony scar, condensing osteitis, and sclerotic bone (Holly, D et al., 2009).

Clinical Features

Focal sclerosing osteitis may be found at any age but is typically discovered in young adults. Patients are usually asymptomatic, and most lesions are discovered on routine radiographic examination (Owlia, F et al., 2011). A majority are found at the apices of mandibular first molars, with a minority associated with mandibular second molars and premolars ((Owlia, F et al., 2011).

Radiographically, one of several patterns may be seen. The lesion may be uniformly opaque, it may have a peripheral lucency with an opaque center, it may have an opaque periphery with a lucent center, or it may be composed of confluent or lobulated opaque masses (Yoshino, A. et al., 2011).

Histopathology

Microscopically, these lesions are masses of dense sclerotic bone; Connective tissue is

scant, as are inflammatory cells (Pedro R et al., 2018).

Treatment

Because it is believed to represent a physiologic bone reaction to a known stimulus, the lesion itself need not be removed. A biopsy might be contemplated to rule out more significant lesions that received serious consideration in the differential diagnosis (Pedro R et al., 2018). The inflamed pulp that stimulated the focal sclerosing osteomyelitis should be treated. The decision about whether the tooth should be restored, treated endodontically, or extracted should be made on a case-by-case basis according to findings.

4. Diffuse sclerosing osteomyelitis

Diffuse sclerosing osteomyelitis is an ill-defined, highly controversial, evolving area of dental medicine. This diagnosis encompasses a group of presentations that are characterized by pain, inflammation, and varying degrees of gnathic periosteal hyperplasia, sclerosis, and lucency. On occasion, diffuse sclerosing osteomyelitis can be confused with secondarily inflamed intraosseous pathoses (florid cementosseous dysplasia) or Paget's disease of bone (Jia, K., et al., 2021).

Diffuse sclerosing osteomyelitis represents an inflammatory reaction in the mandible or maxilla, believed to be in response to a microorganism of low virulence. Bacteria are generally suspected as causative agents, although they are seldom specifically identified. Chronic periodontal disease, which appears to provide a portal of entry for bacteria, is important in the etiology and progression of diffuse sclerosing osteomyelitis. Carious non vital teeth are less often implicated (Marí, A., et al., 2014).

Clinical Features

This condition may be seen in any age, in either sex, and in any race, but it tends to occur most often in middle-aged black women. The disease is typified by a protracted chronic course with acute exacerbations of pain, swelling, and occasionally drainage (Monsour, P & Dalton, B et al., 2010).

Radiographically:

This process is diffuse, typically affecting a large part of the jaw. The lesion is ill defined. Early lucent zones may appear in association with sclerotic masses (Baltensperger, M et al., 2004). In advanced stages, sclerosis dominates the radiographic picture. Periosteal thickening may also be seen. Scintigraphy may be particularly useful in evaluating the extent of this condition.

Histopathology

The microscopic changes of this condition are inflammatory, Fibrous replacement of marrow is noted; a chronic inflammatory cell infiltrate and occasionally a neutrophilic infiltrate are also seen (Kodama, Y., et al., 2013). Bony trabeculae exhibit irregular

size and shape and may be linedby numerous osteoblasts, Focal osteoclastic activity is also present. The characteristic sclerotic masses are composed of dense bone, often exhibiting numerous reversal lines.

Chronic sclerosing osteomyelitis shares many clinical, radiographic, and histological features with florid osseous dysplasia. The two should be separated, because the former is an inflammatory/infectious process and the latter a bony dysplastic process. Treatment and prognosis are therefore dissimilar. Florid osseous dysplasia appears to be an extensive form of periapical cemental dysplasia and, unlike diffuse sclerosing osteomyelitis, may exhibit anterior periapical lesions and traumatic or simple bone cysts. Furthermore, florid osseous dysplasia is usually asymptomatic and appears as a fibroosseous lesion lacking an inflammatory cell infiltrate.

Treatment

The management of diffuse sclerosing osteomyelitis is problematic because of the relative avascular nature of the affected tissue and because of the large size of the lesion. Even with aggressive treatment, the course is protracted. If an etiologic factor such as periodontal disease or a carious tooth can be identified, it should be eliminated. Antibiotics are the mainstay of treatment and are especially helpful during painful exacerbations. Surgical removal of the diseased area is usually an inappropriate procedure because of the extent of the disease. However, decortication of the affected site has resulted in improvement in some cases. Low-dose corticosteroids have also been used with some success. Hyperbaric oxygen therapy may prove to be a valuable adjunct.

Recently, treatment with pamidronate has shown promising results.

5. Chronic Osteomyelitis with Proliferative Periostitis (Garré's Osteomyelitis)

Chronic osteomyelitis with proliferative periostitis, commonly known as Garré's osteomyelitis, is essentially a subtype of osteomyelitis that has a prominent periosteal inflammatory reaction as an additional component (Erişen, M et al., 2014). It most often results from a periapical abscess of a mandibular molar tooth or an infection associated with tooth extraction or partially erupted molars, It is most common in children.

The eponym Garré's osteomyelitis has been applied to this condition after the author, Dr. K. Garrés, who in1893 described the clinical features of 72 patients with osteomyelitis. The disease he described was most common in the femur, with only three cases occurring in the jaws. In the absence of histological and radiographic findings, which were unavailable at the time of the report, it is likely that Garrés was describing a form of recalcitrant, acute osteomyelitis that occurred in both adults and

children. It was not chronic osteomyelitis with proliferative periostitis. Therefore, the term osteomyelitis, although widely used in reference to this condition, is inaccurate. Garré's(Suma, R et al., 2007)

Clinical Features

This variety of osteomyelitis is uncommonly encountered. It has been described in the tibia, and in the head and neck area, it is seen in the mandible. It typically involves the posterior mandible and is usually unilateral. Patients characteristically present with an asymptomatic bony, hard swelling with normal appearing overlying skin and mucosa. On occasion, slight tenderness may be noted.

This presentation necessitates the differentiation of this process from benign mandibular neoplasms. Radiographs and a biopsy provide a definitive diagnosis.

Radiographically, the lesion appears centrally as a mottled, predominantly lucent lesion in a pattern consistent with that of chronic osteomyelitis. The feature that provides the distinctive difference is the periosteal reaction. This, best viewed on an occlusal radiograph, appears as an expanded cortex, often with concentric or parallel opaque layers. Trabeculae perpendicular to the onion skin layers may also be apparent (Kannan, S. K et al., 2006).

Histopathology

Reactive new bone typifies the subperiosteal cortical response. Perpendicular orientation of new trabeculae to redundant cortical bone is best seen under low magnification. Osteoblasts dominate in this area, and both osteoblasts and osteoclasts are seen centrally. Marrow spaces contain fibrous tissue with scattered lymphocytes and plasma cells. Inflammatory cells are often surprisingly scant, making microscopic differentiation from fibroosseous lesions a diagnostic challenge.

Treatment :

Identification and removal of the offending agent are of primary importance in chronic osteomyelitis with proliferative periostitis. Removal of the involved tooth is usually required. Antibiotics are generally included early in this treatment. The mandible then undergoes gradual remodeling without additional surgical intervention (Jayasenthil, A et al., 2015).

6. Alveolar osteitis (drysocket)

After extraction of a tooth, a blood clot is formed at the site, with eventual organization of the clot by granulation tissue, gradual replacement by coarse fibrillar bone, and, finally, replacement by mature bone. Destruction of the initial clot prevents appropriate healing and causes clinical syndrome known as alveolar osteitis (Chow O et al., 2020). Extensive investigations have shown that the clot is lost secondary to

transformation of plasminogen to plasmin, with subsequent lysis of fibrin and formation of kinins (fibrinolytic alveolitis): these are potent pain mediators. Local trauma, estrogens, and bacterial pyrogens are known to stimulate fibrinolysins(). This knowledge correlates well with the increased frequency of alveolar osteitis in association with inexperienced surgeons, traumatic extractions, oral contraceptive use and presurgical infections. In addition, inadequate irrigation at surgery and the use of tobacco products have been related to the development of the problem (Taberner-Vallverdú, M et al., 2015)

Clinical Features

The frequency of alveolar osteitis is higher in the mandible and the posterior areas. After oral contraceptive use is taken into account. They do not appear to be a significant sex predilection. The prevalence is between 1% and 3% of all extractions, but it increases to 25% to 30% for impacted mandibular third molars. The frequency appears to be decreased when impacted teeth are prophylactically removed rather than for therapeutic reasons after development of chronic inflammation of pericoronal tissues (Daly, B. J et al., 2022).

The overall prevalence is highest between 20 and 40 years of age (when the majority of teeth are extracted. although the likelihood of developing alveolar osteitis appears greatest for extractions in the 40- to 45-year-old age group The affected extraction site is filled initially with a dirty gray clot that is lost and leaves a bare bony socket (dry socket). The detection of the bare socket may be hindered by partial retention of the clot or by overlying inflamed tissue that covers the site. The diagnosis is confirmed by probing of the socket, which reveals exposed and extremely sensitive bone. Typically, severe pain, foul odor, and (less frequently) swelling and lymphadenopathy develop 3 to 4 days after extreaction of the tooth. The signs and symptoms may last from 10 to 40days (Blum, I. R. 2002)

Treatment and Prognosis

On evaluation of the patient complaining of postextraction pain, a radiograph should be taken of the affected area to rule out the possibility of a retained root tip or a foreign body. All sutures should be removed. The socket is irrigated with warm saline, followed by thorough clinical inspection of the socket for any unexpected pathosis (Koorbusch GF et al., 2011). Curettage of the socket is not recommended, because this typically increases the associated pain (Daly B et al., 2012). Potent oral analgesics should be prescribed, and the patient should be given a plastic syringe with instructions to keep the socket clean via home irrigation with a chlorhexidine or saline solution. This irrigation should continue until debris no longer collects within the healing socket (usually 3 to 4 week (Bowe DC et al., 2011)

7. Osteoradionecrosis

Osteoradionecrosis is one of the most serious complications of radiation to the head and neck but is seen less frequently today because of better treatment modalities and prevention. The current prevalence rate is less than 4%, whereas the frequency approached 15% less than 20 years ago. Although the risk is low, it increases dramatically if a local surgical procedure is performed within 21 days of therapy initiation or between 4 and 12 months after therapy. Radiation of bone results in permanent damage:' to the osteocytes and microvasculature system. The altered bone becomes hypoxic, hypovascular, and hypocellular. Osteoradionecrosis is the result of nonhealing, dead bone; infection is not necessarily present (Chronopoulos, A et al., 2018).

Radiation therapy is a valuable treatment modality in treating cancer of the maxillofacial region. Radiation therapy can be used alone or as adjunctive therapy in combination with surgery and chemotherapy.

Radiation therapy like any treatment modality has side effects, including mucositis and xerostomia. One of the most important side effects is osteoradionecrosis (ORN). ORN is generally caused by trauma to the radiated area, usually by dental extraction, but it can also occur spontaneously (Nadella, K. R et al., 2015)

The clinical picture of ORN is most commonly seen with pain and exposed bone in the maxillofacial region ORN is more common in the mandible than in the maxilla. A dosage of radiation above 5,000 to 6,000 rads is generally felt to make the mandible susceptible to ORN. Radiographically, the appearance on the orthopantomogram or CT scan resembles conventional osteomyelitis with areas of osteolysis and bony sequestrum. Often there is an appearance of moth-eaten bone present on these films.

Treatment of ORN is aimed at removing the nonviable (necrotic) tissue and allowing the body to heal itself. Minor debridement of exposed bone and Current therapy of HBO consists of 100% oxygen delivered in a pressurized manner ((Hinchy V., et al 2013). Tissues treated with HBO have increased levels of oxygen, which has a negative effect on bacteria and HBO treatment consists of dives or treatment sessions for 90 minutes based at 2.4 atm of pressure. 20 to 30 dives are given preoperatively before any surgical intervention is performed. The area of ORN is then debrided and followed with10 additional HBO treatments. Reconstruction of the maxillofacial region is based on the patient’s response to the treatment protocol. HBO treatments are expensive and available only in medical centers or academic health science centers (Hinchy, N. V., et al 2013)**.**

8. Osteochemonecrosis

A similar type of jaw necrosis may be seen as a complication of bisphosphonate therapy e.g., pamidronate, zoledronic acid . Bisphosphonates are currently used as part of the treatment regimen for patients with multiple myeloma, metastatic cancers to bone e.g.,breast or prostate cancer, Paget's disease, and osteoporosis because of their inhibitory effect on osteoclastic bone resorption. Bisphosphonates, taken for an extended period greater than 1 year but the patient at risk for non infectious jaw necrosis (Almazrooa, S. A., & Woo, S. B 2009).

The typical presenting clinical symptom of bisphosphonate-associated osteonecrosis is pain, and the characteristic sign is bone exposure. The lesion usually follows tooth extraction or other form of jaw surgery, although many cases seem to be spontaneous. As with osteoradionecrosis, the mandible is more commonly affected than the maxilla (Almazrooa, S. A., & Woo, S. B 2009)

The pathophysiological mechanisms leading to bisphosphonate-induced osteochemonecrosis of the jaws are different from osteoradionecrosis (Woo, S. B., et al., 2006). In bisphosphonate-induced osteochemonecrosis of the jaws osteoclastic action is reduced, but osteoblastic production continues, leading to an osteopetrosis like condition. These alterations in bone physiology with eventual increase of the medullary bone as the disease progresses and the inability of osteoclasts to remove superinfected “diseased” bone are regarded as causative factors. In contrast to osteoradionecrosis, where a radiation-induced avascular necrosis is the major cause, avascularity does not appear to be a major cofactor (Woo, S. B., et al., 2006).

Routine dental care can and should be provided, using routine local anesthetics. All procedures should be performed as atraumatically as possible with little tissue trauma, bleeding and risk for postoperative infection. Specific precautions may be necessary for special types of procedures (i.e., orthodontic, endodontic, prosthodontic, others). Of course, oral surgery or periodontal procedures involving the manipulation of bone will present the greatest risk (Bagan, J. Vet al.,2012).

There is no empirical evidence to inform the decision of whether to cease bisphosphonate therapy in the event of development of bisphosphonate osteonecrosis BON. Some guidelines recommend that the indication for bisphosphonate therapy be considered and bisphosphonate therapy stopped only if the systemic condition permits. Hence, management is interdisciplinary and involves ongoing close monitoring. Surgical treatment should be conservative or delayed. In the instance of any infection, aggressive use of systemic antibiotics is indicated (Carlson, E. R., & Basile, J. D., et al 2009).

Conclusion

In conclusion, inflammatory diseases of the jaw bone present complex challenges for affected individuals. Awareness of the causes, symptoms, diagnostic techniques, and management strategies is critical for effective care. Ongoing research holds promise for better understanding and treating these conditions, ultimately improving outcomes for patients everywhere.

Referances

Almazrooa, S. A., & Woo, S. B. (2009). Bisphosphonate and nonbisphosphonate-associated osteonecrosis of the jaw: a review. The Journal of the American Dental Association, 140(7), 864-875

Andre CV, Khonsari RH, Ernenwein D, Goudot P, Ruhin B. Osteomyelitis of the jaws: a retrospective series of 40 patients. J Stomatol Oral Maxillofac Surg 2017;118:261-264.

Baltensperger, M., Grätz, K., Bruder, E., Lebeda, R., Makek, M., & Eyrich, G. (2004). Is primary chronic osteomyelitis a uniform disease? Proposal of a classification based on a retrospective analysis of patients treated in the past 30 years. Journal of Cranio-Maxillofacial Surgery, 32(1), 43-50.

Baltensperger M., Eyrich G. Osteomyelitis of the Jaws. In: Baltensperger M.M., Eyrich G.K.H., editors. Osteomyelitis of the Jaws. Springer; Berlin/Heidelberg, Germany: 2009.
Bagan, J. V., Hens-Aumente, E., Leopoldo-Rodado, M., Poveda-Roda, R., & Bagan, L. (2012). Bisphosphonate-related osteonecrosis of the jaws: study of the staging system in a series of clinical cases. Oral oncology, 48(8), 753-757.

Baur, D. A., Altay, M. A., Flores-Hidalgo, A., Ort, Y., & Quereshy, F. A. (2015). Chronic osteomyelitis of the mandible: diagnosis and management—an institution's experience over 7 years. Journal of Oral and Maxillofacial Surgery, 73(4), 655-665.

Blum, I. R. (2002). Contemporary views on dry socket (alveolar osteitis): a clinical appraisal of standardization, aetiopathogenesis and management: a critical review. International journal of oral and maxillofacial surgery, 31(3), 309-317.
Bowe, D. D. C. (2011). The management of dry socket alveolar osteitis.

Carlson, E. R., & Basile, J. D. (2009). The role of surgical resection in the management of bisphosphonate-related osteonecrosis of the jaws. Journal of Oral and Maxillofacial Surgery, 67(5), 85-95.

Chen, L., Li, T., Jing, W., Tang, W., Tian, W., Li, C., & Liu, L. (2013). Risk factors of recurrence and life-threatening complications for patients hospitalized with chronic suppurative osteomyelitis of the jaw. BMC Infectious Diseases, 13, 1-8.
Chow O, Wang R, Ku D, Huang W. Alveolar Osteitis: A Review of Current Concepts. J Oral Maxillofac Surg. 2020 Aug;78(8):1288-1296.

Chronopoulos, A., Zarra, T., Ehrenfeld, M., & Otto, S. (2018). Osteoradionecrosis of the jaws: definition, epidemiology, staging and clinical and radiological findings. A concise review. International dental journal, 68(1), 22-30.

Conterno, L. O., & Turchi, M. D. (2013). Antibiotics for treating chronic osteomyelitis in adults. Cochrane database of systematic reviews, (9).

Daly B, Sharif MO, Newton T, Jones K, Worthington HV. Local interventions for the management of alveolar osteitis (dry socket). Cochrane Database Syst Rev. 2012 Dec 12;12.

Dym H, Zeidan J. Microbiology of acute and chronic osteomyelitis and antibiotic treatment. Dent Clin North Am 2017;61:271-282.

Erişen, M., Bayar, O. F., & Ak, G. (2014). Garre osteomyelitis: a case report. J Dent Fac Atatürk Univ, 9, 49-53.

Haeffs TH, Scott CA, Campbell TH, Chen Y, August M. Acute and chronic suppurative osteomyelitis of the jaws: a 10-year review and assessment of treatment outcome. J Oral Maxillofac Surg 2018;76:2551-2558.

Hatzenbuehler J, Pulling TJ. Diagnosis and management of osteomyelitis. Am Fam Physician. 2011 Nov 01;84(9):1027-33.

Hinchy, N. V., Jayaprakash, V., Rossitto, R. A., Anders, P. L., Korff, K. C., Canallatos, P., & Sullivan, M. A. (2013). Osteonecrosis of the jaw–Prevention and treatment strategies for oral health professionals. Oral oncology, 49(9), 878-886.
Holly, D., Jurkovic, R., & Mracna, J. (2009). Condensing osteitis in oral region. Bratislavske lekarske listy, 110(11), 713-715.

Jayasenthil, A., Aparna, P., & Balagopal, S. (2015). Non-surgical endodontic management of Garre's osteomyelitis: a case report. British Journal of Medicine and Medical Research, 9(3), 1-4.

Jia, K., Li, T., & An, J. (2021). Is Operative Management Effective for Non-Bacterial Diffuse Sclerosing Osteomyelitis of the Mandible?. Journal of Oral and Maxillofacial Surgery, 79(11), 2292-2298.

Julien Saint Amand M., Sigaux N., Gleizal A., Bouletreau P., Breton P. Chronic Osteomyelitis of the Mandible: A Comparative Study of 10 Cases with Primary Chronic Osteomyelitis and 12 Cases with Secondary Chronic Osteomyelitis. J. Stomatol. Oral Maxillofac. Surg. 2017;118:342–348.

Kannan, S. K., Sandhya, G., & Selvarani, R. (2006). Periostitis ossificans (Garrè's osteomyelitis) radiographic study of two cases. International Journal of Paediatric Dentistry, 16(1), 59-64.

Kesting M.R., Thurmüller P., Ebsen M., Wolff K.-D. Severe Osteomyelitis Following Immediate Placement of a Dental Implant. Int. J. Oral Maxillofac. Implant. 2008;23:137–142.

Kodama, Y., Tanaka, R., Kurokawa, A., Ohnuki, H., Sultana, S., Hayashi, T., ... & Takagi, R. (2013). Severe destruction of the temporomandibular joint with complete resorption of the condyle associated with synovitis, acne, pustulosis, hyperostosis, and osteitis syndrome. Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology, 116(2), e128-e133.

Koorbusch, G. F., Deatherage, J. R., & Curé, J. K. (2011). How can we diagnose and treat osteomyelitis of the jaws as early as possible?. Oral and Maxillofacial Surgery Clinics, 23(4), 557-567.

Lew, D. P., & Waldvogel, F. A. (2004). Osteomyelitis. The Lancet, 364(9431), 369-379.
Marí, A., Morla, A., Melero, M., Schiavone, R., & Rodríguez, J. (2014). Diffuse sclerosing osteomyelitis (DSO) of the mandible in SAPHO syndrome: a novel approach with anti-TNF therapy. Systematic review. Journal of Cranio-Maxillofacial Surgery, 42(8), 1990-1996.

Monsour, P. A. J., & Dalton, J. B. (2010). Chronic recurrent multifocal osteomyelitis involving the mandible: case reports and review of the literature. Dentomaxillofacial Radiology, 39(3), 184-190.

Omami G. The Moth-Eaten Mandible: Osteomyelitis. Ear, Nose & Throat Journal. 2023;102(8)

Owlia, F., Karbassi, M. H. A., Mirjalili, N., Zahir, S. T., Gholami, N., & Karimi, S. (2011). A localized sclerosing osteomyelitis at the periapex of a vital tooth: report of a misdiagnosis. Journal of dental research, dental clinics, dental prospects, 5(3), 102.

Prasad KC, Prasad SC, Mouli N, Agarwal S. Osteomyelitis in the head and neck. Acta Otolaryngol 2007;127:194-205.

Paterson DL. Recommendation for treatment of severe infections caused by Enterobacteriaceae producing extended-spectrum β-lactamases (ESBLs). Clin Microbiol Infect 2000;6:460-463.

Pedro R, José M, Ana D, João R, Eduardo G, Cátia S, António L, Tiago D, Irina X. Condensing osteitis in the diagnosis consultation at Egas Moniz University Clinic. Ann Med. 2019 May 28;51(Suppl 1):129.

Schmitt SK. Osteomyelitis. Infect Dis Clin North Am. 2017 Jun;31(2):325-338.

Schuknecht, B., & Valavanis, A. (2003). Osteomyelitis of the mandible. Neuroimaging Clinics, 13(3), 605-618.
Suei Y., Taguchi A., Tanimoto K. Diagnosis and Classification of Mandibular Osteomyelitis. Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. 2005;100:207–214.

Suma, R., Vinay, C., Shashikanth, M. C., & Reddy, V. S. (2007). Garre's sclerosing osteomyelitis. Journal of Indian Society of Pedodontics and Preventive Dentistry, 25(Supplement), S30-S33.

Taberner-Vallverdú, M., Nazir, M., Sánchez-Garcés, M. Á., & Gay-Escoda, C. (2015). Efficacy of different methods used for dry socket management: A systematic review. Medicina oral, patologia oral y cirugia bucal, 20(5), e633.

Uçkay I, Jugun K, Gamulin A, Wagener J, Hoffmeyer P, Lew D. Chronic osteomyelitis. Curr Infect Dis Rep. 2012 Oct;14(5):566-75.

Ueda N, Nakashima C, Aoki K, Shimotsuji H, Nakaue K, Yoshioka H, Kurokawa S, Imai Y, Kirita T. Does inflammatory dental disease affect the development of medication-related osteonecrosis of the jaw in patients using high-dose bone-modifying agents? Clin Oral Investig. 2021 May;25(5):3087-3093. doi: 10.1007/s00784-020-03632-7. Epub 2020 Oct 14. PMID: 33057886.

Woo, S. B., Hellstein, J. W., & Kalmar, J. R. (2006). Systematic review: bisphosphonates and osteonecrosis of the jaws. Annals of internal medicine, 144(10), 753-761.

مفهوم الالتهابات العظمية المختلفة التي تصيب عظام الفكين: تشخيصها وطرق علاجها

ليلى مصطفى عبدالرحيم

كلية طب وجراحة الفم والأسنان، الجامعة الأسمرية الإسلامية، زليتن، ليبيا
Lailamustafa2014@gmail.com

المستخلص:

يمكن أن تشمل الأمراض الالتهابية في عظم الفك مجموعة متنوعة من الحالات، التي تتميز بوجود التهاب في عظام الفك العلوي أو الفك السفلي. يعد التهاب العظم وهو عدوى بكتيرية تصيب العظام، أحد أبرز الأمثلة على ذلك.

إن التشريح المعقد لعظم الفك، بما في ذلك قربه من الهياكل الحيوية مثل الأعصاب والجيوب الأنفية، يعني أن الأمراض الالتهابية يمكن أن تؤدي إلى مضاعفات كبيرة. التدخل المبكر أمر بالغ الأهمية لمنع الضرر الذي لا يمكن إصلاحه وتخفيف الألم.

مع استمرار الأبحاث، تظهر آفاق جديدة في فهم الآليات الكامنة وراء الأمراض الالتهابية في عظم الفك. يمهد التقدم في البيولوجيا الجزيئية وعلم الوراثة الطريق للعلاجات المستهدفة وأساليب الطب الشخصي.

تهدف هذه الورقة إلى تصنيف التهابات العظام وفي الوقت نفسه تقديم شرح موجز عن التشخيص المرضي والمظهر الشعاعي وطريقة العلاج لكل مرض.

الكلمات المفتاحية: أمراض العظام، التهاب العظم، التشخيص المرضي، العلاج