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CASE REPORT

Maxillary osteomyelitis: A brief review of the literature and case report

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ABSTRACT

Maxillary osteomyelitis is a rare but severe inflammatory disease with significant destructive potential. Although its incidence has decreased in recent decades due to the widespread use of antibiotics and improved oral hygiene, it remains a considerable challenge in terms of early diagnosis and appropriate therapeutic approach.

The authors present a suggestive clinical case, namely a 58-year-old female patient with maxillary osteomyelitis of odontogenic etiology, accompanied by a review of the specialized literature.

KEYWORDS: maxillary osteomyelitis, odontogenic, inflammatory condition.

INTRODUCTION

In the past, osteomyelitis was frequently encountered and feared, considering its progression over time, uncertain outcome, and the potential disfigurement resulting from tooth and bone loss[1]. Infections of the maxilla continue to pose several challenges despite recent advances in diagnosis, surgical management, and antimicrobial therapy[2].

Maxillary osteomyelitis, characterized by bone destruction, necrosis, and sequestrum formation, is often a consequence of untreated dental infections. It has a lower prevalence compared to mandibular involvement, an aspect explained by the richer vascularization of the jaw, which provides greater resistance to infection[3,4]. However, some authors report a higher incidence of maxillary involvement, reaching up to 51.7%[5]. Regarding gender predilection, statistics show a higher frequency among males, with a variation of up to 5.2:1[5,6]. Age distribution varies, with increased incidence reported in the 4th and 5th decades of life[5,7].

An early and accurate diagnosis is crucial for initiating appropriate treatment and preventing potential complications such as involvement of the orbit and skull.

ETIOLOGY AND PATHOPHYSIOLOGY

Maxillary osteomyelitis occurs more frequently in the presence of several predisposing conditions: odontogenic infections (abscesses, deep caries, periodontal diseases), trauma or oral surgery, maxillary sinusitis,

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post-radiotherapy osteoradionecrosis, diabetes mellitus, immunodeficiencies, malnutrition, smoking, alcoholism, poor oral hygiene[2].

Maxillary osteomyelitis is manifested by one or more necrotic bone lesions that are exposed or can be probed through an intraoral or extraoral fistula in the maxillofacial region, persisting for at least 6-8 weeks without response to appropriate therapy. It may be accompanied by pain, inflammation, erythema, suppuration, and abnormal dental mobility. Although it can occur spontaneously, in most cases, it is the result of a dental procedure, for example, a tooth extraction or oral surgery. Dentoalveolar infections may precede the appearance of necrotic bone[3]. Another common cause of osteomyelitis of the jaw bone is represented by maxillary sinusitis. In a retrospective study conducted on a group of 32 patients, Peravali et al.[5] reported that in 74% of cases, the main etiological factor for maxillary osteomyelitis was the odontogenic component, followed by maxillary sinusitis (165 of the patients) and trauma (6.4%).

Maxillary infections usually progress from a dental infectious focus through simple adjacent spread (e.g., pulpal and periodontal infections) or through direct inoculation from trauma or surgical interventions (e.g., dental extractions, oral mucosal injuries, and maxillofacial fractures). Less commonly, the spread is hematogenous from a distant infectious focus, particularly in cases of acute osteomyelitis in infants and children.

Conditions such as diabetes mellitus, malnutrition and immunosuppression can be considered risk factors. In most of the cases found in the literature, the presence of diabetes mellitus has been a consistent finding and represented a negative prognostic factor for disease progression[1,5,8-10]. According to Peravali et al.[5], 68% of patients diagnosed with maxillary osteomyelitis also had diabetes, compared to 20% in mandibular osteomyelitis cases. In the study conducted by Lata et al.[10] on a group of 21 patients with maxillary osteomyelitis, diabetes was identified as one of the most significant contributing factors in 47.6% of cases. An explanation for this mechanism would be the vascular changes associated with diabetes, as well as the compromised immune system of diabetic patients.

Another risk factor for the development of this pathology appears to be the medication taken by the patient, with bisphosphonates being most frequently implicated[3,11].

DIAGNOSTIC AND TREATMENT CRITERIA

The diagnosis of maxillary osteomyelitis is based on clinical symptoms (pain, swelling, local fistula), correlated with imaging, biopsy, and bacteriological and mycological examination of a bone fragment collected under sterile conditions (without oral contamination). A careful clinical evaluation is essential for an accurate diagnosis. Maxillary osteomyelitis is usually considered when there is a destructive maxillary lesion with mild soft tissue and a periosteal reaction, with or without palatal involvement, in a clinical context suggestive of infection.

Radiological imaging plays a crucial role in the diagnosis and assessment of maxillary osteomyelitis, providing detailed information about the extent of the lesion and supporting therapeutic decisions. A key aspect of imaging is its ability to differentiate osteomyelitis from other non-infectious conditions with a similar radiological appearance. Imaging techniques used include panoramic radiography, computed tomography (CT), which provides detailed visualisation of bone structures and lesions, and magnetic resonance imaging (MRI), which can reveal inflammation of the soft tissue surrounding the affected bone[12,13]. These methods are essential for establishing an accurate diagnosis and guiding appropriate treatment. Panoramic dental radiography can identify early changes in osteomyelitis, such as widening of the periodontal ligament space, increased trabecular density of the alveolar bone, thickening of the lamina dura, or bone sequestration[13,14]. In advanced stages of the disease, it can reveal increased bone density, periosteal thickening, areas of opacification, or osteolysis[13,15]. A suggestive radiological appearance is the so-called "moth-eaten" bone. Computed tomography is significantly more sensitive in detecting changes, particularly cone-beam CT[16].

Histopathological diagnosis can be challenging, especially cases involving small specimens, incomplete excisions, or improper orientation of the specimen. Pseudoepitheliomatous hyperplasia is a benign epithelial proliferation, often underestimated in oral pathology practice, which can histologically mimic invasive squamous cell carcinoma, thus complicating the differential diagnosis. This benign entity is observed in association with various oral lesions, including those of infectious, inflammatory, reactive, and degenerative origin. Dense inflammation may mask or distort histological features, making it significantly difficult for pathologists to distinguish benign from malignant lesions. Histopathologically, osteomyelitis is characterized by the presence of bone necrosis with irregular aggregates at the level of the bony trabeculae, absence of osteocytes, absence of osteoblastic lining, chronic inflammatory infiltrate, and local lymphocytosis[17].

Bacteriological diagnosis is also a challenge. Changes in sampling techniques aimed at avoiding salivary contamination during bone sampling,

a strategy that facilitates the distinction between pathogens and contaminants, along with the specific characteristics of the microbial flora involved in the etiology of maxillary osteomyelitis, have a considerable impact on microbiological analysis and should constitute the first step in developing an evidence-based antimicrobial strategy[18,19]. The primary pathogens involved in mandibular osteomyelitis are viridans streptococci and anaerobes. *Staphylococcus aureus*, *Staphylococcus epidermitis*, *Fusobacterium*, *Peptostreptococcus*, and *Prevotella*, are organisms frequently found in cases of maxillary osteomyelitis. A rare cause of osteomyelitis, particularly at the maxillary level, is infection with *Actinomyces*[20]. Osteomyelitis caused by *Actinomyces* frequently presents as the so-called "lumpy jaw disease"[20]. In addition to the classic symptoms of osteomyelitis, cases associated with this bacterium are often marked by significant fistula formation. The infection can also spread to facial regions. Chronic mandibular osteomyelitis caused by *Actinomyces* leads to progressive bone destruction. Therefore, early diagnosis and appropriate multimodal treatment are essential to prevent disease progression. This bacterium, which normally lives as a saprophyte in the oral cavity, can cause mixed infections with other pathogenic bacterial species, initially favouring the development of soft tissue infections, with a tendency to evolve into chronic forms. Fungal infection may also be responsible for the onset of osteomyelitis[5,21-23]. In a 10-year study, Niranjan et al.[22] found the presence of fungi in 52% of osteomyelitis cases. Most studies indicate a polymicrobial involvement in maxillary osteomyelitis.

Close collaboration between clinicians and pathologists enables the correlation of clinical and histopathological findings, facilitating the exclusion of neoplasms and the avoidance of excessive or inappropriate treatments[24].

A multidisciplinary approach is essential for establishing an accurate diagnosis and implementing effective treatment, involving collaboration between ENT specialists, oral and maxillofacial surgeons, infectious disease specialists, radiologists, and pathologists[25]. Therapeutic success largely depends on the early identification of the pathology, close monitoring of clinical progression, and management of associated risk factors.

Treatment includes antibiotic therapy lasting several weeks (especially aminopenicillins and/or clindamycin). This must be complemented by surgical intervention, depending on the extent of the infection[26]. Prolonged bactericidal antibiotic therapy is particularly relevant in the presence of potentially resistant virulent microorganisms and compromised regional vascular perfusion[1]. Adjunctive hyperbaric oxygen therapy may be employed in more refractory forms of maxillary osteomyelitis to enhance the local and regional immune response of the maxilla, as well as to produce microvascular neoangiogenesis to support reperfusion. A correct and aggressive therapeutic approach is key to preventing disease-related morbidities[13].

CASE REPORT

A 58-year-old female patient, from an urban area, diagnosed with a tumor mass in the right paramedian maxillary dental arch that invades the anterior hard palate and the floor of the right nasal cavity, for which two biopsies were performed through an intraoral approach by the oral and maxillofacial surgeon, the histopathological results of which were inconclusive, was admitted to the ENT Department of Zalau County Emergency Hospital for a biopsy through the endonasal approach.

From the patient's medical history, we recall multiple dental treatments, and a dental consultation performed approximately 18 months ago, during which generalized peri-implantitis changes were identified in quadrant 4, incipient peri-implantitis in quadrant 3, the absence of premolars 14, 15, 24 and 25 in quadrants 1 and 2, with tooth 11 showing 5mm vestibularization.

The first pathological changes were observed as the patient underwent a dental CT scan (CBCT) for dental implants in the right upper jaw. As the CT scan showed irregular bone with altered architecture, the patient was referred to an oral and maxillofacial surgeon with a suspected diagnosis of radicular cyst in order to perform a cystectomy. As there were no signs of cystic lesions on the CT scan and an overall loss of bone architecture with widespread inflammatory nodules and fistulas scattered over the entire area of the right hard palate, an incisional biopsy was performed in order to rule out malignancy. The histopathological exam identified fragments of cyst wall lined with stratified squamous epithelium with parakeratosis and a mixed inflammatory infiltrate, along with colonies of *Actinomyces* in the adherent detritus of the fragments. As a result, the patient was prescribed antibiotic treatment with Clindamycin for a duration of 3 weeks. The reevaluation performed after the treatment revealed extensive granulation in the right upper dental arch, which is why a CT scan was recommended. The contrast-enhanced cranio-facial CT examination revealed the absence of bone substance in the right paramedian maxilla, with involvement of the dental alveoli (Figure 1A), the

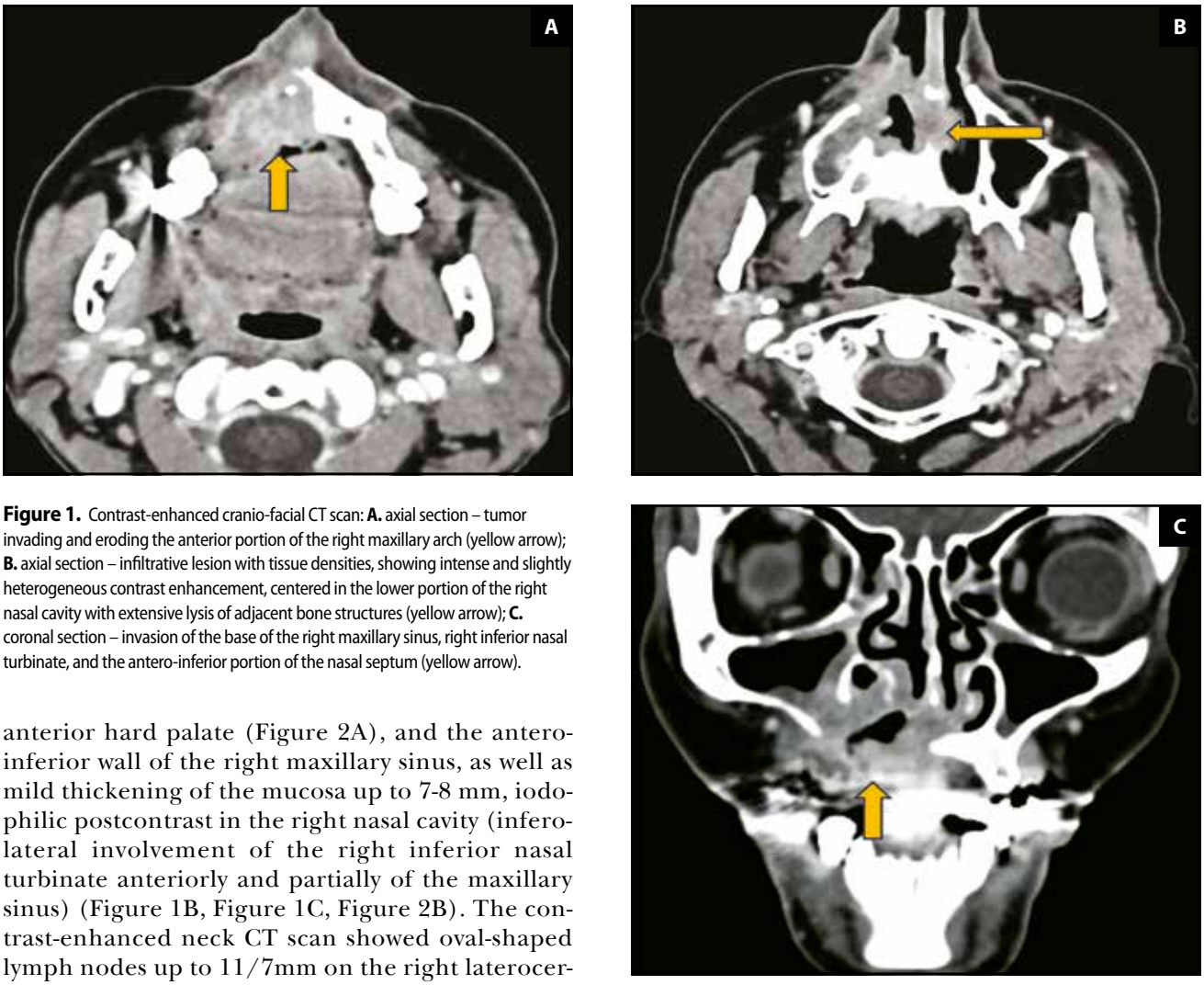

Figure 1. Contrast-enhanced cranio-facial CT scan: **A.** axial section – tumor invading and eroding the anterior portion of the right maxillary arch (yellow arrow); **B.** axial section – infiltrative lesion with tissue densities, showing intense and slightly heterogeneous contrast enhancement, centered in the lower portion of the right nasal cavity with extensive lysis of adjacent bone structures (yellow arrow); **C.** coronal section – invasion of the base of the right maxillary sinus, right inferior nasal turbinate, and the antero-inferior portion of the nasal septum (yellow arrow).

anterior hard palate (Figure 2A), and the antero-inferior wall of the right maxillary sinus, as well as mild thickening of the mucosa up to 7-8 mm, iodophilic postcontrast in the right nasal cavity (infero-lateral involvement of the right inferior nasal turbinate anteriorly and partially of the maxillary sinus) (Figure 1B, Figure 1C, Figure 2B). The contrast-enhanced neck CT scan showed oval-shaped lymph nodes up to 11/7mm on the right laterocervical level, in levels Ib and IIa.

A re-biopsy was performed by the oral and maxillofacial surgeon through an endobuccal approach of the tumor mass, and the histopathological examination described reactive mucosa with a marked, non-specific chronic inflammatory process and intraepithelial abscesses. A request for a second opinion and an additional immunohistochemical study was made to a pathologist from a higher-level competency medical center. The morphological aspect raises the issue of differential diagnosis between pseudoepitheliomatous squamous hyperplasia and verrucous squamous carcinoma. The immunohistochemical staining revealed positive Ki67 in the parabasal layers, P53 focal positivity, variable in the basal layers, focal p16 positivity, positive CkAE1/AE3 at the surface epithelial level and identified the presence of foci of discohesive growth at the level of the conjunctival stroma, raising suspicion of a stromal invasion focus.

In the course of the disease, the patient complained of local pain and fetid halitosis, the local evolution being stationary. The attending physician decided to refer the patient to our ENT Department for re-biopsy through endonasal approach.

The local ENT examination and exploratory nasal endoscopy revealed swelling of the upper lip on the right side, fetid halitosis, and an ulcerative-vegetative tumor mass at the level of the upper dental arch in the region of the right lateral incisor, right upper canine and premolars (otherwise absent) (Figure 3). The tumor invaded the hard palate and the floor of the right nasal cavity. At this level, an oronasal communication was also identified.

Under local anaesthesia and endoscopic control, biopsy fragments were collected from the floor of the nasal cavity. Postoperatively, antibiotic treatment (ciprofloxacin), anti-inflammatory and analgesic medications were administered. The evolution was surprisingly favourable under treatment, with remission of symptoms and a significant improvement in the clinical appearance, leading to the suspicion of maxillary osteomyelitis. Nasal secretion was collected from the oroantral communication for bacteriological and mycological

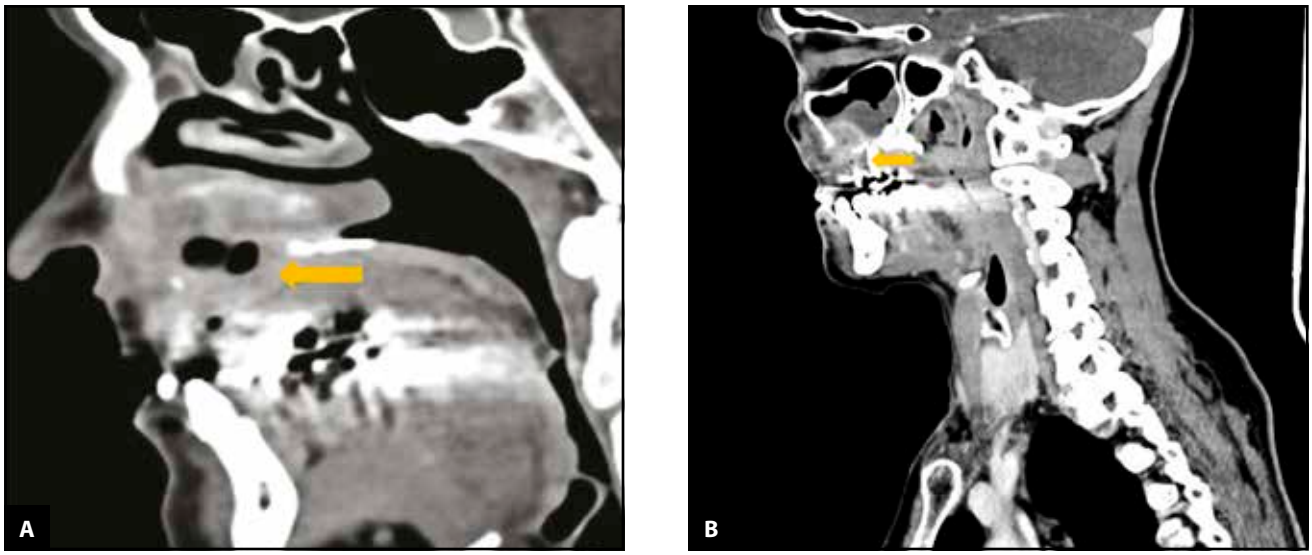

Figure 2. Contrast-enhanced cranio-facial CT scan **(A)** and neck CT scan **(B)**, right paramedian sagittal sections – tumor invading the hard palate with lysis in the anterior portion (yellow arrow).

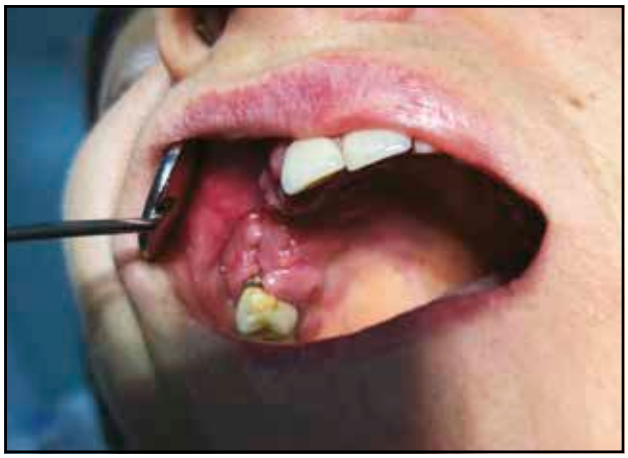

Figure 3. Ulcerative-vegetative tumor mass at the level of the upper dental arch, in the region of the right lateral incisor, right upper canine and premolars.

examination, and a nasal scraping was taken from the floor of the right nasal cavity for cytological examination. Methicillin-sensitive *Staphylococcus aureus* was identified in the nasal secretion. The cytological examination revealed a smear with acute inflammation, with no signs of malignancy. The patient was evaluated in the Infectious Diseases Department, and the infectious disease specialist recommended antibiotic treatment with Doxycycline for 6 weeks.

The histopathological examination, which at the patient's request was performed in the same high-level competence medical center where the first two biopsies were reread, described polypoid fragments of the sinus mucosa, some of them with chronic inflammation changes, others with squamous meta-

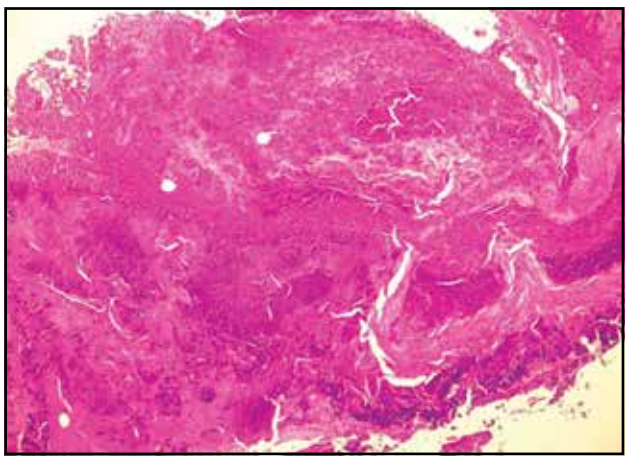

Figure 4. Microscopical image of histopathological examination, hematoxylin-eosin staining (x10) – fibrinoid-necrotic material.

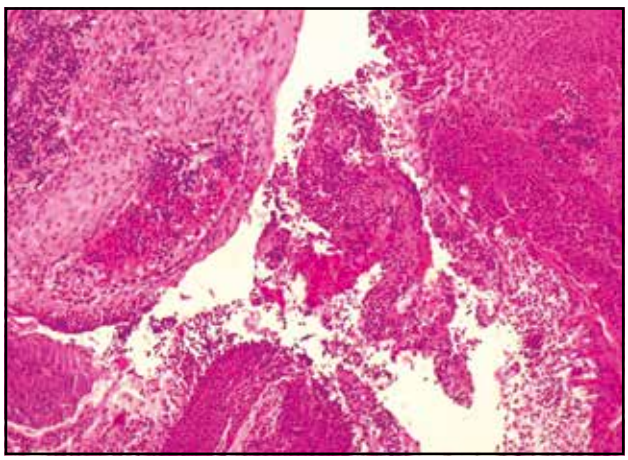

Figure 5. Histopathological examination, hematoxylin-eosin staining (x10) – stratified squamous epithelium with exocytosis of polymorphonuclear neutrophils, reactive atypia, with massive fibrin-leukocyte and hematic exudate on the surface.

plasia on the surface and multiple nuclear atypias in the deeper layers. Immunohistochemical staining revealed Ki67 positivity in the parabasal layers, P53 did not intensely stain the squamous cells, and CkAE1/AE3 identified groups of scattered cells originating from the base of the squamous epithelium but without desmoplastic reaction and apparently surrounded by the basement membrane in the PAS (Periodic acid-Schiff) staining. No definitive histological signs of malignancy were reported in this biopsy.

The radical excision of the tumor was not considered an option due to the lack of a histopathological diagnosis, as well as the tumor's location and extent, which would result in a postoperative defect and mutilation of the region. It was decided to fully reinterpret all biopsies performed in the Department of Anatomopathology of the Zalau County Emergency Hospital, with each biopsy's microscopic appearance, combined with the results of immunohistochemical examinations, supporting the diagnosis of pseudoepitheliomatous hyperplasia (Figure 4, Figure 5, Figure 6, Figure 7). The first biopsy sample showed colonies of *Actinomyces*.

Under antibiotic treatment, the case evolved favourably, with resolution of the symptoms and significant improvement in the clinical appearance. It was decided to discharge the patient with periodic follow-ups in the ENT, Oral and Maxillofacial Surgery, and Infectious Diseases Departments.

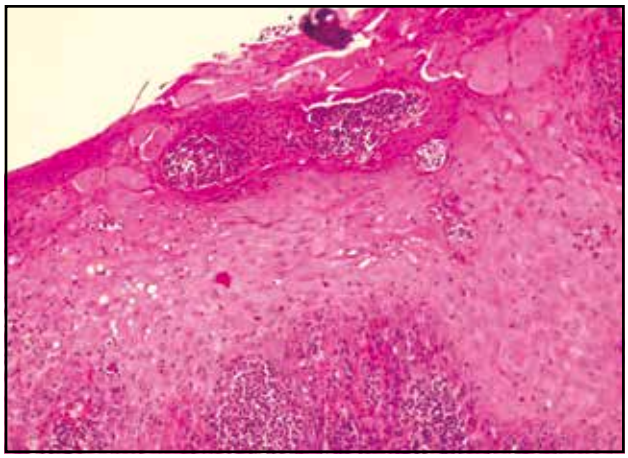

Figure 6. Histopathological examination, hematoxylin-eosin staining (x20) – hyperparakeratosis with intraepithelial abscesses.

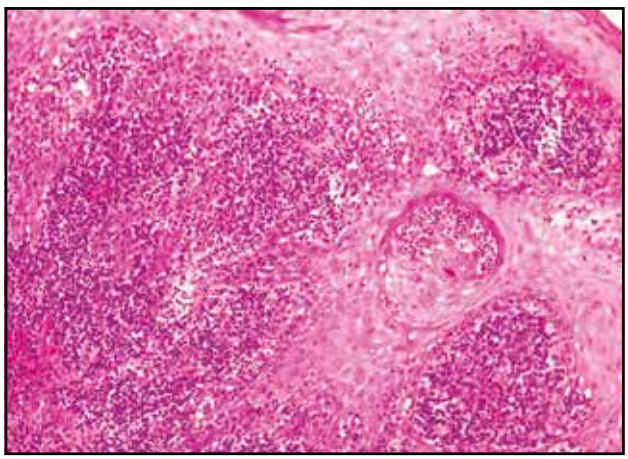

Figure 7. Histopathological examination, hematoxylin-eosin staining (x20) – stratified squamous epithelium with elongated, irregular epithelial ridges, anastomosed at the stroma level, which shows abundant polymorphous inflammatory infiltrate.

DISCUSSIONS

The particularities of the presented case highlight the complexity of diagnosing and managing maxillary osteomyelitis caused by *Actinomyces* sp. infection, a rare but significant condition in clinical practice.

The clinical presentation of the patient, characterized by an ulcerative-vegetative tumor with local invasion and oroantral communication, mimicked a malignant tumor lesion. This pseudotumoral manifestation of osteomyelitis is rare but documented in the literature, often being confused with neoplasms, especially in the absence of obvious infectious signs. The initial inconclusive biopsies and nonspecific histopathological findings contributed to the delay in reaching the correct diagnosis.

The patient's previous dental work may have been a predisposing factor in the development of maxillary osteomyelitis in this case. The literature highlights the frequent association of maxillary osteomyelitis with predisposing factors such as untreated dental infections, iatrogenic trauma or dental work[10,26]. An incorrectly performed prosthetic restoration, especially in the absence of adequate endodontic treatments, can serve as a persistent infection focus. According to specialized studies, fixed prosthetic restorations should only be performed after the complete elimination of periapical infections, following a thorough evaluation of the periodontal and endodontic status.

Actinomycotic maxillary osteomyelitis is a rare entity, representing a significant diagnostic challenge due to its nonspecific clinical manifestations and similarity to malignant lesions. The identification of *Actinomyces* colonies in the first biopsy suggests a rare chronic infection that predominantly affects the mandible but can also involve the maxilla. Actinomycosis is characterized by granulomatous lesions, abscess and fistula formation, and can mimic neoplastic lesions. The diagnosis is often difficult, requiring clinical, imaging and histopath-

ological correlation. In this case, the presence of pseudoepitheliomatous hyperplasia (PEH) further complicated the differential diagnosis. PEH is a benign epithelial proliferation that can mimic invasive squamous cell carcinoma, requiring careful clinicopathological correlation to avoid overdiagnosis and unjustified aggressive treatments[27].

Computed tomography (CT) imaging revealed bone lesions with poorly defined margins and extension into the soft tissue, features suggestive of actinomycosis[28]. The presence of fistulas and fluid collections, along with thickening of the sinus mucosa, supports the diagnosis of actinomycotic osteomyelitis.

The histopathological examination was essential in establishing the diagnosis, identifying *Actinomyces* colonies. In the context of chronic infections, such as actinomycosis, PEH can emerge as a reactive response of the epithelium to persistent inflammation. Histologically, PEH is characterized by elongation and branching of epithelial ridges, without significant nuclear atypia, in contrast to squamous cell carcinoma, which shows cellular atypia and stromal invasion[24].

The favourable evolution under antibiotic treatment with doxycycline and ciprofloxacin supports the diagnosis of actinomycotic osteomyelitis. Actinomycosis responds well to prolonged antibiotic therapies, and extensive surgical interventions can be avoided if the diagnosis is established correctly and promptly. The treatment of actinomycotic osteomyelitis involves prolonged antibiotic administration, especially penicillin G, for a duration of 6 to 12 months. In cases with penicillin allergy, alternatives such as doxycycline may be used. In the presented case, the administration of doxycycline for 6 weeks resulted in significant improvement in both the symptoms and the clinical appearance.

Surgical interventions are reserved for cases with large abscesses, chronic fistulas, or extensive bone necrosis. In this case, radical excision was not considered appropriate due to the location and extent of the lesion, as well as the risk of mutilation.

CONCLUSIONS

The case highlights the importance of a multidisciplinary approach in the diagnosis and treatment of actinomycotic maxillary osteomyelitis. Close collaboration between ENT specialists, oral and maxillofacial surgeons, pathologists and infectious disease specialists is essential for establishing an accurate diagnosis and initiating appropriate treatment. A multidisciplinary approach is imperative in managing complex cases with atypical manifestations.

Additionally, it is crucial to differentiate between PEH and malignant lesions to avoid excessive or inadequate treatments. Careful monitoring and regular follow-up of the patient are recommended to prevent recurrences and ensure a favourable clinical outcome.

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