Clinicopathologic Study of Osteomyelitis from Oral and Maxillofacial Region

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Recently, osteomyelitis from oral and maxillofacial region which is an acute or chronic inflammatory process in medullary spaces or cortical surfaces of bone is uncommon in Korea. And the clinicopathologic study of osteomyelitis in Korea has been rarely reported. The purpose of this study were to examine the clinicopathologic analysis of osteomyelitis patients and to apply its results for treatment, Retrospective analysis of 103 cases of osteomyelitis patients treated in the Department of Oral and Maxillofacial Surgery at DKUDH from 1991 to 2000. There was a male predominance with a 2.3:1 ratio. The mean age of onset of disease was almost the same in cases of acute and chronic osteomyelitis: 29.4 years(range 1-81 years). Swelling, pain, pus discharge, and sequestration were main characteristic features of this disease entity. Acute chronic osteomyelitis of the jaws is caused mostly by a bacterial focus(odontogenic disease, periapical lesion, pericoronitis, periodontal disease, postextraction wounds, and infected fractures). It suggested that acute and chronic osteomyelitis could be basically the same disease separated by the arbitrary time limit of 1 month after onset of the disease by a true bacterial infection. And these results could play an role in the diagnosis and treatment of osteomyelitis of the jaws.

Key words: Osteomyelitis, Clinicopathologic study

Ⅰ. Introduction

The word “osteomyelitis” originates from the ancient Greek words osteon (bone) and muelinos (marrow) and means infection of medullary portion of the bone. In common medical literature, it usually encompasses the cortical bone and periosteum as well. It can therefore be considered as an inflammatory condition of the bone, beginning in the medullary cavity and havasian systems and extending to involve the periosteum of the affected area.

The infection becomes established in calcified portion of the bone when pus and edema in the medullary cavity and beneath the periosteum compromises or obstructs the local blood supply. Following ischemia, the infected bone becomes necrotic and leads to sequester formation, which is considered a classical sign of osteomyelitis

The prevalence, clinical course, and management of osteomyelitis of the jaw bones have changed profoundly over the past 50 years. This is due to mainly one factor: the introduction of antibiotic therapy, specifically penicillin. After the introduction of antibiotics, acute phases were often concealed by these antimicrobial drugs without fully eliminating the infection. Subacute or chronic forms of osteomyelitis have therefore become more prominent.
Osteomyelitis of the jaws is still a fairly common disease in maxillofacial region, despite the introduction of antibiotics and the improvement of dental and medical care. Different terminologies and classification systems are used based on a variety of features such as clinical course, pathological-anatomical or radiological features, etiology, and pathogenesis. Because of these classification systems, it has occurred throughout the literature, leading to confusion and thereby hindering comparative studies, an overview of the most commonly used terms and classification systems in osteomyelitis of the jaws was given in this report.

Because of its unique feature bearing teeth and hence connecting to the oral cavity with the periodontal membrane, osteomyelitis of the jaws differs in several important aspects from osteomyelitis of long bones. The specific local immunological and microbiological aspects determine a major factor in the etiology and pathogenesis of this disease, and hence also have a direct impact on its treatment. This is reflected by the longstanding recognition of osteomyelitis of jaw bones as a clinical entity. Therefore, a wide variety of classifications, specifically for the jaw bones, have been established by several authors in the medical literature, which are based on clinical course, pathological-anatomical and/or radiological features, etiology, and pathogenesis.

The purpose of this study was to examine the clinicopathologic analysis of 103 cases of osteomyelitis patients and to apply its results for treatment.

### II. Material and Methods

Retrospective analysis of the osteomyelitis patients treated in the Department of Oral and Maxillofacial Surgery in DKUDH from 1991 to 2000. Classification system was primarily based on the clinical course and appearance of the disease as well as on radiographic features. Subclassification was based on etiology and histopathologic features of the disease. And also demographic analysis in this study was done.

### III. Results

Analysis of the osteomyelitis patients treated in the Department of Oral and Maxillofacial Surgery at DKUDH showed a clear predominance of cases diagnosed as secondary chronic osteomyelitis at the time of presentation, whereas cases of acute osteomyelitis were significantly less often diagnosed (Table 1).

**Table 1. Classification of Osteomyelitis in this study**

<table>
<thead>
<tr>
<th>Type</th>
<th>Case</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute</td>
<td>18</td>
<td>17.5</td>
</tr>
<tr>
<td>Chronic</td>
<td>80</td>
<td>77.7</td>
</tr>
<tr>
<td>Unidentified</td>
<td>5</td>
<td>4.8</td>
</tr>
<tr>
<td>Total</td>
<td>103</td>
<td></td>
</tr>
</tbody>
</table>

**Table 2. Distribution of Age and Sex of Osteomyelitis in this study**

<table>
<thead>
<tr>
<th>Age/Sex</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-10</td>
<td>4</td>
<td>3</td>
<td>7</td>
<td>6.8</td>
</tr>
<tr>
<td>11-20</td>
<td>10</td>
<td>6</td>
<td>16</td>
<td>15.5</td>
</tr>
<tr>
<td>21-30</td>
<td>18</td>
<td>12</td>
<td>30</td>
<td>29.1</td>
</tr>
<tr>
<td>31-40</td>
<td>19</td>
<td>2</td>
<td>21</td>
<td>20.4</td>
</tr>
<tr>
<td>41-50</td>
<td>10</td>
<td>5</td>
<td>15</td>
<td>14.6</td>
</tr>
<tr>
<td>51-60</td>
<td>2</td>
<td>4</td>
<td>6</td>
<td>5.8</td>
</tr>
<tr>
<td>61-70</td>
<td>5</td>
<td>2</td>
<td>7</td>
<td>6.8</td>
</tr>
<tr>
<td>Above 71</td>
<td>1</td>
<td>0</td>
<td>1</td>
<td>1.0</td>
</tr>
<tr>
<td>Total</td>
<td>69</td>
<td>34</td>
<td>103</td>
<td>100</td>
</tr>
</tbody>
</table>

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**Fig. 1.** Acute Osteomyelitis. Ill-defined area of adioclucency showing central radiopaque mass. **Fig. 2.** Acute Osteomyelitis. Nonvital bone with loss of the osteocytes from the lacunae. **Fig. 3.** Chronic Osteomyelitis. Ill-defined area of radiolucency of the right body of Mn. **Fig. 4.** Chronic Osteomyelitis. Sclerosed trabecular pattern in fibrous connective tissue with chronic inflammatory cell infiltration. **Fig. 5.** Chronic Diffuse Osteomyelitis. Diffuse area of increased radiodensity of Mn. **Fig. 6.** Chronic Diffuse Osteomyelitis. Sclerosed trabecular pattern with little bone marrow in less chronic inflammatory cell infiltration. **Fig. 7.** Chronic Focal Osteomyelitis. Focal area of increased radiodensity Surrounding the apex of he nonvulal Mn 1st molar. **Fig. 8.** Chronic Focal Osteomyelitis. Sclerosed trabecular pattern with little bone marrow and loss of the osteocytes from the lacunae. **Fig. 9.** Proliferative Periostitis. Radiopaque laminations of cortical bone parallel the cortex adjacent to inflammatory process of Lt. Mn molar area. **Fig. 10.** Proliferative Periostitis. Cellular and reactive vital bone with individual trabeculae oriented perpendicular to the surface.
Acute chronic osteomyelitis may affect all ages and both sexes. In our retrospective analysis of 103 cases of acute and secondary chronic osteomyelitis there was a male predominance with a 2.3:1 ratio (Table 2). The mean age of onset of disease in our studied cases was almost the same in cases of acute chronic osteomyelitis: 29.4 years (range 1-81 years) (Table 2).

Most frequent sources were odontogenic foci (78.7%), periapical lesions (30%), extraction wounds (22%), pericoronitis (17.5), periodontal diseases (11%), infected fractures (6%), postoperative infection (1.9%), osteoradionecrosis (1.0%), and unidentified (12.6%). Swelling (55.4%), pain (19.4%), trismus (11.7%), pus discharge (8.7%), and sequestration were typical clinical findings of this disease (Table 3).

Acute osteomyelitis showed ill-defined area of radiolucency showing central radiopaque mass (Fig. 1) and nonvital bone with loss of the osteocytes from the lacunae (Fig. 2), while chronic osteomyelitis showed ill-defined area of radiolucency of the right body of Mn (Fig. 3) and sclerosed trabecular pattern in fibrous connective tissue with chronic inflammatory cell infiltration (Fig. 4). Chronic diffuse osteomyelitis, diffuse area of increased radiodensity of Mn (Fig. 5) and sclerosed trabecular pattern with little bone marrow in less chronic inflammatory cell infiltration (Fig. 6). Chronic focal osteomyelitis, showed focal area of increased radiodensity surrounding the apex of he nonvital Mn 1st molar (Fig. 7) and sclerosed trabecular pattern with little bone marrow and loss of the osteocytes from the lacunae (Fig. 8). Proliferative periostitis showed radiopaque laminations of cortical bone parallel the cortex adjacent to inflammatory process of Lt. Mn molar area (Fig. 9) and cellular and reactive vital bone with individual trabeculae oriented perpendicular to the surface (Fig. 10).

### IV. Discussion

Classification system was primarily based on the clinical course and appearance of the disease as well as on radiographic features in this study. Subclassification was based on etiology and histopathologic features of the disease. In this study, retrospective analysis of the osteomyelitis patients using the abovementioned major classification groups showed mainly two different types of...
osteomyelitis and a clear predominance of cases diagnosed as secondary chronic osteomyelitis at the time of presentation, whereas cases of acute osteomyelitis and chronic osteomyelitis were significantly less often diagnosed. Although acute forms of osteomyelitis were seen only rarely these days, most authors in common medical literature still describe this form as an entity of its own. Mercuri (5) and Marx (6) arbitrarily defined the time element as being 1 month after onset of symptoms. Many authors agree that chronic osteomyelitis involving the jaw bone may be divided in two major categories: suppurative and nonsuppurative forms (3,7).

To our knowledge, Mercuri (5) and Marx (6) were the first and only authors to define the duration for an acute osteomyelitis until it should be considered as chronic. They set an arbitrary time limit of 4 weeks after onset of disease. Pathological-anatomical onset of osteomyelitis corresponds to deep bacterial invasion into the medullar and cortical bone. After the period of 4 weeks, a persisting bone infection should be considered as secondary chronic osteomyelitis. Although the onset of the disease is a debatable point in time, it is still a simple and clear classification criterion and therefore of practical use for the clinician. This same definition was later used by several other authors (8,9). Because of its simplicity and clarity, this criterion was also used in this classification to differentiate acute osteomyelitis from chronic osteomyelitis cases. Acute and chronic osteomyelitis of the mandible affects most commonly the body of the mandible, followed by the symphysis, angle, ascending ramus, and condyle (10,11).

Retrospective analysis of 251 cases of acute and secondary chronic osteomyelitis there was a male predominance with a 2:1 ratio (11). Koobush et al (19) described a male to female ratio of 3:1 in a survey of 35 patients. An equal gender distribution was noted in a larger African patient population (12). The mean age of onset of disease in our studied cases was almost the same in cases of acute and secondary chronic osteomyelitis, which were comparable with those described by previous investigators (9-13).

It was by far the most common osteomyelitis type, which was usually caused by bacterial invasion from a contagious focus, odontogenic foci (78.7%), periapical lesions (30%), extraction wounds (22%), pericoronitis (17.5%), periodontal diseases (11%), and infected fractures (6%). Swelling (55.4%), pain (19.4%), trismus (11.7%), pus discharge (8.7%), and sequestration were typical clinical findings of this disease. Cases with an acute suppurative clinical course usually showed impressive signs of inflammation and intense pain mostly described by a deep sensation within the bone by the patient’s history. Local swelling and edema due to abscess formation also was substantial causing trismus and limitation of jaw function. A general malaise caused by high intermittent fever with 39-40°C was often accompanied by regional lymphadenopathy. In some instances paresthesia or anesthesia of the lower lip was described (Vincent’s symptom), indicating involvement of the inferior alveolar nerve. Pus around the gingival sulcus and through mucosal and, possibly cutaneous, fistulas, and a fetid oral odor caused by anaerobic pyogenic bacteria often were present. Increased mobile teeth even led to malocclusion and showed decreased or loss of sensitivity. It was thought that sequester formation and appositional neosteogenesis might be limited due to the short period since establishment of deep bone infection, which was the definition of acute osteomyelitis.

Most symptoms, such as pain and swelling, were usually less extensive in the chronic than in the acute stage. The deep and intense pain was replaced by a more dull pain. Painful swelling was subsided by a harder palpable tenderness caused by periosteal reaction. Sequester and fistula formation were somewhat more predominant in
advanced stages, and were regarded as classical signs of chronic osteomyelitis. The noted fetid odor was less frequent in patients. A disturbed occlusion sometimes was noted. Clinically and radiographically, a broad spectrum ranging from an aggressive osteolytic putrefactive phase to a dry osteosclerotic phase may be observed\[^{14}\].

One of the most confusing terms among the currently used osteomyelitis nomenclature is "diffuse sclerosing osteomyelitis" (DSO), and diffuse types\[^{3,15}\]. The focal type (periapical osteitis/osteomyelitis or condensing osteitis) is a rather common condition with a pathognomonic, well-circumscribed radiopaque mass of sclerotic bone surrounding the apex of the root\[^{16,17}\]. Since the infection in these cases is limited to the apex of the root with the absence of deep bone invasion, sufficient endodontic treatment with or without apex surgery or extraction of the affected tooth usually leads to regression of these lesions or residual sclerosis may remain as a bone scar. True diffuse sclerosing osteomyelitis, however which is a rare disease of unknown etiology that can cause major diagnostic and therapeutic problems\[^{1,2,16,18}\] is 8 cases in this study. The absence of pus, fistula, and sequestration are characteristic. The disease shows an insidious onset, lacking an acute state.

A further pathological disease entity such as florid osseous dysplasia (FOD) has been confused with diffuse sclerosing osteomyelitis, since it may mimic DSO radiographically by presenting sclerosing opaque and dense masses\[^{19,20}\]. These masses are, however, confined to the alveolar process of either or both jaws in cases of FOD, FOD which is mostly observed in black women and in many cases lacks clinical symptoms has no data in this study. As with all pathologies of the bone which compromise local blood flow and host resistance, FOD makes the jaw more susceptible to secondary infection.

Strictly periostitis ossificans or ossifying periostitis is, like diffuse sclerosing osteomyelitis, a descriptive term for a condition that may be caused by several similar entities\[^{2,21}\]. It is merely a periosteal inflammatory reaction to many nonspecific stimuli, leading to the formation of an immature type of new bone outside the normal cortical layer. Radiographical bony enlargement was main clinical feature and occurred in 6 cases of children in this study.

The clinical term "dry socket" or alveolar osteitis may also be regarded as a localized form of infection generally used in the medical and dental literature to describe an absence of invasion into the bone\[^{1,2}\]. It should therefore not be regarded as a form of osteomyelitis\[^{6}\]. In alveolar osteitis the commonly advocated theory suggests a clot breakdown due to the release of fibrinolysins either from microorganisms or trauma. In both situations the bacteria remain on the surface of the exposed bone, and an actual invasion does not occur. Although not considered a true infection, alveolar osteitis may lead to acute or chronic osteomyelitis once the bacterial invasion into the medullar and cortical bone has occurred and a deep bone infection has been established.

Further subclassification of these major osteomyelitis groups have to be based on presumed etiology and pathogenesis of disease. These criteria will be helpful in determining the necessary therapeutic strategies which may differ somewhat among the subgroups. The nature of these subgroups will be outlined in more detail later.

V. References


