Marc M. Baltensperger • Gerold K. H. Eyrich (Eds.)

Osteomyelitis of the Jaws

Marc M. Baltensperger
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Foreword by Robert E. Marx
With 537 Figures and 47 Tables

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Aspects of the Human Face
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mmbaltartproject.com

Dedication

This book is dedicated to my wife Chrisellee, my son Glen and Kevin. Without their continuous love, support and understanding, this book would not have been possible.

MARC M. BALTENSPERGER

Everything should be made as simple as possible,
but not one bit simpler.

ALBERT EINSTEIN (1879–1955)

Classification and re-classification of a subject over and over again with reference to other differentiating factors, makes one able to achieve a fresh approach each time.

VIMANASTHAANA IN CHARAKA SAMITHA 6:4
ANCIENT AYURVEDIC SUTRA, 7 B.C.

Medicine as a science is continuously changing. Research and clinical experience increase our knowledge, in particular regarding treatment and medical therapy. Where in this work a dosage or an application is mentioned, the reader may trust that the authors, editors, and publisher have gone to great lengths to ensure that this information corresponds exactly to **current knowledge at the time of publication**.

However, each user is required to read the instruction leaflet for the medication to check for himself or herself if the recommended dosage or the contraindications given therein differ from the information given in this book. This applies especially for medication that is rarely used or that has only recently come on the market and for those medications whose use has been restricted by Federal Health authorities.

MARC M. BALTENSPERGER
GEROLD K. H. EYRICH

Special Thanks

This textbook, in its present form, would not have been possible without the support and input of several people who have accompanied us on this journey. Some of them have directly contributed to this book as coauthors; others have given us valuable ideas and thoughts, which we have integrated in this book. Our special thanks go to these friends and colleagues.

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JOACHIM OBWEGESER

JÖRG SCHMUTZ

BERNHARD SCHUKNECHT

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HERMAN SAILER

REINHARD ZBINDEN

WERNER ZIMMERLI

Foreword

Osteomyelitis of the jaws is a disease that has affected mankind since prehistory. In fact, the famous 1.6 million-year-old fossil find of "Turkana Boy" documents this very well. As a 12-year-old prehuman hominid (homo erectus) his nearly complete skeleton clearly showed an osteomyelitis arising from an odontogenic infection around one of his first molar teeth (Fig.1). Paleontologists even conjecture that it was the most likely cause of his premature death. Today, medical and dental specialists continue to treat osteomyelitis of various types with the recognition that osteomyelitis of the jaws differs significantly from osteomyelitis of the long bones and at other skeletal sites. These differences are due to a different group of pathogens, the presence of teeth, a different blood vessel density, an oral environment, a thin mucosa as opposed to skin, one jaw that is mobile and the other that is fixed, the more frequent presence of foreign bodies, and the commonality of head and neck radiotherapy. These differences are also reflected in the confusing array of terms used to describe the different forms of jaw osteomyelitis, e.g., chronic diffuse sclerosing osteomyelitis, Garré's osteo-

Fig. 1 Mandible of the "Turkana Boy". Picture courtesy of National Museums of Kenya, Palaeontology Department, Nairobi, Kenya

myelitis with Proliferative periostitis, periostitis ossificans, etc., as well as many entities that are not primarily infectious etiologies but develop a secondary osteomyelitis by virtue of exposed bone in the oral cavity such as osteoradionecrosis, osteopetrosis, and bisphosphonate-induced osteonecrosis.

In this book, M. Baltensperger and G. Eyrich begin with a straightforward classification and terminology system that is consistent with the scientific evidence of the different clinical forms of jaw osteomyelitis. They further coordinate the initiating factors, host local and systemic factors, and pathogenesis to the clinical presentation. In a logical manner, useful to the student, resident, and senior clinicians alike, a differential diagnostic methodology is offered, inclusive of clear descriptions of what the present imaging technology can and cannot offer. This is followed by separate chapters on the different microscopic pathologies seen with each type of osteomyelitis and the microbiology of the known pathogens. The next three chapters are focused on treatment. They logically begin with the principles of treatments including the selection of the most appropriate surgical procedure and its follow-up by the selection of the appropriate antibiotic, as well as its correct dosing and the role of adjunctive hyperbaric oxygen in selected cases. The next chapter openly discussed the uniqueness of osteomyelitis involving the temporomandibular joint and its treatment, a disease not often discussed in other texts. The final chapter underscores the practical value of this book. In an innovative manner the authors present 20 case reports of actual cases and how they were diagnosed and further managed. These cases encompass nearly the full spectrum of osteomyelitis of the jaws and support the application of the principles and techniques reviewed in the preceding chapter.

The reader should appreciate the flow of this book, from the history of osteomyelitis to its classification, diagnosis, pathology, imaging, and treatment. The reader should also appreciate that a complex disease, such as osteomyelitis of the jaws with numerous variations, can be simplified in approach while still retaining the comprehensiveness of its diagnosis and treatment. The student can use this book as a comprehensive textbook or on a chapter-by-chapter basis. The experienced clinician can use the book for general review or as a case review as he or she may be confronted with their own challenging case. Both groups will find the case reports to be the realistic element that brings everything together.

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Preface

Writing and editing a book naturally gives rise to questions regarding the deeper motivations for undertaking such work. Some topics seem to accompany one during one's daily work life: They even seem to be sitting in the waiting area when you enter the office in the morning. When this happens, we start to become more obsessed with the attendant difficulties of the discipline. Finding oneself wrong, or perhaps ill-equipped with the tools and knowledge available, can often deepen the quest for solutions. For us, osteomyelitis of the jaws has become a complex matter that has not been resolved by current research, but we have certainly made significant progress.

Everyone can visualize the faces and anxiety of the parents of a 12-year-old boy being confronted with the possible diagnosis of a malignant disease. In one case, soon after we had assuaged the initial fear of a life-threatening disease away, we found ourselves confronted with primary chronic osteomyelitis. Fear was clearly seen in the parents' faces when we explained that we did not really have a reliable treatment option, and that neither did we know the cause or prognosis of the disease. Consequently, the disorder has become a "face," insofar as not only the disorder but also the patient "accompanies" you as you try to determine the treatment. This uncertainty is even more astonishing since we encounter osteomyelitis frequently in our daily practice. Many people may assume that osteomyelitis has been thoroughly researched and resolved, especially inexperienced residents. When looking for signposts, guidelines, or definitions, however, the road becomes ambiguous. For example, several terms have been used for the same conditions, and there are numerous classification systems, descriptions, and recommendations. The inevitable question is: Which road should one take, and which opinions should one follow?

Once a path has been chosen, one must constantly verify and justify courses of action and treatment options. This will help us to understand the treatment process and keep focused on the goal. Without engaging in this self-inquiry, however, one cannot make much progress.

Although we use a classification system in this book which is very familiar to us, we have tried to render it understandable to everyone. We have combined our knowledge and experience so that readers will not be prone to the pitfalls which have marked our journey. In retrospect, there have always been signposts along the road. Some of the questions have been solved and others have increased in complexity. Up to now, it appears that no comprehensive book on osteomyelitis of the jaws has been attempted. It is our deepest hope that this book will be a significant contribution that will help guide the reader in choosing the best roads and vehicles on this journey.

MARC BALTENSPERGER AND GEROLD EYRICH

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1 Introduction

Marc Baltensperger and Gerold Eyrich

Osteomyelitis of the jaws is still a very unique disease of the facial skeleton that represents a great challenge for the physician as well as the patient being treated, despite all recent advances in diagnosis and evolved treatment modalities. In the past decades the clinical appearance of osteomyelitis cases has changed dramatically. Not only has the average number of cases seen in a maxillofacial unit decreased, but also the clinical picture of the disease itself has changed significantly. Osteomyelitis of the jaws used to be an infectious disease with an often complicated course, involving multiple surgical interventions and sometimes leading to facial disfigurement as a result of loss of affected bone and teeth and the accompanying scarring. The outcome was usually all but certain; hence, prolonged treatment and frequent relapses have been associated with this disease in the past.

Since the second half of the twentieth century, however, there has been a dramatic reduction in the incidence of osteomyelitis cases involving the jaws and other bones of the skeleton (Hudson 1993). One major probable factor leading to this development is the introduction of antibiotics into the therapeutic armamentarium; however, other factors have also contributed, such as improved nutrition and better availability of medical and dental care, especially including advances in preventive dentistry and oral hygiene. Earlier diagnosis due to more sophisticated diagnostic imaging modalities has additionally improved the morbidity associated with this disease (Hudson 1993; Topazian 2002).

While the abovementioned factors are accounted for in most Western countries, this is still not the case for all of them. There are numerous countries with great deficiencies in their medical and social systems, unable to give adequate medical treatment to all people in need. In such an environment severe courses of osteomyelitis involving the jaws are still frequently observed. The statistics of medical institutions which deal with osteomyelitis of the jaws in these regions resemble those seen in Western maxillofacial units several decades ago (Adekeye 1976; Adekeye and Cornah 1985; Taher 1993).

Despite all the benefits associated with the advances in medicine and dentistry, the development of microorganisms resistant to commonly used antibiotics, the increased number of patients treated with steroids and other immunocompromising drugs, and the rising incidence of AIDS, diabetes, and other medically compromising conditions have led to new problems in the treatment of osteomyelitis of the jaws, leading again to an increase of cases refractory to standard treatments. Radiation therapy leading to osteoradionecrosis has also been a condition which, if super-infected, has contributed to a large number of complicated osteomyelitis cases in the past decades. Due to more localized and fractioned application of radiation, and the consequent prophylactic dental treatment of these patients, these sometimes jaw-mutilating courses of the disease have become less frequent.

Recently, an increasing number of patients treated with bisphosphonates have been noted to develop osteonecrosis of the jawbone. This condition, also known as osteochemonecrosis, presents a condition which favors the development of osteomyelitis. The widespread use of bisphosphonates foreshadows that the number of cases with this condition will even rise in the future; however, presently in most maxillofacial units the number of osteomyelitis cases of the jaws seen by the single physician has decreased over the past decades. This may lead to a lack of experience in managing this disease with its unique manifestations in the jaw.

The numerous reports in the literature on osteomyelitis of the jaws reflects the importance of this disease, especially in the maxillofacial specialty. Several authors have undertaken the task of describing the disease and classifying its various types. This rich diversity of literature has also led to some confusion and many inconsistencies. The various classification systems advocated over the years have resulted in a great variety in terminology and have made comparative studies on an evidence basis extremely difficult, if not impossible. Although classification systems are described in several textbooks and journals, only few authors have demonstrated a classification system on a substantial number of cases and, hence, one of practical use for the treating physician.

The foundation of this textbook lies in a large study which retrospectively analyzed the osteomyelitis cases treated in the past three decades at the Department of Cranio-Maxillofacial Surgery at the University Hospital in Zurich. In this study conducted by my coeditor, Gerold Eyrich, and myself, 290 well-documented cases of osteomyelitis of the jaws were included (Baltensperger 2003). It represents, to our knowledge, the largest examined patient group with this disease in the literature to date. The main purpose of this study was to classify these cases based on the classification system for osteomyelitis used in this unit. Throughout the study, meticulous work-up of all the patient data revealed the benefits as well as the drawbacks of the used classification system. In conclusion, some modifications compared with the commonly used classification systems have been made where we thought them to be beneficial. The definitions of certain categories were refined. Some terms have been abandoned. In the case of primary chronic osteomyelitis a new subclassification was proposed which seemed to be justified based on our patient data and other recent publications.

The advocated classification system for osteomyelitis of the jaws is the core of this book. It is referred to as the Zurich classification for osteomyelitis of the jaws. Most of the coauthors contributing to this book have already been involved in the above-mentioned study and/or have participated in other recent publications on this topic where the same classification was used; therefore, the proposed classification system advocated in this book is consistently applied in each chapter.

It was our purpose to cover every aspect of this disease from classification to diagnosis and treatment. This book consists of 11 additional chapters which are all intended to stand on their own, with separate tables of contents and references and a summary at the beginning. This allows the reader who is particularly interested in one aspect of the disease a quick overview of the topic; however, certain redundancies from chapter to chapter are deliberately taken in account.

Because of the predominant role of diagnostic imaging in the diagnosis and classification of osteomyelitis (see Table 2.7 in Chap. 2), an entire chapter is dedicated to this special topic, highlighting standard conventional radiographic imaging modalities as well as the latest technologies in use.

Other chapters focus on pathology and microbiology, which are necessary topics in understanding the nature of this complex disease. In the past, osteomyelitis of the jaws, in analogy to long bone infection, was believed to be primarily caused by *Staphylococcus aureus*, *Staphylococcus epidermidis*, or hemolytic *Streptococci*. The discovery of several anaerobic bacteria involved in jawbone infection has broadened our knowledge in understanding the microbiology of this disease and is outlined in detail.

Pathology has always been regarded as valuable in the diagnostic process of osteomyelitis, especially in distinguishing it from other diseases of the bone. The specific aspects of infections of the jawbone are discussed in detail.

The chapter which deals with therapy of osteomyelitis of the jaws is divided into surgical therapy, antibiotic therapy, and hyperbaric oxygen therapy, which are considered the major columns of osteomyelitis treatment to date.

Because of its unique location and rare incidence, osteomyelitis of the mandible affecting the temporomandibular joint is very demanding to treat and always represents a great challenge. We therefore considered a separate chapter for this issue to be justified.

The final chapter is designed as an atlas. Typical case reports as well as cases with a complex course of each osteomyelitis category are described and illustrated, rounding out the scope of this book.

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Osteomyelitis of the Jaws: Definition and Classification

2

Marc Baltensperger and Gerold Eyrich

Contents

2.1 Summary

Osteomyelitis of the jaws is still a fairly common disease in maxillofacial clinics and offices, despite the introduction of antibiotics and the improvement of dental and medical care. The literature on this disease is extensive. 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. A mixture of these classification systems 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 is given at the beginning of this chapter.

The Zurich classification system, as advocated in this textbook, is primarily based on the clinical course and appearance of the disease as well as on imaging studies. Subclassification is based on etiology and pathogenesis of the disease. Mainly three different types of osteomyelitis are distinguished: acute and secondary chronic osteomyelitis and primary chronic osteomyelitis. Acute and secondary chronic osteomyelitis are basically the same disease separated by the arbitrary time limit of 1 month after onset of the disease. They usually represent a true bacterial infection of the jawbone. Suppuration, fistula formation, and sequestration are characteristic features of this disease entity. Depending on the intensity of the infection and the host bone response, the clinical presentation and course may vary significantly. Acute and secondary chronic osteomyelitis of the jaws is caused mostly by a bacterial focus (odontogenic disease, pulpal and periodontal infection, extraction wounds, foreign bodies, and infected fractures).

Primary chronic osteomyelitis of the jaw is a rare, nonsuppurative, chronic inflammation of an unknown cause. Based on differences in age at presentation,

clinical appearance and course, as well as radiology and histology, the disease may be subclassified into early- and adult-onset primary chronic osteomyelitis. Cases with purely mandibular involvement are further distinguished from cases associated with extragnathic dermatoskeletal involvement such as in SAPHO syndrome or chronic recurrent multifocal osteomyelitis (CRMO).

2.2 Definition

The word "osteomyelitis" originates from the ancient Greek words osteon (bone) and muelinos (marrow) and means infection of medullary portion of the bone. Common medical literature extends the definition to an inflammation process of the entire bone including the cortex and the periosteum, recognizing that the pathological process is rarely confined to the endosteum. 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 medullar cavity and havarian 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 (Topazian 1994, 2002).

Although other etiological factors, such as traumatic injuries, radiation, and certain chemical substances, among others, may also produce inflammation of the medullar space, the term "osteomyelitis" is mostly used in the medical literature to describe a true infection of the bone induced by pyogenic microorganisms (Marx 1991).

2.3 History

The prevalence, clinical course, and management of osteomyelitis of the jawbones have changed profoundly over the past 50 years. This is due to mainly one factor: the introduction of antibiotic therapy, specifically penicillin. The integration of antibiotics into the therapeutic armamentarium has led to a complete renaissance in the treatment of most infectious diseases, including osteomyelitis (Hudson 1993). Further factors, such as sophistication in medical and dental science as well as the widespread availability for adequate treatment, have additionally led to improvement in the management of this disease. Modern diagnostic imaging allows much earlier treatment of bone infections at a more localized stage.

In the preantibiotic era, the classical presentation of jawbone osteomyelitis was an acute onset, usually followed by a later transition to a secondary chronic process (Wassmund 1935; Axhausen 1934). Massive clinical symptoms with widespread bone necroses, neoosteogenesis, large sequester formation, and intra- and extraoral fistula formation were common presentations, sometimes leading to significant facial disfigurement (Fig. 2.1).

After the introduction of antibiotics, acute phases were often concealed by these antimicrobial drugs without fully eliminating the infection. Subacute or chronic

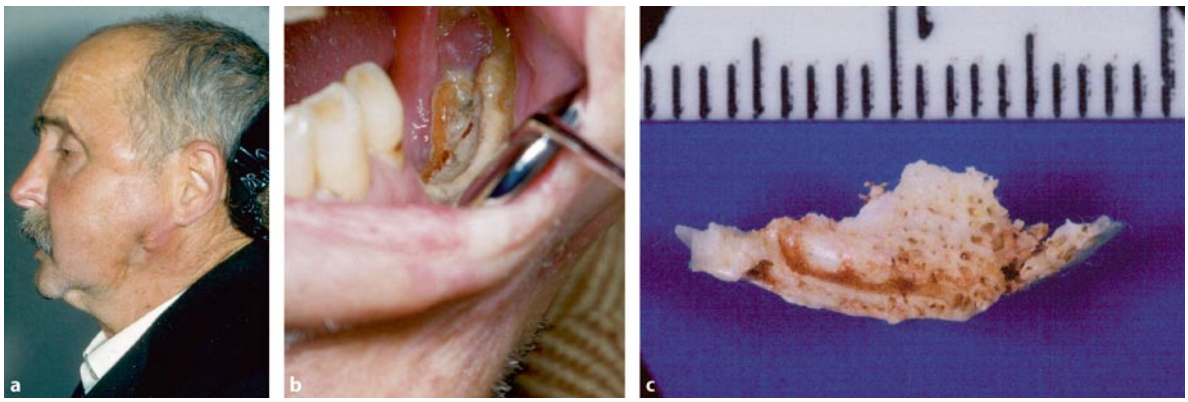

Fig. 2.1a–c Elder case of advanced secondary chronic osteomyelitis of the left mandible. The massive affection of the left mandible demonstrates extraoral fistula and scar formation (**a**). Intraoral view of the same patient with large exposure of infected bone and sequestra (**b**). Large sequester collected from surgery (**c**) (Courtesy of N. Hardt)

forms of osteomyelitis have therefore become more prominent, lacking an actual acute phase (Becker 1973; Bünger 1984).

2.4 Overview of Currently Used Classification Systems and Terminology

One of the first widely accepted staging systems for osteomyelitis in long bones was first described by Waldvogel and Medoff (1970) and Waldvogel et al. (1970a,b). The authors distinguished three categories of osteomyelitis: osteomyelitis from hematogenous spread; from a contagious focus; and due to vascular insufficiency. The classification is primarily based on etiology and pathogeneses of infection and does not readily lend itself to guiding therapeutic strategies such as surgery and antibiotic therapy. A more comprehensive classification proposed by Cieny et al. (1985) and Mader and Calhoun (2000) is based upon the anatomy of the bone infection and the physiology of the host. It divides the disease into four stages combining four anatomical disease types and three physiological host categories resulting in the description of 12 discrete clinical stages of osteomyelitis. Such a classification system, although it may be important in dealing with numerous sites of the skeletal system and allowing stratification of infection and the development of comprehensive treatment guidelines for each stage, is unnecessarily complex and impractical when dealing with infections of the jawbones.

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

Table 2.1 Classification systems described in the literature for osteomyelitis of the jaws

Reference	Classification	Classification criteria
Hudson JW Osteomyelitis of the jaws: a 50-year perspective. *J Oral Maxillofac Surg 1993 Dec; 51(12):1294-301*	I. Acute forms of osteomyelitis (suppurative or nonsuppurative) A. Contagious focus 1. Trauma 2. Surgery 3. Odontogenic Infection B. Progressive 1. Burns 2. Sinusitis 3. Vascular insufficiency C. Hematogenous(metastatic) 1. Developing skeleton (children) II. Chronic forms of osteomyelitis A. Recurrent multifocal 1. Developing skeleton (children) 2. Escalated osteogenic (activity < age 25 years) B. Garrè's 1. Unique proliferative subperiosteal reaction 2. Developing skeleton (children and young adults) C. Suppurative or nonsuppurative 1. Inadequately treated forms 2. Systemically compromised forms 3. Refractory forms (chronic recurrent multifocal osteomyelitis CROM) D. Diffuse sclerosing 1. Fastidious microorganisms 2. Compromised host/pathogen interface	Classification based on clinical picture and radiology. The two major groups (acute and chronic osteomyelitis) are differentiated by the clinical course of the disease after onset, relative to surgical and antimicrobial therapy. The arbitrary time limit of 1 month is used to differentiate acute from chronic osteomyelitis (Marx 1991; Mercuri1991; Koorbusch1992).

Table 2.2 Classification systems described in the literature for osteomyelitis of the jaws

Reference	Classification	Classification criteria
Hudson JW Osteomyelitis of the jaws: a 50-year perspective. *J Oral Maxillofac Surg 1993 Dec;51(12):1294-301*	I. Hematogenous osteomyelitis II. Osteomyelitis secondary to a contiguous focus of infection III. Osteomyelitis associated with or without peripheral vascular disease	Classification based on pathogenesis. From Vibhagool 1993
Hudson JW Osteomyelitis of the jaws: a 50-year perspective. *J Oral Maxillofac Surg 1993 Dec;51(12):1294-301*	I. Anatomic Types Stage I: medullar osteomyelitis – involved medullar bone without cortical involvement; usually hematogenous Stage II: superficial osteomyelitis – less than 2 cm bony defect without cancellous bone Stage III: localized osteomyelitis – less than 2 cm bony defect on radiograph, defect does not appear to involve both cortices Stage IV: diffuse osteomyelitis – defect greater than 2 cm. Pathologic fracture, infection, nonunion II. Physiological class A host: normal host B host: systemic compromised host, local compromised host C host: treatment worse than disease	Dual classification based on pathological anatomy and pathophysiology From Vibhagool 1993 and Cierny 1985
Mittermayer CH Oralpathologie. *Schattauer, Stuttgart-New York 1976*	I. Acute suppurative osteomyelitis (rarefactional osteomyelitis) II. Chronic suppurative osteomyelitis (sclerosing osteomyelitis) III. Chronic focal sclerosing osteomyelitis (pseudo-paget, condensing osteomyelitis) IV. Chronic diffuse sclerosing osteomyelitis V. Chronic osteomyelitis with proliferative periostitis (Garrè's chronic nonsuppurative sclerosing osteitis, ossifying periostitis) VI. Specific osteomyelitis 1. Tuberculous osteomyelitis 2. Syphilitic osteomyelitis 3. Actinomycotic osteomyelitis	Classification based on clinical picture, radiology, pathology, and etiology

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; therefore, to extrapolate from long bone infections to disease of the jaws is only possible with limitations. This is reflected by the long-standing recognition of osteomyelitis of jawbones as a clinical entity, which differs in many important aspects from the one found in long bones; hence, a wide variety of classifications, specifically for the jawbones, have been established by several authors in the medical literature. Classifications proposed are based on different aspects such as clinical course, pathological–anatomical

Table 2.3 Classification systems described in the literature for osteomyelitis of the jaws

Reference	Classification	Classification criteria
Hjorting-Hansen E Decortication in treatment of osteomyelitis of the mandible. *Oral Surg Oral Med Oral Pathol 1970 May;29(5):641-55*	I. Acute/subacute osteomyelitis II. Secondary chronic osteomyelitis III. Primary chronic osteomyelitis	Classification based on clinical picture and radiology
Marx RE Chronic Osteomyelitis of the Jaws *Oral and Maxillofacial Surgery Clinics of North America, Vol 3, No 2, May 91, 367-81* Mercuri LG Acute Osteomyelitis of the Jaws *Oral and Maxillofacial Surgery Clinics of North America, Vol 3, No 2, May 91, 355-65*	I. Acute osteomyelitis 1. Associated with Hematogenous spread* 2. Associated with intrinsic bone pathology or peripheral vascular disease* 3. Associated with odontogenic and nonodontogenic local processes* II. Chronic osteomyelitis 1. Chronic recurrent multifocal osteomyelitis of children 2. Garrè's osteomyelitis 3. Chronic suppurative osteomyelitis – Foreign body related – Systemic disease related – Related to persistent or resistant organisms 4. True chronic diffuse sclerosing osteomyelitis	Classification based on clinical picture and radiology, etiology, and pathophysiology Classification of acute osteomyelitis by Mercuri, classification of chronic osteomyelitis by Marx. The arbitrary time limit of one month is used to differ acute from chronic osteomyelitis * From Waldvogel and Medoff 1970
Panders AK, Hadders HN Chronic sclerosing inflammations of the jaw. Osteomyelitis sicca (Garre), chronic sclerosing osteomyelitis with fine-meshed trabecular structure, and very dense sclerosing osteomyelitis. *Oral Surg Oral Med Oral Pathol 1970 Sep;30(3):396-412*	I. Primarily chronic jaw inflammation 1. Osteomyelitis sicca (synonymous osteomyelitis of Garrè, chronic sclerosing nonsuppurative osteomyelitis of Garrè, periostitis ossificans) 2. Chronic sclerosing osteomyelitis with fine-meshed trabecular structure 3. Local and more extensive very dense sclerosing osteomyelitis II. Secondary chronic jaw inflammation III. Chronic specific jaw inflammations – Tuberculosis – Syphilis – Lepra – Actinomycosis	Classification based on clinical picture and radiology Classification of chronic osteomyelitis forms only

and/or radiological features, etiology, and pathogenesis. A mixture of these classification systems has been used in many instances, leading to confusion and thereby hindering comparative studies and obscuring classification criteria. An overview of the most commonly cited classifications of jawbone osteomyelitis are listed in Tables 2.1–2.4.

Table 2.4 Classification systems described in the literature for osteomyelitis of the jaws

Reference	Classification	Classification criteria
Schelhorn P, Zenk W [*Clinics and therapy of the osteomyelitis of the lower jaw*]. *Stomatol DDR 1989 Oct;39(10):672-6*	I. Acute osteomyelitis II. Secondary chronic osteomyelitis III. Primary chronic osteomyelitis IV. Special forms – Osteomyelitis sicca (pseudo-paget Axhausen) – Chronic sclerosing osteomyelitis Garrè	Classification based on clinical picture
Topazian RG *Osteomyelitis of the Jaws. In Topizan RG, Goldberg MH (eds): Oral and Maxillofacial Infections.* *Philadelphia, WB Saunders 1994, Chapter 7, pp 251-88*	I. Suppurative osteomyelitis 1. Acute suppurative osteomyelitis 2. Chronic suppurative osteomyelitis – Primary chronic suppurative osteomyelitis – Secondary chronic suppurative osteomyelitis 3. Infantile osteomyelitis II. Nonsuppurative osteomyelitis 1. Chronic sclerosing osteomyelitis – Focal sclerosing osteomyelitis – Diffuse sclerosing osteomyelitis 2. Garrè's sclerosing osteomyelitis 3. Actinomycotic osteomyelitis 4. Radiation osteomyelitis and necrosis	Classification based on clinical picture, radiology, and etiology (specific forms such as syphilitic, tuberculous, brucellar, viral, chemical, *Escherichia coli* and *Salmonella* osteomyelitis not integrated in classification)
Bernier S, Clermont S, Maranda G, Turcotte JY *Osteomyelitis of the jaws.* *J Can Dent Assoc 1995 May;61(5):441-2, 445-8*	I. Suppurative osteomyelitis 1. Acute suppurative osteomyelitis 2. Chronic suppurative osteomyelitis II. Nonsuppurative osteomyelitis 1. Chronic focal sclerosing osteomyelitis 2. Chronic diffuse sclerosing osteomyelitis 3. Garrè's chronic sclerosing osteomyelitis (proliferative osteomyelitis) III. Osteoradionecrosis	Classification based on clinical picture and radiology
Wassmund M Lehrbuch der praktischen Chirurgie des Mundes und der Kiefer. *Meusser, Leipzig 1935*	I. Exudative osteitis II. Resorptive osteitis III. Productive osteitis IV. Acute necrotizing osteitis (osteomyelitis) V. Chronic osteomyelitis 1. Chronic course of an acute osteomyelitis 2. Occult osteomyelitis 3. Chronic necrotizing osteomyelitis with hypertrophy 4. Chronic exudative osteomyelitis 5. Productive osteomyelitis	Classification based on clinical picture and radiology (note that classification was developed before introduction of antibiotic therapy)

2.5 Currently Used Terms in Classification of Osteomyelitis of the Jaws

2.5.1 Acute/Subacute Osteomyelitis

Although acute forms of osteomyelitis are seen only rarely these days, most authors in common medical literature still describe this form as an entity of its own. Mercuri (1991) and Marx (1991) arbitrarily defined the time element as being 1 month after onset of symptoms. Endurance past this arbitrary set time limit is then considered as chronic osteomyelitis reflecting the inability of host defense mechanisms to eradicate the responsible pathogen. Many authors have agreed on this classification and have used the term likewise in their publications (Koorbusch et al. 1992; Hudson 1993; Schuknecht et al. 1997; Schuknecht and Valavanis 2003; Eyrich et al. 1999; Baltensperger et al. 2004).

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 (subacute) symptoms. In some instances, subacute osteomyelitis is referred to as a transitional stage within the time frame of acute osteomyelitis and corresponds to the third and fourth week after onset of symptoms (Schuknecht et al. 1997; Schuknecht and Valavanis 2003).

2.5.2 Chronic Osteomyelitis

The classification of chronic osteomyelitis is incoherent and confusing. Different disease processes have been described by this one term in some instances, whereas several terms have been designated for lesions that represent the same entity in other instances (Groot et al. 1996; Eyrich et al. 1999).

Many authors agree that chronic osteomyelitis involving the jawbone may be divided in two major categories: suppurative and nonsuppurative forms (Mittermayer 1976; Hudson 1993; Topazian 1994, 2002; Bernier et al. 1995).

2.5.3 Chronic Suppurative Osteomyelitis: Secondary Chronic Osteomyelitis

Chronic suppurative osteomyelitis is an often preferred term in Anglo-American texts (Marx 1991; Bernier et al. 1995; Topazian 1994, 2002) and can mostly be used interchangeably with the term "secondary chronic osteomyelitis," which is predominantly used in literature from continental Europe (Hjorting-Hansen 1970; Panders and Hadders 1970; Schelhorn and Zenk 1989). It is by far the most common osteomyelitis type, which is usually caused by bacterial invasion from a contagious focus. Most frequent sources are odontogenic foci, periodontal diseases and pulpal infections, extraction wounds, and infected fractures. Pus, fistula, and sequestration are typical clinical findings of this disease. Clinically and radiographically, a broad spectrum ranging from an aggressive osteolytic putrefactive phase to a dry osteosclerotic phase may be observed (Eyrich et al. 1999).

2.5.4 Chronic Non-suppurative Osteomyelitis

The term "nonsuppurative osteomyelitis" describes a more heterogenic group of chronic osteomyelitis forms, which lacks the formation of pus and fistula. Topazian (1994, 2002) includes chronic sclerosing types of osteomyelitis, proliferative periostitis, as well as actinomycotic and radiation-induced forms to this group, whereas Bernier et al. (1995) advocate a more restrictive use of this term. Hudson (1993) uses the term to describe a condition of prolonged refractory osteomyelitis due to inadequate treatment, a compromised host, or increased virulence and antibiotic resistance of the involved microorganisms. This classification therefore also incorporates those cases in which a suppurative form of osteomyelitis can present as a nonsuppurative form in an advanced stage.

2.5.5 Diffuse Sclerosing Osteomyelitis, Primary Chronic Osteomyelitis, Florid Osseous Dysplasia, Juvenile Chronic Osteomyelitis

One of the most confusing terms among the currently used osteomyelitis nomenclature is "diffuse sclerosing osteomyelitis" (DSO). This term has apparently led to great confusion in the medical literature. A variety of denominations were used to describe this disease. One of the first descriptions was by Thoma in 1944, who used the term "ossifying osteomyelitis" and considered that a disease which was caused by a subpyogenic infection that could be found in tertiary syphilis. Sclerosing osteomyelitis was later described and divided into a focal

and diffuse types (Shafer 1957; Shafer et al. 1974; Pindborg and Hjorting-Hansen 1974; Mittermayer 1976; Topazian 1994, 2002). The focal type, also known as periapical osteitis/osteomyelitis or condensing osteitis, is a rather common condition with a pathognomonic, well-circumscribed radioopaque mass of sclerotic bone surrounding the apex of the root. 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, is a rare disease of unknown etiology that can cause major diagnostic and therapeutic problems (Jacobson 1984). The absence of pus, fistula, and sequestration are characteristic. The disease shows an insidious onset, lacking an acute state. It is therefore considered to be primary chronic and has been named primary chronic osteomyelitis by several authors, predominantly in the German and continental European medical and dental literature (Hjorting-Hansen 1970; Panders and Hadders 1970; Schelhorn and Zenk 1989; Eyrich at al 1999). Periods of onset usually last from a few days up to several weeks and may demonstrate a cyclic course with symptom-free intervals. Pain, swelling, and limitation of mouth opening, as well as occasional lymphadenopathy, dominate the clinical picture.

The term DSO is primarily descriptive of the radiological appearance of the pathological bone reaction; however, although the term is usually used synonymously with primary chronic osteomyelitis, it represents a description of a strictly radiological appearance that can be caused by several similar processes. These processes include primary and secondary chronic osteomyelitis, chronic tendoperiostitis, and ossifying periostitis or Garrè's osteomyelitis (Hjorting-Hansen 1970; Ellis et al. 1977; Eisenbund et al. 1981; Bünger 1984; Van Merkestyn et al. 1990; Groot et al. 1992b, 1996; Eyrich et al. 1999). This fact has most likely contributed to this diversity in nomenclature, as the terms are often used interchangeably.

A further pathological disease entity has been confused with diffuse sclerosing osteomyelitis, since it may mimic DSO radiographically by presenting sclerosing opaque and dense masses: florid osseous dysplasia (FOD). These masses are, however, confined to the alveolar process of either or both jaws in cases of FOD. Florid osseous dysplasia is mostly observed in black women and in many cases lacks clinical symptoms. Patients suffering from this disease, similar to true DSO, may in some instances also experience cyclic episodes of unilateral pain and mild swelling. This is usually the case when superinfection occurs (Schneider et al. 1990; Groot et al. 1996)

As with all pathologies of the bone which compromise local blood flow and host resistance, FOD makes the jaw more susceptible to secondary infection. In these instances pus and fistula formation may occur as well as sequestration (Carlson 1994). Many cases like these in the literature have, in retrospect, been incorrectly labeled as diffuse sclerosing osteomyelitis where these symptoms are by definition always absent. The FOD should therefore be considered more a bone pathology facilitating osteomyelitis once infection of the bone has been established and not equated with the infection itself.

As mentioned above, the exact etiology of true DSO remains unknown. A common theory is a low-grade infection of some kind; however, most biopsy specimens taken from the enoral and extraoral approach have failed to be conclusive, showing either no growth in cultures or growth only from suspected contaminants (Jacobson et al. 1982; Jacobson 1984; Van Merkesteyn et al. 1988). A study by Marx et al. (1994) demonstrated a high frequency of *Actinomyces*, *E. corrodens* species, *Arachnia* and *Bacteroides* spp. in cortical and medullar samples from patients with DSO. This study, like many others, still demonstrated insufficiencies regarding the protocol for collecting bone specimens and therefore was inconclusive. Moreover, a variety of antibiotics used over a long period consistently failed to fully eradicate the disease or arrest the symptoms (Jacobson 1984; Van Merkesteyn et al. 1988, 1990). Van Merkesteyn et al. (1990) and Groot et al. (1992a) have advocated other etiologies such as aberrant jaw positioning and parafunction; however, their theory lacks an explanation for those cases of true DSO in edentulous patients.

In our recent publications (Eyrich et al. 1999, 2003; Baltesperger et al. 2004) we used the term "juvenile chronic osteomyelitis," which resembles the clinical and radiological picture of Garrè's osteomyelitis as used by various authors. Heggie et al. (2000, 2003) made a similar observation when analyzing his young osteomyelitis patients and used the term "juvenile mandibular chronic osteomyelitis." This disease usually peaks at puberty and is characterized mostly by voluminous expansion of the mandibular body, periosteal apposition of bone ("periostitis ossificans"), and a mixed sclerotic-lytic appearance of the cancellous bone. The clinical

picture resembles primary chronic osteomyelitis, sharing the lack of pus formation, fistulae, or sequestration. Juvenile chronic osteomyelitis is therefore considered to be an early-onset form of primary chronic osteomyelitis. A further and more detailed description of this disease entity is described later in this chapter.

2.5.6 SAPHO Syndrome, Chronic Recurrent Multifocal Osteomyelitis (CRMO)

In 1986 Chamot et al. described a syndrome associated with synovitis, acne, pustulosis, hyperostosis, and osteitis (SAPHO syndrome). Soon, several case reports and studies were published, concluding a possible relationship between SAPHO syndrome and DSO of the mandible (Brandt et al. 1995; Kahn et al. 1994; Garcia-Mann et al. 1996; Suei et al. 1996; Schilling et al. 1999; Eyrich et al. 1999; Roldan et al. 2001; Fleuridas et al. 2002). Kahn et al. (1994) presented a small series of seven patients with DSO of the mandible out of 85 cases of SAPHO syndrome. Eyrich et al. (1999) presented a series of nine patients with DSO, eight of which also represented a SAPHO syndrome, supporting the hypothesis of a possible association of the two.

Chronic recurrent multifocal osteomyelitis (CRMO) is characterized by periods of exacerbations and remissions over many years. This rare disease is noted in adults as in children, although it is predominant in the latter group. In several articles published in the past few years, a possible nosological relationship between diffuse sclerosing osteomyelitis and chronic recurrent multifocal osteomyelitis has been described (Reuland et al. 1992; Stewart et al. 1994; Suei et al. 1994, 1995; Flygare et al. 1997; Zebedin et al. 1998; Schilling 1998; Schilling et al. 1999). In correlation with advanced age, there seems to be an increased association with palmoplantar pustulosis, a part of the SAPHO syndrome (Shilling et al. 2000). Because of its possible relationship with other dermatoskeletal associated diseases, CRMO has been integrated in the nosological heterogeneous SAPHO syndrome by several authors (Chamot et al. 1994; Schilling and Kessler 1998; Schilling et al. 2000).

2.5.7 Periostitis Ossificans, Garrès Osteomyelitis

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. 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.

Probably the most confusing and misinterpreted term regarding osteomyelitis is "Garrè's osteomyelitis." While most medical pathologists discard the term, it has still enjoyed great acceptance in the medical and dental literature, where occurrence in the jaws has been termed unequivocally (Eversole et al. 1979). Many terms have been used synonymously in the literature and attributed to Garrè, such as periostitis ossificans, chronic nonsuppurative osteomyelitis of Garrè, Garrè's proliferative periostitis, chronic sclerosing inflammation of the jaw, chronic osteomyelitis with proliferative periostitis, and many more. Table 2.5 gives an overview of the use of the term "Garrè's osteomyelitis" in the medical and dental literature; however, in his historical article in 1893, Carl Garrè did not actually describe a singular, specific type of osteomyelitis. Moreover he described special forms and complications of a single disease: acute infective osteomyelitis. He used 72 illustrative cases (98 sites) to discuss ten specific manifestations and complications of acute osteomyelitis. This is a direct contradiction to those authors who assume that he described a new form of chronic osteomyelitis (Wood et al. 1988).

2.5.8 Other Commonly Used Terms

2.5.8.1 Alveolar Osteitis (Dry Socket)

The clinical term "dry socket" or alveolar osteitis may also be regarded as a localized form of infection. Various authors have used this term differently. Hjorting-Hansen (1960) describes three principle forms of dry socket: alveolitis simplex; alveolitis granulomatosa; and an alveolitis sicca. Amler (1973) differentiates among alveolar osteitis, suppurative osteitis, and fibrous osteitis. The author concludes that the three types of osteitis correspond to disturbances during the natural healing process of an extraction alveolus. Meyer (1971) took great effort in demonstrating the histopathological changes in alveolar osteitis. He classifies this condition according to the degree of local invasion of the surrounding bone and uses the terms "osteitis circumscripta superficialis", "media" and "profunda". The term latter may be seen as a localized form of osteomyelitis; however, the

Table 2.5 Use of the term Garrè's osteomyelitis in medical and dental literature

Reference	Term used	Type of Publication
Batcheldor GD, Giansanti JS, Hibbard ED, Waldron CA (1) Garrè's osteomyelitis of the jaws: a review and report of two cases *J Am Dent Assoc 1973;87:892-7* Ellis DJ, Winslow JR, Indovina AA (2) Garrè's osteomyelitis of the mandible. Report of a case. *Oral Surg Oral Med Oral Pathol. 1977 Aug;44(2):183-9* Marx RE (3) Chronic Osteomyelitis of the Jaws Oral and Maxillofacial Surgery Clinics of North America, Vol 3, No 2, May 91, 367-81	Garrè's osteomyelitis	Case report (1 & 2) Review article (3)
Perriman A, Uthman A Periostitis ossificans. Br J Oral Surg 1972; 10:211-6	Periostitis ossificans	Review article
Smith SN, Farman AG. Osteomyelitis with proliferative periostitis (Garrè's osteomyelitis). Report of a case affecting the mandible. *Oral Surg Oral Med Oral Pathol. 1977 Feb;43(2):315-8*	Osteomyelitis with proliferative periostitis	Case report
Eisenbud L, Miller J, Roberts IL Garrè's proliferative periostitis occurring simultaneously in four quadrants of the jaws. *Oral SurgOral Med Oral Pathol. 1981 Feb;51(2):172-8*	Garrè's proliferativ e periostitis	Case report
Panders AK, Hadders HN Chronic sclerosing inflammations of the jaw. Osteomyelitis sicca (Garrè), chronic sclerosing osteomyelitis with finemeshed trabecular structure, and very dense sclerosing osteomyelitis. *Oral Surg Oral Med Oral Pathol 1970 Sep;30(3):396-412*	Osteomyelitis sicca (synonymous osteomyelitis of Garrè, chronic sclerosing nonsuppurative osteomyelitis of Garrè, periostitis ossificans)	Review article
Mittermayer CH Oralpathologie. *Schattauer, Stuttgart-New York 1976*	Chronic osteomyelitis with proliferative periostitis (Garrè's chronic nonsuppurative sclerosing osteitis, ossifying periostitis)	Textbook
Schelhorn P, Zenk W [Clinics and therapy of the osteomyelitis of the lower jaw]. *Stomatol DDR 1989 Oct;39(10):672-6* Bernier S, Clermont S, Maranda G, Turcotte JY Osteomyelitis of the jaws J Can Dent Assoc 1995 May;61(5):441-2, 445-8	Chronic sclerosing osteomyelitis Garrè, Garrè's chronic sclerosing osteomyelitis (proliferative osteomyelitis)	Review article
Topazian RG Osteomyelitis of the Jaws. In Topizan RG, Goldberg MH (eds): Oral and Maxillofacial Infections. *Philadelphia, WB Saunders 1994, Chapter 7, pp 251-88*	Garrè's sclerosing osteomyelitis	Textbook

term "alveolar osteitis" (dry socket) is generally used in the medical and dental literature to describe an absence of invasion into the bone. It should therefore not be regarded as a form of osteomyelitis (Marx 1991). 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 secondary chronic osteomyelitis once the bacterial invasion into the medullar and cortical bone has occurred and a deep bone infection has been established.

2.5.8.2 Osteoradionecrosis and Radioosteomyelitis

Radiotherapy is considered a major column in the treatment of head and neck malignancies. Despite recent advances in radiotherapy, such as using modern three-dimensional techniques, as well as hyperfractionation or moderately accelerated fractionation and consequent prophylactic dental treatment, osteoradionecrosis is still an observed condition in maxillofacial units.

Aside from its effect on the tumor cells, radiation also has serious side effects on the soft and hard tissues adjacent to the neoplasm. Mucositis, atrophic mucosa, xerostomia, and radiation caries are well-known side effects of head and neck radiotherapy. Because of its mineral composition, bone tissue absorbs more energy than soft tissues and is therefore more susceptible to secondary radiation. In cases where the bone is irradiated exceeding a certain local dose, osteoradionecrosis may develop, leading to marked pain in the patient and possible loss of bone leading to functional and aesthetic impairment.

Osteoradionecrosis was once considered an infection initiated by bacteria, which invaded the radiation-damaged bone; hence, the term "radiation-induced osteomyelitis" or radioosteomyelitis was commonly used. Marx (1983) conclusively identified this condition as a radiation-induced avascular necrosis of bone. He was able to demonstrate that radiation caused a hypoxic, hypocellular, and hypovascular tissue, leading to a spontaneous or trauma-initiated tissue breakdown. The result is a chronic nonhealing wound, susceptible to superinfection. As in florid osseous dysplasia and other bone pathologies, microorganisms are responsible for contamination and, if invasion occurs, secondary infection of the bone, resulting in osteomyelitis.

2.5.8.3 Osteochemonecrosis

The medical literature describes several drugs and substances that facilitate or induce conditions known as osteonecrosis of the jaws, such as corticosteroids and other cancer and antineoplastic drugs. Exposure to white phosphorous among workers in the matchmaking industry in the nineteenth century has led to unusual necroses of the jaws, which became known in the literature as phossy jaw or phosphorous necrosis of the jaw.

In the recent years bisphosphonate therapy has become a widely accepted mainstay of therapy in various clinical settings such as multiple myeloma, metastatic cancer therapy, and treatment of advanced osteoporosis. With the increased prescription of these drugs, the incidence and prevalence of bisphosphonate-associated complications of the jaw continues to be elucidated. This trend seems to be even more the case in patients receiving injectable bisphosphonates, such as pamidronate and zoledronic acid, but cases involving osteochemonecrosis of the jaw associated with chronic peroral administered bisphosphonates have also been reported (Ruggiero et al. 2004, 2006).

The pathophysiological mechanisms leading to bisphosphonate-induced osteochemonecrosis of the jaws are yet far from being fully understood; however, it seems apparent that important differences to the pathogenesis of osteoradionecrosis do occur (Hellenstein and Marek 2005). In bisphosphonate-induced osteochemonecrosis of the jaws osteoclastic action is reduced, but osteoblastic production continues, leading to an osteopetrosis-like condition (Whyte et al. 2003). 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 to date; however, inhibition of angiogenesis is currently being actively investigated (Fournier et al. 2002; Wood et al. 2002), and further research will hopefully help fully understanding its role in pathogenesis of this disease.

Regarding the current data and knowledge, we favor the term "bisphosphonate-induced osteochemonecrosis of the jaw" because it is not restricted to a certain pathogenesis. The term "bisphosphonate osteomyelitis" should not be used for the same reasons as the term radioosteomyelitis should be abandoned. The jawbone

with bisphosphonate-induced osteochemonecrosis is far more susceptible to bacterial invasion due to its strongly altered physiology; however, infection of the bone is to be considered a secondary phenomenon and not the primary cause of this disease entity.

2.6 Osteomyelitis of the Jaws: The Zurich Classification System

2.6.1 General Aspects of the Zurich Classification System

Osteomyelitis of the jaw as a clinical entity has long been recognized in the medical literature. As mentioned previously, various classification systems and nomenclatures of the disease have evolved with time. The heterogeneity of the classification systems is borne by the fact that several modalities are used to describe and define maxillofacial osteomyelitis. These modalities include etiology and pathogenesis, clinical presentation and course, radiology, and histopathology. Furthermore, most classification forms represent a mixture of these criteria, causing confusion, thereby hindering comparative studies.

At the Department of Cranio-Maxillofacial Surgery at the University of Zurich, the classification system for osteomyelitis of the jaws uses a hierarchical order of classification criteria. It is primarily based on clinical appearance and course of the disease, as well as on radiological features. Based on these criteria, three major groups of osteomyelitis can be distinguished:

1. Acute Osteomyelitis (AO)
2. Secondary Chronic Osteomyelitis (SCO)
3. Primary Chronic Osteomyelitis (PCO)

Within these major groups, the clinical presentation is similar in the majority of cases; however, as will be described later, a certain variety of the clinical course is noted, especially in cases of primary and secondary chronic osteomyelitis.

Histopathology is considered a secondary classification criterion, taking into account that findings are mostly unspecific and nonconclusive when considered by themselves; however, tissue examinations of biopsies are irreplaceable for confirmation of the diagnosis in cases of unclear and atypical clinical and radiological appearance, and moreover in excluding possible differential diagnosis.

Furthermore, in some cases of osteomyelitis with an atypical appearance a synthesis of medical history, clinical presentation, imaging studies, histopathology, and other diagnostic tools may be necessary to achieve an appropriate diagnosis.

Analysis of the osteomyelitis patients treated in the Department of Cranio-Maxillofacial Surgery in Zurich using the abovementioned major classification groups showed a clear predominance of cases diagnosed as secondary chronic osteomyelitis at the time of presentation, whereas cases of acute osteomyelitis and primary chronic osteomyelitis were significantly less often diagnosed (Table 2.6). In a small group of nine patients, despite meticulous work-up of all data including clinical course and symptoms, diagnostic imaging, laboratory

Table 2.6 Distribution of osteomyelitis cases treated at the Department of Cranio-Maxillofacial Surgery in Zurich, 1970–2000 (Baltensperger 2003)

Major groups of osteomyelitis of the jaws	Cases	
	N	%
Acute osteomyelitis (AO)	48	16.6%
Secondary chronic osteomyelitis (SCO)	203	70.0%
Primary chronic osteomyelitis (PCO)	30	10.3%
Not clearly classifiable/questionable osteomyelitis	9	3.1%
Total	290	100.0%

findings, and histopathology, no clear diagnosis was possible. Most of these cases showed a chronic course resembling primary chronic osteomyelitis or a (diffuse) sclerosing form of secondary chronic osteomyelitis. In some of these cases the diagnosis of osteomyelitis was even questionable. The problems in diagnosis of these challenging cases and possible related differential diagnosis are outlined later in this chapter.

Further subclassification of these major osteomyelitis groups is based on presumed etiology and pathogenesis of disease. These criteria are therefore considered tertiary classification criteria. These tertiary criteria are helpful in determining the necessary therapeutic strategies which may differ somewhat among the subgroups. The nature of these subgroups are outlined in more detail later in this chapter.

An overview of the Zurich classification of osteomyelitis of the jaws and the classification criteria are given in Fig. 2.2 and Table 2.7.

2.6.2 Acute Osteomyelitis and Secondary Chronic Osteomyelitis

2.6.2.1 Definitions

The basic terminology used in the Zurich classification of osteomyelitis of the jaws was promoted by Hugo Obwegeser, among others. The general principles of this classification system were described and published by E. Hjorting-Hanson, a former staff member at the Department of Cranio-Maxillofacial Surgery Zurich, in 1970. Hjorting-Hanson, as many other authors before and after him, gave an excellent description of the clinical and radiological picture of acute and secondary chronic osteomyelitis; however, he fell short of clearly defining at what stage an acute/subacute osteomyelitis should be considered chronic. To our knowledge, Marx (1991) and Mercuri (1991) were the first and only authors to define the duration for an acute osteomyelitis

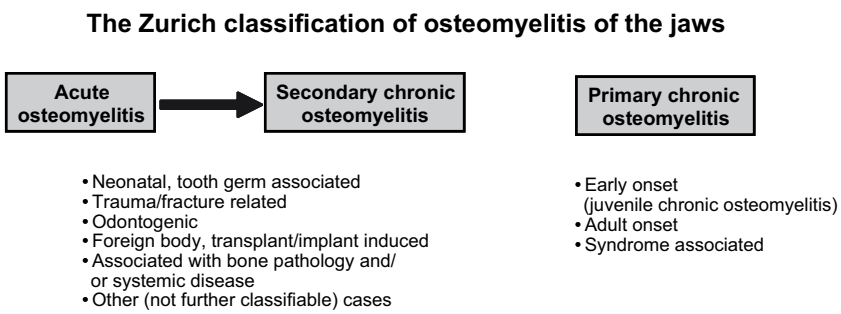

Fig. 2.2 The Zurich classification of osteomyelitis of the jaws: since secondary chronic osteomyelitis is a sequel of the prolonged and chronified acute form, both basically have the same subclassification groups

Table 2.7 Classification criteria upon which the Zurich classification of osteomyelitis is based

Hierarchic order of classification criteria	Classification criteria	Classification groups
First	Clinical appearance and course of disease Radiology	Major Groups Acute osteomyelitis (AO) Secondary chronic osteomyelitis (SCO) Primary chronic osteomyelitis (PCO)
Second	Pathology (gross pathology and histology)	Differentiation of cases that cannot clearly be distinguished solely on clinical appearance and course of disease; important for exclusion of differential diagnosis in borderline cases.
Third	Etiology Pathogenesis	Subgroups of AO, SCO, and PCO