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# Osteomyelitis of the Jaws: Evolution and Clinical Relevance of Classification Systems

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KEYWORDS

Osteomyelitis (OML), Jaws, Classification, Chronic osteomyelitis, Suppurative osteomyelitis

**ABSTRACT:** Osteomyelitis (OML) of the jaws is an inflammatory condition of bone that predominantly arises from odontogenic infections and is influenced by the unique anatomical and vascular characteristics of the maxillofacial skeleton. Unlike osteomyelitis of long bones, jaw osteomyelitis demonstrates varied clinical behaviour, ranging from acute suppurative infections to chronic, non-suppurative sclerosing forms. Over time, several classification systems have been proposed based on clinical course, presence of suppuration, radiographic extent, aetiology, and pathogenesis. These classifications play a crucial role in diagnosis, treatment planning, and prognostic assessment. This narrative review aims to comprehensively summarize and critically analyse the various classification systems of osteomyelitis of the jaws, highlighting their clinical relevance and limitations. Understanding these classifications allows clinicians to tailor management strategies and improve patient outcomes.

**INTRODUCTION:** Osteomyelitis of the jaws remains a unique disease of the facial skeleton that poses a significant challenge for both the physician and the patient, despite recent advances in diagnosis and treatment modalities. In the past, osteomyelitis was encountered frequently & dreaded because of its prolonged course, the uncertainty of its outcome & occasional disfigurement resulting from the loss of teeth & bone. But today the entire scenario has changed; it is much less common. Improved nutrition, dental care, and the availability of antibiotic therapy & intervention based on new imaging modalities have been significant factors in reducing the disease's morbidity. Since the second half of the 12$^{th}$ century, however, there has been a dramatic reduction in the jaws and other bones of the skeleton [3]. Despite all these 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 immunocompromised drugs, the rising incidence of AIDS, diabetes, and other medically compromising conditions have led to the new problems in the treatment of osteomyelitis of jaws, leading again to increase of cases refractory to standard therapies [1].

The clinical presentation of jaw osteomyelitis is highly variable, reflecting differences in etiology, host response,

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microbial flora, and disease duration. As a result, multiple classification systems have evolved to describe and categorize this condition. A clear understanding of these classifications is essential, as they influence diagnostic approaches, treatment modalities, and prognosis. This review focuses exclusively on the various classification systems of osteomyelitis of the jaws and their clinical significance.

**RATIONALE**: Classification of osteomyelitis of the jaws serves several important purposes. It facilitates accurate diagnosis by distinguishing acute infections from chronic and non-infective inflammatory conditions. Classification systems also guide treatment planning, as acute osteomyelitis may respond to antibiotic therapy alone, whereas chronic forms often require surgical intervention. Furthermore, classification aids in predicting prognosis, assessing recurrence risk, and standardizing communication in clinical practice and research. Despite these benefits, the lack of a universally accepted system underscores the complexity of this disease.

**PREDISPOSING FACTORS OF OML:** Osteomyelitis of the jaws is predisposed by a combination of local, systemic, and iatrogenic factors that compromise bone vascularity and host defense. The most common local factors include untreated odontogenic infections, periodontal disease, trauma, fractures, and surgical procedures such as tooth extractions and implant placement. Systemic conditions like diabetes mellitus, immunosuppression, malnutrition, anemia, and advanced age reduce resistance to infection and impair healing. Lifestyle factors such as tobacco smoking and alcohol abuse further compromise the blood supply. Additional risk factors include radiation therapy, medications that affect bone metabolism, and infections caused by virulent or resistant microorganisms, all of which increase susceptibility to jaw osteomyelitis **(Fig. 1)**

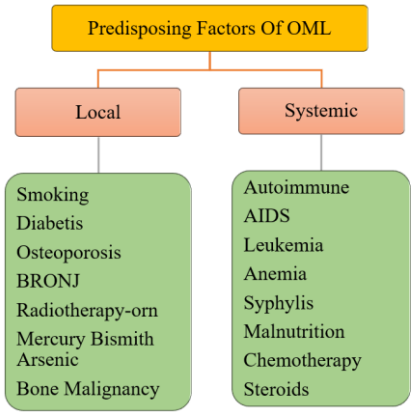


**Fig. 1 Predisposing factors of OML**

**PATHOGENESIS**: The pathogenesis of osteomyelitis of the jaws begins with microbial invasion of the medullary cavity, most commonly from odontogenic infections, trauma, or surgical procedures. The disease spreads through the Haversian and Volkmann canal systems, leading to acute inflammation, vascular congestion, and thrombosis, which compromise the local blood supply. Reduced perfusion results in bone ischemia and necrosis, forming sequestra that act as persistent foci of infection. The surrounding viable bone attempts repair by new bone formation, producing an involucrum. In chronic cases, ongoing low-grade infection and impaired host response lead to fibrosis, sclerosis, sinus tract formation, and recurrent exacerbations **(Fig. 2)**

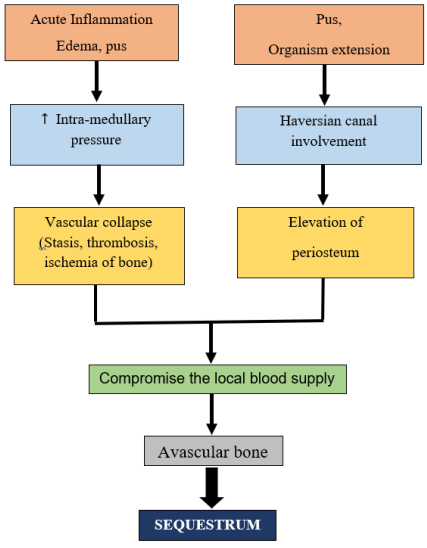


**Fig. 2 Pathogenesis of OML**

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**Table 1: Classification of Osteomyelitis of the Jaw**

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| Wasmund [4] 1935 | **Based on the clinical picture & radiology**<br>• Executive osteitis.<br>• Receptive osteitis.<br>• Productive osteitis.<br>• Acute necrotizing osteomyelitis.<br>• Chronic osteomyelitis. |
| Panders AK & Handers HN [5] 1970 | **Based on clinical picture &radiology [classification of chronic osteomyelitis forms only]**<br>1) Primary chronic jaw inflammation<br>• Osteomyelitis sicca (synonymous osteomyelitis of Gawe OML)<br>• Chronic sclerosing OML with the fine-meshed trabecular structure<br>• Local and more extensive, very dense sclerosing OML<br><br>2) Secondary chronic jaw inflammation<br>Chronic specific jaw inflammations: Tuberculosis, Syphilis, Lepra, Actinomycosis. |
| Hsoring-Hansen E -1970 [6] | **Based on the clinical picture and radiology.**<br>• Acute/ subacute osteomyelitis<br>• Secondary chronic osteomyelitis<br>• Primary chronic osteomyelitis |
| Mitter Mayer CH -1976 [7] | **Based on the clinical picture, radiology, pathology & etiology:** -<br>• Acute suppurative osteomyelitis<br>• Chronic suppurative osteomyelitis<br>• Chronic focal sclerosing osteomyelitis<br>• Chronic diffuse sclerosing osteomyelitis<br>• Chronic osteomyelitis<br>• Specific osteomyelitis<br>✓ Tuberculous osteomyelitis<br>✓ Syphilitic osteomyelitis<br>✓ Actinomycosis osteomyelitis |
| Waldvogel [8] 1970 | **1. Mechanism of done infection**<br>2. Hematogenous -Secondary to bacterial transport through the blood, mainly in children<br>3. Contiguous -Bacterial inoculation from and adjacent focus: Eg, post-traumatic OML inf. Related to prosthetic devices.<br>4. Associated with- Inflections affecting the feet in patients with vascular insufficiency, DM, and peripheral vascular insufficiency<br><br>**Duration of infection:**<br>**Acute**: Initial episodes of OML, edema formation of pus, vascular congestion thrombosis of small vessels.<br>**Chronic**: Recurrence of acute cases, large areas of ischemia, necrosis & bone sequestra. |

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| Cierny and Mader 1985 [9] | **Anatomical stage**<br>1. Medullary infection restricted to the bone marrow<br>2. Superficial inflection restricted to cortical bone<br>3. Localized inflection with clearly defined edges & bone stability preserved.<br>4. Diffuse inflection spreads to the entire bone circumference, with instability before/after debridement.<br><br>**Classification of host:**<br>A) Host healthy- Patients without comorbidities<br>B) Local compromise - smoking, chronic lymphedema, venous stasis arthritis, significant scars, fibrosis by RT<br>C) System compromise - DM, malnutrition, renal/ hepatic failure, chronic hypoxia.<br>D) Poor clinical Condition-surgical treatment will have a higher risk than OML itself. |
| Schelhorn & Zenk 1989 | **Based on the clinical picture**<br>• Acute OML<br>• Secondary chronic OML<br>• Primary chronic OML<br>• Special form: osteomyelitis sicca, Gases OML |
| Marx & Mercuri [10] | **Based on clinical /radiological/etiology/pathophysiology.**<br>**A) Acute osteomyelitis**<br>1. Associated with hematogenous spread<br>2. Associated with intrinsic bone pathology (or)PVD<br>3. Associated with odontogenic/odontogenic local process<br>**B) Chronic osteomyelitis**<br>1. Chronic recurrent multifocal OML of children<br>2. Garris OML<br>3. Chronic supportive OML: Foreign body-related, Systemic disease related to persistent /resistant organisms<br>4. True chronic diffuse sclerosing osteomyelitis |
| Hudson -1993 [3] | **ACUTE FORMS** (suppurative /non-suppurative)<br>Contiguous focus: Trauma, surgery, and odontogenic infection<br>• Progressive: burns, sinusitis, and vascular insufficiency<br>• Hematogenous (metastatic): developing skeleton (children)<br>**CHRONIC FORMS**<br>• Recurrent multifocal: Developing skeleton (children), Escalated osteogenic activity (<age 25 years)<br>• Garre's: Unique proliferative subperiosteal reaction, Developing skeleton (children to young adults)<br>• Suppurative or Non-suppurative: Inadequately treated forms, systematically compromised forms, and Refractory forms (CROML: Chronic Refractive Osteomyelitis)<br>• Diffuse sclerosing: Fastidious organisms, and compromised host or pathogen interface |

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| Topazian -1994 [02] | **A) Suppurative**<br>• Acute suppurative osteomyelitis - Primary chronic suppurative / Secondary chronic suppurative<br>• Chronic suppurative osteomyelitis<br>• Infantile osteomyelitis<br><br>**B) Non-suppurative**<br>• Chronic sclerosing osteomyelitis: Focal sclerosing osteomyelitis & Diffuse sclerosing osteomyelitis<br>• Garre's sclerosing osteomyelitis<br>• Actinomycotic osteomyelitis<br>• Radiation osteomyelitis (osteoradionecrosis)<br>• Specific infective osteomyelitis: Tuberculosis & Syphilis types |
| Bernier -1995 [11] | **I) Suppurative: Acute & Chronic forms**<br>**II) Non-suppurative**<br>• Chronic focal sclerosing osteomyelitis<br>• Chronic diffuse sclerosing osteomyelitis<br>• Garre's osteomyelitis<br>• Osteoradionecrosis |
| Zurish [1] | 1) Acute osteomyelitis < 4 weeks Presence of pus, fistula (or) sequestration<br>**Origin**- Dental trauma/ fracture, foreign body, Hematogenous<br>2) Secondary osteomyelitis > 4 weeks: Presence of pus, fistula /sequestration.<br>**Origin** -Dental trauma/fracture, foreign body, Hematogenous<br>3) Primary chronic osteomyelitis > 4 weeks: Absence of pus, fistula/ sequestration.<br>**Unknown origin**- Associated syndrome-SAPHO Chronic recurrent multifocal OML |

**CLINICAL RELEVANCE OF CLASSIFICATION:** Accurate classification of osteomyelitis of the jaws has direct implications for treatment selection, prognosis, and prevention of complications. Acute forms benefit from early antibiotic therapy, whereas chronic and sclerosing forms often necessitate surgical management. Classification also aids in distinguishing osteomyelitis from conditions such as osteoradionecrosis, medication-related osteonecrosis, and malignancies.

**LIMITATIONS OF EXISTING CLASSIFICATIONS:** Despite numerous proposed systems, overlap between categories and inconsistent terminology remain significant limitations. No single classification comprehensively addresses clinical, radiographic, microbiological, and host-related factors. This lack of consensus can complicate diagnosis and comparison of research outcomes.

**FUTURE DIRECTIONS:** Future classification systems should integrate clinical presentation, imaging findings, microbial characteristics, and host immune status. Advances in molecular diagnostics and imaging may contribute to more precise and standardized classification models.

**CONCLUSION:** Osteomyelitis of the jaws encompasses a spectrum of inflammatory bone disorders with diverse clinical presentations. Multiple classification systems have been proposed to describe this condition, each with distinct advantages and limitations. A thorough understanding of these classifications is essential for accurate diagnosis, effective treatment planning, and improved patient outcomes. Continued efforts toward a unified and clinically relevant classification system are warranted.

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