

The Contagious Orifice: Maxilla and Mandible

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Abstract

Osteomyelitis (OM) is a bony inflammation of the maxillofacial skeleton with the participation of medullary and cortical bone, due to bacterial transgression. OM is described as the Suppurative type with pus/fistula/abscess/sequestration, where infectious agents materialize inconsistently, and the Non suppurative chronic inflammation of obscure aetiology. Conclusive criterion are i) narrative and clinical exposition. ii) imaging methodologies iii) culture specimens iv) histological assessment. Surgical objectives are necrotic debridement and conserving viable bone to expedite bone healing and bone defect rebuilding. Biodegradables employed are polylactic acid, polyglycolic acid, polyparadiioxane polyesters, hydroxyapatite, bioceramics, polymers, ceramic composites calcium phosphates, fibrin sealed implants etc. Bisphosphonates are miraculous for pain relief in PCO, DSO, DSOM, even in long standing illness.

Osteomyelitis (OM) is a bony inflammation of the maxillofacial skeleton with the participation of medullary and cortical bone, due to bacterial transgression, composed of the Greek word Osteon (bone) and Meulinos (marrow) [1]. Consequently, osteomyelitis is epitomized as an inflammation of the mandibular basal and alveolar bone. The exceedingly vascular maxilla with thinner cortex is sporadically affected. The expected mandibular locales are the body, the symphysis, angle, ascending ramus and condyle. The therapy of OM of the jaw is intricate since the chronic patterns tend to reappear. OM is described as the Suppurative type with pus/fistula/abscess/sequestration, where infectious agents materialize inconsistently, and the Non suppurative chronic inflammation of obscure aetiology [1]. An obvious odontogenic infectious aetiology or traumatized mucus membrane/bony tissue with pathogenic microbial influx to the bone marrow or cortex, assigns Secondary chronic OM [2]. Paediatric and immunocompromised population evinces a haematogenous influx of OM of the jaw. Chronic Non suppurative OM has a surreptitious, vague inception with negligible growth of organisms and an unknown aetiology for chronic, bony cortical/cancellous incrimination [3]. Symptoms evolve over a period of time with asymptomatic intervals succeeding exacer-

bations. Primary chronic OM(PCO), Diffuse sclerosing OM (DSO) Diffuse sclerosing OM of the mandible(DSOM) Juvenile mandibular chronic OM(JMCO), Chronic recurrent multifocal OM (CRMO), Chronic non-bacterial OM(CNO) acquire identical annotations [1]. CRMO is dissimilar from PCO, DSO, DSOM & CNO due to confirmation of abundant primary loci. CRMO is the elucidation of SAPHO syndrome (synovitis, acne, pustulosis, hyperostosis and osteitis) [1]. Non suppurative OM of the jaw can be aseptic or pathogenic. Infection with *Actinomyces* and *Eikenella corrodens* manifests with mild/ no symptoms. CRMO is decreed aseptic. Classification: Primary chronic OM(PCO) is a chronic, non-suppurative OM of mysterious aetiology [1]. Secondary chronic OM(SCO) is a designated suppurative infection with abscess/fistula/belated sequestrum composition. Acute and Subacute OM is a continuous infection < 4 weeks while with the Chronic OM(CO) pyogenicity extends

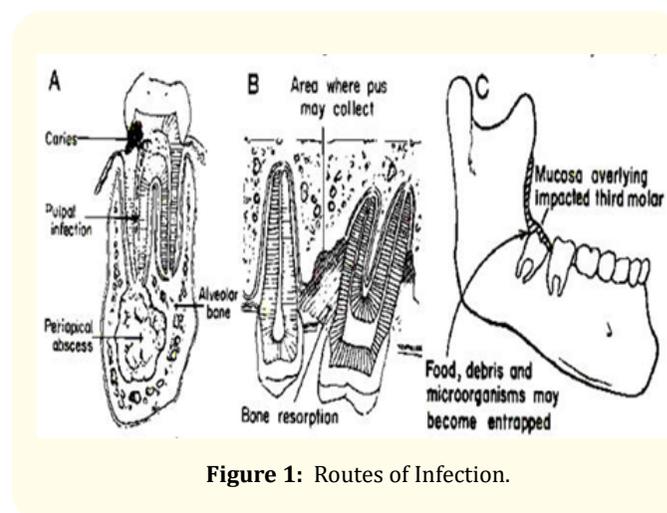


Figure 1: Routes of Infection.

Diagnosis

OM of the jaw is overwhelming for competent diagnoses, therapy and amelioration. Subclassifications and diversifications are basically similar, with inconsistent degree and execution, few with distinct radiologic manifestations, numerous focal afflictions, sup-

uration, victim's age and reoccurrences [4]. Conclusive criterion are. i) narrative and clinical exposition. ii) imaging methodologies iii) culture specimens iv) histological assessment [1]. Prevalent clinical characteristics of suppurative OM are local intense pain, tenderness, fever, painful/painless swelling, purulent discharge, intraoral fistula, skin fistula, trismus, hypoesthesia of the inferior dental nerve and pathologic fractures [5]. Favourable bacterial ingress can imitate acute or prolonged alveolar osteitis. Non suppurative OM of the jaw features recurrent pain, swelling, restricted mouth opening, inadequate secretions, periosteitis and sporadic lymphadenopathy and diminished inferior alveolar sensation [1].

Radiography

PCO of the mandible shows a mixed pattern of sclerosis and osteolysis mandibular expansion, periosteal bone reaction, indistinct corticomedullary margin. Periosteal fresh bone laminates and sequestra are radiologically unique characteristics of OM [1,6]. PCO displays osteosclerosis/osteolysis and onion skinning of the sub-periosteal new bone. IMAGING: Conventional radiographs are for primary assessment and as an addenda to further imaging, ratification and analysis and overview in maxillofacial OM.

Organization of OM: The Zurich system is decisive [2].

Hjorting Hansen.E	Clinical Picture Radiology	Acute/Subacute	Secondary Chronic	Primary chronic
Panders AK. Hadden HN.	Clinical Picture	Acute Special forms	Secondary chronic	Primary chronic
Marx R	Clinicals Radiology Aetiology Pathophysiology	Acute (haematogenous, intrinsic, local odonto- genic)	Chronic, Garre's, Chronic suppurative	True chronic, chronic recurrent multifocal
Hudson JW	Clinicals, Radiology	Acute-Contagious, Progressive, Haematogenous	Chronic-Garre, Re- current multifocal	Suppurative Non suppurative, Diffuse Sclerosing
Topazian RG	Clinical picture, radiology, aetiology	Suppurative Acute, Chronic Infantile Primary/Second- ary Chronic	Non suppurative, Chronic scleros- ing, Focal / Diffuse sclerosing	Garre', Actinomy- cotic, Radiation OM & Necrosis
Bernier's OM of the Jaw	Suppurative Acute/Chronic	Non suppurative OM	Chronic Focal sclerosing Chronic diffuse sclerosing.	Garre's chronic scler- osing Osteoradio necrosis
Zurich 's Classification	Clinical course Imaging	Acute OM	Secondary Chronic OM (SCO)	Primary Chronic OM (PCO)

CT scans

CT scans are handy for primary planning in surgeries of undisguised annihilation of the bone, staging and follow ups. Soft tissue distortions can be perceived with PET CT (positron emission tomography) scans. Integration with classic CT augments the sensitivity and specificity by determining the anatomy and metabolism [1,7]. LASER DOPPLER flowmetry: medullary inflammation reduces mandibular effusion. MRI scans using gadolinium contrasts, appropriate for early, acute, OM, disease monitoring but restricts comparisons between edema/infection and metal implants diminish the diagnostic imagery [1].

Radionuclide Scans

Predominant is scintigraphy with technetium 99m, determining bone development. Alternatives are gallium 67 and indium 111.

Bone scintigraphy with single emission computerized tomography (SPECT) has a greater sensitivity [1,8]. Fluorodeoxyglucose PET is suitable for evaluating disease remission and follow up. Osteoblast activity and bone rejuvenation is signalled by technetium 99m MDP uptake. Initiation of early inflammation within 3 days is evidenced by a consolidation of 99m Tc and gallium 67. SPECT and low dose CT is a substitute to traditional CT curtailing hazardous exposure to radioactive rays and upgraded, coalesced images. Radioisotope scans can recognize additional affected zones [1]. HAEMATOLOGY: Total leucocyte count is markedly raised, as is the erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). Immunoglobulins IgG, IgA, and IgM coordinate poorly with CRP. DIFFERENTIAL is necessitated from bacterial OM of the jaw, Paget's disease, hypercalcaemia, hypercementosis, fibrous dysplasia, primordial malignant bone tumours, RADIOGRAPHIC VARIA-

TIONS are osteogenic sarcoma, fibrous dysplasia. Non suppurative OM of the jaw: PCO duplicates osteosarcoma, chondrosarcoma, Ewing's sarcoma, non-hodgkin's lymphoma, metastasis histiocytosis X, leukaemia and neuroblastoma. Benign deliberations are fibrous dysplasia, ossifying and non-ossifying fibroma, juvenile parotitis, chronic sialadenitis, paget's disease, cementoma, nonspecific chronic lymphadenitis [1]. HISTOLOGY; Mandibular suppurative OM exhibits bony necrosis with depleted osteolytic lacunae, haversian canals and a paucity of osteoblastic rimming. Inflammatory invasion of the marrow comprises of neutrophils, plasma cells, histiocytes, lymphocytes, osteoclastic activity, thrombosed and hyperaemic marrow arterioles and capillaries [1,2]. Decalcification permits microorganisms in 33% cases. The disease commences in the marrow and stimulates a viable, layered periosteum (74%).

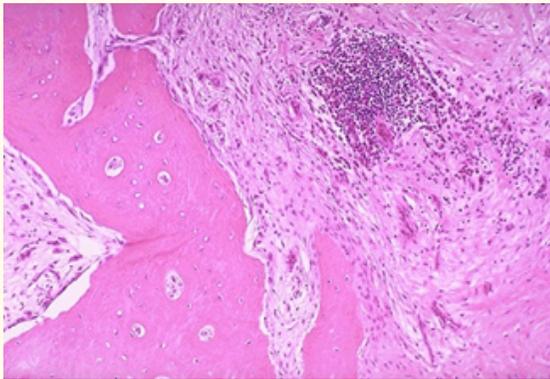


Figure 2: Healing OM.

Suppurative OM, in which the bacterial toxins, initiate an inflammatory reaction, engenders vascular thrombosis and creates an anaerobic milieu, chiefly facultative. Non suppurative OM (PCO) endorses a chronic nonspecific inflammation, with cumulative bone resorption, sclerosis and medullary fibrosis. THERAPEUTICS varies in infective cases. Surgical options are analogous. Suppurative OM warrants adequate vascularization followed by infection control [1]. Surgical objectives are necrotic debridement and conserving viable bone to expedite bone healing bone defect rebuilding. Intervention entails bone decortication with or without bone grafting, sequestromy, saucerisation etc., Teeth sustain bacterial ingress, infection, and therapeutic non-performance, necessitating removal. Partially resected bone is gradually reformed, in critical, discontinuous cases. In septic and aseptic OM of the mandible, decortication is of choice. In non-suppurative OM (PCO, DSO, CRMO) surgery is unsatisfactory. (even with an amalgamation of antibiotics, NSAIDS, hyperbaric oxygen). Non-surgical modalities are antibiotics, NSAIDs, steroids, chemotherapeutic agents as they balance bony modulation and immune reaction. In undiagnosed OM, antibiotics are prescribed. Established OM certifies a varying

antibiotic regimen. Crucial antibiotic objectives of a polymicrobial flora in infective OM, require adaptation as per culture/disease furtherance. Evolving antibiotic resistance can adversely influence consequences Continued antibiotic therapy for 2 to 6 weeks, intravenous, subsequently oral and for post-operative control of infection in PCO, is advocated [1,2]. However, exclusive administration of antibiotics is not advantageous. Transfer of antibiotics, locally, is by non-resorbable and resorbable modalities. A non-resorbable polymethylmethacrylate (PMMA) bead intensifies local antibiotics but necessitates a subsequent surgical evacuation, and accounts for low grade foreign body reaction and an erratic drug dispensation. Biodegradables employed are polylactic acid, polyglycolic acid, polyparadoxane polyesters, hydroxyapatite, bioceramics, polymers, ceramic composites calcium phosphates, fibrin sealed implants, collagen sponges, however in animal models of OM. Notwithstanding, the local antibiotic delivery is calculable, and second surgery is unnecessary [1,2]. Non-steroidal anti-inflammatory drugs (NSAIDS) inhibit cyclooxygenase, an enzyme engaged in prostaglandin, prostacyclin and leukotriene synthesis. Divaricating cellular mechanisms are managed by these cellular mediators e.g. neovascularisation, vascular homeostasis, febrile progression, inflammation and pain receptor regulation [1]. Steroids alleviate the symptoms. Hyperbaric oxygen augments the oxygen tension, assists angiogenesis, ascertains blood flow, enhanced oxygen tension improves neutrophilic killing, fibroblastic and osteoclastic movement, and composing of oxygen radicals, for immediate annihilation of anaerobes and facultative aerobes.

Chemotherapeutics

Bisphosphonates are pyrophosphate analogues competently inhibiting osteoclastic bone resorption/ remodelling. Osteolytic modification relieves pain. Methotrexate has equivocal results and adverse effects, pending additional evaluation. Calcitonin organizes bone turnover, controls calcium balance and homeostasis, restricts prostaglandins, augments endorphins formulation, diminishes bone pain and improves restoration. Clinical remission is maximum with Tumour necrosis factor alpha inhibitor [1,2]. OM of the jaw is affiliated with systemic illnesses like diabetes mellitus, autoimmune deficiencies, rheumatic arthiritis, cancer chronic inflammatory bowel disease, palmoplantar pustulosis malignancies, malnutrition, alcoholism, autoimmune deficiency syndrome (AIDS) and sickle cell anaemia etc. Bacterial pathogenic invasion seconds immunologic derangements e.g. defective cellular immunity and decreased phagocytosis. Pathogenic bacteria are admitted through the mucosal barrier, infected teeth or haematogenously and install a localized bony infection [1,2]. A chronic inflammation originates which endangers the local blood flow and eventuates avascular necrosis. Extensive, prolonged infections alter the bacterial pathogenicity primarily from aerobic to anaerobic, engendering treatment resistance. Immune mediation is favoured when a

localized endarteritis propagates PCO or chronic OM. Bacterial pathogens activate immune reactions which are subsequently autonomous.

Autoimmune disease possibly provokes PCO, evoking altered immune response considering i) Bone/joint molecules are imitated by fragmented microorganisms and immune mechanisms invade normal osteoarticular tissue. ii) Immunoglobulins conjoin with fragmented microorganisms and precipitate in bones/ joints thus instigating non-infected tissue iii) A skin infection demolishes the barrier between immune cells and superficial skin antigens. The mounted immune response assaults the normal skin antigens thereby and initiating an inflammatory cascade [1]. BIOPSY AND CULTURING: Bone cultures in chronic OM are polymicrobial and are often conclusive for oral/skin flora (> 90%) This however, could be due to biopsy contamination, improper handling or culture. Anaerobes are degraded in 15 minutes and aerobes within 2 hours of sampling. The anaerobes are best cultivated at 37 degree centigrade and varying carbon dioxide concentrations. Hence, specific biopsy and culture methodologies are in discord. Blood is an undependable culture medium [1].

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ASEPTIC OM (CRMO, DSO, PCO) evince an autoimmune aetiology aided by concomitant autoimmune disorders e.g. SAPHO (synovitis, acne, pustulosis, hyperostosis, and osteitis), palmoplantar pustulosis, localized pustular psoriasis, diabetes mellitus, chronic inflammatory bowel disease psoriasis vulgaris etc [1,2]. Systemic illnesses viz diabetes mellitus, anaemia, malnutrition modify host resistance and markedly reforming the course of the disease. TREATMENT: Surgical and non-surgical. Surgery is a standard protocol for PCO. However, future research may alter the choice of therapy. Bisphosphonates are miraculous for pain relief in PCO, DSO, DSOM, even in long standing illness. Bisphosphonate associated for OM of the jaw

(BAOMJ) may be precipitated with immunodeficiency, old age > 70 years, surgical interventions, tooth extraction, oral hygiene, infections, glucocorticoids and chemotherapeutics. Conventionally, OM of the jaw is typically localized on one side of the mandible, rarely affecting the maxilla or other cranial bones [1, 9-13].

Conclusion

Osteomyelitis is epitomized as an inflammation of the mandibular basal and alveolar bone. The exceedingly vascular maxilla with thinner cortex is occasionally affected. The mandibular sites affected are the body, the symphysis, angle, ascending ramus and condyle. The therapy of OM of the jaw is intricate since the chronic patterns tend to reappear. The treatment varies in infective cases. Suppurative OM warrants adequate vascularization followed by infection control [1]. Surgical objectives are necrotic debridement and conserving viable bone. Intervention entails bone decortication with or without bone grafting, sequestromy, saucerisation etc. Non-steroidal anti-inflammatory drugs (NSAIDS) manage the cellular mediators e.g. neovascularisation, vascular homeostasis, febrile progression, inflammation and pain receptor regulation [1]. Steroids alleviate the symptoms. Hyperbaric oxygen augments the oxygen tension for immediate annihilation of anaerobes and facultative aerobes. Bisphosphonates are pyrophosphate analogues competently inhibiting osteoclastic bone resorption/ remodelling. Osteolytic modification relieves pain. Calcitonin organizes bone turnover, diminishes bone pain and improves restoration. Clinical remission is maximum with Tumour necrosis factor alpha inhibitor.

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12. Image 1 Courtesy Antimicrobe org.
13. Image 2 Courtesy, The pathology guy.

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