

DENTO-MAXILLARY RADIOLOGY AND IMAGING

Course 5

RADIOIMAGING DIAGNOSIS OF MAXILLARY INFLAMMATORY LESIONS

5.1. ETHIOPATHOGENY

5.2. CLINICS

5.3. BIOCHEMISTRY

5.4. MORPHOPATHOLOGY

5.5. IMAGING DIAGNOSIS METHODS

5.6. PERIOSTITIS

5.7. OSTEITIS - OSTEOPERIOSTITIS

5.7.1. DENTO - ALVEOLAR OSTEOPERIOSTITIS (rarefying osteitis)

5.7.2. SYPHILITIC OSTEOPERIOSTITIS

5.7.3. TUBERCULOSIS OSTEOPERIOSTITIS

5.7.4. ACTINOMYCETES OSTEOPERIOSTITIS

5.8. ACUTE OSTEOMYELITIS

5.9. CHRONIC OSTEOMYELITIS

5.9.1. FOCAL CHRONIC SCLEROSING OSTEOMYELITIS

5.9.2. DIFFUSE CHRONIC SCLEROSING OSTEOMYELITIS

5.9.3. CHRONIC SCLEROSING GARRÉ'S OSTEOMYELITIS

5.10. ASEPTIC MAXILLARY OSTEONECROSIS

5.10.1. THERMIC AGENTS MAXILLARY OSTEONECROSIS

5.10.2. CHEMICAL AGENTS MAXILLARY OSTEONECROSIS

5.10.3. IONIZING PHYSICAL AGENTS MAXILLARY OSTEONECROSIS

5.1. ETHIOPATHOGENY

They are tissular pathologic lesions resulting after regional maxillary inflammatory process onset.

Define a stereotyped response, belonging to innate immunity mechanism, secondary to any injurious stimuli:

- Infection,**
- Chemical,**
- Physical,**
- Immune (antigen) reactions,**

being also accompanied by general manifestations.

5.2. CLINICS

They are characterized by symptoms and signs grouped in a clinico - morphological entity as inflammatory syndrome with different etiology but typical biology and imaging aspects.

Clinical are present the five inflammatory cardinal signs coming from Latin and described by Celsius: rubor (redness), tumor (swelling), calor (heat), dolor (pain) and functio laesa (loss of function).

5.3. BIOCHEMISTRY

Biochemical inflammatory reactions imply mediators releasing as: histamine, kynines, prostaglandins, and leukotriene which corollary primer miscellaneous immunological phenomena.

Biological appear increasing of inflammatory tests values: erythrocytes sedimentation rate, fibrinogen, reactive C protein, miscellaneous glycoproteins.

5.4. MORPHOPATHOLOGY

Any inflammation process will initially pass through the same next phenomena:

- vasomotories (local hyperemia, edema),
- cellular (leucocytes diapedesis, local cells mobilization and metamorphosis)
- tissular (proliferarea fibroblasts proliferation and healing in proliferative or productive inflammation).

Maxillary inflammatory processes will produce characteristic changes in conjunctive and vascular tissues from:

- osseous medullary spaces,
- Havers canals,
- periosteum,

osseous tissue indirectly, reactive, participate by local phenomena as:

- osteolysis,
- osteonecrosis
- osteosclerosis.

Lesional dynamics allow demarcation of three evolutive types of inflammation: acute, subacute and chronic.

Depending on osseous elementary structure localization exist some anatomo-clinic forms, named by adding specific suffix “-itis”:

- Periostitis - periosteal inflammatory process localization,
- Osteitis - osseous cortical inflammatory process localization,

- Osteomyelitis - all osseous structures are involved by inflammatory process.

5.5. IMAGING DIAGNOSIS METHODS

Topo - anatomically detect and localize the maxillary inflammatory changes being able to fine characterise the specific inflammatory lesions.

Every imaging diagnostic method has its anatomical regional exam indication being part in a rigorous quantified algorithm of investigation.

Also imaging offer a morphological picture of typical and atypical inflammatory lesions, with varying degree of sensitivity and specificity according to method, technique and evolutive moment.

ULTRASOUND EXAM

Early detect soft tissue oedema being the first of choice imaging exam to diagnostic assessment of exudative component into inflamed soft tissue.

It has a first position in the imaging diagnostic algorithm of osteomyelitis detection due to the ability of early identification of under periosteal fluid collection.

MRI

Detect the inflamed tissue oedema as a focal hyper T2, hypo T1 signal region with homogeneous or ring shape contrast uptake.

CT - SCAN

Detect the inflamed tissular lesions as unsharply defined nodules, with soft tissue density and ring shape contrast uptake.

RADIOGRAPHY

Detect typical aspects with:

- intralesional radiolucency
- sequestrum radioopacity
- secondary process of osteosclerosis with or without regional periostitis.

5.6. PERIOSTITIS

Maxillary periosteal inflammatory reaction with chronic non-suppurative evolution, more often appearing in children and adolescents, especially in mandible molars region, every active phase being followed by lamellar new periosteal bone formation.

ETHIOPATHOGENY

Resulting process due to periosteal irritation by miscellaneous causes:

- Internal:

- **dental infected follicle (eruption accident),**
- **rarefying osteitis - generic manifestation of apical chronic inflammation;**

- External:

- **suppurative otitis media,**
- **scalp infections determine regional cervical lymphadenopathies, near the mandible, which will irritate the neighboring periosteal;**

- Congenital

- **congenital syphilis**

ULTRASOUND EXAM

It has a first position in the early imaging diagnostic of periosteal reaction due to the ability of early identification of under periosteal fluid collection.

RADIOGRAPHY

Detect periosteal thickening by lamellar new periosteal bone deposition on vestibular face and on the inferior mandible margin.

When coexist with an apical infectious process radiographic exam identify a regional focal characteristic circumscribed radiolucent osteolysis.

5.7. OSTEITIS - OSTEOPERIOSTITIS

Is an inflammatory process that involves maxillary cortical bone, being usually associated by periosteal implying, resulting an anatomo-clinic process of osteoperiostitis.

It was described:

- **an acute suppurative form appeared as a complication of acute apical periodontitis or of pericoronitis in the rarefying osteitis;**

- a chronic or sclerozing form which follow an acute process characterized by draining extinction of acute activity without the cause removal.

RADIOGRAPHY

Detects a focal, apical, radiolucent osteolysis with diffuse limits in acute phase, which at the posterior maxillary level detach antral muco-periosteal due to secretion accumulation resulted from regional rarefying osteitis radiographic identified as a sclerotic marginal halo.

Also the involvement of neighboring regional sinus may determine a sinusitis.

CLINICO - ETHIOLOGICAL

Were identified the following forms of osteo-periostitis:

- Non-specific:

- dento-alveolar osteoperiostitis (rarefying osteitis)

- Specific:

- syphilitic osteo-periostitis;
- tuberculosis osteoperiostitis;
- Actinomyces osteoperiostitis.

5.7.1. DENTO-ALVEOLAR OSTEOPERIOSTITIS (rarefying osteitis)

Also named, Magitot disease is the result of alveolo-dental membrane inflammation secondary to a dental carious lesion.

Appear as a chronic inflammation form, having a common radiographic aspect of apical circumscribed osteolysis diffuse or net outlined, without differentiation possibilities between the determined morbid entities: apical infection, granuloma, chronic abscess, small apical cyst.

5.7.2. SYPHILITIC OSTEOPERIOSTITIS

Appear at the maxillary level in the tertiary stage as:

- circumscribed form – circumscribed syphiloma and
- diffuse form – diffuse syphiloma.

CIRCUMSCRIBED SYPHILOMA

It may be detected as two clinico - radiographic forms, both with preferential localization at the mandible angle: hyperostosis and goma.

SYPHILITIC HIPEROSTOSIS - appear as a regional demineralization of mandible angle associated with intense periosteal sclerozing osteogenesis which deforms the regional osseous contour.

SYPHILITIC GOMA with deep localization and net outlined homogenous osteolysis which lyses one of the compact and then funnel shape perforates it with skin exteriorization or mucosal at the retromolar trigon.

DIFFUSE SYPHILOMA

It may localize in the mandible body or maxillary incisors region appearing as typical osteo-periostitis with osteolysis, sequestrum and regional deformity.

In tertiary congenital syphilis appears Hutchinson clinic triad:

- incisor teeth specific malformed,
- keratitis,
- deafness.

Hutchinson teeth appear with a crescent deepening of the free margin with their superior part retraction, inferior axial convergent deviation of superior median incisors of the second dentition.

5.7.3. TUBERCULOSIS OSTEOPERIOSTITIS

Secondary tuberculosis maxillary involvement appearing in children and young people is predilected localized in mandible often by hematogenous dissemination and rarely by lymphatic or from a superficial regional tuberculous outbreak spread.

May appear as:

- **EXUDATIV-CAZEOUS** form with local osteolysis and fistulous drain with resulting cavern development;
- **PRODUCTIV-PROLIFERATIVE** form in which granulomatous type inflammatory material destroy the regional bone, resulting finally an inhomogeneous radiolucency, diffuse outlined, with or without central sequestrum.

5.7.4. ACTINOMYCETES OSTEOPERIOSTITIS

Is part of a cervico-facial actinomycosis form, infectious disease with chronic evolution caused by soft tissue penetration of actinomycetes, existing microorganism in oral cavity saprophytic microflora and organism defending granulomatous reaction to microbial invasion.

There were described at mandible level several rarefying and pseudoneoplastic clinico-radiologic forms with the identifying of diffuse radiolucent and cystic regional images.

5.8. ACUTE OSTEOMYELITIS

It is an extensive acute evolutive infectious process which involve all diametrical osseous structures: medulla, periosteum, cancellous and compact bone.

May be caused by:

- periapical abscess extension,
- post traumas or surgery,
- hematogenous spread - bacteremia.

RADIOGRAPHY

There is a clinico-radiologic parallelism which appear only after one week of evolution, needed time for sufficiently intense and extended osseous destruction by osteolysis and pus local accumulations.

Radiographic appears a diffuse defined radiolucent osteolysis which in time delimit a zone of necrotic radioopaque bone (sequestrum) usually associated with regional periosteal reaction.

5.9. CHRONIC OSTEOMYELITIS

May be:

- a sequelae of acute osteomyelitis, or
- a long-term, low-grade inflammatory reaction.

Appear in deficiency conditions (nutritional carency, immunologic depression, post radiotherapy) or in the presence of pre-existing systemic factors: Paget's disease, osteopetrosis, talasemia-sickle cell disease.

Radiographic appear as specific "moth-eaten bone" radiolucent osteolysis that may show focal zones of opacification (mottled aspect), with variable extension and without precise limits.

5.9.1. FOCAL CHRONIC SCLEROSING OSTEOMYELITIS

Is believed to represent a focal bony reaction to a low-grade inflammatory stimulus.

Appear as maxillary regional periapical masses of dense radiopaque osteocondensing tissue or as a radiopaque outbreak with periphery or central radiolucency.

It was also described as condensing osteitis, bony scar, sclerotic bone, and focal periapical osteopetrosis.

5.9.2. DIFFUSE CHRONIC SCLEROSING OSTEOMYELITIS

It is a maxillary inflammatory reaction, believed to be in response to a microorganism of low virulence in condition of an existent chronic periodontal disease.

Radiographic appear as a diffuse, ill-defined osteocondensing region, which may be associated with minimum radiolucencies and regional periosteal reaction.

5.9.3. CHRONIC SCLEROZING GARRÉ'S OSTEOMYELITIS

Or chronic osteomyelitis with proliferative periostitis it is a chronic osteomyelitis form in which additionally appears a prominent periosteal inflammatory reaction.

May be caused by: post extraction local infections, eruptive accidents infections or periapical abscess of mandible molars.

Radiographic appear an "moth-eaten bone" type osteolysis outbreak as a mottled predominantly lucent lesion with periosteal trabecular reaction perpendicular on bone cortical.

5.10. ASEPTIC MAXILLARY OSTEONECROSIS

Are osseous destruction processes secondary to direct or vascular intermediated action of miscellaneous iatrogenic or surrounding environment agents, grouped depending to their specific mode of action in:

- thermic,
- chemical,
- ionizing physical.

5.10.1. THERMIC AGENTS MAXILLARY OSTEONECROSIS

Appear after electro cautery tumoral ablation, because resection in oncological safety limits implies regional normal tissue involvement.

Manifest as marginal inhomogeneous osteolysis outbreaks, diffuse outlined, with internal sequestrum, following the anatomic excision trajectory.

5.10.2. CHEMICAL AGENTS MAXILLARY OSTEONECROSIS

Results through several mechanisms:

- direct osseous contact, by regional diffusion of arsenical substance used for dental pulp dieback or by a misapplication with an improperly dressing (may lead to dento-alveolar osteolysis with or without internal sequestrum);**
- indirect osseous contact by salivary elimination in intoxication with: hydrargyrum, phosphorus or bismuth, with initial apparition of an ulceronecrotic gingivitis, osseous lesions appearing latter during evolution of the disease.**

5.10.3. IONIZING PHYSICAL AGENTS MAXILLARY OSTEONECROSIS

Also named, osteoradionecrosis is secondary oro-maxillo-facial malignant tumors radiotherapy being one of the most serious complications.

Repeated ionizing radiations absorption, decrease the organism defending capacity, with miscellaneous metabolic interfering which break the bone normal physiological balance.

Initially, the radiographic aspect is normal, then, in a shorter or longer time (months - years - tens of years), depending of precocious or tardive clinic form of osteoradionecrosis, appear, usually after a minor local trauma, the typical image of inhomogeneous osteodestruction: diffuse outlined and with internal sequestrum.