Bacterial Infections

Noma (Cancrum Oris)

NOMA means to devour (a spreading sore). It is a rapidly spreading mutilating, gangrenous stomatitis that occurs usually in debilitated or nutritionally deficient persons.

SYNONYMS- Cancrum oris; Orofacial Gangrene; Gangrenous Stomatitis ; Necrotizing Stomatitis.

It is a rapidly progressive , polymicrobial, opportunistic infection caused by components of the normal oral flora that become pathogenic during periods of compromised immune status.

CAUSATIVE ORGANISMS - Fusobacterium Necrophorum. Prevotella intermedia.

PREDISPOSING FACTORS

- Poverty
- Malnutrition or Dehydration
- Poor Oral hygiene
- Unsafe drinking water
- Proximity to unkempt livestock
- Recent illness
- Malignancy
- An immunodeficiency disorder ,including AIDS.

CLINICAL FEATURES

Seen in Children's of age 1-10 years, in Adults with major debilitating diseases(eg-diabetes mellitus,leukemia,lymphoma,HIV infection)

Begins as a small ulcer of the gingival mucosa which rapidly spreads and involves the surrounding tissues of jaw, lips and cheeks by gangrenous necrosis.

Initial site is commonly an area of stagnation around a fixed bridge or crown.

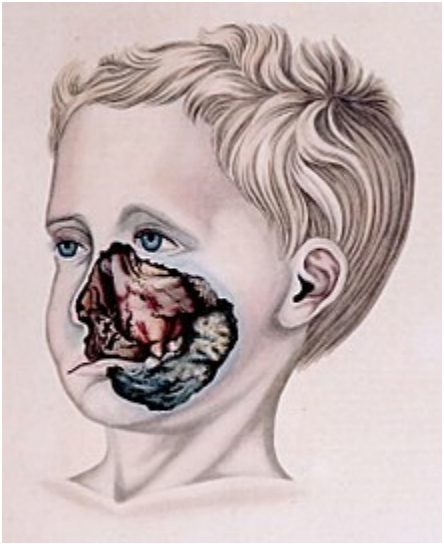
The overlying skin becomes inflamed ,edematous , and finally Necrotic, With the result that a line of demarcation develop between healthy and dead tissues, and large mass of tissues may slough out, leaving the jaw exposed.

- **NOTE:** Gangrene is denoted by appearance of blackening of the skin .

- Subcutaneous fat pad and buccal fat pad undergo necrosis in advance of other adjoining tissues.
- Foul Odor from gangrenous tissue.
- Palate and Tongue may get involved. Patient with 1- increase in temperature . 2-Suffers secondary infection. 3-may die from toxemia or pneumonia.
- **NOMA PUDENDI**- This condition can also Cause tissue damage to the genitals.
- **NOMA NEONATORUM** is a rare gangrenous form of noma, which during its course, causes progressive and mutilating destruction of the soft tissues and the bone. It occurs in newborns at birth or during the first month of life.

TREATMENT AND PROGNOSIS

1. Mortality rate- 75% before the availability of antibiotics.
2. “Penicillin” and “Metronidazole” are the first line therapeutic antibiotics for Necrotizing Stomatitis.
3. Since therapy is directed against the pseudomonas organisms and often consists of Piperacillin, Gentamicin or Clindamycin.
4. Surgical excision of gross necrotic areas is recommended , but aggressive removal contraindicated to stop the extension of the process and create reconstruction process.
5. Necrotic bone is left in ace to help hold the facial form but is removed as it sequestrates. Reconstruction should be delayed by one year to ensure complete surgery.
6. Parenteral fluid should be given to correct dehydration & electrolytic balance.



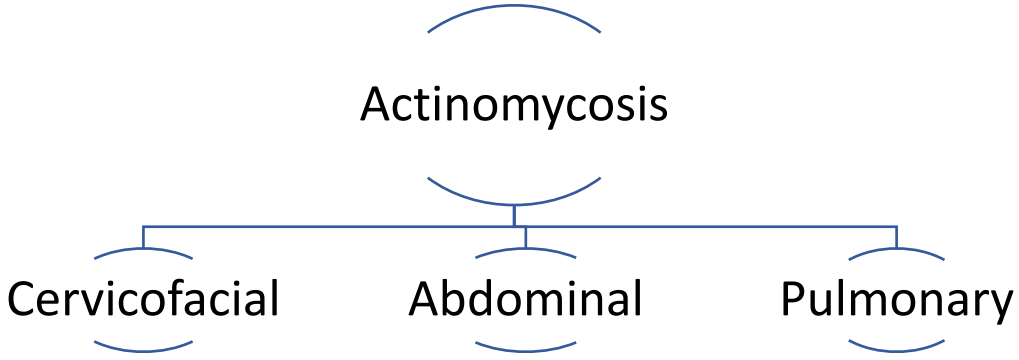
Actinomycosis

Actinomycetes : Gram-positive, pleomorphic non–spore- forming, non–acid-fast anaerobic or Microaerophilic bacilli of the genus Actinomycetes and the order Actinomycetales cause actinomycosis. Actinomyces are very closely related to Nocardia species; both were once considered to be fungal organisms.

Actinomyces is a gram positive, non-spore- forming anaerobic or microaerophilic bacterial rod . Actinomyces israelii causes most Actinomyces infections in humans, although other forms such as Actinomyces Odontolyticus, Actinomyces Viscosus, Actinomyces Meyeri, Actinomyces Gerencseriae, and Propionibacterium Propionicum have also been reported. Actinomyces infections are commonly polymicrobial .

Actinomycosis :

1. It is a chronic granulomatous suppurative and fibrosing disease caused by anaerobic or microaerophilic gram-positive nonacid fast, branched filamentous bacteria.
2. Most of the species isolated from actinomycotic lesions have been identified as A. israelii, A. viscosus, A. odontolyticus, A.naeslundii or A. meyeri.
3. These microorganisms have been identified in dental plaque, dental calculus, necrotic pulp, and tonsils.
4. The usual pattern of this disease is one characterized chiefly by the formation of abscesses that tend to drain by the formation of sinus tracts.



Clinical Features of Cervicofacial Actinomycosis:

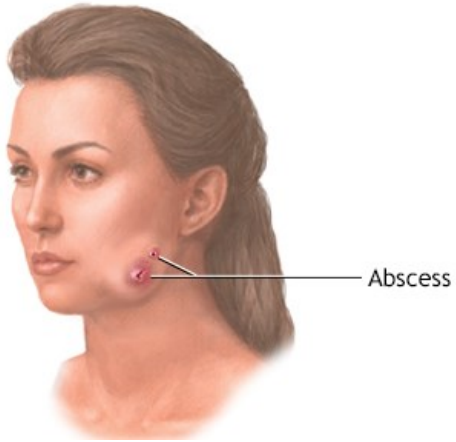
- Most common type seen in adult males
- Submandibular region is the most affected site
- Trismus is the most common feature
- First Sign: Formation of a palpable mass which is painless & indurated
- Non-healing tooth socket, exuberant tissue
- Periosteal thickening of alveolus
- Development of fistula is a common feature
- Disfigurement of face
- Infections involving maxilla & mandible
- Painful lesions on tongue

Radiographical Features of Cervicofacial Actinomycosis:

- Areas of bone destruction with well defines areas of radiolucencies with cortical lining of dense bone.
- Lamina Dura is deficient at the apex of the tooth
- Scattered area of bone destruction separated by normal / sclerosed bone.

Management of Cervicofacial Actinomycosis:

- Two Fold Therapy: Antibiotics & Surgery
- Lesion is removed surgically through debridement
- Penicillin should be given i.e 3-4 million IV, 4 hours till 2- 4 weeks
- If the patient is allergic to penicillin, tetracycline orally 500 mg QDS for 6 months is given



Syphillis

Syphilis is a systemic, sexually transmitted disease (STD) caused by the Treponema pallidum bacterium. The three means of syphilis transmission are:

•Person to person via vaginal, anal, or oral sex through direct contact with a syphilis chancre.
•Person to person during foreplay, even when there is no penetrative sex (much less common).
•Pregnant mother with syphilis to fetus.

PRIMARY SYPHILIS

• Starts as painless, erythematous indurated papule – ulcer

• Lymphadenopathy

• Usually single , painless, clean, well defined, Nontender & markedly indurated • Inguinal lymph node invariably involved

• Site - genital, perineal or anal area; however, any part of the body may be affected

• Multiple, non tender, discrete, rubbery , Usually Bilateral

• Syphilis d emblee- when primary chancre may be overlooked or concealed

• Phagedena – invasion of ulcer with Vincent organisms may result in gangrenous changes

• Heal spontaneously leaving behind tissue paper scar Regardless of stage of disease and location of lesions, histopathologic hallmarks of syphilis include endarteritis (which in some instances may be obliterative in nature) and a plasma cell–rich infiltrate.

Chancre : A chancre is a painless genital ulcer most formed during the primary stage of syphilis. This infectious lesion forms approximately 21 days after the initial exposure to Treponema pallidum, the gram-negative spirochaete bacterium yielding syphilis. Chancres transmit the sexually transmissible disease of syphilis through direct physical contact. These ulcers usually form on or around the anus, mouth, penis, and vagina. Chancres may diminish between four and eight weeks without the application of medication.

Chancres, as well as being painless ulcerations formed during the primary stage of syphilis, are associated with the African trypanosomiasis sleeping sickness, surrounding the area of the tsetse fly bite.

SECONDARY SYPHILIS

• 2-12 weeks of development of primary syphilis – pt. manifests with symptoms of secondary syphilis

• Characterized by low-grade fever, malaise, sore throat, headache, adenopathy and cutaneous or mucosal rash.

• Manifest as- Evanescent copper-colored macular rash

• A few days later, symmetric papular eruption appears, involving entire trunk and the extremities, palm, and soles

• Papules - reddish brown,scaly,discrete,0.5–2 cm in diameter.

• Variants - pustular, combination of these , lichenoid , acneiform nodular ,circinate,corymbose,annular

• 25% of pts have abnormal CSF

Condyloma lata • Reddish-brown papular lesions on the penis or anogenital area can coalesce into large, elevated plaques up to 2-3 cm in diameter, known as condylomata lata, a highly infectious lesion • Lesions usually progress from red, painful, and vesicular to “gun metal grey” as the rash resolves.

Latent Syphilis • The latent (hidden) stage of syphilis begins when primary and secondary symptoms disappear • Without treatment, the infected person will continue to have syphilis infection in their body even though there are no signs or symptoms and are contagious

TERTIARY SYPHILIS

• Morbidity and mortality of syphilis in adults in past years were due to late manifestations of illness • There may be an interval of 1 - 20 years from acute infection to clinical onset of late or tertiary stages of disease • Tertiary syphilis conveniently divided into three main groups Late benign syphilis Cardiovascular syphilis Neurosyphilis

BENIGN TERTIARY SYPHILIS - Characterized by gumma & appear 3-10 years after infection - proliferative granulomatous inflammatory process causing destructive of affected tissues - Most lesions occur in skin and bones, with lesser frequency in mucosa and of viscera muscles and ocular structures Theory: 1. Best evidence in support of hypersensitivity was provided by Magnuson et al. who inoculated volunteers in Sing Sing Prison with the Nichols strain of T. Pallidum . Gummas developed only in persons with a history of previous syphilis .They concluded that superinfection in sensitized patient may explain gumma formation.

CARDIOVASCULAR SYPHILIS • Clinically manifest after latent period of 15–30 years Pathology • Spirochetes appear to have predilection for vasa vasorum of aorta particularly the proximal aorta • Produce transmural inflammatory lesions - endarteritis of these vessels • Obliteration of lumen of vasa vasorum the aortic media develops patchy necrosis with subsequent focal scarring.

NEUROSYPHILIS

• Abnormalities in CSF have been noted in 13% of pts with untreated primary syphilis 25–40% of pts with untreated secondary syphilis • After initial invasion of CNS during early syphilis, untreated infection may resolve spontaneously persist as asymptomatic syphilitic meningitis progress to symptomatic acute syphilitic meningitis progression of early asymptomatic or symptomatic meningeal infection may lead to meningovascular syphilis (usually 5–12 yrs after primary infection) or parenchymatous forms of neurosyphilis such as tabes or paresis (usually 18–25 yrs).

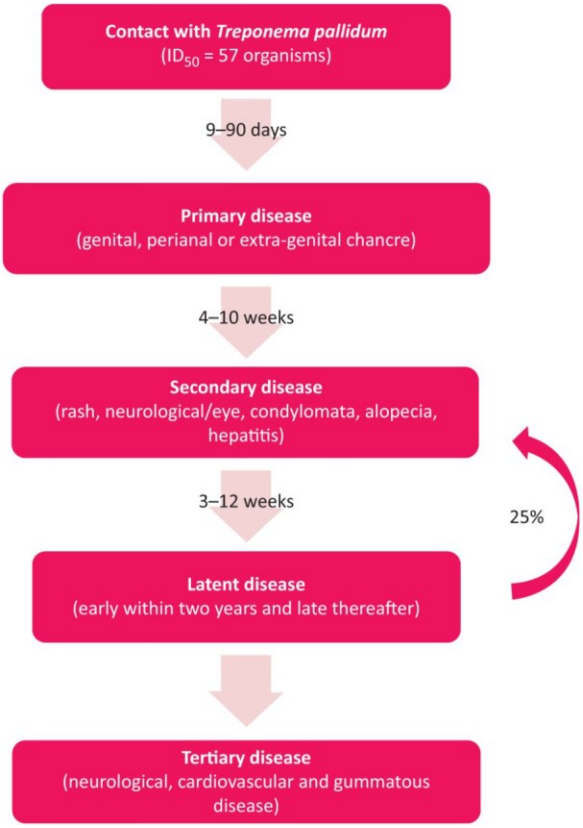
Diagnosis of Syphilis

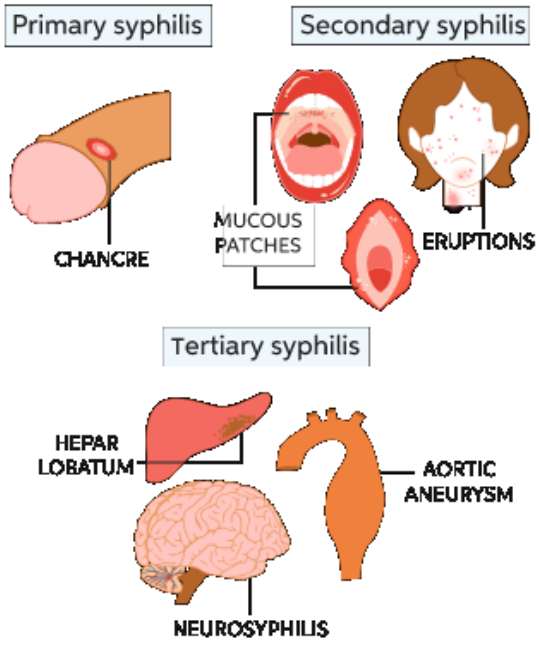
Blood tests: A. **Non-treponemal tests** Like, Venereal Disease Research Laboratory (VDRL) test; Usually positive in early disease, but may be negative in advanced disease, occasionally false positive, confirmation required with Treponemal test B.

Treponemal tests First tests to become positive and are useful for screening Treponemal antibody tests usually become positive 2 to 5 weeks after the initial infection and remain positive indefinitely Cerebrospinal Fluid Examination: Neurosyphilis is diagnosed by finding high numbers of lymphocytes and high protein levels in the CSF Direct testing of serous fluid from a chancre by: Dark ground microscopy or Direct fluorescent antibody testing.

Treatment of Syphillis : There are no home remedies or over-the-counter drugs that will cure syphilis, but syphilis is easy to cure in its early stages. A single intramuscular injection of long

acting Benzathine penicillin G (2.4 million units administered intramuscularly) will cure a person who has primary, secondary, or early latent syphilis.





Tuberculosis

Tuberculosis (TB) is a potentially fatal contagious disease that can affect almost any part of the body but is mainly an infection of the lungs.

Causative Organisms - Mycobacterium tuberculosis (Human) & Mycobacterium Bovis (Animals)

Other causative organisms □ Mycobacterium africanum □ Mycobacterium microti Non-Mycobacterium Genus □ Mycobacterium leprae □ Mycobacterium avium □ Mycobacterium asiaticum M. africanum M. Bovis M. Canetti M. microti M. tuberculosis complex

- Discovered in 1882 by Robert Koch.

Classification

Pulmonary TB - Primary Disease - Secondary Disease Extra pulmonary i. Lymph node TB ii. Pleural TB iii. TB of upper airways iv. Skeletal TB v. Genitourinary TB vi. Miliary TB vii. Pericardial TB viii. Gastrointestinal TB ix. Tuberculous Meningitis x. Less common forms

Primary Tuberculosis :-

- The infection of an individual who has not been previously infected or immunised is called Primary tuberculosis or **Ghon's complex** or **childhood tuberculosis**.
- Lesions forming after infection is peripheral and accompanied by hilar which may not be detectable on chest radiography.

Secondary Tuberculosis : The infection that individual who has been previously infected or sensitized is called secondary or post primary or reinfection or chronic tuberculosis.

Extra Pulmonary TB :- • 20% of patients of TB Patient • Affected sites in body are :-

1) **Lymph node TB (tuberculuous lymphadenitis):-** • Seen frequently in HIV infected patients. • Symptoms :- Painless swelling of lymph nodes most commonly at cervical and Supraclavical (Scrofula) • Systemic systems are limited to HIV infected patients.

• 2) **Pleural TB** :- Involvement of pleura is common in Primary TB and results from penetration of tubercle bacilli into pleural space.

Symptoms :- Dysphagia, chronic productive cough

3) **TB of Upper airways**

4) **Genitourinary TB** :- • 15% of all Extra pulmonary cases. • Any part of the genitourinary tract get infected. • Symptoms :- Urinary frequency, Dysuria, Hematuria.

5) **Skeletal TB** :- • Involvement of weight bearing parts like spine, hip, knee. • Symptoms :- Pain in hip joints n knees, swelling of knees, trauma.

6) **Gastrointestinal TB** :- • Involvement of any part of GI Tract. • Symptoms :- Abdominal pain, diarrhea, weight loss

TB Meningitis & Tuberculoma :- 5% of All Extra pulmonary TB -Results from Hematogenous spead of 10 & 20 TB. 8) TB Pericardiatis :- • 1- 8% of All Extra pulmonary TB cases. • Spreads mainly in mediastinal or hilar nodes or from lungs. 9) Miliary or disseminated TB :- • Results from Hematogenous spread of Tubercle Bacilli. • Spread is due to entry of infection into pulmonary vein producing lesions in different extra pulmonary sites. 10) Less common Extra Pulmonary TB • uveitis, panophthalmitis, painfull Hypersensitivity related phlyctenular conjuctivis.

Diagnosis

1.**Bacteriological test**: a. Zeihl-Neelsen stain b. Auramine stain(fluorescence microscopy)

2. **Sputum culture test**: a. Lowenstein –Jensen(LJ) solid medium: 4-18 weeks b. Liquid medium : 8-14 days c. Agar medium : 7 to 14 days

3.**Radiography**: Chest X-Ray(CXR) 4.Nucleic acid amplification: Species identification ; several hours Low sensitivity, high cost ,Most useful for the rapid confirmation of tuberculosis in persons with AFB-positive sputa, Utility AFB-negative pulmonary tuberculosis .Extra pulmonary tuberculosis

4.**Tuberculin skin test (PPD)** : Injection of fluid into the skin of the lower arm. 48-72 hours later – checked for a reaction. Diagnosis is based on the size of the wheal. 1 dose = 0.1 ml contains 0.04µg Tuberculin PPD.

Preventive measures

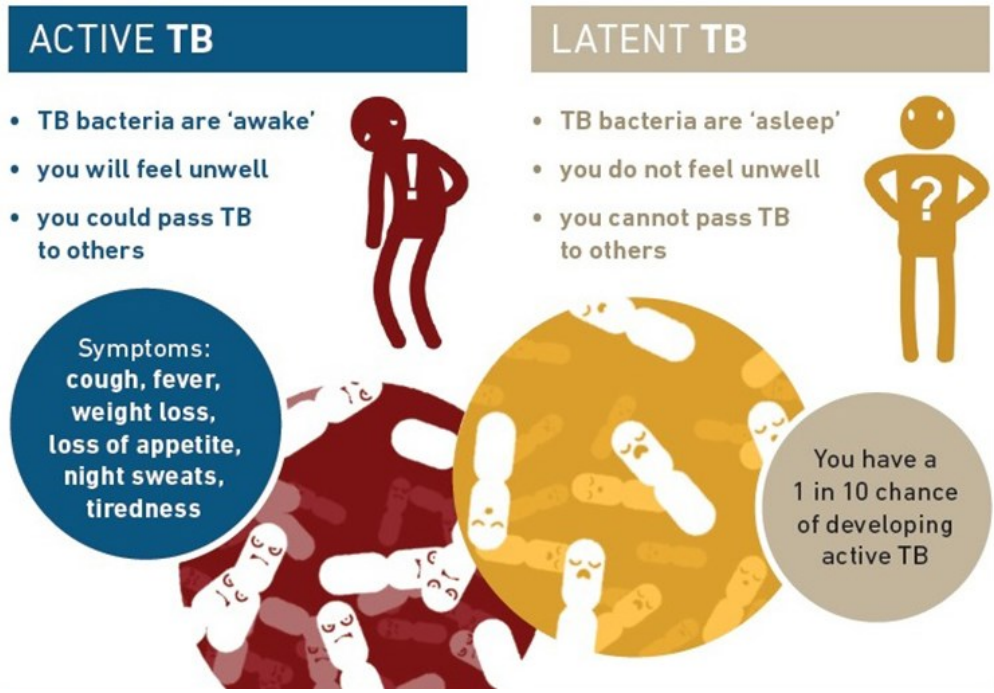
1) Mask 2) BCG vaccine 3) Regular medical follow up 4) Isolation of Patient 5) Ventilation 6) Natural sunlight 7) UV germicidal irradiation

BCG vaccine - Bacille Calmette Guerin (BCG). First used in 1921. Only vaccine available today for protection against tuberculosis. It is most effective in protecting children from the disease. Given 0.1 ml intradermally. Duration of Protection 15 to 20 years. Efficacy 0 to 80%. Should be given to all healthy infants as soon as possible after birth unless the child presented with symptomatic HIV infection.

Treatment regimen according to WHO

ISONIAZID (H) RIFAMPICIN (R) PYRAZINAMIDE (Z) ETHAMBUTOL (E) STREPTOMYCIN (S)

DOTS - Directly observed treatment, short-course DOT means that a trained health care worker or other designated individual provides the prescribed TB drugs and watches the patient swallow every dose.



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