CUTANEOUS TUBERCULOSIS

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Introduction

- Cutaneous TB caused by M. Tuberculosis and rarely M. Bovis.
- Mycobacteria is aerobic, non motile, non- sporing forming, acid fast bacilli
- Acid fastness is due to mycolic acid .
- Cell wall contain arabinogalactan, mycolic acid, peptidoglycan.

Epidemiology

- Total number of cases in 2016 is 1.4 million
- 90% cases are in India, South America, Philippines, Brazil, Africa
- At a time 1billion people carry M. tuberculosis bacteria.
- 5-10% develop overt TB and out of these 3.4% are MDR and 5 lakh people are XDR.
- Cutaneous TB is 0.1% of overall TB.
- 13% pts. are associated with HIV

Immunology

- Protective immunity involves T-lymphocytes
- After first infection organism is recognized by toll like receptors.
- Mycobacteria is phagocytosed by macrophages with help of TLR.
- More macrophages are attracted along with neutrophils and monocytes.
- Later on activate T lymphocytes.
- Main cytokine are TNF ,interferon gamma and IL12.
- Delayed Hypersensitivity and protective immunity develops.

Predisposing Factor

- Steroid therapy
- Nutritional Deficiency
- Chemotherapy
- Associated with HIV

Diagnostic test

1)Tuberculin test using Inj. PPD (I.D): 0.1ml is injected which measures 5 tuberculin units.

- Induration is measured after 48hrs.
- 5mm induration in high risk patient.
- 10mm induration in endemic areas
- 15mm people with no risk factor
- False positive with other Mycobacteria disease.

Other tests:

2) Interferon gamma test

(Gold standard: Quantiferon Tb in spot/ tube test.)

3)PCR test: DNA amplification test

4) Gene Xpert test for Rifampicin resistance.

Classification

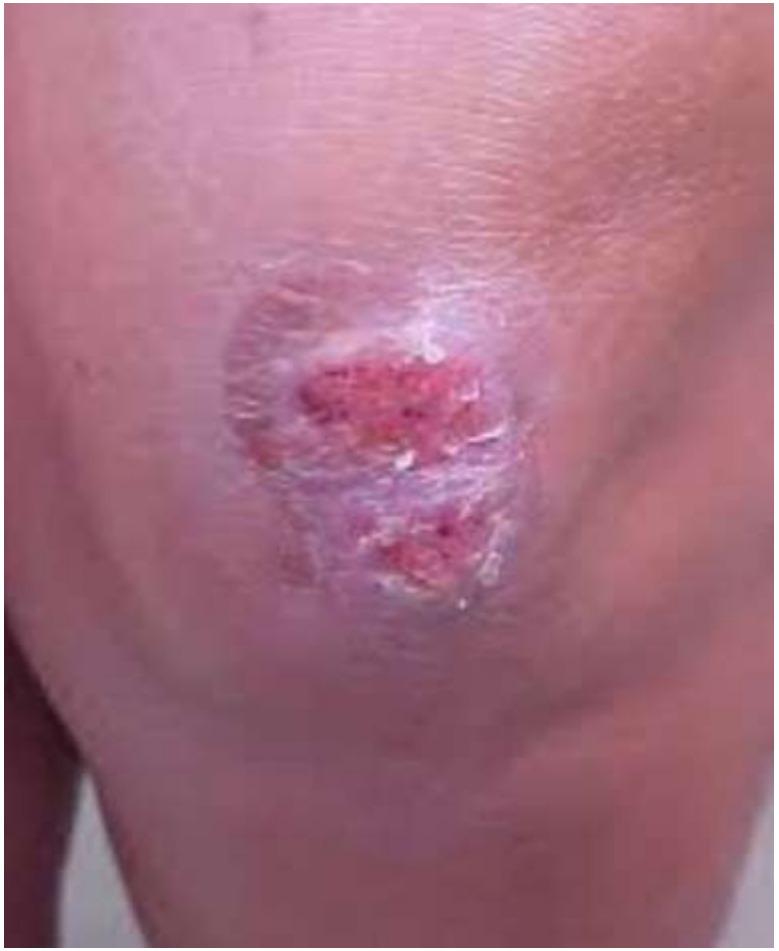
A) Multibaciliary with low resistance

- Tuberculous chancre
- Scrofuloderma
- Tuberculous gumma
- Miliary TB
- Orofacial TB

B) Paucibacillary with high resistance

- TBVC
- Lupus Vulgaris
- Tuberculids

Tuberculous Chancre

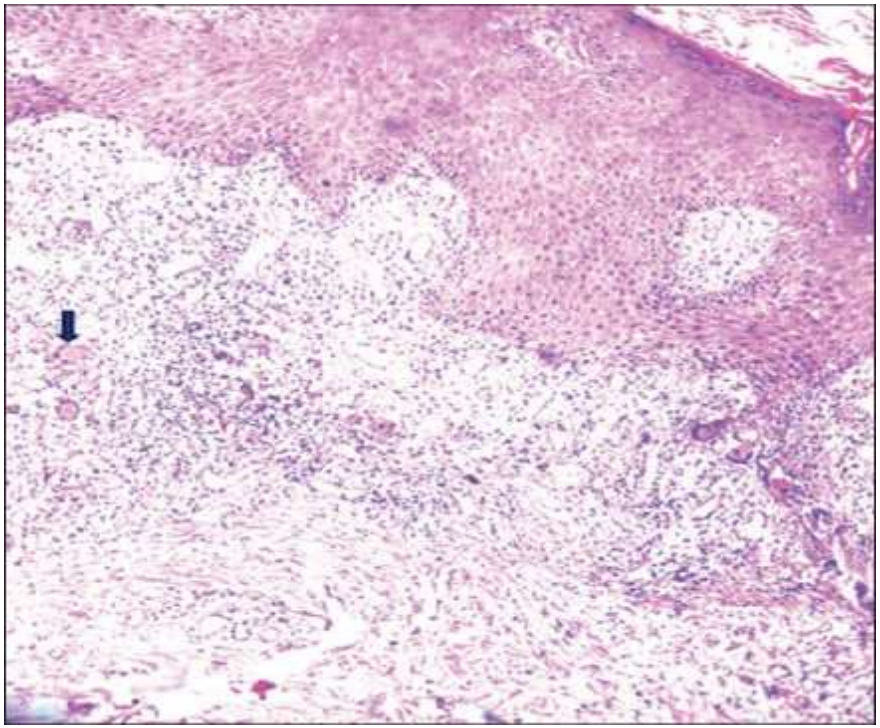


Primary TB

- Primary inoculation of Mycobacteria in skin or mucosa.
- No natural immunity.
- Commonly affects health worker and children.
- Enter through skin abrasion.
- Sites are buttocks, hands.

Histopathology

- Acute neutrophic infiltrate ,
- Multiple AFB seen,
- After few weeks caseation and granuloma formation occurs



Risk factors are:

- Children
- Over crowding
- Positive open cavitory patient in family.
- Low socio- economic status

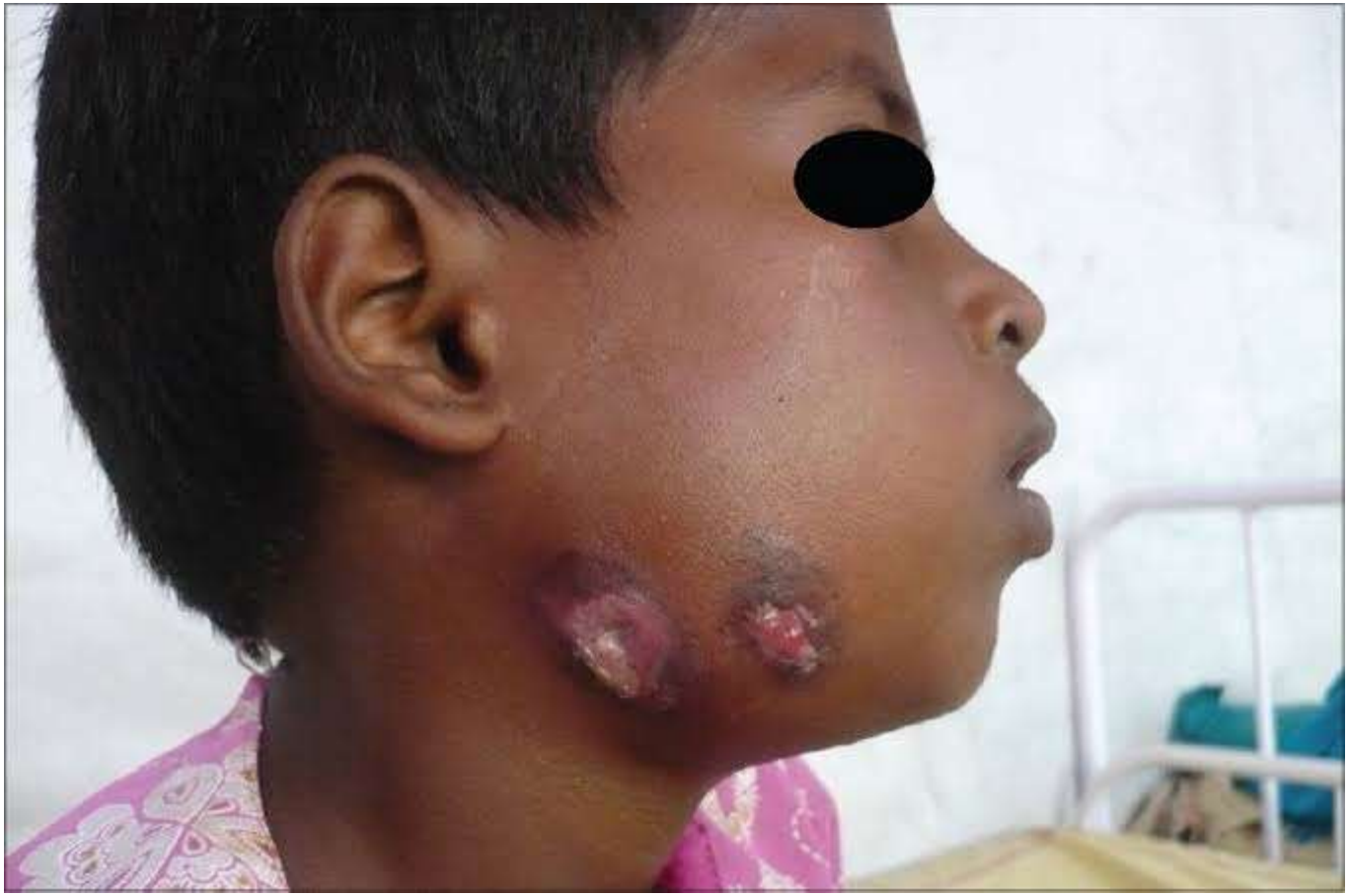
Clinical features:

- Small brownish papule which may ulcerate to form undermined edges, granulomatous base, and adherent crusts.
- Regional lymphadenopathy after 4-8 weeks
- Ghons focus is formed
- Many times heals on there own.
- In patients with low immunity org. may disseminate to form miliary TB.

Course and prognosis:

- Occ. may progress into Lupus Vulgaris and Scrofuloderma.
- Tuberculin test negative in beginning later on may turns positive.

Scrofuloderma

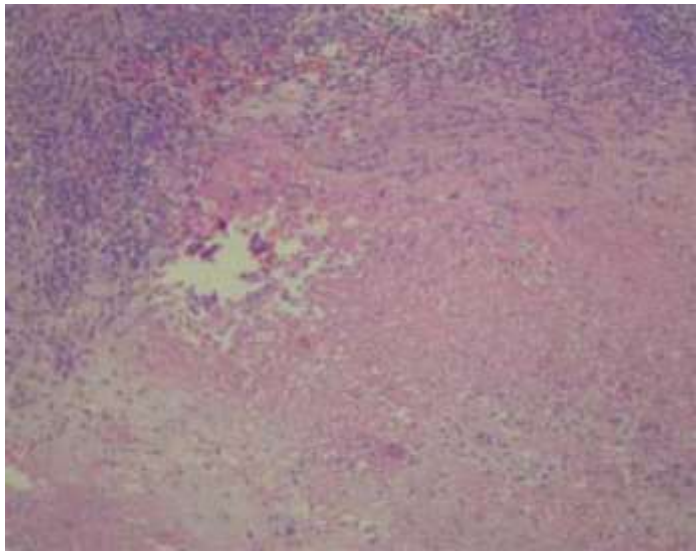


Scrofuloderma

- Spread: Joint, bone, subcutaneous nodules are involved.
- Involvement of skin is secondary
- Commonest site: cervical and retro auricular lymph nodes are involved.
- Age: childhood, adolescent and old age.
- Males more common than females.
- HIV is more common.

Histopathology:

- Ulceration of skin, dermal abscess, Epitheloid or macrophages can be seen and sometimes caseous necrosis is present.



Clinical features:

- Protracts sinuses, non healing ulcers which heals with scaring.
- Liquefactive necrosis matted together that break down to form sinuses which discharge necrotic material and sometimes leads to form ulcers.
- Ulcers have undermined edges.
- Heals with forming puckered scars.

Differential diagnosis:

- Lymphoma
- Sporotrichosis
- Hidradenitis suppurativa.
- Deep fungal infections

Investigation:

- Biopsy
- Tuberculin test
- Tuberculous bacilli in culture

Orofacial TB

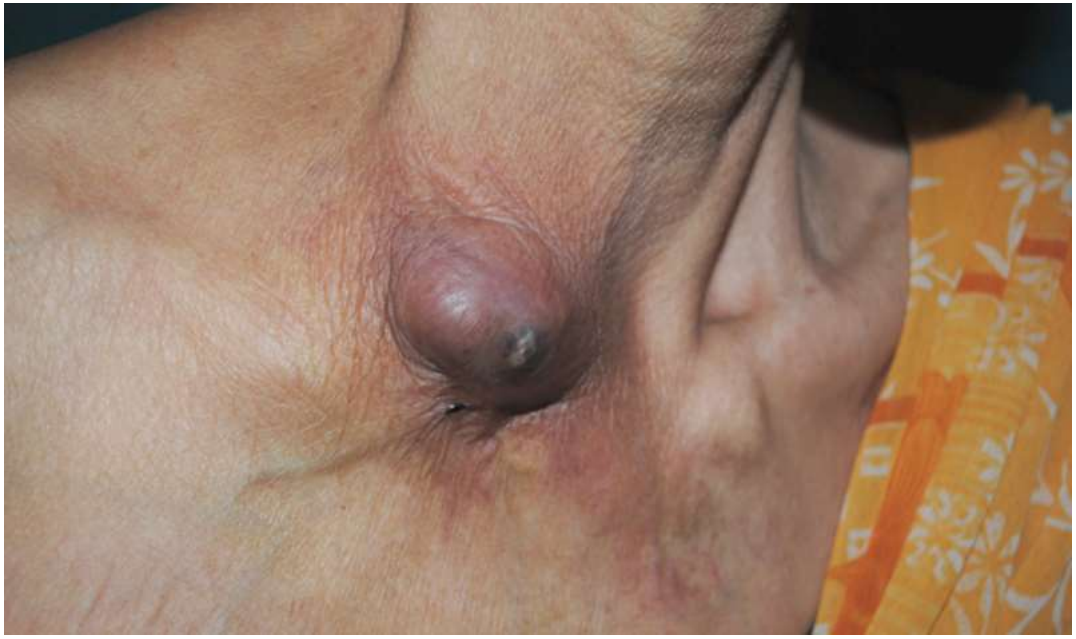
- Characterized by tubercular infection of mucosa or adjoining orifices.
- Most lesion are produced by autoinoculation
- Mainly hematogeous spread
- More common in elderly
- Males more than females.
- Malnutrition, immunosuppresion, are predisposing factors.
- Sites : most common is side and tip of tongue, soft or hard palate, buccal mucosa, or genitals.

- Histopath:
- Non specific inflammation in upper dermis, deep dermis pronounced necrosis with AFB.
- Clinical features :
- Small nodule with rapidly breaks to form ulcer with necrotic base and surrounded by Erythema.
- Ulcer are painful.
- Resistance power is low.
- Prognosis is poor.

- Differential diagnosis:
- Apthous ulcer
- Bechets disease
- Crohns diseases

- Investigations
- Presence of AFB on biopsy culture and pcr
- HIV and malnutrition should be ruled out
- Tuberculin test is negative

Tuberculous Gumma



Tuberculous Gumma

- Due to hematological spread of bacteria from primary lesion.
- Seen in Immuno-compromised, malnourished, individuals.
- Single or multiple lesion children.

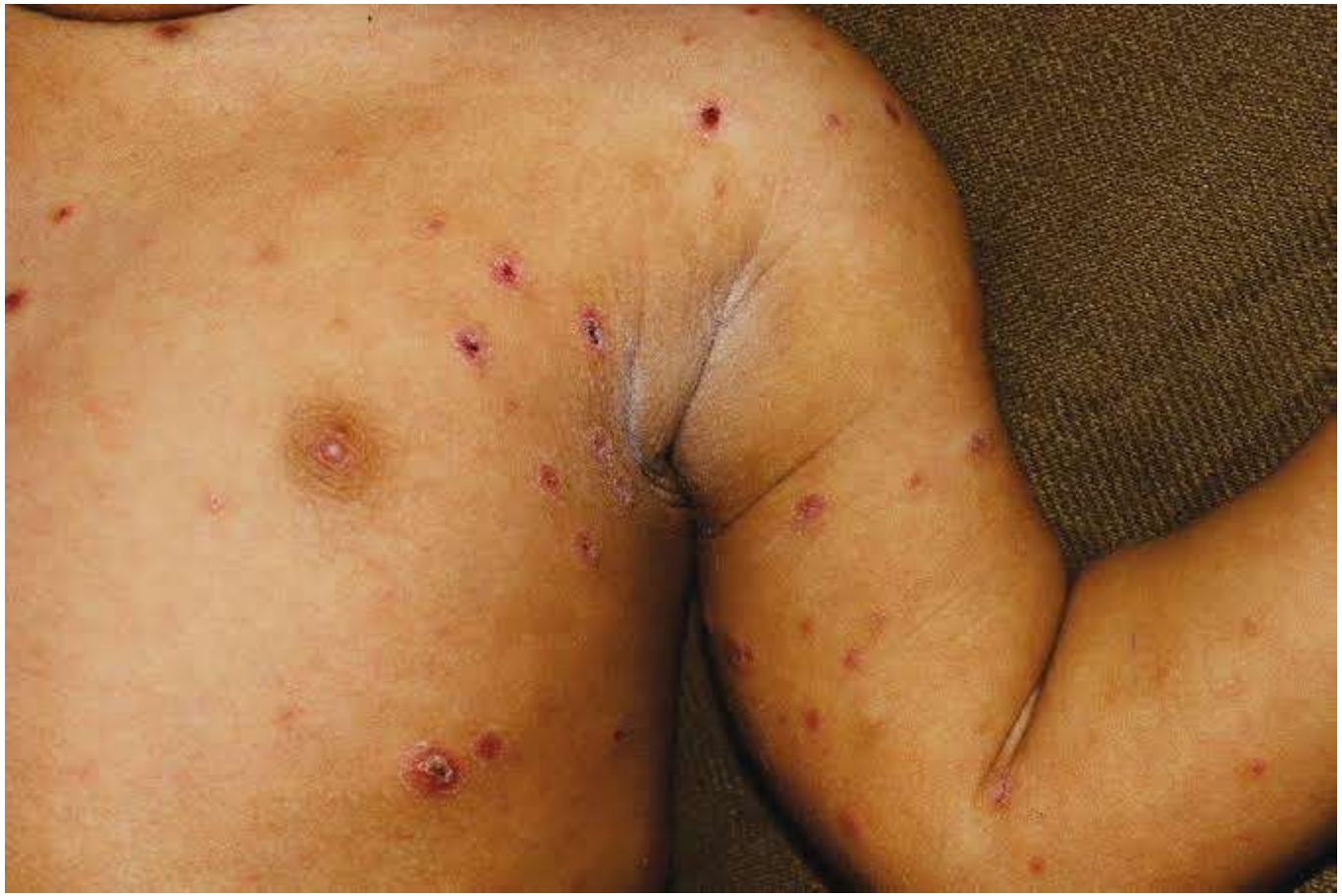
- Histopathology: Massive necrosis with large number of AFB

- Clinical features:
- Multiple tender nodules,
- Overlying skin breaks down forming abscess, sinuses and fistula,
- Prognosis is bad.

Investigation:

- Biopsy
- Tuberculin test can be negative
- Culture is positive
- PCR.

Miliary TB



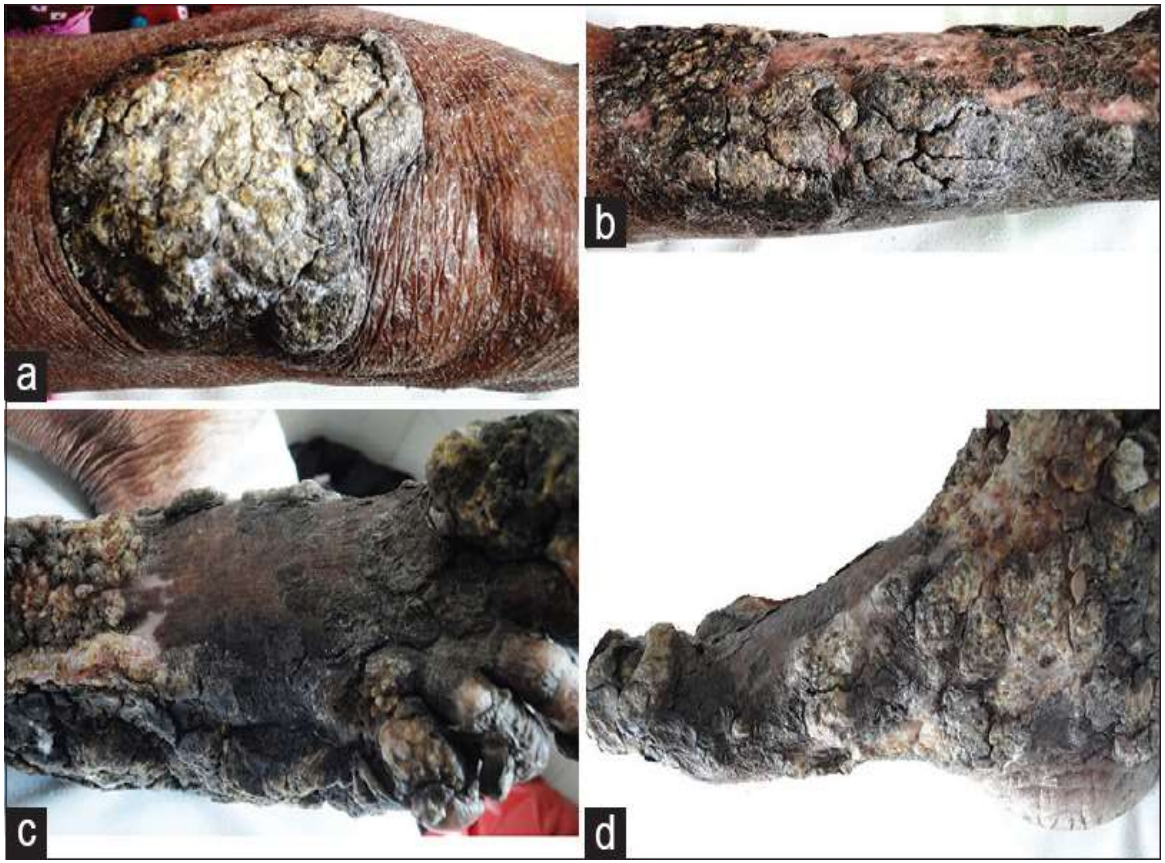
Miliary TB

- It is rare variant, seen with pulmonary disseminated and infectious tuberculosis.
- Seen in young children mainly after measles, malnutrition.
- In adults with HIV infection.
- Histopath: Multiple small abscess with AFB as well as surrounding dermis

- Clinical features:
- Trunk and proximal extremities are involved.
- Bluish vesicles, papules, hemorrhagic lesions which become superficial ulcers.
- Prognosis is poor may respond to treatment.

- Investigation:
- Biopsy
- Culture
- PCR

TBVC



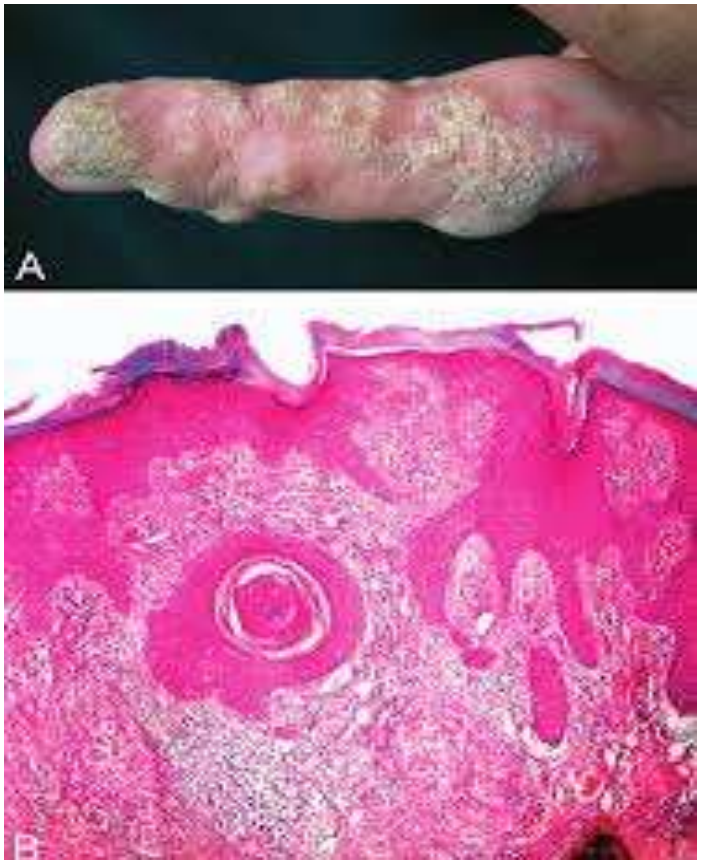
TBVC

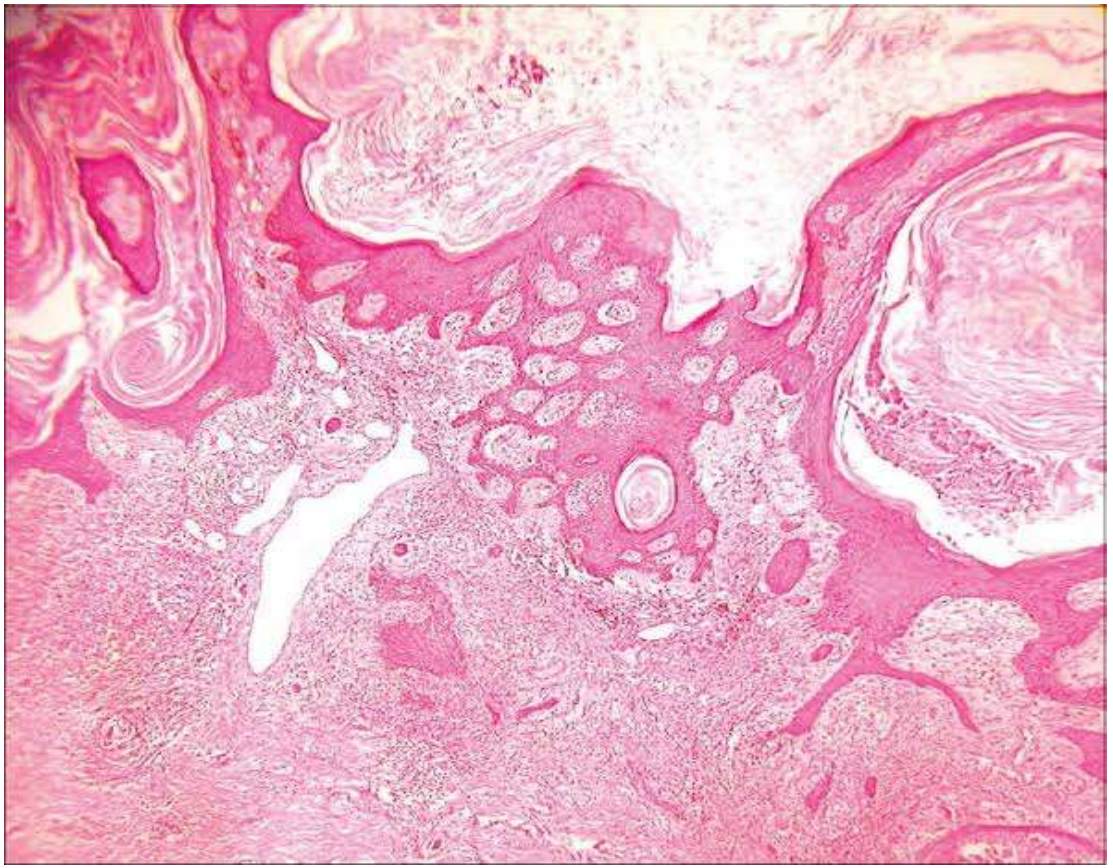
- Caused by exogenous inoculation of M. Tuberculosis in previously infected and sensitized patients.
- Common in healthcare workers and children in endemic area.
- Males more common than females.
- Young male who are previously sensitized are commonly affected.
- India and china

- Histopathology: Pseudoepitheliomatous hyperplasia, neutrophilic infiltrate in superficial dermis is seen and AFB is difficult to detect.

- Sites: Those exposed to trauma and infected sputum. Commonly seen over hands, knees, ankle, buttocks.

Histopathology : TBVC





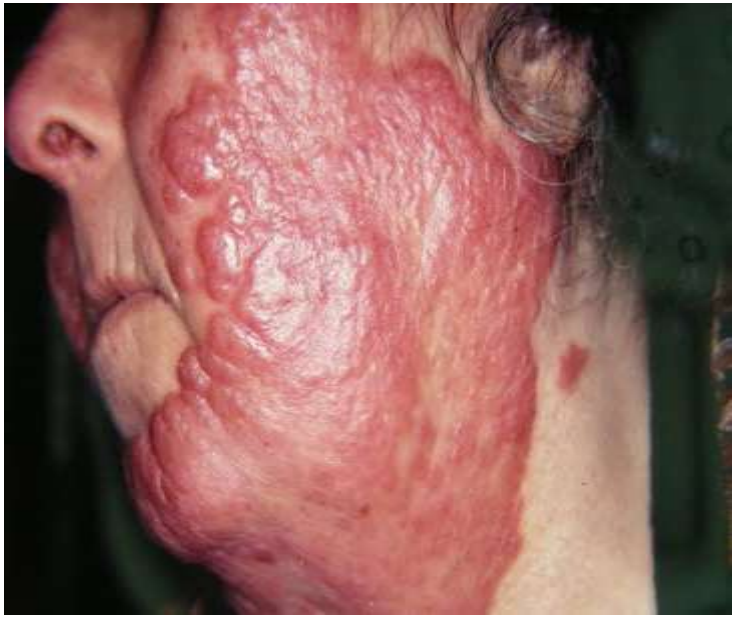
Clinical features:

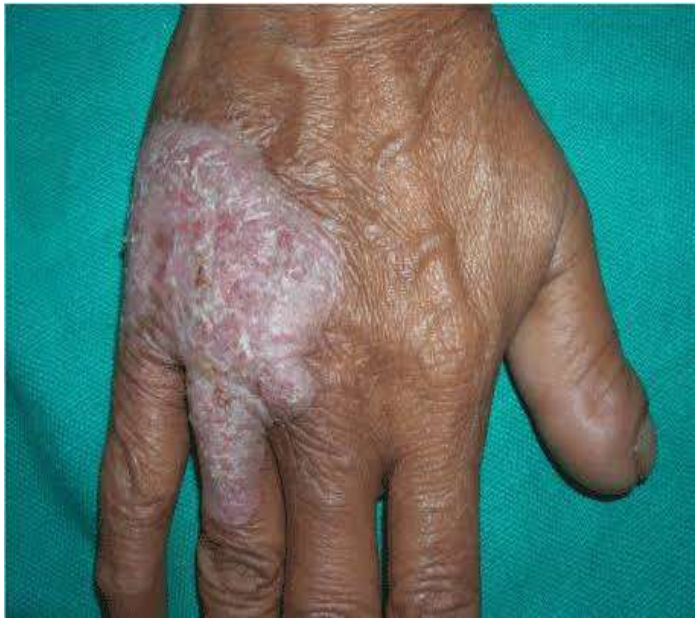
- Starts with small indurated warty papule with erythematous hallow.
- Gradually forms serpiginous lesion with rough papillomatous surface, deep fissures to form granulomatous base.
- Occasionally may be keloidal or crusted lesion.
- Single or multiple lesions may be present.
- Responds well to AKT.

- Differential diagnosis:
- Warts
- Hypertrophic lichen planus
- Prurigo nodularis
- Blastomycosis

- Investigation:
- Biopsy
- Interferon gamma
- PCR

Lupus Vulgaris





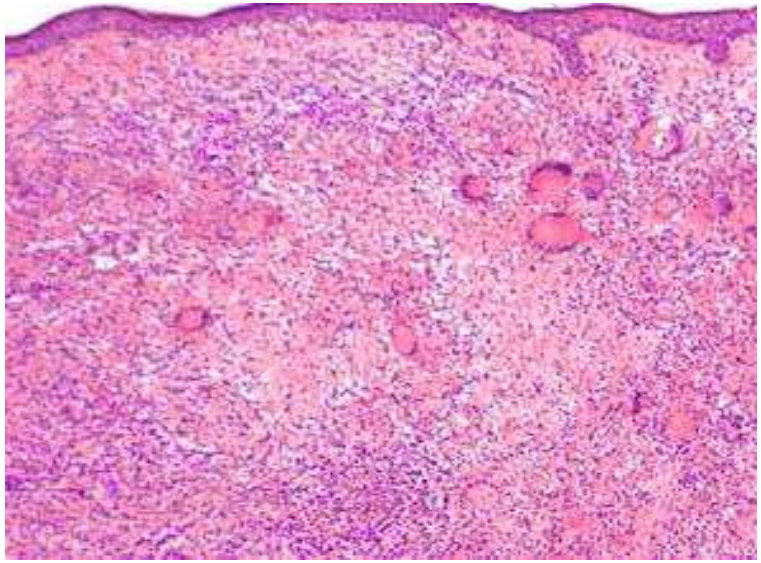
Lupus Vulgaris

- This is chronic progressive paucibacillary form.
- Most prevalent form of cutaneous TB.
- More common in females than males.
- Source is endogenous, contiguity hematogeous spread, from underlying foci.
- Adult females are commonly affected.
- Rarely exogenous infection with BCG vaccination.

Pathology:

- Characteristic tubercle without caseation.
- Epitheloid histocyte, multinucleated giant cell, and langerhans giant cell.
- Overlying epidermis is necrotic.
- Sites : face and neck are common in European population.
- In India multiple lesions over buttocks and trunk are common.

Histopathology: Lupus Vulgaris



Clinical features:

A) Plaque type: Initial lesion is small reddish brown papule extend to form plaques.

- Soft in consistency.
- Diascopy: apple jelly nodules are seen.
- Healing may occur at one end, extension at other end.

B)Hypertrophic form

C)Ulcerative: lesion breaks down to form ulcer, crusting, with deep tissue destruction.

D)Vegetative form

E) Multiple nodular form

Differential diagnosis:

- Lupus Erythematosus
- Sarcoidosis
- Blastomycosis
- Lichen planus
- Leprosy
- Psoriasis
- Leishmaniasis
- Jessener lymphocytic infiltrate.

- Complication:
- Scarring,
- Destruction,
- SCC.

- Prognosis if untreated, lesions are progressive and destructive. Rapid response to treatment.

Investigations:

- Biopsy
- Culture
- PCR
- Interferon gamma

Treatment

- Common drugs used:
- Isoniazid (H) (5mg/kg)
- Rifampicin (R) (10mg/kg)
- Ethambutol (E)(10-15mg/kg)
- Pyrazinamide (Z)(15mg/kg)

Treatment

- Regimen Cat1 is HRZE daily for 2 months along with HRE daily for 4months.
- In MDR, fluroquinolone group + amikacin/ kanamycin+ clofazimine+ ethambutol+ pyrazinamide for 12-18 months is given
- In XDR, add Bedaquiline to above regimen.

THANK YOU!