
C.L.T&R.I
OFFICE
ADMINISTRATIVE BLOCK

# DIFFERENTIAL DIAGNOSIS OF LEPROSY

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# CARDINAL SIGNS OF LEPROSY

- Hypopigmented Anaesthetic / Hypoanesthetic Patch
- Thickened nerves
- Presence of acid fast bacilli in slit skin smear

# CLINICAL PRESENTATION OF LEPROSY

1. Neural
    - pure neuritic hansens
    - visible deformities eg: claw hand , foot drop , lagophthalmos
    -facial nerve palsy
2. Ulcers
    -recurrent trophic ulcers of foot
    -scald injuries in hands
    -chronic non healing ulcer with osteomyelitis
3. Nasal symptoms :
    - epistaxis/ nasal stuffiness

4. SKIN :

- Hypopigmented anesthetic patch
- painful erythematous patch (type 1 reaction )
- painful nodules( type 2/ENL Reaction)
-ulcerative ENL
- Nodule ( histoid hansens )

# NEUROLOGICAL CONDITIONS

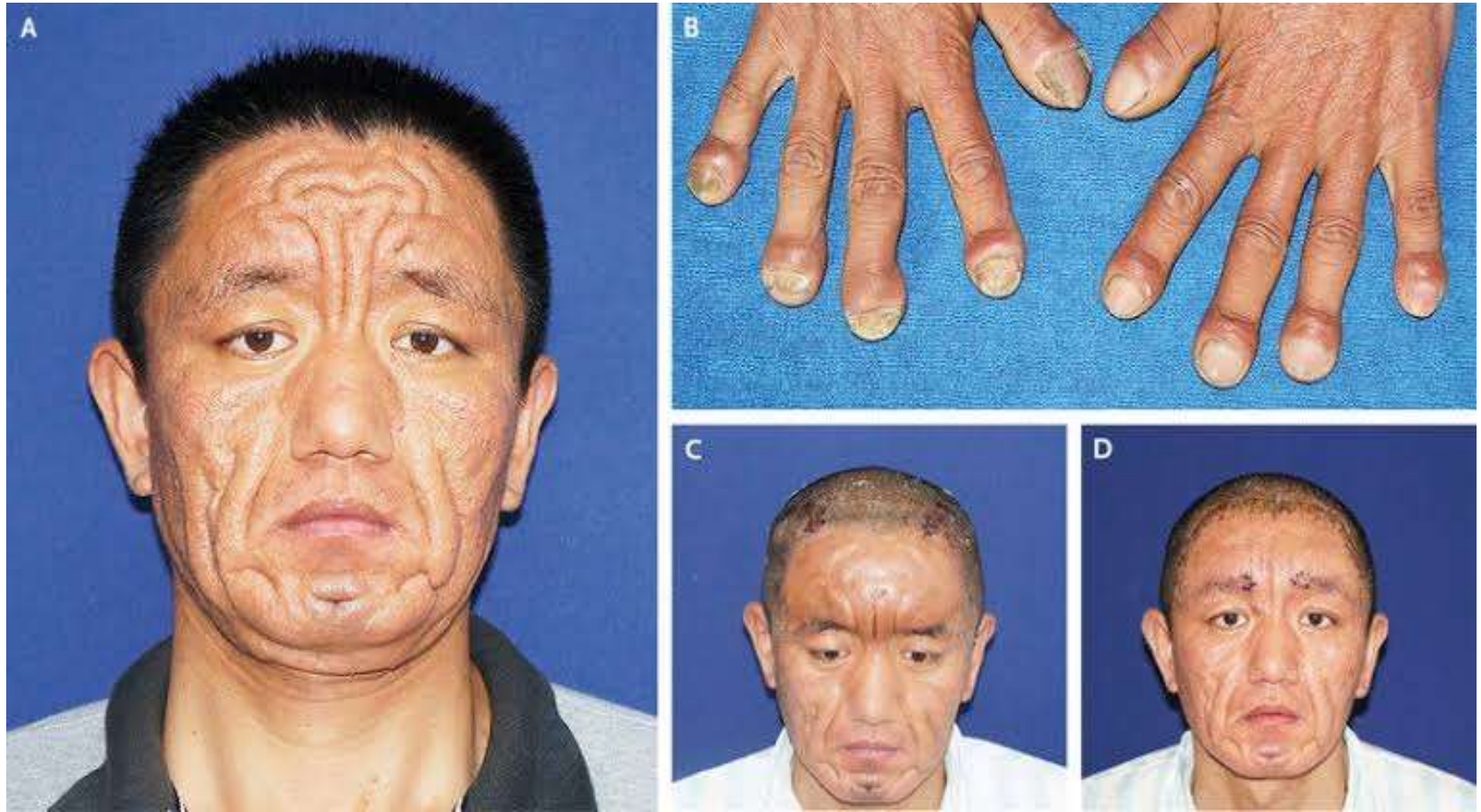
- Palpable Nerve Thickening without Anesthesia or other Signs of Nerve Damage:

1. ***Excessive Muscular Development*** :

This is generalized as in a professional wrestler, and localized as in a person accustomed to carry heavy weights on the head, with resultant thickening of great auricular nerve.

## 2. *Pachydermoperiostosis:*

A condition with generalized thickening of skin, periosteum and bone. Generalized nerve thickening was reported . In addition, there is clubbing of fingers and furrowing of the thickened skin of forehead which can easily be mistaken for the leonine facies of LL.



# PALPABLE NERVE THICKENING WITH/WITHOUT ANESTHESIA OR MUSCLE WASTING 2

- 1. Hereditary Neuropathies(HSN 1 & 3)

## HSMN 1

Inheritance: AD.
Age of presentation: First decade of life.
Symptoms: Poor motor performance, thinning of extremities, paresthesias, cramps.
Signs: Hand muscles may be affected first, while as the disease progresses lower limb is involved. Other features are distal muscle atrophy, skeletal abnormalities (hammer toes, pes cavus), graded sensory impairment and generalized areflexia with normal autonomic functions. Nerve palpation: Generalized, smooth, uniform, non-tender nerve enlargement.

**HSMN 3**

İHMSN-3 is also called Dejerine-Sottas syndrome. It is characterized by segmental demyelination of peripheral nerves, which appear hypertrophic as a result of concentric proliferation of Schwann cells.
Age of presentation: Infancy or childhood.
Symptoms: Delayed motor milestones, weakness and wasting of distal limbs. There is progressive symmetric weakness and deformiti lower limbs, with an equinovarus posture. Signs: Severe sensory impairment and generalized areflexia are noted. Tendon reflexes are or absent, in contrast to leprosy

# Disorders with nerve thickening

| | |
|---|---|
| **Infective** | Leprosy |
| **Hereditary** | Hereditary motor and sensory neuropathy, Refsum's disease, Rud's syndrome |
| **Immune mediated** | Chronic inflammatory sensory and demyelinating polyradiculopathies |
| **Infiltration** | Neurolymphomatosis, lymphoma, leukemia, amyloidos |
| **Tumors of nerve or nerve sheath** | Schwannoma, neurofibroma, neurofibromatosis 1 and 2 |

# Regional Anesthesia with or without Muscle Wasting but without Palpable Nerve Thickening

- Syringomyelia
- Tabes
- Peripheral Neuropathy: HIV, SLE,PAN, Sarcoidosis,Diabetic neuropathy , Alcoholic polyneuropathy.
- Hereditary Sensory Radicular Neuropathy
- Congenital Indifference to Pain
- Hysteria

# DIFERENTIAL DIAGNOSIS OF DERMATOLOGICAL CONDITIONS

## 1.PITYRIASIS ALBA

COMMON IN CHILDREN

MC SITE:FACE

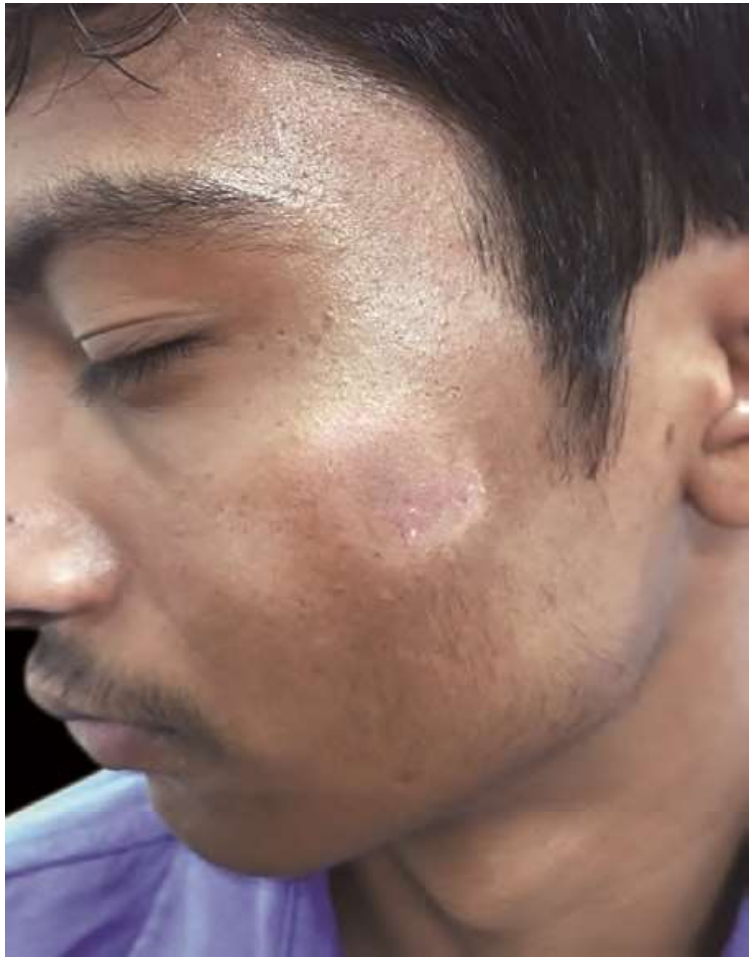
NO OF LESION :SINGLE TO TEN

MORPHOLOGY:HYPOPIGMENTED,ILL DEFINED ROUND TO OVAL

SLIGHTLY SCALY

COURSE:SEASONAL VARIATION & SELF RESOLUTION

RX: EMOLLIENTS OR MILD STEROIDS



# 2.NEVUS ACHROMICUS/ANEMICUS

*N.achromicus* – circumscribed area of hypomelanosis present at birth

Morphology :

- bizarre shaped , variable degree of hypopigmentation

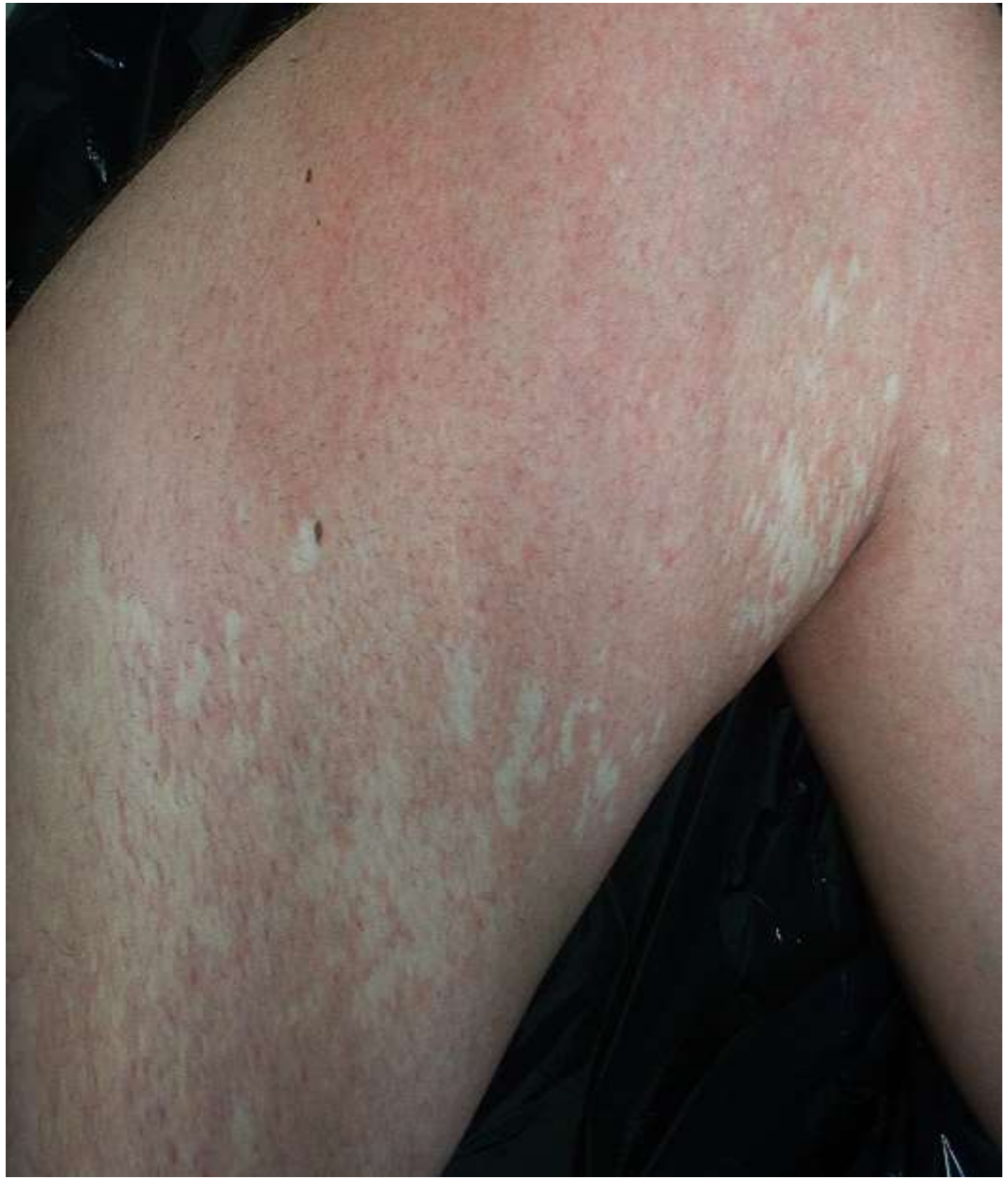
-usually single , has feathery , serrated well defined borders .

-does not disappear on diascopy .

N.anemicus- circumscribed area of hypopigmentation due to vascular anamoly

-disappears on diascopy

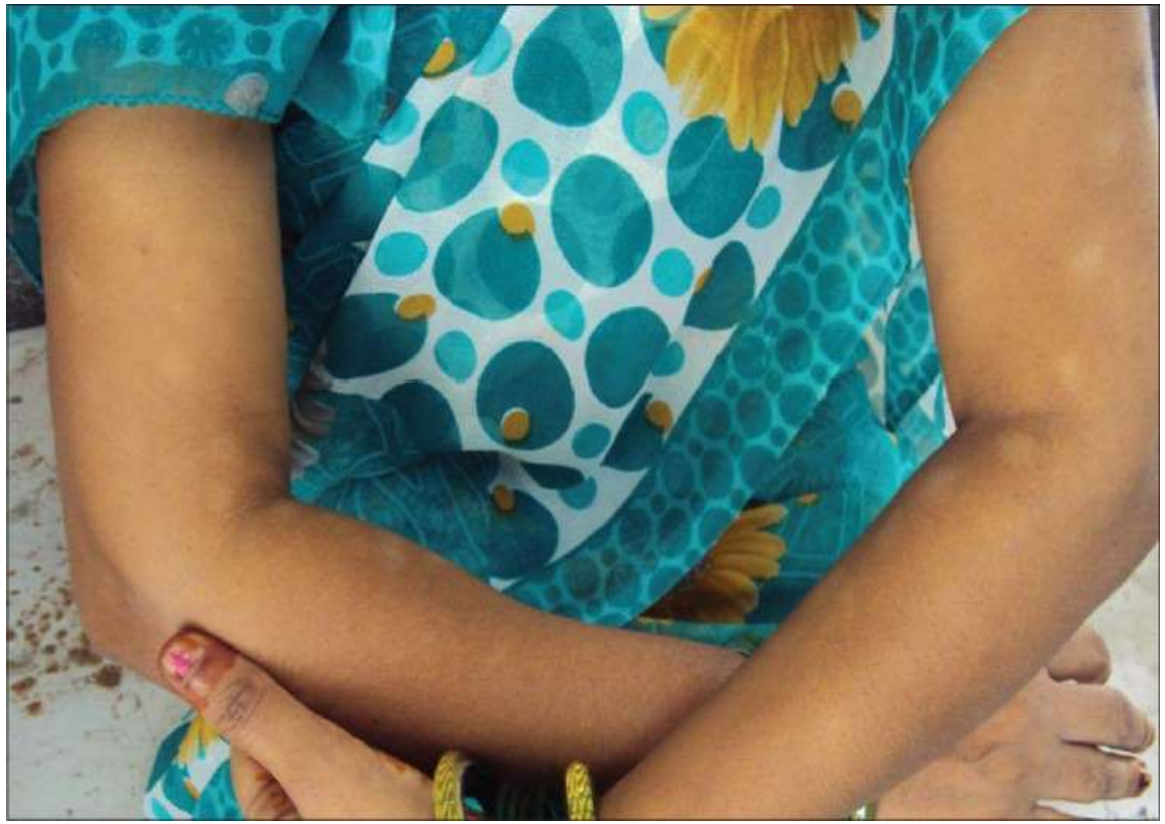
# N.ANEMICUS

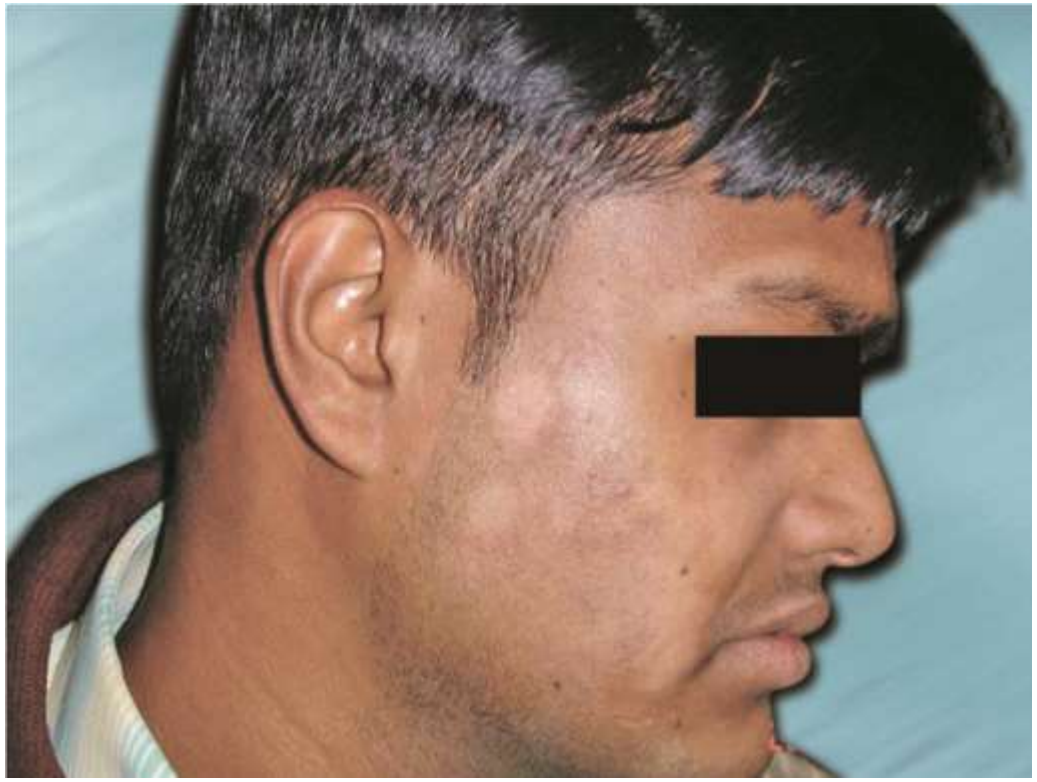


# N.ACHROMICUS

# POLYMORPHIC LIGHT ERUPTION

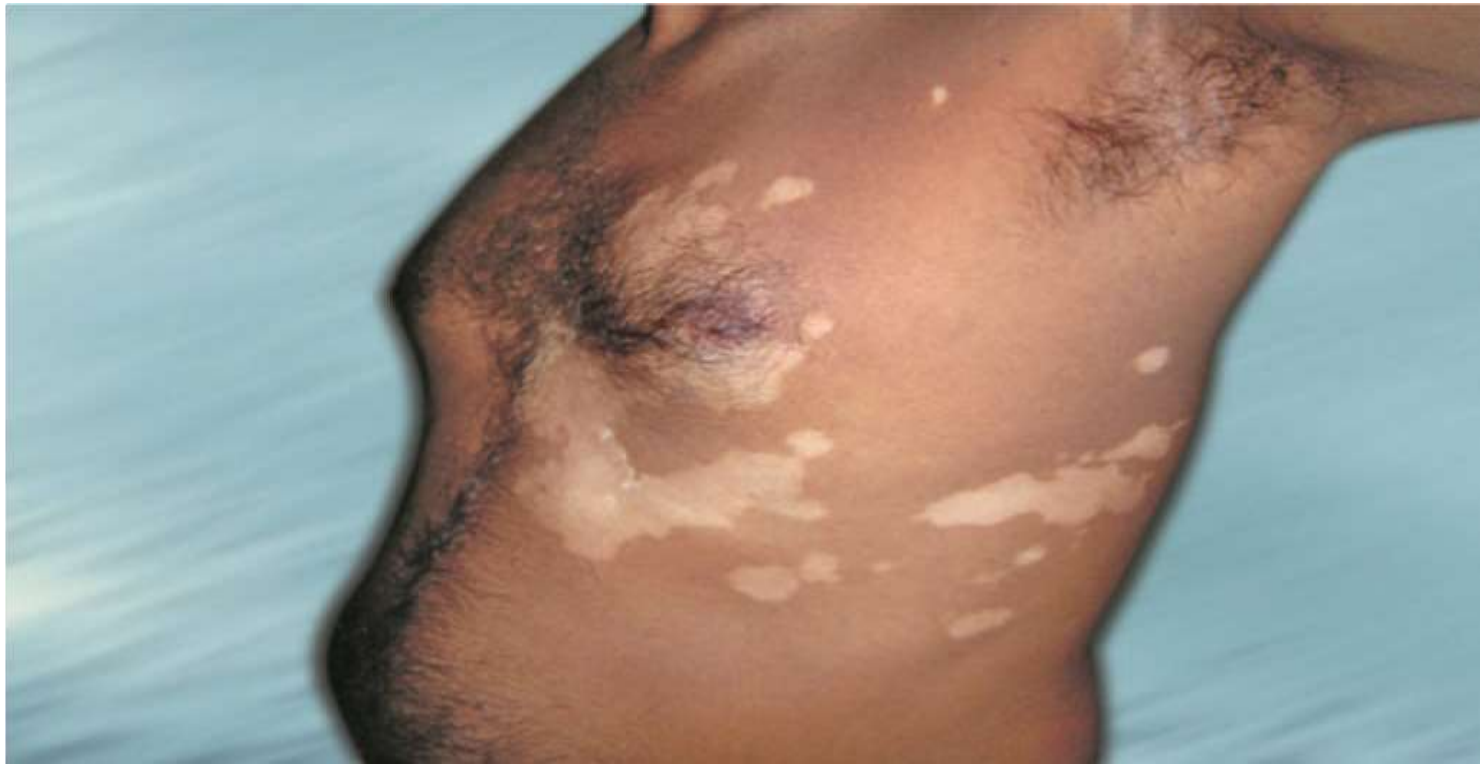
- Hypopigmented lesion – asymptomatic or slightly itchy , mainly over sun exposed parts especially over face which are confused with indeterminate leprosy





# VITILIGO

- Usually depigmentation but hypopigmentation is seen in the incipient stage or during treatment
- No sensory loss , hair loss , decreased sweating
- Skin texture is normal



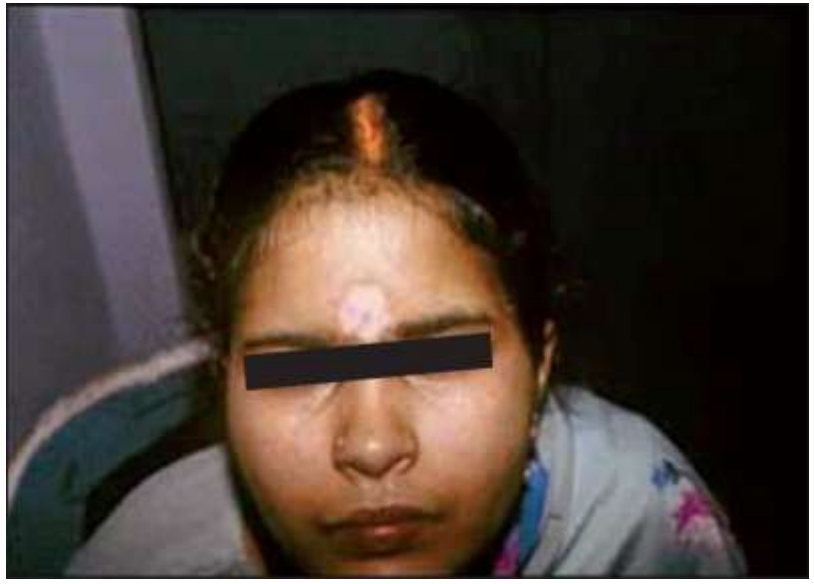
# ACQUIRED HYPOMELANOSIS

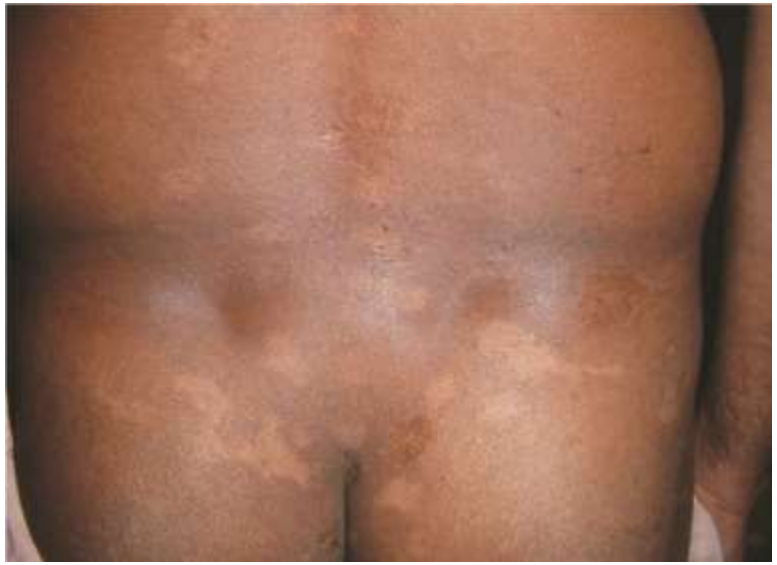
✓Causes

-long term use of potent topical  steroids or intralesional appl

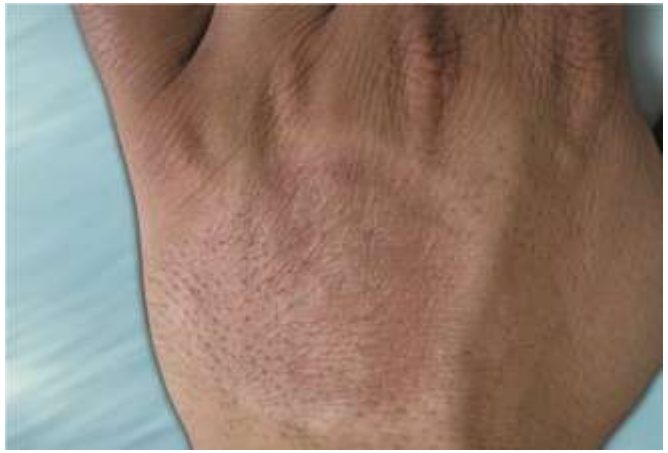
- chemicals leading to contact leucoderma

-follows inflammatory conditions like psoriasis , eczema , lupus erythematosus , sarcoidosis





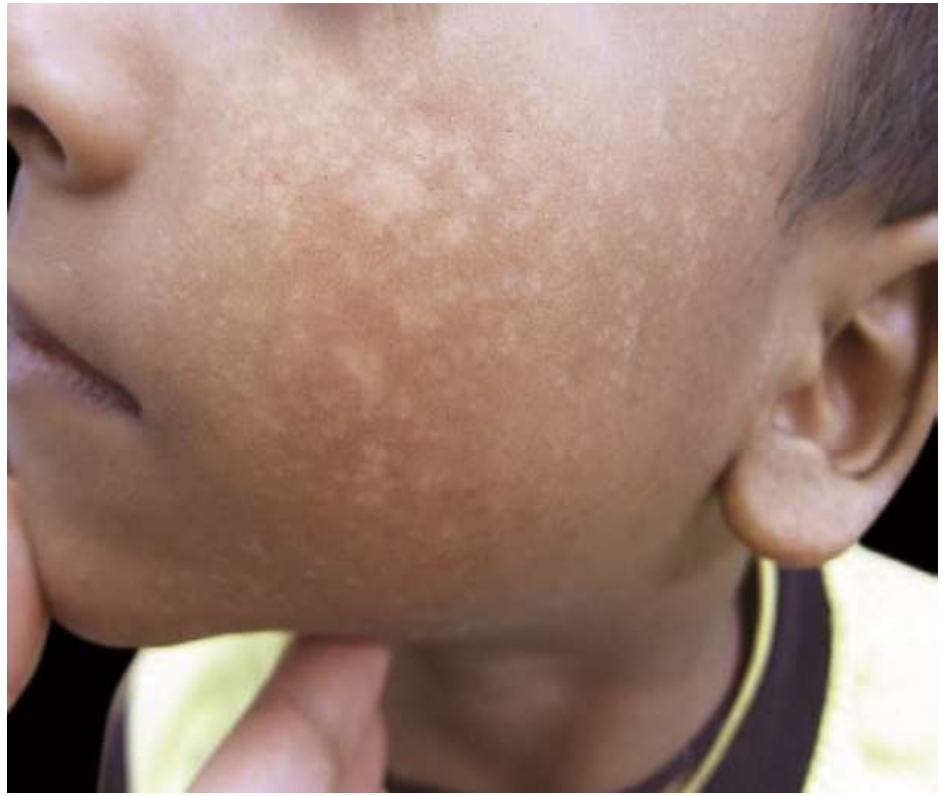
Postinflammatory hypopigmentation after the lesion of psoriasis

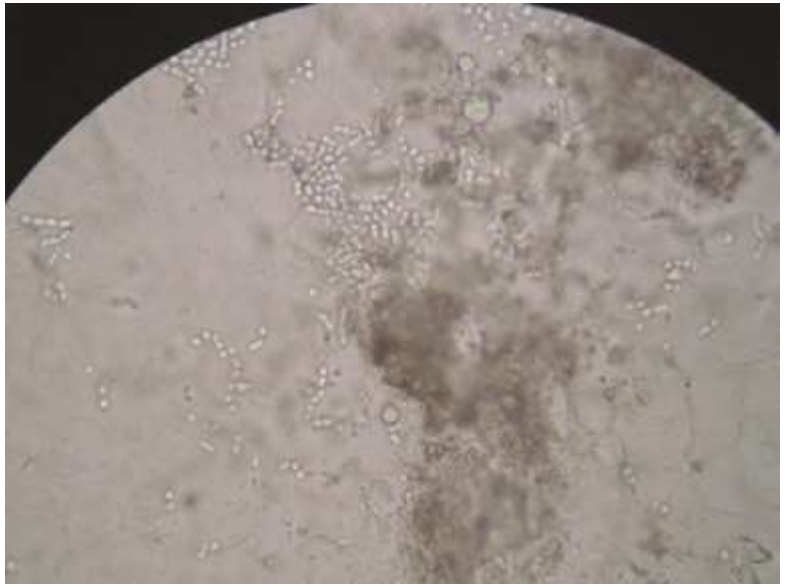


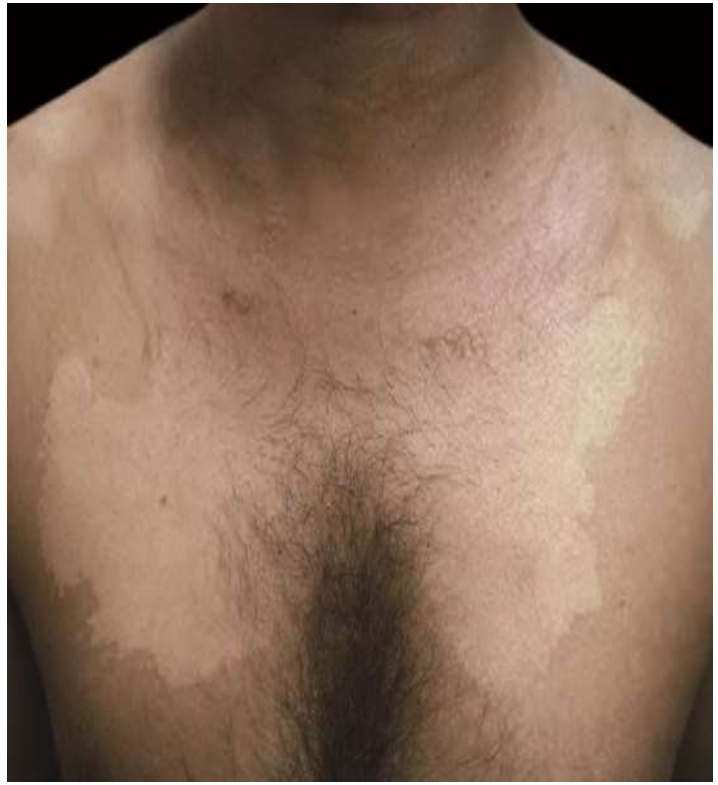
Hp after healing of trauma

# PITYRIASIS VERSICOLOR

- Morphology : smaller than leprosy macules , hypopigmented and show fawn colored fine scaling , asymptomatic , may fuse to form large lesion
- No impairment of sensation or hair loss
- Diagnosis : koh mount

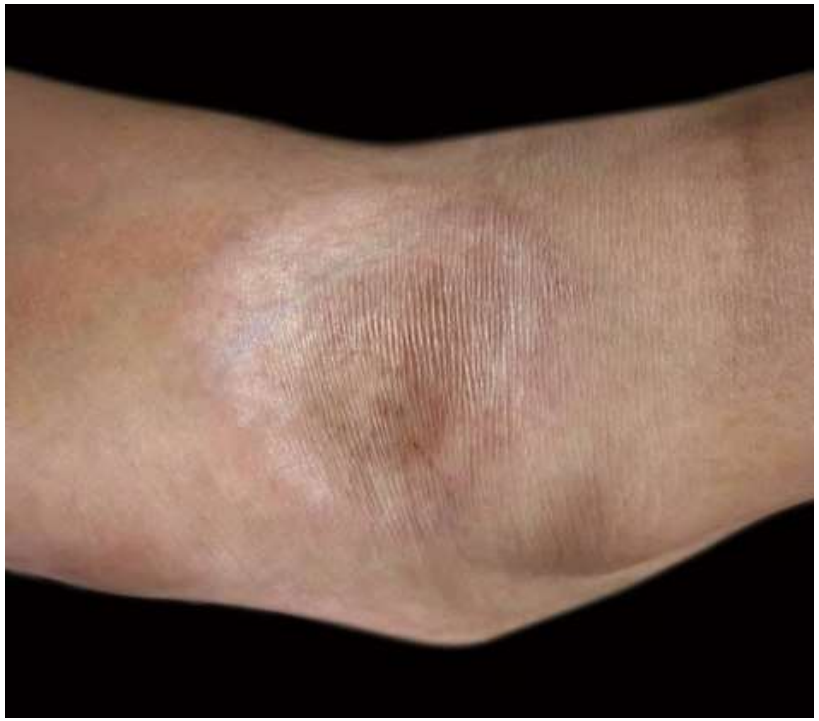


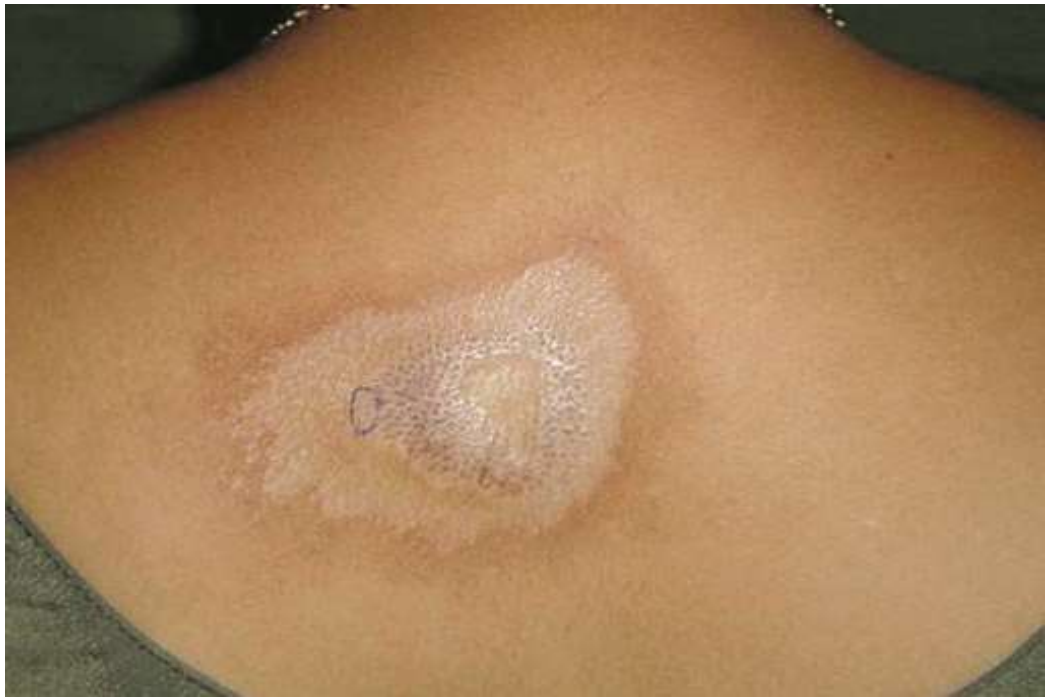




# MORPHEA (LOCALIZED SCLERODERMA)

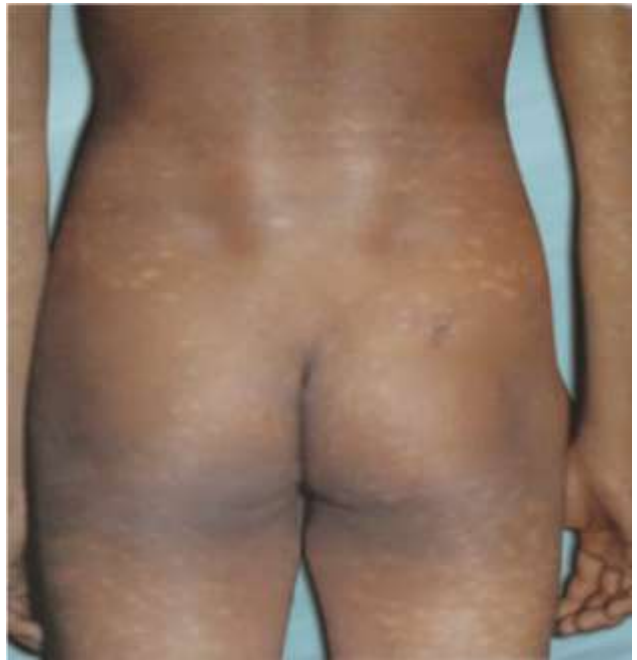
- A white plaque which may be slightly raised in parts, the edge often purple, hair growth and sweating are lost in the lesion, and when sclerosis is marked there is some sensory loss (

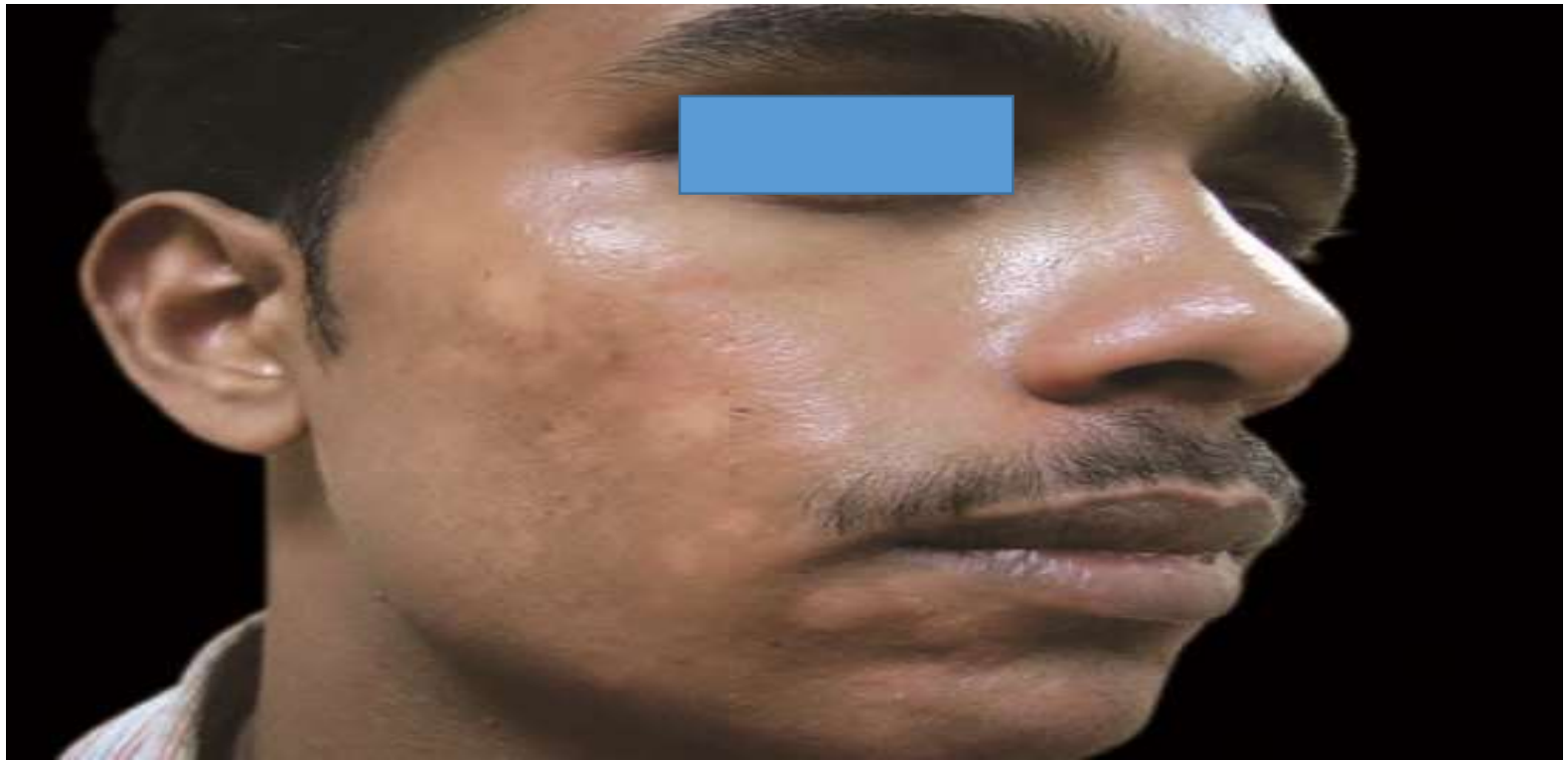




# POST-KALA-AZAR DERMAL LEISHMANIASIS (PKDL)[10]

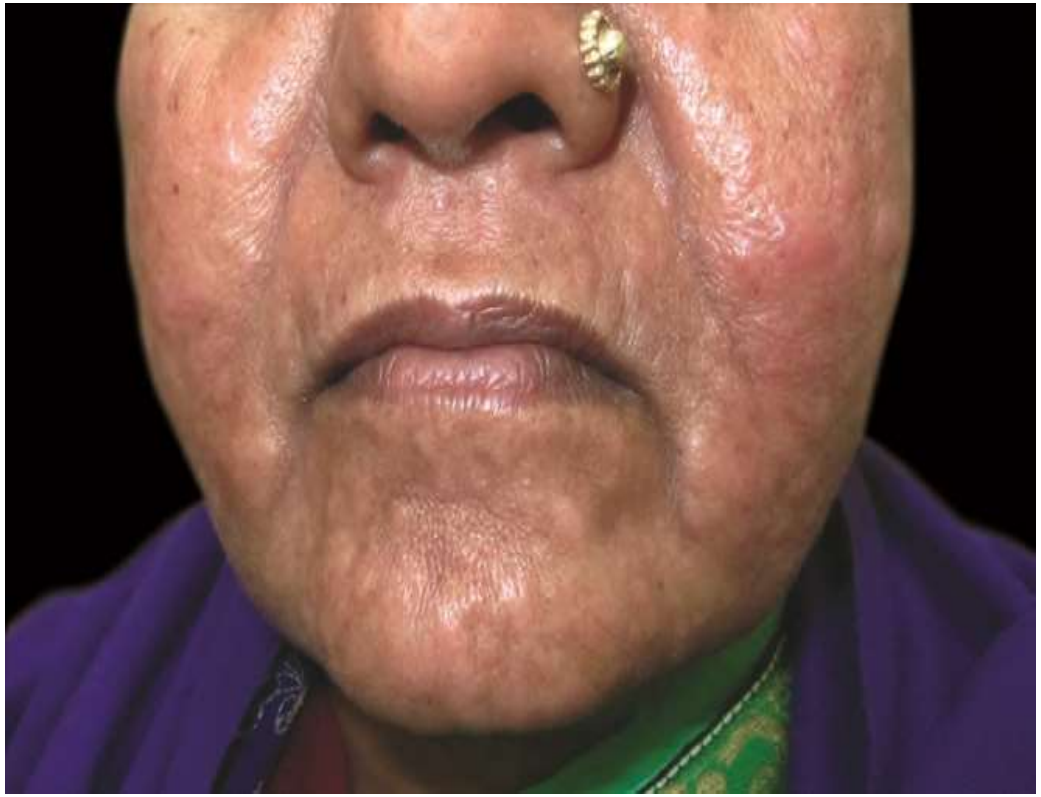
- Geographic distribution : Indian subcontinent and to East Africa
- Macular PKDL - perioral hypopigmented macules that coalesce to form well demarcated, irregular patches.
- The patches then spread over the malar region, followed by the forehead and scalp and then appear on trunk and limbs (Fig. 9.5). In addition, there may be erythematous





-Macular PKDL is mistaken for  borderline or lepromatous leprosy

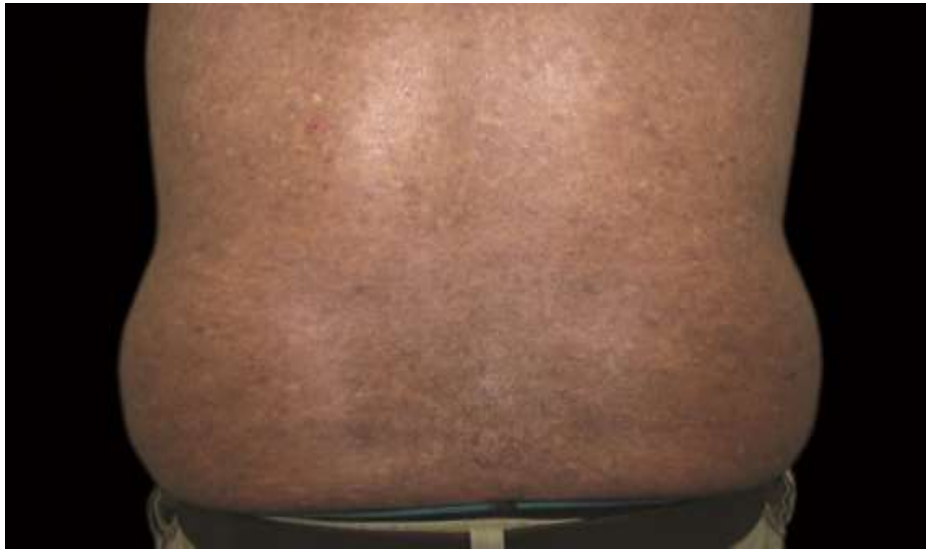
-However the  lesion is b/l and symmetrical



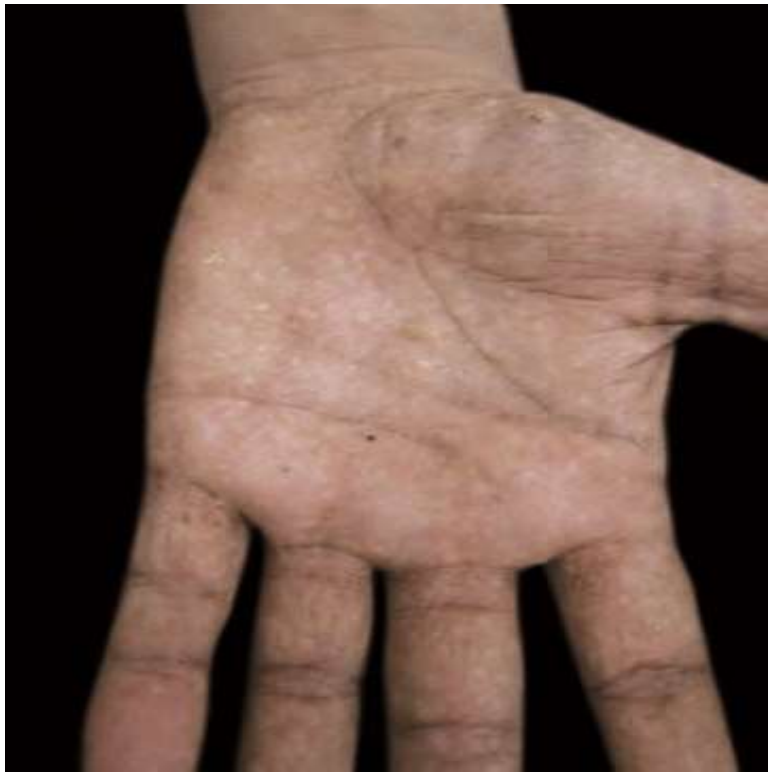
Post-kala-azar dermal leishmaniasis: An admixture of nodules, papules and macules in the perioral area

# CHRONIC ARSENIC EXPOSURE

- Clinical presentation: diffuse pigmentation / fine freckle – like spotty pigmentation ( raindrop pigmentation ) or normal areas of depigmentation on normal / hyperpigmented skin
- Minute papules (<2mm ) – mild form
- Punctate wart like papules 2-5mm -moderate form
- Diffuse hyperkeratosis – severe form



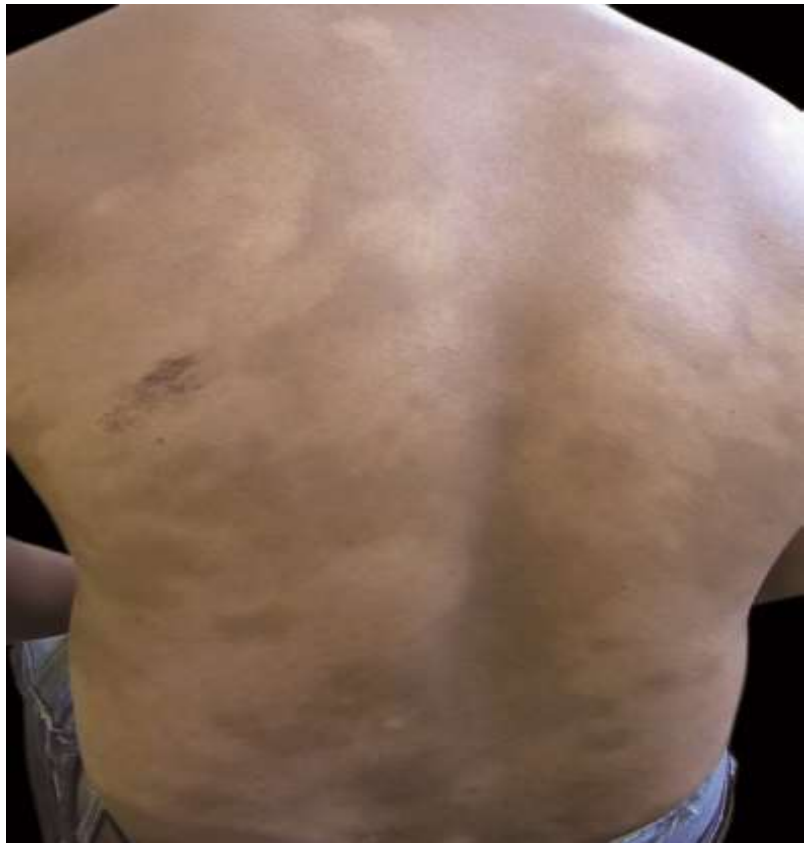
Raindrop pigmentation

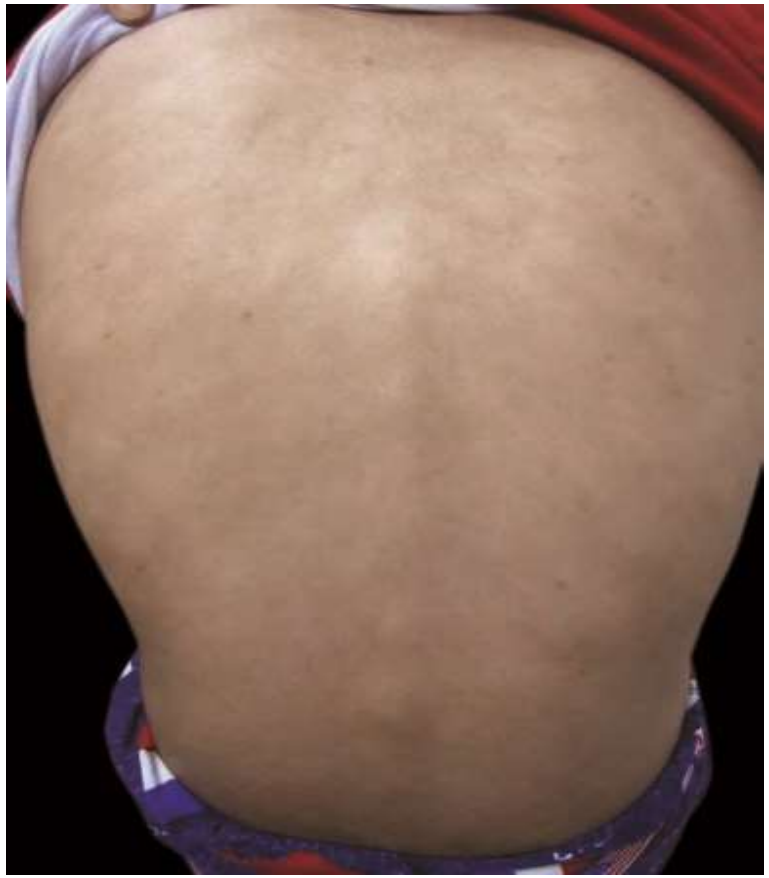


Punctate keratosis of palm

# PROGRESSIVE MACULAR HYPOMELANOSIS

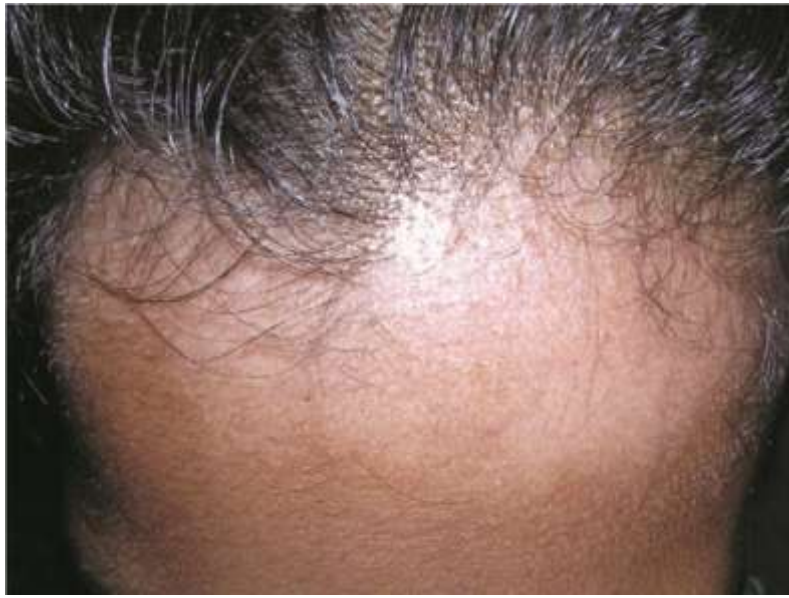
- This typically occurs in young *females*.
- Morphology: *asymmetric*, hypopigmented, poorly demarcated, smooth macules or patches.
- Classical site : mid-lumbar region followed by the abdominal area, face spared

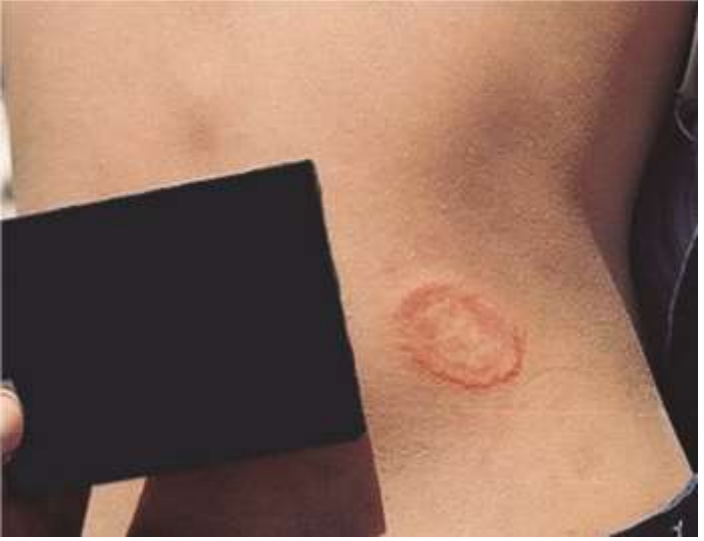




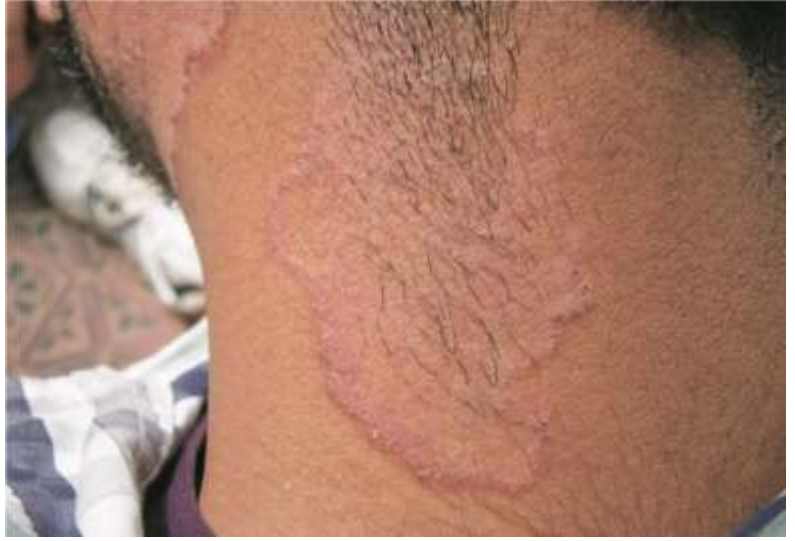
# OTHERS:

- Hypopigmented Mycosis Fungoides
- Idiopathic Guttate Hypomelanosis
- Albinism
- Lichen Sclerosus
- Achromic onchocerciasis
- Tinea corporis
- Incontinentia pigmenti achromicus
- Seborrheic dermatitis
- Leukoderma syphiliticum

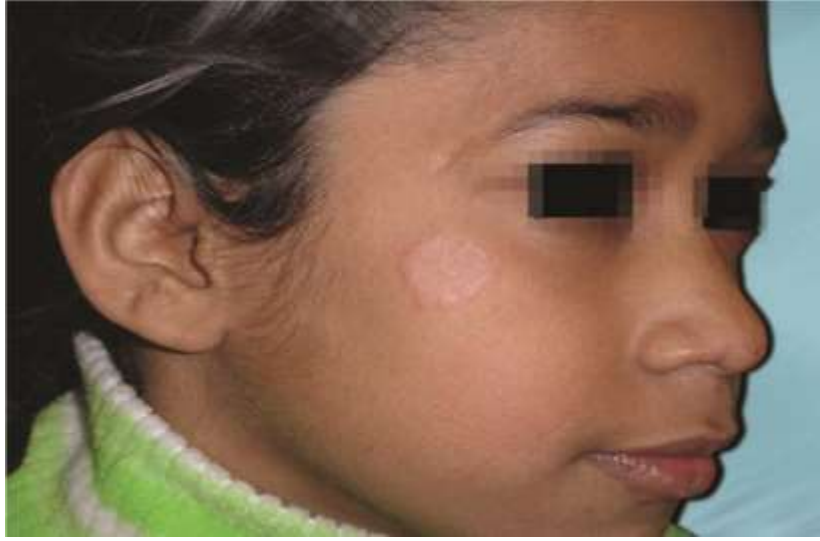




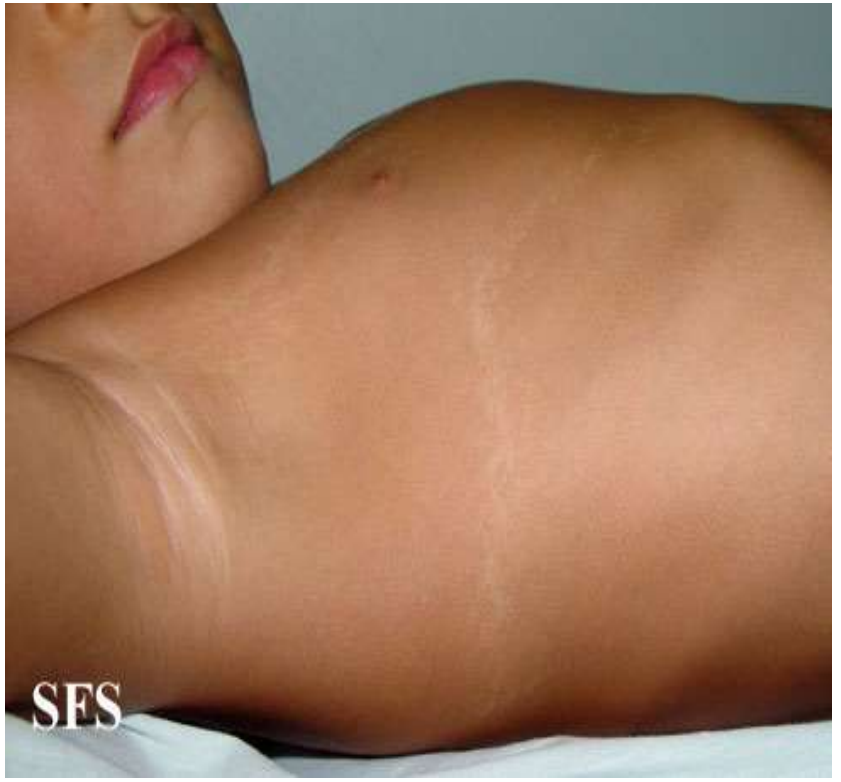
Tinea corporis resembling tuberculoid leprosy

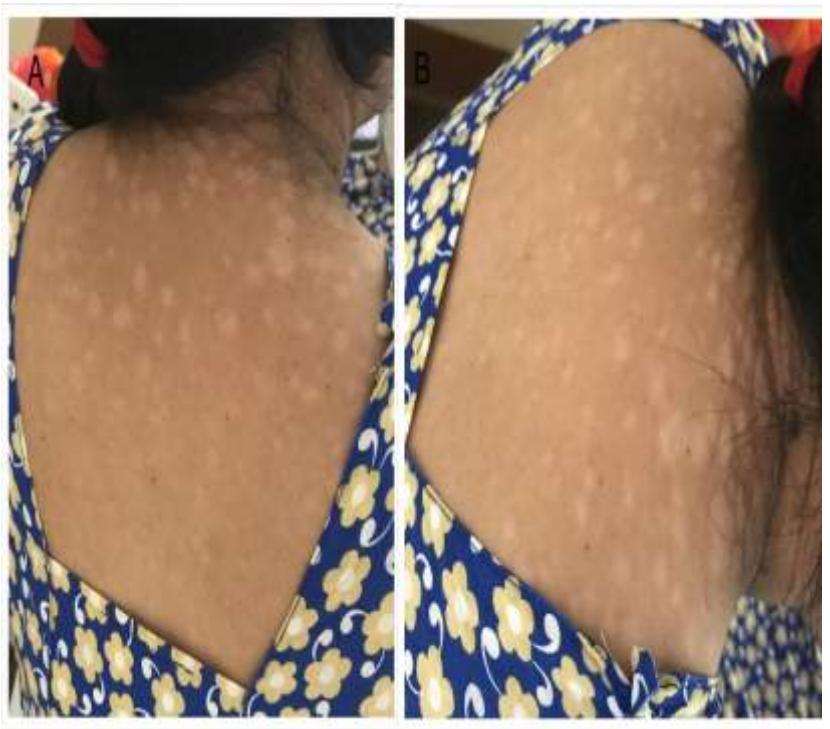


Tinea:Side of neck scaling and peripheral erythema



Tinea facei

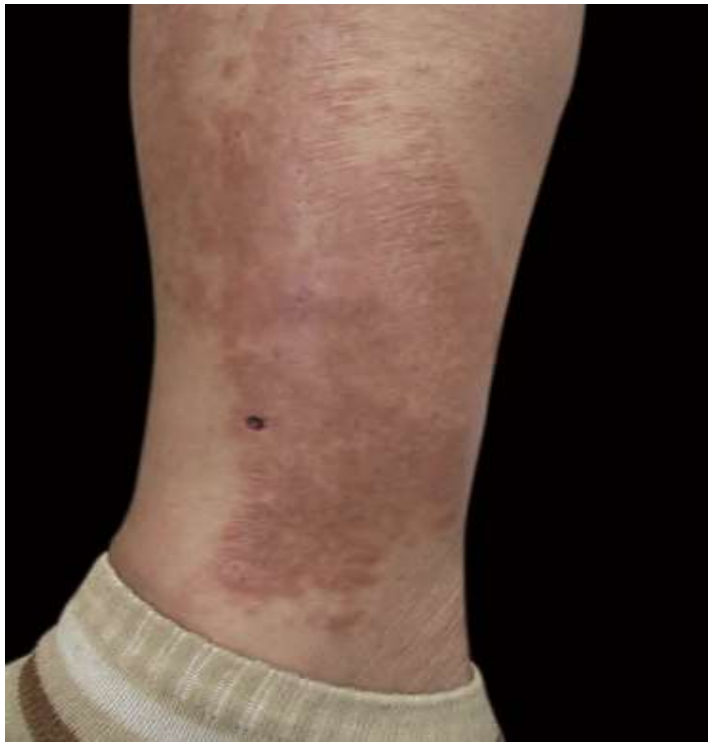


# Lesions which are Raised

*1.Follicular Mucinosis* (*Alopecia Mucinosa*)[21, 22]

Skin-colored or erythematous plaques favor scalp, face, neck, shoulders and limbs (Fig. 9.11a). Lesions are scaly and without hair (alopecia), but hair follicles are prominent.



## *2.Granuloma Annulare*

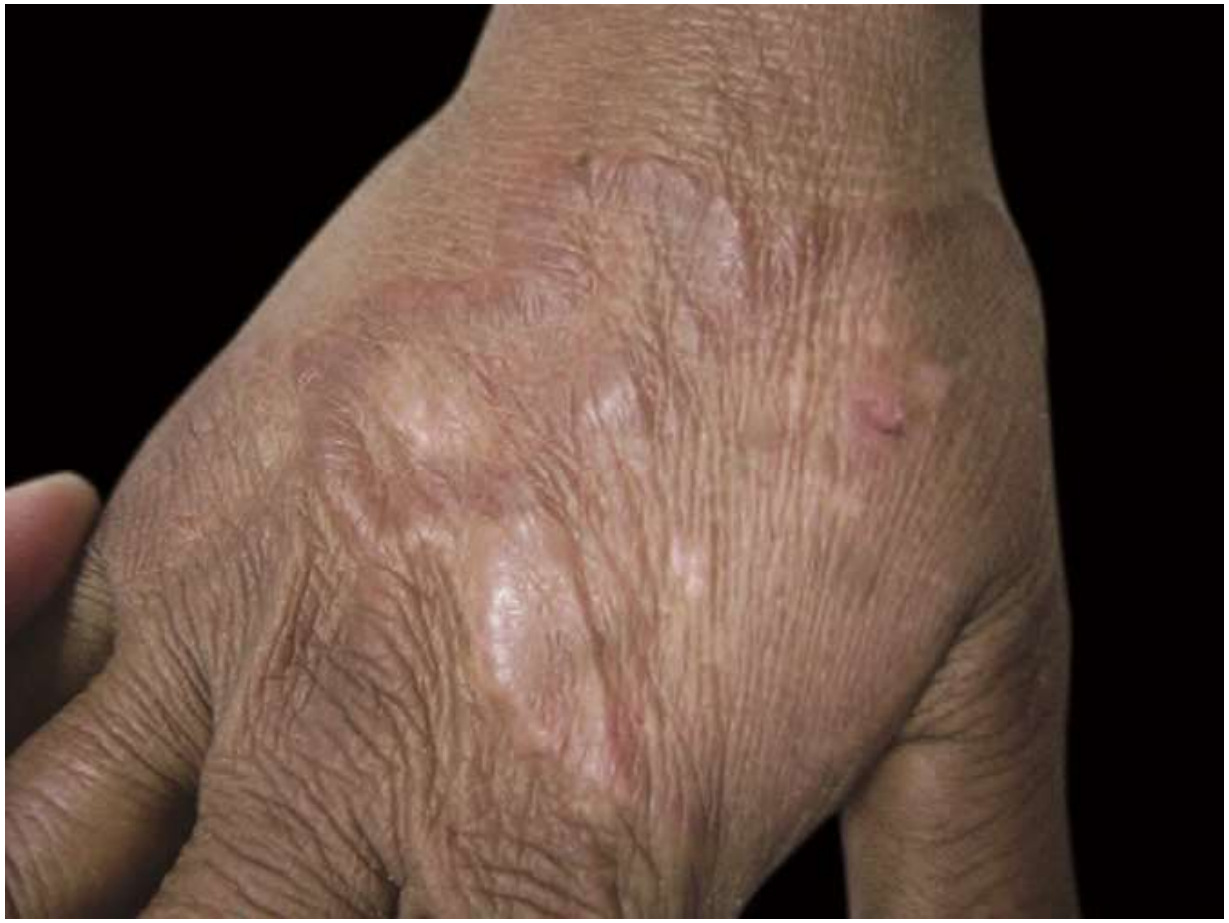
- disease of unknown etiology

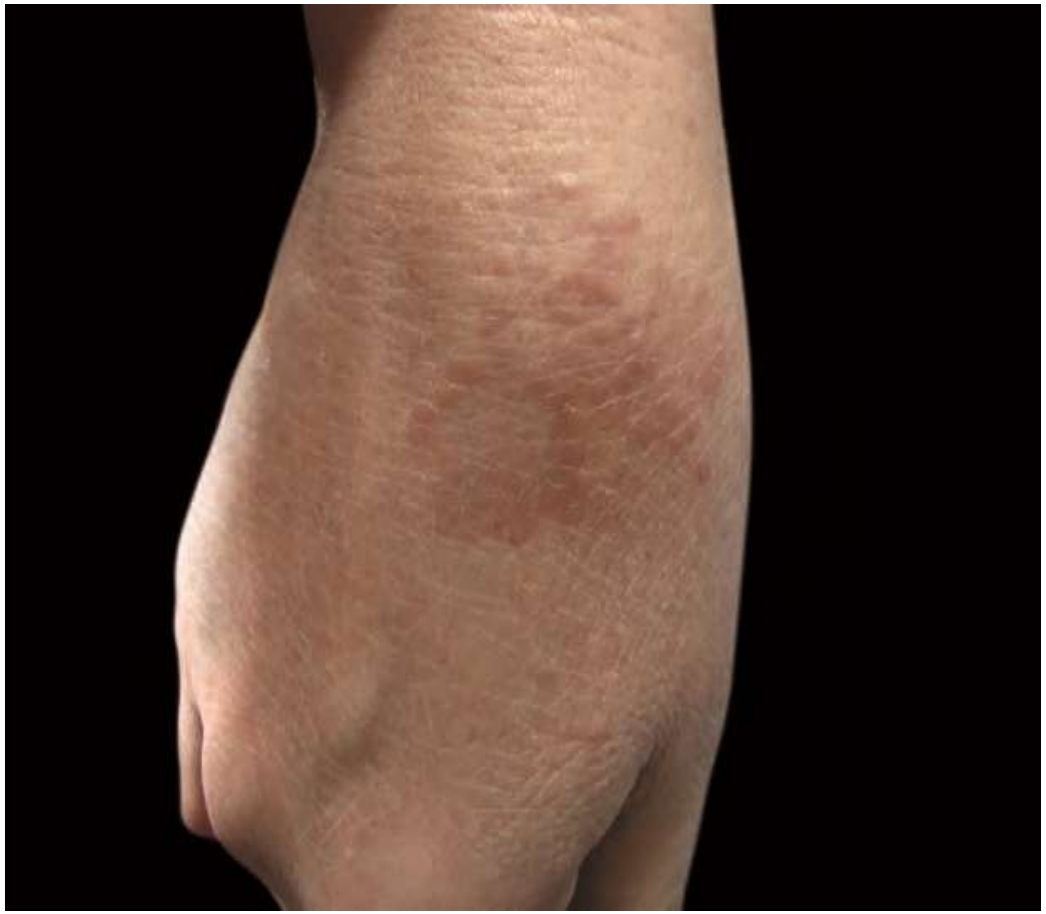
- mc variant seen over dorsa of hands as asymptomatic , skin colored , non scaly annular plaque with beaded margins and centrifugal extension

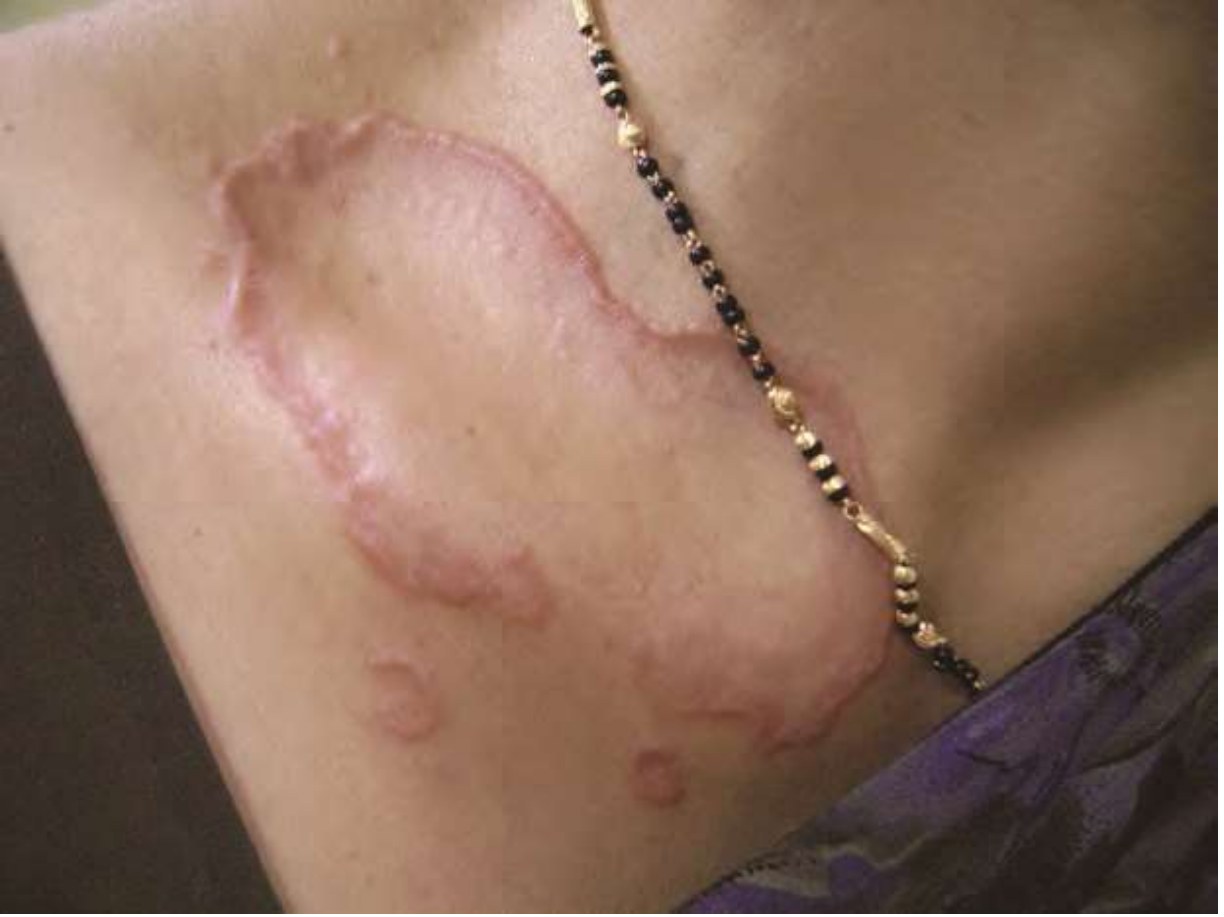
- localized variant should be differentiated from tuberculoid or BT lesion

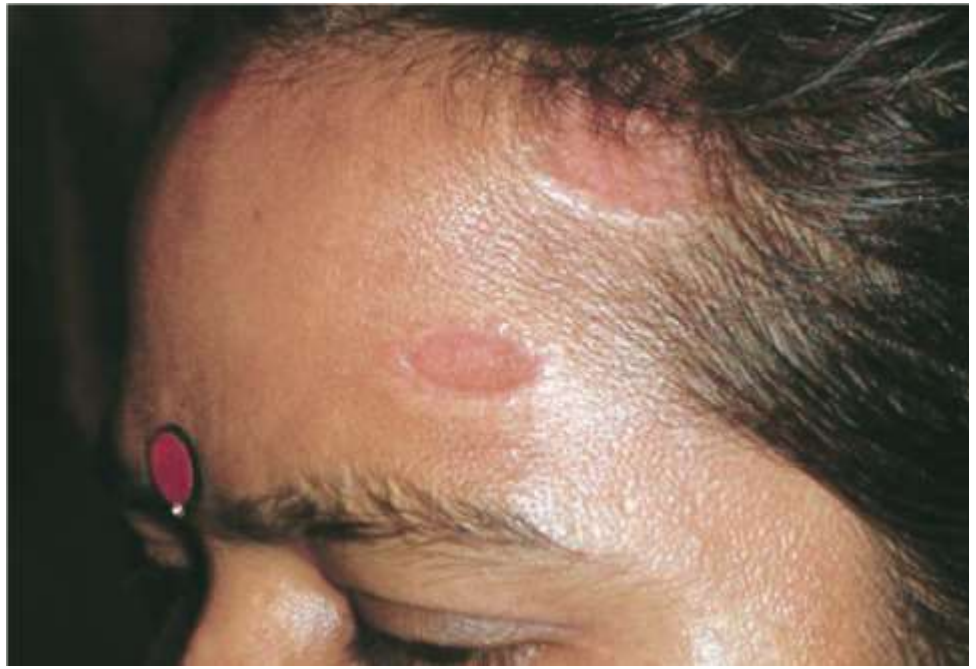
- generalized should be differentiated from lepromatous

# Granuloma annulare

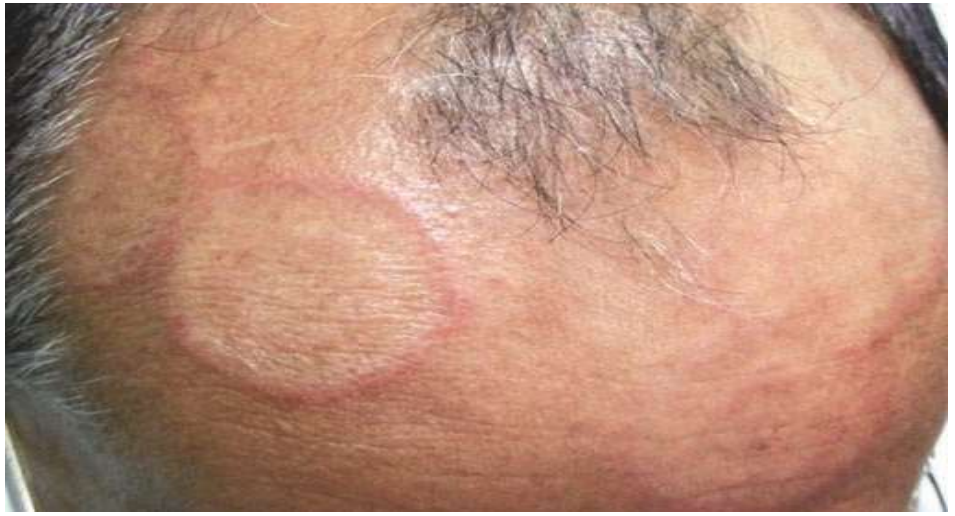






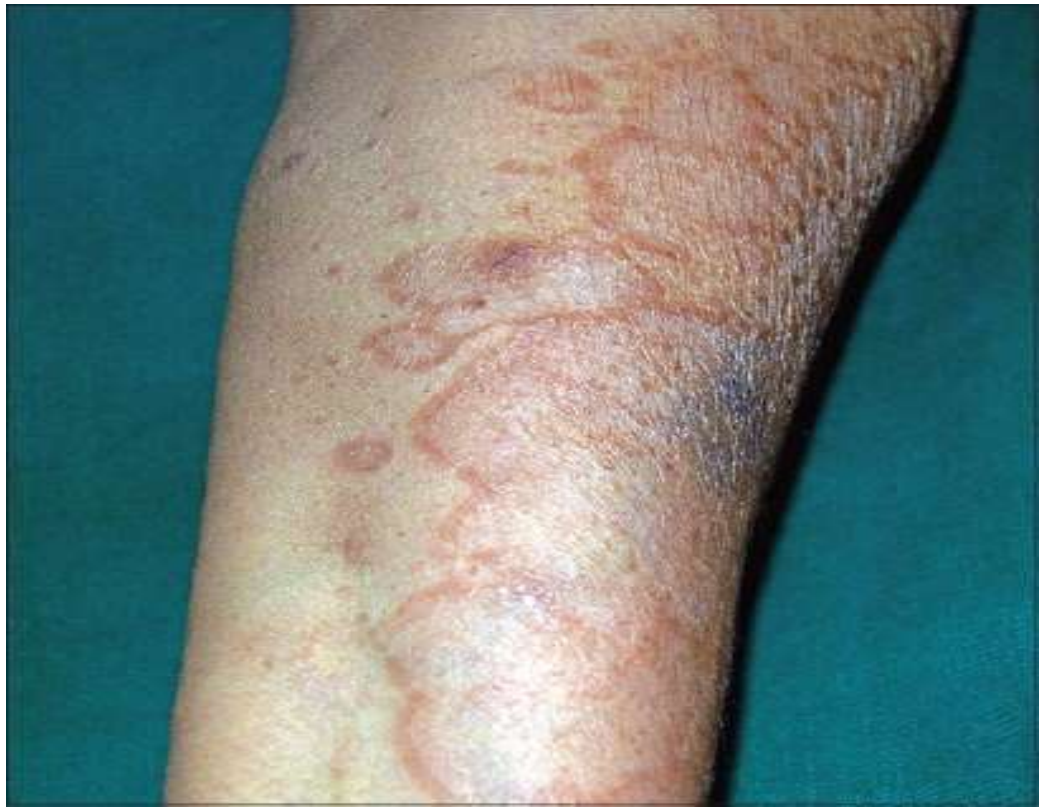


Granuloma annulare

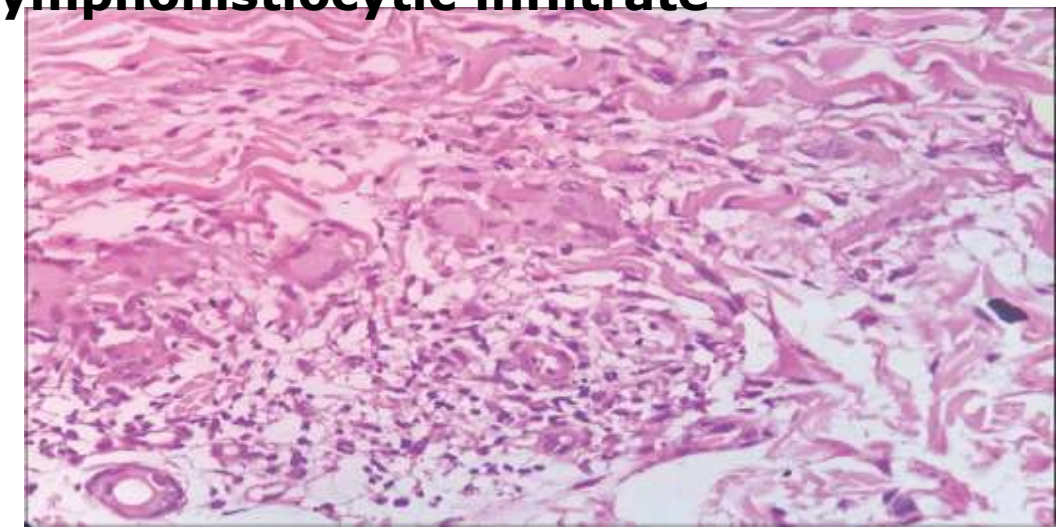


# 3.GRANULOMA MULTIFORME

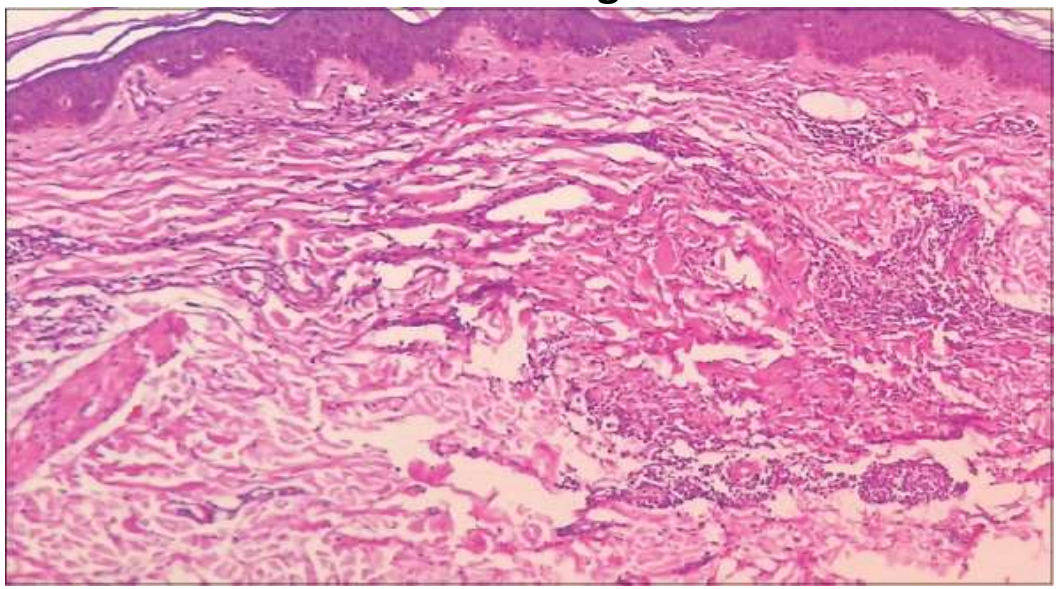
- The etiopathogenesis of GM is still unknown
- Recently, it has been proposed that GM is a photodermatosis, and it has been postulated that the primary event in the pathogenesis could be cumulative damage to collagen. This damage to collagen could be induced by a chemical or biological agent in the environment and further potentiated by immunologic factors. This is supported by the fact that, in almost all cases reported , the lesions have been confined to sun-exposed areas.
- Histology shows foci of necrobiosis surrounded by granulomatous zone with elastotic material within the granulomas. The giant cells contain fewer nuclei and scarring is absent.
- Often confused with tuberculoid lesion
- Rx: resolves spontaneously/ topical steroids



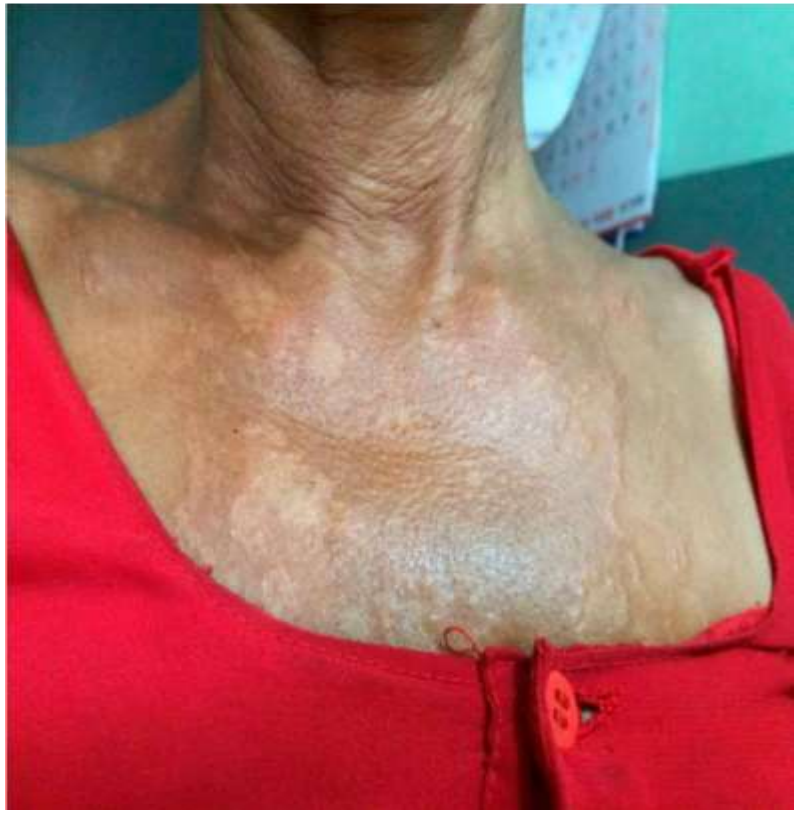
**Dermis showing prominent multinucleated giant cells with perivascular lymphohistiocytic infiltrate**

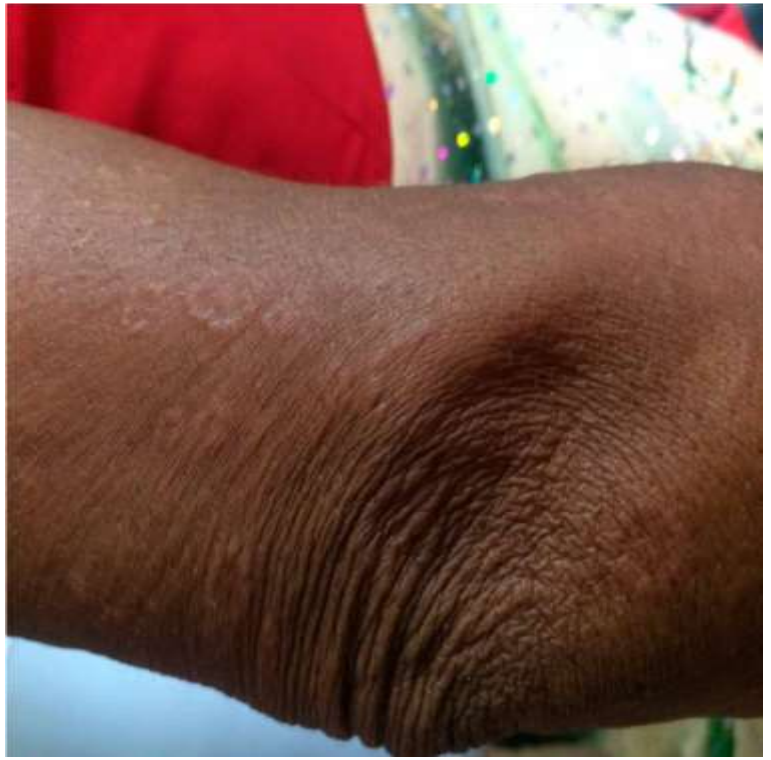


**Necrobiotic granuloma**



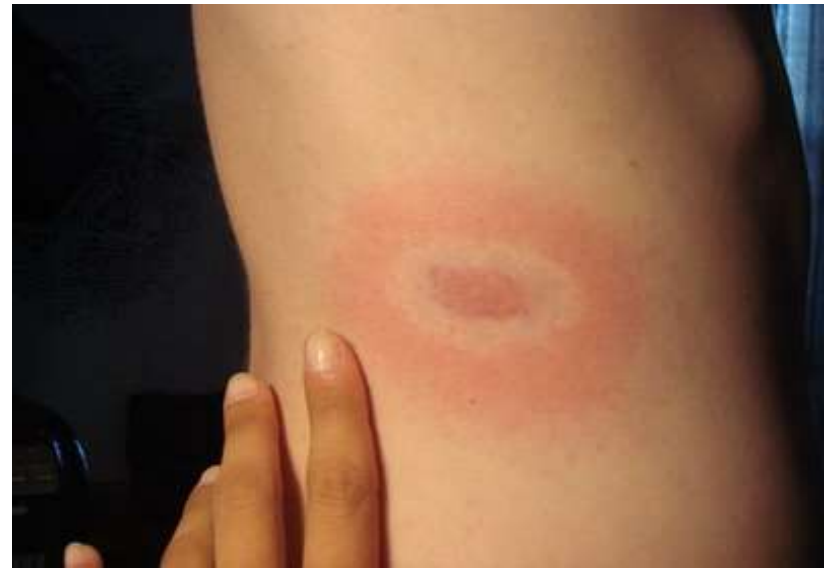
# Granuloma multiforme



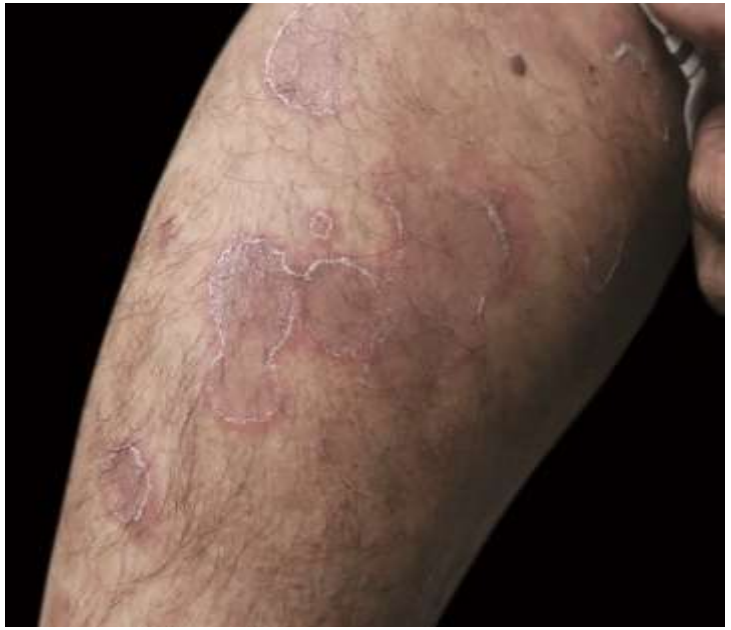


# 4.GYRATE ERYTHEMA

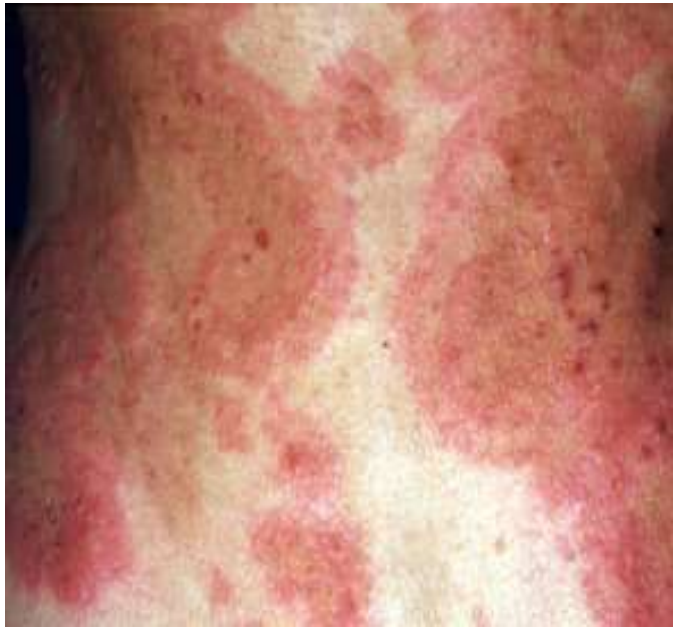
- *Erythema marginatum* (*EM*): rheumatic fever, trypanosomiasis, serum sickness, or streptococcal endocarditis.
- *Erythema chronicum migrans* (*ECM*):first stage of Lyme disease
- *Erythema gyratum repens* (*EGR*):associated with an underlying malignancy and presents as multiple, macular, serpiginous bands of erythema which migrate. Pruritus is common.
- *Erythema annulare centrifugum* (*EAC*): hypersensitivity reaction to tinea infection is suspected



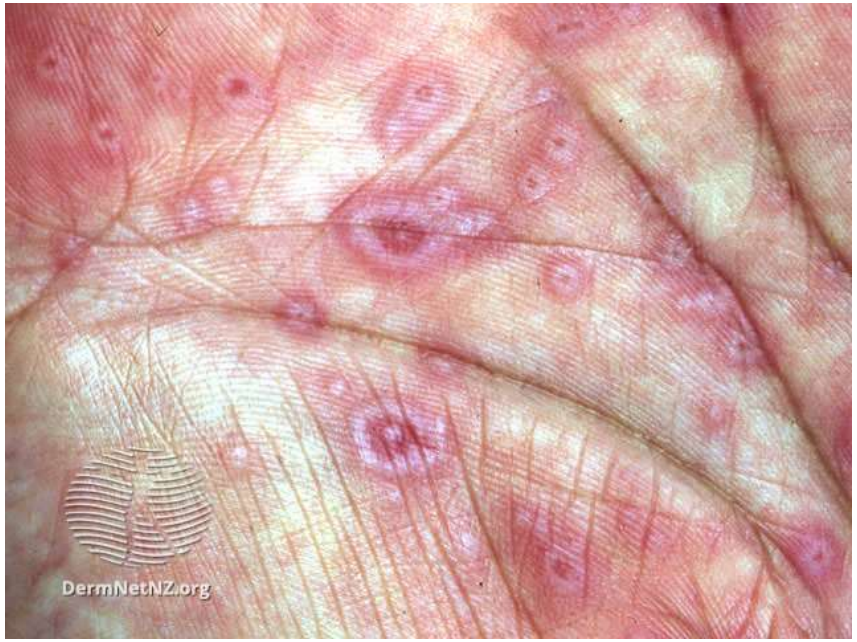
Ery.centrifugum

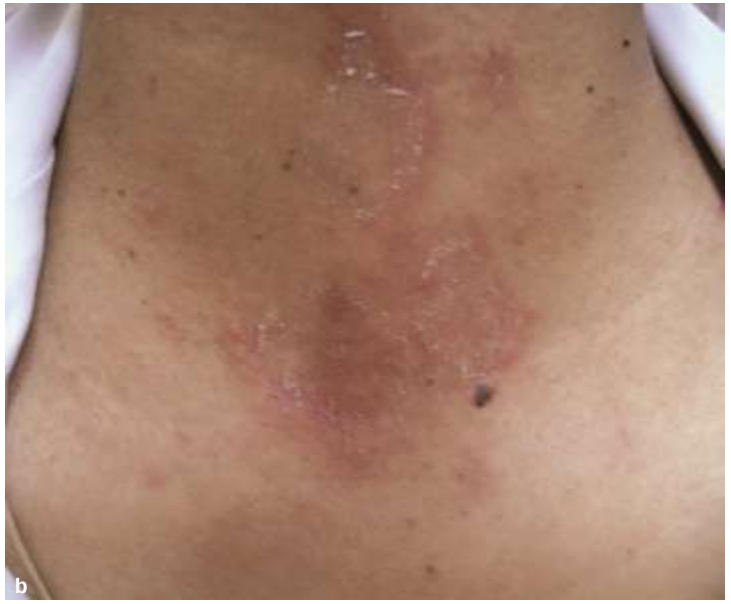


*Erythema gyratum repens*

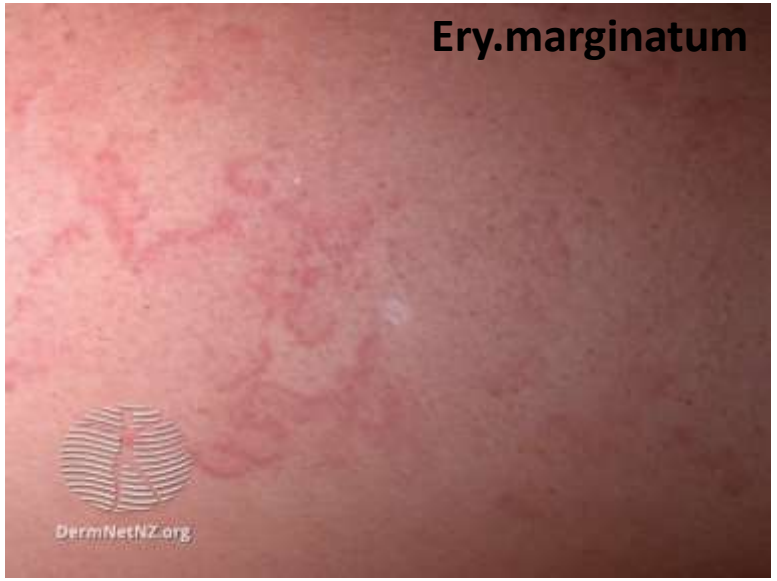


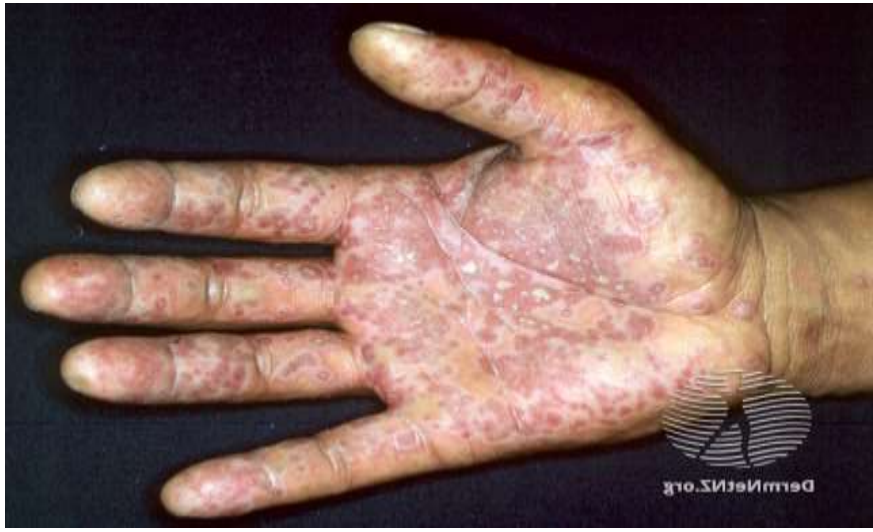
Ery.Multiforme Minor



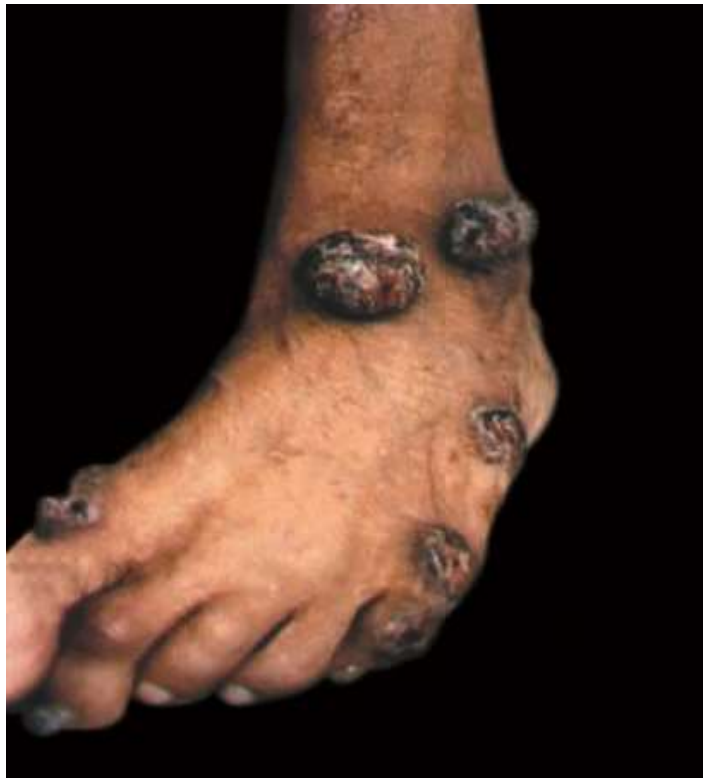

Ery.marginatum



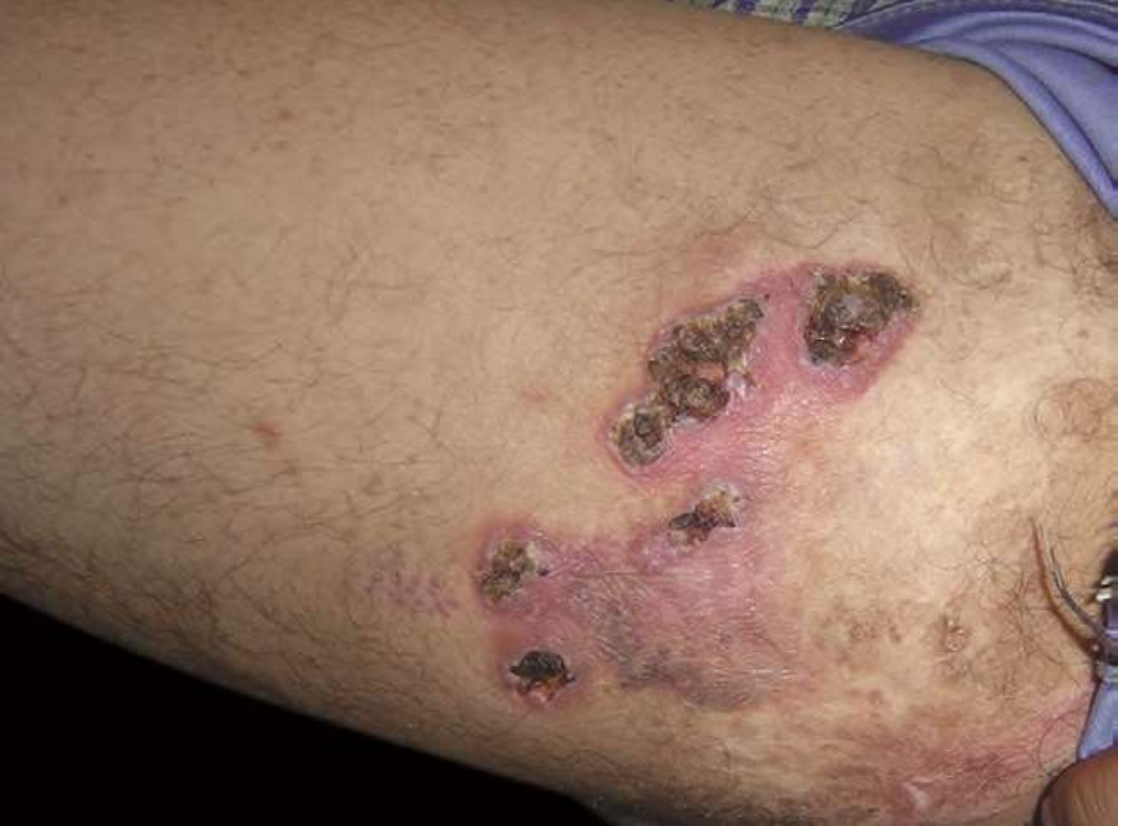
# 5.KAPOSIS SARCOMA

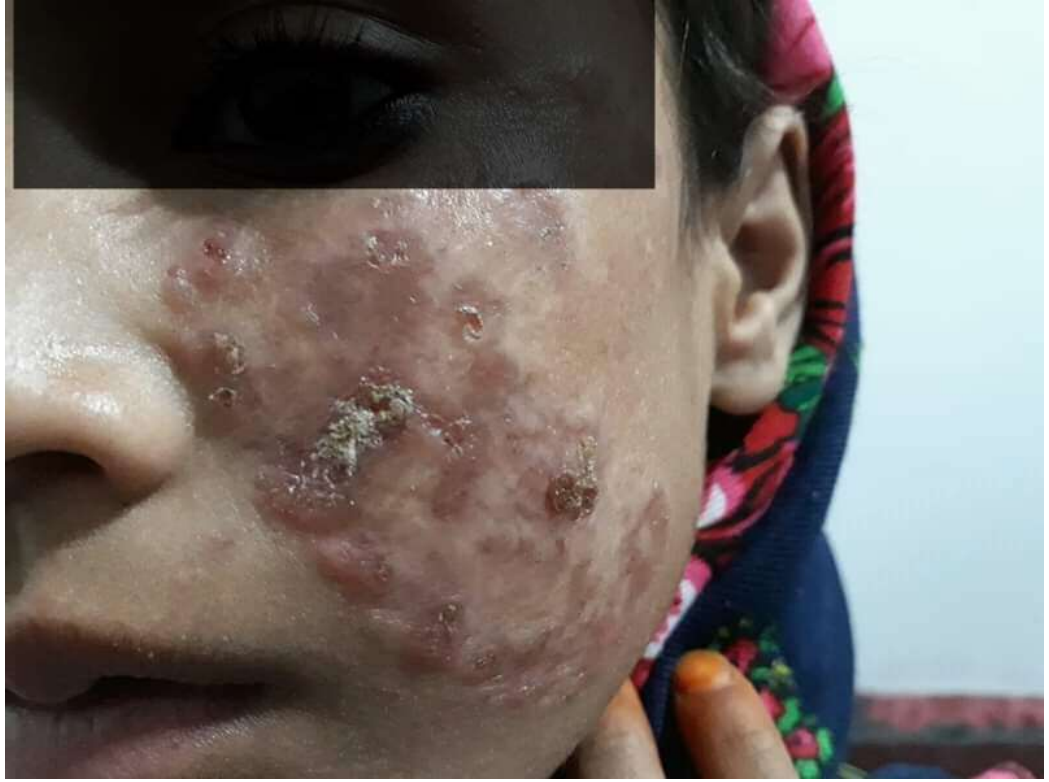
- Age group affected : all ages
- Sex predisposition : males predominantly
- C/F:presents with nodules and chronic edema of affected limbs. Feet and lower legs are usually involved bilaterally, and legs feel hard on palpation, as in neglected LL. Edema of legs may be the first manifestation (compare LL).



# LUPUS VULGARIS

- Papules Coalesce To Form Plaque Which Are Yellowish Red And Irregular In Shape.
- The Lesion Becomes White When A Glass Slide Is Pressed On It, And The Papules Then Appear As Brown ‘Apple-jelly’ Spots.
- Especially If The Lesion Is On The Face, There Can Be A Problem In Diagnosis As Hypoesthesia Is Not Seen In Leprosy Lesions On The Face And Nor Are The Nerves Consistently Enlarged In This Location.
- In This Case, Histological Differentiation Can Be Made From Tt By The Normal Appearance Of Cutaneous Nerves And Caseation. If A Therapeutic Challenge Is Offered With Anti-tubercular Treatment A Failure To Respond In 6 Weeks Is A Reliable Tool In Endemic Areas To Rule Out TB.[28]

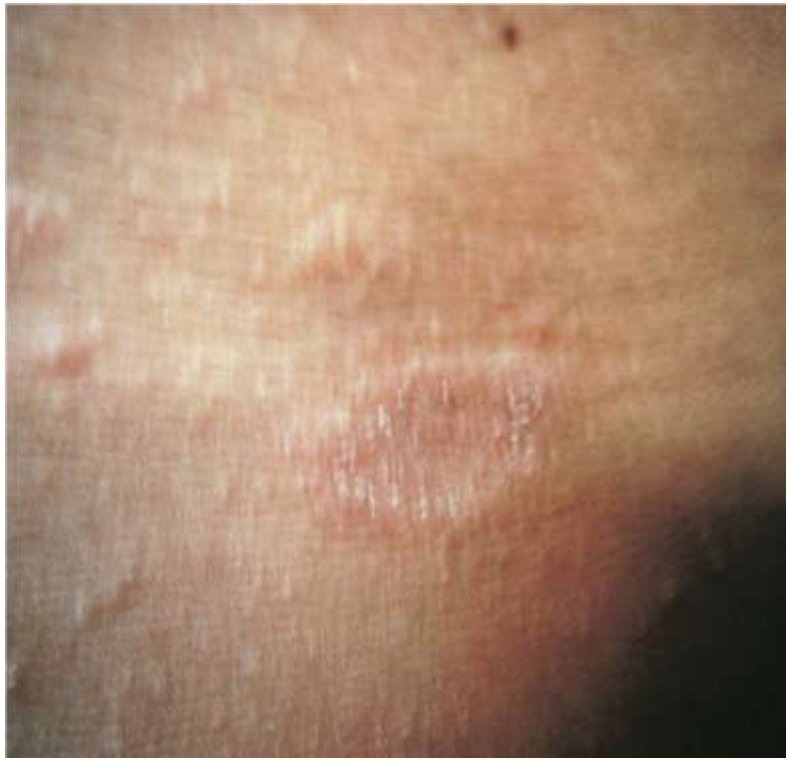




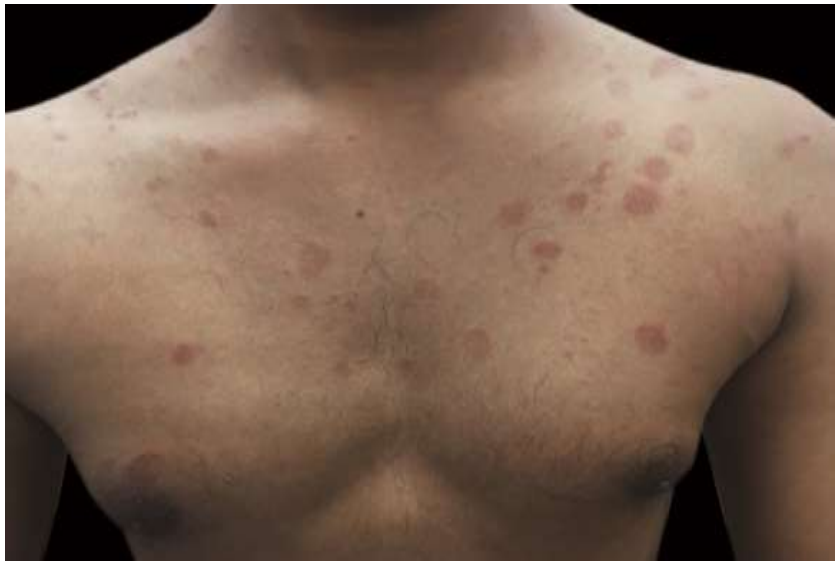
# Other DD's

- Cutaneous leishmaniasis
- Mycosis fungoides
- Subacute lupus erythematosus
- Wegeners granulomatosis
- Syphilis
- Sarcoidosis
- Pirtyriasis rosea
- Necrobiosis lipoidica
- Lupus vulgaris
- My.marinum infection

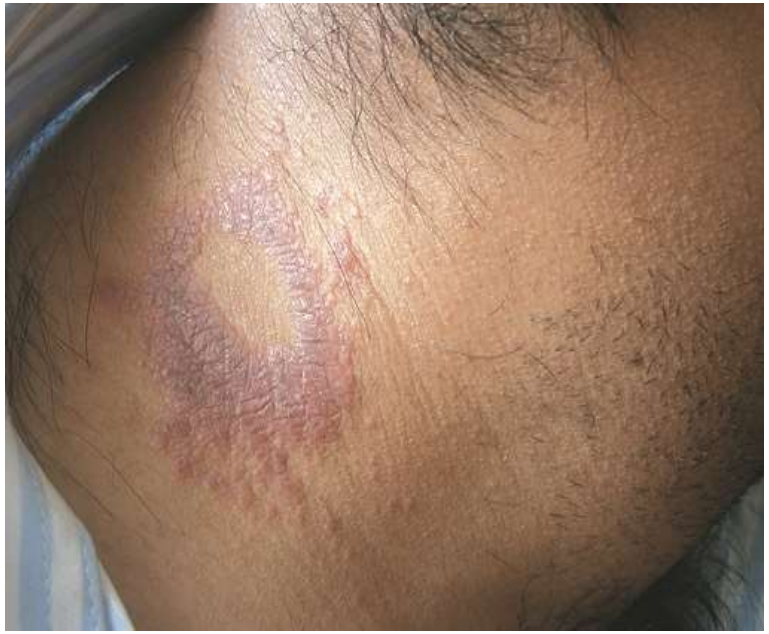
. Acquired necrobiosis of face
.Neurofibromatosis
.Nummular eczema
.Tuberculid
.Cellulitis/erysipelas
.Angiofibroma
.lymphocytoma cutis



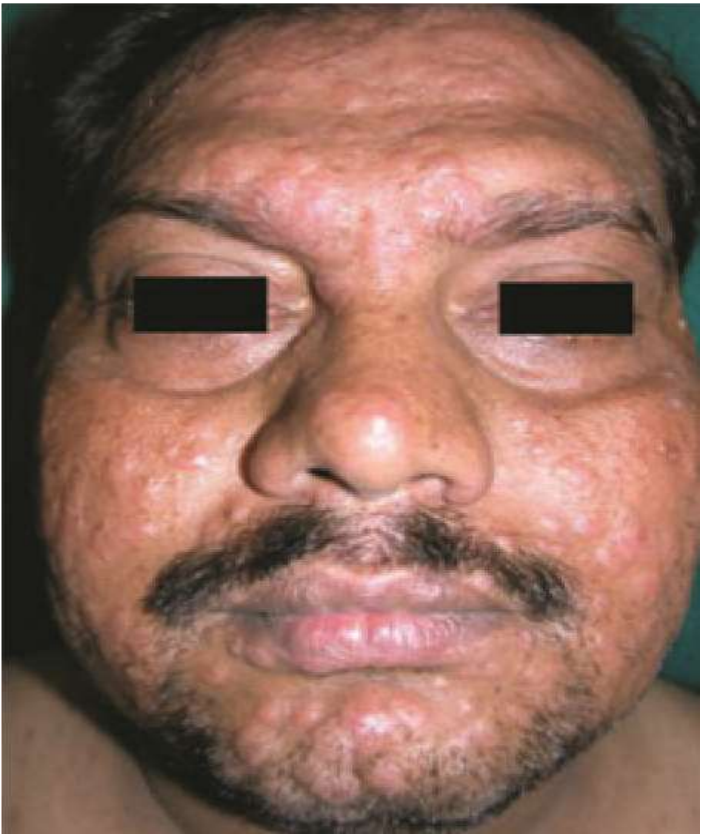
**Pityriasis rosea: Herald patch**

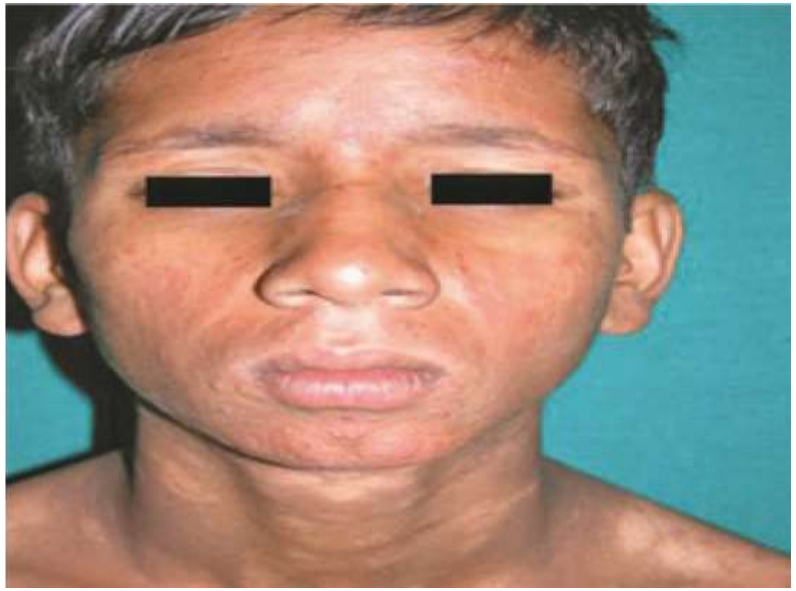


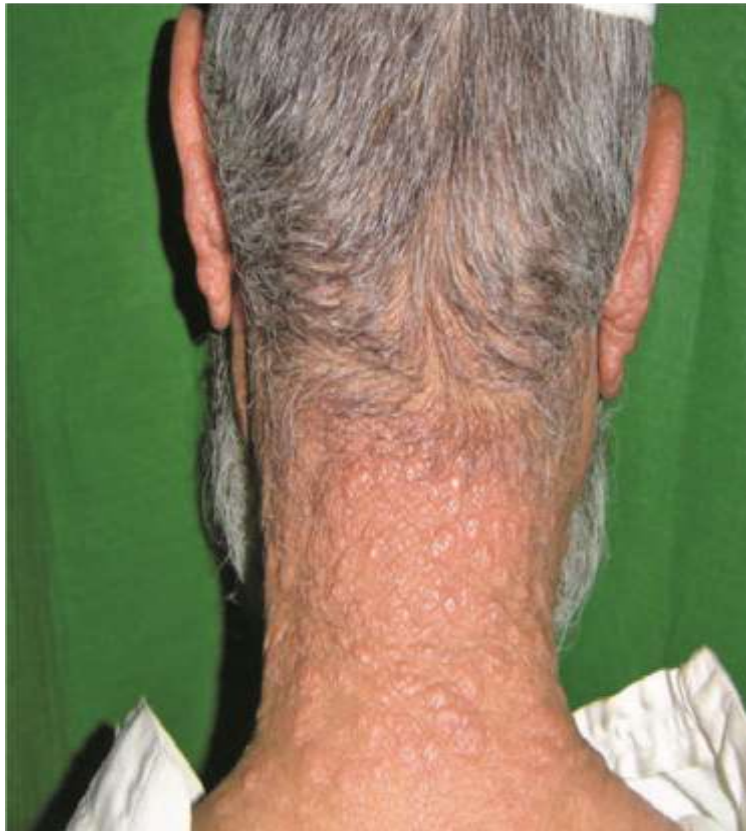
**Nummular eczema**

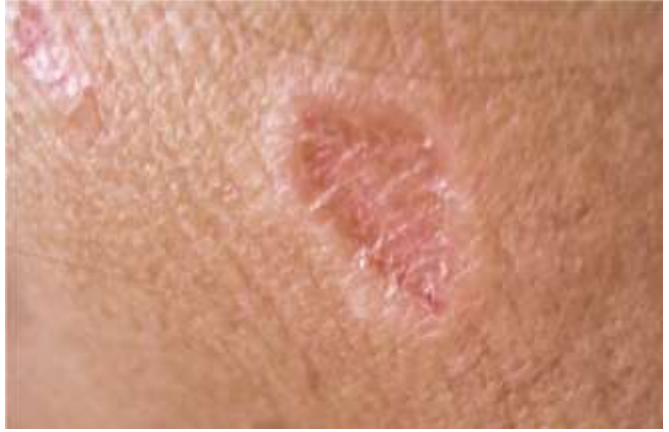


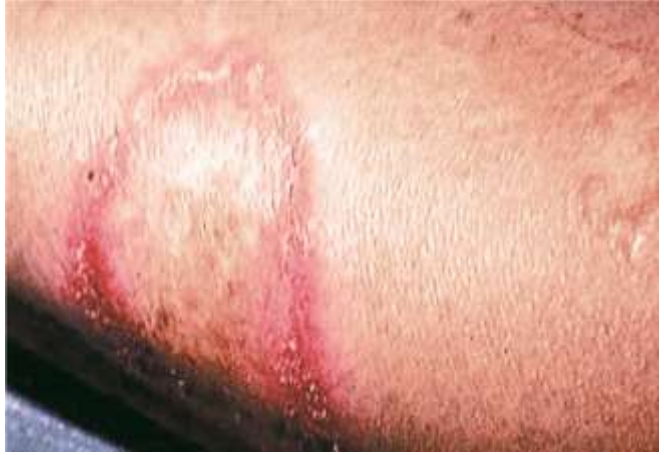
# Papulonodular lesions of PKDL

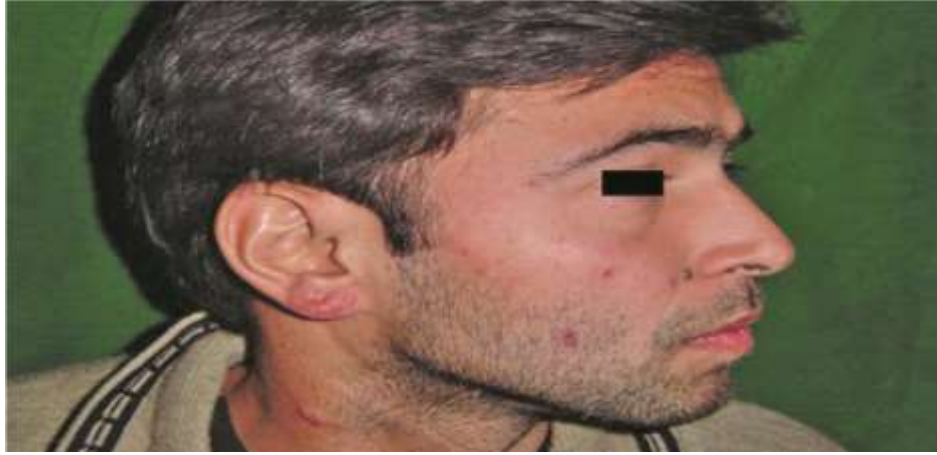


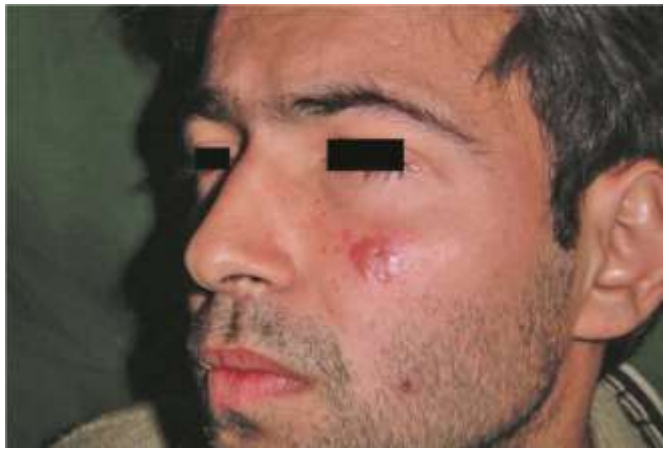


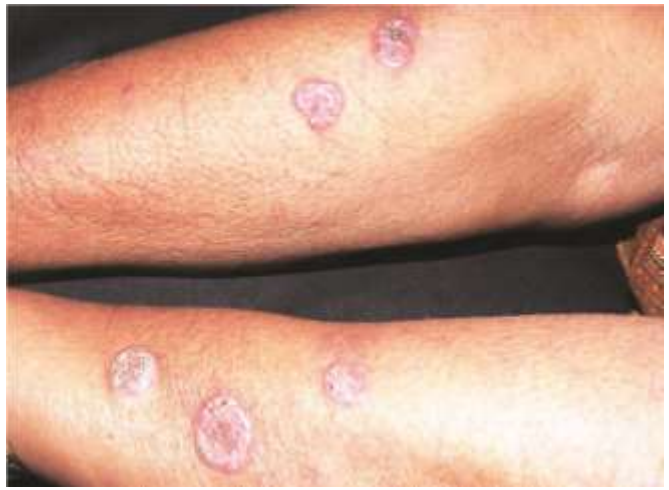


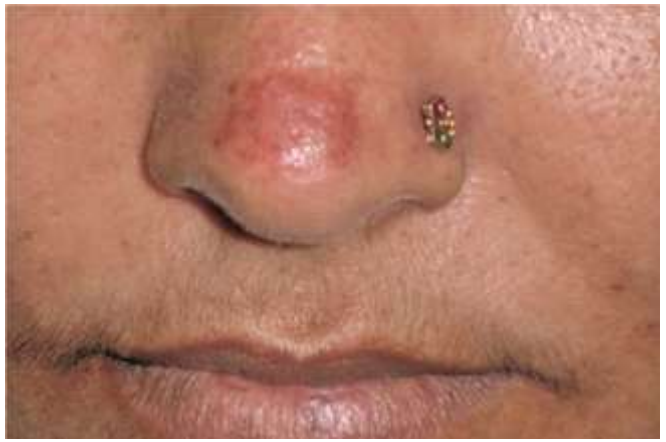


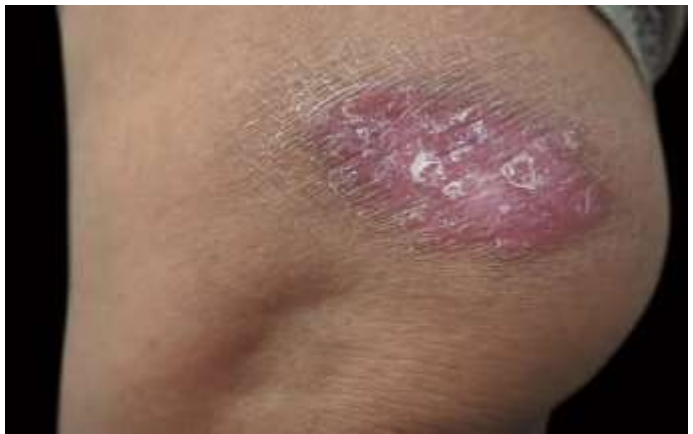


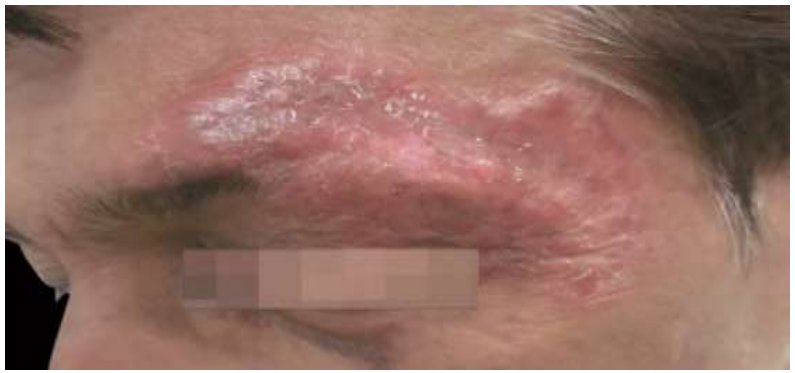




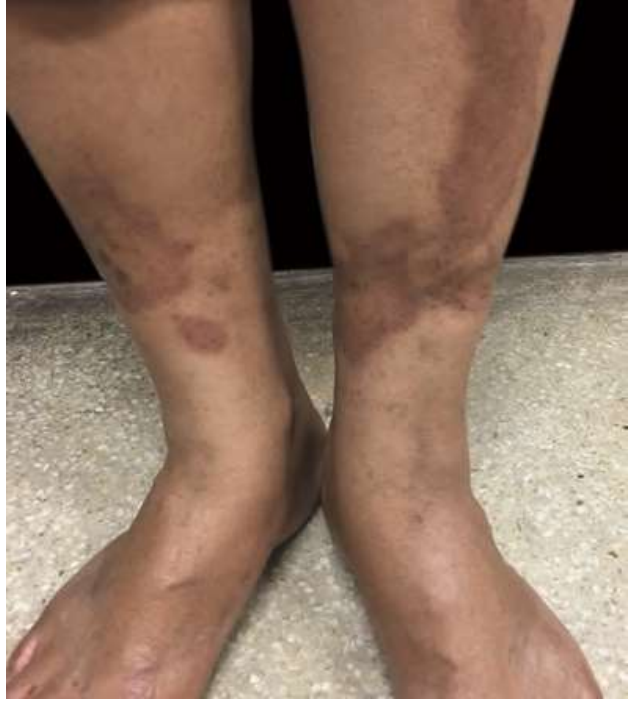






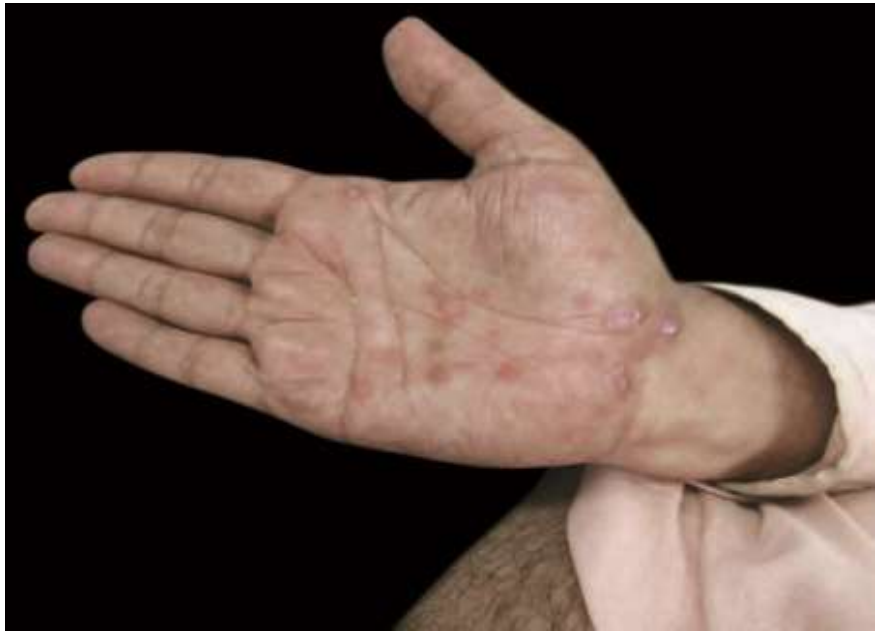


**Sarcoidosis**

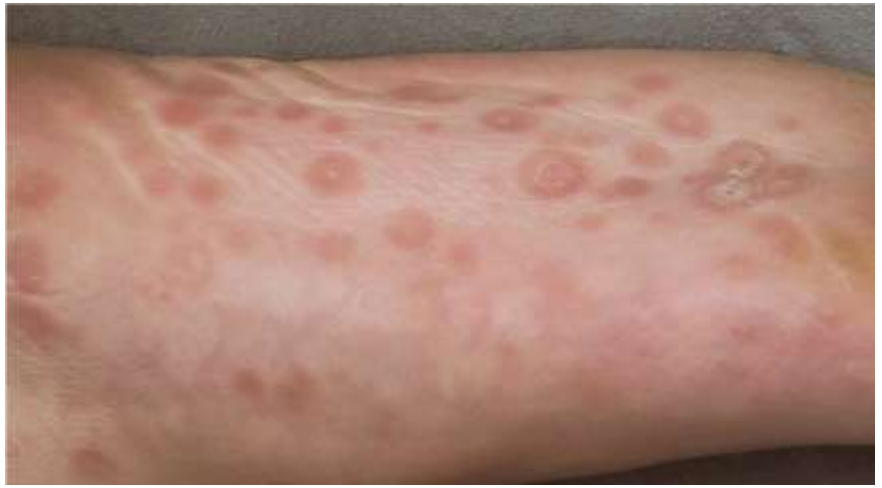


Necrobiosis lipoidica

neurofibromatosis

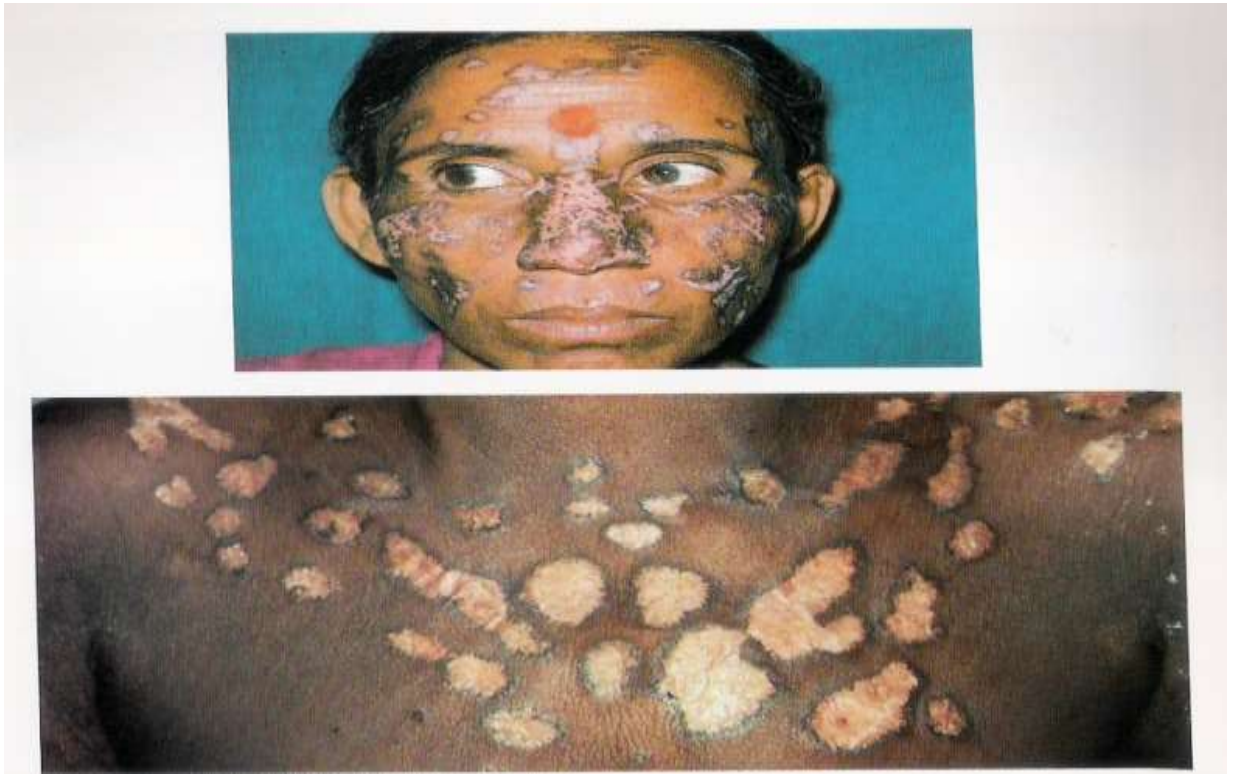


Secondary syphilis



# DISCOID LUPUS ERYTHEMATOUS

- ‘bat – wing’ distribution of lesions on the face, with some tendency towards de-pigmentation (i.e. loss of pigment). This is shown much more markedly in the lower picture of lesions on the upper chest and shoulders. Sensation and sweating are normal and peripheral nerves not affected.



# DD OF ENL LESIONS

- Cutaneous polyarteritis nodosa
- Erythema nodosum
- Nodular vasculitis/erythema induratum
- Erythema multiforme
- Cutaneous small vessel vasculitis
- Papular urticaria/prurigo nodularis

# Differential diagnosis of madarosis

- Hypothyroidism
- Follicular mucinosis
- Alopecia areata

## Differential diagnosis of acquired ichthyotic lesions:

- Nutritional/malabsorption ichthyosis
- Ichthyosis secondary to malignancies
- Drug-induced ichthyosis

# DIFFERENTIAL DIAGNOSIS OF INFILTRATED LESIONS

| | | |
|---|---|---|
| **1.** | **PKDL** | **Infiltration associated with hypopigmented macules, nodules, plaques (Figs 21.20 to 21.24). Giemsa slit-skin smear is diagnostic showing amastigotes (LD bodies). Past history of kala-azar, absence of cardinal signs of leprosy, are helpful clues** |
| **2.** | Rosacea | Diffuse erythema and thickening of skin, telangiectases, papules and pustules over central area of face |
| **3.** | Reticulohistiocytosis | Firm brown or yellow papules/plaques over extensors, face scalp hands with mutilating shortening of fingers due to arthritis. Biopsy is confirmatory |
| **4.** | Sarcoidosis | Papulonodules, diffuse facial papular eruption, plaques, diffuse or patchy ichthyotic lesions.<br>All of these may mimic leprosy (Figs 21.25 to 21.32). Biopsy will show sarcoid granulomas |
| **5.** | Disseminated cutaneous leishmaniasis | Not seen often. Widespread plaques, papules, nodules on face, extensor limbs. Resembles lepromatous leprosy. Slit-skin smear and biopsy helpful |
| **6.** | Lipoid proteinosis | Uncommon disease. Beaded papules or yellow-brown nodules, loss of eyelashes, hoarseness of voice. Histology helps in diagnosis |

*Smile Is Contagious , Leprosy Is Not*

Thank you