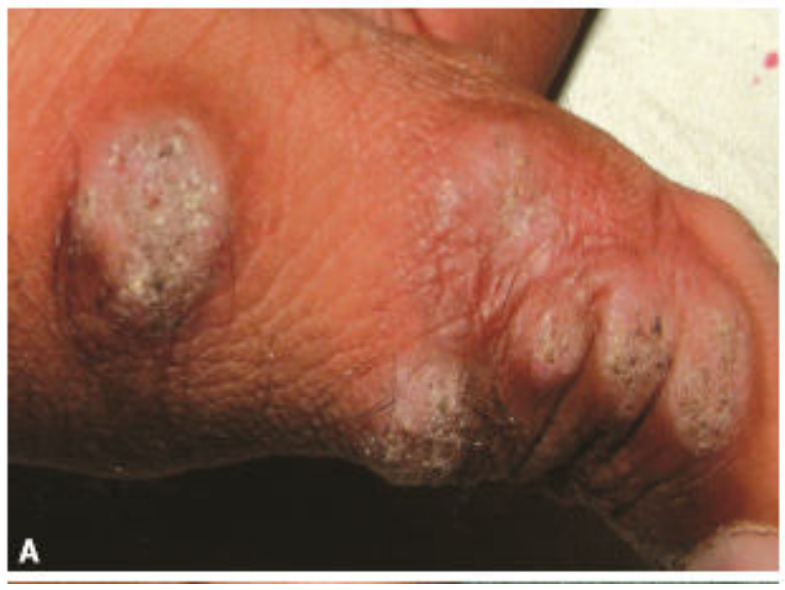
Fungal and viral infections- Problem based learning

VERRUCA VULGARIS



- Describe the lesions

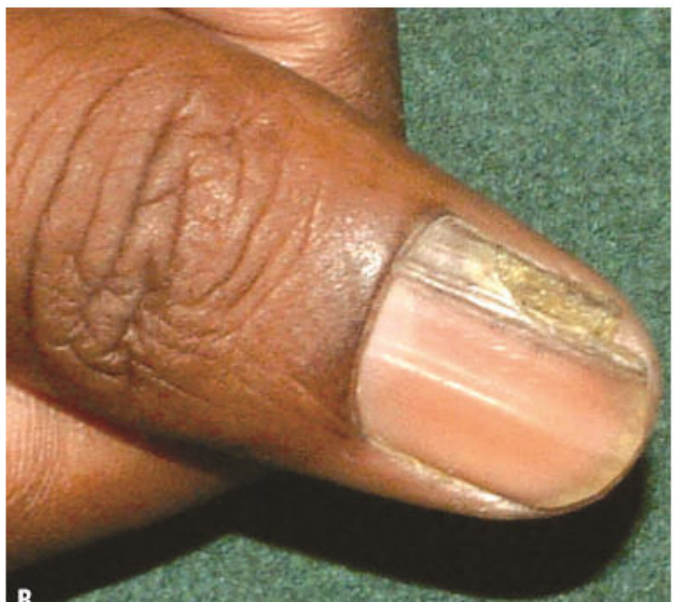
- Multiple circumscribed verrucous papules on the thumb

- Surface shows black dots

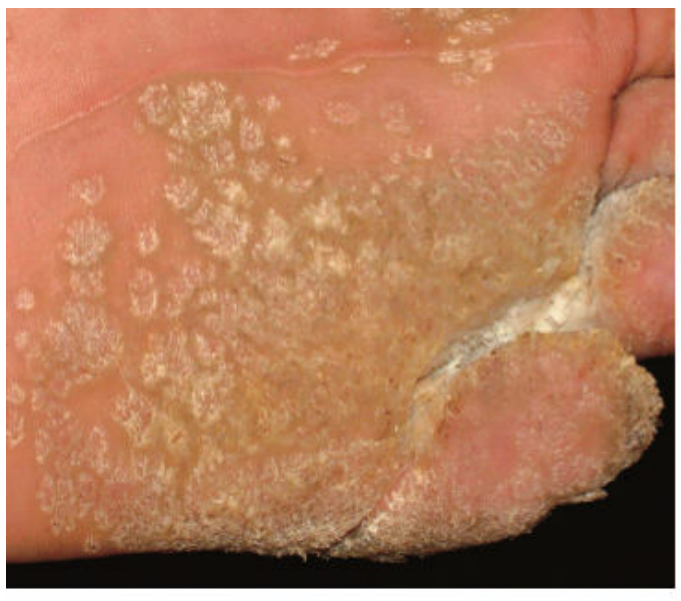
- Diagnosis:

- Verruca vulgaris

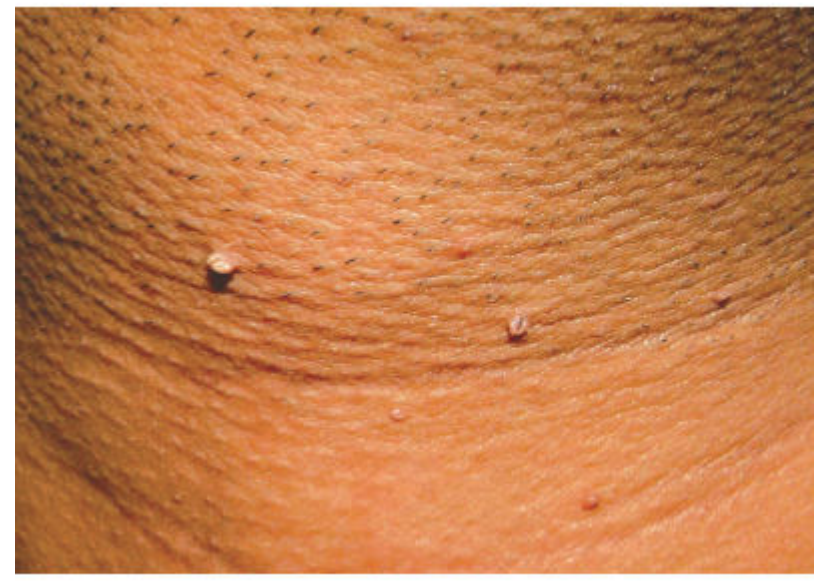
- blackish discoloration-
- Capillary thrombosis
- Wart resolves with no sequalae



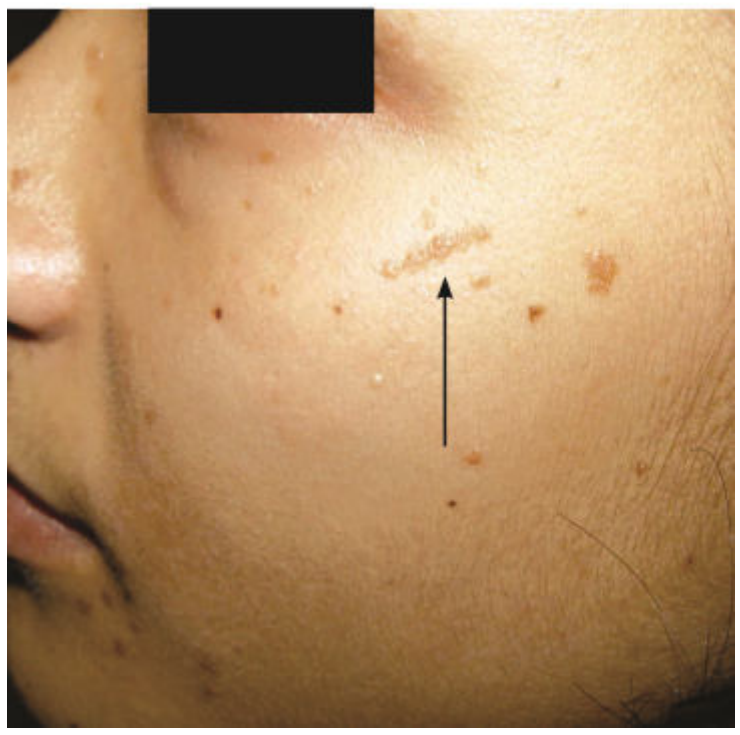
- ??
- Subungual verruca
- Causative agent?



plantar warts

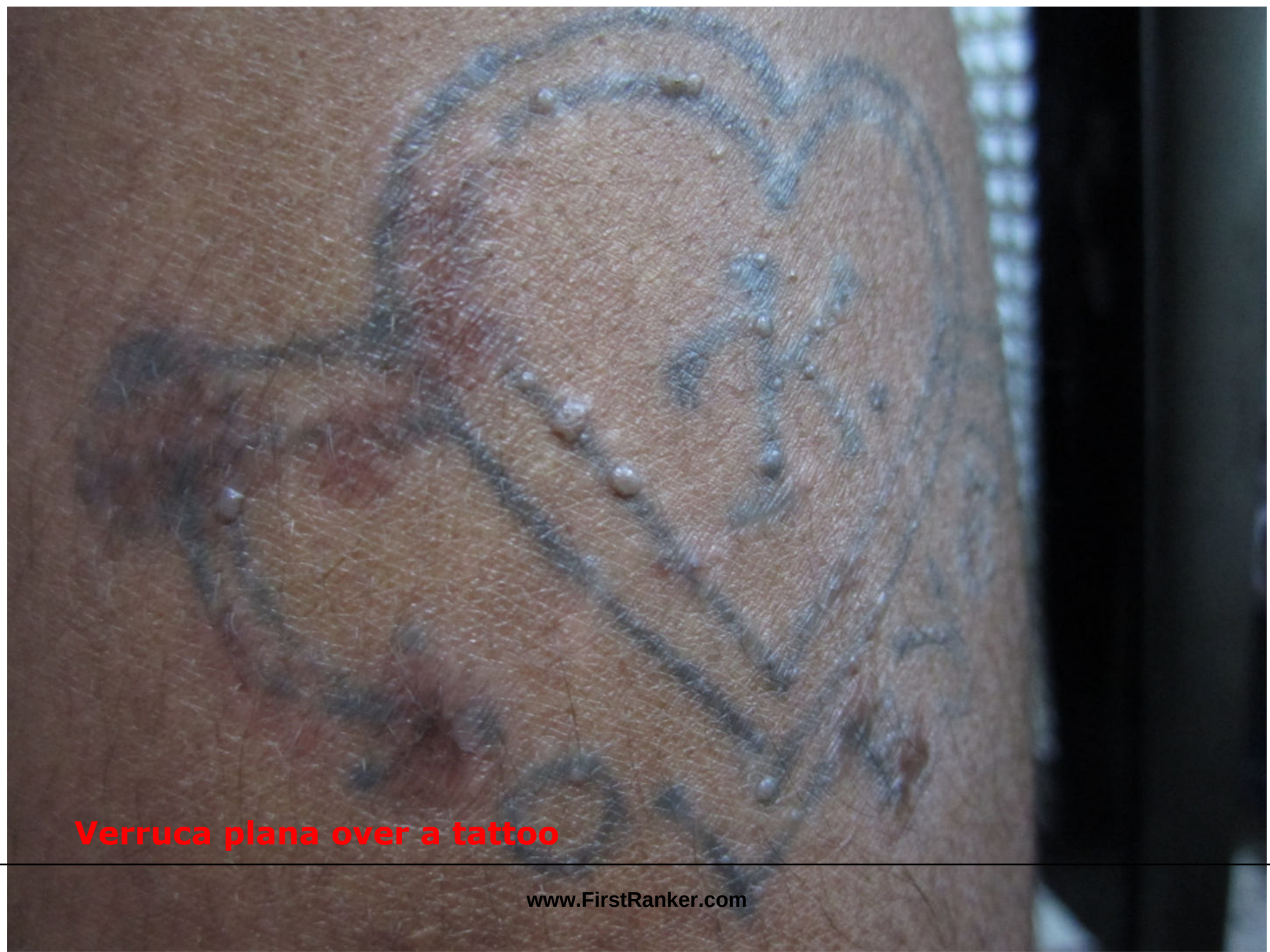


Filiform warts

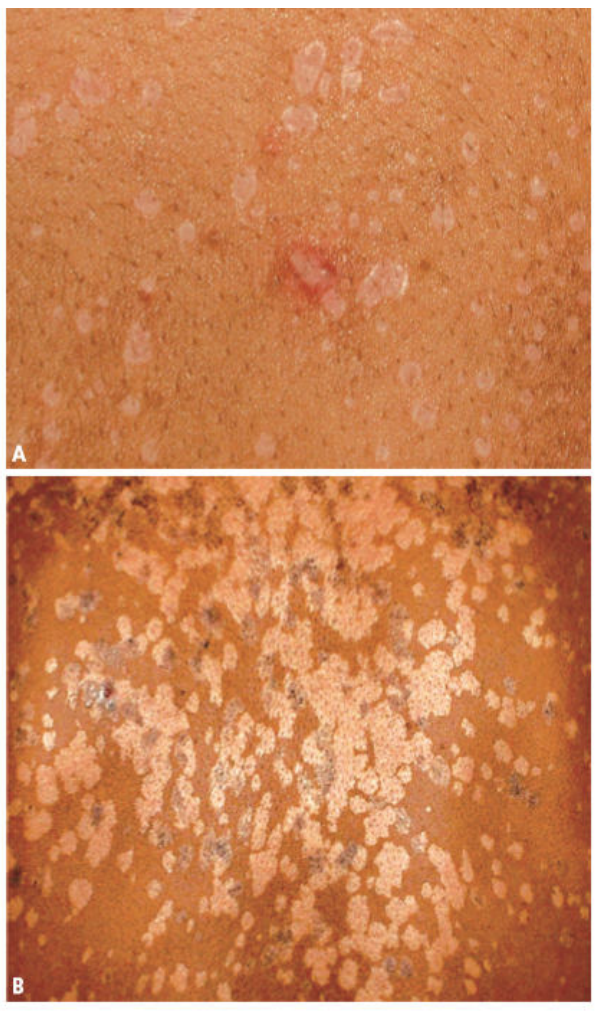


Verruca plana

- ??
- Pseudokoebnerisation- Lesions may be arranged linearly (pseudo Koebner's phenomenon) due to auto-inoculation.

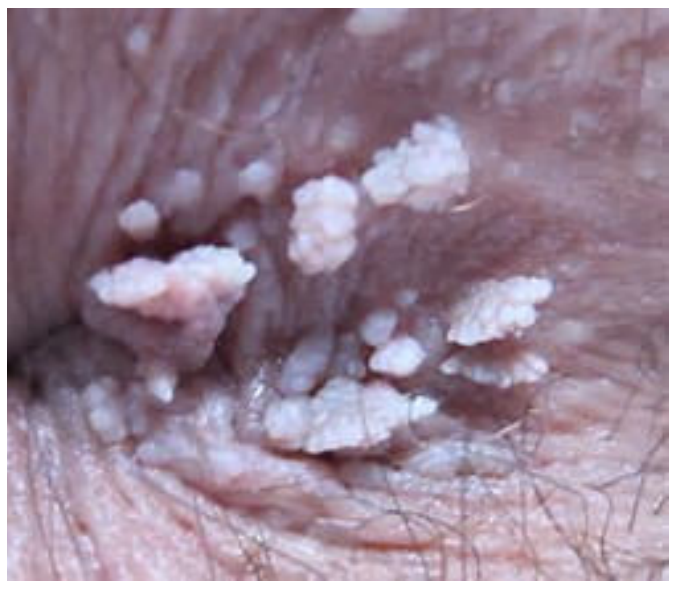


Verruca plana over a tattoo



- Epidermodysplasia verruciformis
  Rare inherited disorder, characterized by defective cell-mediated immunity to certain types of HPV (3, 5, 8, 9) resulting in wide spread lesions.

- Plane wart-like lesion
- Pityriasis versicolor-like irregular, scaly macules

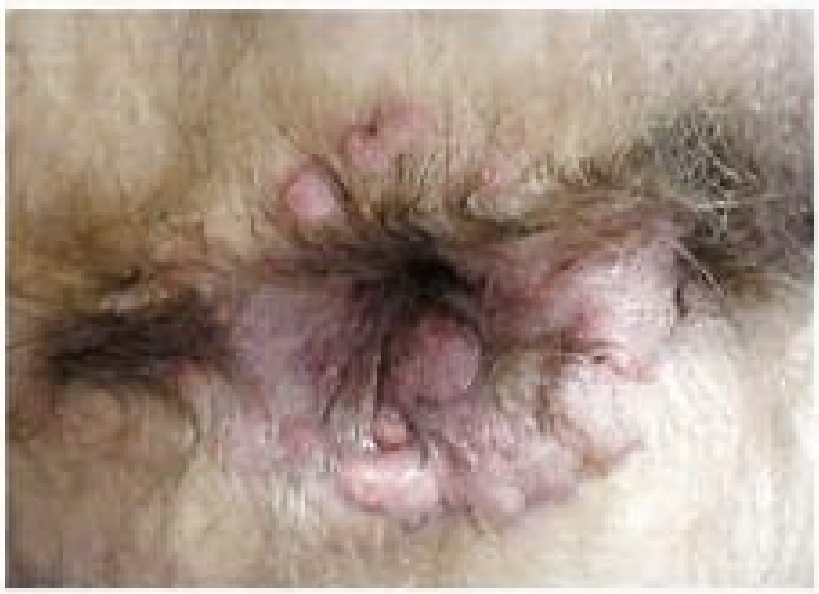


- Anogenital warts
- Sexually transmitted disease.

- A variety of clinical variants, e.g., condyloma acuminata, papular warts, and Bowenoid papulosis.

- Most frequently on the glans, perianal region, vulva, and cervix.

- Differential…..



- Condyloma lata
- Secondary syphilis

Table 14.11. Clinical manifestations and HPV type

Clinical manifestations	*HPV type*
Verruca vulgaris	2, 4, 27
Palmoplantar warts	1, 2, 4, 57
Verruca plana	3, 10
Epidermodysplasia Verruciformis	3, 5, 8, 9
Anogenital warts	6*, 11*, 16**, 18**, 31**, 33**

*Low oncogenic potential
**High oncogenic potential

- Diagnosis
- Characteristic warty appearance with a rough, dry stippled surface.
- Presence of pseudo Koebner's phenomenon, especially in plane warts.
- Typical histology

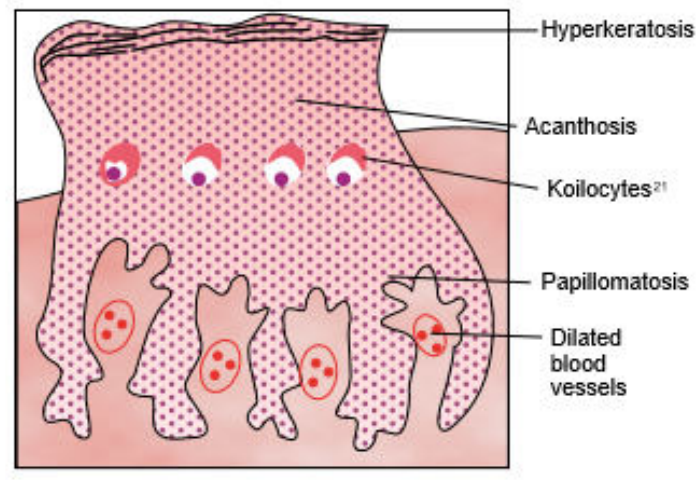
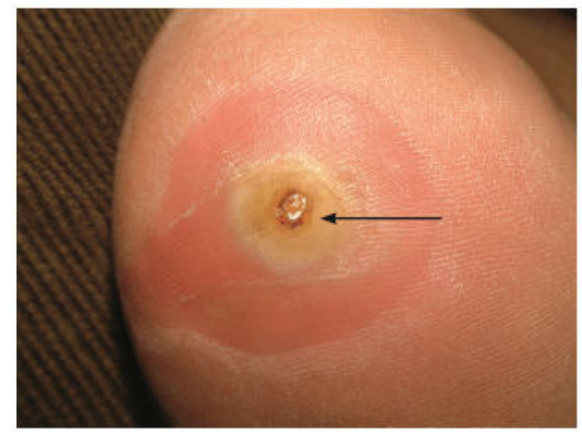


Fig. 14.48. Verruca vulgaris: histology.

Corns	Plantar warts
Location: at points of pressure	Anywhere
Skin markings: continue over lesions	Skin markings interrupted
On paring: keratinous core seen	Black dots seen



- Treatment
- More than 50% of warts resolve spontaneously

- Cryotherapy
- Cryogens: Liquid nitrogen, carbon dioxide, and nitrous oxide. Technique: A cotton-tipped applicator dipped in cryogen is applied firmly to the wart till a small halo of freezing appears on adjoining normal skin. Or can be sprayed using a cryocan.

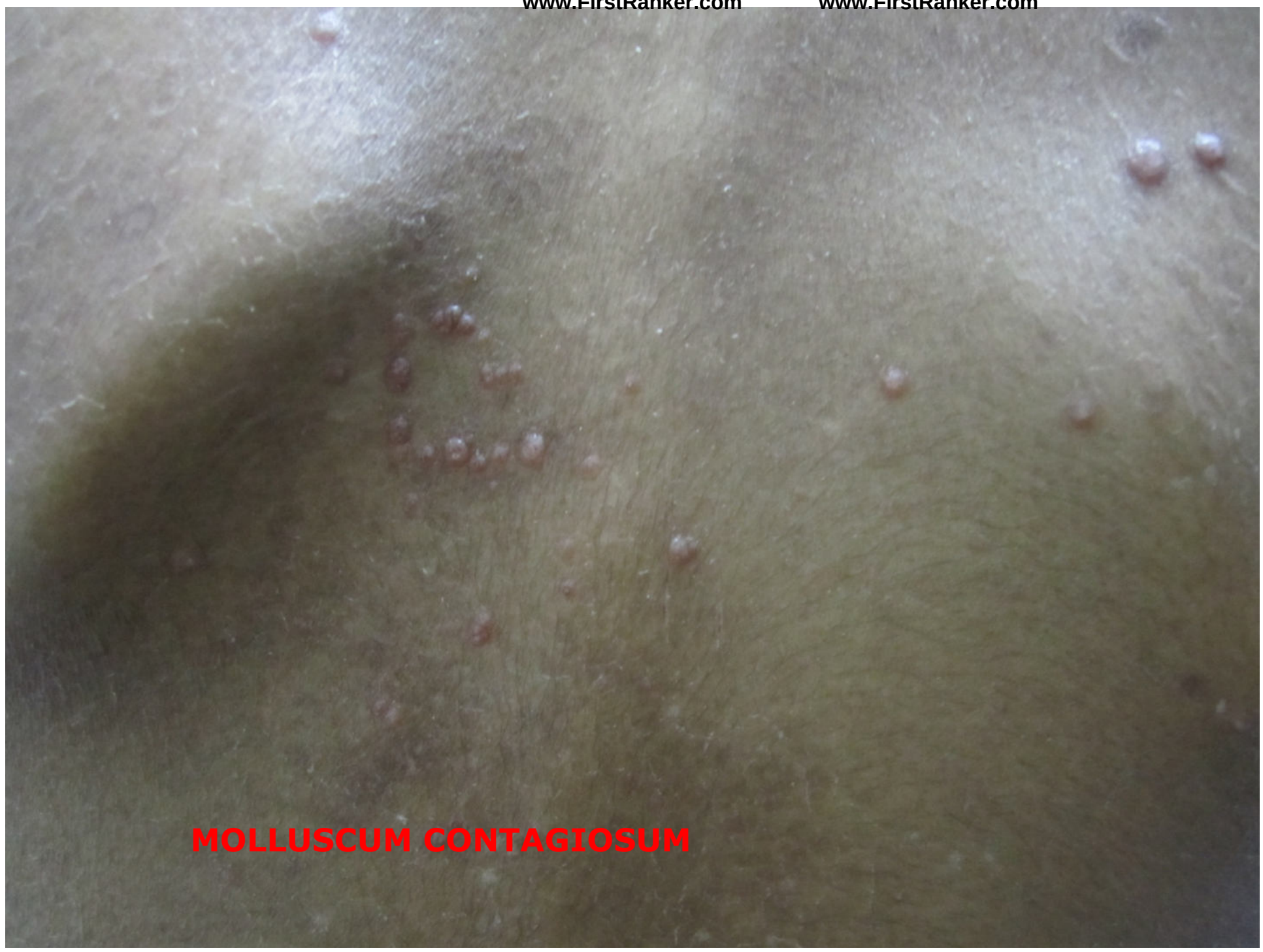
- Electric cautery and radiofrequency ablation (RFA)

- Topical agents
- Salicylic acid (10–25%)

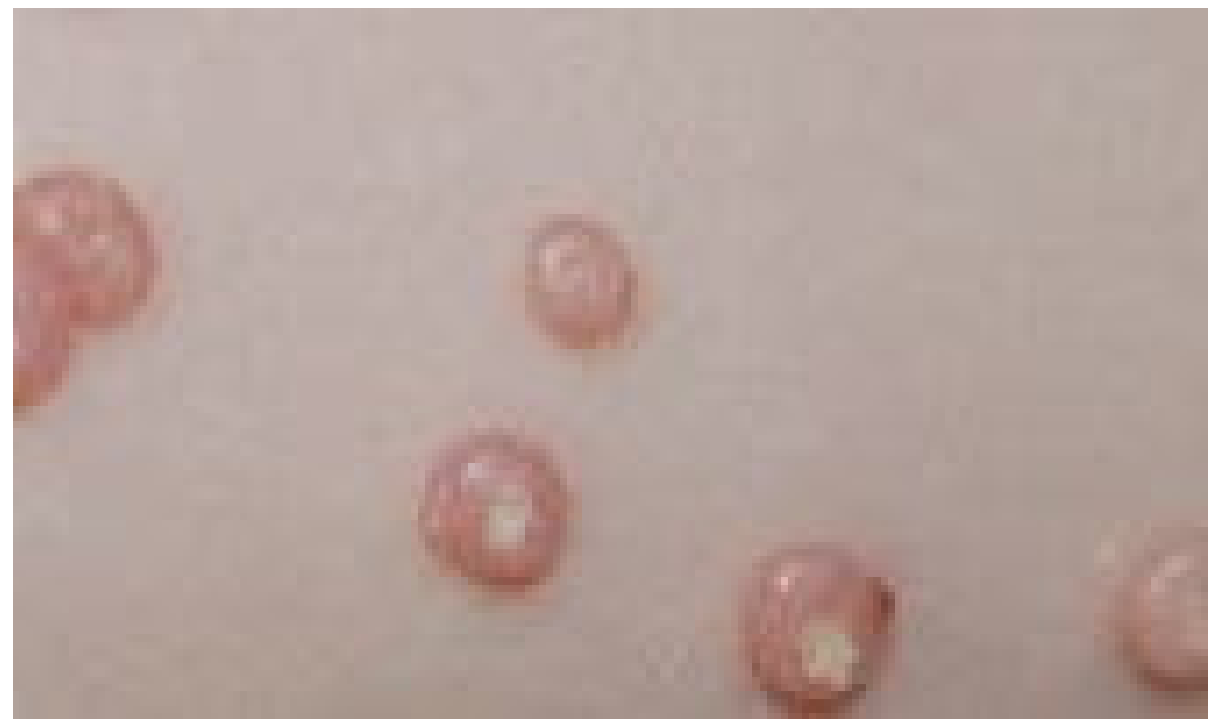
- Wart paint: Contains salicylic acid (a keratolytic agent) and lactic acid in a quick drying collodion or acrylate base.

- Retinoic acid (0.05–0.1%):

MOLLUSCUM CONTAGIOSUM



- Diagnosis of MC is based on:
- Presence of pearly white umbilicated papules.
- Extrusion of the cheesy core through the central crater; characteristic cytological appearance of the expressed material.

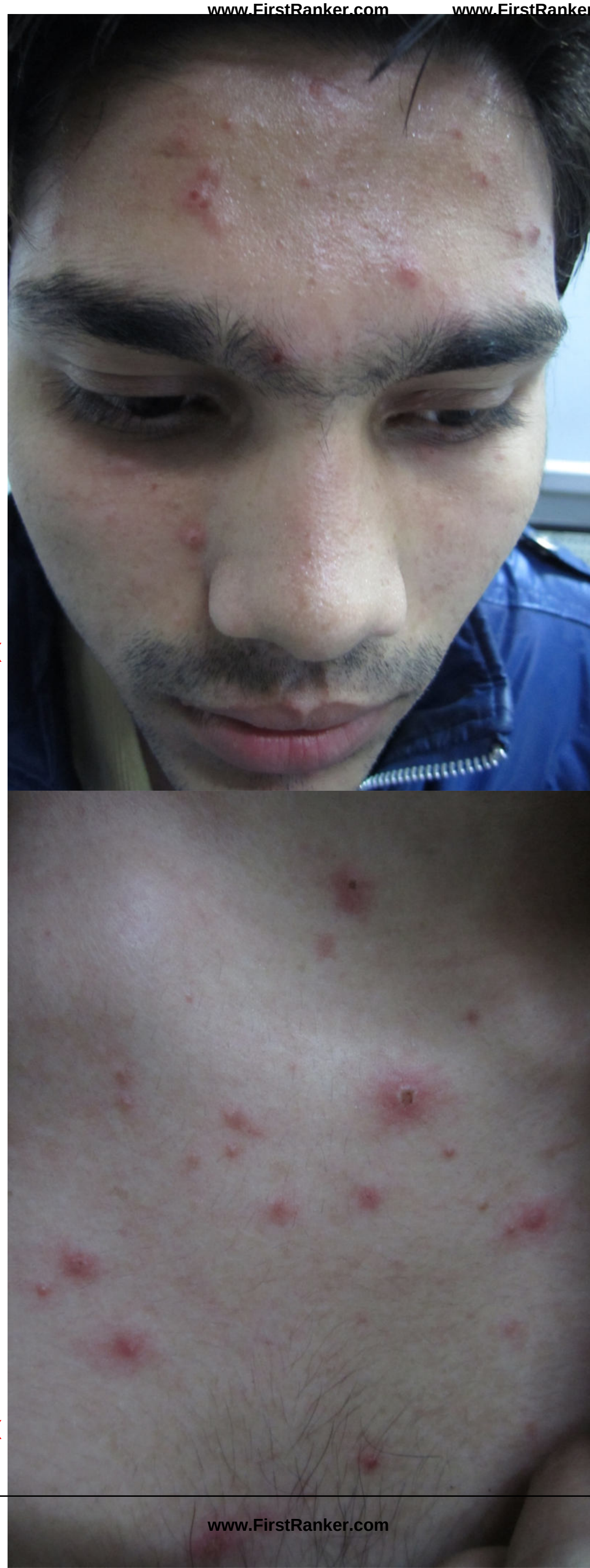


- Pseudoisomorphic phenomenon
- Anogenital region: what should you suspect
- Sexually transmitted MC
- In adult patients with extensive and persistent lesions------- what should you suspect?
- underlying HIV infection should be ruled out.
- Course -Self-limiting.
- Except in immunosuppressed and atopics

Table 14.13. Treatment options in molluscum contagiosum

Children	Few lesions	May resolve spontaneously
	Several lesions	Wart paint Mechanical extirpation followed by chemical cautery after using EMLA[23]
Adults	Few lesions	Mechanical extirpation followed by chemical cautery
	Several lesions*	Cryotherapy Wart paint

*Rule out underlying HIV infection



CHICKEN POX

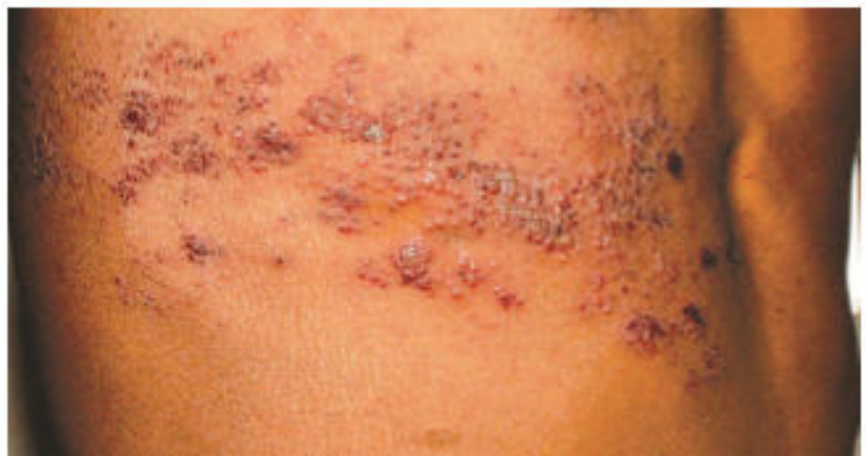
CHICKEN POX

- Morphology:
- Crops of papules with erythematous halo; r
- apidly become vesicular (dew drops on rose petal appearance), then pustular.
- Eruption at different stages present.
- Heal with minimal scarring unless complicated by secondary infection or hemorrhagic lesions (as seen in immunocompromised).
- Adults usually have a more severe eruption.

- Site: Centripetal distribution.

- Treatment: Specific antiviral therapy (acyclovir 800 mg, five times daily × 7–10 days) in adults and in immunocompromised individuals.

- None needed in children.



- ??

- Herpes Zoster
- Causative agent?
- Varicella-zoster virus

- Morphology??

- Very painful, segmental eruption of grouped papules and vesicles on an erythematous, slightly edematous base

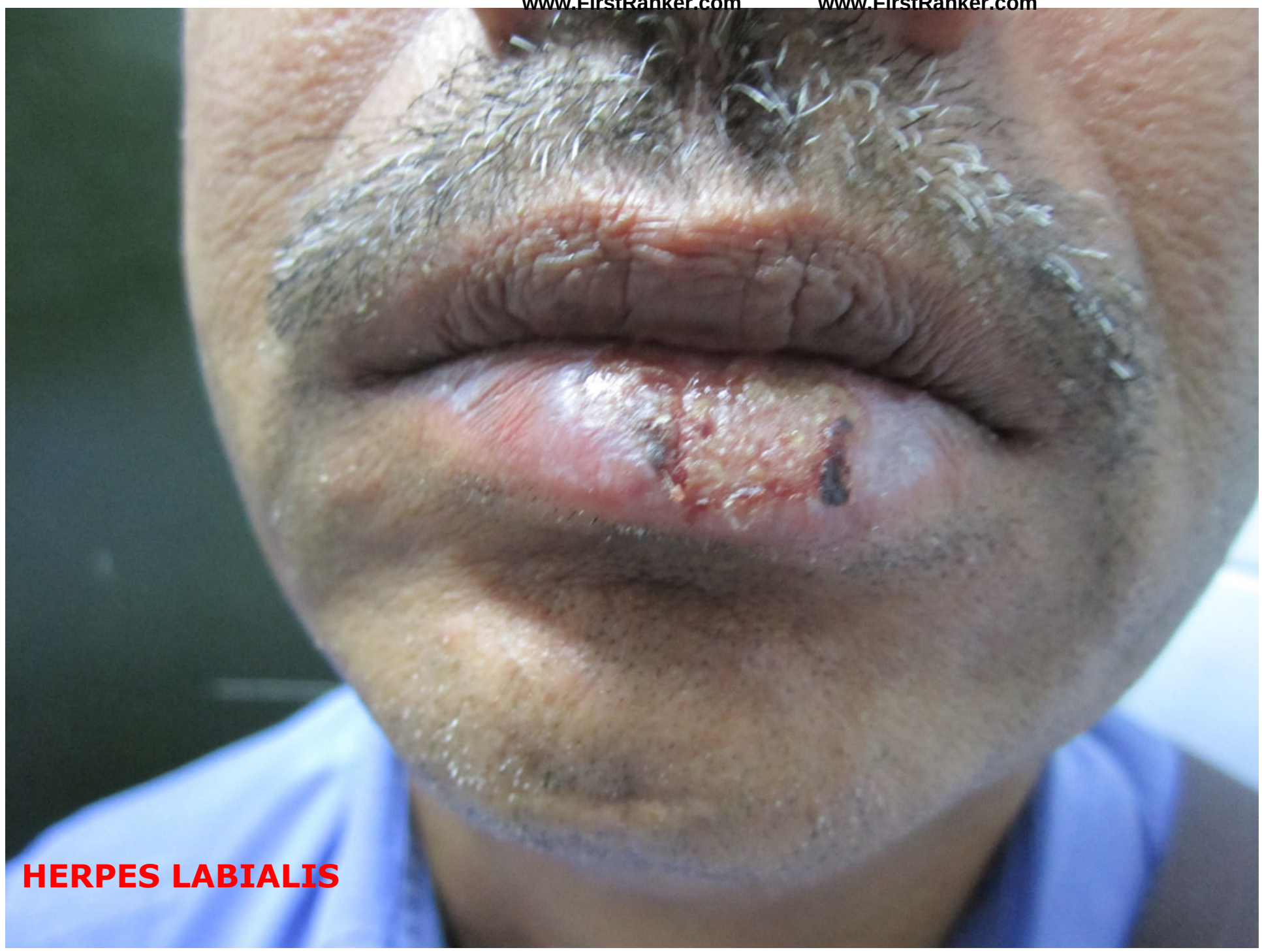
- Predisposing factors for reactivation are:
- Old age.
- Lymphoreticular malignancies, e.g., Hodgkin’s disease and leukemia.
- Human immunodeficiency virus infection.
- Sometimes without apparent cause.

- Sites:
- Thoracic intercostal nerves,
- ophthalmic division of trigeminal nerve.
- Complications ??
- Postherpetic neuralgia
- corneal ulcers and scarring------- Eye involvement is indicated when vesicles are present on the side of the nose (Hutchison's sign).
- Secondary bacterial infection
- Generalized ----in immunocompromised individuals and in those with internal malignancies.

- Investigations
- giant cells on cytopathology is confirmatory.
- if disseminated hemorrhagic lesions present----????
- Rule out an underlying immunodeficiency (lymphoreticular malignancies and HIV infection)

- Treatment:
- Self-limiting.
- Symptomatic treatment with analgesics.
- Specific treatment with antivirals (acyclovir, famciclovir, and valacyclovir) in: (a) ophthalmic zoster (b) immunocompromised (c) severe zoster (hemorrhagic lesions), and (d) elderly (to reduce postherpetic neuralgia).

- Acyclovir: 800 mg, five times a day × 7 days (adult dose).
- Famciclovir: 500 mg, three times a day × 7 days.
- Valacyclovir: 1 g, three times a day × 7 days



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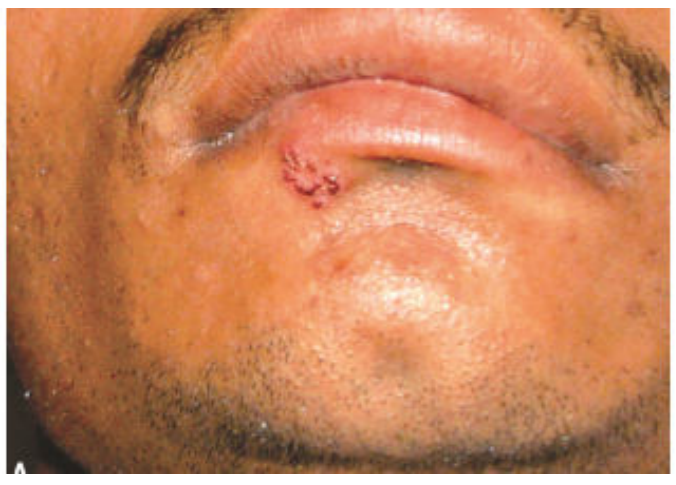
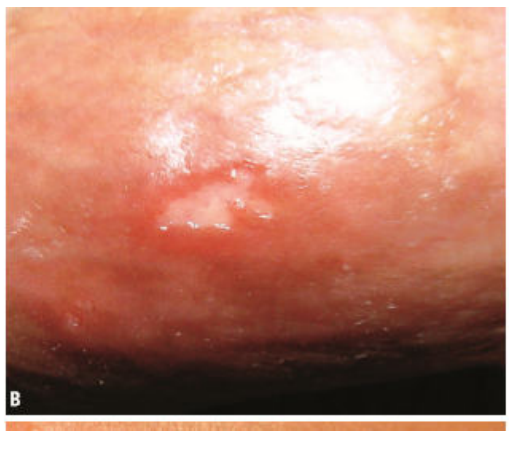
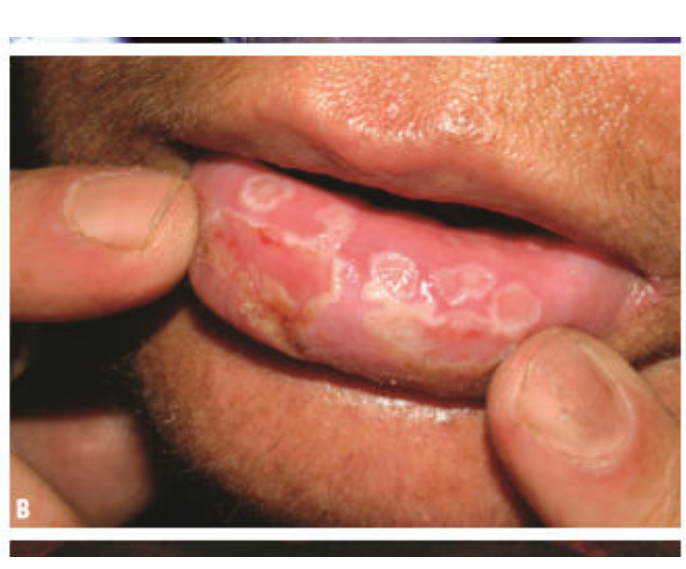


- grouped vesicular lesions



- ??
- erosions with polycyclic margins.



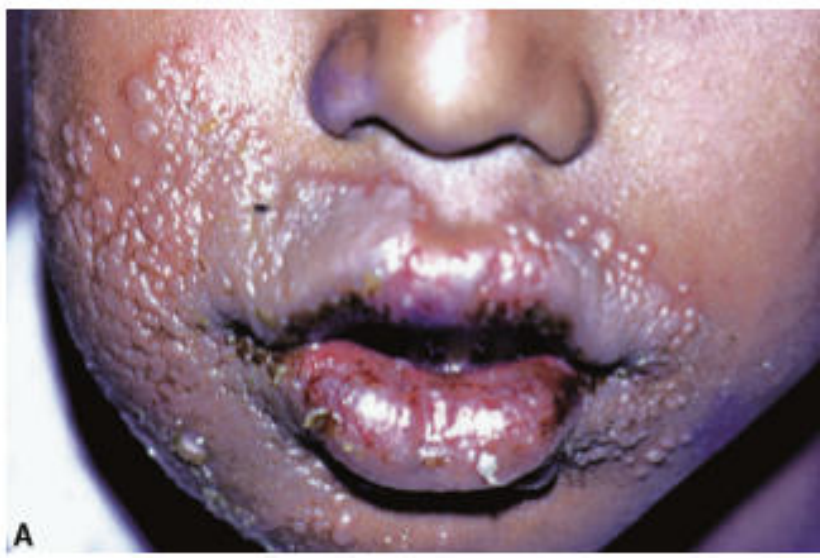
- erosions with polycyclic margins.

- Cause?
- HSV
- Why difference in severity?
- Primary infection, more severe and associated with constitutional symptoms.
- Recurrent infection manifests as grouped papulovesicular lesions which rupture to form polycyclic erosions.

- HSV (type I and type II).
- Type I generally causes lesions above the waist,
- while type II causes genital infection.
- After primary infection (first infection), the virus lies dormant in sensory ganglion and
- gets activated from time to time.

- ?? Transmission
- Direct contact: Usually occurs in children from an infected adult who is often asymptomatic yet shedding virus.

- Sexual contact: Usually in adults, herpes genitalis being the most common cause of infective genital ulcer disease. Again asymptomatic shedding important.



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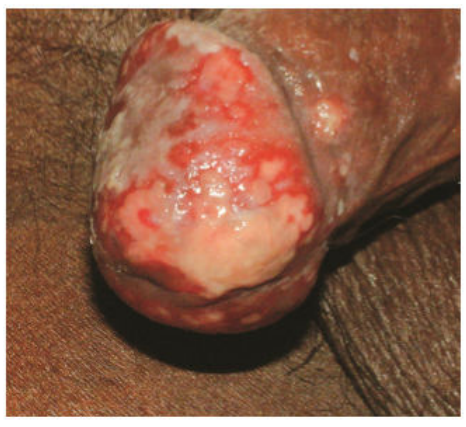
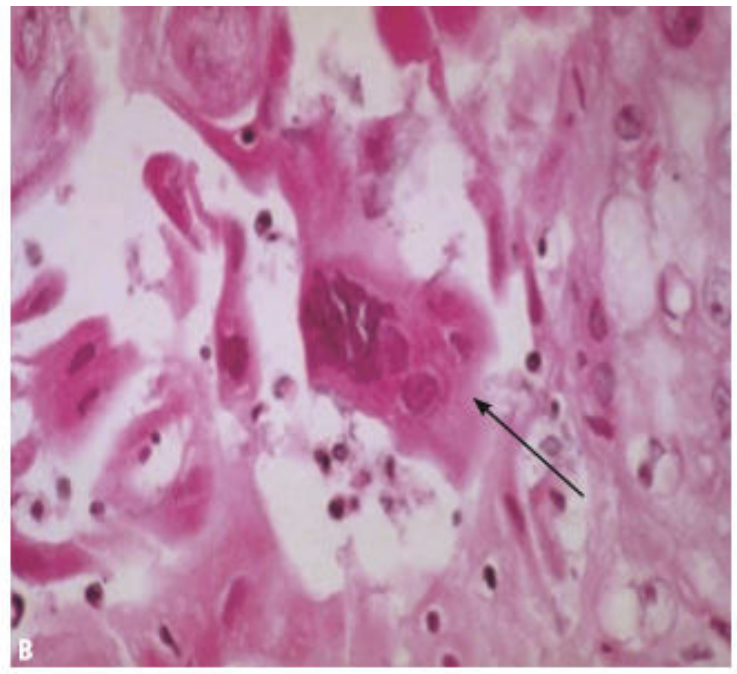
- Primary HSV infection
- may present as acute gingivostomatitis: characterized by closely grouped vesicles which rapidly form polycyclic ulcers covered with a yellow pseudomembrane. Heal in about a fortnight
- Malaise, fever, and lymphadenopathy are frequent.



- ??
- Primary herpetic genital infection

- Bed side test that u would do ????

- Tzanck smear -
- multinucleated giant cells



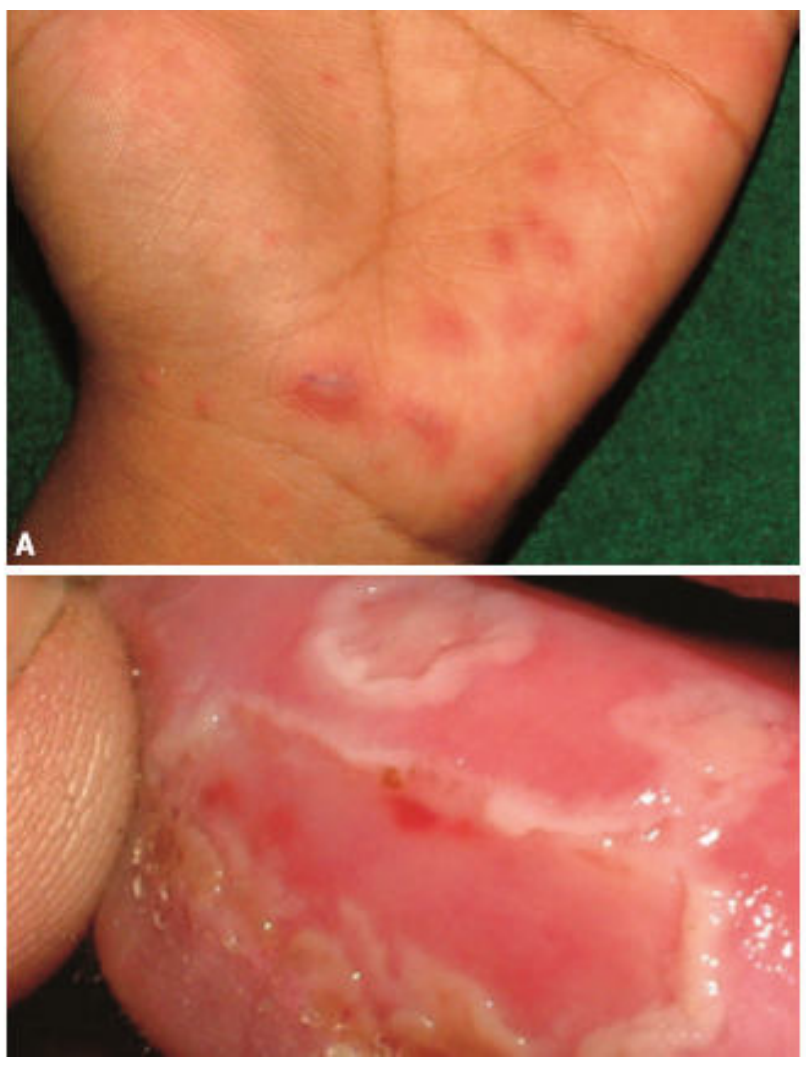
Tzanck smear, showing multinucleated giant cells

- Serology:
- Antibody titers rise with primary infection (IgM initially, IgG later).
- Though IgM levels fall, IgG levels persist.
- Are of doubtful diagnostic significance in recurrent infections but
- help in primary infection.

Herpes zoster	*Herpes simplex*
Prodrome: of dermatomal pain	Of burning and stinging
Morphology: grouped vesicular lesions on an erythematous, edematous skin. Multiple such groups in dermatomal distribution	Grouped vesicular lesions on slightly erythematous skin; lesions in primary episode covered with pseudomembrane
Recurrences: single episode	Recurrences at same site

Table 14.15. Principles of treatment HSV infection

First episode	
	Acyclovir, 200 mg, five times/day × 7 days Famciclovir, 250 mg, three times/day × 7 days Valacyclovir, 1 g, two times/day × 7 days
Recurrent episodes	
Herpes labialis	Symptomatic treatment
Herpes genitalis	Either: ❖ Episodic treatment with acyclovir, 200 mg, five times/day × 5 days ❖ Suppressive treatment (if >6 episodes/year): acyclovir 400 mg twice daily for 12 months.
Complications	
Disseminated infection Eczema herpeticum Encephalitis	Parenteral acyclovir
Immunosuppressed patients	Suppressive treatment

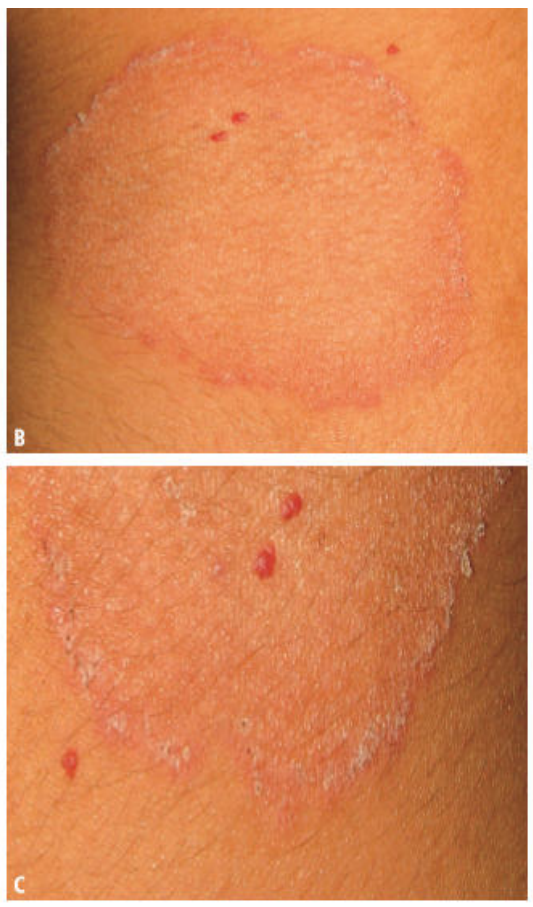


- Morphology??
- painful oblong vesicles on hands
- erosions in oral mucosa

- Hand, Foot, and Mouth Disease

- Treatment: Symptomatic.

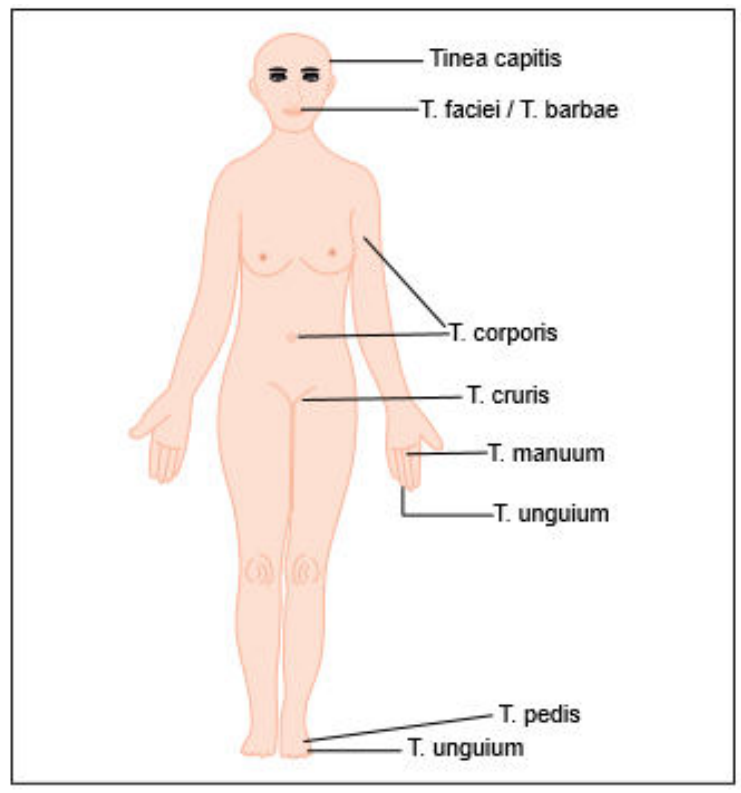
FUNGAL INFECTIONS



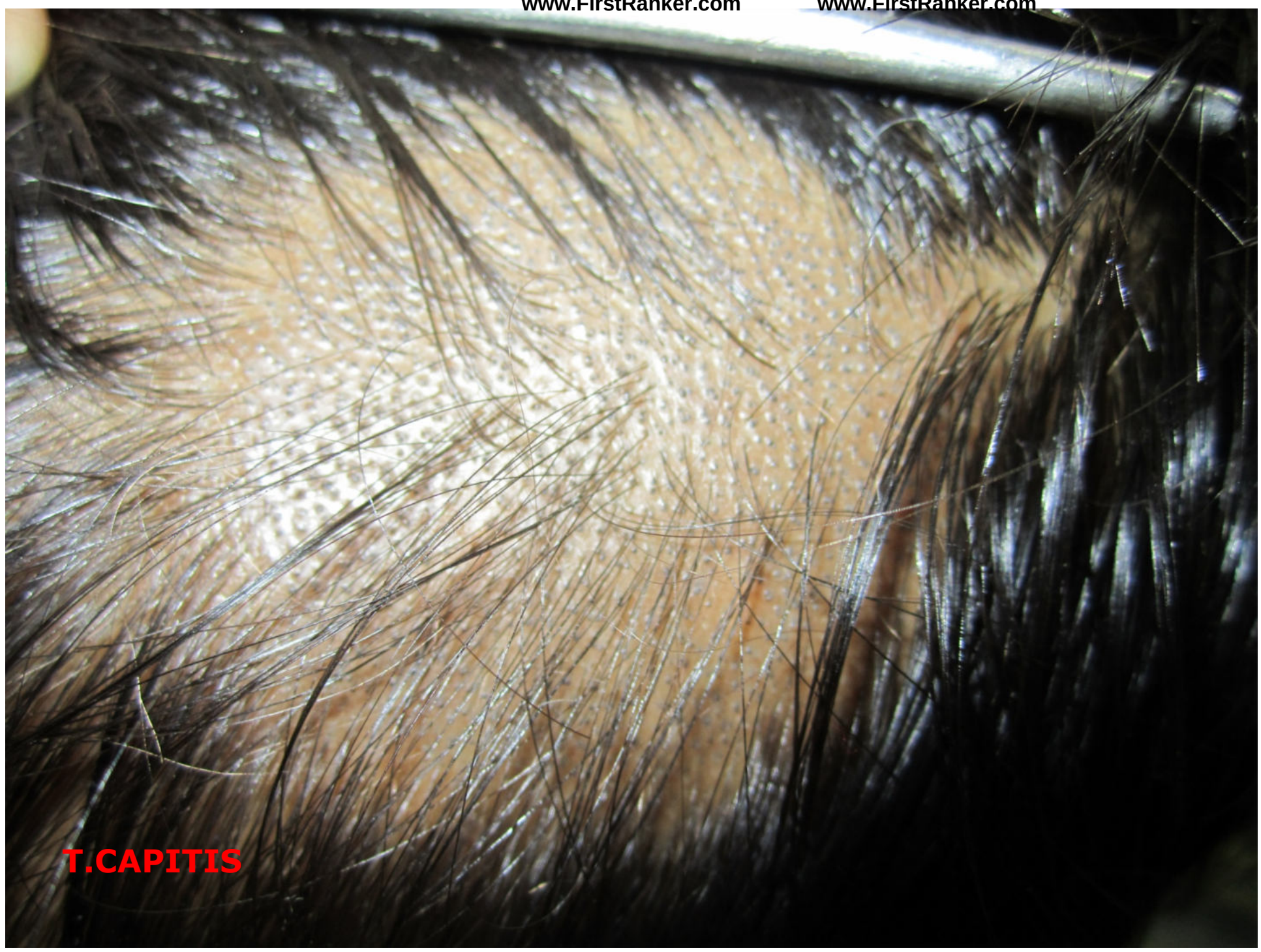
- ??

- ANNULAR PLAQUES
- relative clearing in the center.
- edge of the lesion showing papulovesiculation and scaling.

- TINEA



T. CAPITIS



- Noninflammatory tinea capitis:
- Caused by anthropophilic organisms (e.g., T. verrucosum), so less inflammation.

- Inflammatory tinea capitis (kerion)
- Caused by zoophilic dermatophytes (e.g., M. canis), which elicit intense inflammation.
- boggy swelling with pustulation. Often, the pus discharges from multiple orifices. Hair from such a swelling is easily and painlessly pluckable

Table 14.18. Diagnostic features of tinea infection

Tinea corporis/cruris	❖ Annular or arcuate lesion ❖ Periphery shows papulovesiculation and scaling and centre is relatively clear
Tinea capitis	❖ Noninflammatory or inflammatory patch of alopecia ❖ Easy painless pluckability of hair
Tinea unguium	❖ Asymmetrical involvement of few nails, which begins distally. ❖ Yellowish discoloration and thickening of nail plate. ❖ Nail plate shows crumbling. ❖ Friable debris under nail plate.

- WHICH BEDSIDE TEST WOULD YOU DO??

- Potassium hydroxide (KOH) scraping AND MOUNTING

Table 14.20. Specimen to be take in tinea infection

T. capitis	Plucked hair, black dots
T. cruris	Scales from edge
T. corporis	Scales from edge
T. unguium	Clippings of discolored nail plate; subungual debris

- Technique
- Mount specimen on glass slide, adding 10% KOH (to dissolve the keratin).
- Keep for half an hour;
- nail clippings require longer (2 h) and warming (not boiling).
- Fungus is easily detected using the low power objective lens (10 ×) with the iris diaphragm closed and the condenser positioned down.

Table 14.21. Interpretation of KOH mount

Tinea capitis	Branching hyphae Spores within (endothrix) (Fig. 14.70A) or around the hair (ectothrix)
Tinea cruris	Branching hyphae (Fig. 14.70B)
Tinea corporis	Branching hyphae (Fig. 14.70B)

- Scenario: tinea capitis outbreak in a school
- What investigation should you do?
- Wood's light examination- green fluorescence

- General measures
- Keeping area dry.
- Avoiding use of synthetic clothes.
- In recurrent infection, prophylactic use of anti-fungal talc

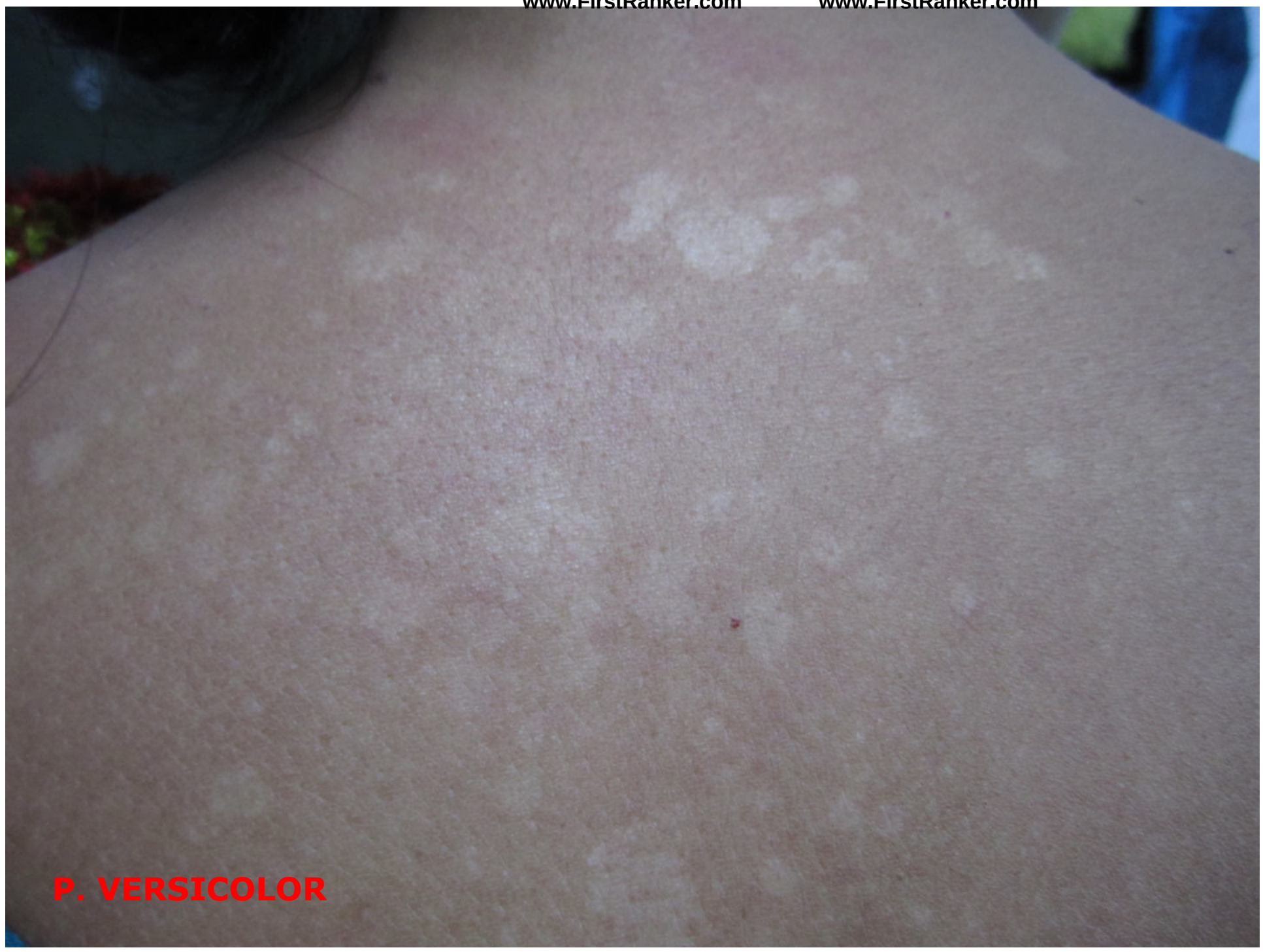
Table 14.22. Topical antifungals

Compound	Preparation	Comments
Azoles		❖ Broad spectrum (effective against dermatophytes, candida and Malassezia, also erythrasma) ❖ Fungistatic, so slow response (4–6 weeks) ❖ Low irritation/sensitization potential
Miconazole, 2%	Cream	
Clotrimazole, 1%	Cream Lotion Powder	
Ketoconazole, 2%	Shampoo	
Econazole, 1%	Cream	
Sulconazole 1%	Cream	
Allylamines		❖ Fungicidal, so rapid response (1–2 weeks) ❖ Narrow spectrum (effective against dermatophytes, Malassezia[38])
Terbinafine, 1%	Cream	
Butenafine, 1%	Cream	
Others		
Ciclopirox olamine, 1%	Cream Nail lacquer[39]	Broad spectrum
Amorolfine, 5%	Nail lacquer[40]	

- Systemic therapy is recommended in the following situations:
- Extensive dermatophytic infections.
- Tinea unguium.
- Tinea capitis

Table 14.23. Treatment protocol for tinea infection

Tinea corporis	
Localized	Topical therapy (4 weeks)
Extensive	Oral terbinafine (2 weeks) Oral griseofulvin (4–6 weeks)
Tinea cruris	
Short duration	Topical therapy (4 weeks)
Chronic	Oral terbinafine (4–6 weeks) Oral griseofulvin (6–8 weeks)
Tinea capitis	Oral griseofulvin (8 weeks) Oral terbinafine (4–8 weeks)
Tinea unguium	Oral terbinafine (6 weeks for finger nails, 12 for toe nails) Oral itraconazole (2 pulses[42] for finger nails, 3 for toe nails) Oral griseofulvin (24 weeks for finger nails, 36 for toe nails)

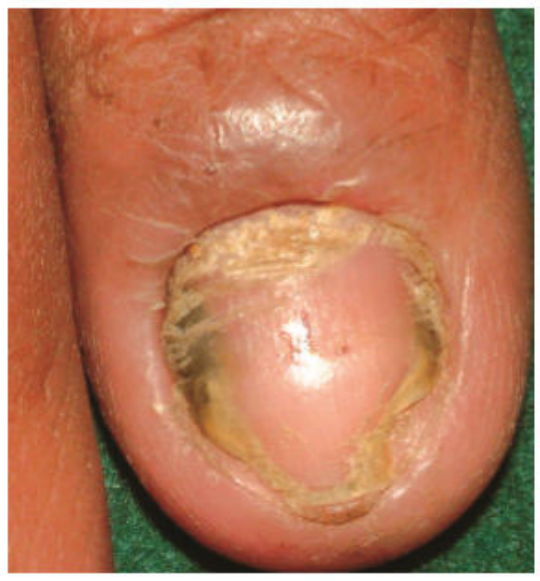


- Perifollicular, hypopigmented (or hyperpigmented), macules surmounted with branny scales.
- Upper trunk, neck, upper arms.
- KOH mount
- shows characteristic ‘spaghetti and meat ball’ appearance.

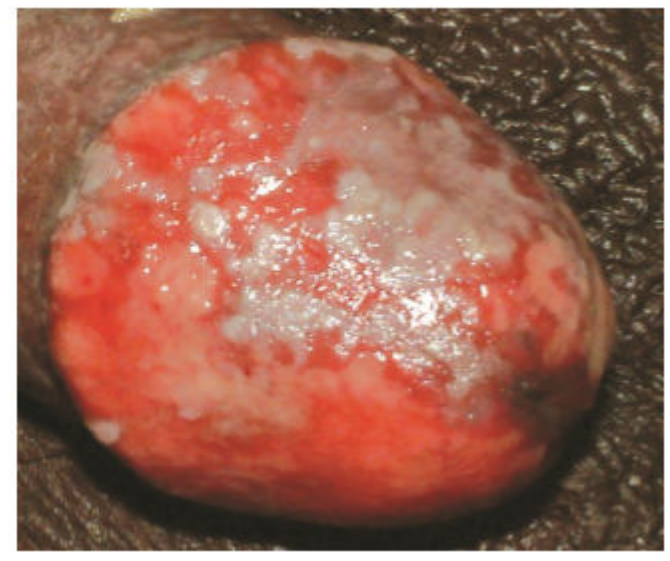
- Topical agents
- Imidazoles:Ketoconazole, 2% applied daily for 4 weeks.
- Selenium sulfide: 2.5% lotion in a detergent base, used weekly for 4 weeks.

- Systemic agents
- Needed in extensive lesions or when recurrences are frequent:
- Ketoconazole, 200 mg daily for three consecutive days.
- Fluconazole, 400 mg single dose.
- Itraconazole, 200 mg daily for 7 days.

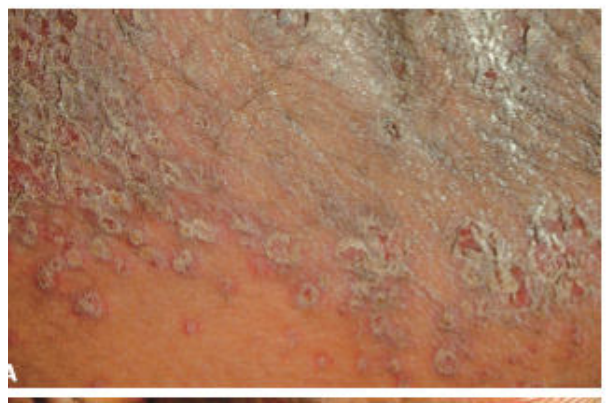
CANDIDAL INTERTRIGO



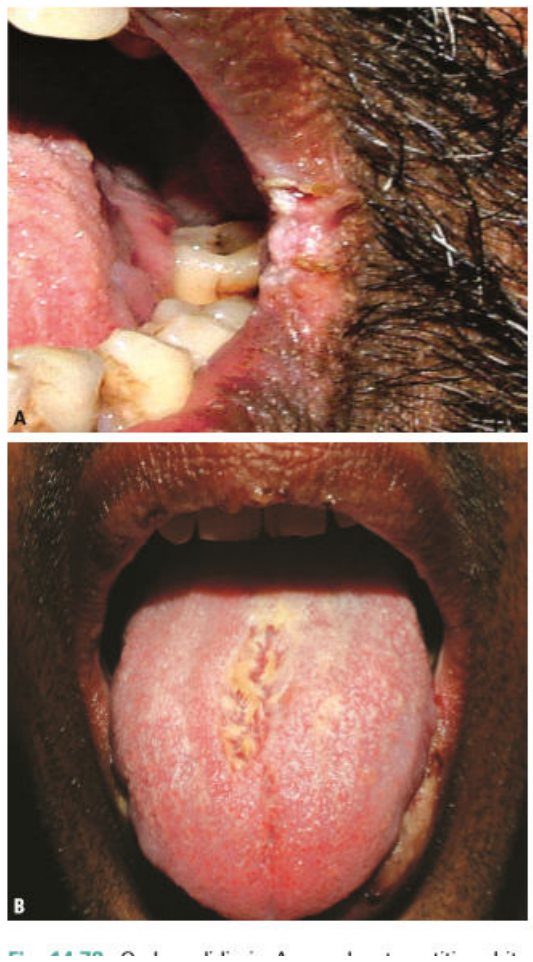
Candidal paronychia



Candidal balanoposthitis



- frayed lesion in the groin with satellite pustules.



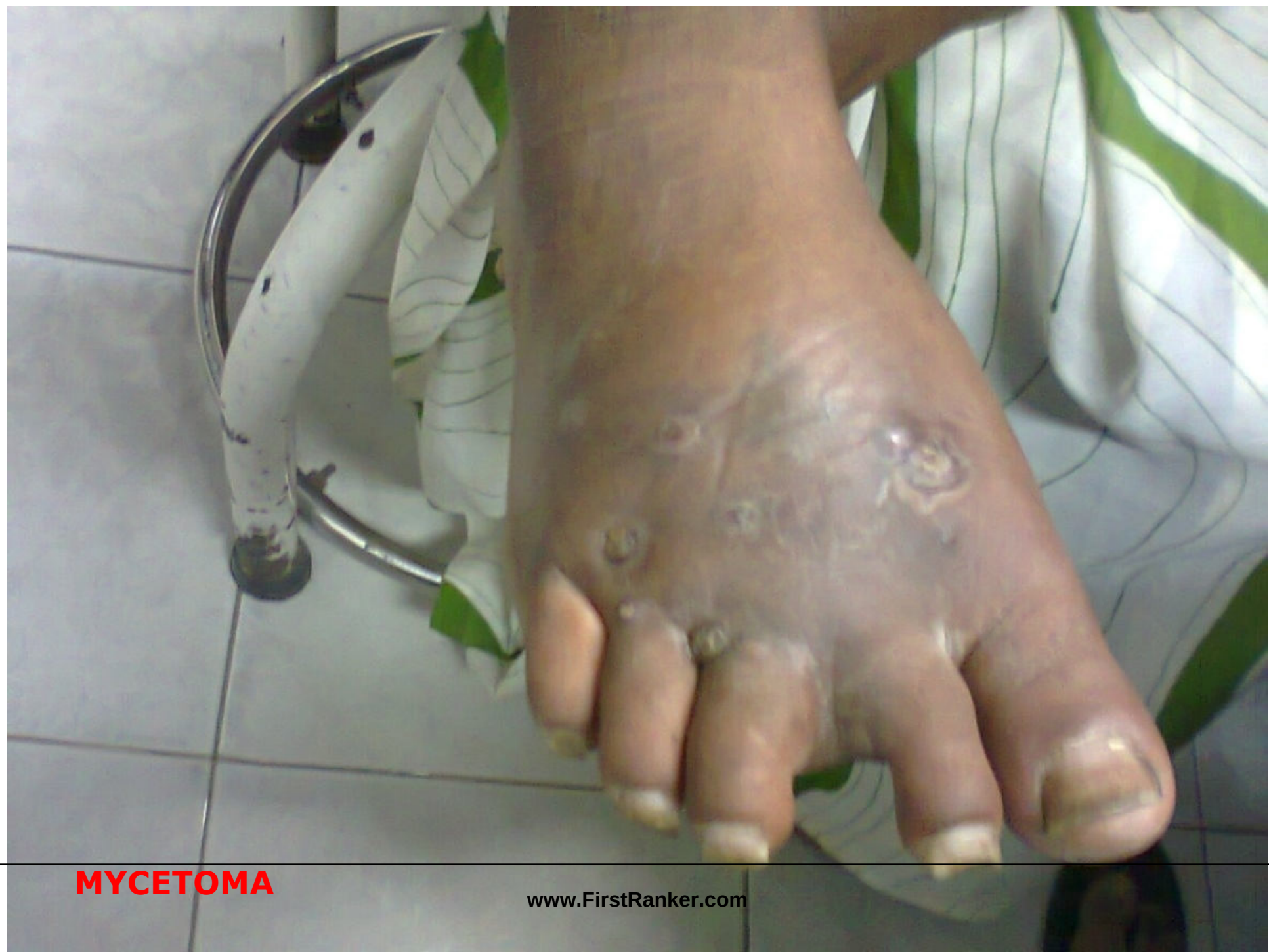
- Oral candidiasis:
- KOH mount shows budding yeasts and pseudo- hyphae.
- Culture

- Rule out diabetes in patients with recurrent infection.
- Rule out immunocompromised states in recurrent/extensive/atypical disease.

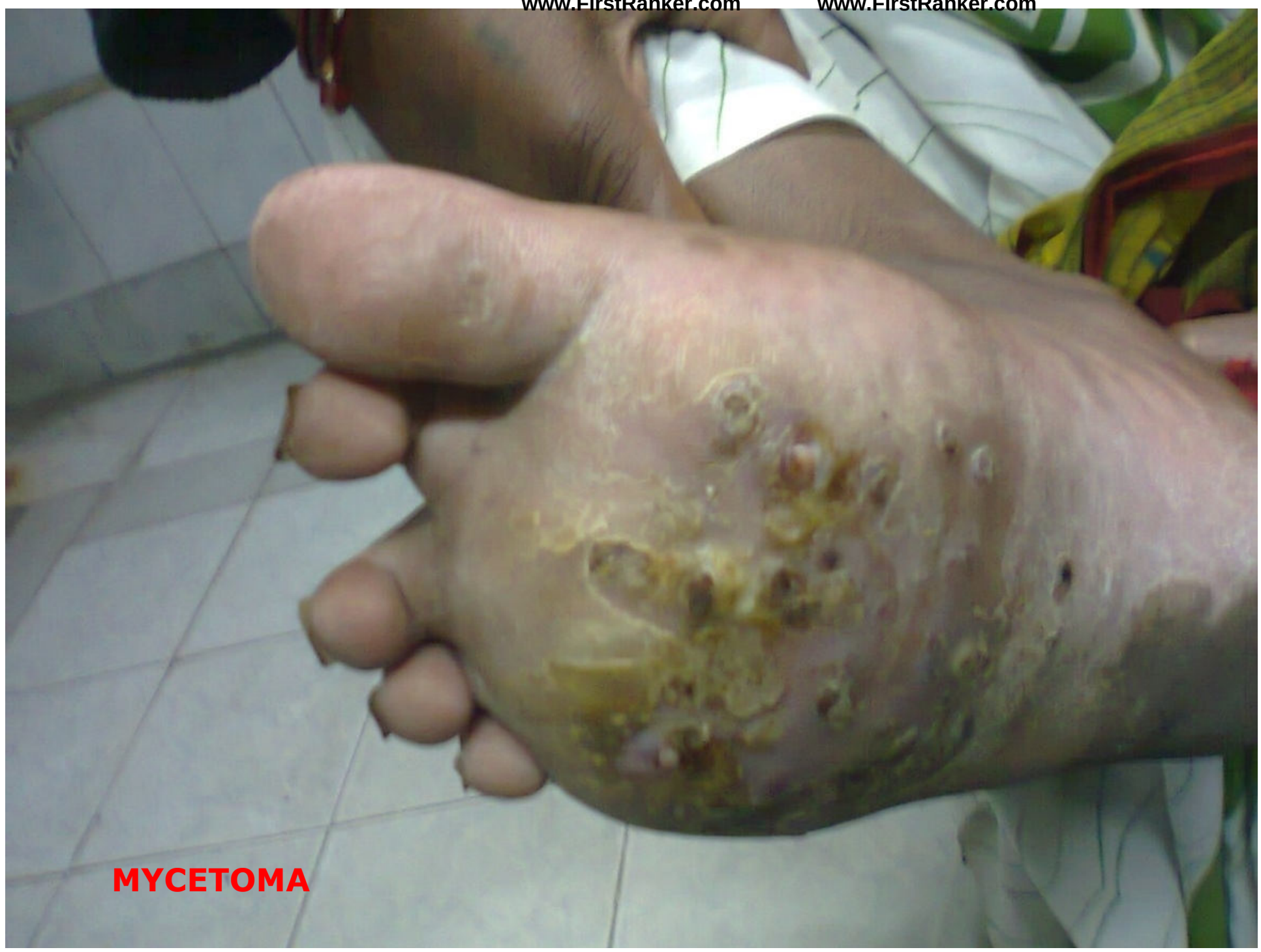
- General measures
- Predisposing factors should be sought and eliminated.
- Intertriginous areas should be kept dry by adequate wiping after a bath.
- In paronychia, prolonged immersion in water is best avoided.

- Topical agents
- Candidal intertrigo:Topical azoles (clotrimazole, miconazole, and ketoconazole) are effective.
- Candidal paronychia: Topical azole lotions
- Oral candidiasis: Lotions and oral suspensions of azoles. Or nystatin.
- Genital candidiasis : Imidazole pessaries for vaginal infection. Topical azoles for balanoposthitis.

- Systemic therapy recommended in the following situations:
- Candidal vulvovaginitis: Single dose fluconazole (150 mg) or itraconazole (400 mg).
- Weekly doses of fluconazole (150 mg) for recurrent problem.
- Recurrent oral candidiasis: In immunocompromised patients (e.g., HIV infection), fluconazole, 150 mg weekly dose.
- Chronic mucocutaneous candidiasis: Requires prolonged therapy



MYCETOMA

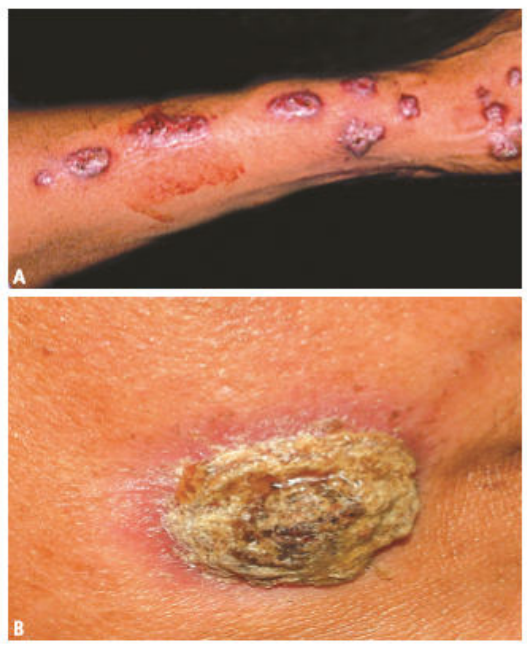


- Begin as subcutaneous nodules,
- which slowly evolve into abscesses and draining sinuses
- Over period of time, the surrounding tissue becomes hard due to fibrosis.

- Investigations
- Establish the diagnosis of mycetoma.
- Identify the causative organism (actinomycetes vs eumycetes).
- Find the extent of local spread

- Examination of pus and granules
- Histology
- Culture
- X-ray of the affected part

- Treatment depends on whether the mycetoma is actinomycotic or eumycotic.
- Actinomycetoma Responds to a 6–9 months course of combination of chemotherapeutic agents like: Streptomycin + dapsone or co-trimoxazole.
- Co-trimoxazole + amikacin.
- Tetracyclines + streptomycin + rifampicin.
- Penicillins + gentamycin + co-trimoxazole



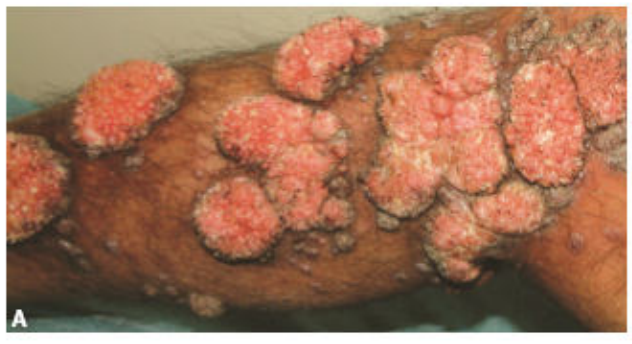
- Eumycetoma
- Ketoconazole
- Itraconazole.
- Amphotericin B in resistant cases

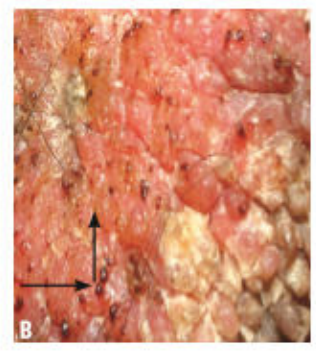
- Surgical intervention:
- Deep debridement and even amputation may need to be done in case of recalcitrant lesions

- ulcerated nodules are arranged in a linear fashion along the lymphatic drainage.

- single infiltrated plaque

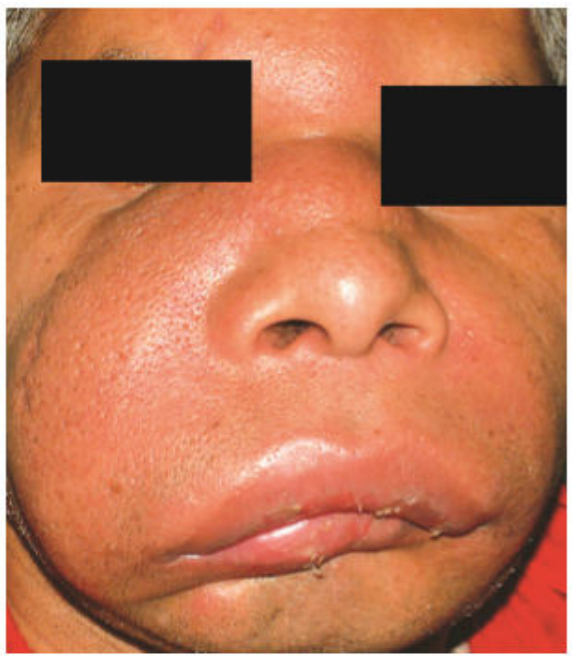
- Sporotrichosis
- Causative agent-
- Sporothrix schenckii.
- Treatment:
- Saturated solution of potassium iodide. Or itraconazole.





- cauliflower-like hypertrophic plaque
- Characteristically, surface is studded with black dots

- Chromoblastomycosis
- Several fungi can cause chromoblastomycosis
- Trauma prone sites.
- Treatment:
- Itraconazole, flucytosin



- painless subcutaneous swelling with smooth edge which can be raised by inserting a finger under it
- Subcutaneous phycomycosis
- Potassium iodide

- GOOD DAY !!!!