**DERMATOLOGY**

* This is the science of skin condition.
* Skin is the largest organ in the body, 1.5 - 1.75 M2.



* The skin is composed of 3 layers
* Epidermis
* Dermis
* Subcutaneous tissue/hypodermis
* Epidermis has four layers from the innermost to outermost they are stratum germinativum, stratum granulosum, stratum lucidum and stratum corneum.
* Dermis has two layers papillary and reticular it is also made up of blood and lymph vessels, nerves, sweat and sebaceous glands.
* Subcutaneous tissue/hypodermis its primarily adipose tissue
* Skin functions
* Protection
* Fluid balance
* Temperature regulation
* Vitamin production
* Immune response function
* Skin is used for per cutaneous absorption
* Sensation-It is tactile for touch ,pain,cold ,itch and pressure
* Excretion –sebum and sweat

 **CLASSIFICATION OF SKIN CONDITIONS**

1. Pruritus

* General pruritus
* Perineal and Perianal pruritus

2. Secretory disorders

* Seborrheic dermatoses
* Acne vulgaris
* Hydradenitis suppurativa

3. Infectious dermatoses

1. -Bacterial skin infections (pyodermas)
* Impetigo
* Folliculitis, Furuncles, Carbuncles

 ii. -Fungal (mycotic) skin infections

* Tinea Pedis -Athlete’s foot
* Tinea corporis – Ringworm of the Body
* Tinea capitis- Ringworm of the scalp
* Tinea unguium-Ringworm of the nails
* Tinea cruris – Ringworm of the groin

 iii. -Viral skin infections

* Herpes zoster
* Herpes simplex
* Genital herpes
* Orlabial herpes

 iv. -Parasitic skin infections;- Pediculosis (Lice infestation)

* Pediculosis capitis- Infestation of the scalp by head louse
* Pediculosis corporis – Infestation of the body by the body louse
* Pediculosis pubis- Infestation of the genital region by louse
* Scabies- Infestation of the skin by the itch mite *sarcoptes scabiei.*

4. Eczema/Dermatitis

* Contact dermatitis
* Atopic dermatitis

5. Non-infectious imflamatory dermatoses

* Psoriasis
* Exfoliative dermatitis
* Pemphigus vulgans

6. Ulcerations of skin

* Pressure ulcer
* Streptococcal ulcer
* Buruli ulcer
* Leg ulcer

7. Blistering diseases/Etythema multifore

* Toxic epidermal necrolysis (TEN) and stevens-johnson syndrome (SJS)
* Pemphigus
* Bullous pemphigoid
* Dermatitis herpetiformis

8. Tumors of the skin

 i. Benign skin tumor

* Cysts
* Seborrheic and keratoses
* Verrucae- wart
* Angiomas
* Pigmented nevi- moles
* Keloids
* Dermatofibroma
* Neurofibromatosis-Von Recklinghausen’s Disease

 ii. Malignant skin tumors

* Basal cell and squamous cell carcinoma
* Malignant melanoma

 iii. Metastastatic skin tumors

* Kaposi’s sarcoma

 9. Other skin conditions

* Necrotizing fasciitis
* Cellulites
* Pellagra
* Erysipelas
* *Ptyriasis versicolor*

 10. Hair disorders

* Alopecia-loss of hair from any cause
* Hirsutism- excessive hair growth
* Buru’s

 **ASSESSMENT OF SKIN CONDITIONS**

1) HISTORY

* Age, gender, occupation, mental status, general appearance.
* Chief complaint –location, duration, type of lesion, dry or wet
* dry particular injuries medication, nutrition, working, environmental, exposure,

2) EXAMS

-Examine the whole body paying attention to the armpit, under breast and private parts.

-Determine the distribution and pattern of lesion e.g herpes –in one side

 Allergic dermatitis –at the site of contact

-Determines the skin colour –, yellow, red, cyanosis

E.g vitiligo is whitish (deficiency of melanin)

-Determine if dry or weeping.

-Determine the configuration e.g. linear, circular bizarre in nature or cluster eg in herpes

-Touch and palpate and determine the degree of infiltration e.g. thin, thick, skin elasticity, oestema, emphysema (some air under the skin)

-Determine whether they are 10 or 20 lesions

* Note the nails

-Convex– >Normal

-Concave –>can be fungal infection.

- should be translucent, capillary refill should be 3sec.

-Spoon shaped nails indicate iron deficiency anemia

-During exam, note **hair** colour**,** texture, examine for parasites. Very fine hair indicates hypothyroidism.

3) LABORATORY INVESTIGATIONS

* Skin biopsy for c/s
* If vesicle –aspirate the contents for c/s
* Immunofluorescence
* Patch testing
* Tzanck smearskin scrapings
* Wood’s light examination
* Clinical photographs

4) ROUTINE EXAMINATION

* Urine for urinalysis
* CBC

 **SKIN LESIONS**

 **1.**PRIMARY LESIONS.

-This are original lesions arising from previously normal skin.

* MACULE,PATCH

-A flat non-palpable discolouration of skin (color may be brown, white, tan, purple, red)

* Macule < 1cm, circumscribed border
* Patch > 1cm, may have irregular border

 *Examples;*

Freckles, flat moles, petechia, rubella, vitiligo, wine stains, ecchymosis

* PAPULE,PLAQUE

-A solid palpable elevation of the skin .it is between 1 -5 mm in diameter .

-found in acne.

* Papule < 0.5cm
* Plagues >0.5cm

*Exampels;*

Papules; Elevated nevi, warts, lichen planus

Plagues; Psoriasis, actinic keratosis

* NODULE,TUMOR

-It is deep seated into the dermis and is larger than 1cm in diameter, solid palpable elevation of the skin

E.g. in rheumatoid arthritis.

* Nodule; 0.5-2cm; circumscribed
* Tumor >1-2cm; tumors do not always have sharp borders

*Exampels;*

Nodule; limpoma, squamous cell carcinoma, poorly absorbed injection, dermatofibroma

Tumors; large limpoma, carcinoma

* VESICLE,BULLA

 =Vesicle

-This is elevation of skin filled with serous fluid which is clear ,it is less than 1cm in diameter. (<0.5cm)

*Exampels;*

Chickenpox, poison ivy, second degree burn (blister) and in early herpex simples/zoster

=Bulla

-A big vesicle more than 1cm . (>0.5cm)

*Exampels*

Pemphigus, contact dermatitis, large burn blisters, poison ivy, bullous impetigo, burus and in late herpes simplex/zoster.

* PUSTULE

-Elevation of the skin i.e vesicle or bulla filled with pus

*Examples;*

Acne, impetigo, furuncles, carbuncles

* WHEAL

-Transient irregular elevation of skin caused by movement of serous fluid into the dermis.

-Does not contain free fluid in a cavity (as for example, vesicle does)

-Is to increased permeability of capillary walls eg insect bites.

 *Examples;*

 Uticaria (hives), insect bite

* CYST

- Is encapsulated fluid filled/semisolid mass in the subcutaneous tissue/dermis.

*Examples;*

Sebaceous cyst, epidermoid cysts

 **2.**SECONDARY SKIN LESIONS

This lesion originate from changes in primary lesions

* SCALES

-Hipped up dead layers of the epithelium/stratum corneum

*Examples;*

Dandruff, psoriasis, dry skin, pityriasis, rosea.

* CRUST

-Dry accumulation/residue of exudates and secretion of pus ,blood ,serum on the skin surface.

-Large adherent crust is a scab

*Examples;*

 Residue left after vesicle rupture: impetigo, herpes, and eczema

* FISSURE

-A small linear or crack in the skin may expose the dermis.

*Examples;*

Chapped lips or hands, athlete’s foot.

* EXCORIATION

-Linear scratch marks confined to epidermis .they are traumatized areas of skin.

* ULCER

-Erosion of the skin due to local destruction extending past the epidermis

-bleeding and scarring possible.

*Examples;*

Stasis ulcer of the venous insufficiency, pressure ulcer

* BURROW

-Linear elevation of skin eg in scabies

* ERYTHEMA

-Redness of the skin caused by the congestion of capillaries

 **TREATMENT –PRINCIPLES OF DERMATOLOGICAL THERAPY.**

1) Apply wet dressing –using right solution e.g. potassium permanganate

2) Topical medications

-Some act by cooling the skin, reducing the itching

-Hydrating the skin –dryness is reduced improves the medication absorption they can also reduce hydration (dehydrates, removes crust, have antiviral in it, antifungal etc)

-Lotions –act by lubricating and cooling.

-Creams –act by drying

 Others

* ointment
* powders
* gels –semisolid
* Paste –mixture of powders and lotions sprays.

3) Systemic medication –antibiotics, corticosteroid, antihistamine, antifungal, sedatives etc.

 **1 SECRETORY DISORDERS**

 **a) SEBORRHEIC DERMATOSES**.

* It is a skin disease affecting areas of the skin where there is a high density of sebaceous gland.

Fungi phytosporum.

* areas commonly affected include the scalp ,eye brow, sides of the nose , ears esp. ear canal ,the axilla, groin, beneath the breast ,the chest and upper back
* it is found in 50% of pts having aids.

 CAUSES /PREDISPOSING FACTORS

* Genetics
* Hormonal changes esp. at adolescent and hormonal therapy
* Emotional stress
* Sex –more in males
* Fat taken in excess.

 CLINICAL FEATURES

* Diffuse erythema
* The scalp is covered with scales which look like dandruff.
* The scales eventually spread to most parts of the body.
* Pustules may accompany the scales or may be present as the only manifestation of the disease
* Local spread from the scalp to the eye brows , side of the nose , ears and the neck are very common
* Blemphatitis and stitis externa are obual accompaniment of the disease.
* Folliculitis may be a predominant feature of the disease.
* In most severe cases ,greasy scales ,wheeping and masceration of the affected area is common

 MANEGEMENT.

* Use of medicated shampoo (betadine shampoo) or substances with antifungatatic or acidic activity e.g. salicylic acid ketoconazole etc – shampoo to stay for 5-10 minutes rotated.
* Use of topical steroids and topical antifungal preparations reduces clinical features (inflammation)
* If the bacterial infections present, give antibiotics.
* For hiv/aids – use systemic antifungals e.g. ketoconazole or greosufulvin.

 NURSING MANAGEMENT

* Advice pt to avoid external irritant, excessive heat and perspiration, scratching prolong the disorder.
* Caution pt. that the disease is a chronic that the problem tends to recur.
* Give instructions on use of shampoo .To use at least 2 or 3 shampoo soaps rotated after each to prevent resistance .shampoo to stay for 5 -10 minutes.

 **b) ACNE VULGARIS**

* This is a chronic inflammation of the sebaceous unit/glands and the associated hair follicles affecting mainly the face, the neck, the back and chest of the adolescents and the young adults.
* It is characterized by plugging of the pillosebacceous follicles by keratin and sebum resulting in an expanded follicle called comedones-primary acne lesions

 PREDISPOSING FACTORS

* Family history
* Hormonal changes during puberty
* Diet –chocolate, cola, fried foods
* Anxiety/stress disorders
* Some chemicals e.g. soruazid
* Exercise vigorous leading to sweat

 AETIOLOGY

* No known cause.
* Androgenic hormones
* Genetic predisposition
* Bacterial factors
* Follicular obstruction/plugging.
* All genders are equally affected and esp. adolescents and ladies who use androgen.

**NB** Acne is aggravated by picking and squeezing the lesion, stress, nervous tension, insufficient sleep, chronic illness, menstruation and some diets (fats)

 PATHOPHYSIOLOGY

* At puberty, the presence of androgens stimulates sebaceous gland causing them to enlarge and produce sebum.
* In childhood , sebaceous glands are small and non-functional due to low levels of androgens
* Acne develops when there is an alteration in keratinisation within the follicles, leading to plugging of the pillosebacious a canal by a mass of keratin and sebum.
* This leads to accumulation of sebum in the follicles and the normal saprophytes propionibacterium acne breaks this sebum into free fatty acids which imitates and eventually break down follicular walls.
* The free fatty acids then invade the surrounding tissues causing inflammatory reaction.

 CLINICAL FEATURES.

* In mild forms, only non-inflammatory lesions are present known as open comedones (white heads) closed comedones (black heads) Their dark colour is due to oxidation of surface keratin. (accumulation of lipids, bacterial, and epithelia debris)
* Papules and pustules are 2-4mm in diameter and have slightly erythematous base.
* Nodules are deeper erythematous lesions from 6-20mm in diameter.
* There may appear and a few superficial pustules
* In most severe forms there are deep nodules , deep pustules and also abscess.

 DIAGNOSIS

* Basically clinical through physical examination and history taking.
* Biopsy of lesion is seldom necessary for a definitive diagnosis

 MANAGEMENT

* Goal is to reduce bacterial colonies to decrease sebaceous gland activity, prevent the follicle from becoming plugged, reduce inflammation, combat secondary infection, minimize scarring and eliminate factors that predispose the person.
* Therapeutic regime depends on the types of lesion/comedonal/popular and may be topical, systemic, intralesional or surgical and includes the following;
* Hormone therapies i.e. give estrogens to suppress effects of androgens (for female).
* Nutritional therapy elimination of specific food products associated with acne e.g chocolate, cola, fried foods, milk products.
* Skin hygiene in mild cases washing the faces at least three times daily with a cleansing soap, but vigorous scrubbing should be avoided.
* Topical pharmacological therapy –this is designed to obtain a mild exfoliation (peeling). Common medication are sulphur-zinc losion, benzoyl peroxide wash gel – depresses sebum production and promote breakdown of comedo plugs , Vitamin A acid (retirioic acid) –has comedolytic effects and also increases the rate of cell production within the hair follicle hence pushing out of the plug itself. (Increases rate of desquamation of epidermis as well as reduce sebum production).
* Systemic pharmacological therapy esp. in severe case of abscess formation oral antibiotics (erythromycin, tetracycline, doxycycline, minocin, penicillins trimethoprim, sulfamethazone).
* Intralesional corticosteroid therapy- cysts should be drained and injected with triamcinolone solution.
* Topical application –use of comedolytics e.g. vitamin A benzoacid (benzoy peroxide) which has antibacterial effects .
* Topical antibiotics e.g. Tetracycline, clindamycin and erythromycin ointment –suppresses multiplication of propillumbacteria
* Oral retinoids e.g. synthetic vitamin A compounds in patients with nodular cystic acne not responding to other treatment.

**NB** these medications are potentially irritating and frequency should be gradually increased

* Surgical management
* Open comedones may be removed with a comedo extractor, incision and drainage of cystic regions.
* Dermabresion- damage epidermis and superficial dermis scars are removed.
* Cryosurgery- freezing with liquid nitrogen

 HEALTH EDUCATION

* The patient should be assured that the disease is not related to uncleanness and in description of any other misconceptions.
* Advice the pt to abide to the treatment regimen as it’s normally long term (4-blocks for any results to be realized).
* Advice the patient to avoid scrubbing the or squeezing pimples (comedones)
* To stop using cosmetics on face –lotions, creams, shaving etc. instead use oil free cosmetics
* Activities causing occlusion such as tight collar shirts should be avoided
* Advice on the need to avoid stress.
* Reduce the junk foods –chips, chocolate, fried food, milk product.

 **2 INFECTIOUS DERMATOSES**

 **i) BACTERIAL SKIN INFECTIONS**

 **a) IMPETIGO**

* It is a superficial skin infection caused by *streptococcus pygenes type- a/staphylococcus aureus , beta haemolytic streptococcus* or both*.*
* Mainly affects children and young adults
* It is associated with poor hygiene and low social economic status.
* More frequent in hot and humid weather.
* Areas mostly affected are the exposed parts of the body mainly the face, nose, arms, the neck, and extremities.
* Epidemics often occur in schools and families through fomites or flies.

 PREDISPOSING FACTORS

* Poor hygiene and dirty in the society
* Malnutrition
* Anemia
* Working place (health providers working in nursery)
* Mode of transmission.
* Direct contact with a lesion
* Indirect contact through contaminated impetigo materials.
* It may appear as a 1 disease or may appear 2 to pediculosis, herpes simplex, scabies, and dermatitis as insect bites.
* May be classified into

 1) Bullae impetigo-large vesicles

 2) Impetigo contangiosum

* Depends on the size of the vesicle e.g. nasal impetigo

 CLINICAL FEATURES

* Usually asymptomatic or mildly symptomatic
* The lesion begins as one or more small red vesicles and over 2-3 days rupture releasing scrum.
* Scrums coagulate or dry up forming up yellow crusts.
* The crusts are easy to remove and moment they are removed they reveal smooth red moist surfaces on which few crust soon develop.
* There is slight itching on the affected areas
* There may be lymph adenopalty and the patient may have fever and generalized malaise.

 DIAGNOSIS

* Clinical feature and history.
* Gram staining.
* Culture and sensitivity to isolate the causative organism.

 MANAGEMENT

* Gentle removal of crusts and treatment of area with antiseptic preparation e.g. providing iodine.
* If localized use topical antibiotics eg neomycin tid, bactroban,
* Systemic abx e.g. cloxacilin 250mg po qid, cefadroxil 500mg po bd, benzathine penicillin 1-2 mega unit, didoxicillin or mecillin/erythromycin for penicillin sensitive patients.

 NURSING MANAGEMENT

* Wear gloves when giving care
* Advice the patient and family members to bath at least once daily with antbacterial soap.
* Towels and soaps should not be shared.
* Those infected in schools should be isolated.
* Keep infected children away from others

 PROGNOSIS

* Complete recovery is the norm and requires upto 2 weeks of treatment.

COMPLICATION

* Glomerulonephritis- after infection with certain strains of streptococcus.

 **b) FOLLICULITIS, FURUNCLES AND CARBUNCLES**

* Folliculitis –is an infection of bacterial or fungi origin that arises within the hair follicles.
* Furuncles(boil) –is an acute inflammation arising deep in one or more hair follicles and spreading into the surrounding dermis
* It is deeper form of folliculitis.
* Carbuncle –is an abscess of the skin and subcutaneous tissue that represent an extension of the furuncle that has invaded several follicles and is large and deep seated.
* Appears where skin is thick and inelastic e.g. back of neck and buttocks
* It is usually caused by staphylococcal infection.

 PATHOPHYSIOLOGY

* The bacterium is mostly stappl. Aureus, gains entry into the hair follicles, multiplies rapidly releasing toxins
* The released toxins damage the cells of the dermis
* Both toxins and contents released during tissue damage triggers a severe inflammatory response.
* Eventually when toxin concentration is sufficiently great, the centre of the inflamed area dies off.
* In the course of inflammation there is massive influx of the neutrophils into the inflamed area and most die in the process of neutralizing toxins and destroying bacteria
* When neutrophils dies , they release enzymes which lead to rapid liquefaction of the dead tissues and a cavity filled with pus in the centre of an area of acutely inflamed tissues.

 CLINICAL FEATURES

* A future begins as a circumscribed tender swelling around a central hair
* Shortly afterwards, it becomes a hard painful bright red and shiny cone shaped nodule 1 -2cm in diameter.
* 48hrs afterwards, suppuration occurs and a yellow point appears on the apex.
* When it breaks ,it discharges a greenish yellow pus and a plug of necrotic material
* Following the discharge of pus , a purplish irregular crater forms which heals with a scar

**NB**-Boils are tender, hot and painful

 MANAGEMENT

* Early lesions should be protected from trauma
* Do not squeeze/brake the lesion when not ripe to avoid septicaemia and recurrence.
* Once the lesion begins to ripen, it should be broken with a sterile needle and the area gently swabbed with an antiseptic solution.
* If the boils are multiple and large or if they are accompanied by constitutional symptoms they should be treated with systemic antibiotic therapy cloxacillin 500mg po bd 1/52 depending on age, dicloxacillin, flucoxacillin, cefadroxil 250mg qid or erythromycin 500mg tid 1/52
* Cephalosporin’s are also effective
* Analgesics are indicated
* Recurrent boils may necessitate long term use of antiseptic e.g. washing with providon iodine for several months
* Bed rest is advised for patients with boils around perineum and anal region
* Resistant boils may need culture and sensitivity and subsequent prescription of the appropriate antibiotics

 NURSING MANAGEMENT

* Warm moist compressions are applied to increase vascularisation and hasten resolution of furuncle or carbuncle.
* IVFS, fever reduction and other supportive tx are indicated for pts who are very ill or suffering from toxicity
* Handle soiled dressing according to standard precautions
* Use gloves not to infect yourself or become a carrier
* Clean surrounding area with antibacterial soap and antibacterial ointment may be applied

 HEALTH EDUCATION

* Hygiene
* Soiled linen should be disinfected
* Complete length of abx adherence

 COMPLICATION

* Septicaemia
* Osteomylitis

 **ii) FUNGAL (MYCOTIC) SKIN INFECTIONS**

* **RINGWORM (TINEAS)**
* Caused by dermatophytes.
* Form ring and only live in dead tissues (stratum corneum)
* Hardly invade living tissues
* There are three main genre of dermatophyte
* Microsporum –causes infection of the skin and hair.
* Epidermic phytes -affects skin and nails.
* Trichophytum –affect the three areas

 TRANSIMISSION SOURCES

 i) Zoophilia –form animals to man

 ii) Arthrophilia –man to another man

 iii) Geophilia –from the soil

 TYPES OF TINEAS

 **a)** **TINEA CAPITIES**

* Ringworm of scalp
* Caused by microsporure or trichophytory spp.
* Common in tropics especially in children
* It is very contagious
* In children, it heals spontaneously by puberty.

 PRESENTATION

* Start as a small duoid scaly patch which spreads to the periphery.
* There is scaling of skin followed by alopecia

 TREATMENT

* Shave the hair and wash the scalp thoroughly.
* Use medicated shampoo 2-3 times daily
* Topical antifungal are prescribed in muconazole, fluconazole, ketoconazole
* Oral antifungal –e.g. ketoconazole, greseofulvin etc.
* Treat the whole family including the pets
* Disinfect the combs.

 **b) TINEA PEDIS (Athlete’s foot)**

* It’s a superficial fungal infection characterized with acute inflammatory process or chronic lesion involving the soles of foot and inter digital spaces
* More common in tropics
* Most common fungal infection.
* More common in dirty swimming pools
* Clinically there are 3 types

 i) INTERDIGIT/INTERTRIGO TYPE

* Especially between 4th-5th toe .there is slow development of maceration (wetness).
* Fissures can also develop.
* There is whitening of inter digital spaces. The whitening rubs off and a red area is left.

 ii) VESICULAR TYPE

* Sudden appearance of group of deeply seated pruritis vesicle, pustules or bulla
* Normally found at side of toes, in step and clorsum of foot.
* They rupture dry up and leave ugly scales

 iii) HYPERKERATOTIC TYPE

* There is excessive thickening of the sole.
* The thickening finely peels apart and the skin creases.
* The creasing is worsened by development of scales white in colour.
* In between the creases, fissuring can occur and infections can take place.

 TREATMENT

* Avoid predisposing places.
* Potassium permanganate solution for feet soaking also silver nitrate solution can be used.
* Topical antifungal.

 c) **TINEA CORPORIS**

* Its superficial infection or the body excluding the hands and feet.
* Common in rural areas.
* It start in erythromatous macules which advance to ring or vesicle with a central healing
* Appear in clusters

 TREATMENT

* Topical antifungal e.g. clotrimazole
* Systemic antifungal.

 **OTHER TINEAS INCLUDE**

1. **TINEA CRURIS** – of the groin
2. **TINEA BARBAE** – of the beard area
3. T**INEA FACIAE** – of the face
4. **TINEA MANUM** – of the hand
5. **TINEA UNGIUM** – of the nails

 **iii) VIRAL SKIN INFECTIONS**

 **a) HERPES ZOSTER/SHINGLES**

* This is an acute painful self –limiting disease usually characterized by unilateral and segmental vesicular eruption.
* It’s caused by the same virus *varicella zoster virus* a member of a group of DNA tissue that causes varicella (chicken pox.)
* It’s normally confined to sensory dermatones or ganglia.
* Herpes zoster occurs in persons with AIDS, lymphoid or bone cancers persons who have not had chicken pox may develop it after exposure to the vesicular lesions of persons with herpes zoster.
* It is a clinical manifestation of the virus infection which remain latent in the tissues
* It spread by direct contact with vesicular lesions fluids or through respiratory secretions

 RISK FACTORS

* Elderly
* Lymphoid cancers
* Immunosuppressed clients
* HIV/AIDS patients
* Hodgkin’s disease pts

 CLINICAL FEATURES

* It begins as red swollen macules which develop into cramp of large tough vesicles, mainly in the chest (thoracic zoster) and forehead (optithalmic zoster).
* Pain is from moderate to severe.
* Very infectious in the 1st 2nd 3rd days.
* It can take 7-26 days
* Regional lymph nodes are inflamed (lymphadenopaty)
* Fever
* Headache
* Malaise and GI disturbances
* Itching and tenderness 4-5 days before eruption
* Severe pain i.e. burning usually accompanied by tingling sensation or dysthaesia

 TREATMENT

* Goal is to release pain and avoid complications e.g. infection, scarring, and post herpetic neuralgia and eye complications.
* Administer strong analgesics preferably opoids for pain relief
* Use antiviral –zoivax cream, Acyclovir (zovirax) (oral acyclovir) accelerates healing and reduces pain and is given in high doses of 400mg – 800mg po 5 times a day 1/52
* If pt reported within 48 hours give acyclovir 800mg tid 7/7 if after 48 hours give GV (geniton ventulin)
* Systemic steroids such as prednisolone 40-80mg tid 4/52 (to reduce post-herpetic neuralgia esp. in elderly and hepatic pain).reduce the dose gradually.
* Sedatives e.g. phenobarbitone 25mg od may be used at bedtime
* Thiamin lone injection SQ under painful areas is effective as an anti-inflammatory agent.
* To reduce itching use histamine antagonist
* Use antidepressant e.g. amitriptiline
* To prevent infection, use broad spectrum antibiotics
* Calamine lotion may also be used to decrease local discomfort
* Burrow’s solution (5% aluminum acetate) applied as cool compress may provide local relief and quickens drying of vesicles.
* For post herpetic neuralgia –use vit.biz(for nerve regeneration).
* For less severe, use acyclovir 5% cream for topical application

 NURSING MANAGEMENT

* Practice reverse barrier nursing
* Asses pt discomfort and response to meds
* Change dressing frequently
* Loose clothing helps minimize contact with affected area.
* Teach patient on how to apply wet dressing or meds on dressing

 HEALTH EDUCATION

* Abstain from sexual intercourse
* Adequate rest
* Good nutrition

 b)  **HERPES SIMPLEX**

* Caused by herpes simplex virus affecting mostly immunosuppressed pple.

 Types

1. *True primary infection episode*-initial exposure to the virus
2. *Non primary initial episode*-initial episode of either type 1 or type 2 in a person previously with infected or other type
* Herpes type 1 affects the mouth
* Herpes type 2 affects the genitalia

c) *Recurrent episode* -subsequent episode of the same viral type

 SINGS AND SYMPTOMS

* Symptoms vary with type
* Pain
* General malaise
* Skin lesions
* Fever

 DIAGNOSIS

* Physical exam and history taking
* Appearance of skin
* Acute vesicular lesions are likely to react positively to rapid essay whereas older crusted patches are diagnosed with viral culture.

 MANAGEMENT

* Hygiene
* Topical tx with drying agent accelerates healing
* Proper feeding
* Severe cases tx with acyclovir 400mg bd 5/7
* For recurrent episode i.e. up to 6 times/year start suppressive therapy-a combination of meds (acyclovir, valacyclovilr or famciclovir) that reduce viral shedding up to 90% hence reducing infection.
* True primary infection during pregnancy start on suppressive therapy to prevent complication during delivery and if in genital tract it is advisable for the mother to undergo c/s

 HEALTH EDUCATION

* Avoid touching lesion
* Wash hands thoroughly
* Inspect genital area
* Abstain from sexual intercourse
* Use condoms btw outbreaks

 COMPLICATION

* Sepsis
* Eczma herpetium
* Severe seborrhoe
* Scabies and other chronic skin conditions
* Herpes whitlow –is the infection of the pulp of the fingertip with herpes type 1 and 2
* Fetal anomalies include skin lesions, microcephally, encephalitis, intra cerebral calcifications.
* Osteomylitis

 **OTHER HERPES**

 c) **Genital herpes**

d) **Orlabial herpes**

 **iv) PARASITIC SKIN INFECTION**

 **a) PEDICULOSIS**

* Pediculosis is lice infestation and can be very prulitic
* There are 3 types
* Pediculosis capitis- Infestation of the scalp by head louse caused by *pediculus humanus capitis*.
* Pediculosis corporis – Infestation of the body by the body louse caused by *pediculus humanus corporis*
* Pediculosis pubis- Infestation of the genital region by louse caused by *phthirus pubis*

 DIAGNOSIS

* Physical exam of appropriate body parts by magnifying glass if need be.

MANAGEMENT

* Consists of topical application of pediculloside such as lindane (kwell scabene) permethin (mix), pyrehin (RID, A-200 pyrinate). A 2nd application in 7-10 days may be necessary.
* Pediculloside are not applied to on eyebrows or eyelashes instead apply petroleum jelly or remove nits manually.

 HEALTH EDUCATION

* Teach the patient and the family about the nature and prevention of pediculosis
* Soak beddings, clothing, combs and brushes in very hot water.

 COMPLICATION

* Severe pruritis
* Dematitis

 **b) SCABIES**

* Very nighty contagious pruritic infection
* It is infestation of the skin caused by female itch mite called *sarcoptes scabiei.* Prevalent during periods of overcrowding.
* Contracted through;
* Prolonged hand shaking
* sharing linen
* kissing
* Sexual transmission.

 PATHOPHYSIOLOGY

* The female itch mite penetrates the stratum corneum and into the skin.
* Within several hours of skin penetration it lays eggs large numbers and deposits fecal pellets.
* The larva mature in 10-14 days and move to the skin surface where females are impregnated.
* The cycle repeats itself
* Incubation period varies but often long period elapse before symptoms are noted
* Delayed hypersensitivity is the major factor in the laps between infestations and symptoms.
* In a previously exposed person incubation period is 4/52-6/52

 CLINICAL FEATURES

* Found at the webs of fingers, flexoral surfaces of the wrist, and at elbow, umbilicus in between buttons, inner thighs, sides of the feet pennies, nipple but hardly in face of adults.
* Itchy mostly at night
* Burrow vesicle –straight or tortuous threadlike ridge and can be a few mm long up to 1cm long.
* The bacterial infection can complicate the burrow
* First month after infection is symptomless
* Common in mentally challenged , malnourish and immunocompromised

 DIAGNOSIS

* Identifying the itch mite and remove from the end of the burrow with a pointed scalpel blade and placed on a slide with glycerol/mineral oil and examined under a microscope.

 TREATMENT

* Balneotherapy
* Treat the whole family members even if asymptomatic.
* Disinfect all the clothes
* Benzyl benzoate emulsion (20%)
* 1st apply and take a bath in 3-12 hours
* 2nd day apply and don’t take a bath
* 3rd day apply and take a bath
* Applied from neck to hands and on a dry body
* All family members esp. those sharing the same bed should be treated simultaneously to eradicate the mite
* If pt. still having severe itching, treat symptomatically with calamine lotion, antihistamine or even steroids.

 PREVENTION

* Good personal hygiene
* Search and treat all infected members
* Avoid skin contact with infected persons clothing esp health workers with patients.

 COMPLICATIONS

* Glumerulonepritis which can result to kidney failure
* Lymphadenopathy

 **3 ECZEMA /DERMATITIS**

* The term *dermatitis* and *eczema* imply inflammation of the skin which could be due to many causes.
* *Eczema* is the relationship pattern in the skin similar to relationship pattern of blistering and swelling.
* *Dermatitis* is inflammation of the skin caused by an allergic response, stress, response to unknown factor.
* Dermatitis is sometimes limited to inflammation of external origin while eczema is reserved to those of indogenous ausation

 ATIOLOGY

* Skin grafts
* Contact allergens
* Infection bacterial fungal or even protozoa
* Drug or factors e.g. xenous stasis in DVT

 CLINICAL FEATURE

* Are classified according to the state of the disease i.e. chronic or acute.

 i) ACUTE STATE

* Redness
* Swelling
* Exudation of serous fluid
* Erythema and hotness of the area
* Itching(pruritis)
* Excoriation and exudation coulpouring of fluid
* Edema

 ii) CHRONIC STATE

* Tick and leathery skin
* Formation of scales (scaling)
* Cracking of the skin i.e. fissuring.
* Lichenification

 CLASSIFICATION OF DERMATITIS

* Exogenous dermatitis e.g. contact dermatitis
* Endogenous dermatitis e.g. atopic dermatitis
* Mixed dermatitis e.g. tropical dermatitis

 **a) CONTACT DERMATITIS**

* It is exogenous type dermatitis
* This is an inflammatory action caused by exposure to agents which can either be physical, chemical or even biological agents
* Contact dermatitis can develop into eczema

HYPERSENSITIVITY REACTIONS

1. *Type I immunoglobulin E(IGE) mediated hypersensitivity,* Once the body is exposed to allergen, it develops antibodies i.e. IgE attached on the surface of the mast cells and basophills hence leads to a cascade of events.
* 1st exposure –body reaction by production of IgE chemical mediators attached to the mast cells.
* 2nd exposure –release of histamine which leads to initiation of the process of inflammation –local redness, vasodilatation, local infiltration, migration of neutrophils etc.

b. *Type II –cytotoxic hypersensitivity* –cross reacting antibody e.g. in gram myasthenia.

c. *Type III* –immune complex mediated hypersensitivity reaction, Result from complex found in the body

* Antigen –antibody complex are phagocytosed, or if not, circulate in blood and deposited in an organ.
* The complement system is activated in the organ affected leading to damage of tissues e.g. in glomeruloneptiritis and RHD.

 d.Type *IV* –Cell mediated hypersensitivity reaction.

* When the body is exposed to an allergen lymphocytes are sensitized.
* The allergen is therefore enclosed in one memory cells leading to production of numerous T-killer cells which destroy the helper cells encoded with the allergen massively hence tissue damage
* Contact dermatitis can be divided into two sub –groups i.e. irritant contact and allergic contact dermatitis

 i. IRRITANT CONTACT DERMATITIS

* This is caused by toxic or damaging effects or some chemicals in the skin.
* Strong irritants injure the skin instantly on 1st contact.
* The immediate inflammatory reaction is the same for all individuals
* Repeated exposure to weak irritant such as solvents and detergents over a long period of time can also cause this type of dermatitis.
* It is usually presents as thickening and dryness of the skin accompanied most often than not with cracking.

 ii. ALLERGIC CONTACT DERMATITIS

* This is a manifestation of delayed hypersensivity reaction type IV caused by allergens
* This is a response to a substance to which the individual has become sensitive to.
* Develops in susceptible following 1st exposure to allergens.
* May results from
* Drug
* Latex
* Rubber
* Jewel
* Dye

 CLINICAL FEATURES

* Rashes develop from the point of contact with allergen or irritants.
* Itching and burning sensation
* Systemic signs e.g. oedema if acid, fever, malaise
* Pain
* Erythama redness
* Oozing or wheeping of serous fluid
* Skin may develop bullae erosions and ulcerations
* Dermatitis caused by strong irritants produce acute localized blisters or swelling that spread.

 MANAGEMENT

* Remove the victim from the cause as soon as possible
* In acute phases use wet compresses to minimize edema and burning sensation.
* Uses weak steroid lotions and cream e.g. hydrocortisone ,low doses of steroids e.g. prednisolone 40mg Od 10/7
* Anti- pruritic agents (antihistamine) to relieve symptoms e.g. calamine brion.
* Sedatives
* Watch for secondary infection

 **b) ATOPIC DERMATITIS**

* It is an inflammatory allergic condition of the skin
* It is a genetically determined condition manifested by familial tendencies to develop allergic reaction e.g. in asthma, utricaria etc.
* All atopics have a tendency to develop a lot of IgE i.e. type I hypersensitivity reaction

 CLINICAL FEATURES

* This is disease does not develop until 3 months of age because before this age a child has not yet developed IgE.
* The disease has a natural cause divided into 3 distinct stages i.e. infantile phase, childhood phase and adult phase.

 i. INFANTILE PHASE.

* Skin rashes are not present at birth but develop between ages 3 -6 months.
* Red rashes develop on the face involving the cheeks, the forehead and behind the ears initially but then they spread to the back, chest, nappy area and feet.
* The rashes then become wheepy (because of fluids) and crust develops.
* There is a lot of itching and scratching which may be severe interfering with steep.
* The infant is very irritable scratching the rashes or every available surface.
* Bacterial infection may occur.
* Excercation may be triggered by URTL etc.

 ii. CHILDHOOD PHASE

* The child passes the disease from infantile phase
* In childhood the disease is more localized on flex oral aspects of elbows ,the knees, wrist and ankles
* Itching is severe and contact scratching causes lichenification giving an appearance of a grey dirty colour to the affected sites.

 iii. ADULT PHASE

* There is persistence of flex oral involvement.
* The forehead, eyelids and sides of the neck are involved in severe cases.
* The areas are hyperpigmented and shows clear discolouration.
* There is severe pruritus
* Excoriation is often common
* Sweat and moisture including pruritus and in some cases the skin may appear very dry (xerosis)

 CAUSES OF THE DISEASE

* Almost ½ of infantile cases clear by age 18 month
* Not all patients pass through the 3 stages; a few will be having the disease in adulthood.
* In most cases the rashes disappear completely at between ages 20 -30 yrs but severe cases may persist longer.

 MANAGEMENT

* In case of infantile and childhood phase atopic dermatitis the parent should be educated on disease and its natural history and they should actively participate in management.
* All the aggravated factors should be determined as soon as possible.
* Very severe cases should be treated.
* In case of acute attack, cold compress should be applied –this helps to cool the skin , minimize oozing and pruritius
* Apply 1% hydrocortisone topical cream tds.
* During flare –ups or recurrences, short course steroid may be given e.g. prednisolone 40mg OD in decreasing doses within a period of one week i.e. 40mg -13mg OD then dose maintained for 2 -3 weeks
* Give calamine ointment to sooth the skin.
* Give antihistamines, sedatives tranquilizers during acute stages.
* Give antibiotics incase of secondary infections.

COMPLICATION

* Asthma
* Seborrheic dermatitis
* Infantile dermatitis

 **4 NONINFECTIOUS INFLAMATORY DERMATOSES**

 **a) PSORIASIS**

* Psoriasis is a chronic inflammatory disease of the skin in which the productions of epidermal cells of the basal layer occur at a rate that is approximately 6 -9 times faster than normal.
* The rapid reproduction of cells is accompanied by rapid transition of cells from the germinative layer of top of the stratum corneam
* The transition is reduced from the normal 30 -40 days to around 7days .this occurs anywhere in the body.
* There`s a genetic predisposition top psoriasis but the cause is idiopathic.
* It has a tendency to improve but keeps on recurring throughout life.
* Some of the factors triggering recurrence include,
* Bacterial infection e.g. pharyngitis
* Emotional trauma
* Mechanical trauma
* Drugs e.g. receptor antagorusta e.g. propranoloc
* Day to day fear and wear
* Seasonal and hormonal changes
* Emotional stress and anxiety

 CLINICAL FEATURES

* Pruritus
* Lesion appear as red raised patches on skin covered with silver scales
* If scales are scooped away, a dark red base of the lesion is exposed producing multiple bleeding points.
* Parts mainly affected include the scalp ,flex oral areas of back and genitalia
* Bilateral symmetry affecting both sides of the body is a feature of psoriasis.
* In about ¼ of all the affected, the nails are affected and they present with pitting, discolouration, crumbling, beneath the free edges, dubbing and separation of the nail plate.
* Pastular lesion may occur if it occurs on the palms and soles.

 DIAGNOSIS

* Based on clinical findings
* Rule out fungal infection

 MANAGEMENT

* Goal is to allow the rapid turnover of epidermis to promote resolution of the psoriatic lesions and control the natural cycles of the disease
* Remove all the aggravating factors
* There is no known cure
* Gentle removal of scales this can be accomplished by baths
* Oils such as olive oil, mineral oil or areeno oil, oatmeal bath or coal tar preparations (balnetar) can be added to water and a soft brush used to scrub the psoriatic plagues gently.
* After bathing the application of emollient creams containing alpha hydroxyl acids or salicylic acid will continue to soften thick scales.
* Cover the skin with tar or antralin
* Three types of therapy are standard; topical, systemic and intralesional
* Use of topical steroids e.g. betamethason covered with a heavy dressing to reduce the multiplication of cells
* Phototherapy /ultra –violet radiation
* Use of systemic cytotoxic drugs e.g. methot

 COMPLICATIONS

* The pt may develop exfoliative psoriasis a condition in which the disease spreads entire body.
* Asymmetrical rheumatic factor, Associated with arithritis of multiple joints causing a crippling disability, the cause of this phenomenon is rarely understood.

 **5**  **SKIN ULCERATION**

* Ulcer is the erosion of the skin due to local destruction extending past the epidermis

 a) **PRESSURE ULCER**

* It is a lesion caused by unrelieved pressure against soft tissue usually over a bony prominence

 EXTRINSIC FACTORS

* Moisture, shear, friction and pressure which can also be internal pressure

 INTRINSIC FACTORS

* Relate to characteristics with pts skin structure determined by amount of collagen in the body, age, nutritional status and use of steroids, presence with spinal injury or other alterations in mobility and perception and degree of perfusion.

 FACTORS AFFECTING SKIN PERFUSION

* Blood pressure
* Extra corporeal circulation serum protein albumin
* Pre-albumin levels of HB and HCL haematocrit level
* Smoking
* Vascular diseases
* Administration of vasoactive meds
* Poor circulation

 PATHOPYSIOLOGY

* Unrelieved pressure causes cellular necrosis as a result with vascular insufficiency
* Pressure applied to soft tissue compress capillaries destroying structure and occluding blood flow.
* Compensation by increased shunting of capillary circulation to the area under pressure increase permeability and fluid leak into tissues endothelial cells are disrupted platelet aggregate thrombi form in capillaries and lead to cellular death which result in tissue necrosis

 CLINICAL MANIFESTATION

* Ischemia is followed by reactive hyperemia
* Tissue edema and inflammation
* Erythema (redness) that may / may not resolve once pressure source is removed
* Visible pressure ulcer and tissue damage

 CLASSIFICATION

* Stage/grade I
* Involves slight erosion of the epidermis with some extension into dermal layer producing redness (erythema) indurations
* Erythema does not blanch with pressure and may progress to dusky blue-gray
* Skin temperature is elevated
* Tissue swollen and congested
* Patient complains of discomfort
* Stage/ grade II
* Involves epidermis and dermis and with extension into subcutaneous layer.
* Skin breaks
* The shallow ulceration shows indurations redness and heat does not blanch to touch
* Edema persists
* Ulcer drains
* Infection may develop
* Partial thickness wound
* Stage / grade III
* Involves epidermis, dermis, subcutaneous layers and with extension down to including muscle
* The ulceration is deep, foul smelling with necrosis, underming of tissue and drainage continue
* Infection develops
* Full thickness wound with borders hyper pigmentation
* Stage / grade IV
* Involves all layers of the skin the underlying muscles and bone or joint
* Deep pocket of infection develop with foul smelling
* Necrosis and drainage continue
* Underming is extensive Borders are darkly pigmented
* Full thickness wound

 MANAGEMENT

* Open draining ulcer may require culture and sensitivity to identify the pathogen and appropriate abx
* Surgical management flaps and skin graft depending on pts condition, compliance and type o f wound
* Debridement agents which may be;
* Enzymatic debridement– use of commercially preparations enzymes agents that the body can produce naturally e.g. collagenase (santyl), accu-zyme, granulex and zymase.
* Keratolytic debridement-agents that promote separation of epidermal tissue and may also be antimicrobial and stimulate granulation
* Autolytic debridement- Use body’s own digestive enzymes that promote softening, liquification and separation of necrotic tissue and keeping ulcers moist with occlusive dressing.
* Absorptive debridement – agents that absorbs products of tissue breakdown

 NURSING MANAGEMENT

* Relieving pressure
* Turning the pt 1-2 hours interval
* Encourage pt to shift weight actively every 15 minutes
* Position the patient
* Recumbent position is preferred to semi-fowler’s position unless contraindicated because it increases supporting body surface area in this position
* Using pressure relieving devices
* Liquid mattress
* Air/rubber ring
* Air fluidized beds
* Heel/elbow ring
* Improving mobility
* Encourage the pt to do ROM or ambulate
* Improving nutritional status (Diet)
* Protein –tissue repair
* Calories- Spare protein

- Restore normal weight

* Water – maintain homeostasis
* Multivitamin – promote collagen formation
* Vitamin C – promote collagen synthesis

 - Support integrity of capillary wall

* Zinc sulfate- cofactor for collagen formation and protein synthesis

 - Normal lymphocyte and phagocyte response

* Vitamin A – stimulate epithelial cells

 - stimulate immune response

***Caution*** *an excess of vitamin A can cause an excessive inflammatory response that could impair healing*

* Reducing friction and shear
* When positioning lift the pt avoid dragging pt across a surface
* Improving sensory perception
* Minimizing irritation moisture
* Keep the pt dry fro urine, stool, drainage by changing the wet linens
* Drying agents and powders are avoided instead topical barrier ointments e.g. petroleum jelly may be helpful
* Absorbent pads that wick moisture away from the body should be used to absorb drainage
* Promoting pressure ulcer healing
* The pt must not sit or lie on the pressure ulcer for even a few minutes

 COMPLICATIONS

* Pyathrusis (formation of pus against bone cavity)
* Oesteomyletis
* Septicemia
* Septic shock

 **b) STREPTOCOCCAL ULCER**

* Common in rural tropics
* Common in men, especially on lower legs and may follow insect bites
* Combine topical and systemic antibiotics

 **c) BURULI ULCER**

* Caused by *mycobacterium ulcerans*
* Highly destructive ulcerating condition
* Dx acid fast bacilli in ulcerated edges
* Surgical repair may be needed
* Prolonged administration of clofazimine may be help

 **d) LEG ULCER**

i) VENOUS ULCER

* Mostly in women with history of thrombophlebitis
* Follows minor surgery
* Not painful

 ii) ARTERIAL ULCER

* Cause atherosclerosis in elderly men than women
* Surgery lumbar sympathectomy
* Very painful and bilateral

 **6 BLISTERING DISEASES**

 **ERYTHEMA MULTIFORE/**

 **a) STEPHENS-JOHNSON SYNDROME (SJS) AND TOXIC EPIDERMAL NECROLYSIS (TEN)**

* These are potentially fatal skin disorders triggered by a reaction to medications e.g. abx sulfonamides i.e. cotrimozazole, ant seizure agents, NSAIDS.

CLINICAL MANIFESTATIONS

* Conjunctival burning (itching)
* Cutaneous tenderness
* Fever
* Cough
* Sore throat
* Headache
* Extreme malaise
* Myalgias (aches and severe pain)
* Oozing pus incase of infection
* In severe cases there is mucosal involvement that is damage to the larynx, bronchi, and esophagus from ulcerations.
* Also fingernails, toenails, eyebrows and eyelashes may shed along with the surrounding epidermis.

DIAGNOSIS

* History of use of medication that precipitate TEN and SJS
* Histological studies of frozen skin cells from a fresh lesion and cytodiagnosis of collections of cellular material from a freshly denuded area are conducted.
* Immunofluorescent studies may be performed to detect atypical epidermal auto antibodies.
* Genetic predisposition to erythema multiforme may have been suggested but is not confirmed

 MANAGEMENT

* The goal of treatment includes control of fluid and electrolyte balance, prevention of sepsis and ophthalmic complications.
* Manage as a severe burn patient
* Supportive care is the main stay of treatment
* All nonessential medications are discontinued immediately.
* Surgical debridement or hydrotherapy in Hubbard tank (large steel tub) may be performed to remove involved skin.
* Administer systemic abx
* Avoid steroids as they delay healing
* Rehydrate the pt with IVFS
* Use aseptic technique
* Provide analgesics

 NURSING MANAGEMENT

* Isolate to prevent infection (Reverse barrier nursing)
* Provide warmth if hypothermia due to severe skin damage
* Regular cleaning
* Use of sterile sheets
* Diet rich in protein insert
* NG if esophagus is ulcerated
* Provide psychological care
* Use of bed cradle

 COMPLICATION

* Sepsis
* Keratoconjuctivitis which can impair vision and result in conjuctival retraction, scarring and corneal lesions.

 **7 SKIN TUMORS**

 **a) BENIGN TUMORS**

 i) **KELOIDS(CHELOIDS)**

* Are overgrowth of the connective tissue
* Occur at the site of previous trauma or scar
* Common in black females

 MANEGEMENT

* Administer intra lesion corticosteroids injection esp. triamcinolone
* Surgical excision followed by radiationothes

 **b) METASTATIC SKIN TUMORS**

 **i) KAPOSI’S SARCOMA**

* This is skin malignancy of the endothelial cells that line the small blood vessels.
* It is common in HIV pts before the era of HIV it presented as lower extremities skin lesions in elderly men.
* It is subdivided into three categories;
* Classic KS occurs predominantly in men of Mediterranean or Jewish ancestry btw 40-70 years. Most pts have nodules or plaques on the lower extremities that are rarely metastasize beyond this area. Classic KS is chronic relatively benign and rarely fatal.
* Endemic (African) KS affects people predominally in eastern half of Africa near the equator. Men are affected more than women and children can be affected as well. The disease may resemble classic KS or it may infiltrate and progress to lymphadenopathic forms.
* Immunosuppression-associated KS occurs in transplant recipients and people with AIDS.

 CAUSES

* Idiopathic
* Virus
* Immunosuppressant agent

 SINGS AND SYMPTOMS

* Reddish dusty or bluish black nodules
* Bloody or ulcerated nodules

 MANAGEMENT

* **Surgical excision of the lesion**
* **Dress the wound (wet dress)**
* **Administer abx when infected**
* **Treat the underlying condition**
* **Manage HIV/AIDS if pt is infected**
* **Chemotherapy with cytotoxic drugs**
* **Radiotherapy**
* **Health education on health compliance**

 **8 OTHER SKIN CONDITINS**

 **a) NECROTIZING FASCIITIS**

* **This is edema and necrotizing of subcutaneous tissue including fescla of tissue**
* **It is infection of the skin function usually caused by *beta haemoyltic streptococcus, but staph aureus* and other organisms may be involved peptostreprococcus, and bacteroids.**
* **The infection is spreads following the superficial and deep fascial planes. It follows surgery or perforating wounds.**

 **SINGS AND SYMPTOMS**

* **Sudden onset often following trauma**
* **Skin resembles that of deep cellulitus**
* **Progressive fever (acute on onset)**
* **Cutaneous lesions**
* **Redness, swollen and tenderness**
* **Deep necrosis may ulcerate and open like a crater**

 **PHATHOPHYSIOLOGY**

* **Bacteria->inflammation (redness, pain, tenderness, swelling)-> poor blood supply-> skin changes to pale/purple->necrosis, gangrenous, anaethetic-> death of cells**

 **DIAGNOSIS**

* **History taking of abdominal surgery, DM, alcoholism**
* **Biopsy for culture and sensitivity**

 **MANAGEMENT**

* **Immediate hospitalization and iv penicillin are imperative**
* **Wide surgical exploration to remove necrotic tissue may be needed (Debridement)**
* **Wound left open**
* **Administer broad spectrum abx**

 **COMPLICATION**

* **Septicaemia**

 **b) CELLULITUS**

* **This is the skin infection where there is inflammation of the cells by *group A beta haemolytic streptococcus, staph. aureus***
* **Can occur as a single isolated events or a series of recurrent events. It is often misdiagnosed as a recurrent thrombophlebitis or chronic venous insufficiency.**

 **CLINICAL FEATURES**

* **Red, tender swelling with ill-defined borders**
* **Edema**
* **Malaise**
* **Low grade fever**
* **Chills**
* **Increased W.B.C**
* **Warmth and pain**
* **Sweating**
* **Enlarged lymph nodes**

 **PATHOPHYSIOLOGY**

* **The pathogen penetrates the normal skin barrier via a minor injury, wound or ulcer and releases their toxins in the subcutaneous tissues causing inflammation response with the tissue.**
* **The inflammation spreads horizontally through the tissue.**

 **DIAGNOSIS**

* **History taking and physical exam**

 **MANEGEMENT**

* **Mild cases should be treated as O.P.D basis with oral abx therapy e.g. erythromycin, doxycycline,**
* **Analgesics for pain**
* **Administer tetanus toxoid vaccine**
* **Administer aqueous procaine and benzathine penicillin injection**
* **Debridement if necrosis is there and skin graft**

 **NURSING MANEGEMENT**

* **Elevate the affected area to prevent edema**
* **Bed rest and provide ADLs and exercise ROM**
* **Apply warm moist packs to the sites every 2-4 hours but caution be taken on DM and paralysis pt to avoid burns**
* **Wound care daily use aseptic technique**
* **Skin care and oral hygiene**
* **Tepid sponging**

 **COMPLICATIONS**

* **Septicaemia**
* **Thrombophlembitis**
* **Necrotizing fasciitis**

 **c) PELLAGRA**

* **This is a skin condition that occurs due to inadequate amounts of nicotinic acid.**
* **Pellagra is found in areas where maize is the staple diet.**
* **Adults more affected than children**
* **Onset is insidious (gradual)**
* **Affects those parts exposed to sunlight e.g. the back of the hands (often sparing the last digit), face, wrists and forearms**
* **The face usually show a butterfly pattern and the V of the neck often presents a well outlined border (casal’s necklace)**

 **CLINICAL FEATURES**

* **Itching**
* **Pain**
* **Burning**
* **Diarrhea and vomiting**
* **Poor appetite**
* **Weight loss**

 **MANAGEMENT**

* **Topically soothing compression with calamine lotion can be used**
* **Antihistamine, steroid, antdiarrhoea and abx preparations are useful**
* **Niacinamide 100mg po qid and a well balanced diet (fruits, vegetables, high protein poultry & fish meat and eggs,) are rapidly effective**
* **Treatment should be reinforced with riboflavin**
* **Analgesics for pain**
* **Rehydrate to treat diarrhea**
* **Dress exposed wounds**
* **Psychological care**

 **d)JIGGERS INFESTATION (TUNGIASIS)**

* **Refer communicable diseases 4th edition page 28**

e)ptyriasis versicolor

* Caused by fungus called *mala ssezia furfur*.
* Common in young people, pregnancy and steroids reduced immunity.

 CLINICAL PRESENTATION

* Start as scaly macules at the back
* The macules form a collision to give irregular maps
* The colour of the area changes
* No itching unless when it goes to the chest, shoulders and can spread to hands and neck.
* If it reaches the face the person is immunocompromised.

 TREATMENT

* Antifungal ointment e.g. Whitefield salicylic acid, clotrimazole etc.