Derma Summary

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Good luck ..

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			Skin Infectio	ns		
Disease		Description	Organism	+ info	Management	Picture
			Bacterial	·	·	
Impetigo	Non- bullous	 Most common. Clusters of vesicles or pustules with rim of erythema that rupture and develop golden-yellow (Honey) crust 	 S. Aureus Group A beta-hemolytic streptococcus 	 Warm humid climate Very contagious <u>Complications</u>: 	 Swab Remove crust Localised topical abx (bactroban, bacitracin) 	
	bullous	 Affect newborn and older children. Flaccid bullae on grossly normal skin Face, hands , diaper area 	- S. Aureus	 Guttate psoriasis post-streptococcal glomerulonephritis. 	 recurrent → Mupirocin Severe → systemic Abx (cephalosporins) 	
Erysip	ielas	 Superficial infection with lymphatics involvement. Well-defined, unilateral, red edematous. (face & leg) 	- Group A beta-hemolytic streptococcus.	LeukocytosisFeverChillsBacteremia	 Smear + culture Cold compressor. Oral Abx (penicillin) or I.V. for severe infection 	Адам
Cellulitis		 Deeper involvement of the dermis and subcutaneous tissue. Acute, ill-defined, raised, hot, tender, erythematous (leg). 	- Strep. Pyogenes, - S. Aureus.	 Palpable tender LN Leukocytosis Fever RF: DM, HTN, obesity Complication → lymphedema 	 Swab + blood culture Acetaminophen IV penicillinase-resistant penicillin or Erythromycin if allergic. 	PAR
Follici	ılitis	 Superficial hair follicle infection Multiple small papules / pustule on an erythematous base. Heals without scarring 		- <u>Complications</u> : abscess formation and cavernous sinus thrombosis.	- Topical and systemic Abx	and
Furuncle (boil)		- Inflammation of deep portions of hair follicle	- S. Aureus	-	Swab: Culture and GSAntibacterial soapAntibiotics.	AC.
Carbu	ncle	 Infection of multiple hair follicles Larger more deep seated Drainage through multiple points in the skin. 		-	 Swab : Culture and GS Screen for carrier state Antibiotics (IV) 	

Disease		Description	Organism	+ info	Management	Picture		
Viral								
	Plane (Verruca planae)	 Affects Face, back of hands . Flat skin colored papules, no hyperkeratosis. Not painful. 	- HPV type 3,10	-	Topical retinoids.CryotherapyLaser			
Warta	<mark>Common</mark> (verruca vulgaris)	 Affects the hands. Common in Children Skin color hyperkeratotic papules. Koebner phenomenon. 	- HPV type 1,2,4	-	- Topical keratolytics			
Warts	Plantar (verruca plantares)	Affects the soles.Often painful.Black dots, thrombosed BV.	- HPV type 1,2,4	-	Topical retinoids.CryotherapyLaser			
	Genital wart	 Most common STD Condylomata accuminata Penile, vulvar skin, perianal area Cauliflower like 	 HPV 6, 11 Oncogenic: HPV 16, 18 	- Treat the partner	 Podophyllotoxin: cytotoxic Imiquimod: immunmod. Physical: cryotherapy, laser 			
Mollus contag ، المعدي	iosum	 Affects the face, neck Skin colored papules with central punctum (umbilication) Koebner phenomenon. 	- Pox-virus	 Hunderson-patterson bodies. In children by contact. Adults: STD, immunosuppression. 	 Involute spontaneously. Curettage, cryotherapy. 	9 33 9 9 0		
Herpes simplex		 Group of small blister. Mucocutaneous Painful Recurrent 	- HSV-1(H. labialis) - HSV-2 (genital herpes)	 Herpetic whitlow (fingertips) Eczema herpeticum. 	 Tzanck Smear. Serology (IgG, IgM). Direct fluorescent ab. Viral culture. Acyclovir. 			
Herpes zoster		 Grouped blisters and vesicles on background of erythema. Prodromal pain—dermatomal post-herpetic neuralgia. 	- Chickenpox virus.	 Complications: conjunctivitis & keratitis. Facial nerve affected → Ramsay Hunt Syndrome. 	 Tzanck Smear. Direct fluorescent ab Analgesia, drying agent Acyclovir. 			

Dise	ase	Description	Organism	+ info	Management	Picture			
	Fungal								
Candio	diasis	 Satellite lesions → Napkin candidosis & Intertrigo (skin folds). Paronychia, oral thrush Very bright, ill-defined, oozing. 	- Candida albican.	-	 Swab and KOH Dry. Nystatin cream. Imidazole Antifungal (itraconazole). 				
	T. pedis	 Sole of foot (athlete's), webs. Erosive interdigitalis. Well defined scaly erythematous plaque, Itchy. 	 Hyperkeratotic → T. rubrum Inflammatory → T. mentagrophyte 	- Itchy, chronic.	-				
	T. Ungum	 Affects the nails Onycholysis. Subungual hyperkeratosis. 	 T. rubrum T. mentagrophyte 	-	- KOH and culture.				
Dermato	T. manun	 Diffuse dry scaling over the palm. Hyperkeratosis with vesicle and pustules. 	-	-	-	A DAY			
phyte Infection S	T. Cruris	Infection of genital skinActive border.	-	-	-				
	T. capitis	- Well circumscribed pruritic scaling area of <u>hair loss</u> (grey patch)	 Black dot → T. tonsurans Ectothrix → M. canis, M. Audouinii 	- Itchy, chronic.	 KOH and culture Wood's light greenish- yellowish. Topical (terbinafine, daktarin) 				
	Tinea corporis	- Well-demarcated annular red hyperkeratotic plaque with central clearing (Ring worm)	 Hyperkeratotic → T. rubrum Inflammatory → T. mentagrophyte 	-	-	S JA			

Disease	Description	Organism	+ info	Management	Picture
Pityriasis versicolor النخالة المبرقشة	 Yellowish brown → (in white skin) Hypopigmented → (in dark skin) Trunk Asymptomatic. 	 Malassezia furfur (hyphae). Pityrosporum orbiculare (yeast). 	-	 Wood's lamp → coppery-orange fluorescence. Topical imidazole (nizoral). 	
		Protozoal			
Leishmaniasis	 Slowly growing painful papule into ulcerated nodule or plaque Exposed sites. Transmitted with sandfly. 	 Sand fly L.tropica , L. major Sand fly (promastigote). 	 Clinical patterns: Cutaneous: (localised or diffuse). Mucocutaneous. Visceral (post kala-azar): liver & spleen 	 Skin biopsy (amastigotes) Tissue impression smears (touch preparations) smears of dermal scrapings (edge of ulcer) Giemsa stain. PCR-based method. Rx: Parenteral pentavalent antimonials (Sodium stibogluconate) 	
		Infestation	n		
Scabies الجرب	 Severe itching worse after bathing and at night. Sites → finger webs, flexor of the wrist, axillae, areolae, umbilicus, lower abdomen and scrotum. Linear burrows (tunnel of mite) Small erythematous papules+ variable degree of excoriation vesicles, indurated nodules or crustacean. 	 Sarcoptes scabiei var. Hominis 60-90 eggs in her 30-day lifespan. 	 Might be complicated by secondary bacterial infection. Other member of the family also having severe pruritus. 	 India ink or gentian violet. Tx of family members. Washing clothing & linen Permethrin cream Lindane lotion or cream (not safe in children d.t neurotoxicity or pregnancy). Crotamiton cream. 	
Pediculosis capitis القمل	 Common in school children. Severe itching of the scalp (occipital) Posterior cervical LN. 	 pediculus humanus var capitis → 3-6 eggs (nits) per day 	- Secondary bacterial impetigo.	 Identification of nit/adult Examination of other family members Wash all fomites Combing: metal nit comb Pyrethrin. Permethrin lotion 	

Papulosquamous

Disease	Types	Pic	Features	Notes
	Plaque psoriasis (Psoriasis Vulgaris)	 Most common Silvery scaly round-to-oval red plaques distributed over extensor body surfaces and the scalp 		
	Guttate	- di 	 Small, droplike, 1-10 mm in diameter, salmon-pink papules, usually with a fine scale Younger than 30 years Trigger: streptococcal infection 	Risk factors: - Drugs (lithium, anti-malarial drugs, NSAIDs and beta-blockers) - Koebner phenomenon
Psoriasis Inflammatory skin condition with reactive	Erythro- dermic		 Scaly erythematous lesions over large areas with few typical psoriatic plaques Hair shed; nails become ridged & thickened Unwell, fever, leukocytosis Increase cutaneous blood flow > Excessive body heat and hypothermia > Increase percutaneous permeability > Increase percutaneous loss of water, protein & iron 	Histology: - Parakeratosis - Auspits sign: due to thick dermis & thin epidermis - Epidermal polymorphonuclear leucocyte infiltrates (Epidermo-Tropism) = (Munro abscesses)
abnormal epidermal differentiation & hyper proliferation (within 10 days).	Pustular		 Sterile small pustules on erythematous background 1- Generalized type (von Zumbusch variant): Generalized erythema studded with interfolecular pustules. Fever, tachypnea and tachycardia. Absolute lymphopenia with polymorphnuclear leukocytosis up to 40,000/µL. 2- Localized form (in palms and soles) 	Causes of Pustular Psoriasis: - Idiopathic - Withdrawal of systemic steroids - Drugs; including: Salicylates, Lithium, Phenylbutazone, Hydroxychloroquine, Interferon - Strong, irritating topicals: Tar, Anthralin,
	Psoriasis inversus (Sebopsoriasis)		 Occurs in armpits, groin and skin folds The erythema and scales are very similar to that seen in Seborrhoeic dermatitis (it has no or very thin scales) 	Steroids under Occlusion, and Zinc Pyrithione in shampoo - Infections - Sunlight (or Phototherapy)
	Psoriatic Arthritis	Distal Onycholysis Subungual hyperKeratosis Nail pitting	 Most commonly a seronegative oligoarthritis. Asymmetric oligoarthritis occurs in as many as 70% of patients with psoriatic arthritis DIP joint involvement occurs in approximately 5-10 Arthritis mutilans: 5% of patients 	- Cholestatic Jaundice - Hypocalcemia.

Disease	Types	Pic	Features	Notes
	Hypertrophic		These extremely pruritic lesions are most often found on the extensor surfaces of the lower extremities, especially around the ankles.	-Associated with: Ulcerative colitis, alopecia areata, vitiligo, dermatomyositis, HCV infection, chronic active hepatitis, and
	Atrophic		- Characterized by a few lesions, which are often the resolution of annular or hypertrophic lesions	primary biliary cirrhosis - Drug induced: Thiazide, Antimalarials, Propranolol.
	Erosive	Typical appearances and locations of lichen planus Classic lichen planus Vielet, flat-topped papules Nail scarring papules	- Risk of squamous cell carcinoma	Clinical features:
	Follicular	Oral Lichen planus White Licy appearance	 Keratotic papules that may coalesce into plaques A scarring alopecia may result 	 Initial lesion on the flexors > generalized eruption develops with within 2-16 weeks
Lichen Planus	Annular	(Widhan shite) Erosion	- Annular lesions with an atrophic center can be found on the buccal mucosa and the male genitalia	 Papules are violaceous, shiny, & polygonal; varying in size from 1mm to greater than 1 cm in diameter
rialius	Vesicular and bullous	lypervignentation	 Develop on the lower limbs or in the mouth from preexisting LP lesions 	 Fine, white lines, called Wickham Stria, are often found on the papules Nail plate thinning causes longitudinal
	Actinic		 Mildly pruritic eruption Nummular patches with a hypo-pigmented zone surrounding a hyperpigmented center 	 grooving and ridging Subungual hyperkeratosis and Onycholysis. Pterygium unguis (Dorsal pterygium)
	Pigmentosus		 Common in persons with darker-pigmented skin Usually appears on face and neck. 	forms as a result of scarring between the proximal nailfold and matrix.
	Plano-pillaris	-	- Patchy progressive permanent hair loss mainly on the scalp	-
Pityriasis Rosea نخالة وردية	-	Hend path	 Increased incidence in Spring and Autumn Begins with <u>herald spot/patch</u> Salmon-colored macule over trunk and proximal limbs, looks like secondary syphilis but it spares hands & soles Pruritus is common, usually of mild-to-moderate severity 	 Human herpesvirus (HHV)-7 & HHV-6 Drug induced: Captopril, metronidazole, isotretinoin, penicillamine, bismuth, gold, barbiturates, & omeprazole

Papulosquamous diseases management								
Psoriasis	Lichen Planus	Pityriasis Rosea						
 1st line Topical agent for mild to moderate psoriasis: Emollients Keratolytics (salicylic acid) > for scales Coal tar & Anthralin > for increased mitosis Vitamin D Corticosteroids Retinoids 2nd line If no response to topical therapy or >20% BDA involved: Phototherapy Natural sunlight UVB UVA & Psoralen 3rd line If no response or + psoriatic arthritis Methotrexate > check for LFT Others: Acitretin (oral retinoid) Cyclosporine Biological therapy (Infliximab) > check for TB 	 Self-limited disease that usually resolves within 8-12 months. Treat to prevent hyperpigmentation. Antihistamine (for pruritus). Topical steroids, particularly class I or II ointments Systemic steroids for symptom control and possibly more rapid resolution Oral Acitretin (Retinoid). Photo-therapy 	 Tends to resolve over the next 6 weeks without scarring Reassurance that the rash will resolve Relief of pruritus Topical menthol-phenol lotion Oral antihistamines Topical steroids Systemic steroids Ultraviolet B (UVB) light therapy Antiviral 						

General principles of eczema /dermatitis

Disease	Description	Clinical features	Management	notes	Picture
Eczema (Dermatitis)	 Skin inflammation characterized by → itchy, scaly, patches of erythema. 	 <u>Subacute Dermatitis:</u> misdiagnosed as tinea <u>Chronic Dermatitis:</u> misdiagnosed as psoriasis 	-	 Eczema classified: <u>Acute</u>: erosion, oozing and vesicles <u>Subacute</u>: Redness + swelling, crust±scale + infection <u>Chronic</u>: lichenification, dark pigmentation and thick papules and plaques. 	
Atopic Dermatitis	 Chronic <u>relapsing</u> itchy skin disease in genetically predisposed patients Defective epidermal differentiation (filaggrin mutations) → impaired barrier function of the skin 	 Colonized by S aureus. Infantile → acute itchy vesicular eczema on extensor/facial involvement (Diaper area is spared) Childhood → subacute and chronic at antecubital and popliteal fossae, neck, wrists and ankles, lichenification, post-inflammatory hyperpigmentation. Adult → chronic inflammation on flexors and hand dermatitis. 	 70% remission before adolescence Diagnostic Features: <u>Major</u> → pruritus, chronicity, Personal or family. <u>Minor</u> → Xerosis (dry), early onset, cheilitis, nipple eczema, Dennie morgan fold, Pityriasis alba,keratoconus. Treatment: Education, Emollient. Topical steroid Topical immunomodulators. Oral antihistamine Oral Antibiotic. 	 Associated with personal or family history of other atopic conditions as asthma, allergic rhinitis, conjunctivitis or atopic eczema 85% begin before 5 yrs AD associated with local infiltration of Th 2 that secrete IL-4, IL-5, IL-13, IL-31 Complication: Eczema Herpeticum, Impetiginized dermatitis, Mollusca Contagiosa. المليساء المعدي 	
Seborrheic dermatitis	 In regions with high sebum production & the body folds Erythematous red-yellow patches & thin plaques with bran-like to flaky (greasy) scales. 	 Commensal yeast Malassezia furfur (pityrosporum ovale) 	 Medicated shampoo Topical antifungal potency topical steroid. Topical immunomodulators (tacrolimus & pimecrolimus) Salicylic acid 	- Cradle cap: scaly & crusty mass covering most of the scalp & can be seen in infantile seborrheic dermatitis.	

Disease		Description	Clinical features	Management	notes	Picture
	Allergic	- Dermatitis resulting from type 4 reaction.	 Acute form present with crusted erythematous papules, vesicles & bullae that is well demarcated & <u>localized</u> <u>to the site of contact</u>. 	Dx: - Hx and Examination.	 Nickel. Fragrances. Preservatives. Hair dyes. Rubber latex. 	
Contact Dermatitis	Irritant	 Non immunologically mediated inflammatory reaction → direct cytotoxic effect. 	 Similar to ACD but ICD never extend beyond the area of contact. Painful rather than pruritic. Occur from the 1st exposure to the irritant unlike ACD which only occur in previously sensitized individual. 	 PATCH testing (ACD). Treatment: Topical steroid. systemic steroid. Oral antihistamine. 	 Water Abrasives Chemicals, e.g. Solvents and detergents. Napkin dermatitis Lip licking 	

Cutaneous manifestations of SLE and other CTD (dermatomyositis , scleroderma)

Disease	Description	Diagnosis	Management	Notes	Picture
Acute Cutaneous Lupus Erythematosus	 Acute malar "butterfly rash" or more generalized photo-distributed eruption. Non scarring. 	 Hx & examination. Skin Biopsy. Lupus Band Test → non-lesional non-exposed skin. CBC, ESR → high. Urine analysis. ↓ C3, C4 → active disease + renal involvement. Serology: ANA: +ve in 95%. Anti-dsDNA → Specific. (indicates renal disease). Anti-smith → specific. Anti-histone Ab → drug induced lupus. Rheumatoid factor 	 Sun protection. Potent topical steroids Antimalarial drugs. Oral steroids. Methotrexate. 	 ALL patients with ACLE will have SLE. ACLE is transient, improves with improvement of the SLE. Minocycline can cause all the type SLE symptoms except renal involvement or CNS involvement. 	
Subacute Cutaneous Lupus Erythematosus	 Scaly, superficial, inflammatory macules, patches, papules and plaques that are photo-distributed, Morphologic subtypes: Annular/polycyclic lesions. Papulosquamous lesions. Psoriasiform Pitryasiform 	 Clinical diagnosis Skin biopsy. Lupus band test. Anti-Ro/SS-A. Anti-La/SS-B. 	 Sun-protection Topical steroids Antimalarial drugs 	 About 1/2 patients with have SLE. Skin disease, photosensitivity, MSK complaints. SCLE could occur in patients with Sjögren syndrome, deficiency of the C2d, or it may be drug induced (thiazide diuretics). 	CE.

Disease	Description	Diagnosis	Management	Notes	Picture
Chronic Cutaneous Lupus Erythematosu	- Scarring.	- Serologic abnormalities are uncommon.	 Sun-protection Potent topical steroids. Intralesional steroids on the active borders. Antimalarial drugs. 	 Most common type → Discoid lupus erythematosus "DLE". Risk of developing SLE is 5% Chilblain lupus: Painful bluish-red papular or nodular lesions of the skin in acral locations (including the dorsal aspects of <u>fingers</u> and toes, heels, nose, cheeks, ears) 	
Subacute Neonatal Lupus Erythematosu		 Anti-Ro/SS-A. anti-La/SS-B antibodies. 	- Transient, resolving within a few months.	 Infants develop skin disease, heart disease. Disease usually manifests as isolated complete heart block → pacemaker Thrombocytopenia / liver. 	
Lupus Panniculitis	- Inflammation involving the <u>subcutaneous tissue</u> , resulting in inflamed nodules that often resolve with depressed scars .	- Excisional biopsy.	- Antimalarial drugs.	 They could have overlying DLE lesions "<u>Lupus</u> <u>Profundus</u>" About 1/2 patients with have SLE. 	
Drug-Induced Lupus Erythematosus	 Nephritis and central nervous system features are not commonly present. 	 No antibodies to native DNA or hypocomplementemia are present. 	- Drug discontinuation.	Drugs induces LE: - Chlorpromazine - Hydralazine, Minocycline - Methyldopa, Anti-TNF - Penicillamin, Isonizide - Procinamide - Quinidine. - IFN-alpha, IFN-beta.	

Disease	Description	Diagnosis	Management	Notes	Picture
Dermatomyositis	 Idiopathic chronic inflammatory disease involving the skin and skeletal muscles. Pathognomonic findings: Gottron's papules → erythematous to purplish flat papules on the extensor surfaces of the interphalangeal joints. Gottron's sign → symmetric violaceous erythema ± edema, over the dorsal knuckles of the hands, elbows, knees, and medial ankles. Other findings: Heliotrope rash → periorbital edema Shawl sign → ILD Calcinosis. 	 History & examination. Progressive proximal symmetrical weakness. Elevated muscle enzyme levels (creatine phosphokinase). Electromyogram. MRI. Muscle biopsy. Serology: ANA. Anti-Jo-1 (anti-histidyl-t-RNA synthase). Anti-Mi-2 -> good prognosis 	 Oral steroids. Methotrexate. Topical steroids. Antimalarial. Physiotherapy. Surgical excision or Co2 laser. 	 Diseases associated with dermatomyositis: Lupus, rheumatoid arthritis. Scleroderma. Sjogren's syndrome. Malignancies (ovaries cancer). 	

Disea	se	Description	Diagnosis	Management	Notes	Picture
Scieroderma Chronic disease that involves the microvasculatur e and connective tissue \rightarrow fibrosis. \uparrow dermal collagen & \downarrow elastic tissues \rightarrow typical thickening & immobility.	Morphea	 Localized, Sclerotic, indurated plaques that may be solitary, multiple, linear. The surface is usually smooth, with the center of the lesion a <u>whitish or</u> <u>ivory color</u>, whereas the border of active lesions is usually violaceous. 		 Treatment is symptomatic. Raynaud's phenomenon: Stop smoking Keep hands warm. Ca channel blockers (nifedipine) Aspirin. Vasodilating drugs (iloprost) Calcinosis cutis: Nifedipine. Surgical Laser excision. 	- They don't develop SLE.	
	CREST Syndrome	 C = Calcinosis cutis R = Raynaud's phenomenon E = Esophageal dysfun ction S = Sclerodactyly. T = Telangiectasia 	 History & examination characteristic skin changes. Serology: ANA (often +ve) Anti-centromere antibodies (CREST) Anti-Scl-70 		-	
	Progressive Systemic Sclerosis	 Swelling of the hands and feet a ± Raynaud's phenomenon. Telangiectasia. Proximal nail fold changes. Thickening and sclerotic changes involving the face & extremities→ Sclerodactyly, madonna fingers, Loss of normal facial lines Thinning of lips, (microstomia). 	"anti-topoisomerase I" - Skin biopsy.	 Skin sclerosis: Physiotherapy. Phototherapy. GI: PPI. Surgery for strictures. Kidney: ACE inhibitors. In severe cases: Immunosuppressant. D-Penicillamine. 	 Raynaud's Phenomenon Digital ischemia due to exposure to cold and/ or emotional stress. Causes Rheumatic disorders. Diseases with abnormal blood proteins Drugs (β blockers, nicotine, cyclosporine) Arterial disease. 	

Hair Disorders

Disease	Description	Tests	Management	+ info	Picture				
	Non scarring (reversible) alopecia								
Alopecia areata	 Immune mediated (T-cell) Targeting anagen follicle. Incidence 15-29, familial 50%. Patterns: Round or oval patches Loss of all scalp hair (alopecia totalis) Loss of all body hair (alopecia universalis) -check eyebrow- Ophiasis: loss of occipital hair Sisaipho: Sparing temporal & occipital Diffuse pattern: after stress or illness. Nail abnormality (Nail pitting, Koilonychia, Longitudinal ridging). 	 Pull test (+) Trichoscopy: Exclamation marks, tapered hair. 	 Spontaneous resolution. Topical sensitizers like diphencyprone. Weekly application to induce mild eczematous reaction & retroauricular lymphadenopathy. Steroids: Topical, intralesional, oral ± methotrexate. Minoxidil 2-5%. Anthralin. NBUVB: Risk of melanoma. 	 Associated diseases: Atopy (allergic rhinitis, asthma, atopic dermatitis). Autoimmune disease: Thyroiditis & Vitiligo. Down syndrome. Psychiatric diseases. Bad prognostic factors: Earlier age of onset. Positive FHx of alopecia. Extensive involvement. Prolong duration >5 y Ophiasis. 	-				
Androgenetic alopecia (female pattern hair loss)	 Decline in hair production Reduction of the anagen duration 5 alpha reductase (5AR) causing miniaturization Male pattern: Has 2 components: Frontotemporal recession (M shaped), Hair loss over vertex. Hairs become short. Total loss of hair except occipital region. Female patterned hair loss: Sometimes there is history of increased hair shedding. Widening of the parting width. Reduction of hair density over frontal scalp and crown. 	 Pull test: Miniaturized short telogen. Part width test: Wide over crown Trichogram: <u>Telogen is more</u> <u>on the crown</u> than occiput. 	 Male: Minoxidil 2% or 5% → #Scalp irritation, hypertrichosis. Finasteride: Reduce incidence of prostate cancer, In females a risk of abnormal genitalia in male fetus. Surgery (hair transplant). Women: Minoxidil 2% and 5%. Anti-androgens: Cyproterone acetate, combined with OCPs → # ↑ Weight, Fatigue, Mastodynia & ↓ libido. Spironolactone → #Breast soreness, menstrual irregularity, safest. Flutamide: ineffective in post menopause, Hepatotoxic. 	- Female patterned alopecia might be associated with PCOS, acne, hirsutism.	-				

Disease	Description	Tests	Management	+ info	Picture
Telogen effluvum	 Premature conversion of Anagen to Telogen. Types: Acute (< 6 months). Chronic (> 6 months). Chronic repetitive. Hair shedding starts 2 weeks after the trigger and peaks between 6-8w, and tapers in about 6-8 w. Follicular regeneration is common within 4-6 months. 	 Trichogram: anagen: telogen ratio is 70:30, Telogen is equal on different parts of the scalp Empty hair follicles, minimal Miniaturized hairs. Pull test is positive IF patient uses no shampoo/comb. 	 Identify the triggering factor and treat it. Minoxidil topically 2%. Biotin 3-5mg daily. L- cysteine supplement. Iron & vitamin D supplement. 	 Triggers: Stress. Nutritional deficiencies Endocrine disorders (thyroid disease). Crash diet. Surgery. Drugs (Heparin, Retinoid, Propranolol, Captopril). Fever. Childbirth. 	-
Anagen effluvum	- Severe insult to the dermal papilla producing 80% hair loss.	- Pull test: Anagen predominant	- No treatment	 Cytotoxic drugs: Doxorubicin (Adriamycin), Cyclophosphamide Paclitaxel (Taxol) Etoposide. 	-
Trichotillo- mania	different hair		 Behavioral treatment. A psychiatrist should be consulted. SSRI (fluoxetine). 	 Trichobezoars (ingestion of hair) result in intestinal obstruction, MRI and US required for diagnosis. In children prognosis is good In adult patients, the prognosis is poor. 	-
Traction Alopecia	 Traction-inducing hairstyles. Scarring in late stages. Frontotemporal margins of scalp Fringe sign: short terminal hairs bordering areas of marginal hair loss 	- Dermoscopy:peri pilar whitish hair casts.	 Changing hair style 2% minoxidil. 	-	-

Disease	Description	Tests	Management	+ info	Picture				
	Scarring (irreversible) alopecia								
Chronic Discoid Lupus	 Onset: 20 and 40 years. <u>Inflammation is in the center</u> Presents with scaly erythematous plaques, follicular plugs, pigmentation and scarring alopecia 	- Trichoscopy: erythema, scales, pigmentary changes and follicular plugs.	 Corticosteroids topically intralesional injection. Antimalarials. 	- Hair follicles are destroyed	-				
Lichen Planopilaris	 Middle aged women. Itching , pain and burning. 	- Trichoscopy: perifollicular erythema & perifollicular scales at the margin of the lesion.	- Antimalarials - Dapsone - Retinoids.	 & replaced by scar tissue & permanent hair loss. The diagnostic hallmark of all scarring alopecia is visible loss of follicular ostia. Compound follicles are also seen. Sebaceous gland may be central to the 	-				
Dissecting Cellulitis of the Scalp	 Occurs predominantly in black males, in their second to fourth decade of life. Perifollicular pustules, nodules, and abscesses, with interconnecting sinus tracts that drain pus or blood. Thought to result from occlusion of the pilosebaceous unit. Acne conglobata, hidradenitis suppurativa, and pilonidal cysts are frequent concomitant diseases. 	-	 Oral isotretinoin is the treatment of choice. Intralesional corticosteroids (eg, triamcinolone acetonide). Antibiotics such as doxycycline, ciprofloxacin, rifampicin, and dapsone. Biologic agents such as adalimumab and infliximab 	 pathogenesis of scaring alopecia. Sebaceous gland involvement is noted in lichen planopilaris and dissecting cellulitis in follicular occlusion triad. 	-				

Pigmentary diseases

Disease	Description	Diagnosis	Management	Notes
Vitiligo	 Circumscribed depigmented macules and patches secondary to absence of functional melanocytes in vitiligo skin Onset: 10-30 years. Koebner phenomenon. Sites → bony prominences, extensor forearm, ventral wrists, dorsal hands. Leukotrichia → body hair depigmentation. (poor prognosis) Extracutaneous Manifestation: Choroidal abnormalities, Iritis, Uveitis. Vogt-Koyanagi-Harada syndrome → Vitiligo, Uveitis, Aseptic meningitis, Dysacusis, Tinnitus, Poliosis, Alopecia. 	 Woods light shows milky white depigmentation. TSH, ANA, CBC . Serum antithyroglobulin and antithyroid peroxidase antibodies. HbA1c. 	 Narrow-band UV-B phototherapy (311nm) Safe in children, pregnant women, and lactating women. #pruritus and xerosis. Psoralen photochemotherapy: Widespread vitiligo. Excimer laser (308nm): < 30% of the body surface. Biologic therapy (JAK inhibitors): Tofacitinib + light. Topical therapy: Steroid, Tacrolimus (head and neck involvement). Vitamin D analogs. Depigmentation: > 40% of surface area. monobenzylether of hydroquinone cream is applied twice daily. Surgery: Unilateral vitiligo(stable form). 	 Theories: Autoimmune mechanisms → Thyroid disorders (Hashimoto and Graves), Addison disease, DM, Alopecia Areata. Cytotoxic mechanisms. Intrinsic defect of melanocytes (Abnormal rough endoplasmic reticulum). Oxidant-antioxidant mechanisms (Accumulation of free radicals). Neural mechanisms (nerve injuries). Classification: Localized → Focal (one area), Segmental (dermatomal pattern, poliosis), Mucosal. Generalized → Acrofacial, Vulgaris, Mixed: Universal → Associated with multiple endocrinopathy syndrome. DDX: Pityriasis Alba, Post Inflammatory depigmentation, Chemical leukoderma, Vogt-Koyanagi-Harada Syndrome.
Melasma	 Sun-exposed areas. Symmetrical tan to brown hyperpigmented macules. Blue or black → dermal melasma. Patterns: Centrofacial → forehead, cheeks, nose, upper lip, and chin. Malar → nose and the cheeks. Mandibular → ramus of the mandible. 	- Woods light: accentuation of the pigmentation implying epidermal rather than dermal type of pigmentation.	 High-SPF sunscreens (50+) Topical depigmenting agents: Hydroxyquinone (HQ) (Side effects → Phototoxic reactions, Irreversible exogenous ochronosis) Combination treatment HQ + retinoic acid +steroid + Vit.C. Azelaic acid. Oral proanthocyanidin. Tranexamic acid (antifibrinolytic) 	- Risk factors: melanocytic nevi thyroid disease, female, pregnancy, OCP.
Freckles	 Multiple tanned macules1-5 mm with uniform pigmentation. Sun-exposed areas. The macules may be discrete or confluent. 	-	 Sunscreens. Chemical peels, cryotherapy, and laser. 	 Associated with fair skin and red or blonde hair. Association with systemic disease → : Xeroderma pigmentosum, Neurofibromatosis (found in the folded regions)

Cutaneous Manifestations Of Systemic Diseases

	Disease	Features	Sites	Treatment	+ info	Picture
			Enc	docrine		
	Acanthosis Nigricans	- Hyperpigmented velvety plaques.	- Body folds and creases.	Weight reduction.Reducing insulin resistance.	 Indicator of insulin resistance. ↑ conc. of insulin → ↑ affinity binding to IGF-1 → stimulation of keratinocytes and dermal fibroblasts. 	
	Acrochordons [Skin Tags]	- Small, skin colored, pedunculated papules.	- Neck, axilla, eyelids.	-	 Associated with obesity and insulin resistance, acanthosis nigricans. Related to IGF activity. 	Contraction of the second seco
D I A	Diabetic Dermopathy	 Red papules → atrophic, hyperpigmented papules and plaques. 	- Shins.	- Blood glucose control.	 Related to diabetic neuropathic and vascular complications. Most Common cutaneous sign of diabetes. 	
B E T E S	Necrobiosis Lipodica Diabeticorum	 Erythematous papules → evolve into yellowish brown plaques with dilated blood vessels and central epidermal atrophy. Sometimes they ulcerate. 	- Pretibial area	 Topical and intralesional steroids. Tacrolimus. Phototherapy, cyclosporine. 	 Very rare and very characteristic. Histopathology: granulomatous reaction. 	8 1
	Bullae Diabeticorum	- Spontaneous blister- like lesions.	- Hands and feet.	- Heals without scarring.	- Rare but distinct marker of diabetes.	
	Scleredema Diabeticorum	- Woody induration and thickening of the skin.	 Mid upper back, neck, and shoulders. 	-	 Common in males [obese with longstanding uncontrolled DM with complications. Controlling DM DOESN'T affect the course of scleredema. 	

	Disease	Features	Sites	Treatment	+ info	Picture
D	Acquired Perforating Dermatosis	 Transepidermal elimination of collagen and elastic fibers 2-10mm, firm, umbilicated, hyperkeratotic, papules. 	- Trunk. - Extremities.	 Topical keratolytics (Urea). Topical/systemic retinoids. Steroids, Phototherapy Antihistamines, cryotherapy. 	 Associated with chronic renal failure, diabetes mellitus. 	
A B E T E S	Bacterial	- Staphylococcus aureus and beta-hemolytic strept.	-	-	 Corynebacterium minutissimum: → Erythrasma. Pseudomonas aeruginosa → malignant otitis externa. 	
	Fungal Infections	 Candida. Tinea pedis, tinea cruris, tinea corporis, onychomycosis. 	- Warm, moist folds of the skin.	 Rhinocerebral mucormycosis → Depriment + IV Amphotericin 	 Rhinocerebral mucormycosis: Extensive, life threatening. Begins in the nasal passages and spreads into the orbit and cerebrum. 	
C	ushing's Syndrome	 Weight gain, Bruises easily. Purple stretch marks. Acne, Glucose intolerance. 	-	-	 Pituitary gland → ↑ ACTH → adrenals ↑ cortisol. Large doses of steroids. 	
	Addison's Disease	 Hyperpigmentation Oral mucous membrane hyperpigmentation is PATHOGNOMONIC. 	 Skin. Mucous membranes. 	-	 Adrenocortical insufficiency. ACTH binding with MELANOCORTIN 1 → Hyperpigmentation. 	
T Hyperthyroidism		 Palmoplantar hyperhidrosis. Pruritus. Non-scarring alopecia. Plummer nails. Pretibial myxedema. 	-	-		P
r o i d	Hvnothvroidism	 Coarse, rough, dry skin. Pallor, Pruritus. Carotenemia. Diffuse hair loss. Loss of 1/3 of the eyebrow. Myxedematous facies. 	-	-	-	Hair dry, coarse, sparse Lateral eyebrows thin Periorbital edema Puffy dull face with dry skin

Disease	Features	Sites	Treatment	+ info	Picture
		Gastro	ointestinal		
Dermatitis Herpetiformis Clustered Vesicular papules or plaques. - Tense subdermal blisters. - Lesions are cutaneous manifestation of Celiac		- Symmetrically distributed over the extensor surfaces (elbows, knees, buttocks and shoulders).	-Gluten free diet. - Dapsone – 1 st line. -Sulfapyridine- 2 nd line. -Tetracycline (localized). -Nicotinamide (localized).	 Autoimmune blistering disorder. Associated with a Gluten Sensitive Enteropathy. Tissue Transglutaminase Antibodies (tTG). Direct immunofluorescence → granular IgA in dermal papillae. 	12 52
Acrodermatitis Enteropathica	 Scaly erythematous patches and plaques → progress to vesicles, erosions, pustules. Blisters with crustation are periorofacial. When the infant is weaned from breast milk. 	- Acral. - Perioral. - Perianal.	- Life long dietary Zinc supplementation	 Triad of → Periorificial, acral dermatitis, alopecia, and diarrhea. Autosomal recessive → zinc absorption impairment in the jejunum and ileum. Acquired in alcoholics. 	
Pyoderma Gangrenosum	 Painful ulcerative with well-defined undermined violaceous border. Starts as a small red papule or pustule that subsequently burst and expand to form a large non-infectious ulcer. 	-	-	 +ve pathergy test (Behcet's disease) Associated with ulcerative colitis, crohn's disease, RA, and leukemia. Surgery is contraindicated. 	
Peutz-Jeghers Syndrome	 Hyperpigmentation (Brown macules). Intestinal hamartomatous polyp 	 Mucous mem. Lips and buccal mucosa Jejunum. 	-	 Autosomal dominant. ↑ risk of malignancy in younger individuals. Follow up for colon cancer 	
Porphyria Cutanea Tarda	 Photosensitivity. Skin fragility after trauma → erosions and bullae. Facial hypertrichosis. Healing leaves milia, hyperpigmentation and atrophic scars. 	- Sun exposed areas (hands).	- Frequently associated with hepatitis C infection.	- Inherited metabolic disorder where uroporphyrinogen decarboxylase (UROD) is deficient.	

Disease	Features	Sites	Treatment	+ info	Picture
Hemochromatosis	 Cutaneous pigmentation → brownish bronze or slate gray. Ichthyosis, koilonychia, hair loss (pubic area). 	- Sun exposed areas (face).	 Phlebotomy. Chelation Therapy. Surgery Indications → End-stage liver disease/Hepatocellular carcinoma, Severe arthropathy. 	- Abnormal accumulation of iron in several organs leading to organ toxicity.	
		l	Renal		
Uremia	 Xerosis → acquired ichthyosis.• Pruritus → Excoriations, prurigo nodularis, lichen simplex chronicus (scratching). Half-and-half nails → dark reddish brown distal band and a white proximal band. 	ichthyosis.• Pruritus \rightarrow Excoriations, prurigo nodularis, lichen simplex chronicus (scratching). Half-and-half nails \rightarrow dark reddish brown distal band		- Most common <u>metabolic</u> cause of pruritus	
Nephrogenic Systemic Fibrosis	- Large areas of thick, indurated skin with fibrotic nodules and plaques on the	- Extremities. - Trunk.	 Extracorporeal photopheresis - Immunosuppressive Phototherapy. IVIG. Topical steroids. 	 Fibrosis of the skin and internal organs (similar to scleroderma). Caused by Gadolinium exposure → imaging patients who have renal insufficiency. 	
		Нуре	rlipidemia		
Xanthelasma Palpebrarum	Xanthelasma - Asymptomatic, and usually - Inner canthus. - Palpebrarum - Upper eyelid. -		 Surgical excision. CO2 laser ablation. Chemical cauterization (trichloroacetic acid). Electrodesiccation. Cryotherapy. 	 Associated with any type of 1^{ry} hyperlipoproteinemia. 	

Disease	Features	Sites	Treatment	+ info	Picture
Tendinous Xanthomas	-	 Achilles tendon,. hands, feet, elbows, and knees. 	- Least responsive to treatment.	- In patients with familial hypercholesterolemia	
Tuberous Xanthomas	 Firm non-tender, cutaneous and subcutaneous yellowish nodules. 	- Extensor surfaces.	- Lipid lowering agents.	- Associated with familial dyslipoproteinemia.	
Eruptive Xanthomas	 Painless, yellowish papules on an erythematous base. 	 Grouped lesions → trunk, elbows, and buttocks. 	- Resolves in few weeks after therapy.	 Associated with hypertriglyceridemia. Could be seen in poorly controlled DM and acute pancreatitis. 	
Plantar Xanthomas	- Elevated cutaneous yellowish-orange deposits.	- Plantar creases	-	- Associated with dysbetalipoproteinemia.	

Purapura & vasculitis

Class	Disease	Clinical features	Histology	Management	notes	Picture
Cutaneous small vessel (leuko- cytoclastic vasculitis) - Palpable purpura is the hallmark - Do Not blanch under pressure (diascopy)	Henoch- Schonlein	 Characterized by: purpura, arthralgias (can progress to arthritis with swelling around knees and ankles), abdominal pain (GI bleeding acute surgical abdomen, paralytic ileus) & renal disease (GN & renal failure). Pulmonary hemorrhage can be fatal Multiple palpable purpura on the extensor aspects of the extremities: lower legs, forearms & buttocks. 	 Perivascular infiltrate of neutrophils (nuclear dust) Blood vessel wall thickening 	 Supportive: D/C drug, bed rest, treat infection & pain killers Abdominal pain: H2 blockers, corticosteroids NSAIDs are best avoided (renal & GI complications) 	 Triggers: viral infection, streptococcal pharyngitis, bacterial infections, foods, drugs (aspirin, penicillin), lymphoma Affects children IgA-mediated 	
	Urticarial lesions "urticarial vasculitis"	 Urticarial vasculitis VS. urticaria: Painful, rather than pruritic Last > 24h & fixed postinflammatory hyperpigmentation Normal complement levels: idiopathic limited to skin Self-resolving Low complement levels: leukocytoclastic vasculitis + diffuse interstitial neutrophils Not limited to the skin: arthritis, arthralgia, angioedema eye symptoms, asthma, GI symptoms 	 Erythrocyte extravasation Fibrin deposits within the blood vessel wall Endothelial necrosis (more serious illness) immunoglobulin & complement deposits 	 Ix: CH50, C3, C4, C1q, ANA, dsDNA, Anti-SSA & Anti-SSB, hepatitis, B&C, lupus band test Cutaneous involvement: NSAIDs & antihistamines, if these fail > Colchicine, hydroxychloroquine, dapsone Systemic disease: corticosteroids + steroid sparing agent (azathioprine, mycophenolate mofetil, rituximab) 	 Complement levels (CH50, C3, C4, and anti-C1q) Diseases associated with urticarial vasculitis: gammopathies (IgG & IgM), SLE, Sjögren syndrome, serum sickness, viral infections (HCV) 	
	Other	 Idiopathic Infection- streptococcal, bacterial en Drugs- NSAID,sulfonamides, penicilli Malignancy- leukemias, lymphoma, r 	ins, barbiturates, propylt	thiouracil		

Class	Disease	Clinical features	Histology	Management	notes	Picture
Small- medium vessels	Churg Strauss syndrome Microscopic polyangiitis Granulomatosis with polyangiitis (GAD) Cryoglobulinemic vasculitis		-	-	-	-
Medium vessel	Polyarteritis Nodosa	 Necrotizing vasculitis Cutaneous findings: subcutaneous nodules associated with livedo reticularis that may ulcerate on the legs & feet Peripheral neuropathy: tingling, numbness, sensory disturbances, weakness and absent reflexes 	 Nodular arteritis Polymorphonuclear infiltration involving medium sized arteries of the deep reticular dermis & subcutaneous tissue + extensive fibrinoid necrosis 	- Most patients respond well to: aspirin, NSAIDs, prednisone, sulfapyridine, or methotrexate	 Has been associated with HBV & HCV infection, Crohn's disease, streptococcal infections, TB, and medications (minocycline) The only laboratory abnormality is ESR 	
Large vessel	Giant cell arteritis Takayasu arteritis	-	-	-	-	-
-	Vitamin C deficiency (scurvy)	 Perifollicular petechiae Keratotic plugging of hair follicles Hemorrhagic gingivitis 	-	-	-	

Dermatological Emergencies

Disease	Description/Features	Management	+ info	Picture
Urticaria	 Wheals or hives. Evanescent. Disappear within 24 hours. Blanch with pressure, Intense pruritus. 	 Antihistamin. Systemic steroids (Severe). 	 Release of mediators from mast cells → ↑ in vascular permeability. Immunologic Type I (IgE mediated) or Type III. Major mediator → IgE, major effector → Mast Cell. Life threatening especially when associated with angioedema of the larynx. Serum sickness → type III immune complex mediated reaction (Fever, Urticaria, Angioedema, Joint pain and swelling, lymphadenopathy). 	
Angioedema	- Edema Involving the deep dermis or subcutaneous and submucosal areas.	 Non Sedating H1 antagonists. (loratadine, claritin). Sedating H1 antagonists (itching). Corticosteroids. Epinephrine (lifesaving). Hereditary Angioedema (doesn't respond to steroids) → antiandrogens (danazol), oral tranexamic acid 	 Airway involvement → EMERGENCY. Causes → Antibiotics, ACEI, Foods Hereditary Angioedema → autosomal dominant caused by C1 esterase inhibitor deficiency (C1 usually presents in the form of angioedema and gastric involvement without urticaria) 	
Anaphylaxis	 Laryngeal edema, bronchospasm. Erythema, pruritus, urticaria or angioedema. Vomiting, cramps, diarrhea. Hypotension, cardiac arrhythmia or shocks. 	- ABCs. - Epinephrine. - Antihistamine.	 Within minutes to hours, severe may be fatal. IgE mediated. Causative agents: Drugs, Foods, Vaccines and Antisera. 	
Erythroderma & Exfoliative Dermatitis	 Erythema with or without scales Affecting >90% of the body. It's common in extreme of ages 	-	 Complication → Hypothermia , fluid and electrolyte loss, infection. Drug Etiology: Sulphonamides, antimalarials, penicillin, phenytoin. Other causes → psoriasis, cutaneous T-cell lymphoma. 	

Disease	Description/Features	Management	+ info	Picture		
Erythema multiforme	 Target lesions little or no mucosal involvement (EM minor), Fever , arthralgia ,sore throat. 	- Acyclovir.	- Follow an infection by HSV.			
Steven Johnson Syndrome	 Mucous membrane erosions. < 10% BSA of epidermal detachment. Subcorneal blister 	 Corticosteroids (early in the disease for a short period of 3-5 days). 	 Severe form of EM (5% mortality). Causes: Mycoplasma pneumonia infection, drugs (50%). Histopath → splitting at the level of the subcorneal epidermal layer. 			
Toxic Epidermal Necrolysis	 Severe mucosal involvement in conjunctiva and the eye < 30% BSA of epidermal detachment. 	 Withdrawal of suspected drug. Patient cared in a burn unit or I.C.U. Replacement of I.V. fluids and electrolytes. 	 Severe form of SJS (30% mortality), major cause of death: Septicemia, electrolyte imbalance. Causes: Drugs (95%) → anticonvulsants, sulphonamides, allopurinol, NSAID, carbamazepine. Risk factors: Slow acetylators, Immunosuppression (HIV, Lymphoma) → acquired glutathione deficiency, HLAB1502. Histopath → all the epidermis is removed DDX: Pemphigus vulgaris. 			
Kawasaki Syndrome	 Fever > 5 days, Conjunctivitis. Red fissured lips, strawberry tongue. Rash - erythema exanthem Edema of palms and soles. Cervical lymphadenopathy. Coronary artery aneurysms. 	<mark>- IV aspirin.</mark> - Immunoglobulin,	 Affects children. Complications → CAD and heart problem. 			
Drug Eruptions (most sensitizing root is topical)						
Maculopapular	 Exanthematous. Morbilliform No pustule, vesicle , blister. 	-	- Seen in children on antibiotic(cefaclor).	Ky		

Disease	Description/Features	Management	+ info	Picture
Hypersensitivity Syndrome Reaction	 Triad of fever, skin eruption and internal organ involvement. 	-	 Life threatening syndrome. Anticonvulsants, sulfonamide, dapsone, allopurinol, Azathioprine, Minocycline. 	
Latex Allergy	- Contact Urticaria, Fatal Anaphylaxis	-	 Type 1 reaction to natural rubber latex. Foods that cross react with latex proteins → Banana, Kiwi, Avocado, Chestnuts. 	
Fixed Drug Eruptions	 Dark brown , grayish , round well demarcated. Pigmented or inflammatory in the form of blistering. 	-	 Drug reaction that happens immediately. In males in the form of a blister in the glans penis. Young males on minocycline for acne. 	
Lichenoid Eruptions	 Resembles lichen planus. Violaceous , scaly , papules , diffuse , mucosal. 	-	- Psoriasiform \rightarrow with lithium , BB, anti- malarial	
Photosensitivity	-	-	 Photoxic → non-immune mediated more of an irritation e.g. psoralen , doxycycline for acne. Photoallergic. 	
Drug induced Pemphigus	- The more superficial the more crustaceans.	-	 Superficial pemphigus (foliaceus). Classical drug causing it is penicillamine and ACEI. Separation (intraepidermal) above the basement membrane . In bullous pemphigoid → Tense blister because the split under the epidermis (subepidermal.) 	
Lupus Erythematosus- like syndrome	-	-	 Causes → Hydralazine, procainamide, sulfonamide, Minocycline, INH. Antihistone Antibodies. 	ant-histone antibodies
Erythema Nodosum	-	-	 Due to inflammation of subcutaneous fat (panniculitis). Causes: TB, penicillin, tetracycline, OCP. 	

Disease	Description/Features	Management	+ info	Picture			
Blistering Diseases							
Pemphigus	 Flaccid blisters intraepidermal. Weeping painful erosions. Sloughing of the intact skin next to the blister. Positive Nikolosky's sign. Positive asboe – Hansen sign or (bulla spread sign). Acantholysis. Intra-epidermal cleft. 	 Systemic corticosteroids Immunosuppressive therapy (Azathioprine, Mycophenolate, Mofetil). IVIG → resistant cases. 	 Vulgaris: IgG autoantibodies attack desmosomplyin → detachment of keratinocytes → floating of cells. IgG and C3 in the intercellular space of epidermis. Age - sixth decade of life. Classification: Vulgaris → skin and mucous membrane involvement. Vegetans, Foliaceus. Fogoselvagem → black fly insect pits. Paraneoplastic → chronic lymphocytic leukaemia. 				
Pemphigoid	 Bullous Pemphigoid: Large bullae on erythematous plaques or normal appearing skin. No or only mild involvement of mucous membrane. Sub-epidermal tense blister. So it's tense (lower abdomen , groin, flexors of Arms & legs). Negative Nikolsky's sign 	 Systemic: Steroid. Antibiotics: Tetracycline, Minocin. Dapsone. Immunosuppressive therapy (Azathioprine, Mycophenolate, Mofetil). 	 Autoimmune subepidermal blistering disorders with circulating IgG and basement membrane zone (BMZ)-bound IgG antibodies (ABS) and C3. Diagnosis: CBC: Eosinophilia, IgE:, HIST, DIF 				
Pemphigoid gestationis:	 Erythematous urticarial Plaques, alone or with papules, vesicles, blisters, erosions (abdomen, proximal extremities). Intense pruritus. Subepidermal blister 	 Potent steroids (avoid the systemic in the 1st tri). Topical drugs, drying agents and antiseptic 	 Resemble herpes because its vesicular. C3, IgG at BMZ, H.G. Factor in Patient's Sera. Starts at the 2nd, 3rd trimester. Flares at postpartum, OCP . Purely anti-HLA antibodies from the paternal placenta. 				