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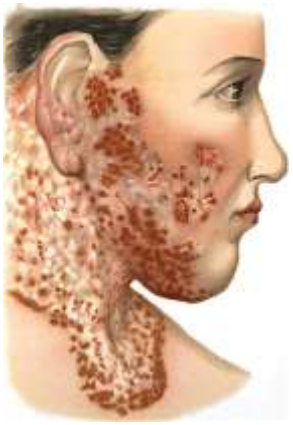
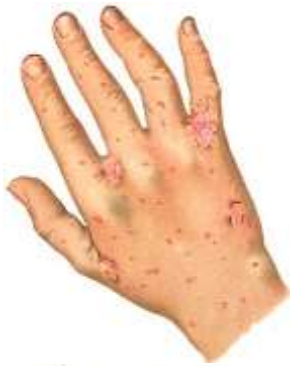
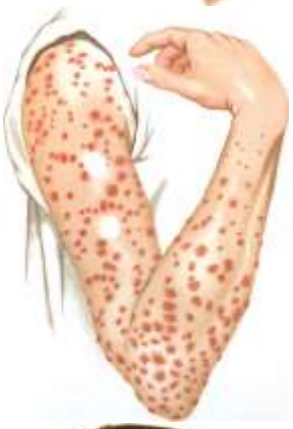
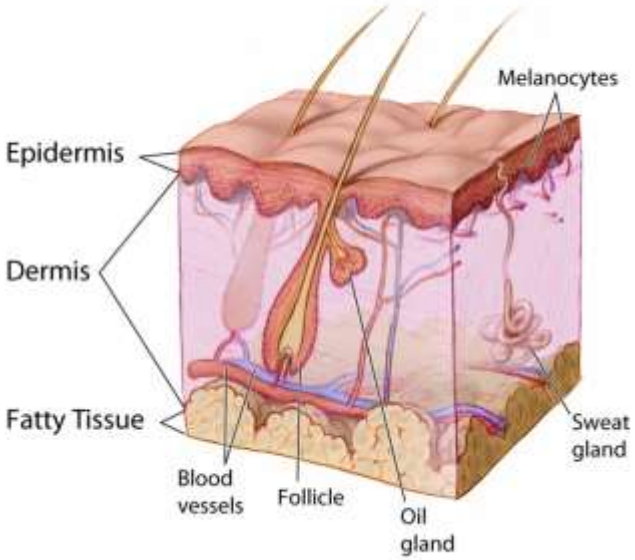
King Saud University







432 Teams
Dermatology

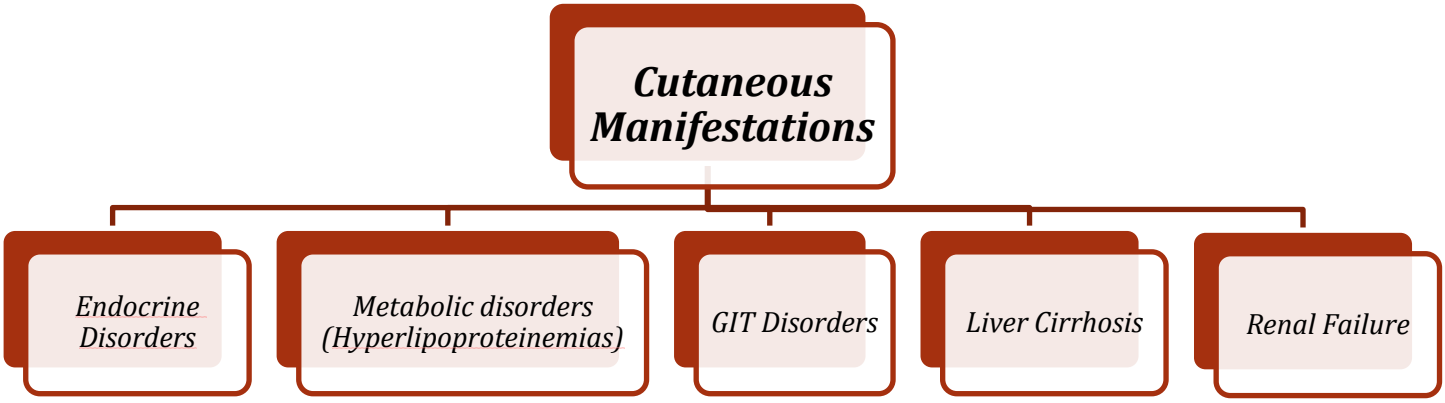


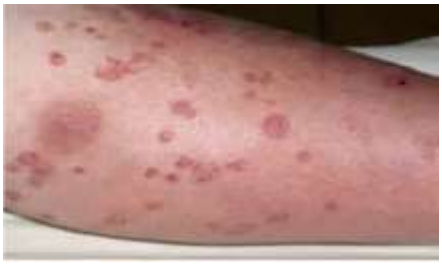
CUTANEOUS MANIFESTATIONS OF SYSTEMIC DISEASES

Color Code: Original, Team's note, Important, Doctor's note, Not important, Old teamwork

Done by: ***Rawan AlQuaiz and Reem AlOtaibi***
Reviewer: ***Lama AlTawil***
Team Leader: ***Basil Al Suwaine&Lama Al Tawil***

Mind Map


Cutaneous Manifestations
Endocrine Disorders
Metabolic disorders (Hyperlipoproteinemias)
GIT Disorders
Liver Cirrhosis
Renal Failure



Endocrine disorders

1) Diabetes Mellitus:

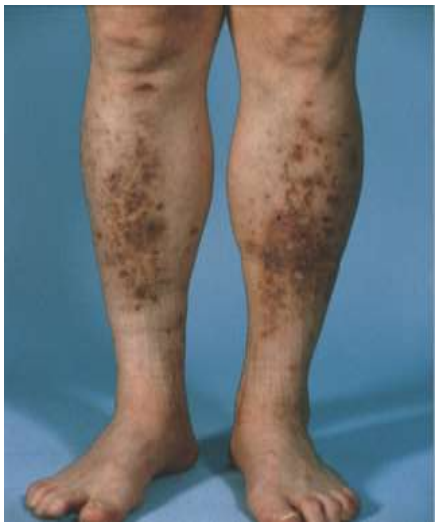


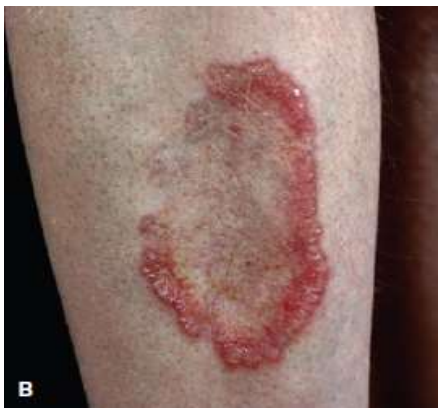
Image: bilateral asymptomatic red-brown atrophic macules and patches over the shins (mainly) and could appear on other sites of the body.

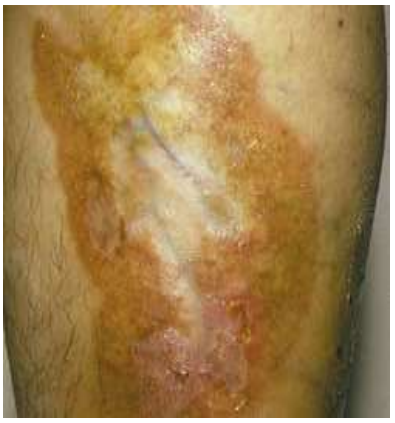
A) Diabetic Dermopathy "Shin Spots"

- Most common cutaneous manifestation of diabetes.
- Affects males more than females. Usually, males over the age of 50 with long standing diabetes. Uncontrolled
- Possibly related to diabetic neuropathy and vasculopathy.
- There is no effective treatment. Controlling diabetes itself will not improve them.

B) Necrobiosis Lipoidica Diabeticorum (NLD)

- The dermis layer is composed of elastin and collagen.
- Necrobiosis: since collagen is considered fibrous tissue and not living tissue, the death of collagen is called "necrobiosis". Unlike in living tissue where it is called necrosis.
- Lipoidica: lipid deposition in the skin → causes yellow discoloration.
- Diabeticorum: specific in diabetic patients.
- Patients classically present with single or multiple red-brown papules, which progress to sharply demarcated yellow(lipid deposition) -brown atrophic, telangiectatic (central) plaques with a violaceous, irregular border.
- Common sites include shins (most common site for diabetic skin disorders) followed by ankles, calves, thighs and feet.
- Ulceration occurs in about 35% of cases. Cutaneous anesthesia, hypohidrosis (decreased sweating) and partial alopecia can be found.
- Pathology: palisading granulomas containing degenerating collagen (necrobiosis) with lipid deposition. Pathogenesis is thought to involve the **non-enzymatic** glycosylation of dermal collagen and elastin.
- Approximately 60% of NLD patients have diabetes and 20% have glucose intolerance. Conversely, up to 3% of diabetics have NLD.
- Women are more affected than men.



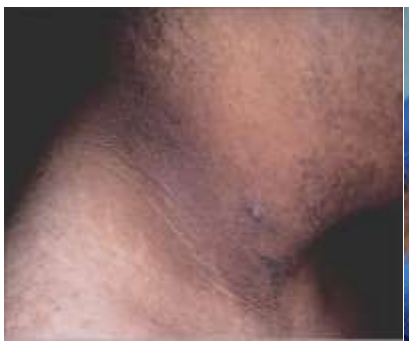


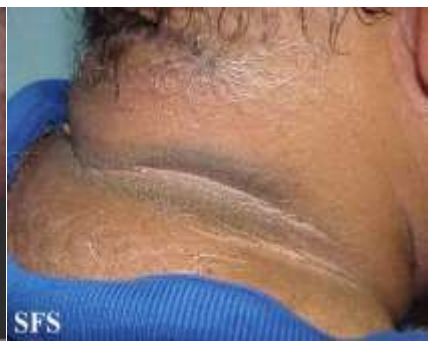
- Treatment: Ulcer prevention. No impact of tight glucose control on likelihood of developing NLD.
  - To reduce inflammation: in the initial stages of the disease, there are red papules and the skin is inflamed → Intralesional steroids.
  - To increase the blood supply to the area for better healing:
    - Aspirin
    - Antiplatelet
    - Pentoxyfylline
    - Preilesional heparin injection

Image: atrophic brownish plaques with violaceous borders (purple borders that are not clear in long standing disease) with central telangiectasia and atrophic skin, which is susceptible to ulceration after mild trauma.

C) Acanthosis nigricans

- Causes:
  - Obesity & insulin resistance & endocrinopathy (DM, acromegaly, cushing syndrome, hypothyroidisim & hyperandrogenic state as HAIRAN syndrome (hyperandrogen, insulin resistance, acanthosis nigricans , PCO
  - Malignancy (esp. GIT, Lung & Breast CA). Patients presenting with **extensive acanthosis** nigricans involving the eyelids, umbilicus, palms and soles → always think of malignancy as a cause and patients need to be screened.
  - Medications (nicotinic acid, niacinamide, testosterone, OCP & Glucocorticoid).
- Pathogenesis:
  - Genetic sensitivity of the skin to hyperinsulinemia.
  - Aberrant keratinocyte and fibroblast proliferation stimulated by excess growth factor (e.g., Insulin like growth factor). The hyperpigmentation in acanthosis nigricans is due to **thickened skin** and NOT increased melanin; bleaching will not treat it. Instead, it's treated using a keratolytic agent (eg. salicylic acid).
- Clinical picture: hyperpigmented velvety plaques (raised, thick and black skin) of the flexures (axilla, inguinal and inframammary folds). The face, external genitalia, medial thighs, dorsal joints, lips and umbilicus can be involved in extensive cases.
- Treatment:
  - Use a keratolytic to remove excess skin.
  - Treat the underlying cause:
    - ✓ Tight blood glucose control.
    - ✓ Treatment of underlying malignancy.
    - ✓ Weight control.
    - ✓ Discontinuation of offending agent.





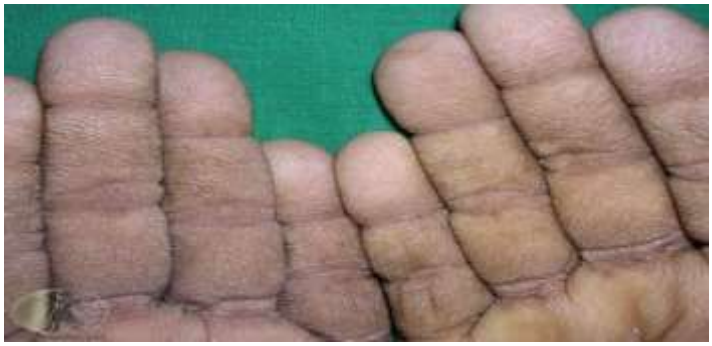
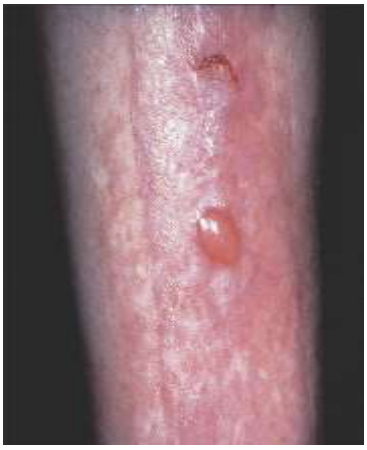
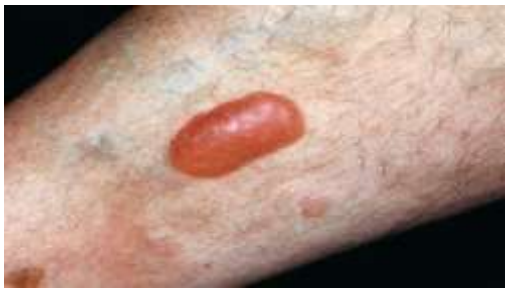


Image: acanthosis nigricans of the palms (tripe palm). It's called a tripe palm because it resembles "tripe", which is the rubbery inner lining of the stomach of farm animals.this indicates an extensive disease i.e. rollout malignancy

D) Diabetic Bullae or Bullae Diabeticorum

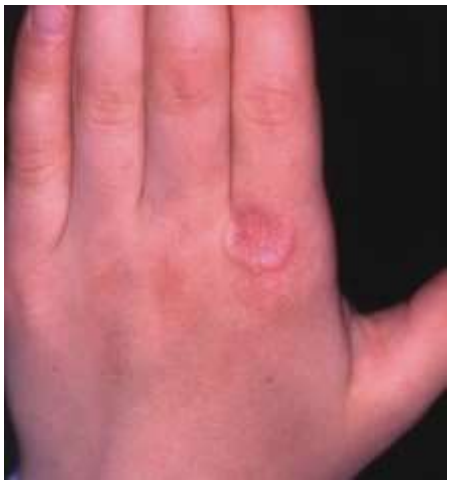


- Bullae = fluid filled blister or sac of > 0.5 cm in diameter. if less, it's called a vesicle.
- Bullae can arise from a separation in the epidermis, basement membrane, or subepidermal layer.
- **Rarest** cutaneous complications of diabetes.
- M > F.
- Long standing diabetes.
- Cause: trauma and microangiopathy may play a role.
- Clinical picture: **overnight, rapid onset** of one or few painless tense blisters on the hands and feet.



- Pathology: In diabetic bullae, the level of separation is either intraepidermal and/or subepidermal layer without acantholysis (acantholysis is usually seen in autoimmune bullous diseases) and negative immunofluorescence (because it's not an autoimmune disease).
- Treatment: Spontaneous healing without scarring.

E) Granuloma Annulare



- Not specific for diabetics and can occur as an idiopathic disease.
- Annulare: from annular, meaning ring like.
- Association between granuloma annulare and diabetes is controversial.
- **Generalized** form of GA is the most closely associated with DM. (if the patient has multiple lesions or generalized GA → you have to rule out diabetes).
- Cinical picture: asymptomatic red-purple dome shaped papules arranged in annular configuration. It has a chronic and relapsing course.

- Treatment :
  - Intralesional (IL) steroids.
  - Systemic steroids.
  - PUVA: stands for psoralen combined with ultraviolet A (UVA) phototherapy

F) Scleredema Diabeticorum

- Occurs in diabetics with poorly controlled, long-standing disease, and **obese men.**
- Clinical picture: painless, symmetric woody "peau d'orange" induration of the upper back and neck. With time, it might limit the movement of the shoulder joint and neck.
- Treatmeant:
  - No specific treatment is available.
  - Control of hyperglycemia does not improve the scleredema.

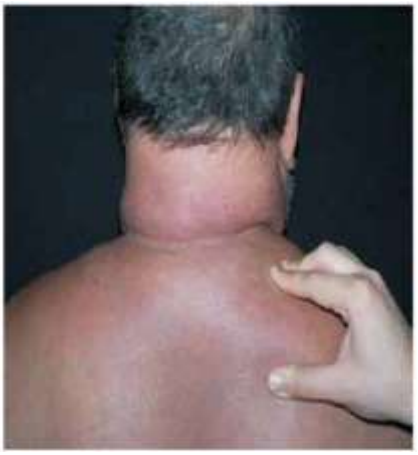


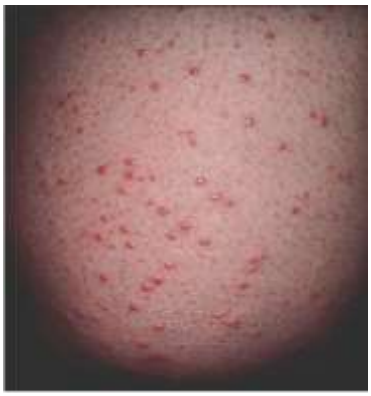
Image: sclerosis of the skin: localized area where the skin is tight.

G) Cutaneous Infections

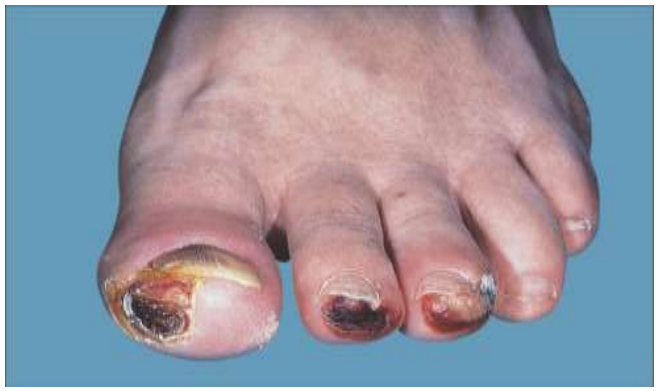
- Diabetic patients are predisposed to develop cutaneous infections due to poor microcirculation.
- Bacterial infections: staph infections.
- Fungal infections: candida infections, dermatophyte infections.

H) Other manifestations of DM:

- Skin tags , perforating dermatosis , Eruptive Xanthomas, and Diabetic foot



Eruptive Xanthomas: multiple xanthomas erupting in a short duration.



Diabetic foot: diabetic neuropathy (peripheral) (anesthesia), Neuropathic and ischemic ulcers (painful).

2) Thyroid diseases:

A) Hyperthyroidism: non-specific manifestations

Table 53.5 Dermatologic manifestations of hyperthyroidism.

DERMATOLOGIC MANIFESTATIONS OF HYPERTHYROIDISM	
Cutaneous changes Hyperpigmentation → due to association of other endocrinopathies such as addison's disease.	Fine, velvety, or smooth skin + **flushing of the face, head and neck** Warm, moist skin due to increased sweating Hyperpigmentation – localized or generalized
Cutaneous diseases	Vitiligo **(Vitiligo and urticaria are due to autoimmune association)** Urticaria, dermatographism Pretibial myxedema and thyroid acropachy
Hair changes	Fine, thin Mild, diffuse alopecia **(Reversible) → telogen effluvium**
Hair disease	Alopecia areata **(Autoimmune association)**
Nail changes **Plummer nails: koilonychia + distal onycholysis.**	Onycholysis **(Separation of the nail from the nail plate)** Koilonychia **(Spoon shaped nails)** Clubbing from thyroid acropachy **Thyroid acropachy: clubbing + hypertrophy of distal phalanx.** Drumstick like **Faster rate of growth**

- Rule out hyperthyroidism before giving botox injections for patients with sweaty hands.
- Patients with hair loss → order thyroid function test (TFT) because hair loss could be associated with hypo and hyperthyroidism.
- Thyroid dermopathy (pretibial myxedema):
  - Occurs in **Graves' disease.**
  - Clinical picture: bilaterally symmetric, non-pitting yellowish-brown to red waxy papules, nodules and plaques on the shins.
  - The clinical findings are due to an increase in hyaluronic acid in dermis (the dermis is composed of collagen, elastin and ground substance "hyaluronic acid"). This substance is used in cosmetic dermatology
  - Treatment regimens include treating the underlying cause and **high potency** topical steroids & intralesional steroid.

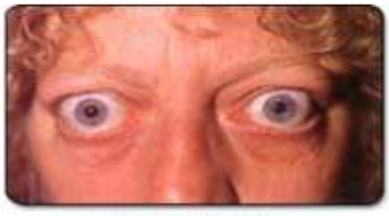


Image: exophthalmos

In Graves'

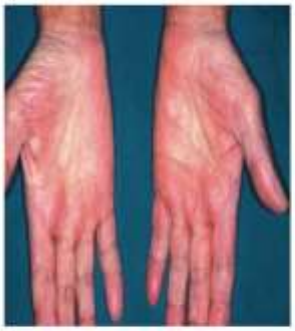


Image: palmar erythema due to hyperthyroidism.

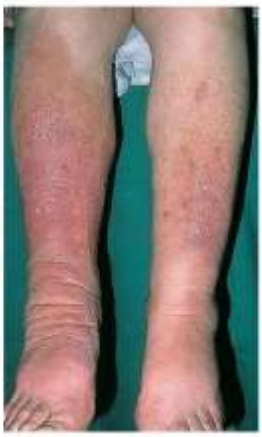
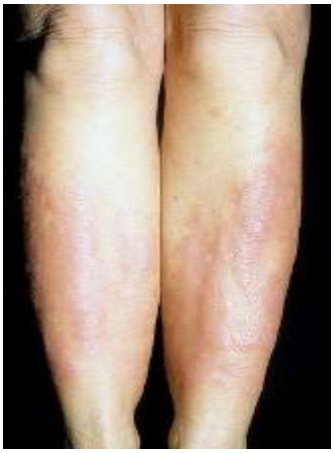
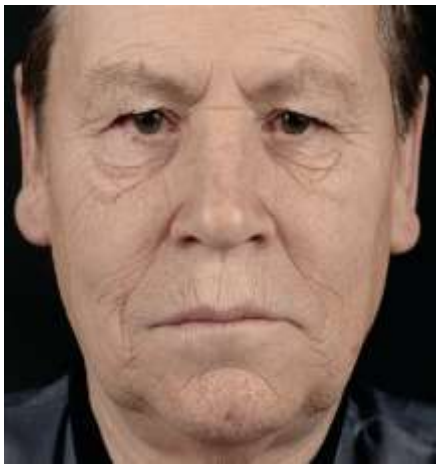


Image: pretibial myxedema



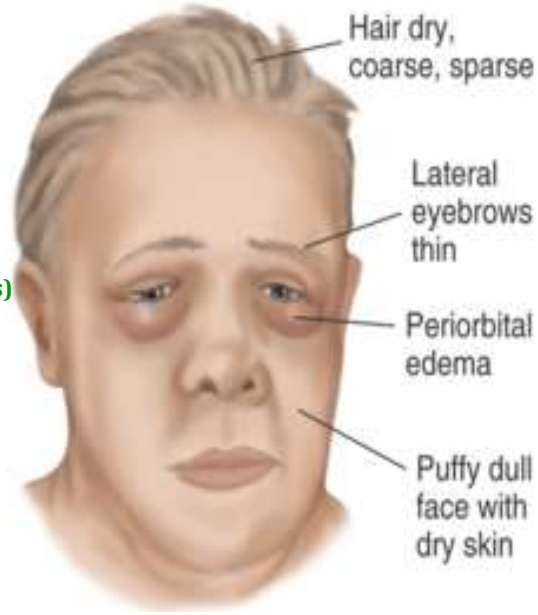
B) Hypothyroidism: non-specific manifestations



- Skin changes:
  - Cool, dry, pale.
  - Xerosis (dryness).
  - Hypohidrosis (decreased sweating).
  - Yellowish hue secondary to **carotenemia.**
  - **Generalized** myxedema: swollen waxy appearance.
  - Swollen lips, broad nose, macroglossia.
  - Purpura secondary to impaired wound healing.
  - Urticaria especially with hashimoto's thyroiditis (autoimmune association).
- Hair changes:
  - Dry, brittle, coarse hair.
  - Diffuse alopecia, Telogen effluvium.
  - Loss of lateral third of eyebrow **(madarosis).**

Table 53.6 Dermatologic manifestations of hypothyroidism.

DERMATOLOGIC MANIFESTATIONS OF HYPOTHYROIDISM	
Skin changes	Dry, rough, or coarse; cold and pale, boggy and edematous (myxedema) Yellow discoloration as a result of carotenemia Easy bruising (capillary fragility)
Cutaneous diseases	Ichthyosis and palmoplantar keratoderma **(Due to skin dryness)** Eruptive and/or tuberous xanthomas (Like in DM)
Hair changes	Dull, coarse, and brittle Slow growth (increase in telogen hair phase) Alopecia of the lateral third of the eyebrows
Nail changes	Thin, brittle, striated Slow growth Onycholysis (rare)



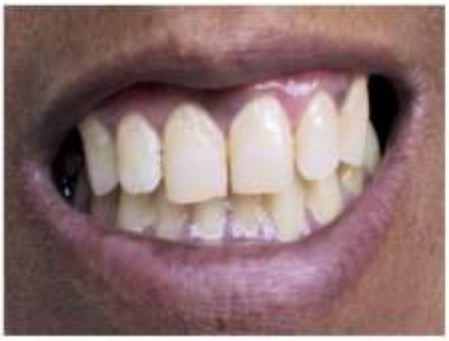
- **Ichthyosis: fish like scales on the skin.**
- **Palmoplantar keratoderma: increased thickness of the skin of the palms and soles.**

Generalized ichthyosis is seen in an inherited case called Harlequin ichthyosis

3) Addison's disease (hypocorticism):

- **Generalized hyperpigmentation** that is more prominent in light exposed areas, scars, genitalia, palmar and finger creases, and under the nails. The pigmentation **characteristically affects the mucous membranes.**
- Loss of pubic and axillary hair in females. (due to decreased androgens)
- Improvement of acne. (due to decreased androgens)
- Causes of hypoadrenalism:
  - Primary (idiopathic)
  - Secondary (pituitary causes)
  - Diseases in the adrenals (TB, malignancy)
- Hyperpigmentation is due to the MSH- like (melanocyte stimulating hormone) effect of ACTH on the skin. In addison's disease → ACTH is increased causing hyperpigmentation.

Addison's disease:



- Note the generalised skin pigmentation (in a Caucasion patient) but especially the deposition in the palmer skin creases, nails and gums.

- She was treated many years ago for pulmonary TB. What are the other causes of this condition? www.sitamol.net

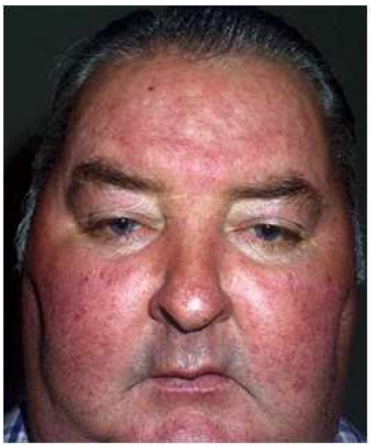
Table 53.8 Selected dermatologic manifestations of Addison's disease.
MSH, melanocyte-stimulating hormone.

SELECTED DERMATOLOGIC MANIFESTATIONS OF ADDISON'S DISEASE

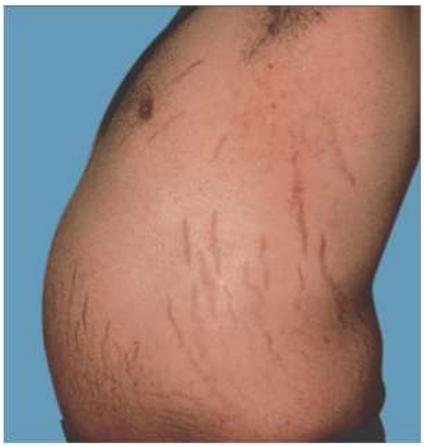
• Hyperpigmentation (MSH-like effect due to secretion of ACTH)
  • Diffuse with sun-exposed accentuation
  • Sites of trauma
  • Axillary, perineum, and nipples
  • Palmar creases
  • Nevi
  • Mucous membranes
  • Hair
  • Nails
• Loss of ambisexual hair in postpubertal women
• Fibrosis and calcification of cartilage including the ear (rare)
• Vitiligo
• Chronic mucocutaneous candidiasis **Mainly in people with polyendocrinopathy (hypothyroidism, hypoadrenalism and candidiasis)**

4) Cushing syndrome:

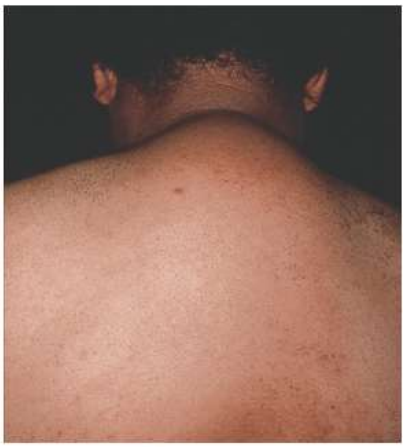
- Causes: endogenous or exogenous.
- Deposition of fat over the clavicles and back of the neck "Buffalo hump".
- Rounded erythematous face with telangiectasia "Moon face". The face is plethoric (red) due to thinning of the skin and since steroids are anabolic hormones it will lead to increased vascularity of the face and rosacea
- Note: Generally steroids can cause acne (non-comedonal and monomorphic acne) and plethora rosacea on the cheeks
- Truncal obesity with slender wasting limbs.
- Thin atrophied skin.
- Striae distensae. Steroid induced striae are violaceous (red-purple in color) and are called striae rubra.
- Hirsutism, acneform rash, androgenetic alopecia.
- Easy bruising of the skin on simple trauma.



Moon face



Striae distensae



Buffalo hump

Table 53.7 Dermatologic manifestations of Cushing's disease (syndrome).
*This same change is indicative of insulin resistance and occurs in HIV-associated lipodystrophy.

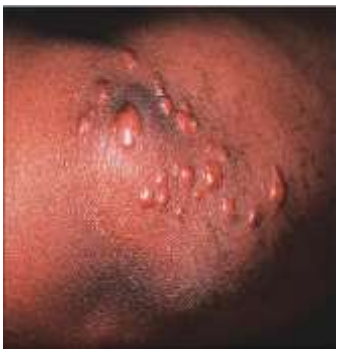
DERMATOLOGIC MANIFESTATIONS OF CUSHING'S DISEASE (SYNDROME)
Altered subcutaneous fat distribution*
• Rounded facies • Fullness of the cheeks ('moon' facies) • Dorsal cervical vertebral fat deposits (buffalo hump) (Fig. 53.20) • Pelvic girdle fat deposition • Reduced fat in the arms and legs
Skin atrophy
• Global atrophy with epidermal and dermal components affected • Multiple striae on abdominal flanks, arms, and thighs (Fig. 53.21) • Cutaneous fragility and prolonged wound healing • Purpura with minor trauma due to reduced connective tissue support
Cutaneous infections
• Pityriasis (tinea) versicolor • Dermatophytosis and onychomycosis • Candidiasis
Appendageal effects
• Corticosteroid-related acne **and steroid induced rosacea** • Hirsutism

5) Metabolic disorders (hyperlipoproteinemia):

- **Type I**
  - Familial lipoprotein lipase deficiency (AR) or apoprotein CII deficiency
  - Increased chylomicrons
  - Associated with hepatomegaly, pancreatitis
- **Type IIa**
  - Familial hypercholesterolemia, common hypercholesterolemia (AD)
  - Increased LDL
- **Type IIb**
  - Familial hypercholesterolemia (AD)
  - Increased LDL and VLDL
- **Type III**
  - Familial Dysbetalipoproteinemia (AR)
  - Increased IDL
- **Type IV**
  - Familial hypertriglyceridemia (AD)
  - Increased VLDL
- **Type V**
  - Familial type V hyperlipoproteinemia, familial lipoprotein lipase deficiency (AD)
  - Increased chylomicrons and VLDL

- Hyperlipoproteinemias are associated with **xanthoma and xanthelasma**.
- There are 6 clinical types of xanthomatosis:

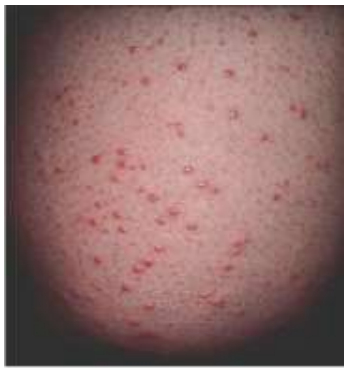
1) Tuberous Xanthoma
   - Flat or elevated, rounded, grouped, yellowish-orange nodules or papules over joints (particularly elbows and knees).
   - Types II, III, and IV. (you dont have to remember each type, just remember that xanthomas are associated with hyperlipoproteinemia and you should order a lipid profile)
   - Might be associated with **biliary cirrhosis** (order liver function tests).



Tuberous xanthoma

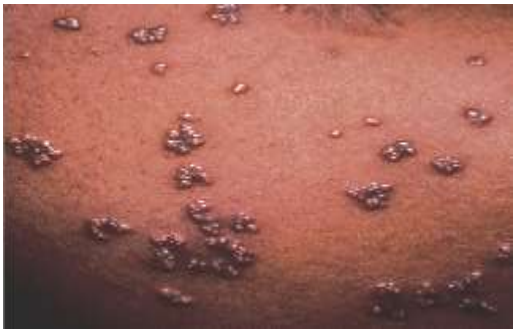
2) Tendinous Xanthoma
   - Papules or nodules over tendons (extensor tendons on dorsum of hands, feet, and achilles tendon).
   - Types II, III.

3) Eruptive Xanthoma
   - Small yellow hue /orange/red papules appearing in crops over entire body (over a short period of time) → buttocks, flexor surfaces, arms, thighs, knees, oral mucosa and may koebnerize.
   - Associated with markedly elevated or abrupt increase in triglycerides (elevated chylomicrons).
   - Types l ,lll , lV , and V.



Eruptive xanthoma

- Associated with diabetes, obesity, pancreatitis, chronic renal failure, hypothyroidism, oral estrogen therapy, oral corticosteroids, isotretinoin (acne treatment), acitretin (acne treatment , so before treating the patient make sure you run a liver function test and check for hyper-TG).

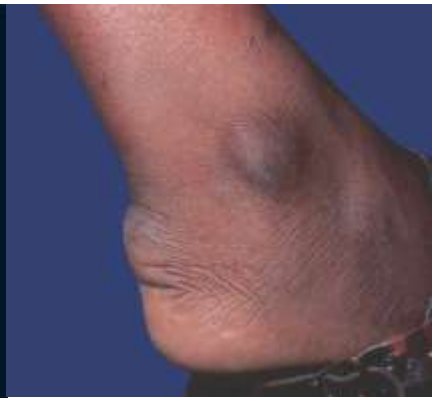


Eruptive xanthoma note the yellowish hue

4) Planar Xanthoma
   - Flat macules or slightly elevated plaques, yellow/tan color.
   - Associated with biliary cirrhosis, biliary atresia (order LFT), myeloma, monoclonal gammopathy, lymphoma (order CBC with differential).
   - Characteristically around eyelids, neck, trunk, shoulders, or axillae.
   - Types ll, lll. (Order lipid profile)

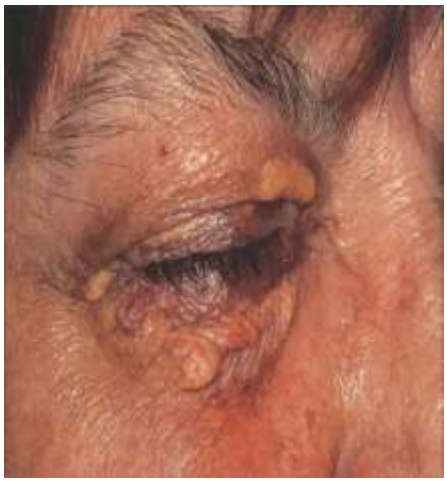
5) Palmar Xanthoma
   - Nodules and irregular plaques on palms and flexural surfaces of fingers.
   - Type III.

Tendinous xanthomas of the fingers in a patient with homozygous familial hypercholesterolemia.

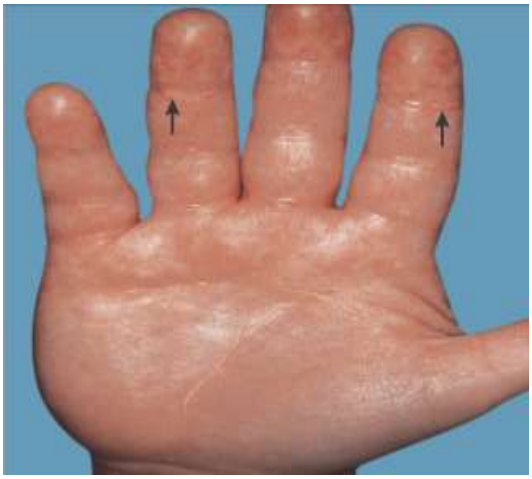


Tendinous xanthoma. Linear swelling of the Achilles area representing a tendinous xanthoma in a patient with dysbetalipoproteinemia.

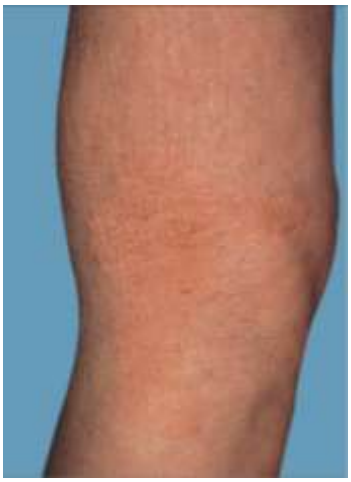
6) Xanthelasma
   - **Most common type of xanthoma.**
   - Eyelids.
   - Usually present without any other disease (idiopathic), but can occur in types II and III.
   - Common among women with hepatic or biliary disorders, also seen in myxedema, diabetes.
   - All types of xanthomas are best treated with surgical excision.



Xanthelasma



Plane xanthomas of the palmar creases in a patient with dysbetalipoprotenemia (arrows).



Plane xanthoma in a patient with a monoclonal IgG gammopathy

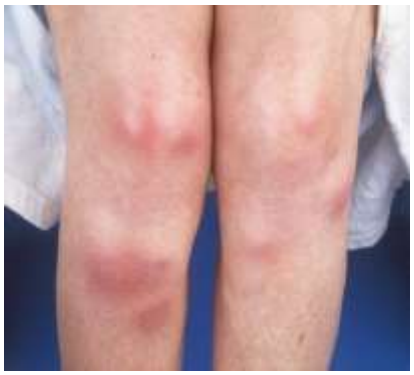
6)CUTANEOUS MANIFESTATIONS OF GASTROINTESTINAL DISORDERS:

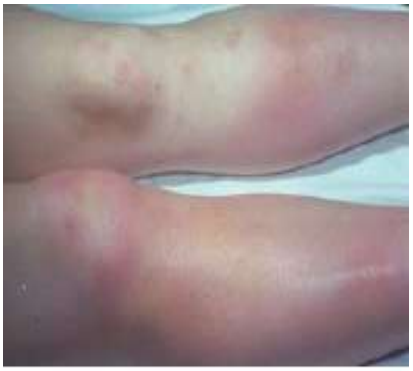
1- Manifestations of Inflammatory Bowel Disease (IBD):

	Association	Cutaneous Findings
Fissures and Fistulas	CD > UC	Commonly involves perineum associated with edema and inflammation (perianal skin tags)
Oral Crohn's	CD	Edema, cobblestone of buccal mucosa, ulcerations, nodules over the oral mucosa
Metastatic Crohn's	CD	Nodules, plaques, ulcerations; commonly on extremities or intertrigenous regions mimics Erythema Nodosum (when we take skin biopsy it will be pathologically the same as intestinal crohn's disease.)
Erythema nodosum	UC>CD	**Tender** red nodules on anterior lower legs; precedes or occurs simultaneous with IBD flare
Pyoderma Gangrenosum (PG)	UC>CD	Papules, pustules, hemorrhagic blisters → enlarge, ulcerate with dusky undermined edges; exacerbated by trauma; frequently on legs
Pyoderma Vegetans	UC	(It is subtype of pyoderma Gangrenosum) Vegetating plaques, vesiculopustules of intertrigenous (place where two areas of skin rub like breast fold or anogenital area); **heal with hyperpigmentation**; when process involves mucosa =Pyostomatits vegetan
Chronic Aphthous Ulcers	UC>CD	Identical to common aphthous ulcers; develop with IBD flares

Note: CD = Crohn's disease ,UC = Ulcerative Colitis , Other less common manifestation: Epidermolysis bullosa acquisita, erythema multiforme, urticaria, clubbing, psoriasis, vitiligo.

Metastatic Crohns' Disease





a- Erythema Nodosum:

• Erythematous, tender nodules (deep) on anterior shins; also seen on thighs, lateral aspects of lower legs, arms, and face , bilateral , symmetrical.

• Often accompanied by fever, chills, malaise, and leukocytosis

• 70% have associated arthropathy **(ankle and knee joint)**

• Occurs at any age, but most prevalent between **20 and 30** years of age

- **Causes :**

MNEMONIC (SHOUT BCG)

S=Sarcoid, Sulfa drugs, Strept.(most common cause of Erythema nodosum is post streptococcal pharyngitis) / H=Histoplasmosis (fungal infrction) / O=Oral contraceptives , pregnancy / U=Ulcerative colitis / T=TB / B=Bechet's / C=Crohns / G=GI (Yersinia, salmonella).

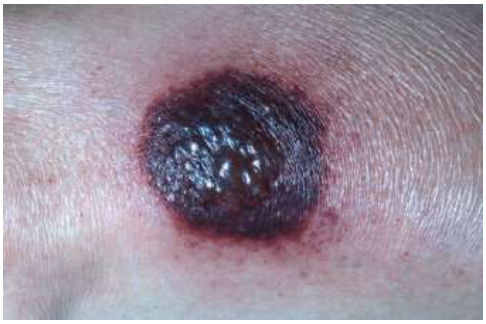
- **Work up:**

1. Hx *(exclude drugs (OCP) , hx of infection & GI symptoms(IBD vs. infection) &pulmonary symptoms for TB and sarcoidosis & pregnancy)*
2. CBC ,diff.
3. ESR (high in TB)
4. Throat swab
5. ASO titre (Anti-streptolysin O positive in streptococcal infection)
6. CXR (to rollout sarcoid, TB)
7. PPD(for TB)
8. Stool for occult blood
9. Skin biopsy (you have to take DEEP biopsy involving subcutaneous fatty tissue)

- **Histology: Septal panniculitis without vasculitis**
- **Treatment:**

- Spontaneous resolution usually occurs within three to six weeks **without scarring**
- NSAIDs such as indomethacin or naproxen
- Systemic steroids effective in severe cases and can be dangerous if infection is etiology
- Potassium iodide in severe cases

b- Pyoderma gangrenosum (PG):

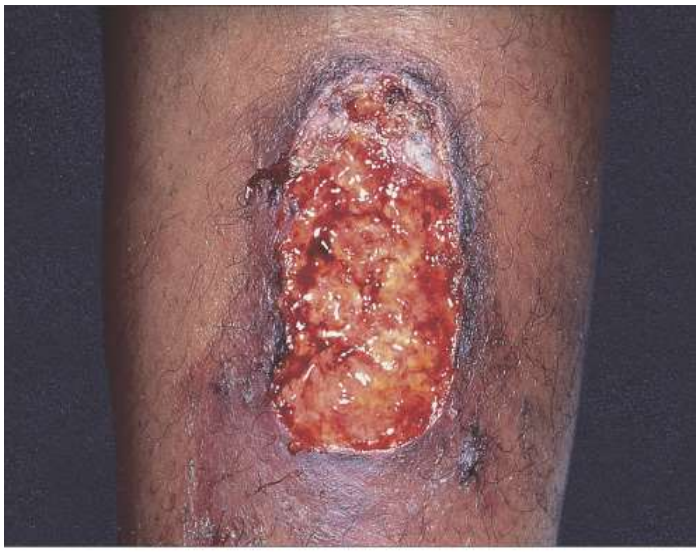


-1.5-5% of patients with IBD develop PG

• Associated with leukemia, myeloma , monoclonal gammopathy (IgA), polycythemia, chronic active hepatitis, HCV , HIV , SLE & pregnancy(so if you have patient with PG you need to screen them for hematological malignancies , inflammatory bowl disease, connective tissue disease and infection)

• Associated with PAPA syndrome → pyogenic arthritis, pyoderma gangrenosum, severe cystic acne

• May be associated with arthritis



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Distinct rolled edges and show satellite violaceous papules that break down and fuse with central ulcer (make sure you rule out heam malignancy , IBD , SLE)

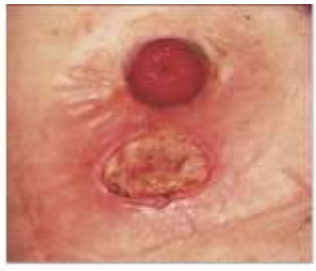
Four Types:

1. Ulcerative
2. Pustular
3. Bullous
4. Vegetative

Histology: Massive dermal edema with epidermal neutrophilic abscesses.

Treatment :

- Treat underlying cause
- Potent topical steroids or IL steroids
- Topical tacrolimus (immunodilator acts as calcineurin inhibitor)
- Systemic steroids when ulcer is big or advanced PG you must treat aggressively
- Cyclosporine,Sulfapyridine, sulfasalzine, and dapsone (orally)
- Infliximab (used for IBD)
- Other agents: thalidomide, SSKI, azathioprine, cyclophosphamide

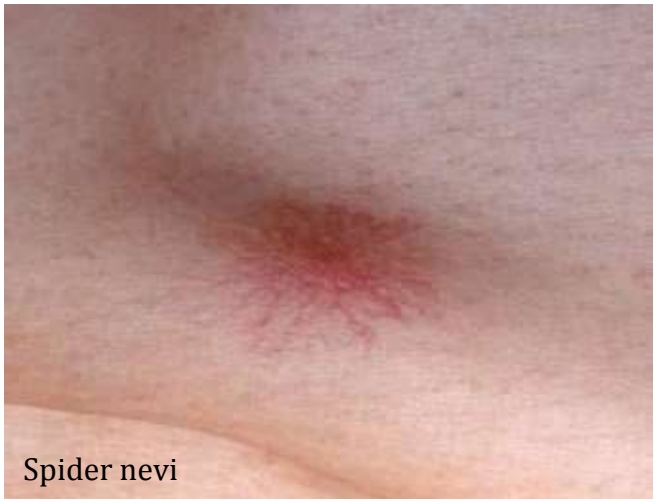


Peristomal Pyoderma Gangrenosum

7)Cutaneous Manifestation of Liver diseases:

- Pruritus: generalized itching especially in the presence of **biliary obstruction or jaundice**.
- Jaundice.
- Spider nevi: small telangeictatic blood vessels especially on the face and upper chest.
- Palmar erythema.
- Thinning of the hair and sometimes loss of sexual hair in the axillae and pubic areas. (because the estrogen level will increase when liver fails and so they androgen dependent hair is lost)
- Porphyria cutana tarda.
- Xanthoma

Terry's nail (the distal third of the nail is normal and proximal 2/3rd is luckonychia)



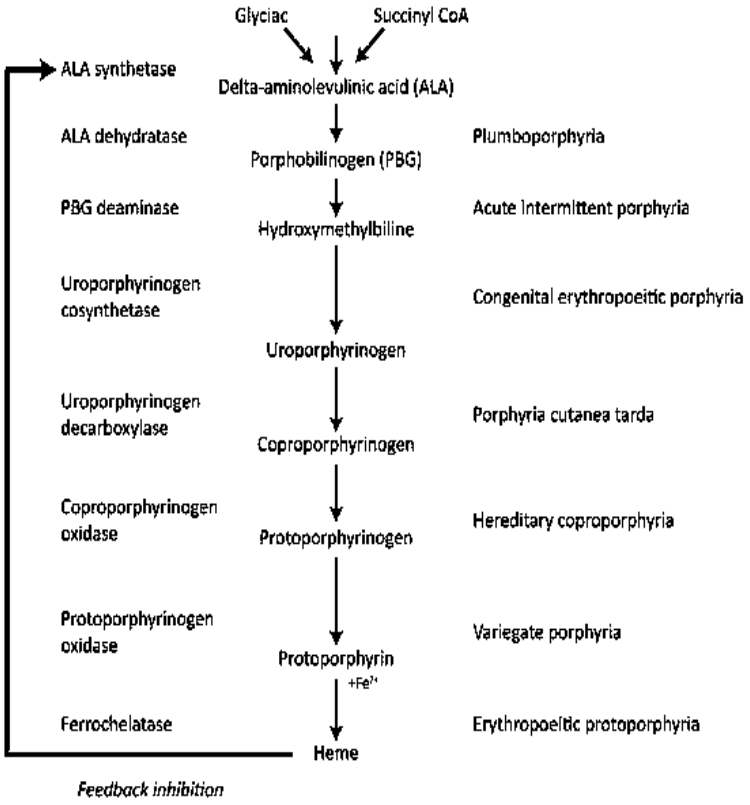
Spider nevi

Porphyria Cutanea Tarda (PCT)

(Dracola myth came from this manifestation)

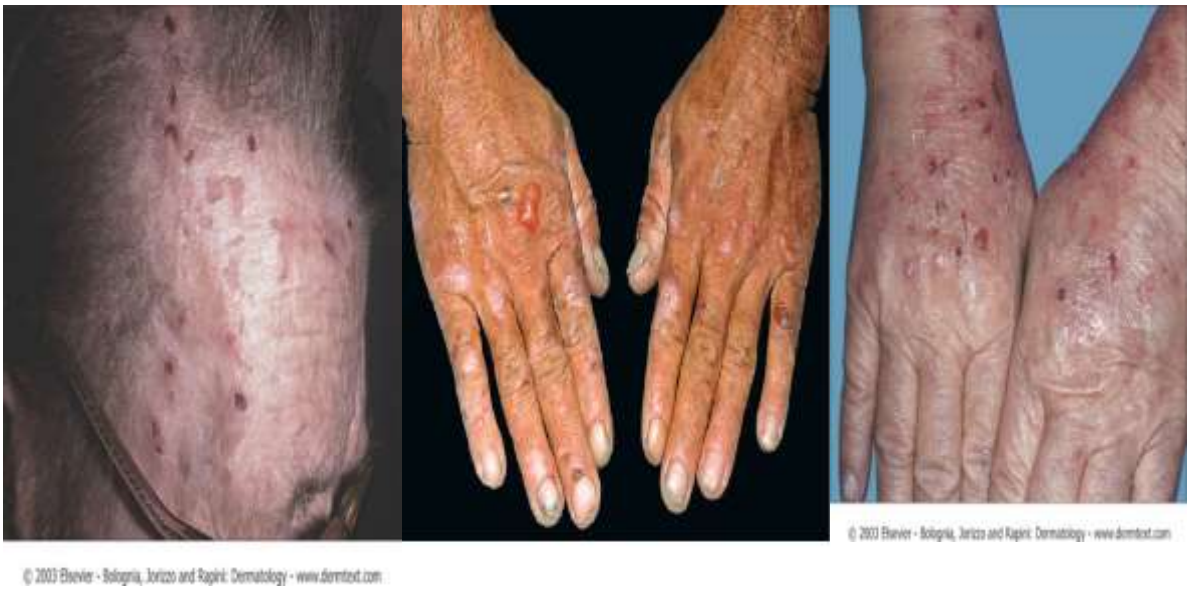
• The pathogenesis may be related to the suboptimal clearance of uroporhyrins (product of heme synthesis pathway) from the circulation which is a photosensitizer .

(the hemoglobin is formed from heme which require multiple steps and the defect involves a problem in the uroporphyrinogen decarboxylase enzyme which then accumulates the porphyrin ; photosensitizer .so if the poryphrin level is high



it will result in blisters formation whenever the patent is exposed to sun)

• Patients may present with photodistributed bullae, skin fragility, hyperpigmentation and hypertrichosis (increase in hair growth)



8)CUTANEOUS MANIFESTATIONS OF RENAL DISEASE:

End Stage Renal Disease (ESRD) and Dialysis:

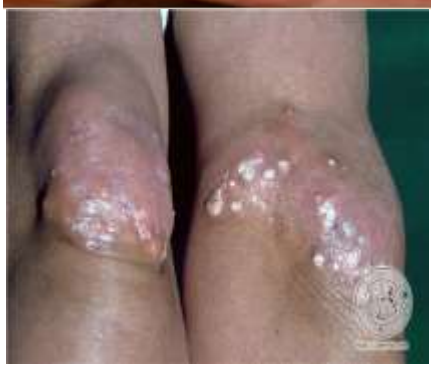
1- Pruritus: the most common cutaneous manifestation of ESRD secondary to increased serum urea

2-Half and half (Lindsay's) nails result from edema of the nail bed and capillary network and give the proximal half of the nail an opaque white appearance

3-Metastatic Calcification

• Deposition of calcium within tissue secondary to abnormal calcium and or phosphate metabolism

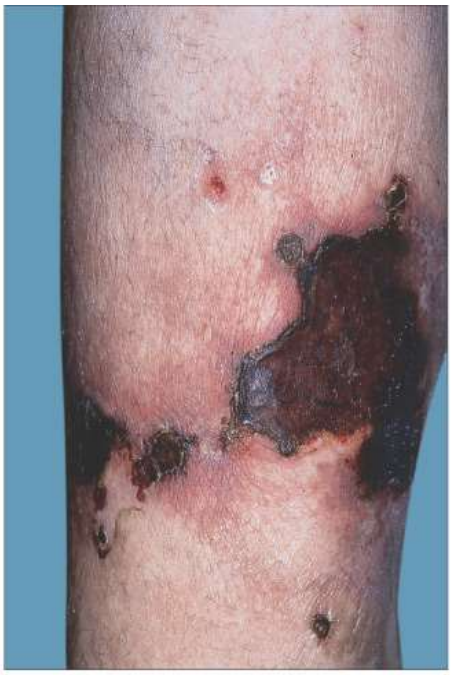
• It can manifest in the skin as benign nodular calcifications (calcinosis cutis (knee pic)) or as a more serious condition (calciphylaxis: the deposition of Ca will be in blood vessels so it will result in infarction and ischemia of the supplied organ) with an associated mortality rate between 60-80%.(this will develop because of vitamin D deficiency which will result in abnormal level of Ca and phosphate secondary to hyperparathyroidism)



Calciphylaxis presents as painful purpuric plaques and retiform (netlike) pupura with progression to ulceration and necrosis.

- Histological finding of medial calcification/intimal hyperplasia of small arteries and arterioles
- Management of these patients includes total or subtotal parathyroidectomy (if PTH levels are elevated), increasing the frequency of the dialysis to help washout the Ca from blood or give oral Ca binding salt, wound care, and avoidance or precipitating factors. Mortality is related to Staphylococcal superinfection of ulcers with resultant sepsis

The work up here includes biopsy , blood ca ,.phosphate , and PTH levels



4-Pseudo-Porphyria Cutanea Tarda

•Similar clinical and histological findings of PCT, in setting **of normal porphyrin profile**

- Usually **due to certain medications** such as furosemide, naproxen, tetracycline, nalidixic acid, or amiodarone or in patient with renal failure on dialysis

Male Team 432:

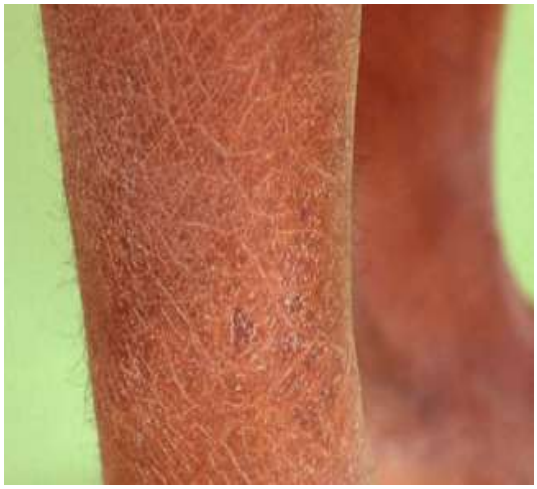
Xerosis:

Definition: It is abnormal dryness of the skin, mucous membranes, or conjunctiva .

Xerosis occurs in 50-92% of the dialysis population.

Etiology: The exact cause of xerosis in ESRD remains unknown.

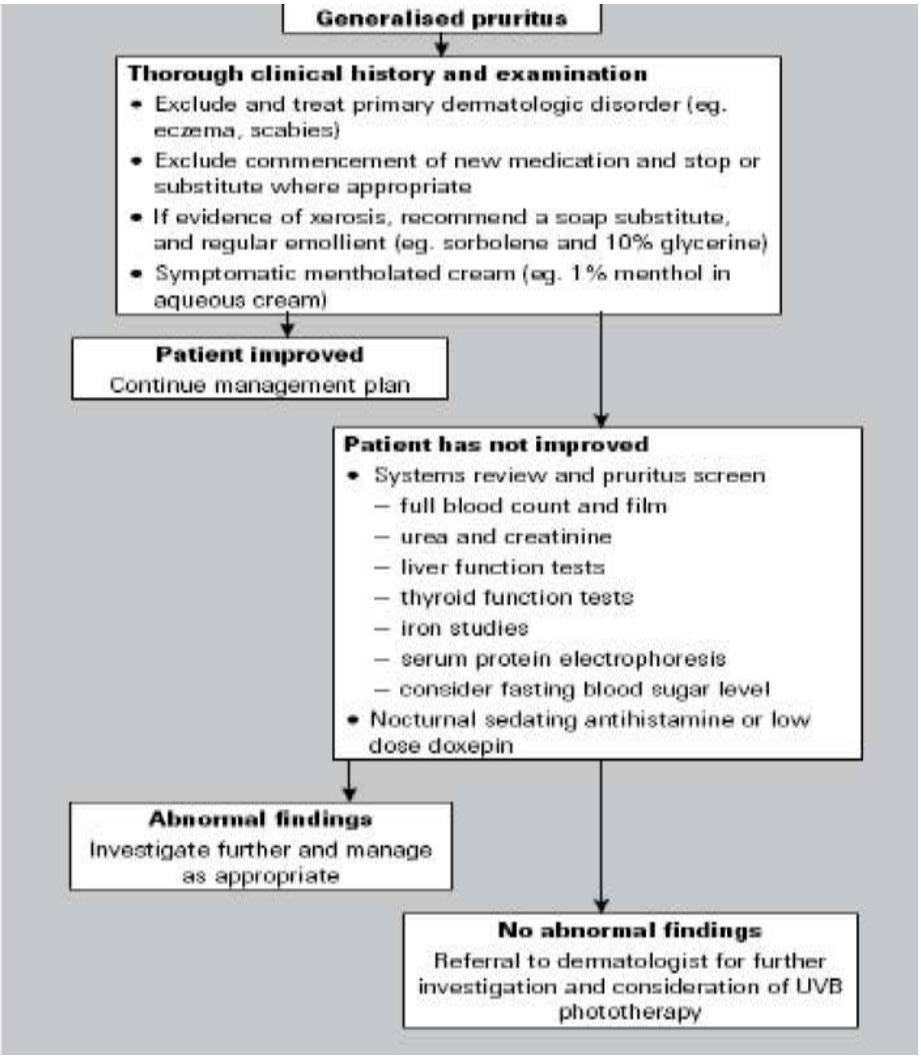
Management: Many patients respond to routine use of emollients.



9)Generalized Pruritus:

-Generalised pruritus in the absence of a rash requires investigation and exclusion of an underlying systemic disorder
-it is important to distinguish these from an underlying primary skin disease such as scabies or eczema

Conditions that Cause Pruritus:
1-Chronic Renal Disease
2-Cholestasis
3-Endocrine Disease
• *Thyrotoxicosis – often due to increased skin blood flow which raises skin temperature*
• *Hypothyroidism – pruritus secondary to the dry skin*
4-Malignancy
• *Most common association: Hodgkin's disease and polycythemia rubra vera*
5-HIV Infection
6-Iron deficiency anemia
7-Parasetic infection



Workup of Generalized Pruritus:
•History and Physical exam
• CBC, diff , Blood film → for hodgkins'
• Stool for Ova&Parasite , occult blood
• CXR
• Thyroid, renal, and liver function tests

Note :
Rheumatic fever :
Erythema marginatum
Subcutaneous nodules
Osler nodes
Janeway lesions
Splinter hemorrhages and clubbing