



The skin and systemic disease

Clues to possible systemic disease:

- ? Rash ass. with joint pain, fever, weight loss, weakness, SOB, altered bowel function
- ? Rash not responding to topical tx
- ? Erythema of the skin (inflammation around blood vessels)
- ? Non blanching palpable purplish fixed lesion , painful and blistering >> vasculitis
- ? Unusual changes in pigmentation or texture of the skin.
- ? Palpable dermal lesion 2ry to granuloma , mets , lymphoma and so on...

Characteristic rash that indicates underlying systemic disease :

- ❑ Erythema multiforme >> HSV, Hep B/C, mycoplasma pneumonia
- ❑ Pyoderma gangrosum >> IBD , RA , hematological malignancy
- ❑ Erythema nodosum >> IBD , streptococcal infxn , TB , sarcoidosis , behcet's
- ❑ Vasculitis >> Hep B /C , SLE , lymphoma , leukemia



Skin reactions associated with infections

1. Toxic erythema



Toxic Erythema

- ? Widespread symmetrical Maculopapular erythema (morbilliform)
- ? starts on the trunk then spreads to the limbs
- ? Usually asymptomatic (itchy)
- ? Blanching
- ? Triggered by viruses, bacteria, drugs
- ? Classic presentation??
- ? Tx: No need, can use;
 - ? emollient and topical steroid if symptomatic
 - ? treat the underlying disease



Gianotti-Crosti syndrome

2. EM

- ? Epidemiology
- ? Presentation : target lesions
- ? Pathophysiology: Type IV HSR
- ? Classification : EM Minor vs Major
- ? Causes: according to classification, most common cause: HSV
- ? Dx : Clinical
- ? TX
- ? Recurrence ?
- ? Coarse
- ? Rare associations

EM

- ❓ The most common infectious trigger HSV
- ❓ Systemic symptoms of the underlying infection usually precede the EM rash by 2–14 days.



EM

Acrally { face
hands
feet. } Target

Doll's eye

3 shades
of colors

Dusky red (vesicle/necrosis
pinkish Red again

sometimes).



HSV

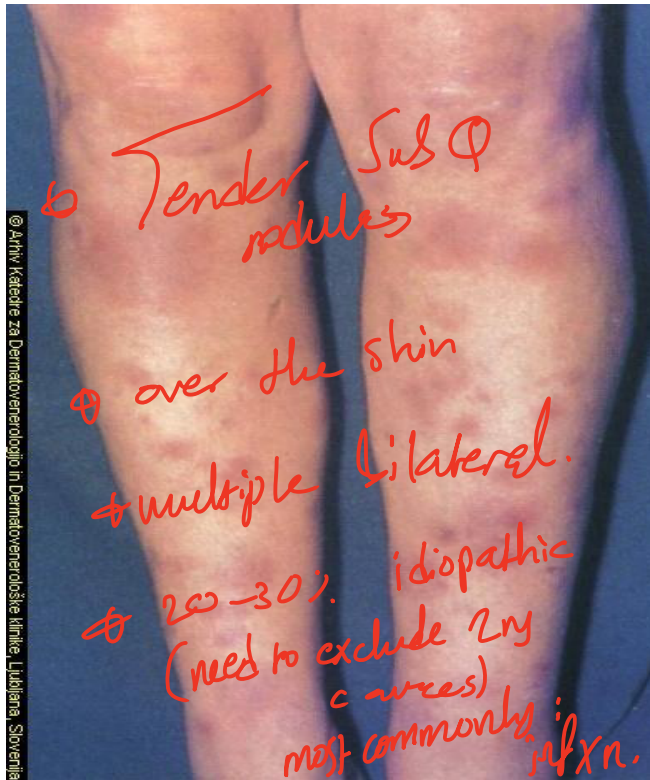
5-14 days.

Erythema Nodosum

Panniculitis (adipose tissue inflammation)

- ? Most common Panniculitis.
- ? Epidemiology: F >> M
- ? Presentation: acute, painful nodules (Shin), especially by palpation.
- ? Idiopathic (50%) vs Secondary
- ? Secondary causes: Multiple, shown in the table
- ? Most Common Sec : Strep
- ? Skin lesions: very tender nodules(3–20 cm), not sharply margined
- ? Dx & management: Bacterial Culture, Imaging, Dermatopathology
- ? Course: Spontaneous resolution occurs in 6 weeks
- ? Tx: treat secondary; rest + NSAIDs (steroids ?)

3. Erythema nodosum



EN: Causes

TABLE 7-1 CAUSES OF ERYTHEMA NODOSUM^a

Infections

Bacterial

Streptococcal infections, tuberculosis, yersiniosis
Other: *Salmonella*, *Campylobacter*, *Shigella*,
brucellosis, psittacosis, *Mycoplasma*

Fungal

Coccidioidomycosis, blastomycosis, histoplasmosis,
sporotrichosis, dermatophytosis

Viral

Infectious mononucleosis, hepatitis B, orf, herpes
simplex

Other

Amebiasis, giardiasis, ascariasis

Other

Drugs

Sulfonamides; bromides and iodides
Oral contraceptives
Other: minocycline, gold salts, penicillin, salicylates

Malignancies

Hodgkin and non-Hodgkin lymphoma, leukemia,
renal cell carcinoma

Other

Sarcoidosis
Inflammatory bowel disease: ulcerative colitis, Crohn
disease
Behçet disease

4. Erythema annulare centrifugum

Redness

تَبَوُّ سَوِي
centrifugally.



Vs Tinea Corporis

5. Erythema chronicum migrans

like syphilis

Bacteria ← spirochete.

? Caused by Borrelia burgdorferi (lyme disease)

Ticks transmitted

? Migrating erythema

? Cutaneous inflammatory response to Borrelia.



←
hematogenous spread
CVS
CNS
flu like symptoms.



Sarcoidosis

Sarcoidosis

- CXR: Bilateral hilar lymphadenopathy
- Non caseating (unlike TB)
- Multi systemic
- 20% cutaneous Big mimic .

- ? Unknown etiology, atypical mycobacterium may be the trigger
- ? **Presentation:**
- ? May occur with or without cutaneous disease
- ? **Skin lesions :** Specific vs Non-specific
- ? Earliest: skin-colored papules, on the face, heal without scarring.
- ? Then: Plaques; annular, extremities and Buttocks, **SCARRING, Chronic disease**
- ? Lupus Pernio: violaceous plaques on the nose and cheeks
- ? EN: m/c NS skin lesion, early sarcoid.
- ? Scarring sarcoidosis.

Sarcoidosis

*Psoasiform
excemations*

scaly plaques



Dusky infiltrated lesions: **Lupus Pernio**

Acrally ↙ nose

Bad prognostic sign
(URI, LRI, osteolytic
lesions ... ?)



Lupus pernio vs. Rosacea (midfacial erythema).
Sharp demarcation & violaceous hue



Scarring sarcoid

periocular

DDx:
hypertrophic pre-existing scar → sarcoid



Löfgren syndrome

Acute syndrome

multisystemic

Benign / resolves spontaneously

-Erythema nodosum

-Fever

-Arthritis

-Hilar adenopathy

- Iritis

Löfgren Syndrome



Hilar
lymphadenopathy



Acute polyarthritis
(usually ankles)



Erythema nodosum

Diagnosis and management

- ❓ Imaging
- ❓ Labs
- ❓ Lesional biopsy
- ❓ Tx:
- ❓ Systemic involvement: systemic steroids
- ❓ Limited cutaneous: topical steroids, if extensive: systemic steroids, mtx, hydroxychloroquine.



Skin changes ass with hormonal imbalance

Hyperpigmentation

Generalized Addison's
mucous membrane
creases.
Localized
(melasma)

- ? Increase in circulating hormones with melanocyte-stimulating activity
- ? Hyperthyroidism, acromegaly, **Addison's disease**
melasma
- ? Pregnancy, OCPs → melasma/chloasma (localized on the forehead and cheeks)
- ? It may fade slowly if ultraviolet light is excluded from the affected skin using daily sun block.



→ Strict sun protection

Hypopigmentation

- ❓ Widespread partial loss of melanocyte function
- ❓ Hypopituitarism (absence of MSH)

Acanthosis nigricans

↳ Increased thickness of epidermis

→ Black

- Insulin resistance.
- axilla, inguinal, neck
- obese areas



extensive
involvement:

Gastric
adenocarcinoma

Necrobiosis lipoidica

- destruction of collagen (in dermis)
- Atrophy + translucent BVs.
- yellowish hue (deposition of lipids)



Associated with DM

65% of ppl with ~



Not related to the severity of DM

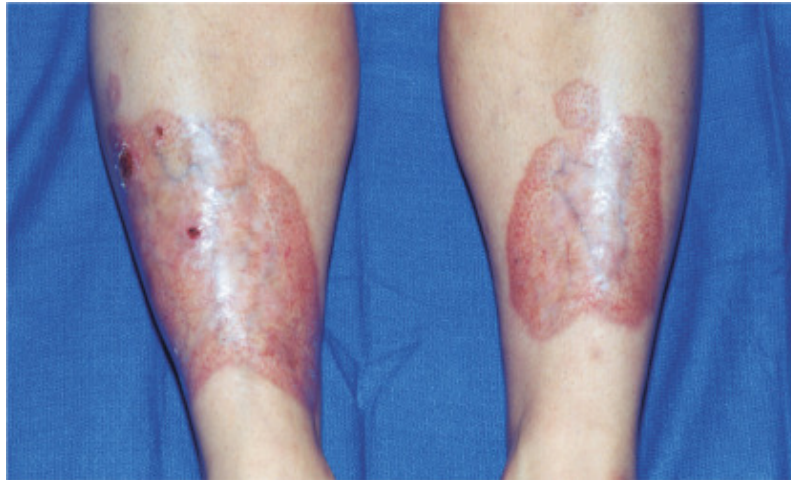


Coarse not affected by controlling blood sugar



Tx: intralesional steroids, surgery.

check HgA1C





Thyroid disease

Table 10.1 Clinical signs of thyroid disease.

Hypothyroidism

Dry skin
Oedema of eyelids and hands
Absence of sweating
Coarse, thin hair; loss of pubic, axillary and eyebrow hair
Pale 'ivory' skin
Brittle poorly growing nails
Purpura, bruising and telangiectasia

Generalized myxoedema

Hyperthyroidism

Soft, thickened skin • edema → pitting (fluid shift)
• GAGs accumulation (non pitting).
Pretibial myxoedema
Increased sweating (palms and soles)
Thinning of scalp hair
Diffuse pigmentation
Rapidly growing nails
Palmar erythema
Facial flushing

Grave's disease

Graves disease

- ❓ Hyperthyroidism with diffuse goiter, Ophthalmopathy and dermatopathy.
- ❓ Dermopathy: *pretibial myxedema*
- ❓ Tx: *steroids*





Skin changes with GI and liver diseases

Zinc deficiency

? Acrodermatitis enteropathica : genetic disorder of Zinc absorption (seen in neonates)

Bottle fed → no zinc
Breast fed → full of zinc
in the transport of zinc.

? Acquired zinc deficiency (AZD) occurs in older individuals due to dietary deficiency or failure of intestinal absorption of zinc (malabsorption, alcoholism, prolonged parenteral nutrition)

? Skin changes usually appear within weeks of birth with erythematous inflamed scaly skin around the mouth, anus and eyes

← genetic
acquired (malabsorption).



Zinc def

- ? If the condition is not recognised and treated promptly then the skin can become crusted, eroded and secondarily infected.(candida, S.aureus)
- ? Other findings:
- ? Zinc supplementation (1mg/kg/day), should be continued until zinc levels normalise (or lifelong in inherited forms).

Vitamin C deficiency

• imp 4 collagen synthesis

Scurvy

- ? occurs in those with malabsorption problems, those on a poor diet, the elderly and alcoholics
- ? Vascular fragility
- ? **Skin lesions:** Petechiae, follicular hyperkeratosis with perifollicular hemorrhage, especially on the lower legs
- ? Other findings:
- ? Labs: Normocytic, normochromic anemia, Serum ascorbic acid level zero. X-ray findings are diagnostic
- ? Tx: Ascorbic acid

- * Hyperplastic.
- * easy bleeding after teeth brushing.



3. Pyoderma gangrenosum

• look for 2nd cause

? Rapid and painful ulcerated

necrotic skin areas with

hypertrophic undermined

purplish margins.

? Ass with UC, Crohn's, RA,

leukemia, monoclonal

gammopathy.

Tribial trauma

*papule → pustule
rapid and sudden.*

PG.



Bullous disease

4. Dermatitis herpetiformis

Celiac disease.

• extensor surfaces
• highly pruritic



5. Liver disease

Box 10.4 Liver disease and the skin

Obstructive

- Jaundice

- Pruritus.

Liver failure

- Multiple spider naevi

- Palmar erythema

- White nails: hypoalbuminaemia

- Porphyria cutanea tarda.

Cirrhosis

- Xanthomas (primary biliary cirrhosis)

- Asteatosis.

Porphyria cutanea tarda

heme synthesis defect

• according to which enzyme of heme metabolism is affected

• not all porphyrias have cutaneous manifestations



porphyrins accumulate

↑ sensitivity to sunlight.

•

• recurrent inflammation cause scarring (milia).

Xanthomas

& M^o h^ol of lipids due to genetic diseases
→ yellowish = lipid



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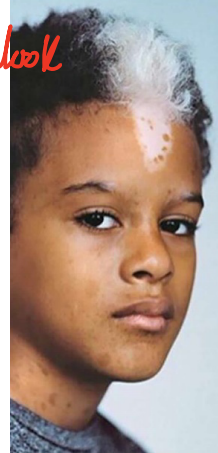
Congenital

? **Albinism**, AR, loss of pigment of skin, eyes and hair, no pigment production → there are melanocytes (eye problems, nystagmus) defective tyrosinase enzyme.

? **Piebaldism**, AD, triangular hypopigmented patches, disorder of melanocyte development forehead, forelock

? **Vitiligo** localized depigmentation, sharply demarcated, symmetrical macular lesions, loss of melanocytes and melanin Desmulation of melanocytes البرص

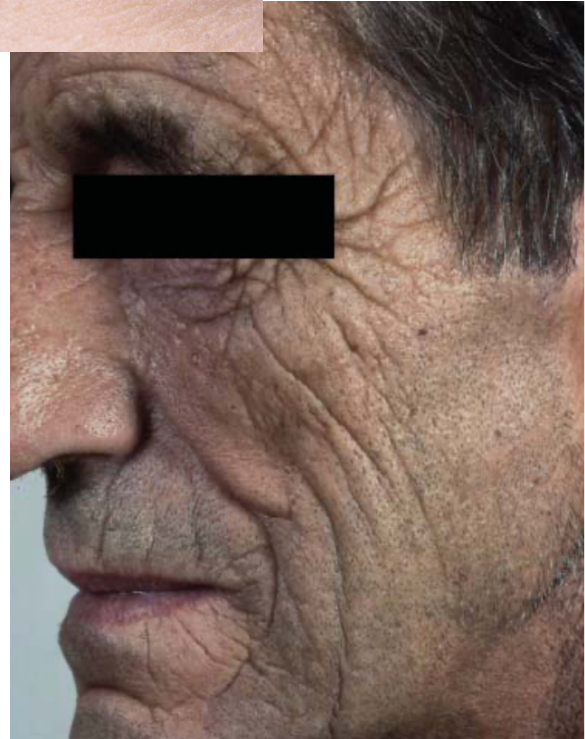
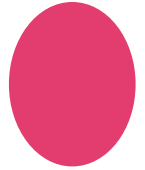
? **+ Post-Inflammatory** such as psoriasis, eczema, lichen planus and lupus erythematosus; infections, chemicals, reactions to pigmented naevi, genetic diseases



There is wide variation in the pattern of normal pigmentation as a result of hereditary factors and exposure to the sun.

Darkening of the skin may be due:

- 1. An increase in the normal pigment melanin
- 2. Deposition of bile salts from liver disease
- 3. Iron salts (haemochromatosis)
- 4. Drugs or metallic salts from ingestion
- 5. AN is characterised by darkening and thickening of the skin of the axillae, neck, nipples and umbilicus
- 6. Post-inflammatory pigmentation is common, often after acute eczema, fixed drug eruptions and lichen planus.
- 7. In malabsorption syndromes such as pellagra and scurvy, there is commonly increased skin pigmentation.



Skin changes of underlying malignancy

Skin disorders associated with malignancy:

| Skin disorder | Associated malignancies |
|------------------------------------|---|
| Acanthosis nigricans | Gastric cancer |
| Acquired ichthyosis | Lymphoma |
| Erythroderma | Lymphoma |
| Acquired hypertrichosis lanuginosa | Gastrointestinal and lung cancer |
| Erythema gyratum repens | Lung cancer |
| Dermatomyositis | Bronchial and breast cancer |
| Migratory thrombophlebitis | Pancreatic cancer |
| Necrolytic migratory erythema | Glucagonoma |
| Pyoderma gangrenosum | Myeloproliferative disorders |
| Sweet's syndrome | Hematological Ca e.g. Myelodysplasia - tender, purple plaques |
| Tylosis | Oesophageal cancer |

Mycosis fungoides

* 1st cutaneous T cell lymphoma

> different clinical presentations.



Poikeloderma (telangiectasia, reticulate pigmentation)

radiotherapy, ?, T cell } idiopathic

Hyper + Hypo pigm
+ telangiectases.
→ atrophy.



Skin and Pregnancy

? **Prurigo gestationis-** is a benign non-specific pruritic (itchy) papular rash that arises during pregnancy and is generally more severe in the first trimester.

? **Polymorphous eruption of pregnancy (PEP)** is a pruritic erythematous rash that usually starts in the striae of the abdomen during the third trimester and can become widespread. No effect on baby

? **Pemphigoid gestationis (PG)** is a rare disorder that may initially resemble PEP but develops pemphigoid-like vesicles, spreading over the abdomen and thighs (Figure 10.23). PG is an autoimmune disorder, (Small baby and higher mortality).

*Benign
Doesn't
affect
baby*

*low weight
low gestation age.*



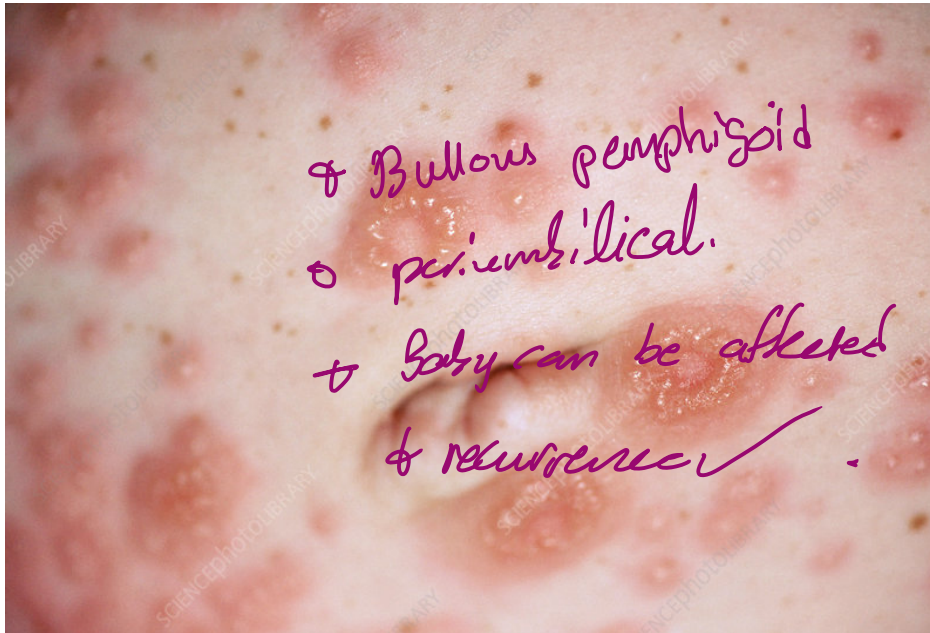


poly
not vesicles
(eczematous
papules
due to tension,
molecular changes
of spreading to the
umbilical region.

& no recurrence.

PEP

Pemphigoid gestations





GOOD LUCK