

Seborrhoeic dermatitis

#

Acne vulgaris

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**Cutaneous and oral symptoms
of internal diseases**

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Seborrhoeic dermatitis

Seborrhoeic dermatitis

- very common, incidence: 2-5 % of the population
- more common in males
- age of onset: infancy, puberty, 20-50 years

Pathogenesis

- pathogen: *Malassezia furfur* (*Pityrosporum ovale*), a yeast
- skin symptoms are located on regions where sebaceous glands are most active: scalp, external ear, retroauricular region, eyebrows, glabella, eyelashes, face, nasolabial folds, presternal area, body folds
- dry, scaling dermatitis - erythematous plaques, covered by white dry scales
- greasy, crusted form
- moist form

Skin symptoms

scalp: „dandruff”

face (frontal region)

Differential diagnosis

- psoriasis vulgaris
- impetigo
- tinea capitis, corporis
- pityriasis versicolor
- intertrigo
- SCLE
- secondary syphilis

Treatment

- scalp: selenium sulfide, zinc pyrithione or ketoconazol containing shampoos
- skin: topical corticosteroid solutions, gels, creams; topical ketoconazol cream
- oral retinoid treatment: in severe cases only

Acne vulgaris

Definition

- very common, affects 85 % of young patients
- age at onset: 10-17 years (puberty), most cases are seen during the middle-to-late teenage period
- acne could persist through the third decade or even later particularly in women
- disease involves the pilosebaceous unit
- affected sites: face, neck, upper part of chest and back

Pathomechanism

1. Follicular hyperkeratosis
2. Increased sebum production
3. Colonisation of *Propionibacterium acnes*
4. Hyperandrogenism

1. Follicular hyperkeratosis

- primary change: hyperkeratinization within the hair follicle, increased cellular turnover in keratinocytes
- keratin plug formation
- block in the discharge of sebum

2. Increased sebum production

- sebum plays an important role, because:
 - acne appears in puberty when sebaceous glands start to produce sebum
 - acne can be controlled by drugs inhibiting the sebaceous glands (antiandrogens, estrogens, oral retinoids)
- sebum is comedogenic, causes inflammation

3. Colonisation of Propionibacterium acnes

- predominant microorganism in the follicular flora:
Propionibacterium acnes
- P. acnes produces lipases, other enzymes (proteases, hyaluronidases), secretes chemotactic factors and produces inflammation in the perifollicular dermis after disruption of the distended follicle

4. Hyperandrogenism

- androgens regulate the development of sebaceous glands and the sebum production
- may play a role in the follicular hyperkeratinization
- hyperandrogenism should be considered a contributing factor for the development of acne in female patients

Clinical signs of acne

- comedo: open (blackhead)
closed (whitehead)
- papule
- pustule
- nodule
- cyst
- atrophic or hypertrophic scar
- keloid

Most common clinical forms

- comedos → **Acne comedonica**
- papulopustules → **Acne vulgaris**
- nodules → **Nodular acne**
- cysts → **Cystic acne**
- scars (hypertrophic, keloid) → **Acne keloidalis**

Treatment of acne vulgaris

I. Topical:

- benzoyl peroxyde
- antibiotics
(erythromycin,
clindamycin)
- retinoid

II. Systemic:

- antibiotics
(tetracyclines,
erythromycin)
- retinoid (isotretinoin)
- antiandrogens

Recommended treatment for various acne forms

For comedos:

- benzoyl peroxyde
- topical antibiotics
- topical retinoids
- oral isotretinoin
- oral tetracycline

To decrease sebum production:

- oral isotretinoin
- antiandrogens

To eliminate *P. acnes*:

- benzoyl peroxyde
- topical antibiotics
- oral isotretinoin
- oral tetracycline
- spironolactone

To decrease inflammation:

- benzoyl peroxyde
- topical antibiotics
- topical retinoids
- oral isotretinoin
- oral tetracycline
- spironolactone

Rare forms of acne

1. Acne conglobata
2. Acne fulminans
3. Acne excoriée
4. Neonatal acne
5. Occupational acne
6. Corticosteroid acne
7. Drug induced acne
8. Acne cosmetica
9. Gram negative folliculitis

Corticosteroid acne

- monomorphic papules, pustules (mainly on the trunk)
- provoked by topical or systemic steroid treatment
- treatment: conventional acne therapy

Drug induced acne

- antibiotics: penicillin, macrolids
- ACTH, nystatin, isoniazid, itraconazole, hydroxychloroquine, naproxen, clonazepam, lithium, vitamin B

Skin symptoms of internal diseases

Skin symptoms of **diabetes mellitus**

- infections
- pruritus
- microangiopathy
- macroangiopathy
- neuropathy
- necrobiosis lipoidica
- xanthoma, xanthelasma
- bullosis diabeticorum
- scleroedema

Immunodeficiency in diabetes

- derangements of immunoregulatory mechanisms frequently occur in diabetes
- hyperglycemia and ketoacidosis diminish chemotaxis, phagocytosis, and bactericidal ability of white blood cells



Infections

- Streptococcus
- Staphylococcus
- yeasts
- dermatophytes
- deep mycoses

Bacterial infections

Folliculitis

Furuncle

Carbuncle

Erysipelas

Mycotic infections

Onychomycosis

Candida intertrigo

Candida paronychia

Angulus infectiosus oris

Candidosis of the tongue

Black hairy tongue

Genital candidosis

- **Vulvovaginitis**
 - mainly the complicated form (recurrent disease, caused by non-albicans *Candida* species)
- **Balanitis, balanoposthitis**

Causes of itching in diabetes

- skin dryness
- intercellular hyperglycaemia
- osmotic changes in the epidermis
- diabetic thick skin
- decreased cellular immunity

Diabetic microangiopathy

- basal membrane thickening
- increased permeability of blood vessels due to glycolysation of the basal membrane proteins (collagen type IV, laminin, heparan sulphate)
- decreased response of vessels to sympathetic innervation
- decreased ability to respond to thermal and hypoxemic stress
- concomittant atherosclerosis of large vessels

Symptoms of macro- and microangiopathy

- ecchymosis, suffusion
- subungual hematoma
- rubeosis diabetica
- palmoplantar erythema
- malum perforans pedis
- trophic ulcers
- gangrena diabetica

„Diabetic foot”

Malum perforans pedis

Necrobiosis lipoidica

Alterations in lipid metabolism in diabetes

- insulin deficiency resulted in dysregulation of lipid metabolism, because activity of the lipoprotein lipase is directly dependent on the presence of insulin
- lipoproteins, chylomicrons permeate through the cutaneous vessel walls and accumulate in the dermal macrophages
- skin symptoms of hyperlipoproteinaemia: **xanthelasma**, **xanthoma**

Xanthoma

Skin symptoms of **liver diseases**

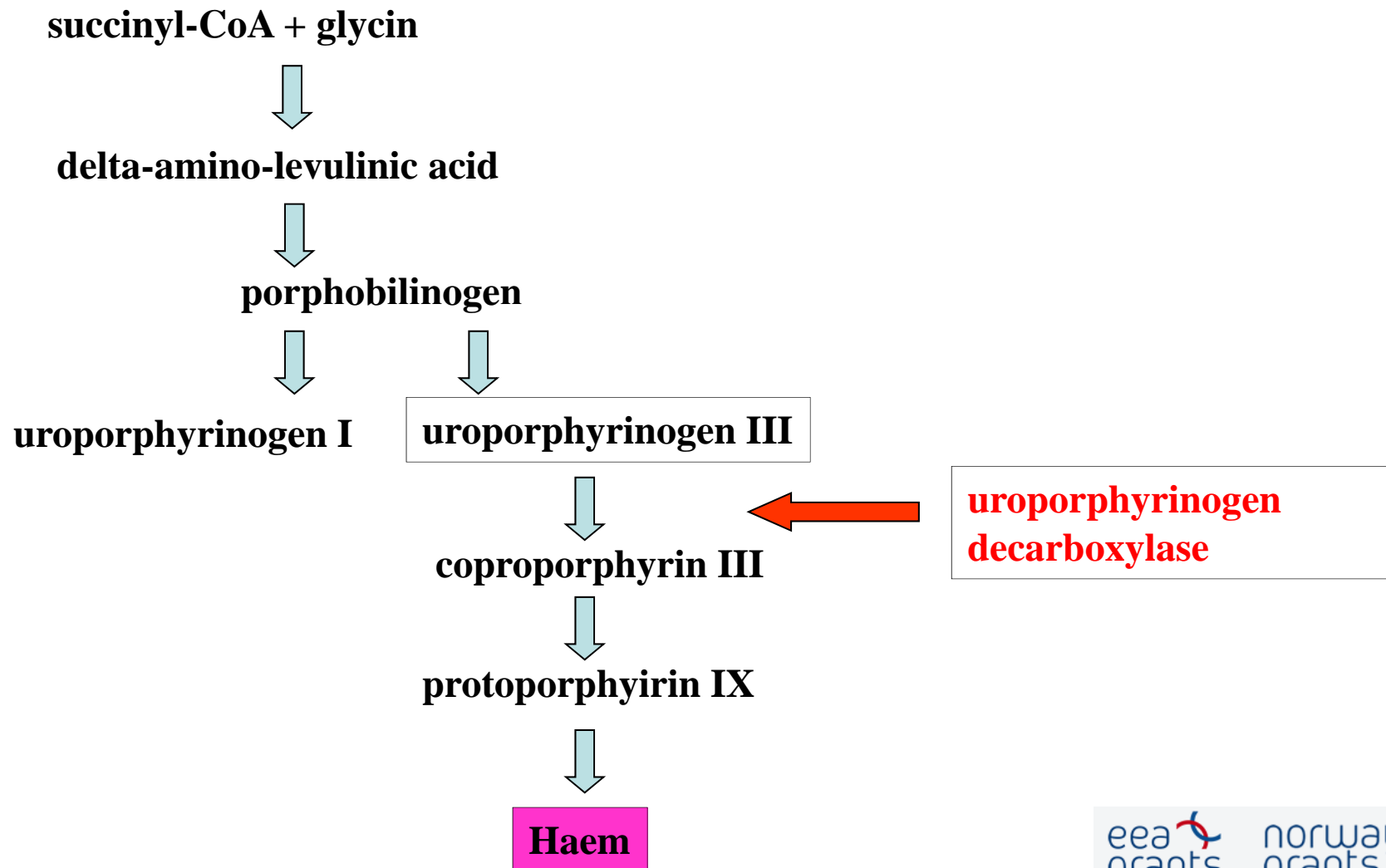
- yellow discoloration: jaundice
- hyperpigmentation (chloasma, melasma)
- erythema palmare (liver palm)
- teleangiectasia, spider nevus, caput Medusae
- purpura, suffusion
- gynecomastia, hypogonadism, hypotrichosis
- porphyria cutanea tarda
- nail abnormalities - leukonychia
- xanthoma, xanthelasma
- skin dryness, pruritus

Jaundice

Porphyria cutanea tarda

- precipitated by exposure to hepatotoxic chemicals (**alcohol**, hexachlorobenzene), or drugs metabolized in the liver (estrogens)
- **Type I** - sporadic form (alcohol, HCV, estrogen, SLE)
- **Type II** - familial form (chemicals - hexachlorobenzene)
- main symptoms: fragility and photosensitivity of the skin

Cause: abnormal haem biosynthesis - uroporphyrinogen decarboxylase enzyme deficiency



vesicle, bulla formation

scars, erosions

temporal hypertrichosis

Pruritus in other internal diseases

- renal failure (uremia)
- obstruction of bile duct (primary biliar cirrhosis)
- endocrin disorders: thyreotoxicosis, hypothyreosis, diabetes mellitus, carcinoid
- hematological disorders: Hodgkin's disease, polycythaemia, non-Hodgkin's lymphoma, multiple myeloma
- visceral cancers
- anemia (iron deficiency)