COMMON SKIN DISORDERS
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I. DERMATITIS:

Acute: Erythema, edema, vesiculation
Subacute
Chronic: Mild erythema, scaliness, lichenification (thickening of the epidermis with exaggeration of normal skin lines), with or without hyperpigmentation.

Classification of dermatitis:

1st. Exogenous dermatitis (contact dermatitis): Dermatitis follows contact pattern.

1. Primary irritant contact dermatitis: Direct chemical (acids or alkalies) or physical action on the skin. Not immunologic. Reaction can be elicited in all individuals.

2. Allergic contact dermatitis: A form of cell-mediated (tuberculin-like, type IV) response. The hypersensitivity is based on a specific immunologic alteration requiring an incubation period of several days. Re-exposure to allergen causes dermatitis that appears eight to ninety six hours after exposure. Usually, only a small percentage of the population is affected.

Mechanism of sensitization: Most environmental allergens are haptens, that is, simple chemicals that must link to proteins to form a complete antigen before they sensitize. The antigen combines with binding sites on membrane of Langerhans cell. It is taken up and processed by this cell which then migrates from the epidermis to the draining lymph node where it sensitizes lymphocytes. Sensitized lymphocytes enter the blood circulation. The whole skin becomes sensitized.

1st. Endogenous dermatitis (constitutional): Several types exist. The commonest are:

1. Atopic dermatitis: Increased susceptibility to atopic diseases, e.g. asthma, hay fever, urticaria, allergic conjunctivitis and systemic drug allergy. There is increased production of IgE antibodies, the role of which in dermatitis is not fully clarified.

The skin is inherently irritable (sensitive) and dry with low threshold for itching. Itching leads to scratching and lichenification. The diaper area is
particularly affected in infants while flexures are affected in childhood and later on in life. The majority of patients clear by puberty.

2. Seborrheic dermatitis: Cause is unknown. Associated with excessive sebaceous secretion (seborrhea) and is usually localized in the areas of greatest sebaceous activity. Onset is during the first few month of life and at puberty. Lesions consist of subacute dermatitis covered by greasy scales. Dandruff is a form of seborrheic dermatitis. The dermatitis is frequently associated with acne. The dermatitis in adults lasts for a lifetime.

II. BACTERIAL INFECTION:

Classification according to depth of infection.

1st. Infection localized to hair: All caused by Staphylococcus aureus, caogulase positive.

1. Folliculitis: Infection of most superficial part of hair follicle. Clinically, pustule pierced by hair.

2. Furuncle: Infection of whole length of hair follicle. Painful.

3. Carbuncle: Infection of a group of follicles (grouped furuncles).

1st. Infections affecting the skin as a whole:

1. Impetigo contagiosum: Most superficial. Mainly an epidermal reaction. Caused by beta-hemolytic streptococci (Streptococcus pyogenes), Staphylococcus aureus, or both. Lesions are characterized by yellowish crusts. Bullous variety is caused by certain strains of Staphylococcus aureus. Nephritis due to beta-hemolytic streptococcus may result.

2. Ecthyma: Affects epidermis and dermis (ulcerated impetigo). Bacteriolobially similar to impetigo.

III. VIRAL INFECTIONS:

1. Herpes simplex:

   One. Type 1: Usually over lips but can occur anywhere. Proto-type lesion is clustered vesicles (intraepidermal). Recurrent attacks: virus residing in ganglia. Precipitating factors for relapse include sunburn, URT infections, GI upsets, menstrual cycle.


2. Herpes zoster: Caused by the varicella virus. Virus resides in ganglia. Clinically: Unilateral vesicular rash along nerve distribution. Does not cross midline. Usually associated with pain which may precede the skin lesions.

3. Human papilloma virus infections: More than 70 strains.

   One. Skin:

      (a) Verruca vulgaris (common warts): 75% of cases. Papule with rough surface.

      (b) Plantar wart: 20% of cases. Usually deep and painful. Different from corn in that it shows clotted blood vessels.

      (iii) Plane warts: 5% of cases. Most often on face.

   One. Mucous membranes:

      Genitalia: Condyloma acuminate. Sexually transmitted. Some strains may be oncogenic.


IV. FUNGAL INFECTIONS:
1st. Superficial infections: Worse during spring and summer due to sun, heat and humidity.

1. Tinea versicolor: Caused by overgrowth of hyphal form of Pityrosporum ovale, a lipolytic yeast that resides in keratin of skin and hair follicle. Affects the most superficial layer of the stratum corneum. Lesions consist of slightly scaly macules that show increased or decreased pigmentation. KOH smear: Mycelia and spores.

2. Dermatophytic infection (ringworm infection): caused by true dermatophytes of the genus Microsporum, Epidermophyton, Trichophyton. Proto-type lesion is a ring. Pruritic. Affect skin, hair and nails. KOH smear: Mycelia. Culture on Sabouraud’s agar.

2nd. Subcutaneous and systemic fungal infection. e.g. Actinomycosis, sporotrichosis, blastomycosis.

3rd. Facultative pathogens: e.g. Candida albicans

Candida albicans is part of the resident flora of the mouth, GI tract and vagina. Predisposing factors for infection include local trauma and maceration (intertrigo), diabetes, steroids and immunosuppressive agents, oral antibiotics. Clinically: Whitish patches on oral mucosa, intertrigo with satellite lesions in body folds. Chronic paronychia. KOH smear: Mycelia and spores. Culture on Sabouraud’s agar.

I. PARASITIC INFECTIONS:

4th. Scabies: Caused by the mite Sarcoptes scabiei. Female burrows in stratum corneum. Transmission is by close and prolonged contact. Itching is worse at night. Sites of predilection are areas where the skin is soft and thin. Penile papules are helpful in diagnosis. Secondary bacterial infection due to scratching.

5th. Pediculosis (lice): Three types exist:

1. Pediculosis capitis: Louse lives on scalp. Eggs are attached to hair (nits). Itching and secondary infection. Skin lesions on posterior part of neck.

2. Pediculosis corporis: Louse lives and deposits eggs in seams of clothes. It comes upon the body for feeding. Lesions consist of hemorrhagic points and erythematosus macules, and linear scratch marks.
3. Pediculosis pubis (crab louse). Usually transmitted by sexual contact. Affects groin, hypogastrium, axillae and eyelashes. Itching may be marked.

6th. Insect bites: Common during spring and summer and on exposed sites. Variable in morphology depending on kind of insect and immunologic background of individual. Proto-type lesion is an erythematous macule or plaque with a central punctum. Urticarial lesions, vesicles and bullae may occur.

I. VASCULAR REACTIONS:

The reaction pattern consists of a spectrum ranging from simple dilatation to inflammation and necrosis of the vessel wall.

Classification according to degree and depth of injury:

1. Simple dilatation causing erythema.
2. Dilatation with seepage of fluid resulting in an erythematous elevated lesion, which has a blanched centre (weal). Classical example is urticaria. Localized swelling may occur, called angioedema, usually eyes and lips.
3. Extravasation of erythrocytes, e.g. purpura.
4. Inflammation around vessels, e.g. erythema multiforme.
5. Inflammation of vessel wall without necrosis:
   i. Vessels only, e.g. allergic vasculitis (hypersensitivity angiitis)
   ii. Vessels and surrounding fat, e.g. erythema nodosum and erythema induratum.
6. Inflammation of vessel wall with necrosis, e.g. panarteritis nodosa.

The causes are varied. They range from food items such as in urticaria to underlying neoplasms. Infections of all kinds, drugs, inhalants, and autoimmune factors such as in connective tissue disorders, all may be causative factors.
II.  PAPULOSQUAMOUS DISORDERS:

Psoriasis, lichen planus, pityriasis rosea.

These diseases are characterized by scaly erythematous papules.

1. Psoriasis: Genetic disorder. Several clinical expressions. Most common is psoriasis vulgaris which affects 2% of the population. Primary lesion in psoriasis vulgaris is a well-demarcated erythematous papule or plaque, covered by silvery scales. Usually affects extensor surfaces. Nails may be affected (commonly pitting and onycholysis). Isomorphic response may occur following trauma.

   Pathophysiology: Accelerated epidermal turnover rate. Immune mechanisms may be causing an ongoing autoreactive immune response.

2. Lichen planus: Unknown etiology. Lichen planus-like eruption may be caused by drugs. Primary lesion is a violaceous lichen, sometimes with whitish streaks (striae) over surface. Affects skin, mucous membranes, nail, and hair. Itching is a fairly consistent feature.

   Distribution of lesions: inner wrists, lumber region, shins, scalp, and glans penis. Actinic lichen planus occurs on sun-exposed sites.

3. Pityriasis rosea: A self-limited disease, thought to be viral in origin, commonly seen in early spring and later summer (early autumn). Lasts about six weeks. Primary lesion is an erythematous, oval scaly macule. The rash is preceded by a large, usually annular lesion with a scaly border resembling ringworm infection, called the “herald patch”. The rash is symmetrical and affects mainly the trunk and proximal parts of the extremities. The lesions are oriented in the planes of cleavage running parallel to the ribs. The rash is usually asymptomatic.

   The differential diagnosis includes secondary syphilis and drug eruptions.

III. ACNE VULGARIS:

Acne vulgaris is a chronic inflammatory disorder of the pilosebaceous follicle associated with seborrhea. Genetically determined. Onset is at puberty.

Lesions are distributed over the face, neck, upper trunk, and proximal part of the upper extremities where sebaceous glands are largest and most numerous.

Hormones that influence the sebaceous gland:

1. Testosterone: Increase size and secretion of gland.
2. Estrogen: Decrease size and secretion of gland.
3. Progesterone:
   a. Physiologic amounts: no effect

Characteristics of the acne follicle:

Abnormal keratinization of lining of sebaceous duct and follicle with resultant comedo formation and plugging. Hyperplastic sebaceous gland.

The comedo (or comedone) is the initial acne lesion. Plugging leads to rupture of hair follicular epithelium with resultant inflammation leading to the formation of papules, pustules, nodules, and cysts. The mechanism of comedo formation is disputed but is probably influenced by sebum and free fatty acids derived from sebum triglycerides under the effects of lipase enzyme.

IV. HAIR DISORDERS:

7th. Non-scarring hair loss (non-cicatricial alopecia): No destruction of hair follicle. Regrowth may occur.

1. Alopecia areata: Sudden hair loss from the scalp or other body sites. Usually localized but may be generalized. May be associated with autoimmune disorders such as thyroid diseases, vitiligo, diabetes. The affected site is normal with no signs of inflammation, scaling or scarring. Spontaneous re-growth takes place after 2-6 months in the majority of cases. Poor prognostic signs: Extensive and widespread alopecia, repeated attacks, involvement of occipital region (ophiasis), and nail changes.

2. Androgenetic alopecia: Androgenetic stimulation of hair follicles predisposed to this response by interdependent influences of genetic factors and of aging. The initial stage in the condemned follicles is probably the accumulation of 5-alpha-dehydrotestosterone, the tissue-active androgen which inhibits the metabolism of such follicles. The earliest changes is a reduction in the duration of anagen and an increase in telogen follicles which become vellus-like and undergo atrophy. Males usually show bitemporal recession and vertex thinning. On the other hand females usually show diffuse thinning.

3. Telogen effluvium: In normal adults 80-90% of hair follicles are in anagen or growing phase of hair cycle. Telogen effluvium consists of premature precipitation of anagen follicles into telogen (resting phase) which results in increased daily hair loss (normal is
less than 100 hairs per day). Hair loss occurs diffusely throughout the scalp and includes the sides and back of the head. Causes: Different types of stress such as fever, difficult childbirth, hemorrhage, “crash” dieting, and emotional stress. Prognosis: Complete regrowth of hair usually occurs.

8th. Scarring hair loss (cicatricial alopecia): Destruction of hair follicle. No regrowth. A variety of causes including physical and chemical trauma, some cutaneous diseases such as cutaneous lupus erythematosus, lichen planus, localized scleroderma (morphea), and neoplasms. Some cases are idiopathic.

I. MELANIN PIGMENTARY DISORDERS:

A. Hypo - and de-pigmentation


2. Vitiligo: It is a genetic disease and most probably has an autoimmune etiology. It may be associated with autoimmune disorders such as thyroid diseases, pernicious anemia, Addison's disease, diabetes and mellitus, alopecia areata. Histologic examination shows a marked absence of melanocytes. Localised, scattered or confluent depigmented macules, more over friction areas are seen. May become generalised. Course is protracted.

3. Idiopathic guttate hypomelanosis: Unknown etiology, however sun may be playing a role. Commonly present on the legs and to a lesser extent on the forearms. Lesions consist of porcelain-white macules, usually 2-6 mm in diameter. Persistent.

4. Post-inflammatory: Usually hypopigmented macules following of resolution of areas of inflammatory skin disease such as eczema and psoriasis. Clear with time.

B. Hyperpigmentation:

1. Freckles (ephilides): Frequent in individuals with red or blonde hair and blue eyes. With the light microscope the only abnormality detectable is an increase in the quantity of melanin in the epidermis. Freckles appear on light-exposed skin. They increase in number, size and depth of pigmentation during summer.
2. Neurofibromatosis (von-Recklinghausen's disease): The cutaneous features in this disease include macular hyper-pigmentation (cafe-au-lait patches) and neurofibromata. Cafe-au-lait marks are present in 90% of cases. They are present in 10% of normal subjects. The presence of one or two are not diagnostic in the absence of other signs of the disease, but if six or more are present the probability of neurofibromatosis is high. In 20% of cases, freckle-like pigmented macules are present in the axillae. Pigmented hamartomas of iris (Lisch nodules).

3. Melasma (Choasma): A blotchy hypermelanosis of exposed areas, most often on the face, mainly cheeks, forehead and moustache area, usually in women. Commonly seen in pregnancy. A similar type of pigmentation may be induced by oral contraceptives. Photosensitization to cosmetics especially perfumes may play a role.

4. Post-inflammatory: Commonly follows acute or chronic inflammatory processes. The intensity and persistence of the hypermelanosis are greater in dark-skinned subjects. Disorders where there is disruption of the basal cell layer of the epidermis such as lichen planus frequently develop areas of slate brown hypermelanosis.