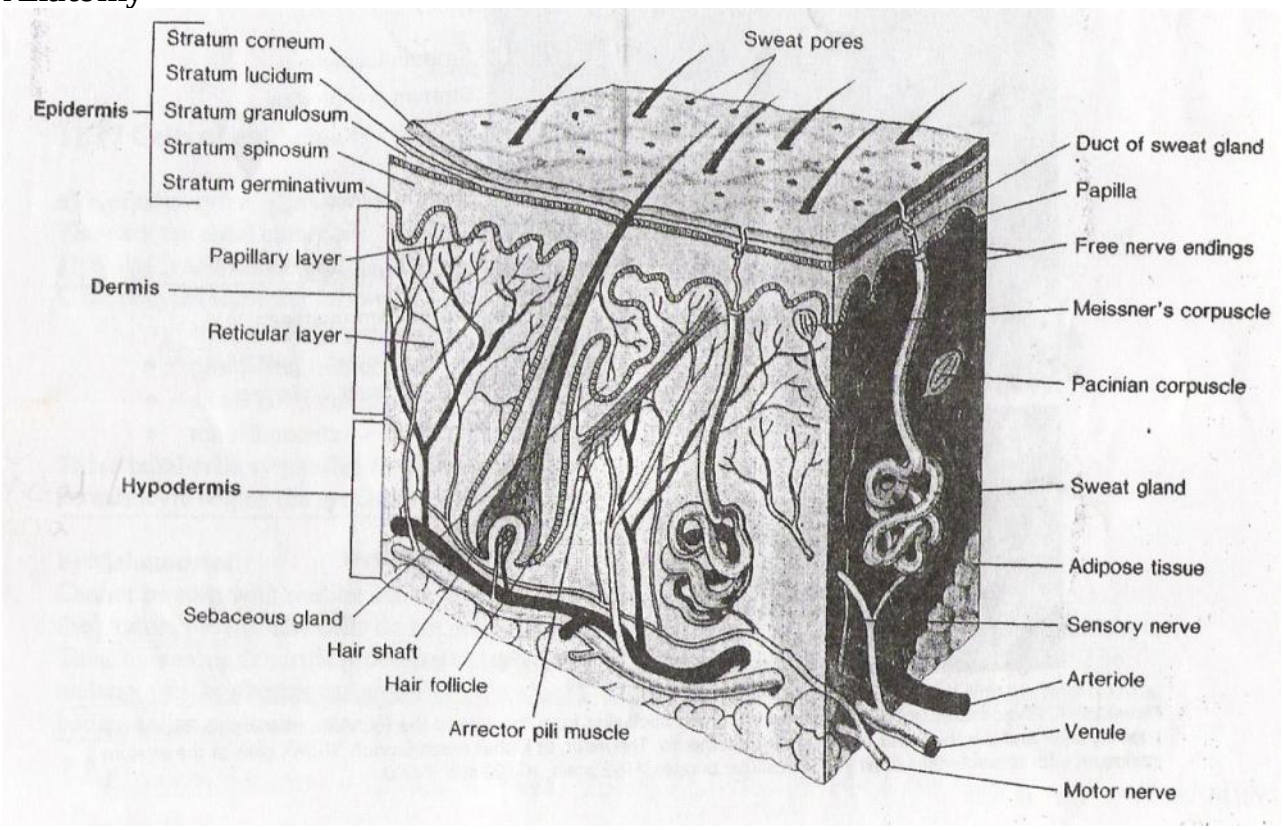
General

Monday, April 11, 2005
3:36 PM

Anatomy



The skin is the largest organ of the body;

* Covers about **1.5-2m²** on average in an adult.
* **16%** total body weight

Functions;

- Protective;
  - Micro-organisms
    * Stratum corneum (physical barrier)
    * Langerhan's cells, Mast cells & lymphocytes
  - UV protection - Melanin
  - Fluid loss
- Regulation of body temperature
  - Heat loss
    * Evaporation of sweat
    * Exposure of peripheral blood vessels
  - Heat retention
    * Erection of hairs
    * Fatty layer of skin
- Sensory organ
- Excretory organ
- Synthesis of Vitamin D
- Facilitation of drug administration & other biochemical reactions
- Cosmetic purposes
- Facilitation of movement of various body parts

Classification of skin diseases & disorders

- Infections & infestations
  - Bacterial
    - Systemic
      - Leprosy
      - Tuberculosis
      - Syphilis
    - Localised (superficial)
      - Impetigo
      - Eccthyma
      - Folliculitis
    - Localised (deep)
      - Cellulitis
      - Erysipelas
      - Boils, Carbuncles
      - Abscesses
  - Viral
    - Measles
    - Herpes - HSV, VZV
    - Molluscum contagiosum
    - HPV
  - Fungi & Yeasts
    - Superficial (Dermatophytes)
      - Tinea
      - Candida
      - Pityriasis versicolor
    - Deep (mycetoma)
      - Chronoblastomycosis
      - Actinomycosis
      - Histoplasmosis
  - Protozoal
    - Trypanosomiasis
    - Leishmaniasis (cutaneous)
  - Helminthic
    - Onchocerciasis
    - Intestinal worms
    - Dracunculus medinensis (Guinea worm)
  - Arthropodal
    - Scabies
    - Pediculosis
    - Tungiasis
    - Fleas
  - Nutritional
    - Vitamin A deficiency
    - Pellagra
    - Scurvy
    - Kwashiakor
  - Neoplastic
    - SCC
    - BCC
    - Melanoma
    - KS
  - Blistering/Bullous conditions
    - Erythema multiforme/SJS/TEN
    - Pemphigus
  - Inflammatory
    - Eczema
    - Psoriasis
    - Acne vulgaris/nodular/cystic/keloidalis
    - Pityriasis rosea/alba
    - Lichen simplex chronicus/planus/nitidus
  - Connective tissue disorders
    - SLE, Discoid lupus
    - Sclerosis
    - Cervical spondylitis
  - Disorders of hair
    - Alopecia areata
    - Trichiasis
    - Pseudosycosis barbae
    - Trichotillomania
  - Disorders of nail
    - Onycholysis
    - Onychogryphosis
    - Pitting, ridging & colour changes
    - Dystrophy
  - Pigmentation disorders
    - Vitiligo
    - Piebaldism
    - Post-inflammatory hypopigmentation
    - Genetic
      - Albinism
      - Itcthyosis
      - Neurofibromatosis
      - Harlequin baby syndrome
  - Allergic conditions
    - Urticaria
    - Drug eruptions
    - Food allergy
    - Perioral dermatitis
  - Traumatic
    - Cut wounds
    - Ulcers
    - Burns
    - Dermatitis artefacta
    - Ingrown toe nails
    - Cones, calluses
  - Growths (lumps & bumps) on the skin
    - Naevus
    - Lentigos
    - Lipomas
    - Kelloids
    - Actinic keratosis
  - Miscellaneous
    - Dermatomyositis
    - Mycosis fungoides
    - Lichen sclerosus

Important points in History Taking - describing the lesions

- **S**ite - Initially up to today (localised, regionalised, generalised, universal/global)
- **S**ize
- **S**hape & Margins (discrete, indiscrete, active)
  - Nummular - Discoid or coin-shaped
  - Herpetiform - resembling herpes (an eruption of groups of deep-seated vesicles on erythematous bases)
  - Verrucous - wartlike elevations
  - Morbilliform -resembling measles (generalized maculopapular eruption of a dusky red colour)
- **S**urface
- **C**olour;
  - Depigmented - No melanocytes
  - Hypopigmented - Melanocytes present but reduced production of melanin
  - Hyperpigmented
  - Erythematous - Redness of the skin arising from oxygenated hemoglobin and is usually indicative of increased blood flow through the upper dermis due to capillary dilatation.
- **C**onsistency
- **P**attern (dermatomal, vascular, lymphatic, linear, reticular)
- **O**nset
- **C**haracter
  - Pruritus
  - Erythema
  - Painful - character of pain
- **R**adiation
- **A**ssociations
  - Systemic manifestations
  - Pruritus
  - Drug history
  - Hypersensitivity to drugs & chemicals
  - Occupation
  - Atopy
  - Origin & history of travel
  - Previous lesions
- **T**imings
- **E**xcercebators & Alleviators
- **S**everity

SKIN LESIONS

Primary lesions - Early skin changes that have not yet undergone natural evolution or change caused by manipulation or trauma.

- **Macule-** an alteration in skin colour but cannot be felt. **>1 cm patch**.
- **Papules** are palpable solid lesions **<0.5cm**, whereas **nodules** are **>0.5cm** & **have a deep component** (pathology in the dermis)**. Tumours** are usually larger than nodules and vary considerably in mobility and consistency. **Plaques**- palpable flat topped lesions **>0.5cm** & **lack a deep component** (pathology in the epidermis) or a group of confluent papules.
- **Vesicles** are raised, fluid-filled lesions **<0.5cm** in diameter; when larger, they are called **bullae**.
- **Pustules** contain purulent material.
- **Wheals/Hive** are flat-topped, pruritic palpable lesions of variable size and configuration that represent dermal collections of oedema fluid.
- **Cysts** are circumscribed, thick-walled lesions that are located deep in the skin; are covered by a normal epidermis; and contain fluid or semisolid material.

Secondary lesions - result when primary lesions undergo a natural evolution or are manipulated by the patient

- **Scales** consist of compressed layers of stratum corneum cells that are retained on the skin surface.
- **Erosion** is focal loss of part or all of the epidermis & heals without scarring.
- **Ulcers** are full thickness (dermis + epidermis) excavations of necrotic or traumatized tissue. Ulcerated lesions inflicted by scratching, rubbing, or picking are often linear or angular in configuration and are called **excoriations**.
- **Fissures** are vertical loss of both epidermis & dermis with sharply defined walls caused by splitting or cracking; they occur usually in diseased skin.
- **Crusts** consist of matted, retained accumulations of blood, serum, pus, and epithelial debris on the surface of a weeping lesion.
- **Scars** are end-stage lesions that can be thin, depressed and atrophic, raised and hypertrophic, or flat and pliable; they are composed of fibrous connective tissue & represent prior disruption of the **epidermal-dermal junction**
- **Lichenification** is a thickening of skin with accentuation of normal skin lines that is caused by chronic irritation (rubbing, scratching) or inflammation.

Other skin lesions

- **Telangiectasia** - Dilation of the previously existing small or terminal vessels of a part that **blanch** with pressure.
- **Petechiae** - Minute hemorrhagic spots **<0.5cm**, of pinpoint to pinhead size, in the skin, which are **not blanched** by **diascopy** (Examination of superficial skin lesions with a flat glass plate through which one can examine superficial skin lesions by means of pressure) & are 2° to extension of red cells in dermis
- **Erythema** - Redness of the skin arising from oxygenated hemoglobin and is usually indicative of increased blood flow through the upper dermis due to capillary dilatation. **Blanch** on pressure
- **Purpura** - A condition characterized by haemorrhage into the skin **>0.5cm**. Appearance of the lesions varies with the type of purpura, the duration of the lesions, and the acuteness of the onset. The colour is first red, gradually darkens to purple, fades to a brownish yellow, and usually disappears in 2 or 3 weeks; colour of residual permanent pigmentation depends largely on the type of unabsorbed pigment of the extravasated blood; extravasations may occur also into the mucous membranes and internal organs. Does **not blanch** on pressure
- **Ecchymosis** - A purplish patch **>2cm** caused by extravasation of blood into the skin, differing from petechiae only in size
- **Burrow** - A superficial epidermis tunnel or tract made by a parasite, such as the itch mite.
- **Comedo** - A dilated hair follicle infundibulum filled with keratin squamae, bacteria, particularly **Propionibacterium acnes**, and sebum; the primary lesion of acne vulgaris.
- **Kobner's phenomenon (**isomorphic reaction**)** lesions in **psoriasis**, **lichen planus**, **vitiligo** and **flat warts** mimic the shape of trauma to the skin (e.g., from scratching, rubbing, or other injury).

Vitiligo

An **autoimmune** reaction to preexisting melanocytes causing slowly progressive depigmenting condition in small or large areas of the skin due to the disappearance of previously active melanocytes.

Classification;

- Type A is **non-dermatomal** and widespread. It represents 75% of cases
- Type B is **dermatomal or segmental**. It represents the remaining 25% of cases

S/S;

- Loss of pigment
- Locally increased sunburning
- Predilection for acral areas and around orifices such as eyes, mouth, anus
- Pruritus (10%)
- Premature graying (35%)
- Koebner's phenomenon (aggravation by trauma)

Risk factors;

- Positive family history
- Autoimmune disorders including hemolytic anemia and adrenal insufficiency

DDx;
Any condition that causes acquired hypomelanosis, including;
- Tinea versicolor
- Leprosy
- Lupus erythematosus
- Pityriasis alba
- Steroid exposure

Ix;
- The depigmentation of vitiligo can be differentiated from hypopigmented lesions by its **ivory-white** colour under **Wood's light**.
- Skin scraping and a potassium hydroxide (KOH) preparation can be examined microscopically to rule out tinea versicolor

Treated by;
- **Camouflage cosmetics** & **sunscreens** with a sun protective factor (SPF) of > 15 (± steroid creams ± dermabrasion).
- PUVA

Icthyosis
Congenital disorders of keratinization characterized by noninflammatory dryness and scaling of the skin, often associated with other defects and with abnormalities of lipid metabolism.
Secondary causes;
- Malignant diseases (e.g., Hodgkin's disease, lymphosarcoma)
- Paraneoplastic syndromes
- Autonomic neuropathy especially in HIV
- Leprosy
- Severe nutritional deficiencies

Scleroderma
A chronic disease of **unknown cause,** characterized by diffuse fibrosis; degenerative changes; and vascular abnormalities in the skin (**scleroderma**- thickening and induration of the skin caused by new collagen formation, with atrophy of pilosebaceous follicles and adhesion to underlying tissues (especially of the **hands** and **face**), articular structures, and internal organs (especially the esophagus, GI tract, lung, heart, and kidney).
M:F - **1:4**

Clinical forms;
- **Localized** forms of scleroderma (**morphea**);
  - **Linear sclerosis** of the integument and immediately subjacent tissues without systemic involvement. Circumscribed patches; No cure; monitor every **6mo**; Plastic surgery may be used to correct disfiguration
- **Mixed connective tissue disease** - combines features of scleroderma (eg, Raynaud's phenomenon, esophageal dysfunction) with clinical and serologic features of **SLE, polymyositis, or RA.**
  - **Limited cutaneous scleroderma** (**CREST syndrome**) with restricted skin involvement (often just the **fingers** and **face)** and **slow progression**, often several decades, before full manifestation of characteristic internal involvement.
    * **C**alcinosis - The abnormal deposition of calcium salts in a part or tissue of the body
    * **R**aynaud's phenomenon - A vascular disorder that is marked by recurrent spasm of the capillaries & especially those of the fingers & toes upon exposure to cold, that is characterized by pallor, cyanosis & redness in succession usually accompanied by pain, & that in severe cases progresses to local gangrene. Precedes onset of other symptoms
    * **E**sophageal dysfunction with dysphagia due to loss of peristalsis and submucosal fibrosis of the esophagus
    * **S**clerodactyly - insidious swelling of the acral (peripheral) portions of the extremities, with gradual thickening of the skin of the fingers.
    * **T**elangiectasia

- **Systemic sclerosis** with **diffuse scleroderma** - generalized cutaneous thickening which may cause **rapidly progressive** and often fatal visceral involvement
    * **Dysphagia** due to loss of peristalsis and submucosal fibrosis of the esophagus
    * **Dyspnea** due to pulmonary fibrosis
    * **Myocardial fibrosis**
    * **Renal vascular changes** resembling those of malignant hypertension
    * Raynaud's phenomenon
    * Atrophy of the soft tissues
    * Osteoporosis of the distal phalanges (**acrosclerosis**), sometimes with gangrene at the ends of the digits, are common findings.
    * Poikiloderma - variation in skin pigmentation

Ix
Rheumatoid factor tests are positive in 33% of systemic sclerosis patients, and serum ANA are present in ≥ 90%

Mx
- No drug has significantly influenced the natural history of systemic sclerosis
- **Corticosteroids** may be helpful for disabling **myositis, synovitis, or MCTD**
- Prolonged oral administration (> 1.5 yr) of **penicillamine** can reduce **skin thickening** and may delay the rate of new visceral involvement.
- **Nifedipine** 20 mg tid or as tolerated may help control **Raynaud's phenomenon.**
- **Reflux esophagitis** is relieved by **frequent small feedings, antacids, and H2 blockers or with proton pump inhibitors and by having the patient sleep with the head of the bed elevated. Esophageal strictures** may require **periodic dilation**; successful correction of gastroesophageal reflux by gastroplasty has been reported. **Tetracycline** 1 g/day po or another broad-spectrum antibiotic suppresses overgrowth of intestinal flora and may alleviate intestinal malabsorption symptoms caused by bacterial colonization of dilated bowel loops. Physiotherapy may help preserve muscle strength but is ineffective in preventing joint contractures.
- For **renal disease**, **ACE inhibitors** are the drugs of choice. Other vasodilators (eg, minoxidil) also have been used with some success. All of these drugs effectively control hypertension and help preserve renal function. When end-stage renal disease is unpreventable, dialysis and transplantation can be used, although the mortality rate remains high.

Pyoderma gangrenosum

Chronic noninfectious condition that is marked by the formation of **purplish nodules & pustules which tend to coalesce & form ulcers with a necrotic base** & that is associated with;
- Idiopathic in **50%**
- Ulcerative colitis
- Malignancy e.g. leukaemia, metastatic adenocarcinoma of the intestine
- Rheumatoid arthritis

These lesions commonly occur on the **lower extremities**, but may present anywhere on the skin.
Management of PG may require aggressive treatment such as **high-dose oral corticosteroids**, pulse corticosteroids, dapsone, azathioprine, cyclosporine A, clofazimine, or even resection of the severely inflamed bowel.

Dermatitis herpetiformis

Chronic disease of the skin marked by a symmetric **itching** eruption of **vesicles and papules** that **occur in groups resembling hives typically in clusters on an erythematous base** & usually in the **knees** & **elbows** & over the **sacrum** & **scapula**; relapses are common; associated with **gluten-sensitive enteropathy** (**coeliac disease**) and **IgA immune complexes** beneath the epidermis of lesioned and normal-appearing skin.
Remarkably responsive to **dapsone** therapy usually within 24hrs of the first dose.

Molluscum contagiosum

A contagious disease of the skin caused by intranuclear proliferation of a virus of the family **Poxviridae** and characterized by the appearance of few to numerous **small, pearly, umbilicated papular expansible epidermal down growths** that contain numerous cytoplasmic inclusion bodies (m. bodies).
Curettage or **cryotherapy** are treatments of choice.

Nevus

A circumscribed malformation of the skin, especially if colored by hyperpigmentation or increased vascularity; a nevus may be predominantly epidermal, adnexal, melanocytic, vascular, or mesodermal, or a compound overgrowth of these tissues. It also describes a benign localized overgrowth of melanin-forming cells of the skin present at birth or appearing early in life.
Keratinolytic agents can be used for temporary relief
Because moles are extremely common and melanomas are uncommon, prophylactic removal is not justified. However, a mole should be excised and examined histologically if;

* it enlarges suddenly (especially with an irregular border)
* darkens or becomes inflamed
* shows spotty color changes
* begins to bleed, ulcerate, or itch
* becomes painful.

If the mole is too large for simple excision, a biopsy should be deep enough for accurate microscopic diagnosis, which must be obtained before wide primary excision because many lesions are clinically misdiagnosed as melanomas. Simple excision or biopsy does not increase likelihood of metastasis if the lesion is malignant, and it avoids extensive surgery for a benign lesion. Moles can be removed for cosmetic purposes without fear of subsequent malignant change, but all moles removed should be examined histologically.

Stevens-Johnson Syndrome & Toxic Epidermal Necrolysis

SJS is a generalized hypersensitivity reaction, usually to a drug, in which the skin and mucus membrane lesions are early manifestation. It may progress to its more severe form, **toxic epidermal necrolysis** which has a high morbidity and up to **40% mortality.**
The condition is classified as **SJS** if **epidermal detachment** affects **<10%** of the skin, as **toxic epidermal necrolysis** (TEN) if **epidermal detachment > 30%**, **or** if it **> 10%** in the **absence of discrete skin lesions**. Cases with discrete skin lesions and between 10% and 30% epidermal detachment are in the overlap between SJS and TEN.
Associated drugs;

- Sulfonamides
- Barbiturates
- NSAIDs
- Phenytoin
- Allopurinol
- Penicillin

The disease typically begins with painful localized erythema that disseminates rapidly. At the sites of erythema, flaccid blisters occur or the epidermis peels off in large sheets with gentle touching or pulling (**Nikolsky's sign**). Malaise, chills, myalgias, and fever accompany the denudation. Widespread areas of erosion, including all mucous membranes (eyes, mouth, genitalia), occur within **24 to 72 h**, and the patient may become gravely ill. Affected areas of skin often resemble second-degree burns. Death is caused by fluid and electrolyte imbalance and multiorgan sequelae (eg, pneumonia, GI bleeding, glomerulonephritis, hepatitis, infection).
Rx - Administration of steroids is controversial. If there is no clear benefit within a few days, they should be withdrawn.

Staphylococcal Scalded Skin Syndrome (RITTER-LYELL SYNDROME)

Acute, widespread erythema and epidermal peeling caused by staphylococcal exotoxin which almost always occurs in infants, children < 6 yr old, and immunosuppressed adults or adults with renal failure.

Etiology

Group II coagulase-positive staphylococci, usually phage type 71 and often resistant to penicillin, elaborate **exfoliatin** (also called **epidermolysin**), an epidermolytic toxin that splits off the upper part of the epidermis just **beneath the granular cell layer**. The inciting infection may be on the **skin** but usually is in the **eye** or **nasopharynx**. The toxin enters the circulation and affects the skin systemically, as in scarlet fever.

Differentiating feature	SSSS	TEN
Patient affected	Infants, young children, immunosuppressed adults	Older patients
Patient history	Recent staphylococcal infection, renal failure	Drug use
Level of Epidermal cleavage (Blister formation)*	Within the granular cell (outermost) layer of the epidermis	Between the epidermis & dermis or at the level of the basal cell

* Determined by the Tzanck test or by a frozen section of a fresh specimen

DDx (but none of these causes a painful rash)

- Drug hypersensitivity (most notably, TEN)
- Viral exanthemas
- Scarlet fever
- Bullae, erosions, and an easily loosened epidermis occur in;
    - Thermal burns
    - Genetic bullous diseases (eg, some types of epidermolysis bullosa)
    - Acquired bullous diseases (eg, pemphigus vulgaris, bullous pemphigoid).

Mx

- Systemic penicillinase-resistant antistaphylococcal antibiotics (eg, cloxacillin, dicloxacillin, cephalexin) must be started as soon as the clinical diagnosis is made, without waiting for culture results
- Corticosteroids are **contraindicated,** and topical therapy and patient handling must be minimized.

Localised (superficial) Bacterial Infections

- **Impetigo** - An acute contagious staphylococcal or streptococcal skin disease characterized by vesicles, pustules, & yellowish crusts
- **Eccthyma** - a cutaneous eruption marked by large pustules that have a hardened base surrounded by inflammation, heal with pigmented scar formation, & occur especially on the lower legs
- **Folliculitis** - inflammation of one or more follicles especially of the hair

Pityriasis

A dermatosis marked by **branny (dandruff) desquamation.**

- **Pityriasis alba**, patchy hypopigmentation of the skin resulting from mild dermatitis.
- **Pityriasis capitis** - dandruff.
- **Pityriasis rosea**, a self-limited eruption of macules or papules involving the trunk and less frequently extremities, scalp, and face; the lesions are usually oval and follow the crease lines of the skin (**Christmas tree pattern**); the onset is frequently preceded by a single larger scaling lesion known as the **herald patch**.
- **Pityriasis rubra** - exfoliative dermatitis.
- **Pityriasis rubra pilaris** - an uncommon chronic pruritic eruption of the hair follicles, which become firm, red, surmounted with a horny plug, and often confluent to form scaly plaques; it is most conspicuously noted on the **dorsa of the fingers and on the elbows and knees**, and is associated with

erythema, thickening of the palms and soles, and opaque thickening of the nails.

Psoriasis

Monday, April 11, 1994
9:54 PM

Papulosquamous disease

Epidemiology
Two peaks of onset, age **25-30** and age **55-60**; can develop in infants; Male = Female

Description
A common **chronic, recurrent epidermal proliferative** disease characterized by **dry**, **erythematous** well-circumscribed, **silvery** (due to lack of melanin due to the reduced transit time of the keratinocytes)**, scaling papules** and **plaques** of various sizes.

Aetiology
- Also ? **Immune mediated** inflammatory disease
- Possible **genetic error in mitotic control**. Activation of lymphocytes (antigen? autoimmune?). Epidermal cell cycle 10 times shorter than normal, leading to epidermal hyperproliferation.

Pathogenesis
Basal cells in normal skin take **14days** to keratinize & migrate to the top & last another **14days** at the top before exfoliation.
In Psorioasis, there is a marked increase in the epidermal cell proliferation rate with a corresponding reduction in the transit time of keratinocytes through the epidermis (**2-7days**).
Pathological findings;
- Parakeratosis (focal) (Retention of nuclei in the cells of the stratum corneum of the epidermis, observed in many scaling dermatoses such as psoriasis and subacute or chronic dermatitis), especially with neutrophils
- Hyperkeratosis - Thickening of the stratum corneum of the epidermis or mucous membrane.
- Hypogranulosis
- Acanthosis - An increase in the thickness of the stratum spinosum of the epidermis
- Elongation and thickening of rete ridges
- Dilated tortuous capillary loops
- Munro's microabscess - Aggregates of neutrophils in the stratum corneum due to production of neutrophilic cytokines
- Thin epidermis above dermal papillae
- Spongiform pustule of Kogoj
- Abnormal mitoses
- "Squirting" papillae

Clinical forms:
- **Discoid or plaque psoriasis** - most common, patches appear on scalp, trunk and limbs, nails may be pitted and/or thickened
- **Guttate psoriasis** - occurs most frequently in children, numerous small papules over wide area of skin, but greatest on the trunk; also following infections in genetically predisposed persons
- **Pustular psoriasis** - small **sterile intraepidermal** pustules filled with neutrophils (2° to increased chemokine production) over the body or confined to one area (i.e., palms and soles) or arranged in annular patterns (especially children). Acute pustular psoriasis (Von Zumbusch's) is a pustular eruption on an erythematous base with total body distribution.
  DDx - Pyoderma
- **Inverse, flexural psoriasis** - affects the flexural areas, lesions are moist and without scales (common in older people) - **Negative** Auspitz sign
- **Ostraceous** - grossly hyperkeratotic

- **Erythroderma (exfoliative psoriasis or red man syndrome)** - is a clinical syndrome characterized by generalized or nearly total diffuse erythema of the skin accompanied by variable degrees of desquamation.
  Causes;
  - Underlying dermatoses which have generalized
    * Eczematous conditions e.g. Contact, atopic or seborrheic dermatitis
    * Psoriasis
    * Lichen planus
    * Pityriasis rubra pilaris
    * Pemphigus foliaceous
  - Drugs e.g. Systemic steroids aggravate it, Carbamazepine, Barbiturates, Phenytoin, Sulfur drugs, Isoniazid, Furosemide
  - Malignancies;
    * Hodgkin's disease
    * Miscellaneous lymphomas & leukaemias e.g. Cutaneous T cell lymphoma, Sezary syndrome, the leukaemic form of mycoses fungoides which is an indolent cutaneous lymphoproliferation occurring in adults presenting with generalized erythroderma
  - Immunosuppression
  - Idiopathic

  Death is usually due to loss of the protective function of the skin;
  * Hypothermia 2° to vasodilatation
  * Dehydration (7times more loss)
  * Infections
  * Protein loss due to scaling (normally <10 gm but in psoriasis >100gm)
  * Oedema 2° to protein loss
  * Hyperdynamic cardiac failure 2° to shunting of blood in skin

S/S

- **Psoriatic arthropathy** occurs in about **5%** of individuals with psoriasis, especially those with psoriatic nail disease. It is a seronegative spondyloarthropathy i.e. Rheumatoid factor negative
  Characteristics;
  - Nail pitting
  - Transverse depressions
  - Subungual (below finger or toe nail) hyperkeratosis
  - Distal interphalangeal arthritis.

  Also, **Arthritis mutilans** - a destructive, resorptive arthropathy with shortening of the distal phalanges. Produces the so-called opera-glass hand.
- Pruritus
- Silvery scales on red oval plaques
- Distributed on the **extensor surfaces** of the **knees** & **elbows**& on the **sacrum** & **scalp** (due to koebner phenomenon 2° to combing hair (trauma))
- Nail changes - **pinhead-sized pits, onycholysis**, and a **red-brown discoloration** resembling a "drop of oil"- due to keratin degeneration
- Positive **Auspitz sign** - Underlying pinpoints of bleeding following scraping due to;
  * Hypervacularity of the lesions
  * Dilated tortuous dermal capillary loops
  * Elongation and thickening of rete ridges
- **Koebner's phenomenon** (psoriatic response in previously unaffected area 1-2 weeks after skin injury)

Risk/Aggravating/Precipitating factors

- Local trauma; local irritation
- Infection (streptococcal pharyngitis can stimulate acute guttate psoriasis, HIV)
- Immunosuppression
- Endocrine changes
- Stress (physical and emotional)
- Drugs (lithium, ACE inhibitors, beta-adrenergic blockers, tetracycline, NSAID's, amiodarone, morphine, procaine, potassium iodide, salicylates, sulfapyridine, sulfonamides and penicillin. Pustular flares may occur with steroids). Also antimalarial medications (aminoquinolone compounds)
- Alcohol use
- Obesity

Ix

- Negative rheumatoid factor
- Latex fixation test
- Leukocytosis and increased sedimentation rate often seen, especially in pustular psoriasis
- Fungal studies - may show a superimposed infection
- Uric acid increases in 10-20%
- In severe cases, anaemia, B12, folate and iron deficiency can be present
- Biopsy

DDx

- Trunk - pityriasis rosea, pityriasis rubra pilaris, tinea corporis
- Lichen planus
- Mycosis fungoides
- Nails - onychomycosis
- Scalp - seborrheic dermatitis

Mx

- Supportive therapy;
  - · Outpatient usually
  - · May require inpatient for severe or resistant cases based on the **Psoriasis area & severity index**
  - · Solar radiation
  - · Mild disease - ultraviolet radiation (UVA, UVB)
  - · Medication to soften scale, followed by soft brush while bathing
  - · Oatmeal baths for itching
  - · Tar shampoos
  - · Avoid excessive sun exposure
  - · Desert climates provide a favourable effect for some patients
  - · Wet dressings may help relieve pruritus
  - · For extensive, recalcitrant psoriasis, a referral to a specialist in psoriatic therapy is suggested
- Definitive therapy;

Mild disease - Low PASI

- **Anthralin ointment 0.1% - Short Contact Anthralin Therapy (SCAT)** - applied for 10 minutes, then washed off, useful adjunctive treatment. Use prior to ultraviolet light (UVA, UVB). Indicated for quiescent or chronic psoriasis, **contraindicated in acute or actively inflamed psoriatic eruptions**. Irritates unaffected skin, protect areas with zinc oxide or petrolatum. Avoid face, eyes, mucous membranes.
- **Coal tar** (Estar, PsoriGel)

Severe disease - High PASI

- **Vitamin D analogs** (calcipotriene ointment 0.005% for moderate plaque psoriasis). Too irritating for facial lesions. Watch for hypercalcemia. Associated with little or no tachyphylaxis.
- **Retinoids**
  - * Etretinate (Tegison) - especially **pustular and erythrodermic psoriasis** disease not responsive to standard treatments. Remains in body up to 2 years after treatment. Extremely fetotoxic. Do not use if pregnant or could become pregnant within 2 years of treatment. **Safe for use on face.**
  - * Isotretinoin: may work on some patients

- **PUVA** (psoralen plus ultraviolet light) - very effective, but causes skin-aging, cataracts and increases risk of skin cancers
- **Methotrexate**; Also cyclosporine, though not common.

Complications

- Pustular psoriasis
- Exfoliative erythrodermatitis
- Hypercalcemia with excessive calcipotriene

Age related factors

Pediatric:

- Onset common <10, rarely <3
- Disease may be atypical in its course

Geriatric:

- About 3% of psoriasis patients acquire the disease after age 65
- Detailed drug history important, since many drugs (e.g., beta-blockers) can exacerbate psoriasis
- If using cytotoxic medications for treatment of psoriasis, closely follow hepatic and renal functions, and creatinine clearance
- Elderly patients may have difficulty with application of topicals over all affected body parts

Pregnancy

- Unpredictable effect on disease. Avoid tars, topical corticosteroids, calcipotriene, and systemic therapies. Etretinate is fetotoxic.

Pemphigus

Tuesday, April 12, 1994
5:43 PM

Vesiculobullous Disease

Introduction
The term pemphigus refers to a group of potentially fatal autoimmune bullous diseases characterized by **intraepidermal bullae** and **extensive erosions** on apparently healthy skin and mucous membranes & that share two distinctive features:

- **Acantholysis** - detachment and rounding of adjacent epidermal cells from each other
- **Autoantibodies** against specific antigens at the site of epidermal damage.

Pemphigus vulgaris

Chronic systemic autoimmune disease

Epidemiology

M=F; **40-60yrs**

C/P

- Painful **oral erosions -** Lesions typically occur first in the mouth, where they rupture and remain as chronic, often painful, erosions for variable periods before the skin is affected.
- **Skin lesions - flaccid bullae** that rupture rapidly, leaving persistent erosions that become **crusted** and often infected. All **mucous membranes with stratified squamous epithelium** may become involved (mouth, pharynx, esophagus, nose, conjunctiva, glans, vagina, and anus). In extensive disease the **scalp** and **upper trunk** are consistently involved. **Itching** is usually **absent**.
  Nikolsky's sign positive
  * Pressure applied to an **intact bulla** leads to peripheral extension of the lesions or
  * Shearing pressure applied to **normal skin** induces bulla formation .

Ix

- **Histologic examination** of an early lesion reveals **acantholysis** in a characteristic **suprabasilar** location. Basal cells remain attached to the basement membrane. Ultrastructurally the initial site of separation of epidermal cells from each other is within nondesmosomal areas. Ultimately desmosomes split, resulting in complete acantholysis of epidermal cells.
  Tzank Preparation. This is a cytologic examination of epidermal cells. Vesicles should be ruptured and the fluid blotted away (or cultured). The base is scraped gently with a no. 15 blade and streaked onto a slide, which may be stained with methylene blue, toluidine blue, **and** Wright or Giemsa stains. Numerous single keratinocytes, such as large oval cells with abundant cytoplasm and a small nucleus, are indicative of acantholysis
- **Direct immunofluorescence (IF)** performed on normal-appearing skin adjacent to a lesion consistently reveals deposition of IgG and to a lesser degree complement components like C3 on epidermal cell surfaces.
- **Indirect immunofluorescence** tests usually show pemphigus antibodies directed against **desmoglein III** in the patient's serum & the antibody titer may correlate with disease severity.

Mx

- General;
  - Active skin infections are treated with **systemic antibiotics.**
  - Reverse isolation procedures may be required.
  - Generous use of **talc** on the patient and sheets may prevent oozing skin from adhering
  - Hydrocolloid dressings may be useful.
  - Silver sulfadiazine cream used on erosions can prevent secondary infection.
- Therapy must be aimed at clearing the pathogenic IgG autoantibodies by long-term **systemic corticosteroids** such as prednisone in a 1-2 mg/kg/day dose divided **2/3** in the morning & **1/3** in the evening
- Immunosuppressives such as **azathioprine, methotrexate,** or **cyclophosphamide plus plasmapheresis** are required for their **steroid-sparing effects**

DDx

- Bullous pemphigoid
- Staphylococcal scalded-skin syndrome.
- Drug eruptions
- Toxic epidermal necrolysis

Pemphigus foliaceus (Superficial pemphigus**)**

Presents either in a sporadic form which most commonly affects the elderly throughout the world or in an endemic form

C/P

- The primary lesion is a **superficial vesicopustule**, which may spread peripherally and then rupture, leading to the formation of **scale-crust**. Lesions tend to appear and predominate over **seborrheic areas** such as the **scalp**, **chest**, and **back**. Lesions may become secondarily infected. Unlike pemphigus vulgaris, mucosal lesions are extraordinarily rare and essentially "**never**" occur.

Ix

- **Histological examination** of an early lesion reveals acantholysis within the **granular cell layer** (upper layers of the stratum spinosum or stratum granulosum) with an occasional infiltration of neutrophils and/or eosinophils. **Tzank preparation**
- **Direct IF** examination of normal-appearing skin adjacent to a lesion reveals deposition of IgG (predominantly IgG4) and occasionally C3 on the epidermal cell surfaces.
- **Indirect immunofluorescence** tests usually show pemphigus antibodies in the patient's serum directed against **desmoglein I** & the antibody titer may correlate with disease severity.

Mx

- Most patients with pemphigus foliaceus can be controlled by a **lower corticosteroid dose** than is needed with pemphigus vulgaris. A dose in the range of 0.50 to 1.0 mg/kg/day of prednisone is usually sufficient.

  Most treated patients respond rapidly and the dose can be tapered over several months.

  The necessity to use steroid-sparing immunosuppressive agents in pemphigus foliaceus is uncommon.

DDx

- Exfoliative dermatitis
- Psoriasis,
- Drug eruption
- Other forms of dermatitis.

Pemphigoid

Wednesday, April 20, 1994
6:18 AM

Vesiculobullous Disease

Pemphigoid gestationis (Herpes gestationis (HG))

Affects pregnant women during the late second or third trimester and rarely women with ovarian tumors and secondary hormonal changes. The etiology remains uncertain and the importance of the underlying hormonal changes is unexplained. There may be a slightly increased risk of fetal morbidity and mortality, but this is controversial.

C/P

- Lesions appear first on the **abdomen** then spread to the rest of the **trunk** and **proximal extremities** and consist of **urticarial papules** and **plaques** followed by **tense vesicles** and **bullae**. Lesions cease to appear and the disease resolves within several days to a few weeks after delivery. The disease recurs during subsequent pregnancies and occasionally, in a mild degree, with menses and the intake of oral contraceptive hormones.

Ix

- **Histologic examination** of an early lesion reveals **separation of** the **epidermis from** the underlying **dermis** through the **lamina lucida** and the **lamina densa** in the **basal cell membrane** and a variable infiltrate of eosinophils and lymphocytes in the upper dermis
- **Direct IF** of perilesional skin consistently reveals intense deposition of C3 and relatively weak deposition of IgG (predominantly IgG4) at the DEJ within the lamina lucida in close proximity to the basal cell hemidesmosomes.
- **Indirect IF** reveals circulating **anti-basement membrane zone antibodies** (predominantly IgG4) in 70% of patients. These antibodies, like those of PV, bind only to stratified squamous epithelia

Mx

- **Moderate-dose systemic corticosteroid therapy**.

Bullous Pemphigoid

Chronic benign bullous eruption considered to be an **autoimmune disease**. Most frequently affects people **over 60***.

Female > Male*

S/S (***** differences between Bullous pemphigoid & Pemphigus vulgaris)

- Large bullae 2 to 5 cm in diameter. Occasional tiny peripheral vesicles.
- **Bullae that arise from erythematous skin*** (usually) or normal-appearing skin (sometimes)
- Bullae stay intact for many days
- **Located on extremities at first, trunk later***
- Occasionally located on the scalp, palms, and soles; mucous membranes (infrequently)
- **Intact blisters outnumber erosions (reverse is true with pemphigus)***
- Clear fluid fills bullae (usually) & (sometimes) blood tinged
- Itching (sometimes severe)
- Some patients are asymptomatic
- 10-20% of skin surface is continuously involved
- **Pruritus may antedate onset of blisters (weeks to months)***

Risk factors;

- Female, age over 60
- Drug associated: furosomide, phenacetin, various penicillins

Pathological findings;

- Bullae located in a **subepidermal*** location
- Light microscopy reveals **subepidermal** blister with perilesional inflammation containing many **eosinophils** and **mononuclear** cells
- Immunofluorescent studies - deposition of C3 (100%) and IgG (65-90%) in lamina lucida

Mx

Systemic Steroids

Complications

- Superimposed infection (may result in death in elderly debilitated patient)
- Complications of steroid therapy
-

Eczema (Dermatitis)

Wednesday, April 13, 2005
12:04 PM

Inflammatory disease

Generic term for inflammatory conditions of the skin, particularly with **vesiculation** in the acute stage, typically **erythematous**, **edematous, papular, and crusting**; followed often by **lichenification and scaling** and occasionally by duskiness of the erythema and, infrequently, hyperpigmentation; often accompanied by sensations of **itching** and burning;

Classification

- Chronological;
  - Acute;
    * Erythematous & edematous usually with well defined margins
    * Vesicles, papules & more rarely, large blisters; the vesicles form by intraepidermal **spongiosis** (Inflammatory **intercellular edema** of the epidermis)
    * Exudation ('Weeping') & cracking
    * Scaling
  - Subacute - histological classification; in between acute & chronic
  - Chronic
    * Less vesicular & less exudative
    * **Lichenification**, a dry leathery thickening with increased skin markings, is secondary to rubbing & scratching
    * Fissures (in pals & soles) & scratch marks
    * Pigmentation changes (hypo- & hyper-)
    * Parakeratosis (focal) - (Retention of nuclei in the cells of the stratum corneum of the epidermis,
- Aetiological;
  - Exogenous - Originating or produced outside of the body
    - Contact Irritant eczema
    - Contact allergic eczema
  - Endogenous - Originating or produced within the body; has a **genetic component**
    - Atopic
    - Seborrhoeic
    - Discoid/Nummular
    - Asteatotic
    - Gravitational/Stasis/Varicose
    - Lichen simplex
    - Pompholyx (palmar & plantar)

1. Contact Irritant Eczema

- Contact irritant dermatitis is caused by an irritant contacting the skin & **breaking down the protective keratin** of the skin and allow penetration of substances into the skin **or irritate existing dermatitis**. Susceptibility to irritants varies immensely among individuals. Irritant contact dermatitis is most frequently seen on the hands, as this tends to be the area of greatest exposure. It is seen more commonly in individuals who are constantly wetting and drying their skin, exposed to cold outdoor winds or hot indoor forced heat, and exposed to any factors that dry the skin or strip away the outermost cells of the skin or surface water-holding substances. The most common irritants are detergents, alkalis, acids, solvents & abrasive dusts.
  If a harsh chemical has been encountered, the clinical change seen at the point of contact is that of a **scalded, erythematous, moist area** with **peeling** back of the overlying epidermis, leaving a fine, lacy **scale** at the border. In cases in which a mild irritant is encountered, the acute change is that of **mild macular erythema**. If exposure becomes chronic, secondary changes of **scale** and **plaque** formation occur, again localized to the area of contact.

Rx

- Eliminating the contactant(s) and the use of bland emollients and mild **soap-free** cleansing agents.

2. Contact allergic eczema

Unlike irritant contact dermatitis where a toxic effect occurs on the surface of the skin, allergic contact dermatitis involves an **external antigen turning on an endogenous delayed hypersensitivity immune response**. The manifestation of this reaction requires an initial **sensitization phase** followed by a subsequent **elicitation phase** where on reexposure to the sensitizer, patients may develop

Allergen	Present in
Nickel	Jewellery, Jean stud, bra clips
Dichromate	Cement, leather, matches
Rubber chemicals	Clothing, shoe, tyres
Paraphenylenediamine	Hair dye, clothing
Neomycin, Benzocaine	Topical applications

Also Poison Ivy (causes tissue necrosis), Euphobia & cashewnuts
Most often, persons having allergic contact dermatitis do not present with an acute dermatitis, but instead with a chronic dermatitis consisting of **erythematous papules** and **lichenified plaques** in the areas of contact. A meticulous history regarding both work and home exposures must be taken;

Ix

- **Epicutaneous patch testing** is performed in an attempt to reproduce the reaction. This involves applying a small amount of various suspected chemicals under occlusion for **48 hours**; the sites are examined for localized reactions (**erythema and edema, papules or vesicles**) at **48, 72** and **96 hours.**

Rx

- Avoidance of exposure is the cornerstone of treatment
- Midpotency to high-potency **topical corticosteroids** clear the chronic dermatitis
- Extended use of emollients and avoidance of irritants protects the altered skin from further damage.

3. Atopic Eczema

Atopy is a **genetic** predisposition to form excessive IgE which leads to a generalised & prolonged hypersensitivity to common environmental antigens, including pollen & the house dust mite. Atopic individuals manifest one or more of a group of diseases that includes;

- Asthma
- Allergic conjunctivitis - 'Muddy Sclera'
- Allergic rhinitis
- Hay fever - an acute allergic rhinitis & conjunctivitis that is sometimes accompanied by asthmatic symptoms
- Urticaria
- Food & other allergies
- Atopic eczema

Aetiology

- These atopic conditions tend to run true to type within each family - concordant in **86% of monozygotic twins** but only in **21% of dizygotes**; Inherited more often from the mother than from the father (**maternal imprinting**) & has a worse prognosis in females.

Pathogenesis

- Considered as an interplay of **genetic susceptibility** that causes **epidermal barrier dysfunction** & **abnormal immune responses**, which are then stimulated by different environmental factors.

C/P

· Pruritus is the most common symptom
· Distribution of lesions
- Infants (mostly between **3-6mo**) - face (cheeks & forehead with **central pallor**), trunk, & extensor surfaces (outer limbs)
- Children - antecubital and popliteal fossae
- Adults - face, neck, upper chest, and genital areas
- In adults with limited distribution of lesions a history of childhood eczema is a clue to diagnosis

· Morphology of lesions

- Infants - erythema and papules; may develop oozing, crusting vesicles
- Children and adults - lichenification and scaling are typical with chronic eczema
-

· Associated features (Atopic diathesis)
- Facial erythema, mild to moderate
- Infraorbital fold (Dennie's sign/Morgan line)
- Perioral pallor
- Dry skin
- Increased palmar linear markings
- Pityriasis alba (hypopigmented asymptomatic areas on face and shoulders)
- Keratosis pilaris - A condition marked by the formation of hard conical elevations in the openings of the sebaceous glands especially of the thighs & arms that resemble permanent goose bumps

Diagnostic criteria

Major;
* History of asthma/hay fever (or in a first-degree relative if <4yrs)
* Visible flexural eczema (cheeks, forehead, outer limbs if <4yrs)

Minor;
* Dry skin (xeroderma)
* Higher tendency for bacterial infection due to higher bacterial index on skin
* Hazy conjunctiva, periorbital hyperpigmentation, ruptured styes, tendency to develop cataracts (2° to disease process & long-term use of steroids)
* Tendency to get corneal ulceration due to frequent itching, convexity of the cornea is increased - keratoconus (A conical protrusion of the cornea caused by thinning of the stroma; usually bilateral)
* Dry eyes, uveitis, conjunctivitis
* Increased palmar linear markings
* Pityriasis alba (hypopigmented asymptomatic areas on face and shoulders)
* Keratosis pilaris - A condition marked by the formation of hard conical elevations in the openings of the sebaceous glands especially of the thighs & arms that resemble permanent goose bumps

DDx
- Seborrheic dermatitis (especially in infants)
- Contact dermatitis (especially if only the face is involved)
- **Photosensitivity rashes** - usually on sun exposed areas & may be 2° to;
  * Collagen vascular diseases
  * Use of topical substances
  * Pellagra
- Scabies
- Psoriasis or lichen simplex chronicus if only localized disease is present in adults

Mx
- Explanation, reassuarance & encouragement as treatment is lifelong
- Avoidance of contact with irritants
- Use of mild **soap-free** cleansing agents. Soap is a salt made from saponification of long chain fatty e.g. stearic acid & caustic soda (Sodium hydroxide) creating polar ends that bind water & thus acting as an emulsifier (binding oils & water) thus causes xerosis by emulsifying sebum
- Regular use of **greasy emollients** ("moisturizers") - prevent excessive water loss from an already dry skin & reduce the amount of local steroid used.
- Appropriate use of **topical steroids** (1% hydrocortisone)- **lotions** (aqueous base) **& creams** (oil/water mixture) are preferable in **acute eczema** & **ointments** (in an oily base) in **chronic** cases; they are usually applied **BD**

  Absorption of topical steroids can be enhanced by;
  - Occlusion by bandaging with e.g. 'wet wraps', tar & ichthamol paste bandages
  - Use of penentrants e.g. Salicylic acid, urea
- **Topical immunosuppresants**, including tacrolimus & pimicrolimus, can be used

Complications
- Cataracts are more common in patients with atopic dermatitis
- Skin infections (usually Staphylococcus aureus); sometimes subclinical
- Eczema herpeticum - generalized vesiculopustular eruption caused by infection with herpes simplex or vaccinia virus. Patients are acutely ill and require hospitalization.
- Irritant reactions due to defective barrier function
- Children with atopic eczema have an increased incidence of food allergy particularly to eggs, cow's milk, protein, fish, wheat & soya. These foods cause an immediate urticarial reaction rather than exacerbating their eczema

Expected course/prognosis;
- Chronic disease that tends to burn out with age. **95%** of patients have spontaneous resolution by age **5yrs**.
- Some adults (**1%**) may continue to have localized eczema, e.g., chronic hand or foot dermatitis, eyelid dermatitis, or lichen simplex chronicus; may be generalized
- May have adult onset eczema

4. Seborrhoeic Eczema
- It is a feature of AIDS
- Predominant age: Infancy, adolescence, and adulthood

Aetiology;
- Caused by **Pityrosporum ovale** infection of the skin.
- Genetic and environmental factors also contribute to disease, i.e., disease flares are common with any stress or illness
- Disease also seems to parallel increased sebaceous gland activity in infancy and adolescence or as a result of some acnegenic drugs

S/S

· Infants
- **Cradle cap - greasy scaling** of scalp, sometimes with associated mild erythema; disappears at **3mo age**
- **Diaper** and/or **axillary rash; Ears** and **retroauricular folds**
- Onset typically about age one month
- Usually resolves by age 8-12 months

· Adults
- **Bilateral and symmetrical Red, greasy, scaling rash** in most locations, consisting of patches and plaques with indistinct margins
- Red, smooth, glazed appearance in skin folds
- Minimal pruritus
- Chronic waxing and waning course
- Most commonly located in **hairy skin areas** with numerous sebaceous glands, e.g., **scalp** and **scalp margins, eyebrows** and **eyelid margins, nasolabial folds, ears** and **retroauricular folds, presternal area** and **mid-upper back**

Risk factors;
- Parkinson's disease
- AIDS (CD4 <200)(disease severity correlated with progression of immune deficiency)
- Emotional stress
- Stroke
- Drugs

Rx

Antipityrosporal agents e.g. Ketoconazole shampoo supplemented with **weak corticosteroids**

RELATIVE POTENCIES OF REPRESENTATIVE TOPICAL CORTICOSTEROID PREPARATIONS

Potency	Compound	Formulation
I. Very high	Clobetasol proprionate Halobetasol proprionate	Cream or ointment 0.05% Cream or ointment 0.05%†
II. High	Betamethasone diproprionate Betamethasone valerate Fluocinolone acetonide Hydrocortisone valerate	Cream or ointment 0.05% Ointment 0.1% Cream 0.02% Cream or ointment 0.01%
III. Medium	Betamethasone valerate Fluocinolone acetonide Hydrocortisone valerate Triamcinolone acetonide	Cream 0.1% Cream or ointment 0.025% Cream or ointment 0.2% Cream, ointment, or lotion 0.1% or 0.025%
IV. Low	Hydrocortisone***	Cream, ointment, or lotion 2.5% or 1.0%

† Ointments are more potent than creams containing the same corticosteroid in the same concentration.
***** 1% Hydrocortisone preparations** are specifically used for application on the face due to the thin skin & increased vasculature thus increased systemic absorption & toxicity.

SE;

* Skin atrophy (with striae, fragility & purpura)
* Acne (perioral/periorbital facial dermatitis)
* Hypopigmentation
* Enhanced or disguised infections e.g. Tinea incognito - Previous application of topical steroids may alter the appearance of tinea cruris causing a more extensive eruption with irregular borders and erythematous papules.
* Systemic absorption (causing suppression of the hypothalamic-pituitary-adrenal axis & even cushingoid features)

Tinea (Dermatophytosis)

Wednesday, April 13, 1994
10:46 PM

Papulosquamous disease

Infections caused by **dermatophytes** (mould fungi) that invade **only dead tissues** of the skin or its appendages (stratum corneum, nails, hair).
Also;

- Traumatized skin
- Areas of skin that's usually wet or macerated (softened by steeping or soaking)
- Occlusion by non-porous material - 2° to increased temperature & hydration

Aetiology;
Trichophyton, **Epidermophyton,** and **Microsporum** are most commonly involved, but clinical differentiation of dermatophytes is difficult. Transmission is usually from person to person or animal to person. Fomites are **not** usually responsible.

Pathogenesis;
Dermatophytes invade keratin by enzymic digestion & mechanical pressure; the hyphae grow into newly differentiated keratin as it is formed, keeping pace with the keratin growth. In tissue the dermatophytes take the form of branching hyphae, which may eventually break up

C/P;

- In skin infections of the **body (Tinea corporis)**, **face (tinea faciale)** & **scalp (Tinea capitis)**, spreading **annular lesions with raised, inflammatory border** & a **healed centre** are produced.
- Lesions in body folds, such as the **groins (Tinea cruris)**, tend to spread outwards from the flexures.
- In **foot ringworm (Tinea pedis**), infection is often confined to the **toe clefts**, but it can spread to the sole; sometimes painful secondary bacterial infection occurs in the toe clefts. **Infected nails** become discoloured, thickened, raised & friable - due to **T. rubrum**
- Infections of the groins, **hands (tinea manuum)** & nails are nearly always 2° to infection of the feet.
- In **scalp infections**, the fungus invades the hair shaft & then the hyphae break up into chains of **arthroconidia**. In some species (T. tonsurans & T. violaceum) the arthroconidia are **retained within the hair shaft** (**endothrix** invasion), whereas in others (Mirosporidium spp, T. verrucosum) they are **produced in a sheath surrounding the hair shaft** (**ectothrix** invasion)
- In **endothrix** infection, the **hair breaks off at, or just below, the mouth of the follicle**, which then becomes plugged with dirt & sebum to give what is described as a **black dot** ringworm, but in **ectothrix** infection the hair usually breaks off **2-3mm above the mouth of the follicle** leaving **short stumps of hair**.

Stages;

- Incubation period - no clinical features but skin scrapings are +ve with KOH wet preparation up to 6cm around lesion.
- Established infection;
  - If epidermal **cell turnover > organism growth rate**, **fungus is shed**
  - For **infection** to take place, **growth rate** of organism must be **=/> epidermal cell turnover**

At the inflammatory ring, the epidermal cell turnover is 4 times the normal & fungus is shed off. There is fungus 6cm around the lesion with increased cell turnover. The lesion extends centrifugally (peripherally) & becomes bigger

Ix
Direct demonstration of fungi - In a KOH preparation, scales are scraped onto a slide, covered with **10% KOH** and a coverslip, and gently heated under a flame to **digest** preferentially **keratinocyte cell walls**. **Fungal hyphae** are seen as translucent structures with even, parallel sides, that are periodically septate and often branching, and that traverse multiple cell boundaries

Mx
Topical (terbinafine or miconazole cream) or systemic (terbinafine, griseofulvin or itraconazole) **antifungals**; Endothrix responds faster due to systemic access of the drugs

Complications
Scarring & permanent hair loss

Leg Ulcers

Thursday, April 14, 2005
11:02 PM

An ulcer is a break in the continuity of an epithelial surface characterized by a progressive destruction of the surface epithelium & a granulating base

Clinical Examination of an Ulcer

i) Site
ii) Size
iii) Shape
iv) Edge
v) Floor - That which can be seen by an observer
vi) Base - What can be palpated
vii) Discharge
viii) Lymph Nodes
ix) Pain
x) General examination
xi) Pathological Examination
xii) Marjolin's ulcer - This eponym is used to describe carcinomatous change occuring at the edge of any long standing benign ulcer e.g. a chronic venous ulcer

Signs of a Malignant Ulcer

- Cancer is more common (10%) in ulcers **>2 cm** in diameter
- The deepest penetration of the ulcer is not beyond the expected border
- Rapid progression & poor healing
- Raised & everted edge (flat shelved edge in benign)
- Indurated base (necrotic in benign)
- Enlarged, Hard, Fixed Lymph nodes.

Leg Ulcers

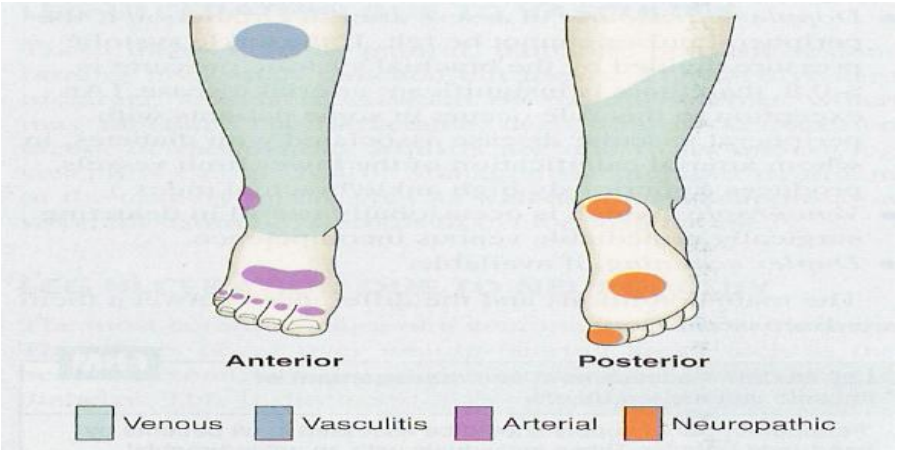
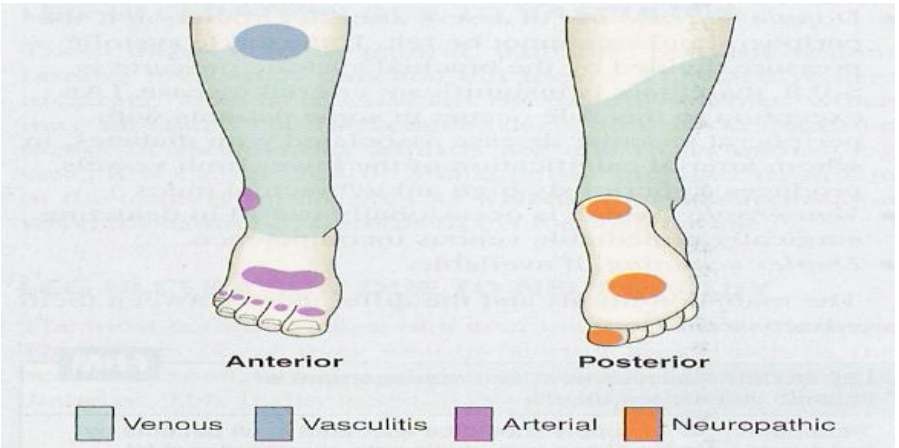


Fig. 21.8 Causes of lower leg ulceration.

21.15 MAIN CAUSES OF LEG ULCERATION

Venous hypertension	
• See text	
Arterial disease	
• Atherosclerosis • Vasculitis	• Buerger's disease
Small vessel disease	
• Diabetes mellitus	• Vasculitis
Abnormalities of blood	
• Sickle-cell disease • Cryoglobulinaemia	• Spherocytosis • Immune complex disease
Neuropathy	
• Diabetes mellitus • Leprosy	• Syphilis
Tumour	



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Neuropathy	
• Diabetes mellitus • Leprosy	• Syphilis
Tumour	
• Squamous cell carcinoma • Basal cell carcinoma	• Malignant melanoma • Kaposi's sarcoma
Trauma	
• Injury	• Artefact

Leg Ulceration due to venous disease

Damage to the venous system of the leg results in oedema, haemosiderin deposition, eczema, fibrosis & ulceration.

Aetiology

In the normal leg, there is a **superficial low-pressure** venous system connected to the **deep, high-pressure** veins by perforating veins. Muscular activity, aided by valves in the veins, pumps blood from the superficial to the deep system & towards the heart.

Incompetent valves in the deep & perforating veins due to;

- Previous DVT
- Congenital or familial valve incompetence
- Infection
- Deep venous obstruction (e.g. from a pelvic tumour)

results in the retrograde flow of blood to the superficial system ('venous hypertension') causing a rise in capillary hydrostatic pressure & stasis leading to **hypoxia** & **necrosis**. Fibrinogen is forced out through the capillary walls & fibrin is deposited as a pericapillary cuff. One theory postulates that growth & repair factors are trapped in the macro-molecular cuff so that minor trauma cannot be repaired & ulcers develop.

Management

- General management includes dietary advice for the obese & encouragement to take gentle exercise
- Oedema should be reduced by the regular use of compression bandages, keeping the legs elevated when sitting & the judicious use of diuretics
- The **exudate & slough** should be removed with **normal saline solution or 0.5% aqueous silver nitrate**. If the ulcer is very purulent, soaking the leg for 15mins in **potassium permanganate** may be helpful.
- Dressings commonly used for venous ulceration include antibiotic-impregnated tulle dressings, non-adhesive absorbent dressings (alginates, charcoals, hydrogels, or hydrocolloids) & dry non-adherent dressings.
- The frequency of dressings depends on the state of the ulcer. Very purulent & exudative ulcers may need daily dressings whilst the dressing on a clean healing ulcer may only require changing every week.
- Paste bandages, impregnated with zinc oxide or ichthammol, help to keep dressings in place & provide protection.
- Surrounding venous eczema is treated by a mild or moderately potent **topical corticosteroid**. The steroid should **not** be applied to the ulcer itself. An anabolic steroid , stanozolol, may help lipodermatosclerosis but side effects (fluid retention, hepatotoxicity) may limit its use
- Oral antibiotic therapy, given in short courses, is only necessary for the treatment of overt infection.
- In the absence of any evidence of compromised arterial supply, graduated **compression bandages** applied from the toes to the knees enhance venous return & have been shown to be most beneficial in the healing of venous leg ulcers
- Vein surgery may help some younger patients with persistent venous ulcers. **Pinch grafts** may hasten the healing of clean ulcers but do not influence their rate of recurrence.

Scabies

Friday, April 15, 2005
7:17 AM

Eczematous Disease

Scabies is caused by burrowing and release of toxic or antigenic substances by the **female** mite **Sarcoptes scabiei var. hominis.**

Clinical Manifestations.

- Intense pruritus, particularly at night.
- 1–2 mm red papules, some of which are excoriated, crusted, or scaling. **Threadlike burrows** (S-shaped - 3-5mm) are the classic lesion of scabies but **may not be seen in infants.**

- In **infants**, bullae and pustules are relatively common; the eruption may also include wheals, papules, vesicles, and a superimposed eczematous dermatitis; and the **palms, soles, face, and scalp** are often affected.
- In **older children adolescents**, **and adults**, the preferred sites are the **interdigital spaces, wrist flexors, anterior axillary folds, umbilicus and belt line, groin, genitalia in men, and areola in women, ankles, buttocks**; the head, neck, palms and soles are generally spared.
- **Norwegian (or crusted) scabies**, in which there are huge numbers of mites, has been described in immunocompromised or debilitated individuals. This highly contagious condition features an **oozing-encrusted, nonpruritic dermatosis of the hands and feet**; there also may be a generalized **erythematous scaling of the abdomen and back.**

Diagnosis

This can often be made clinically but can be confirmed by microscopic identification of mites by application of a drop of mineral oil on the selected lesion, scraping of it with a No. 15 blade, and transfer of the oil and scrapings to a glass slide.

Differential diagnosis

- Chickenpox
- Drug eruptions
- Dermatitis herpetiformis
- Atopic dermatitis
- Seborrheic dermatitis

Treatment.

- **Permethrin 5% cream (Elimite)** is a slightly more effective scabicide than lindane but is more expensive. **Caution:** Do not use on broken or secondarily infected skin
- Application of **1% lindane cream or lotion** to the entire body from the neck down, with particular attention to intensely involved areas and is left on the skin for 8–12 hr; if necessary, it may be reapplied in 1 wk for another 8–12-hr period. The vulnerability of small infants to percutaneous absorption of this potentially neurotoxic substance dictates caution in prescribing it for them. Signs of toxicity include nausea, vomiting, weakness, tremors, irritability, disorientation, seizures, and respiratory compromise.
- **Ivermectin** in a single dose PO has been used , in combination with topical drugs for the treatment of hyperkeratotic (crusted or 'Norwegian') scabies that doesn't respond to topical treatment alone.

Acne Vulgaris

Monday, April 18, 1994
7:00 AM

Inflammatory disease

A common inflammatory disease of the pilosebaceous glands characterized by comedones, papules, pustules, inflamed nodules, superficial pus-filled cysts, and (in extreme cases) canalizing and deep, inflamed, sometimes purulent sacs and, occasionally, scarring.
50% of affected individuals have a family history of acne; Male = Female

Aetiology

- Overproduction of androgens
- Hyper responsiveness of follicle/sebaceous gland to androgens
- Hypersensitivity to **Propionibacterium acnes** & its metabolic products
- **Pathophysiology**
    * Androgens stimulate **sebum production** & **proliferation of keratinocytes** in hair follicles
    * Keratin plug obstructs the follicle opening, resulting in sebum accumulation and follicular distension
    * P. acnes, an anaerobe, colonizes & proliferates in the plugged follicle
    * P. acnes **hydrolyzes sebum triglycerides into free fatty acids**, **produces chemotactic factors** and **proinflammatory mediators**, and **activates complement**, all of which result in inflammation of the follicle and surrounding dermis

Risk factors

- Adolescence
- Male sex
- Androgenic steroids, e.g., steroid abuse, some birth control pills
- Oily cosmetics, including cleansing creams, moisturizers, oil-based foundations
- Rubbing or occluding the skin surface, as may occur with sports equipment (helmets and shoulder pads), holding the telephone or hands against the skin
- Drugs - iodides or bromides, lithium, phenytoins
- Systemic corticosteroids
- Virilization disorders
- Hot, humid climate

S/S

Factors which influence symptomatology

- Sex:
    - males - later onset, greater severity;
    - females: earlier onset, lesser severity
- Seasonal variation - less severe in summer
- May be worse immediately prior to menses
- Closed comedones (whiteheads)
- Open comedones (blackheads)
- Nodules or papules
- Pustules, with or without redness and edema ("cysts")
- Scars - ice pick, atrophic macules, hypertrophic, depressed
- Lesions occur over the **forehead**, **cheeks** and **nose**, and may extend over the **central chest** and **back**

Grading system

Grade 1: Comedonal - closed/open
Grade 2: Papular > 25 lesions on face & trunk
Grade 3: Pustular > 25 lesions, mild scarring
Grade 4: Nodulocystic - inflammatory nodules and cysts, extensive scarring

DDx

- Occupational exposure to tars, oils, grease
- Steroid induced acne
- Folliculitis
- Pseudo-folliculitis barbae
- Acne rosacea
- Acne cosmetica
- Perioral dermatitis
- Chloracne

Pathological findings

- Oiliness, thickening of the skin
- Hypertrophy of the sebaceous glands
- Perifolliculitis
- Scarring

Ix

Testosterone and its metabolites can be measured in those very rare cases when acne arises de novo in the previously unaffected adult

Mx

- There is **no cure** for acne; Treatment only controls the lesions & prevents scarring
- Treatment measure takes a minimum of 4 weeks to show results
- Picking at or popping lesions may increase inflammation and scarring
- Drugs;
    - Comedonal acne
        * Topical retinoids e.g. Isotretinoin
        * Azelaic acid - Antibiotic & Anticomedonal effects
        * Benzoyl peroxide - reduce inflammation
    - Mild to moderate Inflammatory acne
        * Topical benzoyl peroxide
        * Topical antibiotic e.g. Erythromycin, Clindamycin, Metronidazole
        * Adapalene - retinoid-like drug
    - Severe Inflammatory acne
        * Isotretinoin (Accutane)
        * Oral contraceptives (females) - Co-cyprindiol (cyproterone acetate with ethinylestradiol) contains an anti-androgen.

Complications

· Acne conglobata - a severe confluent inflammatory acne with systemic symptoms
· Facial scarring
· Psychological scarring