An overview of dermatology

Dermatology is the branch of medicine that deals with skin, mucous membranes, hair and nails. Although relatively straightforward to examine, the skin is the largest organ and has numerous potential abnormalities - there are about 1500 distinct skin diseases and many variants. We are relatively ignorant about the pathogenesis of the majority of these although knowledge is rapidly increasing especially in the fields of molecular medicine and genetics.

This course will discuss the impact of skin diseases, outline the biology of normal skin, and describe how to examine the skin and how its diseases may be effectively treated. A range of skin infections, inflammatory skin diseases and neoplastic conditions will be briefly described.
UK data suggests an average of 15% of consultations in general practice relate to a skin problem and between 50 and 75% of individuals may have a skin problem at any time. Although most of these are relatively harmless and asymptomatic, (warts, athletes' foot, dandruff, insect bites and so on), many result in significant disability.

Symptoms of skin disease include:

- Pain, especially stinging and burning
- Itch, which may be intermittent or persistent, localised or generalised
- Functional disability

Signs may be described in terms of single areas of altered skin (lesions) or widespread eruptions. The distribution, configuration, colour, morphology, surface and secondary changes may be helpful in making a diagnosis and planning management.

Classification of skin diseases

Skin diseases are classified in various ways.

- Site of involvement such as facial rashes, lesions on sun-exposed sites
- Pathogenesis (when known) such as genetic abnormalities, infectious aetiology or autoimmune mechanisms
- Main structure affected such as epidermal diseases, abnormalities of melanocytes, vascular changes.

The general classification of skin diseases, which is here with suggested, is based on the idea that every abnormal skin condition may be thought of as a reaction against one of three offenses, each sub-divided into two classes:

I. Living offenders may be:

1. Parasites.
2. Bacteria.

II. Abnormal forces may be:

1. Mechanical.
2. Waves or emanations like light, heat, cold, electricity, x-rays.

III. Chemical substances may be:

1. Externally applied.
2. Endogenous
   (a) Exanthemata, including syphilis.
   (b) Allergic dermatoses, including drug reactions.

It must be understood that almost any combination of these irritants may be acting in a given case, and that any one of them may initiate the trouble, to be joined or replaced by another. The eczema may become infected, or it may follow scabiesor a coccigenic infection which has been cleared up.
As we noted above the reaction which is a sign or symptom of diseases may be either increase or decrease of some normal function or some unaccustomed function. In the case of the skin it is perhaps helpful at this point to call attention briefly to some of its more normal functions. Physically, chemically, and vitally it protects the body against things and forces in its environment which might be harmful. It is also, along with its modifications, the organ of sensation; e.g., special, such as heat and cold, hardness and softness, motion and position. Physical protection is given by the skin-in the following ways: mechanically, by resisting trauma, insulation against heat and cold and electricity, radiation or conservation of heat as required, protection against strong light by pigment. Chemically, the skin protects more sensitive underlying tissues against most of the weak acids and alkalis found under natural conditions; against many artificial ones as well.

Vitally protective functions include the following : the skin is not quickly water logged nor desiccated. Irritating chemicals are neutralized or washed away or absorbed and carried to more definitely excretory organs. Every bacterium and parasite cannot be kept outside of the epidermis, butmore blood is

brought to the affected area, and skin cels, leueocytes, andserum oppose and, if possible, destroy the in vaders. We may note in passing that one of the most important and vital functions of the skin seems to be to stimulate the reticulo-endothelial system in preparing the bacteria-fighting mechanism of the entire body.

When the normal functions of the skin, or, indeed, some abnormal functions, reach the point of causing disfigurement or discomfort, those by-products of reaction against offense may cause the patient to seek relief; and he will describe his symptoms in terms of appear- ance (rash, abnormal colour, swelling, weeping, scabs, pus, scaling, etc.), or feelings (itchiness, tenderness, pain, etc.). Given a patient with a group of these symptoms, how can the physician tel against what the reaction is taking place? We cannot here give the differential diagnosis of, say the 326 skin diseases mentioned above; but perhaps a few dogmatic general observations will suggest how useful the classification and viewpoint can be in practice.

Most of the commoner skin conditions are readily recognized by sight by anyone who has seen a fair number of cases. Many doubtful cases can be recognized nearly enough to give satisfactory treatment, even if a technical name is not applied, and even if the detailed causes cannot be stated. Certain of the cases can be diagnosed by their response to treatment in a comparatively short time. In yet other cases it is necessary to undertake prolonged study which may involve laboratory examination of pus, skin, scalings, etc., or search for an allergen.

Some points in diagnosis are: (1) Particular area of skin involved. (2) Appearance of reaction, e.g., redness, discrete border, etc., (3) Suggestive symptoms, e.g., itchiness, pain, tender ness, etc. (4) Signs and symptoms in other organs, e.g., the exanthemata. (5) Allergic symptoms in other organs. (6) Temperature, rapid pulse, etc. (7) History, presence of similar cases in family or district. Let us consider again our simplified classification of skin diseases, mentioning a few of those commonly met with as examples, and giving a few notes on treatment as we go along.

I

Parasites. In this group a sensible procedure seems to be to kill the parasites without destroying or irritating the skin cels. Most cases of scabies are identical in appearance. Occasionally a certain type of allergic reaction resembles it closely. Fortunately sulphur ointment does not seem to greatly irritate the rare case of eczema on which it is mistakenly applied, but it does

kill the parasites or scabies. The commoner of the other two or three conditions due to animal parasites are insect bites and pediculosis. Appropriate treatment is effective.

For practical purposes it does not seem to be essential to differentiate between the various fungi, there action to which are listed variously as trichophytos is, dermatophytosis, tinea, ring- worm. Mercurials usually meet the requirements, asbi-chloride compresses or ammoniated mercury ointment, or both. Whit field's ointment, chrysarobin, potassium permanganate, and some of the newer dyes also have their advocates.

Bacteria do not multiply readily in or on the outer layer of skin. It is possible that their presence helps maintain general body immunity (a sort of perpetual vaccine). Some- times the bacteria reach the warmth and moisture of the dermis in sufficient numbers to multiply and cause reaction. The offenders are usually the cocci. The mercurials will generally clear up impetigo. Slightly deeper skin infections, accompanied by possible pus formation, are treated by cutaneous antiseptics, attention to diet, tin by mouth, manganese by injection, vaccines, etc. MacKenna wisely remarks : "Remember that weak or non-irritant antiseptics applied frequently are better than strong antiseptics which damage the skin more than the bacteria". May we express this idea in other words by recalling that the reaction of the skin is capable by itself of overcoming the offenders in many cases. We do not need to feel responsible for killing off all the bacteria present, but only to insist that they practise "birth control".

In all conditions it is well to remember that the reaction of the skin may be so much in the nature of an allergic condition that the line of treatment adopted will have to have more consideration for that aspect of the case than for the matter of killing off the offenders.

II

Mechanical force sufficient to destroy the skin produces a wound, usually treated under the heading of surgery. "Dermatology",- knowledge and study of the skin as a living organ,-should be of great assistance to all who have to do with the healing of wounds. Some skins are hypersensitive to mechanical irritations, e.g., dermatographism is a severe degree of this. Dermatitis is another type of reaction against mechanical trauma. Most cases of dermatitis are more or less hyper-sensitive to mechanical irritation and therefore one should avoid rubbing with a towel, scratching, etc. The writer has seen an

eczema- tousarea of skin which was completely dry develop a tiny rash which oozed serum in a few minutes after one rub over it with the finger.

Certain persons and certain skin areas are affected more than others by physical conditions, e.g., heat or cold. It has been suggested that some cases of apparent drowning are due to cold allergy, hence allergic persons before swimming in cold water should be tested by immersing their hands in very cold water. Electricity may produce a dermatitis. X-rays can cause severe reaction when used improperly, and sometimes when used properly.

III

When chemical substances externally applied are severely caustic, destruction of tissues result, just as when the abnormal force is applied, and the dermatologist may then need to refer the patient to a surgeon. A mild irritating drug produces more or less dermatitis and requires complete removal of the offender, soothing of the area from further insult, either chemical or physical, and avoid- ance of infection.

Another type of skin reaction occurs when any material comes in contact with a skin area which is hypersensitive to that particular sub-stance. Then we have an allergic reaction which shows itself as urticaria, dermatitis, eczema, etc. The offending allergen may be almost anything, e.g., bacteria, something connected with the patient's work, toilet preparations, clothing, plants, medicines, etc. These allergic reactions can be discussed in more de- tail a little later, suffice it to say here that the treatment of contact dermatitis and eczema includes removal of the causes and soothing of the irritated area.

The skin diseases which are caused by chemical substances, which reach the dermis from endogenous sources including (a) the exanthemata, (b) allergic dermatoses. Exanthemata are characterized definitely by skin reactions to toxins. Probably, for convenience, syphilis can be included with this group; but every skin rash in persons whose serological tests are positive is not due to lues. The patient is stil susceptible to all the other ills that skin is heir to; e.g., scabies, eczema, drug rashes, etc. It was well known to the “grannies" of a less scientific day that “when the rash comes out the child will be better”. The steps they took to bring out the rash may not have been so ill-advised as many would have us believe. This recalls what has been said about the skin as a stimulator of the immunizing processes of the body. Is the rash in the exanthemata allergic or immunological or both; or are those conceptions mutually antagonistic? In their excellent book on scarlet fever the Dicks spend seven pages arguing that scarlet fever is not allergic, after wards

admitting that in a broad sense- “allergy is manifest in practically al infectious dis- eases'. In their anxiety to point out the importance of the toxin and antitoxin nature of scarlet fever they fail to notice that the two conceptions are not mutually antagonistic.

In an article of this kind there is no point in going into the theory of allergic diseases. In the allergic dermatoses the skin happens to be the sensitized or shock organ. Indeed in most of them it is only a certain area of the skin which has become sensitized. The group includes eczema, hives, angioneurotic oedema, purpura, erythema nodosum, etc. The sensitizing agents may reach the cells through external contacts or by way of the blood stream. In the latter case of antigen (or allergen) may reach the blood stream by absorption through some other parts of the skin or through the mucus membrane, particularly those of the intestine. Various article of the food are therefore very commonly causes of this group of skin disease.

Skin tests are not extremely important in discovering the allergen, as pointed out by several writers. For one thing the cases in which the skin tests are usually positive are chiefly those in which the reaction follows ingestion of the offending substance so immediately that the patient (or in the case of a child its parents) already have noticed the connection, and so will report. But in a larger group the reaction is so delayed that the immediate causal connection is not noticed by the layman.

The existence of drug rashes was known to the profession long before the idea of allergy was developed. The association between the drug and the skin reaction is often sufficiently close in time to be evident either to the patient or to his physician, although sometimes the fact that a sensitivity has developed to a drug which the patient has been taking for a considerable period may be missed. No drugs are usually in general use very long before some- one is able to report a case of allergic reaction to it. The writer has seen one extensive urticarial eruption following a course of sulfapyridine, and has reported a case of mild skin reaction to the ingestion of tablets of stilbestrol.

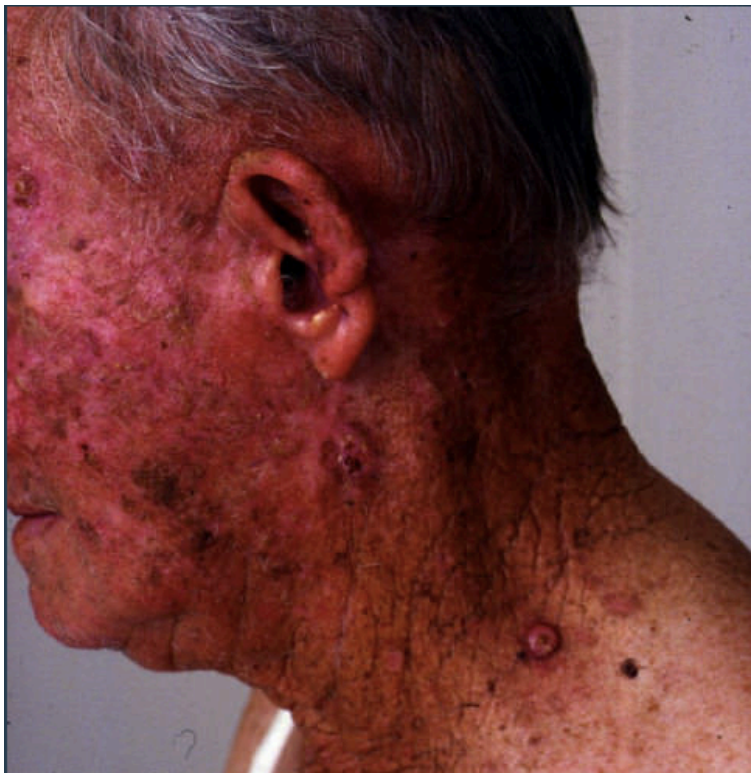
The treatment of the allergic skin diseases includes such simple and logical procedures as removing the allergen, applications of soothing ointments, lotions, etc.; and the avoidance of irritating substances, or procedures which might not affect a healthy skin at all but which, in an area giving an allergic reaction, can be suficiently strong to keep that reaction alight. It is also well to remember that psychology plays an important part in

many skin conditions, particularly those of an allergic nature. It is claimed that while psychoneurosis may not initiate an allergic phenomen on the reaction having been once produced by an allergen may be reproduced by emotional or other psychic conditions.

The Epidemiology and Burden of Skin Disease

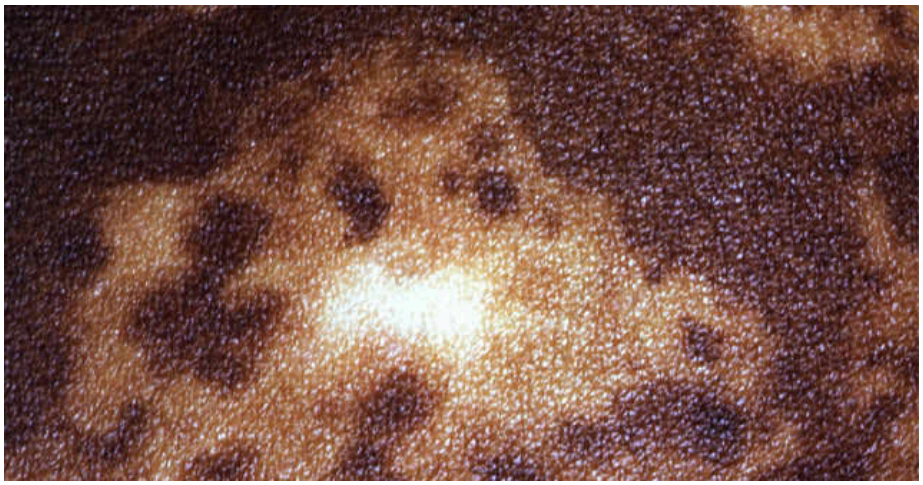
Despite the high frequency of certain skin diseases in developing countries, they have so far not been regarded as a significant health problem in the development of public health strategies.

White-skinned New Zealanders are particularly prone to conditions relating to excessive exposure to ultraviolet radiation in skin that has inadequate natural protection. These include photoageing changes (e.g.. dryness, freckling, fine wrinkles) and malignancies (e.g. actinic keratoses, basal and squamous cell carcinoma, melanoma).



Old skin

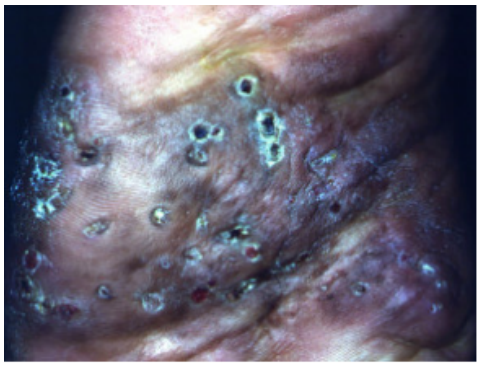
Black skin is particularly prone to pigmentary disorders and hypertrophic or keloidal scarring.

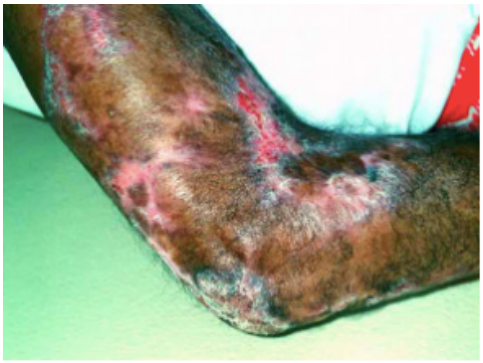


Trichrome vitiligo

Skin diseases prevalent in the tropics often have infectious origins.

- Bacterial infections, usually impetigo but also tropical ulcers, yaws, cutaneous tuberculosis and pinta
- Widespread fungal infections, usually dermatophytes but also mycetoma
- Parasitic infections, usually scabies but also leishmaniasis and schistosomiasis
- Viral haemorrhagic fevers

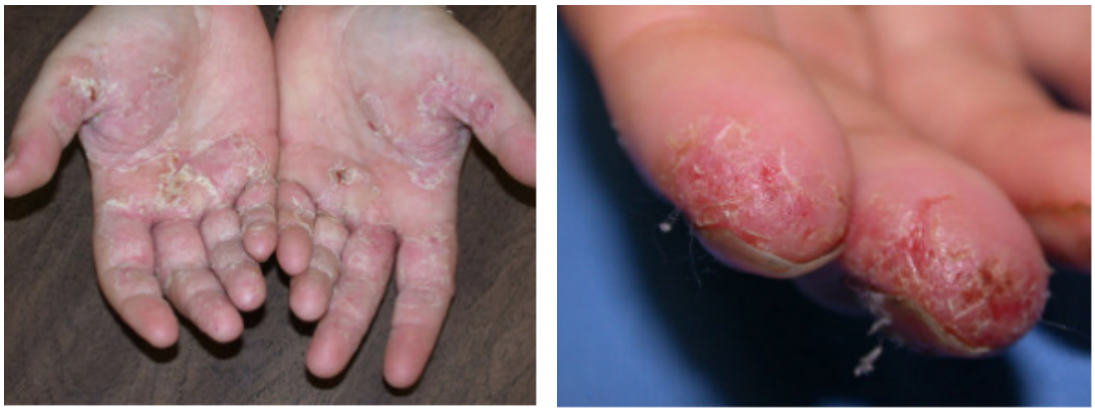




Tropical skin infections

Occupational dermatological diseases often relate to the irritant nature of material with which workers are in contact and sometimes to immune reactions to specific allergens. Hand dermatitis is the most common occupational skin problem. Examples:

- Cleaners develop irritant hand dermatitis due to water, detergents and solvents; and may be allergic to fragrances, formaldehyde or preservatives.
- Hairdressers develop irritant hand dermatitis due to water, shampoo and hair fibres; and may be allergic to nickel, hair dye, bleach or perming solution
- Builders develop irritant hand dermatitis due to cleansing agents, friction, fibreglass and cement (strongly alkaline); and may be allergic to epoxy resin (especially boat builders), potassium dichromate (a component of cement) or formaldehyde (timber treatment).
- Dairy farmers may develop chilblains (early morning milking), paronychia (cold wet fingers) or contact dermatitis due to allergy black rubber hosing.



Occupational hand dermatitis

Psychosocial impact of skin diseases

Most dermatological conditions are highly visible and may invoke disgust, shame and self-consciousness that can have profound psychosocial effects. Disfigurement can result in negative self perception, depression, social rejection and social isolation related to unfavourable self-image. Emotional abuse, verbal abuse and bullying may take place. These in turn can lead to self-contempt, frustration and torment leading to deliberate self harm or even suicide (a recognised complication of disfiguring skin conditions such as acne).

- Skin conditions affecting the face may require aggressive treatment even if they are clinically relatively mild.

- Disturbance of body image is particularly serious if it arises during childhood or adolescence, as is the case for birthmarks, atopic eczema and acne.

- Patients with body dysmorphic disorder (dysmorphophobia) believe they are ugly, unattractive or even repulsive despite a normal appearance. Preoccupation with their appearance may be a sign of obsessional-compulsive disorder and may lead to severe depression.

In addition, psychiatric disorders may manifest as apparent skin disease. Therefore the management of skin diseases requires recognition of psychological aspects as well as treating the affected skin.

- Psychoses may present as delusions of parasitosis, a false but unshakeable belief that they are infested and resulting in scratching and gouging the skin.
-
- Other patients deliberately harm their skin and may present with strangely shaped burns, ulcers or rashes. This is called dermatitis artefacta.

The prevalence of skin diseases

An analysis of hospital attendances for skin diseases conducted in Perth in 1992 reported the most common conditions encountered were actinic keratoses, psoriasis, malignant tumours and dermatitis.

The range of skin conditions seen by dermatologists depends on:

- The actual prevalence of skin disease
- The potential clinical morbidity and mortality of the disease
- The clinical severity of the disease in the individual
- Ethnicity and skin colour
- The availability of effective therapy
- The impact on the individual (employment, educational and psychosocial effects)
- Public awareness and concern (especially relating to skin cancer)
- The referring doctor's interest and knowledge about skin disease (increased knowledge may increase or reduce referral numbers)

- Availability of diagnostic and treatment services
- Cost of diagnostic and treatment services

Skin disease prevalence in the community is largely unknown. Several population surveys have been carried out in the Maryborough community in Victoria, Australia. Light exposed sites were examined in 2113 adults in a 1983 report; 49 (2.32%) had at least one skin cancer and 1202 (56.9%) had at least one actinic keratosis.[3] Total body examinations in 1457 adults was reported in 1999. The age- and sex-adjusted prevalence of warts was 7.1%, acne 12.8%, atopic dermatitis 6.9%, seborrhoeic dermatitis 9.7%, asteatotic dermatitis 8.6%, psoriasis 6.6%, culture-positive tinea 12%, seborrhoeic keratoses 58.2%, and Campbell de Morgan spots 54.4%. Subsequent examination of a sample of the non-respondents revealed they were more likely to have skin cancers and Campbell de Morgan angiomas than the respondents. The authors comment that as population-based surveys on the frequency of common skin diseases rely on voluntary presentation, there is a risk of response bias which may compromise the quality of the data obtained. Some skin disorders are more common in those with black skin (tinea capitis in children and acne keloidalis nuchae in adults for example)[6] and others less common (skin cancer).

A significant percentage of the workload of a general practitioner is dermatological. A study of a practice in Cornwall published in 1999 found 21% of 11,191 patients seen had a dermatological diagnosis (21%). The most common skin diseases seen were viral warts, eczema and benign tumours. Skin disease is not simply a cosmetic problem and may have similar impact to other medical disorders. Dermatologic problems can result in psychosocial effects that seriously affect patients' lives comparable to arthritis and other disabling illnesses.

Structure of the epidermis

The skin of an adult occupies an area of 1.5 to 2 m^2. It varies in thickness from 0.3 to several centimetres in thickness. The thinnest sites are the eyelids (a few cells thick) and scrotum. The thickest are the soles and palms (about 30 cells thick). The total weight of skin can reach 20 kg, about 16% of total body weight.

Skin is made up of:

- Epidermis
- Basement membrane zone
- Dermis
- Subcutaneous tissue.

Normal skin

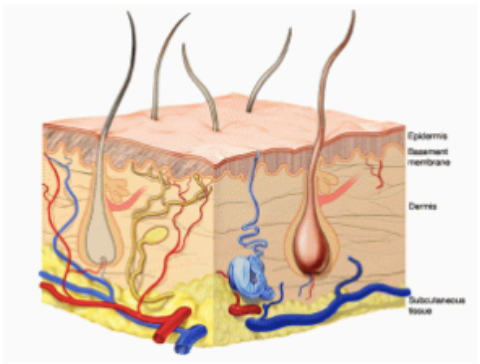
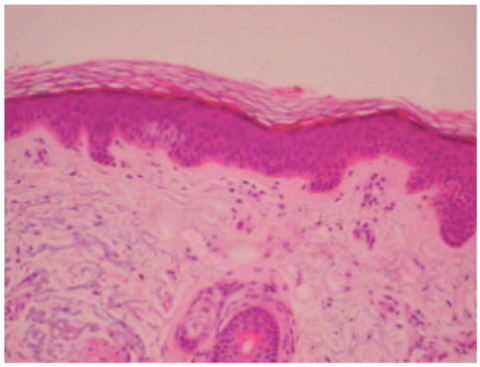


Diagram showing structural



Haematoxylin and eosin stained

These layers are modified according to the needs of the specific area of the body. For example, the scalp is covered with thick hair, the palms have particularly thick epidermis and the face contains large numbers of sebaceous glands.

Each square centimetre of skin is said to have approximately:

- 6 million cells
- 5,000 sense end organs
- 400 cm nerve fibres
- 200 pain sensors
- 100 cm blood vessels
- 100 sweat glands
- 15 sebum glands
- 12 cold receptors
- 5 hairs
- 2 heat receptors

Acid mantle

Skin has an average pH value of 5.5, creating the acid mantle. This is the result of acidic substances such as amino acids, lactic acid and fatty acids in perspiration, sebum and the hormones. There are resident protective

microflora (bacteria and yeasts) but the acid mantle repels pathogenic micro-organisms and reduces body odour.

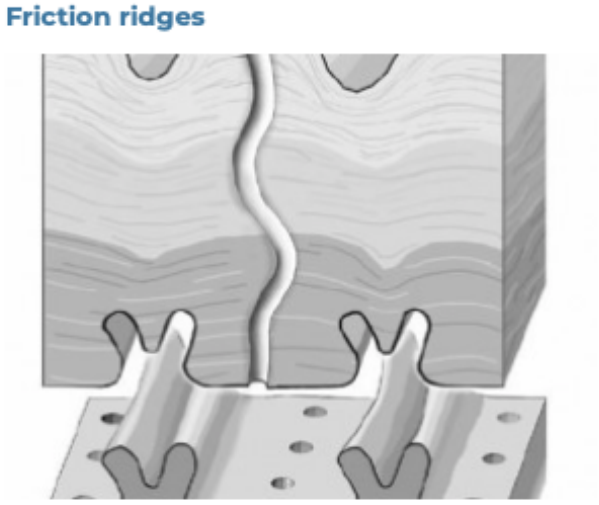
Epidermis

The epidermis is a dynamic structure acting as a semi-permeable barrier with a layer of flat anuclear cells at the surface (stratum corneum). The epidermis regenerates in orderly fashion by cell division of keratinocytes in the basal layer, with maturing daughter cells becoming increasingly keratinised as they move to the skin surface. Immune cells within the epidermis recognise and process small molecules penetrating the skin surface. Pigment cells in the basal layer (melanocytes) protect the skin from ultraviolet radiation. The basement membrane zone is the communication channel between epidermis and dermis.

The epidermis has a complex structure designed to protect from the environment. It has an undulating surface with cross-crossing ridges and valleys, with invaginations due to follicles and sweat duct ostia. Epidermis is thickest on palms and soles, and thinnest on eyelid and scrotum.

Friction ridges

Ridges are particularly well developed on the fingers and toes where they are known as friction ridges with characteristic patterns commonly referred to as fingerprints. The science of ridgeology has been well developed for forensic purposes.



Friction ridges

Proliferation

Keratinocytes make up 95% of the skin surface and are normally renewed every 15 to 30 days. The speed of renewal is greater if the epidermis is injured and in certain skin diseases (particularly psoriasis). Keratinocytes are created in the basal layer and gradually move towards the surface, flattening out and becoming more differentiated towards the anuclear horny cell of the stratum corneum.

The appearance and structure of normal skin varies according to the site of origin of the tissue and the age, sex and ethnicity of the subject.

Basal cell layer	
Keratinocytes	• Columnar cells derived from ectoderm. • Stain with haematoxylin (i.e. pink on H&E sections). • Produce protein (keratin) and lipids. • Produce inflammatory cytokines including interleukin-1. • Express adhesion molecules. • Attached to surrounding cells by desmosomes. • Dividing cells with a roughly 19-day cycle. Daughter cells move to surface to form stratum corneum (28 to 60-days). • On haematoxylin and eosin (H&E) routine sections keratinocytes appear pink (taking up eosin dye) with blue nuclei (haematoxylin).

Melanocytes	• Dendritic cells with clear cytoplasm and small dark-staining nuclei. • Derived from neural crest. • Ratio to basal keratinocytes is 1:10. • Produce melanin in melanosomes. • Dendritic processes allow transfer of melanin to adjacent keratinocytes by pinocytosis (tips of dendrites pinched off and engulfed). • Similar number of melanocytes in all races but melanogenesis is variable. • White skin: melanin mainly in basal layer. Blacks: melanin throughout epidermis. Tanning: melanin shifts into keratinocytes, production increased.
Merkel cells	• Touch (sensory-mechanical) receptors. • Scarce small round cells found only using electron microscopy. • Most numerous in palms and soles. • Probably neural crest derived, possibly from keratinocytes. • Attached to keratinocytes by desmosomes.

Squamous cell layer (stratum spinosum, prickle cell layer)

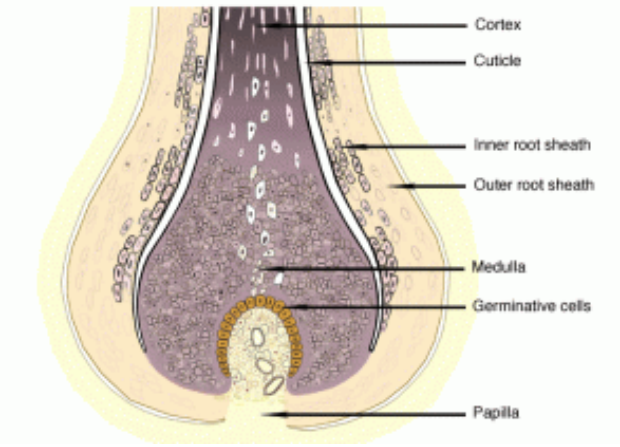
Keratinocytes	• 5-12 layers of polygonal cells that become flatter near surface. • Daughter cells produced by basal cells progress outwards. • Communicate with surrounding keratinocytes, melanocytes and Langerhans cells.
Langerhans cells	• Dendritic immune cells with clear cytoplasm on haematoxylin-eosin (H&E) stained sections; identified by special stains (such as gold). • Characteristic “tennis racquet” granules on electron microscopy. • Derived from bone marrow. • Antigen-presenting cell: collects contact antigens and presents them to sensitised T lymphocytes. • Circulate via dermal lymphatics to regional lymph nodes. • Surface receptors for C3, Fc portion of IgG; express Ia antigens on surface.
Desmosomes	• Attached to cell membrane opposite similar complex on adjacent cell. • Tonofilaments connect the keratinocyte cytoplasm with the desmosome. • In the intercellular space there is a lattice-work transmembrane linker. • Desmosomes make and break as keratinocytes move from basal layer to surface.

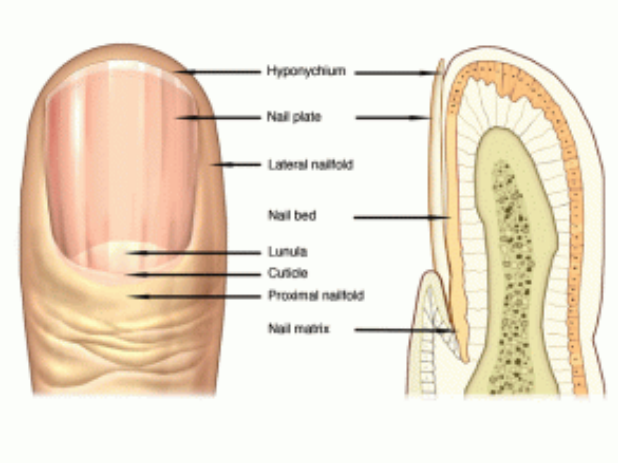
Inflammatory cells	Migrating neutrophils, eosinophils, lymphocytes, erythrocytes can be present in epidermis transiently in diseased states.

Epidermal appendages

Structure	Description
Eccrine glands	• Sweat glands produce hypotonic solution of water, sodium chloride, urea, ammonia and uric acid. • Abundant, except vermilion of lips, labia minora, glans penis, prepuce. • Most dense on palms, soles, axillae and forehead. • Sweat on palms and soles enhances grip. • Secretory coil deep in dermis, duct opens directly onto skin surface. • Duct composed of two layers of small cuboidal cells. • Immune function: secrete IgA. • Hypothalamic control via specific nerve fibres results in increased production with heat, emotional stress and spicy foods. • Develop tolerance to high ambient temperatures by increased sweat production and increasing hypotonicity.

Apocrine glands	• Scent glands that become active after puberty. • Mainly found in axillae and perianal regions. • Primary secretion is thick and odourless; smell derives from bacterial colonisation. • Modified as Moll's glands (eyelids), ceruminous glands in ear canal, mammary glands, • Open into pilosebaceous follicle.

Pilosebaceous structures	• Terminal hair on scalp; vellus hair on body surface (short, thin, light coloured). • None on palms and soles. • Root sheath buds downward from epithelium at a consistent angle, depending on site. • Growth phase (anagen) with pointed tip lasts several years; short involutional phase (catagen); resting phase for several months, with clubbed or bulbous tip (telogen). • Hair cortex is produced at a rapid metabolic rate from medulla (loose cuboidal cells) within hair bulb. Cortex contains densely packed keratin with extra sulphur and cystine. • Cortex surrounded by cuticle: a single layer of shingle-like cells. • Hair colour depends on amount of melanin in cortex during anagen: dark hair has more eumelanin melanosomes; fair hair fewer with more lamellated phaeomelanin; red hair has erythromelanin; grey/white hair very few melanocytes. **Anatomy and physiology of hair**  Structure of hair bulb

Sebaceous glands	• Most concentrated on scalp and face where circulating androgens induce increased secretion at puberty. • Several lobules lead into common excretory duct, mostly opening into outer portion of hair follicle. Opens directly onto skin surface on labia, prepuce, nipple and areola. • Modified in Montgomery's tubercles of areolae secreting into lactiferous duct. • Produce sebum: triglycerides, phosopholipids, esterified cholesterol.
Nails	• Nail matrix produces cornified cells of nail plate; outer cells formed by proximal matrix, innermost by distal matrix (lunula). • Fingernails grow 0.1mm per day; toenails 0.03mm per day. • Cuticle protects matrix from environment. • Nails protect and scratch. **Normal nail**  Normal nail

Structure of the dermis and subcutis

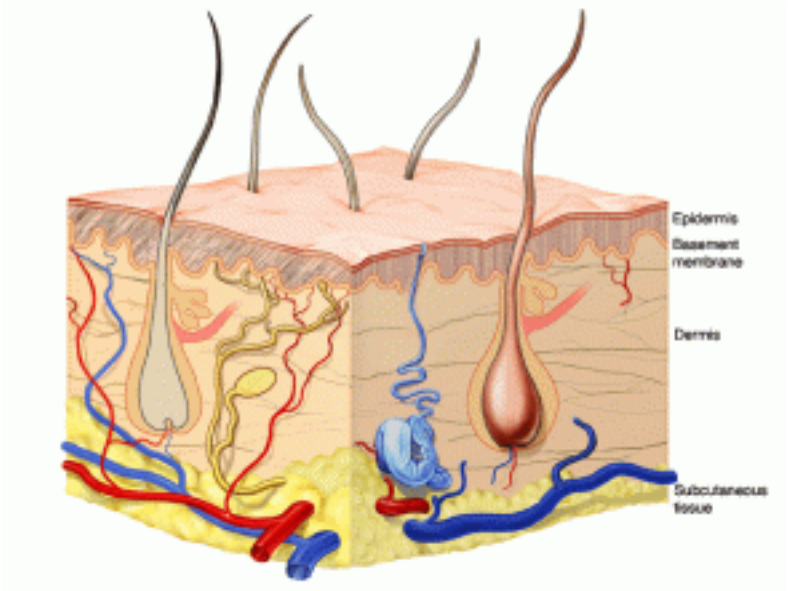
The basement membrane zone is the communication channel between epidermis and dermis. The dermis supports the epidermis, providing nutrients and protecting it.

The dermis supports the epidermis by providing it with nutrients and toughness. It is made up of fibres and ground substance, with nerves, blood vessels and cellular infiltrations. The papillary dermis is the upper portion beneath the epidermis, characterised by thin haphazardly arranged collagen fibres, thin elastic fibres and ground substance. The lower portion is the reticular dermis, composed of coarse elastic fibres and thick collagen bundles parallel to the skin surface.

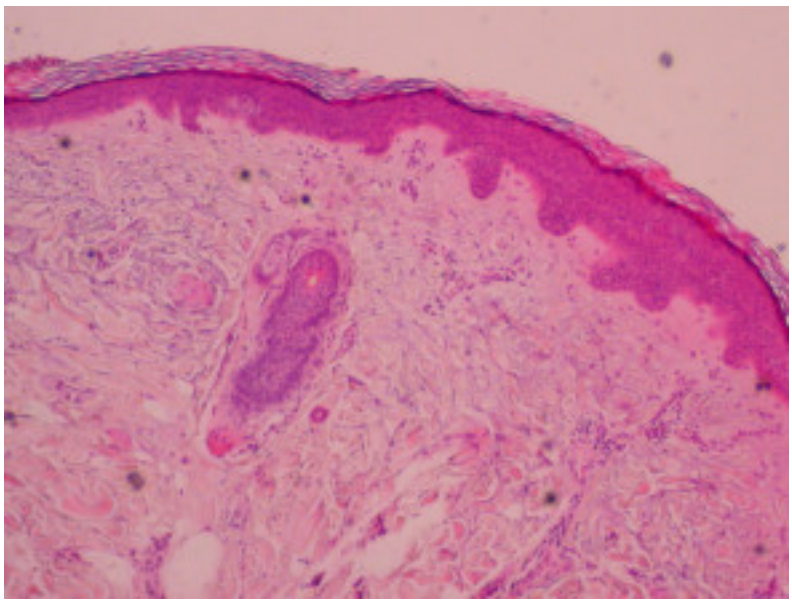
The dermis is full of double rows of peg-like formations called papillae under the basement membrane zone. Each double row underlies an epidermal ridge.

The papillary dermis is the portion of the dermis just below the epidermis. The reticular dermis extends from the papillary dermis to the fat.
Below this is subcutaneous tissue, the shock absorbing, and insulating and energy storage layer.

Normal skin



Structural components of skin



Haematoxylin and eosin stained

Basement membrane zone

Structure	Description
Basal cell membrane	• Selectively permeable membrane. • Hemidesmosomes on dermal side.
Lamina lucida	• Although appears clear under electron microscope, it is a dense structure holding anchoring filaments (type 7 collagen), fibronectin and laminin (a glycoprotein). • Contains pemphigoid antigens.
Lamina densa	• Appears dense under electron microscope. • Made of type 4 collagen and heparan sulphate. • Contains epidermolysis bullosa acquisita antigen.
Sublamina densa	Cross banded fibrils bind lamina densa to papillary collagen.

Dermis

Structure	Description
Collagen	• The major protein of the dermis and 90% of the dermal fibres. • Provides tensile strength. • Fibres are cross-linked triple helix of polypeptide chains. • Continuously synthesised by fibroblasts and degraded by collagenase. • Haphazard arrangement in papillary dermis; bundles are parallel to surface in reticular dermis. • Fine network of Type 3 collagen around blood vessels stains with silver.
Elastic fibres	• Comprise 10% of the fibres in the dermis. • Return deformed skin to its resting state. • Made of amorphous elastin protein, surrounded by microfibrils. • Synthesised by fibroblasts. • Thin fibres in papillary dermis; thicker in reticular dermis.

Ground substance	• Amorphous viscoelastic gel made of anionic polysaccharides (glycosaminoglycans): hyaluronic acid, dermatan sulphate, and chondroitin-6-sulphate. • Produced and degraded by fibroblasts and mast cells. • Binds water (up to 1000 times own weight); controls flow of solutes.
Fibroblasts	• Derived from mesenchyme. • Produce collagen, elastin, ground substance and fibronectin (a glycoprotein).
Blood vessels	• Arteries in subcutaneous tissue send arborising branches into the dermis. • Lined by endothelial cells; sheathed in collagen; surrounded by pericytes, dendritic macrophages and T-lymphocytes. • Hypothalamic control results in constriction and dilatation with heat, emotional stress (blush with embarrassment, anger; blanch with fear) and spicy food.
Lymphatics	Extensive network throughout dermis.
Nerves	• Extensive network of sensory and autonomic nerve fibres. • Distinct sensory nerve endings for touch, heat, cold, pressure, pain.

Arrector pili muscles	• Originate near the basement membrane zone and attach to the hair follicle near its base. • Smooth muscles. • Cause erection of the hairs on exposure to cold or fear (goose bumps). • Best developed on areolae and tunica dartos of scrotum.
Immune cells	• Lymphocytes, neutrophils, monocytes (histiocytes and activated macrophages), mast cells. • Most often found around blood vessels.

Subcutaneous tissue (subcutis, hypodermis)

Structure	Description
Adipose cells	• Connective tissue (lipocytes). • Groups of cells form lobules with wide variations. • Surrounded by connective tissue, larger blood vessels and nerves. • For fat storage, heat insulation, shock absorption.

Functions of the skin

Intact skin is essential for life, illustrated by the serious nature of extensive thermal burns - the mortality risk of a 40% total body surface area burn in a 70-year-old patient is 94%.

Secretions

The surface of the scalp, face and upper trunk of adults is a hydrolipid film made up of sebum, water, salts and metabolic products. Sebum is produced by sebaceous glands within the hair follicular apparatus. The lipids protect against irritants, allergens and certain toxins and prevent water loss.
Eccrine sweat glands produce a salty solution. They are found over most of the body but are often profuse over the scalp and forehead, axillae, palms and soles. This arises in response to exercise and high temperature (internal or environmental).

Apocrine glands are found in axillary, pubic and perianal regions resulting in a sticky secretion prone to malodour. The smell is due to colonization by bacteria.

Protective properties

Barrier function	
Physical	• Protects the body from mechanical damage i.e. friction and impact. • Mainly impermeable to water. • Adherent flat cells of stratum corneum, supporting keratinocytes and collagen provide tensile strength. • Sebum aids pliability. • Elastin allows skin to recover shape after deformation. • Subcutaneous tissue absorbs shock.
Thermal	• Poor conductor of heat, thus a barrier to heat injury, tolerating <40 deg C. • Higher temperatures may cause marked tissue destruction. • Severe burns rare on palms and soles because of thick epithelial cover. • Deep epidermal appendages (scalp) are also protective.

Antimicrobial	• Protects from bacteria, fungi and viruses. • Keratinocytes provide physical barrier and produce cytokines. • Sebum and acid mantle repel pathogenic organisms. • Commensal bacteria/yeasts protect against pathogens. • Skin is a major immunological organ.
Chemical	• Keratinocytes protect from chemicals. • Excessive exposure causes irritant dermatitis and chemical burns.
Radiation	• Superficial layers of epidermis protect dividing DNA in basal layer and below from excessive UV radiation • Due to thickness of keratinocyte layer, melanin, urocanic acid and appendages. • Sunburn rare on palms and soles because of thick epithelial cover. • Sunburn rare in black skin because of melanin throughout epidermis. • Sunburn greater after bathing because of loss of water-soluble urocanic acid. • Hair provides a physical barrier.
Maintain fluid balance	
Evaporation	• Adherent keratinocytes and sebum prevent skin from drying out. • Sweat moistens skin surface.
Permeation	Adherent keratinocytes and sebum prevent absorption of excess water.

Excretion	Waste products and salt disposed of in sweat.
Regulation of body temperature	
Cooling	• Evaporation of sweat. • In hot weather cutaneous blood vessels dilate to give off heat.
Warming	• Insulating subcutaneous tissue. • In cold weather cutaneous blood vessels constrict to conserve heat. • Hair protects scalp. • Arrector pili contraction (goose bumps) produces heat.
Immunological function	
Infection	Protect against microbial attack.
Allergy	Protect against allergic reaction.
Metabolic function	
Synthesis	• Vitamin D (subcutaneous tissue) • Structural proteins, glycans, lipids, signalling molecules
Storage	• Fuel in subcutaneous adipose cells • Electrolytes, water, vitamins, carbohydrates, protein
Wound healing	Secrete fibronectin and other compounds required for restoration of skin integrity.
Communication	
Sensory nerves	• Warn of heat, cold, contact, mechanical injury, infestation. • Temperature, pain, touch, vibration, pruritus.

Physical state	• Aesthetic appearance. • Odour (sexual attraction): apocrine gland secretion. • Deodorant (low level of bacterial decomposition).
Emotional state	• Facial expressions. • Skin colour (erythema, pallor). • Sweating, goose bumps.

Skin permits a stable internal environment, not just holding the body together but also protecting internal organs from environmental dangers.

Skin colour

Skin colour varies from white (absence of pigmentation) to black (densely melanized). The colour depends on the quantity and depth of melanin and other chromophores.

Oxidised haemoglobin	Red
Deoxidised haemoglobin	Blue
Haemosiderin	Red-brown
Carotene	Orange-yellow
Bilirubin bound to elastin	Green-yellow
Dermal exogenous pigment	Tattoos, makeup, drugs, metallic compounds

The thickness and quality of keratinocytes and dermal components may also affect skin colour. A thick layer of horny cells can appear white (psoriasis), yellow (seborrhoeic dermatitis), dirty-brown (ichthyosis) or black (eschar). Inflammatory infiltrates may result in plaques that are yellow-brown (granulomas), violaceous (lichen planus) or scarlet (psoriasis).

The pigment melanin is formed by the action of tyrosinase on dopamine metabolites in melanosomes (specialised pigment granules) in the cytoplasm of melanocytes. Skin produces predominantly brown to black eumelanin and

a little phaeomelanin; the hair of red-headed celts contains mainly phaeomelanin.

The amount of melanin depends on:

- Genetic factors: ethnicity/phototype.
- Active melanogenesis.

There are melanocytes in the normal skin of all races. However, dark-skinned people produce more melanin, and it is distributed to keratinocytes throughout the epidermis. The more superficial the pigment, the more effective the protection it provides against damage caused by ultraviolet radiation. Albinos have inactive melanocytes.

Melanin is found in the dermis in some melanocytic naevi (moles) and as a result of inflammation affecting the level of the basement membrane (postinflammatory pigmentation).

Melanin pigmentation is promoted by:

- Ultraviolet radiation
- Hormones (MSH, ACTH, androgens, oestrogens, progesterones)
- Inflammation
- Friction

It may be reduced by:

- Melatonin
- Corticosteroids

Symptoms of skin disease

The predominant cutaneous symptom is itch (pruritus). Itch can be localised or generalised, intermittent or continuous. It can occur in the absence of skin disease but is more frequently caused by a dermatological problem. Little is known about the mediators or the neurological processes involved in either the detection of an itch stimulus or the induction of the main response to itch, scratch. Symptom control remains unsatisfactory.

Pain is experienced when the integrity of the skin is broken but may also result from a neuropathic stimulus, as in various forms of neuralgia. It is described as stinging, burning, rawness, stabbing, boring, sharp or dull. Neuropathic symptoms may be accompanied by altered sensation (dysaesthesia), often hyperaesthesia.

Pathophysiology of pruritus

Itch is a subjective and variable sensation resulting in scratching. Although itch is mostly unpleasant, scratching is a pleasure, perhaps because the pain of scratching reduces the itch.

Peripheral nervous system

There are complex chemomediators on free nerve endings transmitted by C–fibres. It is possible that itch is low intensity pain at dermoepidermal junction, but specific itch fibres and nerve endings are now considered more likely. Mechanical and electrical stimuli and chemicals including histamine and substance P may induce itch.

Central nervous system

Itch activates a cerebral network resulting in a strong motor effect – itch provokes scratch, whereas pain results in withdrawal from the painful stimulus. Lesions of the lateral spinothalamic tract disrupt itch, pain and temperature sensations. Itch can be induced by centrally acting serotonin and opioids.

Evaluation of the itchy patient

History should determine what areas are affected by itch and its severity, exacerbating and relieving factors and the time course of the symptoms and signs.

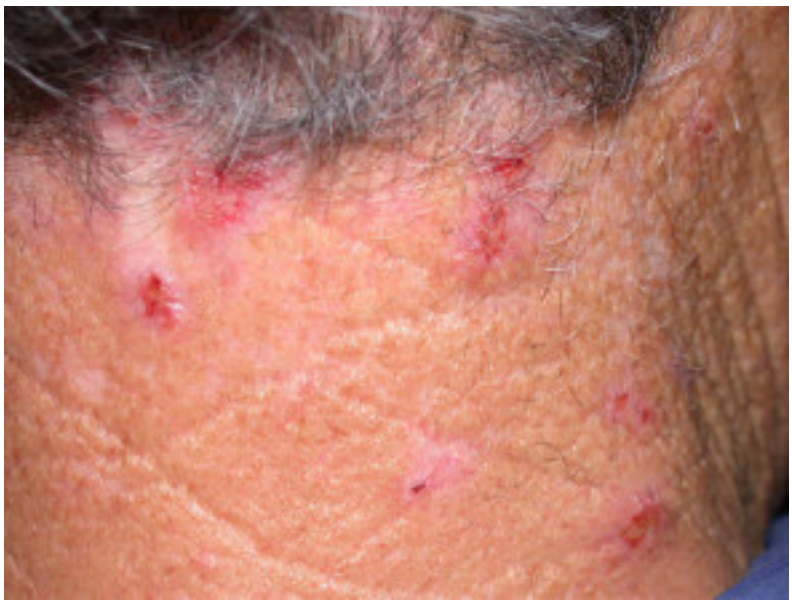
Examine the patient's skin all over and note the distribution of the itch/rash. Identify primary and secondary skin lesions. Scratching results in picked or linear excoriations, bruises and broken-off hair. Nails used for rubbing appear highly polished.

Perform a full medical examination, particularly if there appears to be generalised itch without a primary skin rash.

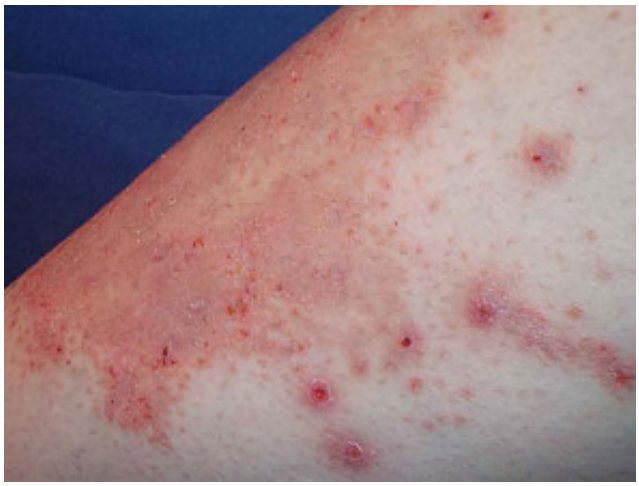
Laboratory tests could include swabs and scrapings for viral, bacterial, fungal culture if infection is possible. Consider the possibility of scabies and look for burrows along the sides and between the fingers and on the wrists. Perform microscopy on the contents of a burrow.

Skin biopsy of a typical primary lesion may be very helpful but these can be hard to find – pathology of a scratched spot shows non-specific inflammatory and healing changes and could be misleading.

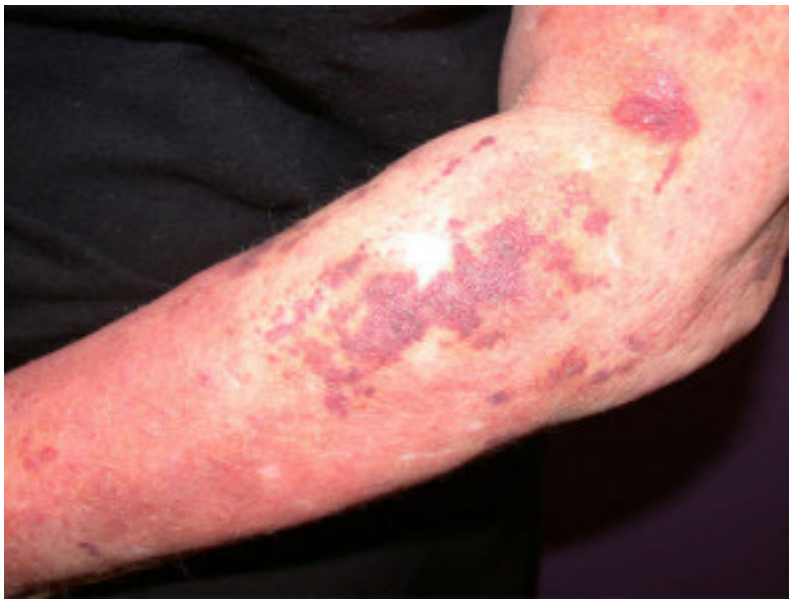
Effect of pruritus



Excoriations but no primary lesions



Excoriated dermatitis



Rubbing the skin resulting in bruising

Laboratory tests are indicated if there is generalised pruritus without an obvious primary skin rash. The range of tests should include as a minimum:

- Complete blood count and differential
- Iron studies
- Liver function tests
- Renal function tests
- Thyroid function tests

A chest X-ray may be indicated if there are pulmonary symptoms or signs or unexplained lymphadenopathy.

Causes of localised itch

In most cases itch is related to an inflammatory condition affecting the skin, although sometimes the itch appears to be caused or at least aggravated by scratching (the itch-scratch cycle). Effective treatments may include emollients, topical steroids and oral antihistamines.

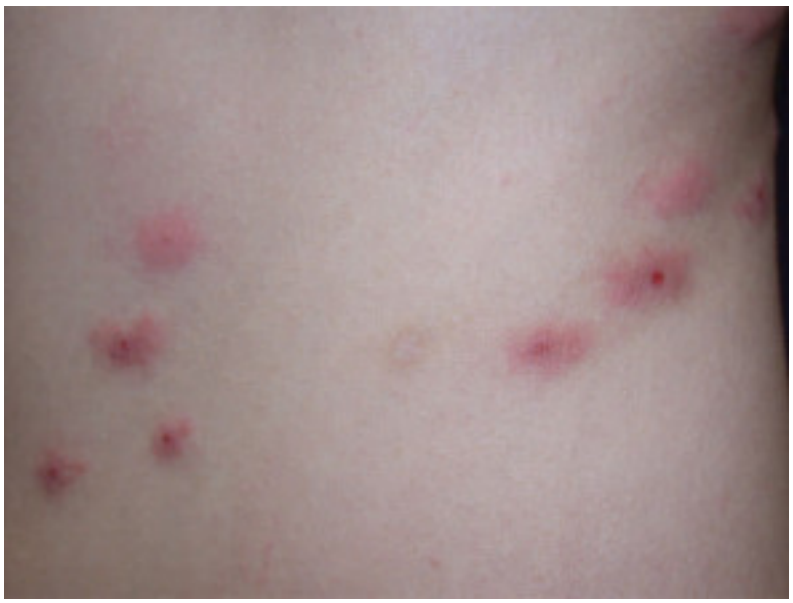
These conditions can occur on any site:

- Insect bites: severity of reaction depends in part on the individual's specific immune response. Insect bites often appear as grouped crops

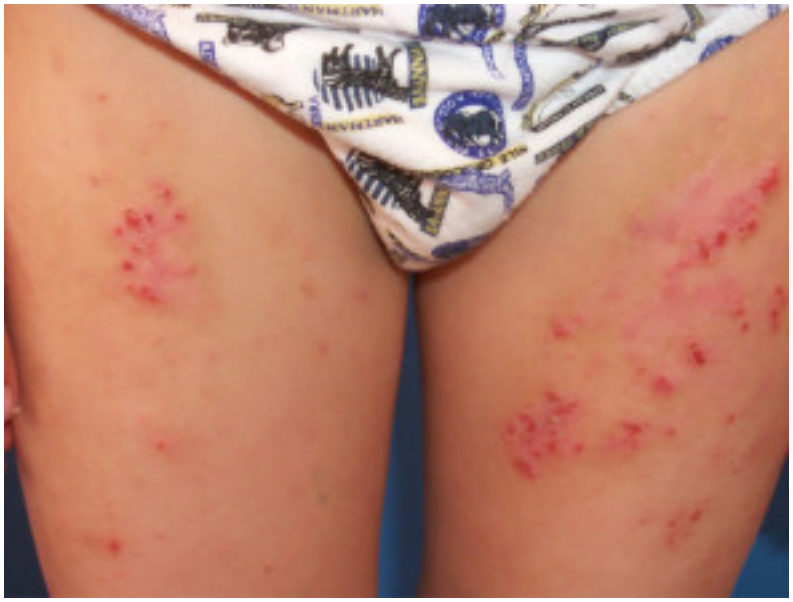
of urticated papules or blisters (‘breakfast, lunch and tea’) on exposed areas, especially the lower legs and waistline.

- Dermatitis: especially atopic dermatitis, nummular dermatitis, lichen simplex and neurodermatitis. Dermatitis mostly results in skin surface changes including dryness, blisters and crusting. The distribution helps determine the aetiology.
- Nodular prurigo and prurigo simplex present as multiple intensely itchy nodules and papules respectively, distributed symmetrically usually either in the cape area (shoulders and proximal upper limbs) or the distal limbs.

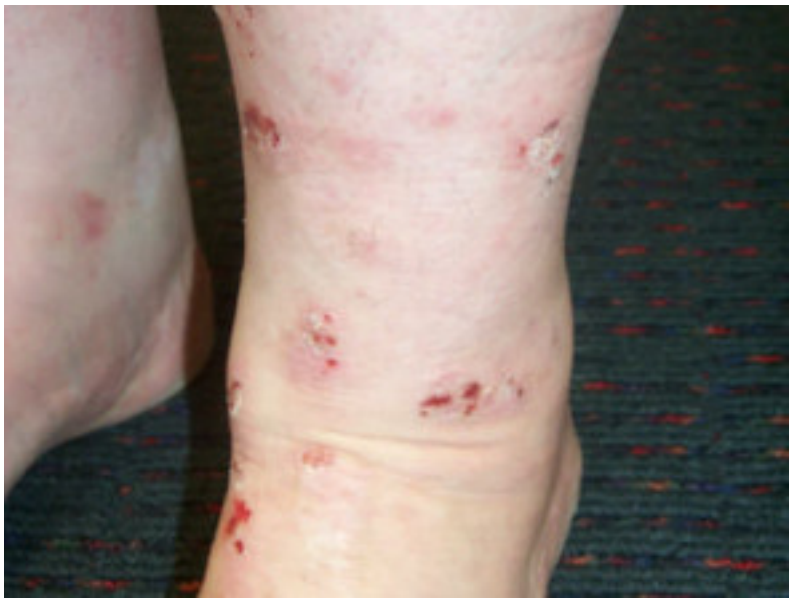
Scratched skin diseases



Excoriated insect bites



Excoriated atopic dermatitis



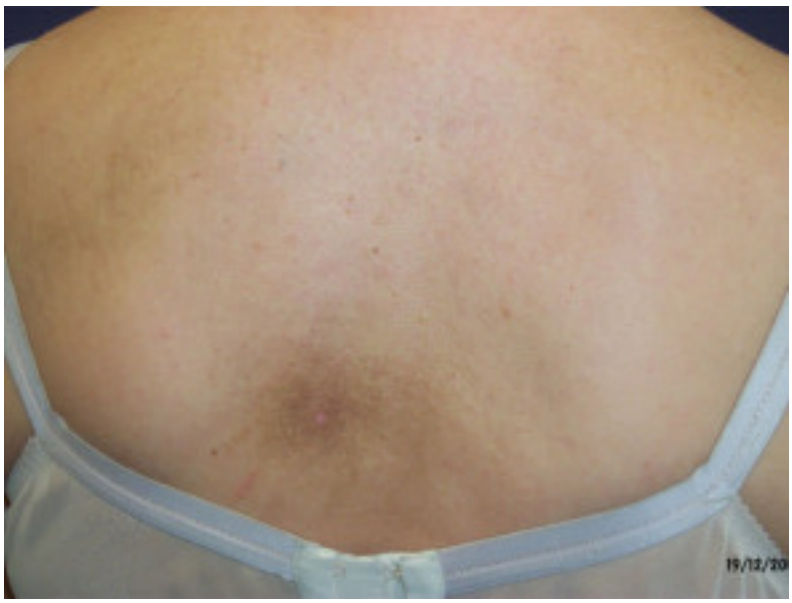
Excoriated nodular prurigo

Localised itch with no primary rash may be due to nerve root impingement resulting in dermatomal neuropathic pruritus. Scratching or rubbing the affected areas may result in secondary hyper/hypopigmentation and lichen simplex (localised lichenified eczema).

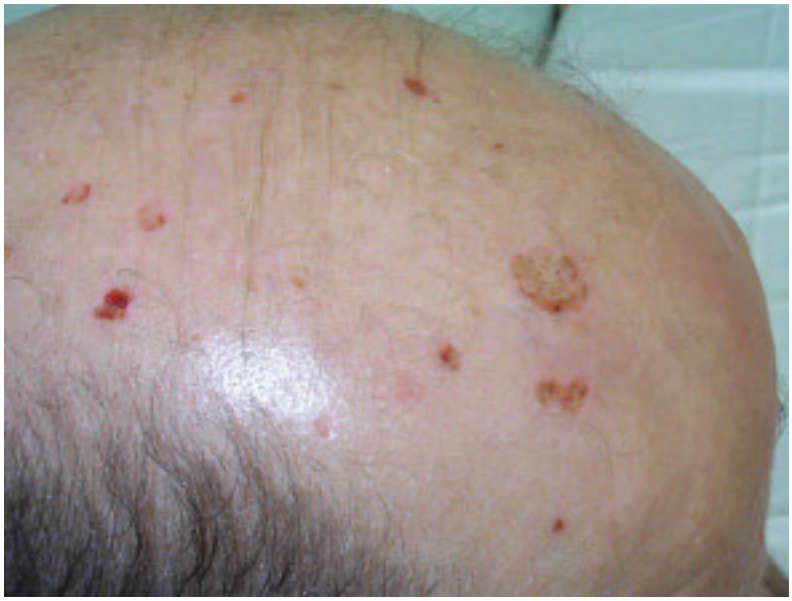
- Notalgia paraesthetica T2-6
- Meralgia paraesthetica L4
- Brachioradial pruritus C4-5
- Pudendal neuralgia S2-4
- Postherpetic neuralgia
-

Treatment may include non-steroidal anti-inflammatory drugs, tricyclic antidepressants such as amitriptyline and physiotherapy.

Neuropathic pruritus



Notalgia paraesthetica



Postherpetic neuralgia

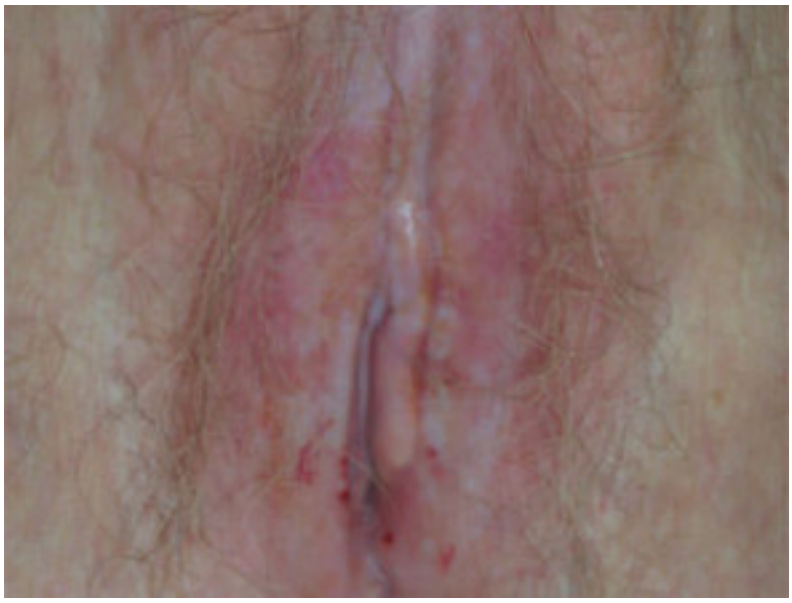
Some regions are particularly prone to chronic itching.

- Pruritus vulvae (itchy vulva) should be distinguished from vulvodynia (burning vulvar pain). Often related to candida, infection with herpes simplex and human papilloma virus should also be considered. Dermatitis can be primary or secondary. Lichen sclerosus favours this site.
- Pruritus ani may be provoked by infection with streptococci, staphylococci, candida and herpes or infestation by threadworms.

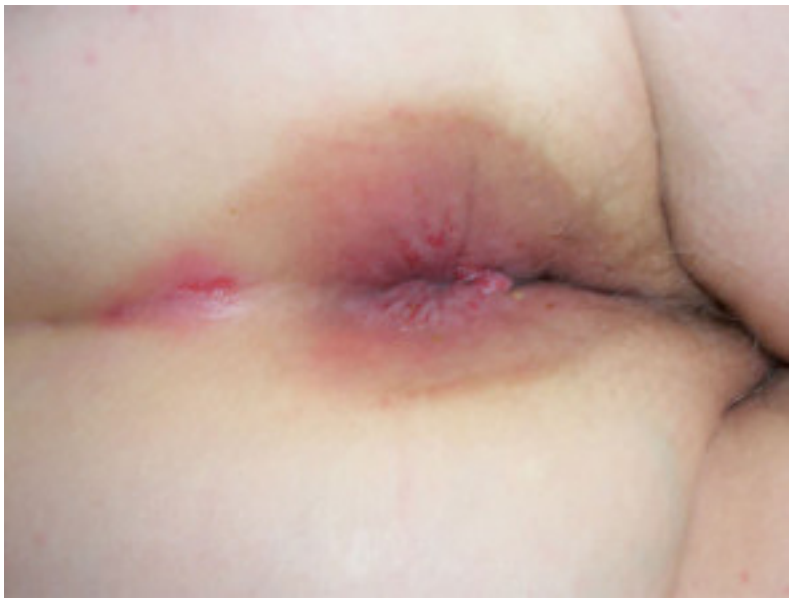
Anorectal disease, fissures, poor hygiene and dietary influences should be considered.

- Scalp itch in children is most often due to pediculosis but in adults is more likely caused by pityriasis capitis (dandruff), seborrhoeic dermatitis or psoriasis.
- An itchy chest and/or back in an elderly man may be due to Grover disease (acantholytic dermatosis), resulting in crops of 2-3 mm crusted papules.

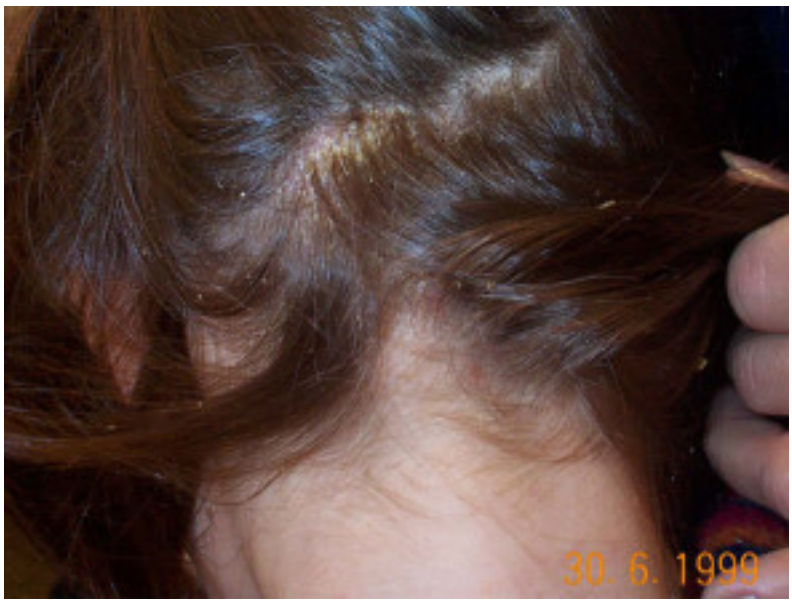
Localised itch



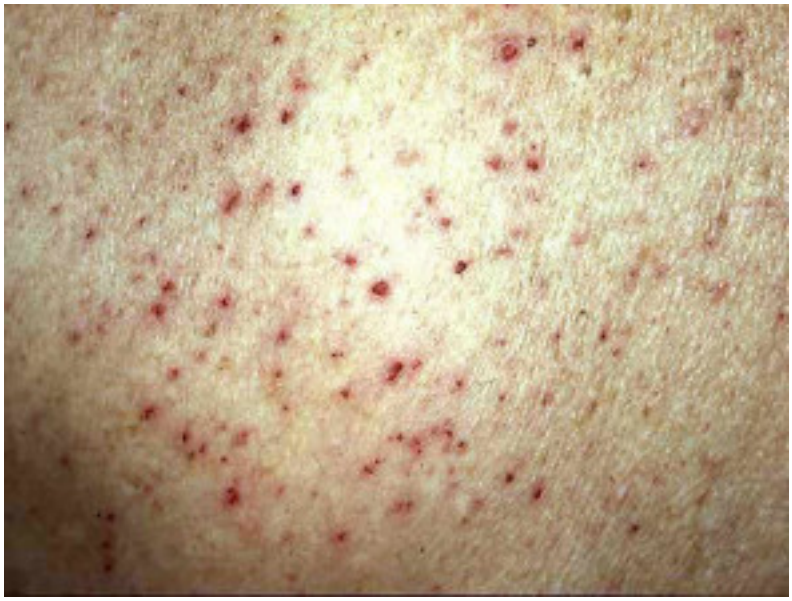
Pruritus vulvae



Pruritus ani



Scalp itch



Excoriations

Generalised itchy rashes

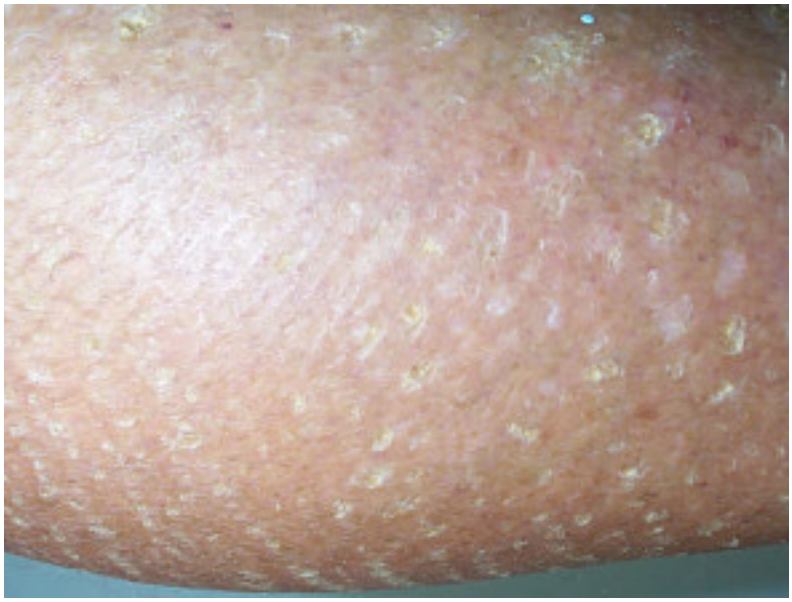
A few primary skin disorders are intensely itchy even when there isn't much rash to see.

- Winter itch affects the elderly and is thought related to xerosis (dry skin). Check for hypothyroidism and treat as dermatitis.
- Scabies: look for burrows and nodules between fingers, wrists and elbows. Signs may be subtle. Immune response results in the rash and

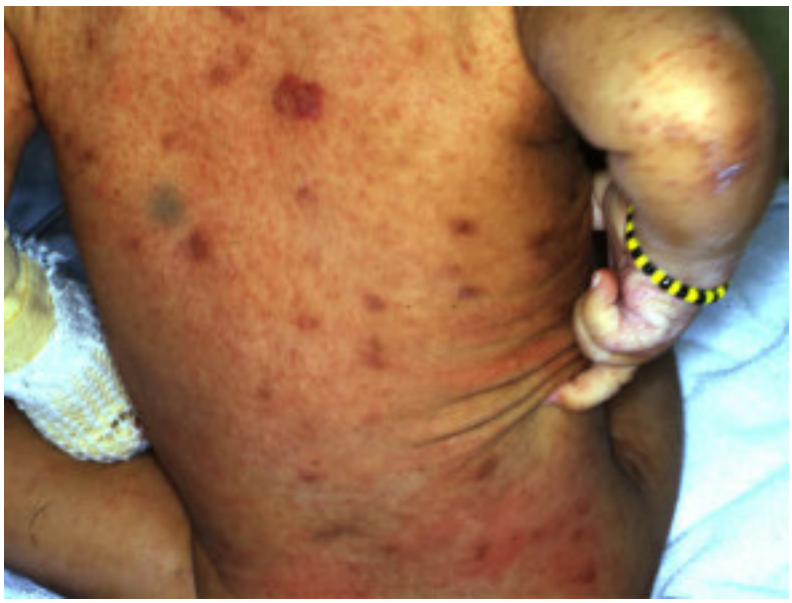
generalised itch; mediators are toxic to mites and evoke scratch. Treat with insecticides.

- Dermatitis herpetiformis is an immunobullous disease affecting extensor surfaces due to gliadin hypersensitivity and associated with gluten enteropathy.
- Urticaria: shifting itchy wheals that may be absent at the time of examination or may not occur at all. Urticaria tends to result in rubbing rather than scratching.

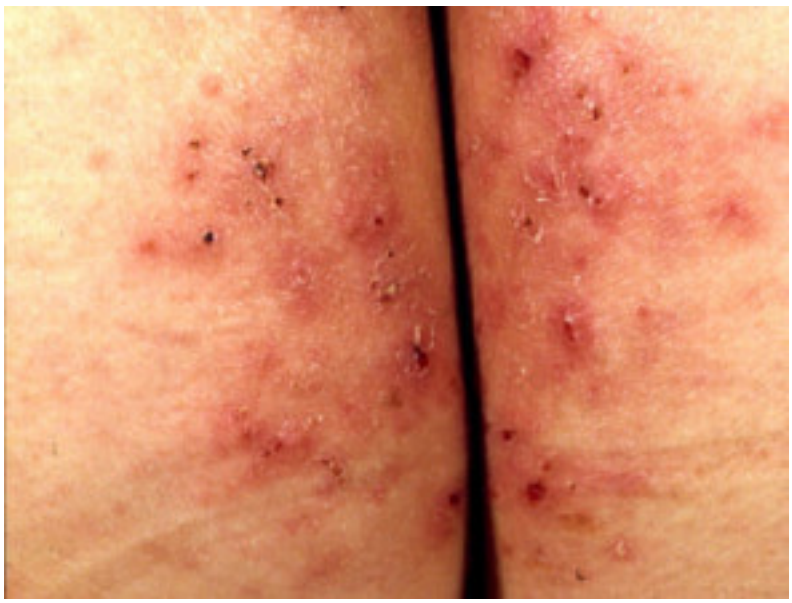
Generalised itch



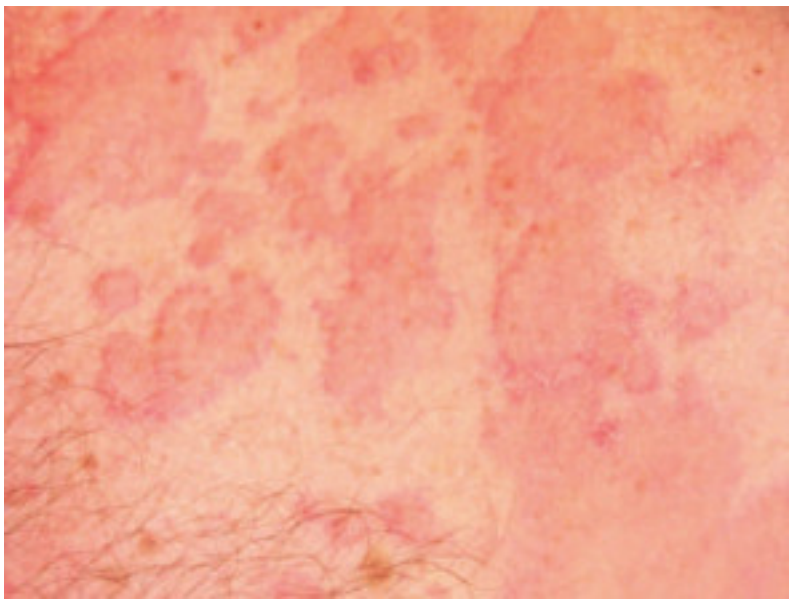
Dry skin can be itchy



Scabies



Dermatitis herpetiformis



Urticaria

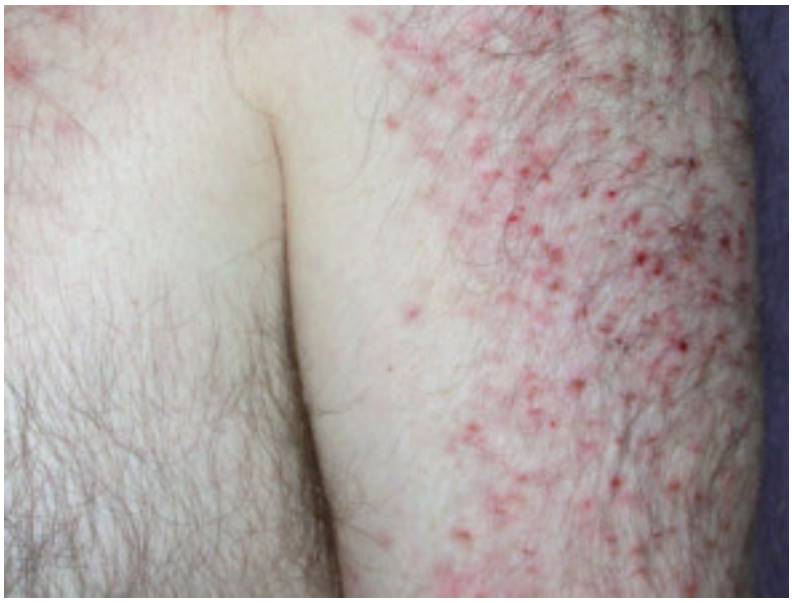
Generalised itch without primary skin disease

Itch may due to various systemic diseases, but in most cases the mechanism is not understood and symptoms may be very severe. Increased opioidergic tone may have a role.

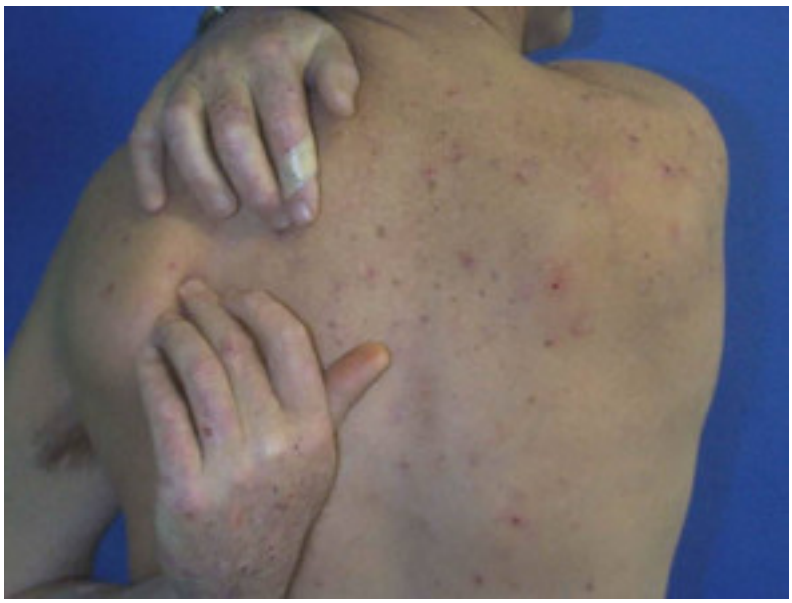
- Biliary or obstructive liver disease especially primary biliary cirrhosis
- Chronic renal failure especially in dialysis patients

- Haematological disease especially iron deficiency, polycythaemia rubra vera, lymphoma and myeloma
- Advanced malignancy
- Thyroid disease (hypo- or hyper-)
- Human immunodeficiency virus infection
- Anorexia nervosa
- Neurological disease including some strokes, multiple sclerosis and brain tumours (neurogenic origin)
- So-called ‘neurotic excoriations’ of psychogenic or central nervous system origin
- Drugs: opioids, chloroquine, minocycline, acetylcholinesterase (ACE) inhibitors, aspirin, antidepressants, hydroxyethyl starch plasma expanders and others
-

Pruritus of systemic origin



Pruritus associated with



Chronic renal failure

Treatment of pruritus

Clearly it is important to identify the cause and treat the primary skin condition and dermatitis, which may be secondary to scratching and rubbing. General measures include:

- Advise short nails to minimise damage
- Wear cool, loose and smooth clothing
- Apply emollients to treat dryness and relieve itch
- Cool affected areas using water and creams containing menthol and/or camphor
- Burning-hot showers frequently provide relief
- Relaxation: music therapy, self-hypnosis and exercise programmes may help
- Phototherapy with UVB and/or UVA

Specific topical antipruritic agents include:

- Topical steroids; use mild products to avoid skin atrophy
- Topical anaesthetics/antihistamines; these may sensitise, i.e. provoke contact allergic dermatitis
- Crotamiton, a scabicide, has antipruritic properties
- Strontium chloride lotion is useful for localised itch

Oral agents

- Conventional antihistamines may act centrally as they are sedative
- Antidepressants such as amitriptyline have antihistamine and neural effects
- Slow release serotonin re-uptake inhibitors and in some instances anti-psychotics may help
- Opiate antagonists especially naltrexone
- Thalidomide probably is effective because of its potent anti-TNF action.

Cutaneous pain

Pain obviously arises when the skin is injured or from a painful infection or skin disease. Pain information is conveyed by a peripheral system of slow unmyelinated C and faster myelinated A-delta neurons. The cell bodies of these fibres exist in the posterior (dorsal) roots of the spinal cord. They extend another process into the spinal cord itself and bundles of these form Lissauer's tract. The fibres terminate in the dorsal horns of the spinal column. Secondary neurons then convey pain information to centres in the brain-stem, thalamus and cerebral cortex.

Biochemical mediators released as a result of tissue injury (noxious stimuli, mechanical injury and extremes of temperature) have been implicated in nociceptive activation and sensitisation (hyperalgesia). These mediators include serotonin, bradykinin, histamine, prostaglandins, substance P (sP) and various ions (H+ or K+). Substance P, synthesised by cells of the spinal ganglia, has also been identified at the peripheral terminal of C fibres.
Pain in the skin due to stimulation of the myelinated A fibres results in sharp, pricking pain and stimulation of the unmyelinated C fibres results in stinging, burning pain.

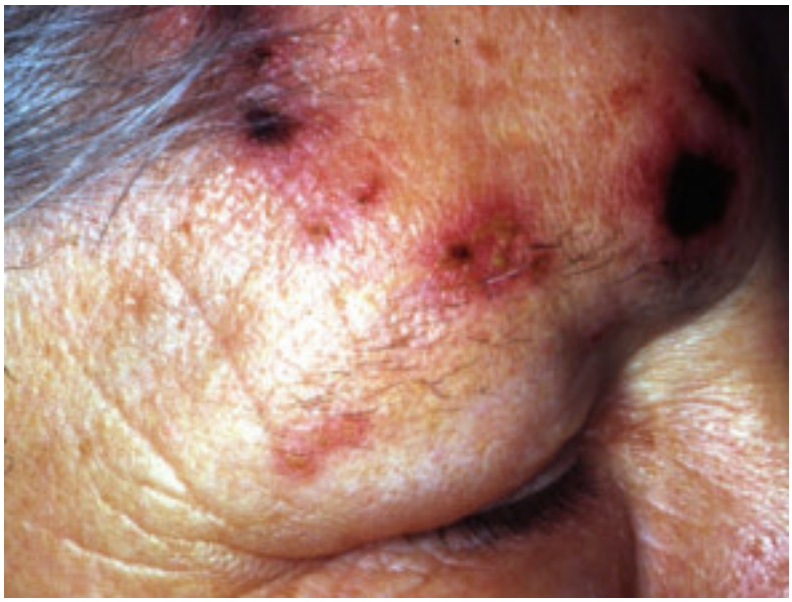
Deep pain is caused by injury of visceral organs and deep muscle and skeletal structures. It is characteristically diffuse, aching and poorly localised. However, pain within deep structures may also be referred to the skin.

Specific cutaneous neuropathic syndromes include:

- Postherpetic neuralgia
- Trigeminal neuralgia
- Pudendal neuralgia
- Vulvodynia
- Glossodynia

Postherpetic neuralgia is a radiculopathy arising from Herpes zoster (shingles). This double-stranded DNA virus establishes latent infection in dorsal root ganglion after primary chickenpox. Viral reactivation in about 15% of adults (most often over 60 years) results in dermatomal mucocutaneous blistering, neuralgia and sometimes, transverse myelitis. Neuralgia may begin before, during or after the rash appears and persist for months after resolution of the rash. It is characterised by aching, burning, dysaesthesias and allodynia (severe pain from light touch) in the same segment as the rash. It is most likely in older patients. Recovery may be partial or complete (90% within six months). Treatment is with topical anaesthetics and analgesics and oral analgesics, tricyclic antidepressants, gabapentin and anticonvulsants. Transcutaneous stimulation and physiotherapy may also be helpful. In some cases systemic steroids have a role. Identification and prompt treatment of early herpes zoster with oral antiviral agents prevents postherpetic neuralgia or reduces its severity.

Zoster ophthalmicus



Herpes zoster ophthalmicus

Trigeminal Neuralgia (tic douloureux) results in frequent sharp facial pain, most often in the region of the second division of the trigeminal nerve. The pain is sometimes mistaken for disease of the teeth, gums and sinuses. The cause is unknown.

Pudendal neuralgia affects the perineum (mons pubis, vulva or scrotum, anal area or medial thighs) and is typically more severe when sitting. It may be unilateral or bilateral and result in burning discomfort, sharp stabbing pain, hyperalgesia or dysaesthesia. It is one cause of vulvodynia (burning pain

affecting the vulva) or scrotodynia (affecting the scrotum). Vulvodynia may also present as localised tenderness in the introitus (vulvar vestibulitis) or as more generalised dysaesthetic vulvodynia associated with inflammatory processes.

Glossodynia or burning mouth syndrome presents as unexplained, prolonged pain and/or burning inside the oral cavity. It is often accompanied by dryness, paraesthesia, and changes in the sensations of taste and smell, and occurs more frequently in middle-aged women. It appears in some to be related to menopause, salivary disturbances, candidiasis, or diabetes.

Examination of the skin

The entire skin surface should be examined as well as hair, nails and mucosal surfaces. This may require a chaperone. Explain the necessity of complete examination to the patient. Use an appropriate light source and magnification. Identify the presenting complaint and incidental skin conditions (especially skin cancers). Have you found solitary lesions or a widespread rash?

Assess distribution, morphology and arrangement i.e. the number, size and colour of skin lesions, which sites are involved, their symmetry, shape and arrangement. What types of lesions are present? (The next section of this course outlines the correct terminology to use.)

Touch the skin to palpate individual lesions and more diffuse rashes, noting surface and deep characteristics. Does the lesion involve epidermis, dermis or subcutis? If scaly, does the surface flake off easily? If crusted, what is underneath?

Look carefully for signs of systemic disease, such as xanthomas (hyperlipidaemia), café-au-lait macules (neurofibromatosis), acanthosis nigricans (insulin resistance) etc.

Examine the hair and nails (described in later sections of this course). Mucosal surfaces include conjunctivae, lips, gums, tongue, and buccal mucosa. Genital examinations are only required if related to the presenting complaint. Specific permission should be obtained for genital examination and a chaperone should be available to the patient.

Finally, where indicated by symptoms or clinical signs, perform a general physical examination that may include height, weight, temperature, cardiovascular/respiratory assessment and so on.

Special examinations

Specialised techniques used in examination of the skin include:

- Dermoscopy for pigmented lesions to diagnose melanoma.
- Skin biopsy for histology and direct immunofluorescence.
- Patch tests to identify type 4 contact hypersensitivity reactions.
- Skin scrapings or nail clippings for mycology (fungal infections).
- Wood's light (long wave UVA) examination for pigmentary changes and fluorescence resulting from certain infections.

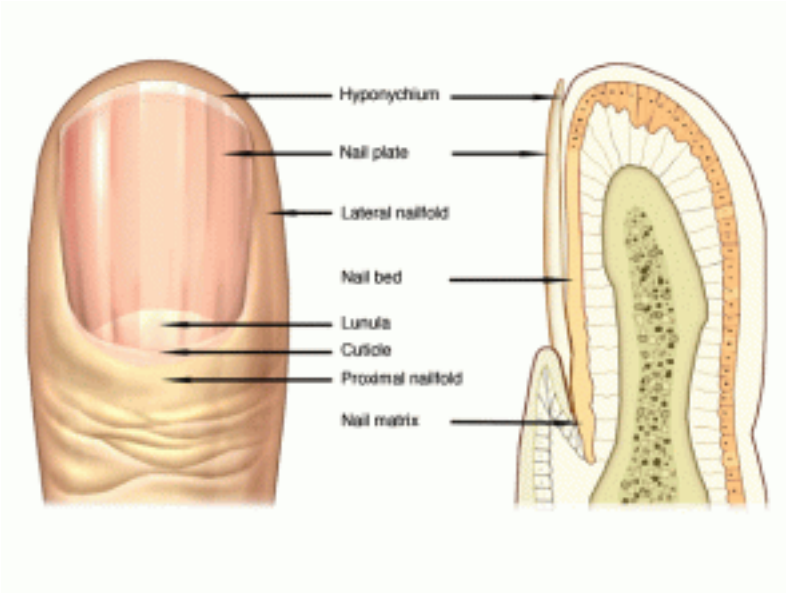
Less dermatologically specific tests include:

- Skin swabs and smears for bacteria yeast and viral infections.
- Blood tests for culture, serology, haematology, biochemistry etc.
- Urine tests for culture, biochemistry etc.
- Faecal tests for culture, occult blood testing, biochemistry
- X-rays for systemic disease, bone abnormalities
- Selected prick tests to identify Type 1 hypersensitivity reactions.

These procedures will be described in later topics where relevant to specific skin conditions.

Examination of the nails

This section provides a glossary of terms used to describe abnormal fingernails and toenails. Proper use of language is necessary for diagnosis and to communicate with other health professionals.
Nails are a specialised form of stratum corneum and are made predominantly of keratin. Their primary functions are for protection, scratching, and picking up small objects. When looking at the nails carefully inspect the nail plate and surrounding skin.



Normal nail

If the patient presents with a nail problem, it is important to ask about skin disease elsewhere and examine them generally. Fungal nail disease (onychomycosis) is nearly always associated with fungal skin disease (check feet, hands, groin). Nail changes may be the first sign of psoriasis (check scalp, elbows, knees, flexures), lichen planus (check oral mucosa, lower back, scalp, wrists, ankles), or another skin disease.

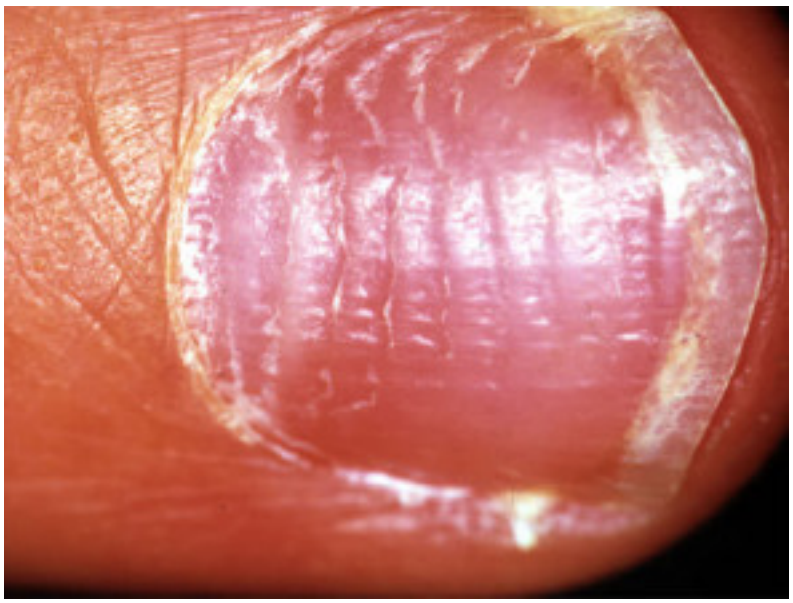
Psoriasis may result in haphazard nail pitting, onycholysis, subungual hyperkeratosis, ridging and/or yellow hypertrophied nail plate.

Eczema is associated with irregular pitting, ridging and paronychia.

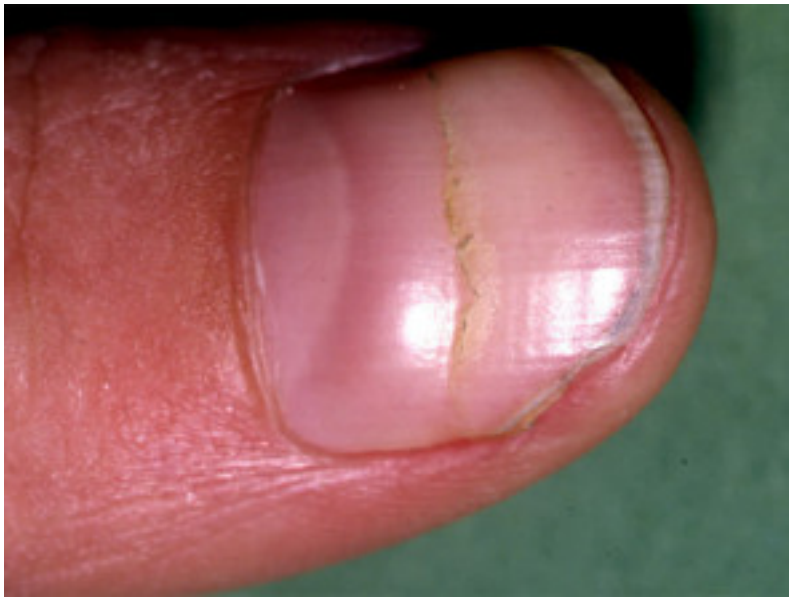
Lichen planus thins the nail plate, which may become grooved and ridged. The nail may darken, thicken or lift off the nail bed.

Abnormalities of the nail plate surface

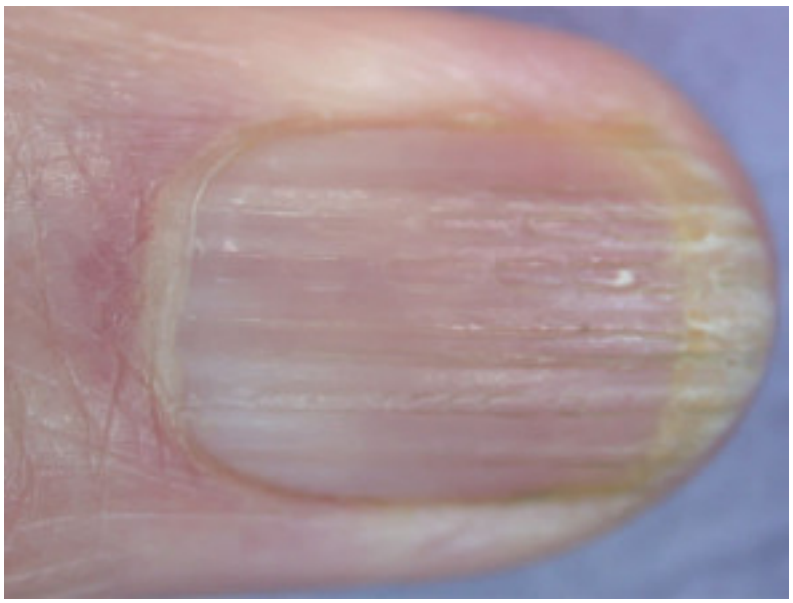
Nail plate abnormalities are often due to inflammatory conditions affecting the matrix or nail bed. Specific diagnoses may be made from characteristic appearances, which are generally self-explanatory.



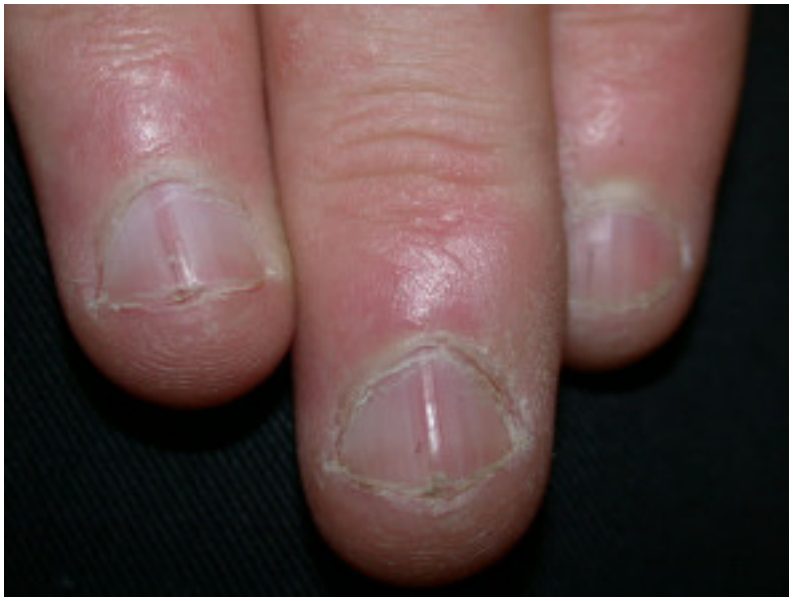
Nail pitting



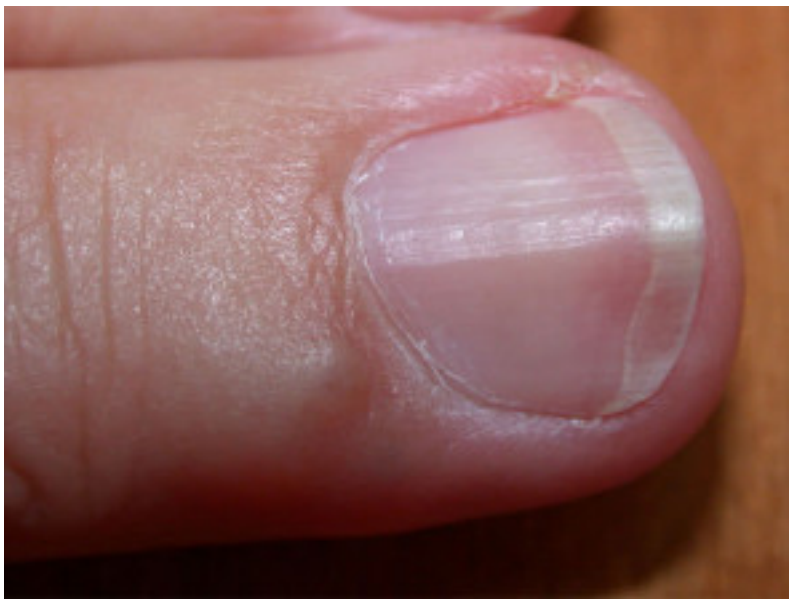
Beau line



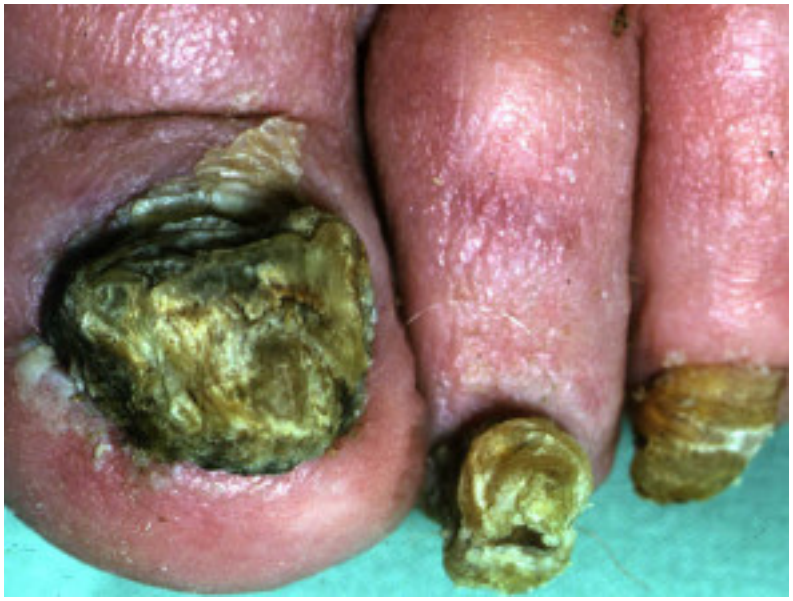
Nail ridge



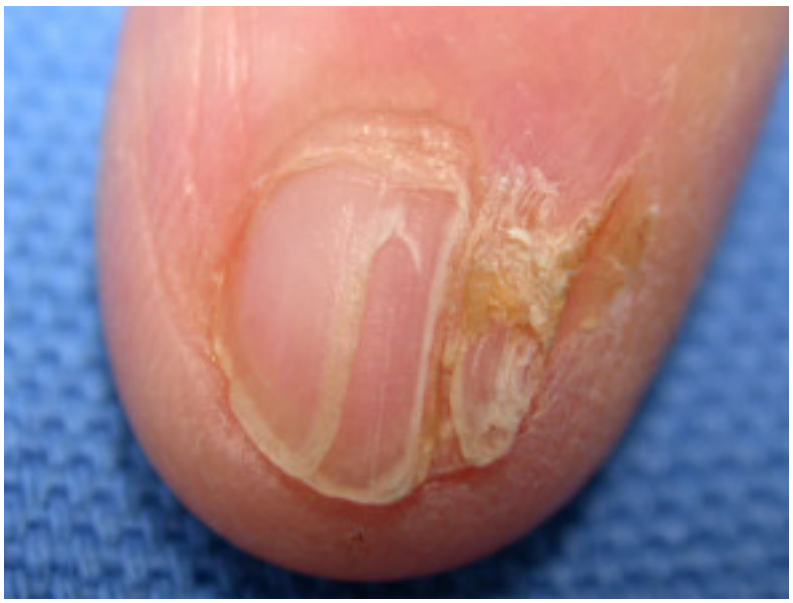
Nail notching



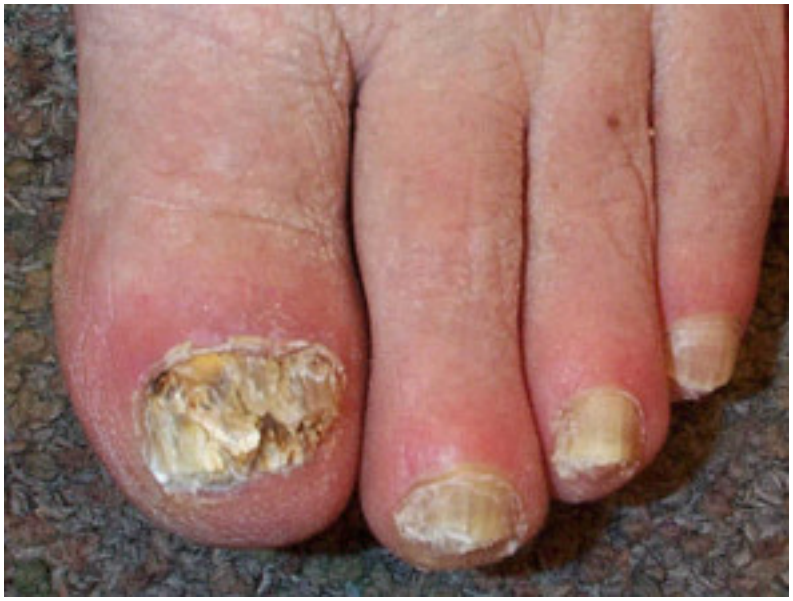
Nail groove



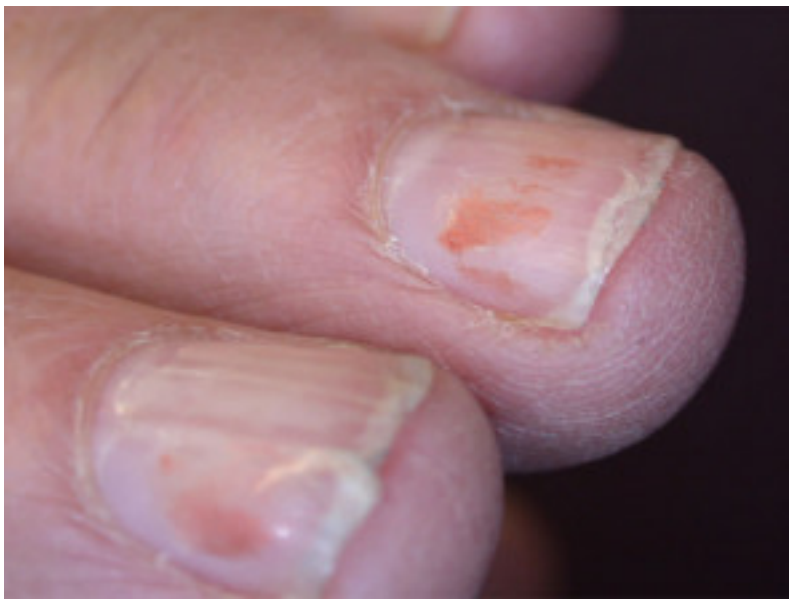
Onychogryphosis



Angelwing deformity

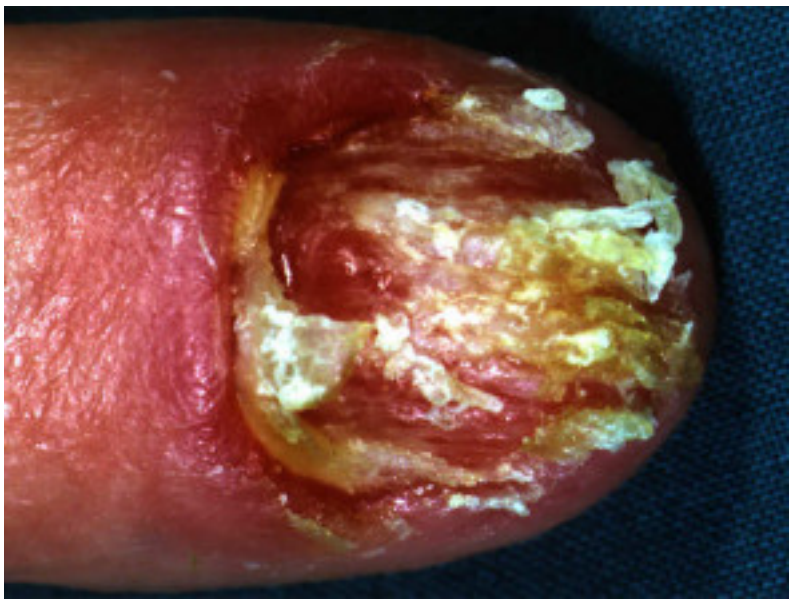


Crumbling nail



Onychoschizia

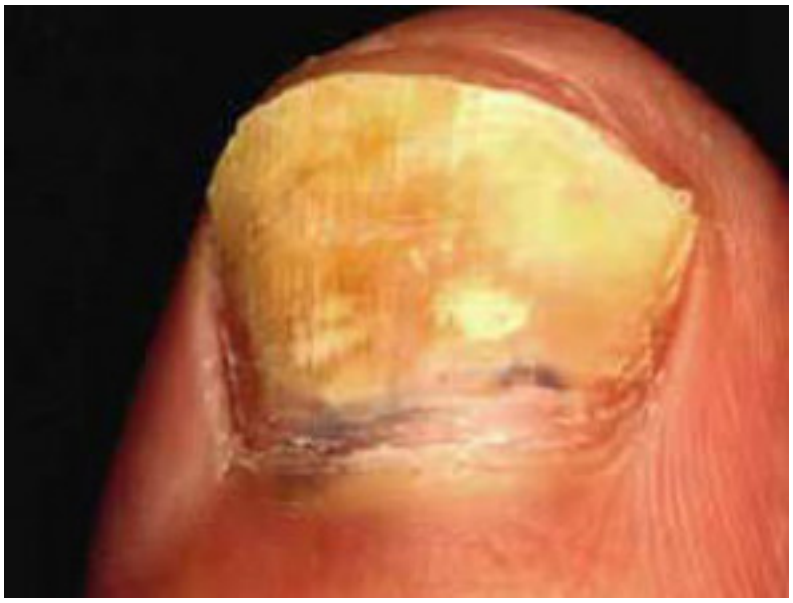
Fingernail trachyonychia in alopecia areata



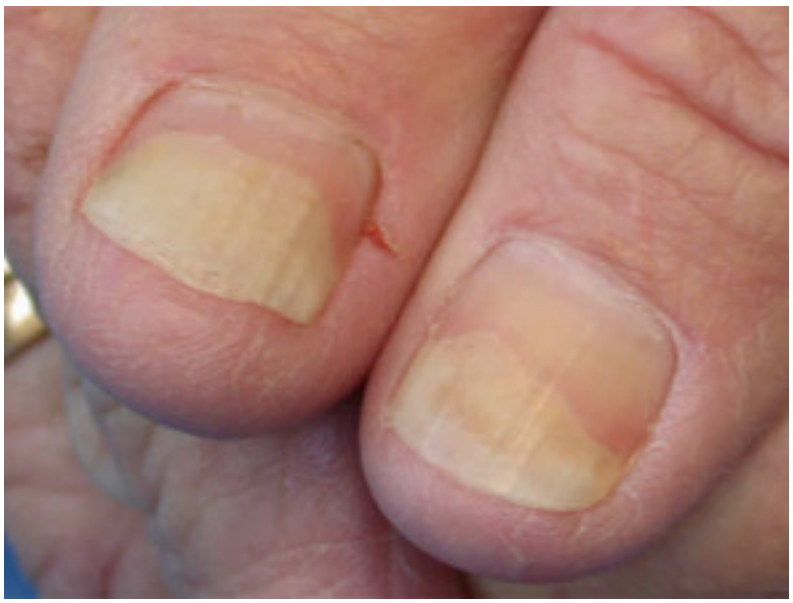
Nail melanoma

Discolouration of nails

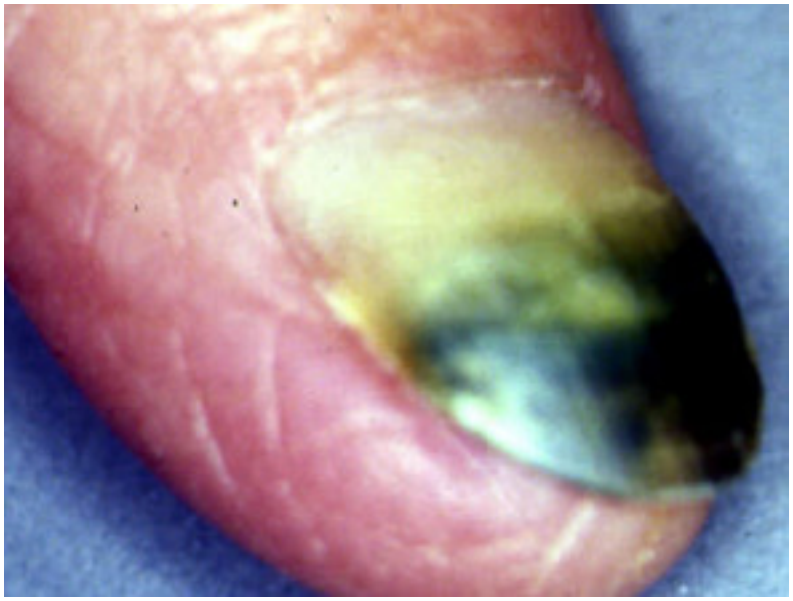
Distinguish a discoloured nail bed from a discoloured nail plate. The most important diagnosis to exclude is subungual melanoma, which presents as a pigmented linear band in the nail plate, which slowly expands at the proximal border and may extend to involve the proximal or lateral nail fold or eponychium.



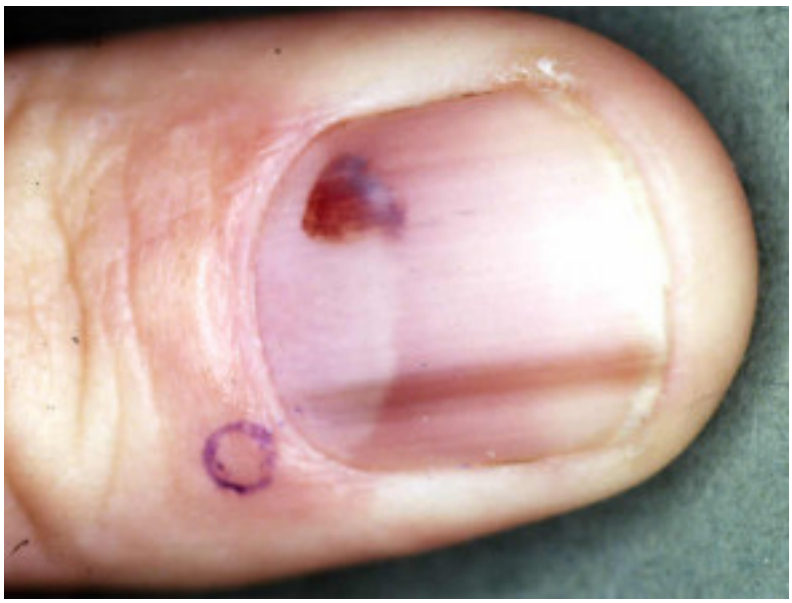
Onychomycosis



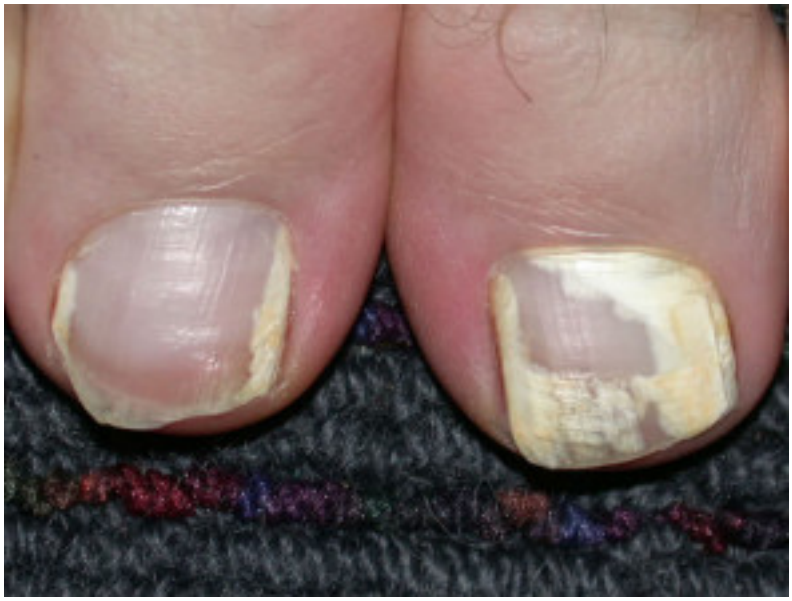
Onycholysis



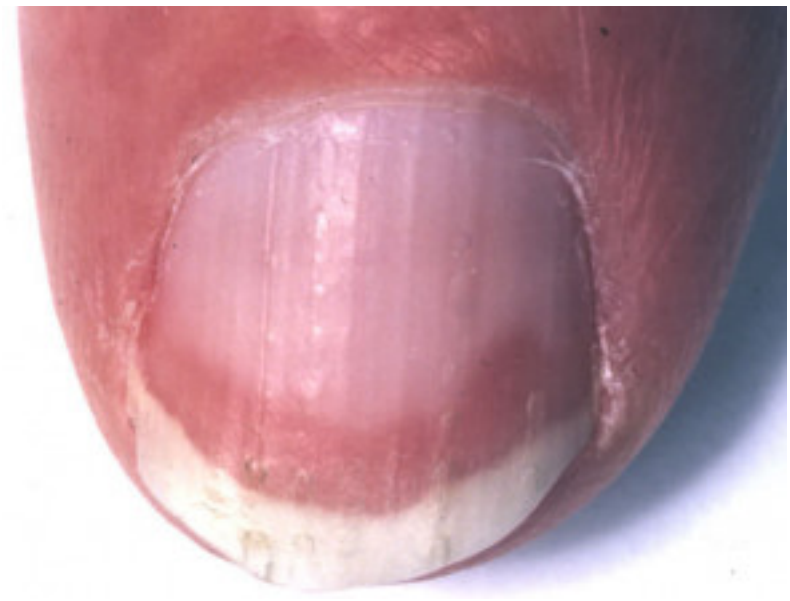
Paronychia



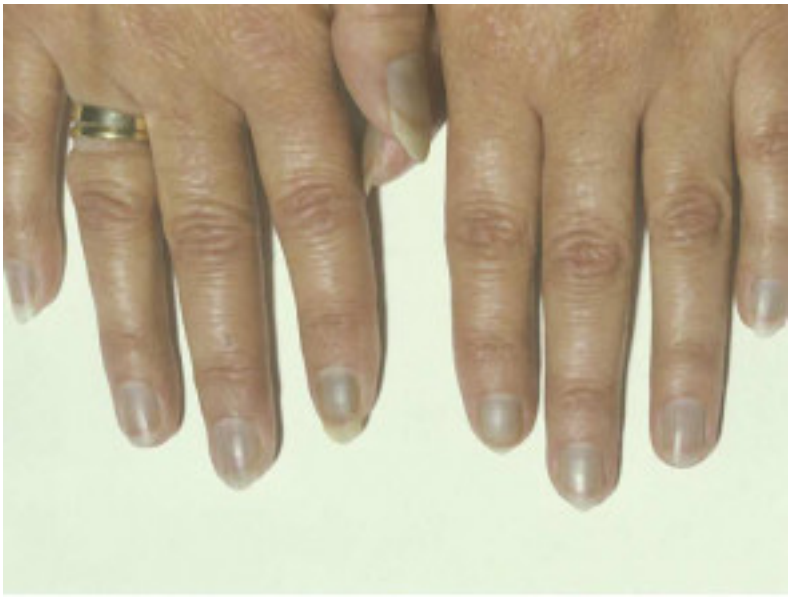
Melanocytic naevus on nail



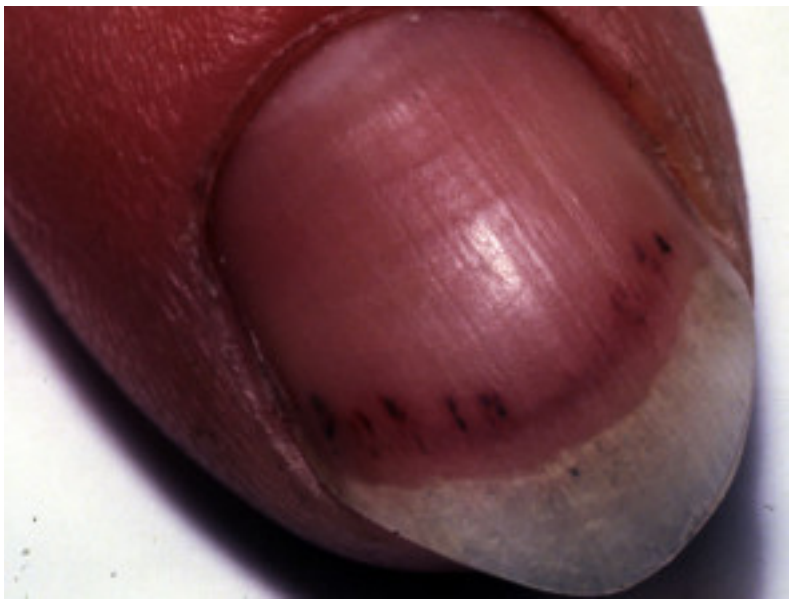
Onychomycosis



Terry nail



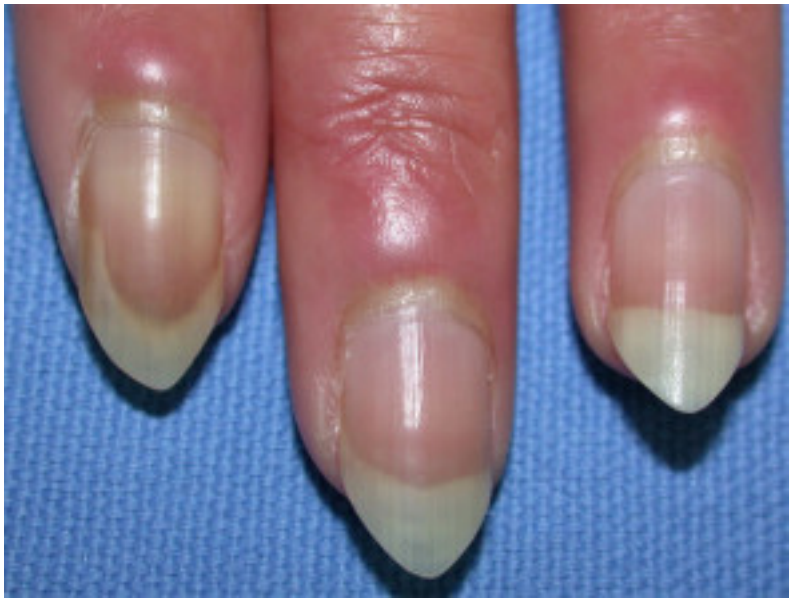
Blue nail



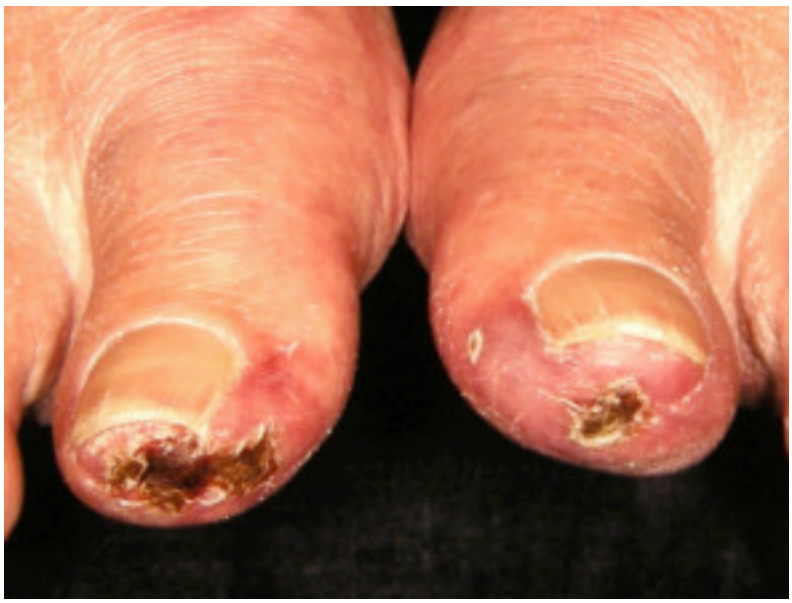
Splinter haemorrhage

Cuticle and nail fold abnormalities

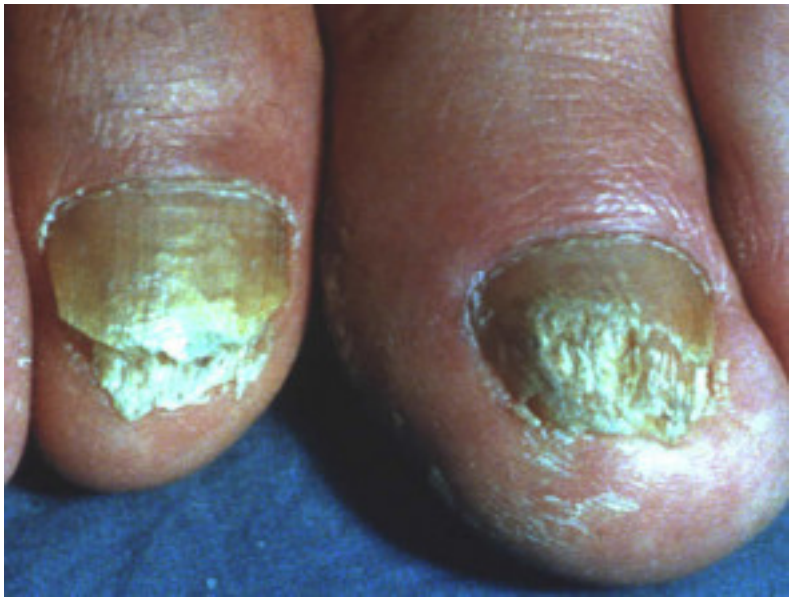
The cuticle (eponychium) is an area of keratin joining the skin of the posterior nail fold to the nail plate. Loss of cuticle results in paronychia: an acute or chronic inflammatory reaction involving nail fold (swelling, tenderness, sometimes pus).



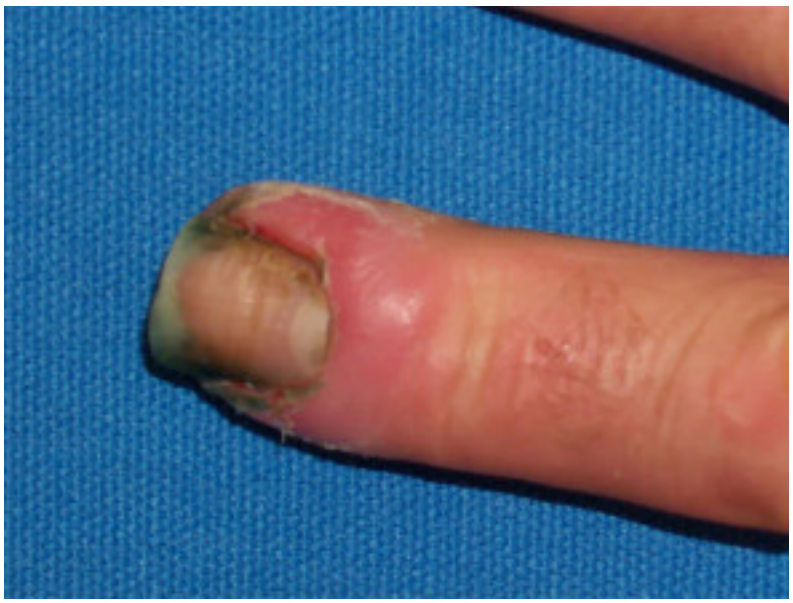
Nailfold telangiectases



Vasculitis



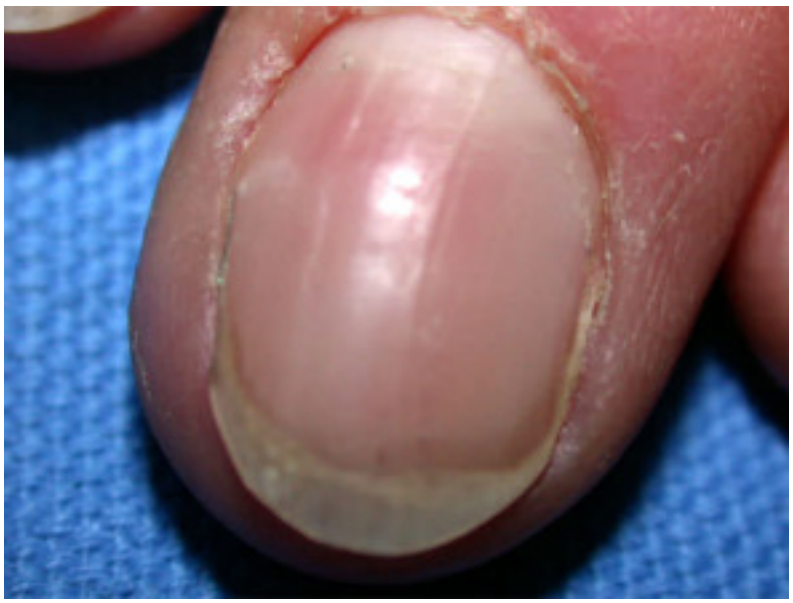
Psoriatic nail



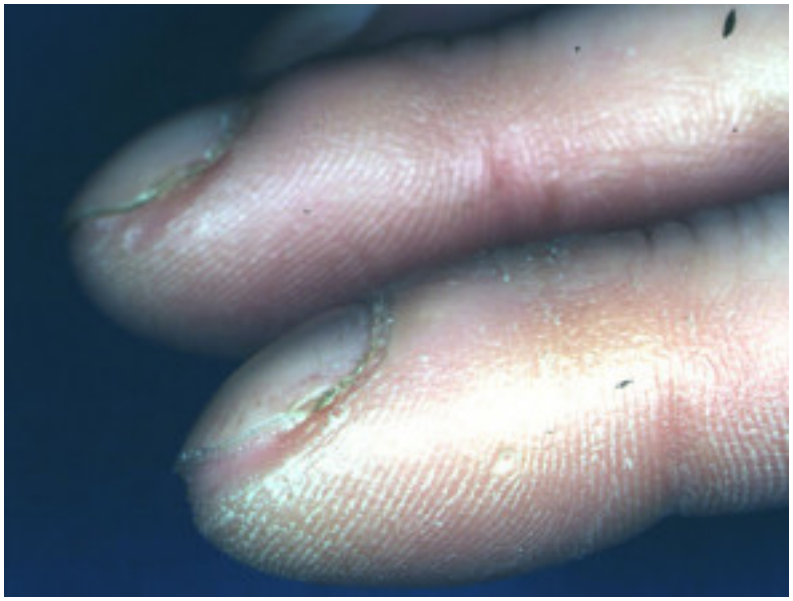
Candida paronychia

Abnormalities of nail shape

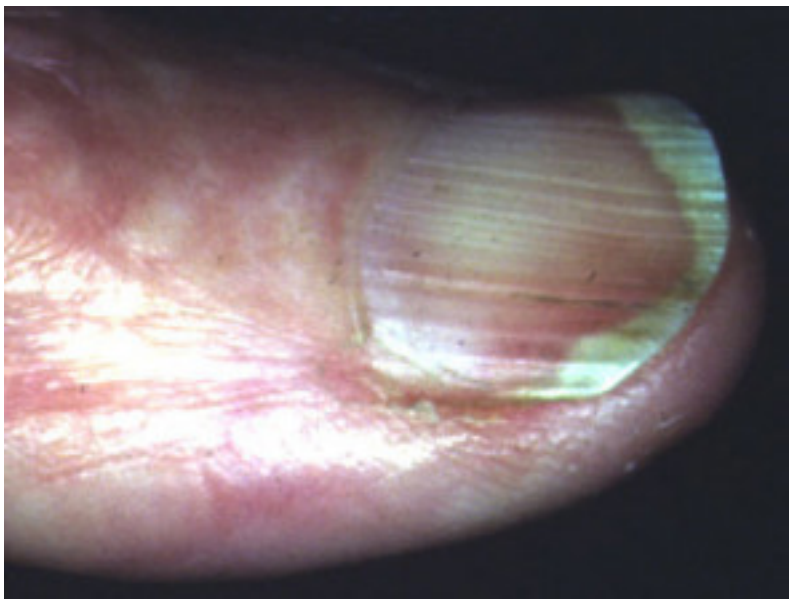
Long nail



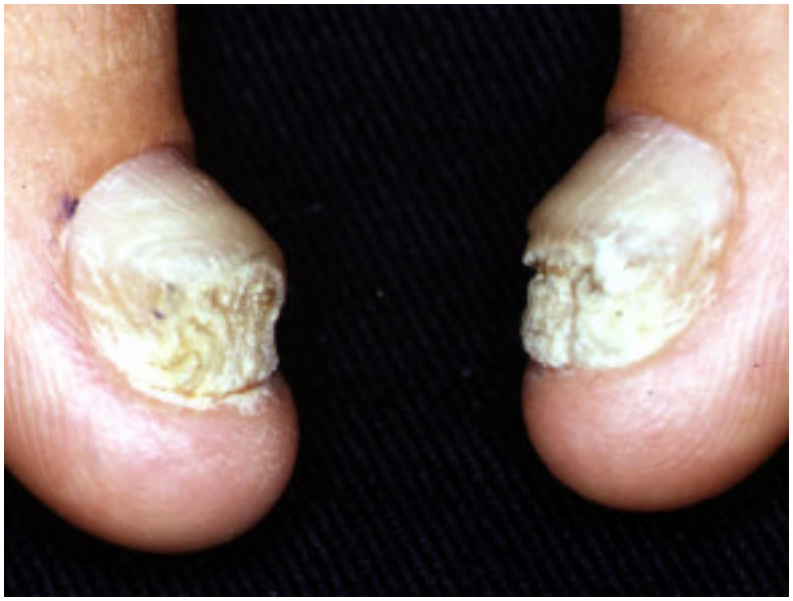
Curved nail



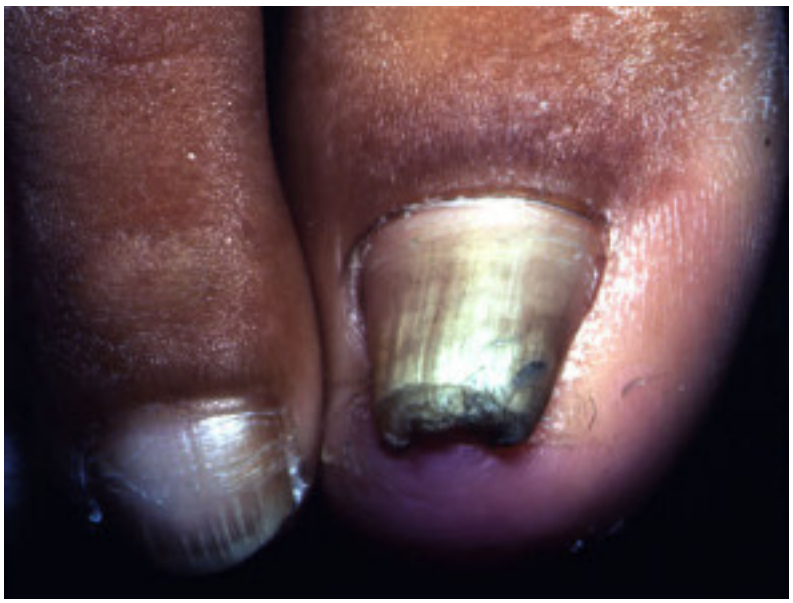
Clubbing



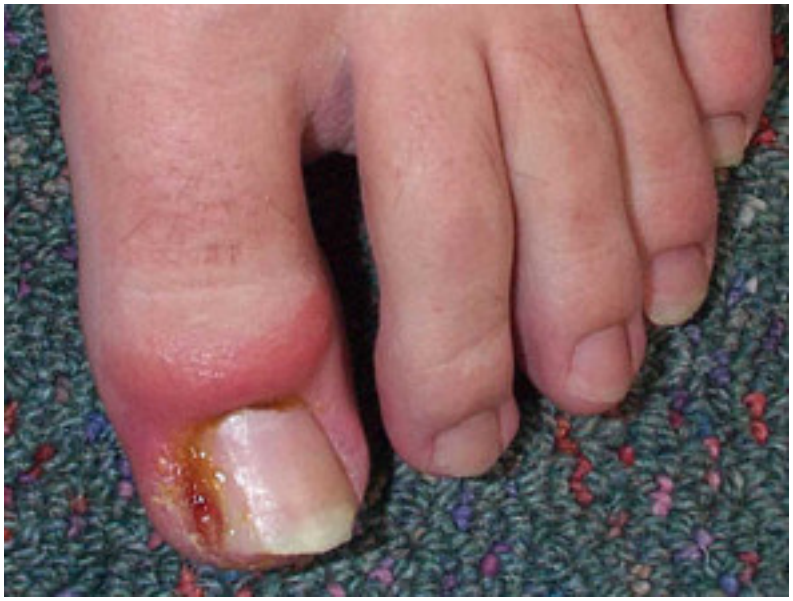
Koilonychia



Pachyonychia

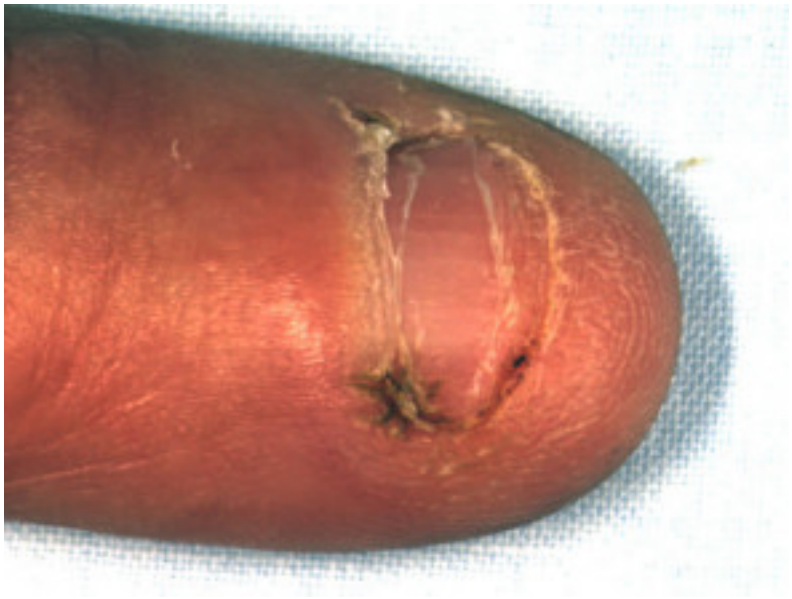


Pincer nail

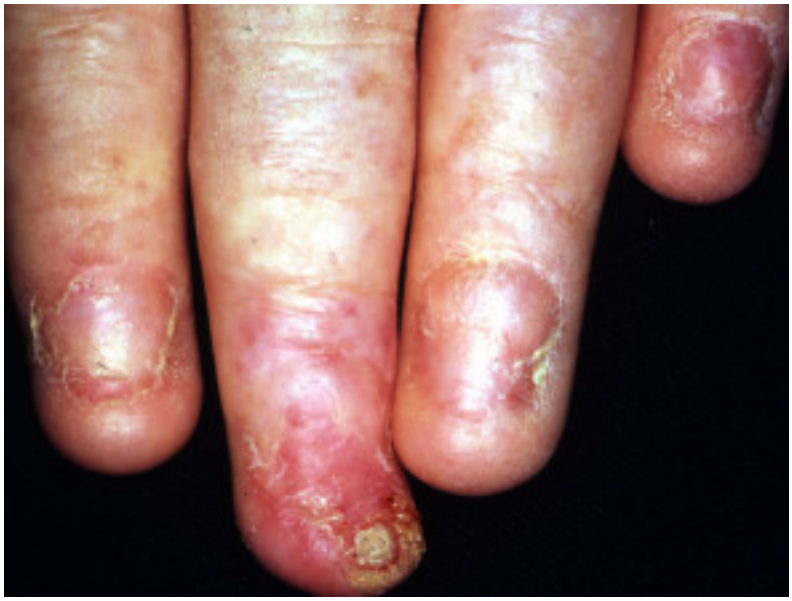


Ingrown nail

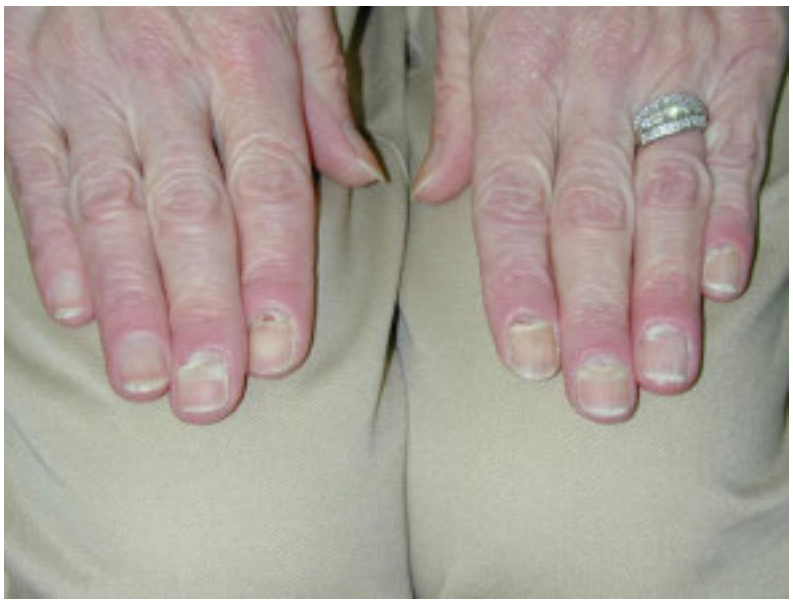
Loss of nails



Bitten nail



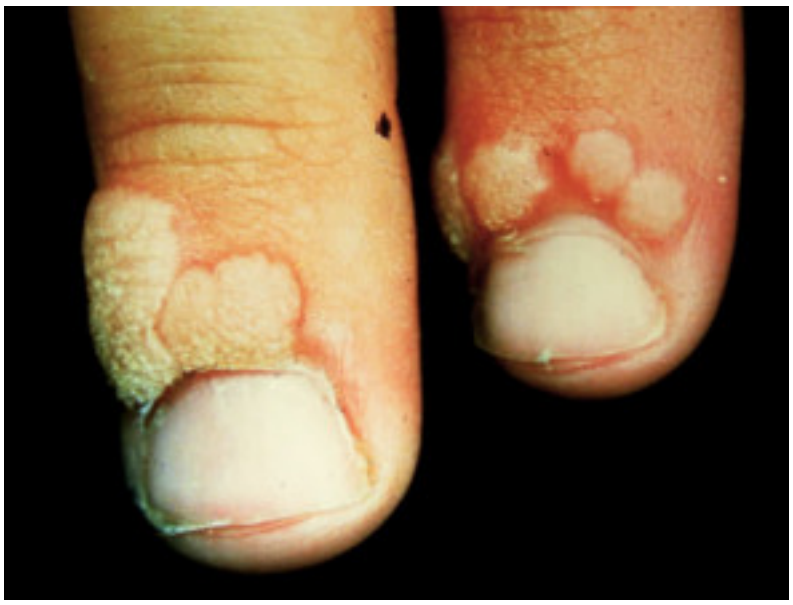
Onychomadesis

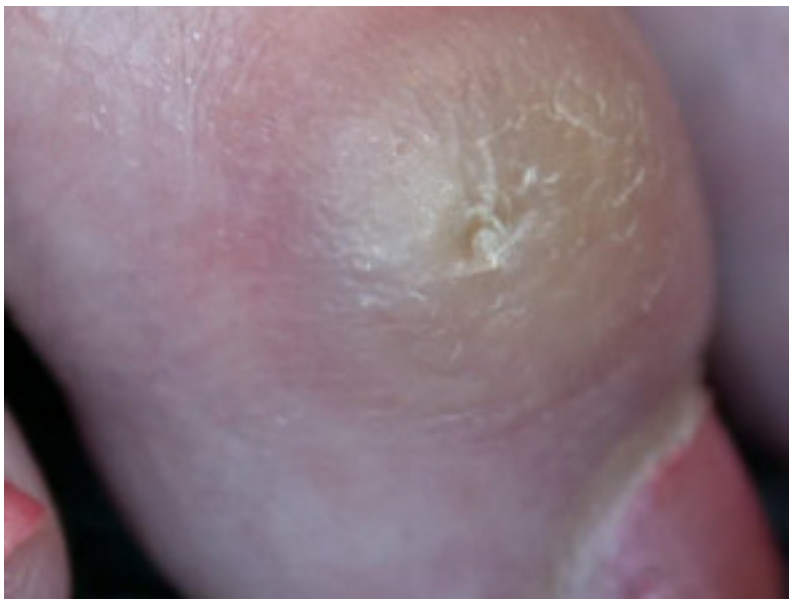


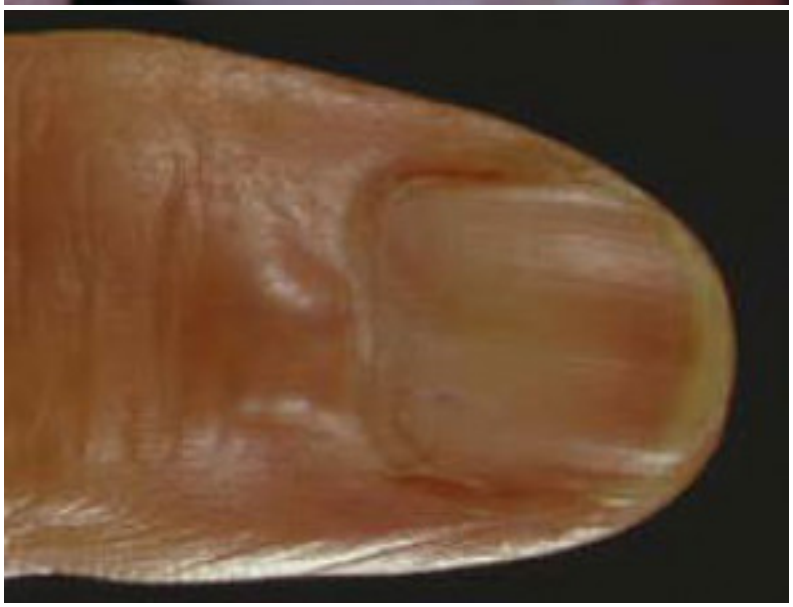
Lichen planus

Lesions around nails

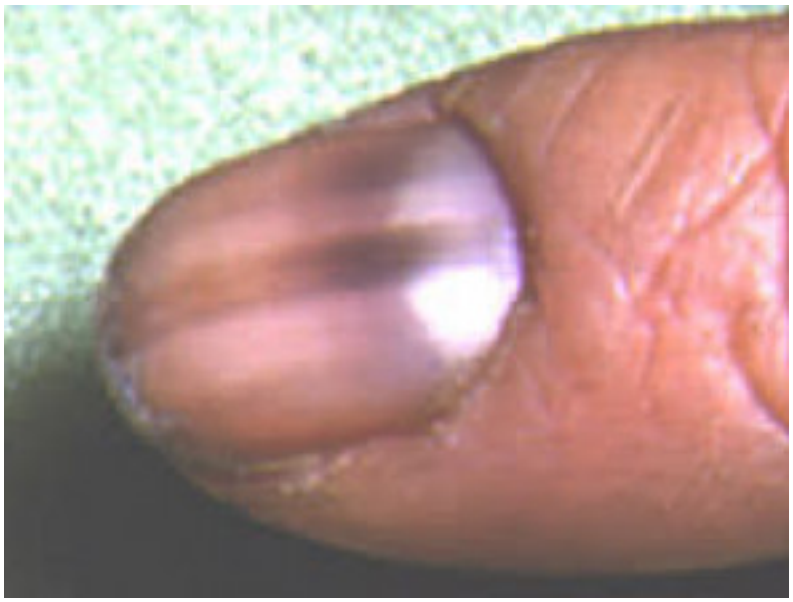
Common skin lesions may arise on the skin close to nails. Benign lesions such as myxoid cyst in the nail matrix area can affect nail growth thus causing a linear depression in the nail plate. Malignant tumours such as squamous cell carcinoma or melanoma can destroy the nail plate.



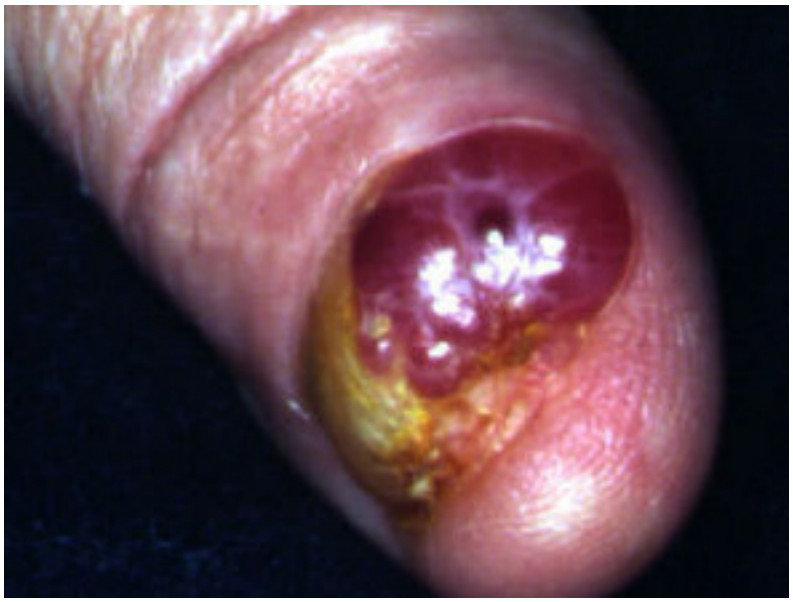




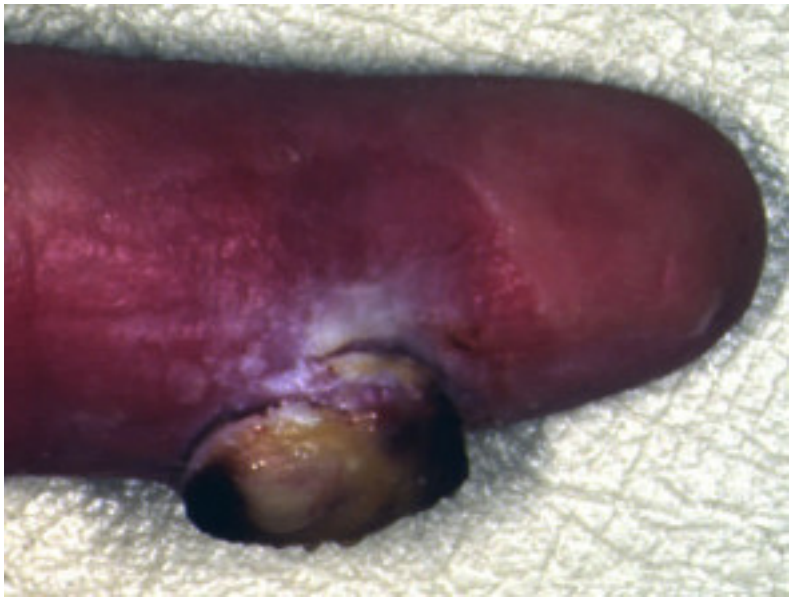
Myxoid cyst



Nail naevus



Nail melanoma



Pyogenic granuloma

Nail psoriasis

What is nail psoriasis?

Nail psoriasis is nail disease associated with psoriasis. It is also known as psoriatic nail dystrophy.

Who gets nail psoriasis?

Only 5% of patients present with typical nail psoriasis as an isolated disorder; most patients have chronic plaque psoriasis. About 50–80% have psoriatic arthritis, particularly arthritis mutilans.

Patients with nail psoriasis may be of any age or race. Nail dystrophy is often precipitated or aggravated by trauma.

What causes nail psoriasis?

Nail psoriasis arises within the nail matrix. The specific pathogenesis of nail psoriasis is unknown.

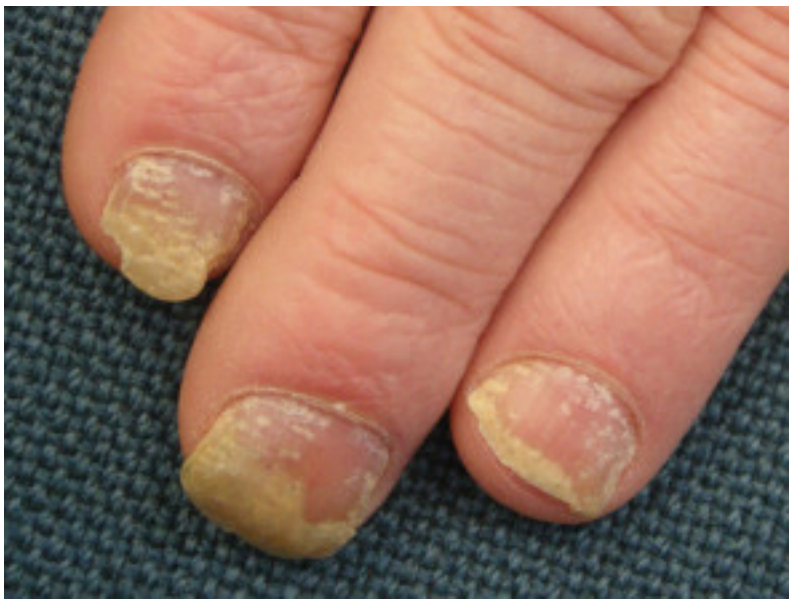
What are the clinical features of nail psoriasis?

Nail psoriasis can affect any part of one or more nails. There are often scaly plaques on the dorsum of the hands and fingers due to associated plaque psoriasis. Signs depend on the part of the nail affected. Its severity may or may not reflect the severity of the skin or joint psoriasis.

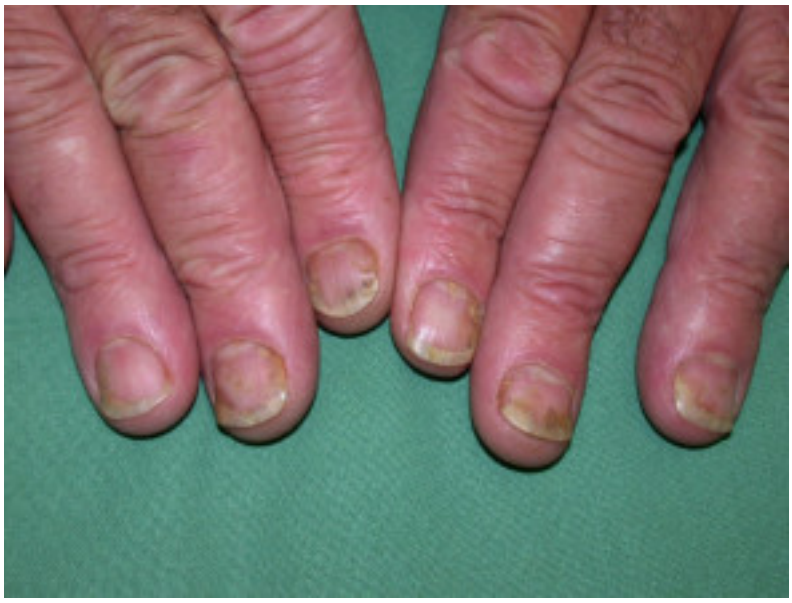
- Psoriasis can enhance the speed of nail growth and thickness of the nail plate.
- Pitting is a sign of partial loss of cells from the surface of the nail plate. It is due to psoriasis in the proximal nail matrix.
- Leukonychia (areas of white on the nail plate) is due to parakeratosis within the body of the nail plate and is due to psoriasis in the mid-matrix.
- Onycholysis describes the separation of the nail plate from the underlying nail bed and hyponychium. The affected distal nail plate appears white or yellow.
- Oil drop or salmon patch is a translucent yellow-red discolouration in the nail bed proximal to onycholysis. It reflects inflammation and can be tender.

- Subungual hyperkeratosis is scaling under the nail due to excessive proliferation of keratinocytes in the nail bed and hyponychium.
- Transverse lines and ridges are due to intermittent inflammation, causing growth arrest followed by hyperproliferation in the proximal nail matrix. The lines and ridges move out distally as the nail grows.
- Psoriatic inflammation can also lead to nail plate crumbling, splinter haemorrhage, and a spotted lunula.
- Acrodermatitis continua of Hallopeau is a rare pustular eruption that affects the nail bed, nail matrix and tips of digits.

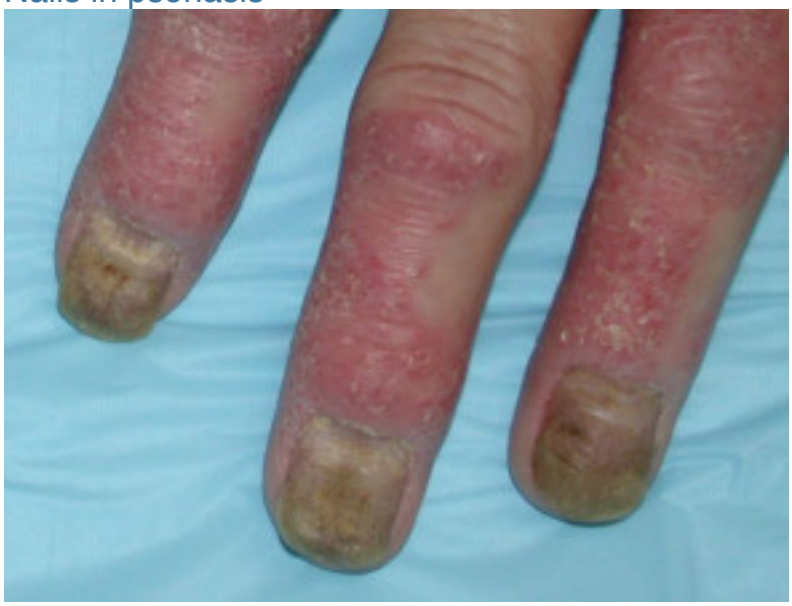
Psoriatic nails



Nails in psoriasis



Nails in psoriasis



Nails in psoriasis

Complications of nail psoriasis

Nail psoriasis is unsightly. It can also lead to:

- Pain and tenderness
- Functional disability
- Psychological distress
- Secondary bacterial infection (acute paronychia) or fungal infection (chronic paronychia, onychomycosis).

How is nail psoriasis diagnosed?

Psoriatic nail disease is readily recognised in a patient with current or prior chronic plaque psoriasis. It is frequently confused with fungal nail infection. Fungal infection can also complicate nail psoriasis.
If in doubt, or antifungal treatment is planned, nail clippings and scrapings of subungual debris should be sent for potassium hydroxide microscopy and fungal culture.

A biopsy of the proximal nail matrix is occasionally needed to confirm the diagnosis of nail psoriasis, particularly if dystrophy affects a single nail and a

tumour is a possible explanation. The biopsy can lead to permanent nail deformity.

What is the treatment for nail psoriasis?

It is difficult to treat nail psoriasis effectively.

Topical treatment must be applied to the nail matrix and hyponychium for months or years, and its effects are often disappointing. Options include:

- Calcipotriol solution twice daily
- Topical high-potency corticosteroid solution or ointment as weekend pulses under cellophane occlusion at night
- Clobetasol propionate 8% in a lacquer vehicle twice daily for 3 weeks then twice weekly.

Other options include:

- 5-fluorouracil cream twice daily
- Topical ciclosporin
- Intralesional triamcinolone acetonide injections into proximal nail folds; this is painful
- Localised phototherapy with UVB or photochemotherapy (PUVA)
- Systemic treatment with methotrexate, acitretin, ciclosporin and biologics.
-

Note: acitretin thins the nail plate and reduces its speed of growth, which can be helpful or not, depending on the type of nail psoriasis.
Topical and oral antifungal treatment may be prescribed if fungal infection is present.

Chemical or surgical avulsion therapy, complete removal of the nail, is occasionally recommended. A risk is that the regrowing nail may be as bad, or more severely affected than prior to the procedure.

How can nail psoriasis be prevented?

At this time, we do not know how to prevent nail psoriasis. Avoidance of trauma is essential.

What is the outlook for nail psoriasis?

Nail psoriasis varies in severity over time. In some patients, it resolves completely spontaneously or as a response to systemic treatment. In others, it persists long term.

Dermatitis

What is dermatitis?

Dermatitis refers to a group of itchy inflammatory conditions characterised by epidermal changes.
Dermatitis affects about one in every five people at some time in their lives. It results from a variety of different causes and has various patterns.
The terms dermatitis and eczema are often used interchangeably. In some cases, the term eczematous dermatitis is used. Dermatitis can be acute or chronic or both.

- Acute eczema (or dermatitis) refers to a rapidly evolving red rash which may be blistered and swollen.
- Chronic eczema (or dermatitis) refers to a longstanding irritable area. It is often darker than the surrounding skin, thickened (lichenified) and much scratched.

An in-between state is known as subacute eczema.

Psychological stresses can provoke or aggravate dermatitis, presumably by suppressing normal immune mechanisms.

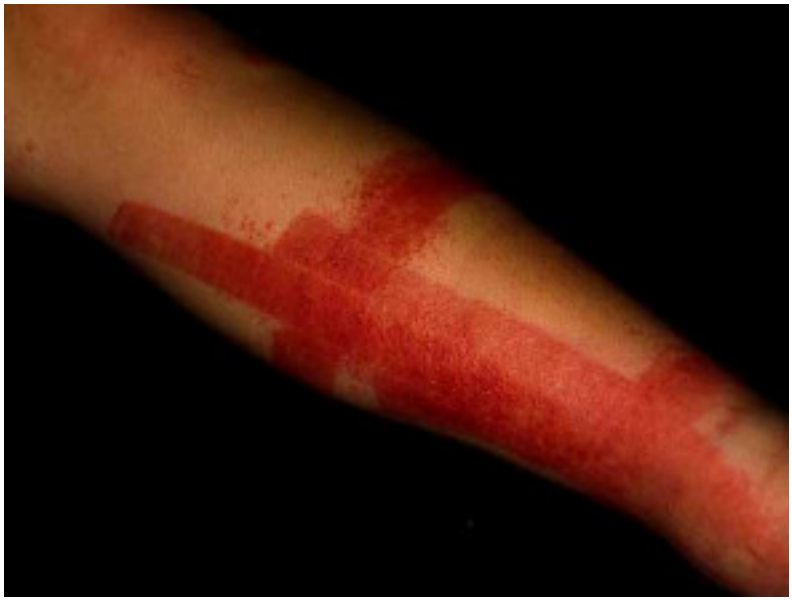
Some types of dermatitis

- Atopic dermatitis is particularly prevalent in children; inherited factors seem important, as there is nearly always a family history of dermatitis or asthma.
- Irritant contact dermatitis is provoked by body fluids, handling water, detergents, solvents or harsh chemicals, and by friction. Irritants cause more trouble in those who have a tendency to atopic dermatitis.
- Allergic contact dermatitis is due to skin contact with substances that most people don't react to, most commonly nickel, perfume, rubber,

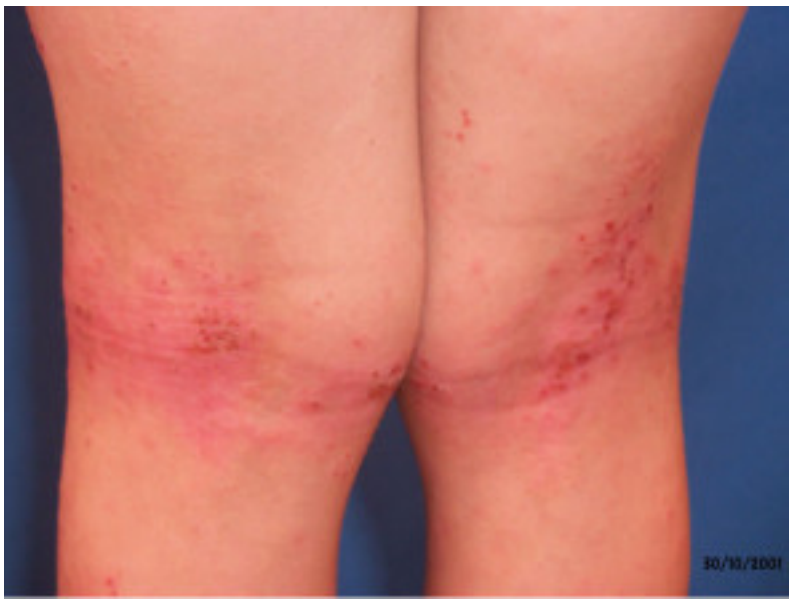
hair dye or preservatives. A dermatologist may identify the responsible agent by patch testing.

- Dry skin: especially on the lower legs, may cause asteatotic dermatitis, also called eczema craquele.
- Nummular dermatitis (also called 'discoid eczema') may be set off initially by an injury to the skin: scattered coin-shaped irritable patches persist for a few months.
- Seborrhoeic dermatitis and dandruff are due to irritation from toxic substances produced by Malassezia yeasts that live on the scalp, face and sometimes elsewhere.
- Infective dermatitis seems to be provoked by impetigo (bacterial infection) or fungal infection.
- Gravitational dermatitis arises on the lower legs of older people, due to swelling and poorly functioning leg veins.
- Otitis externa – dermatitis affecting the external ear canal
- Meyerson naevus – dermatitis affecting melanocytic naevi (moles)

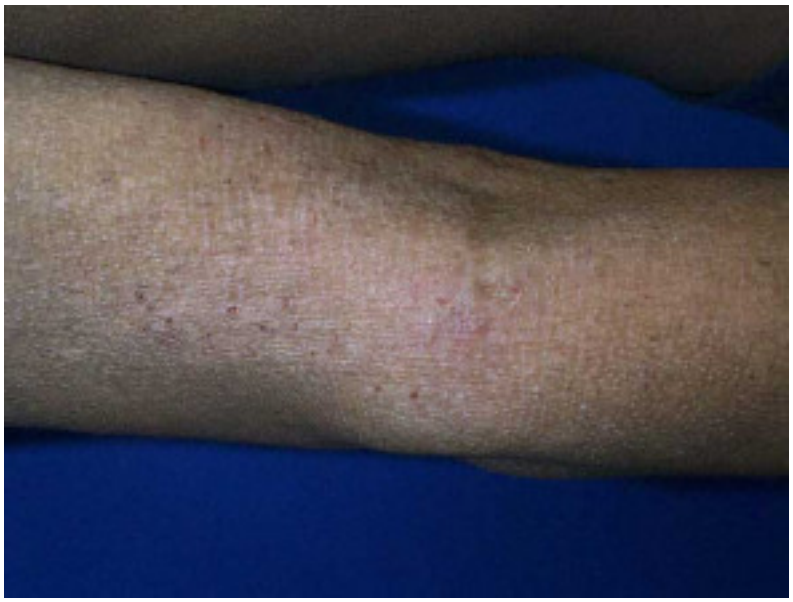
Dermatitis



Acute dermatitis



Atopic eczema



Chronic dermatitis

Atopic dermatitis

What is atopic dermatitis?

Atopic dermatitis is a chronic, itchy skin condition that is very common in children but may occur at any age. It is also known as eczema and atopic

eczema and was formerly known as Besnier prurigo. It is the most common form of dermatitis.

Atopic dermatitis usually occurs in people who have an 'atopic tendency'. This means they may develop any or all of three closely linked conditions; atopic dermatitis, asthma and hay fever (allergic rhinitis). Often these conditions run within families with a parent, child or sibling also affected. A family history of asthma, eczema or hay fever is particularly useful in diagnosing atopic dermatitis in infants.

Atopic dermatitis arises because of a complex interaction of genetic and environmental factors. These include defects in skin barrier function making the skin more susceptible to irritation by soap and other contact irritants, the weather, temperature and non-specific triggers: see Causes of atopic dermatitis.

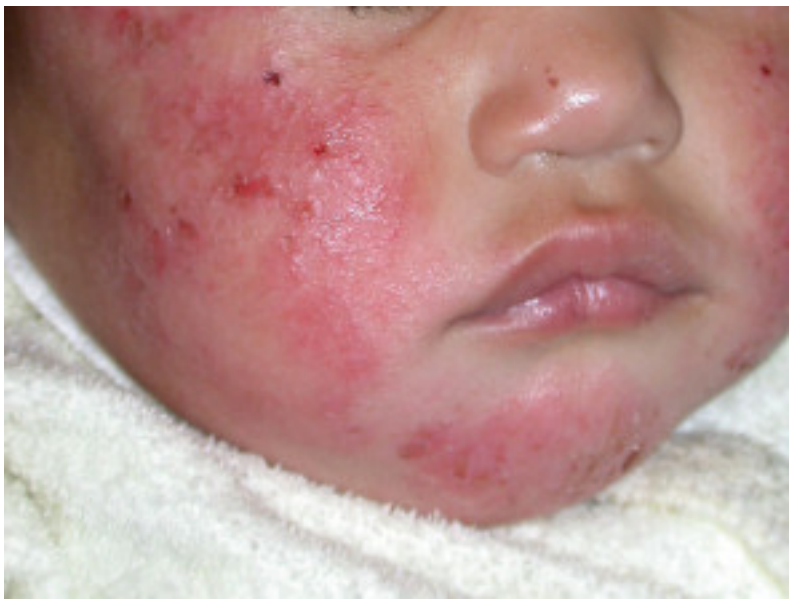
What does atopic dermatitis look like?

There is quite a variation in the appearance of atopic dermatitis between individuals. From time to time, most people have acute flares with inflamed, red, sometimes blistered and weepy patches. In between flares, the skin may appear normal or suffer from chronic eczema with dry, thickened and itchy areas.

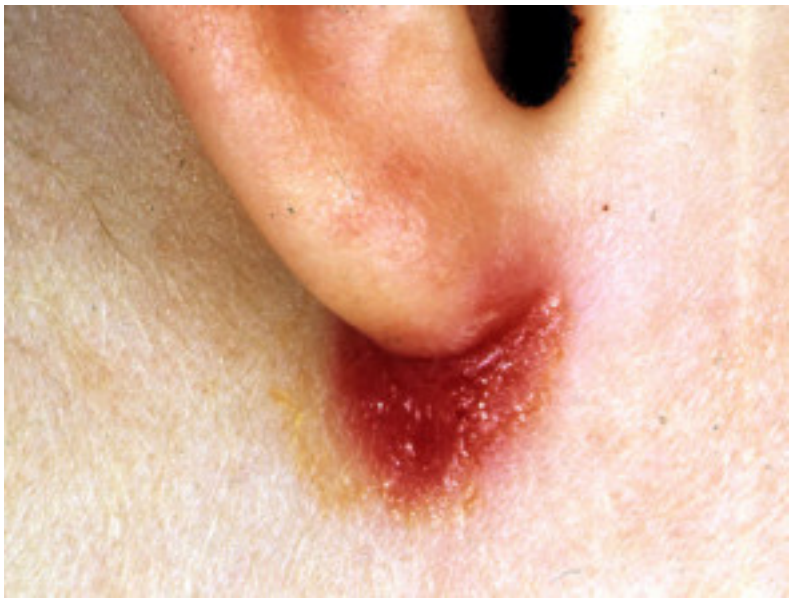
The presence of infection or an additional skin condition, the creams applied, the age of the person, their ethnic origin and other factors can alter the way eczema looks and feels.

There are however some general patterns to where the eczema is found on the body according to the age of the affected person.

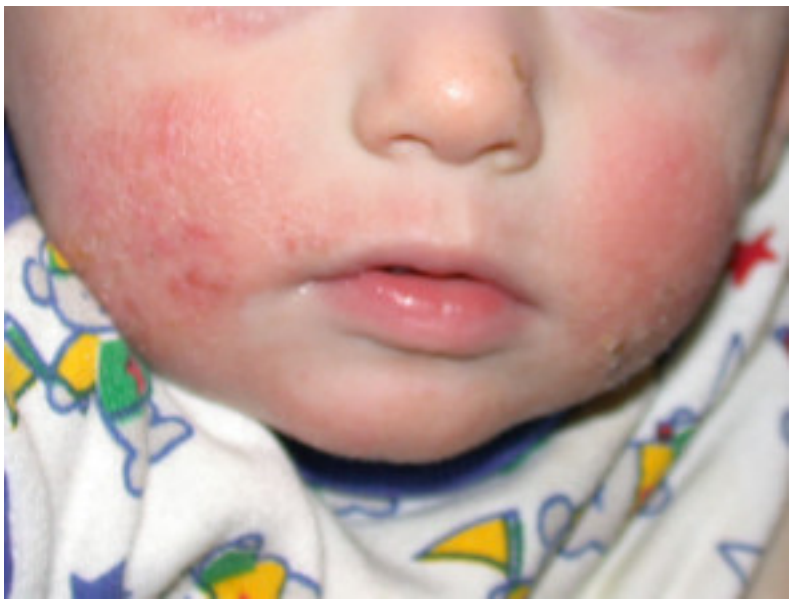
Infantile atopic dermatitis



Atopic eczema



Atopic eczema

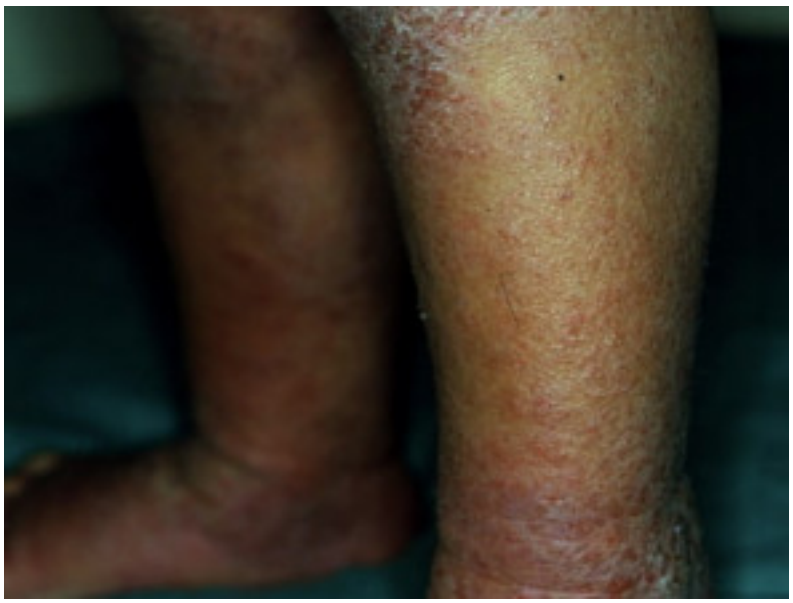


Atopic eczema

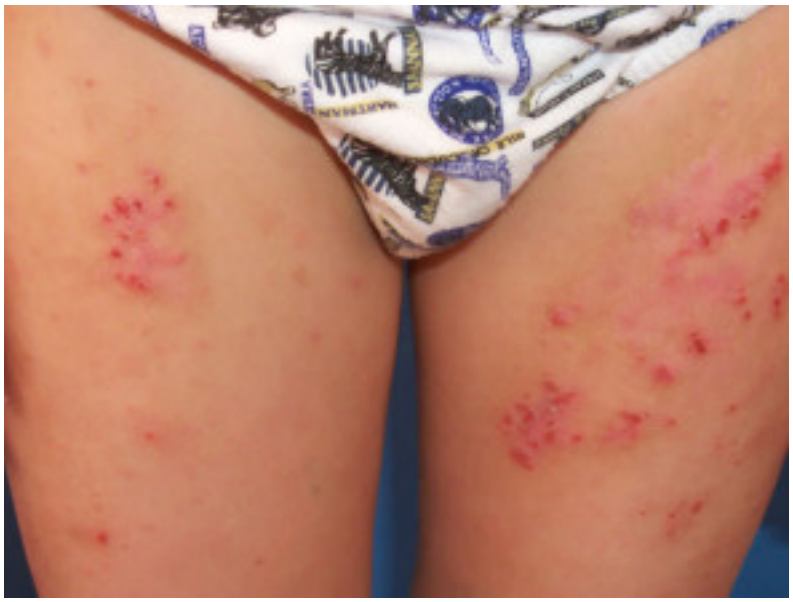
Infants

- Infants less than one year of age often have widely distributed eczema. The skin is often dry, scaly and red with small scratch marks made by sharp baby nails.
- The cheeks of infants are often the first place to be affected by eczema.
- The napkin area is frequently spared due to the moisture retention of nappies. Just like other babies, they can develop irritant napkin dermatitis, if wet or soiled nappies are left on too long.

Atopic dermatitis in pre-schoolers



Atopic eczema



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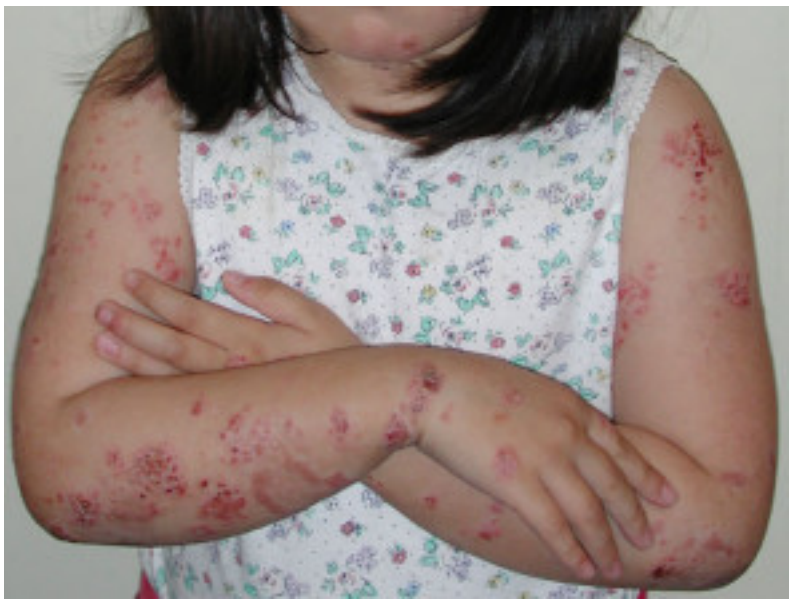
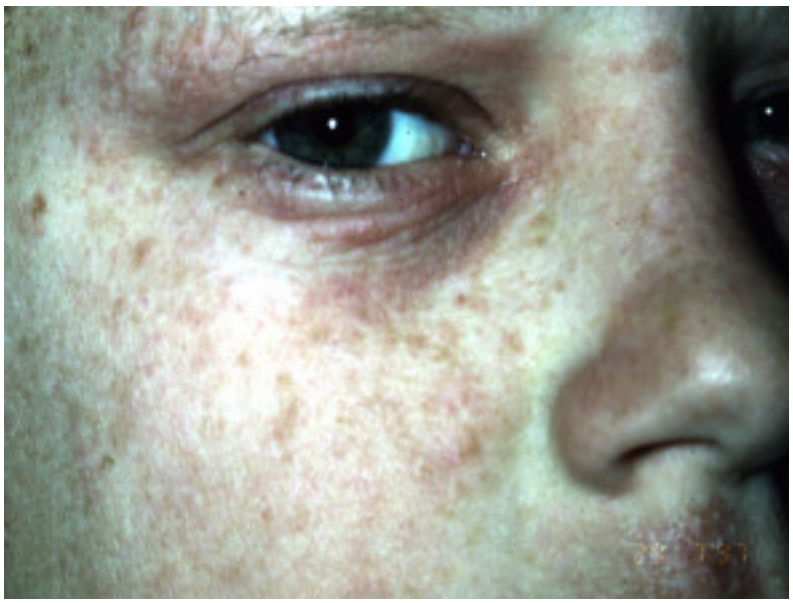


Atopic eczema

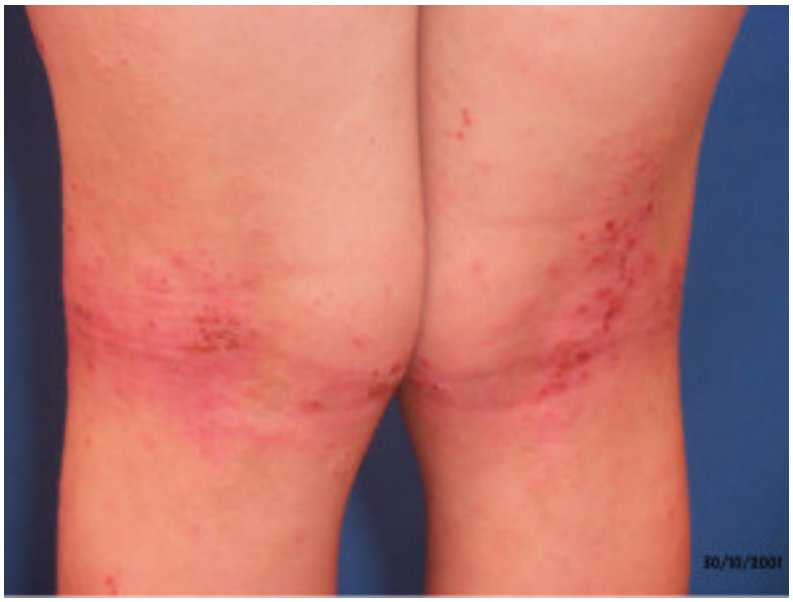
Toddlers and pre-schoolers

- As children begin to move around, eczema becomes more localised and thickened. Toddlers scratch vigorously and eczema may look very raw and uncomfortable.
- Eczema in this age group often affects the extensor (outer) aspects of joints, particularly the wrists, elbows, ankles and knees. It may also affect the genitals.
- As the child becomes older the pattern frequently changes to involve the flexor surfaces of the same joints (the creases) with less extensor involvement. The affected skin often becomes lichenified i.e. dry and thickened from constant scratching and rubbing,
- In some children, the extensor pattern of eczema persists into later childhood.

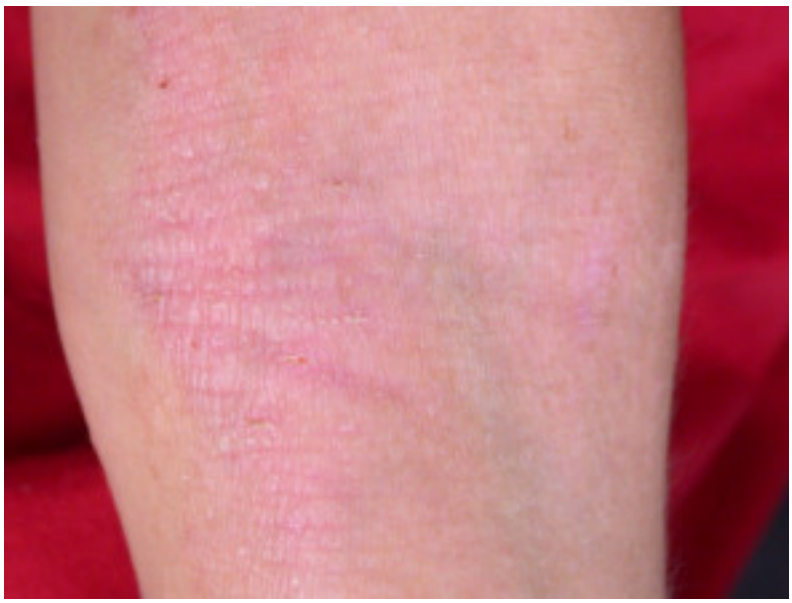
Atopic dermatitis in school-age children



Atopic eczema



Atopic eczema

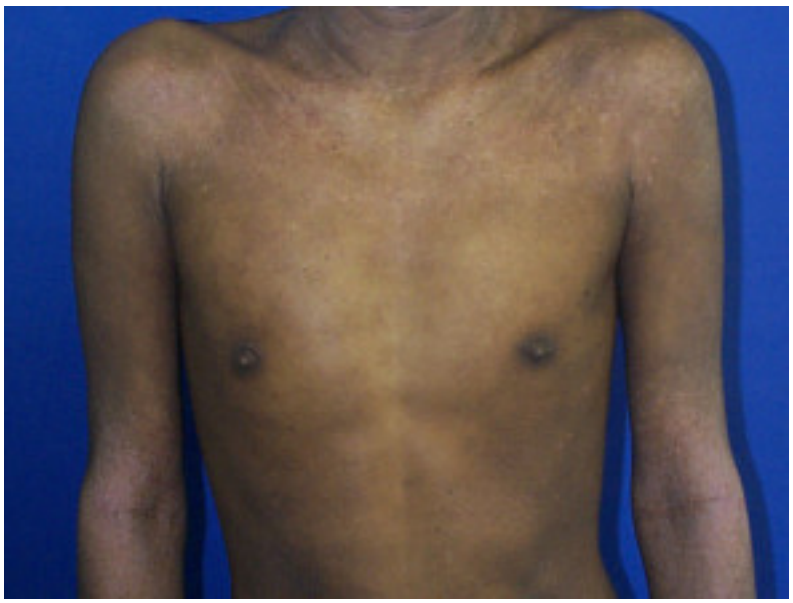


Atopic eczema

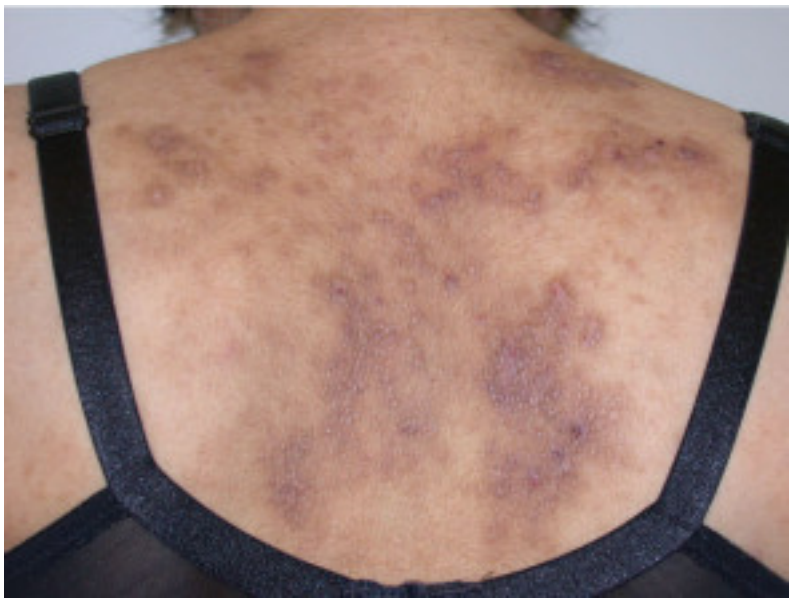
School-age children

- Older children tend to have the flexural pattern of eczema and it most often affects the elbow and knee creases. Other susceptible areas include the eyelids, earlobes, neck and scalp.
- They can develop recurrent acute itchy blisters on the palms, fingers and sometimes on the feet, known as pompholyx or vesicular hand/foot dermatitis.
- Many children develop a 'nummular' pattern of atopic dermatitis. This refers to small coin-like areas of eczema scattered over the body. These round patches of eczema are dry, red and itchy and may be mistaken for ringworm (a fungal infection).
- Mostly eczema improves during school years and it may completely clear up by the teens, although the barrier function of the skin is never entirely normal.

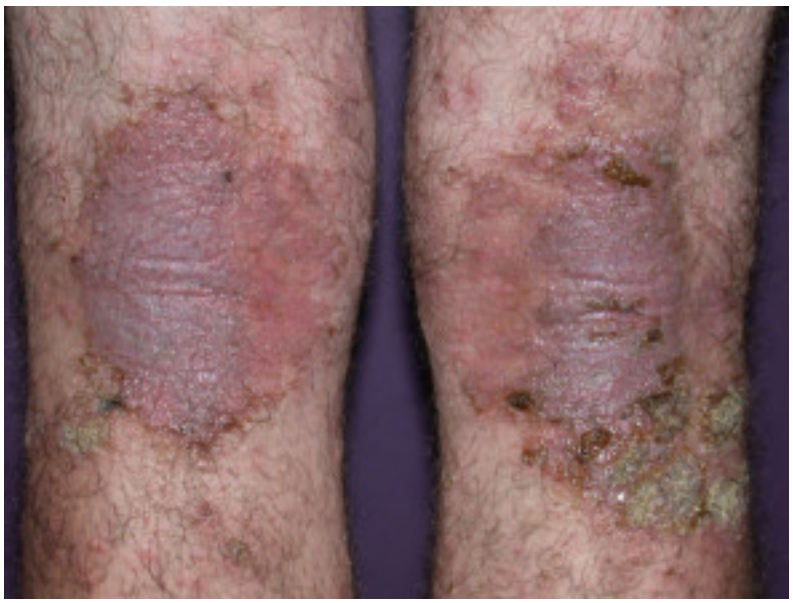
Atopic dermatitis in adults



Atopic eczema



Atopic eczema

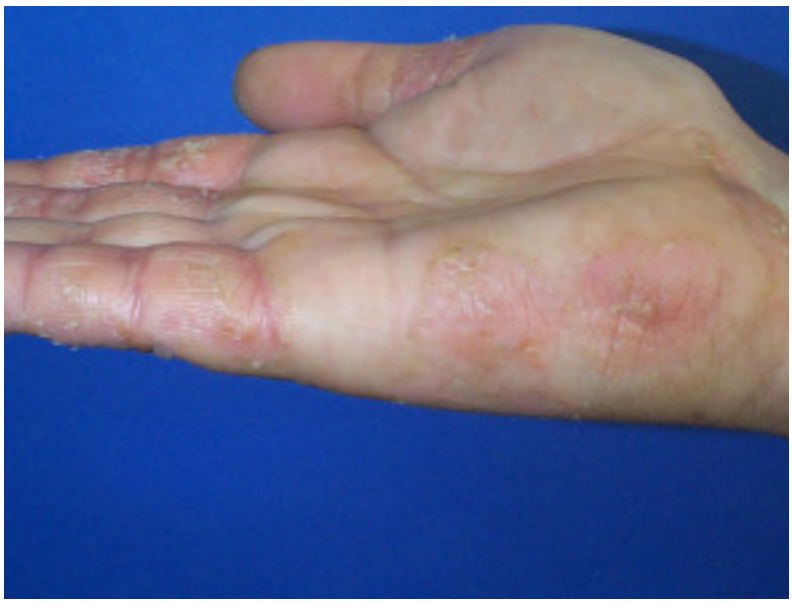


Infected dermatitis

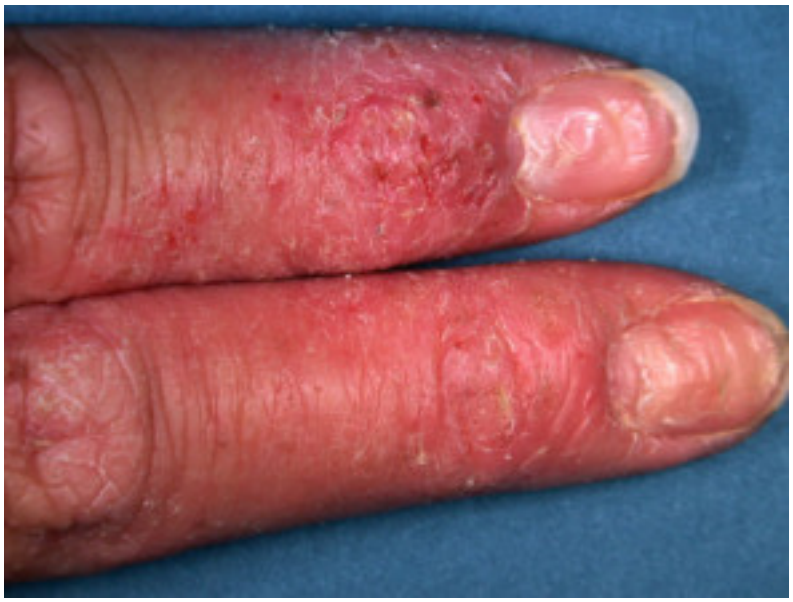
Adults

- Adults who have atopic dermatitis may present in various different ways.
- They may continue to have a diffuse pattern of eczema but the skin is often more dry and lichenified than in children.
- Commonly adults have persistent localised eczema, possibly confined to the hands, eyelids, flexures, nipples or all of these areas.
- Recurrent staphylococcal infections may be prominent.
- Atopic dermatitis is a major contributing factor to occupational irritant contact dermatitis. This most often affects hands that are frequently exposed to water, detergents and /or solvents.
- Having atopic dermatitis does not exclude contact allergic dermatitis (confirmed by patch tests) in children and adults)
- Hand dermatitis in adult atopics tends to be dry and thickened but may also be blistered.

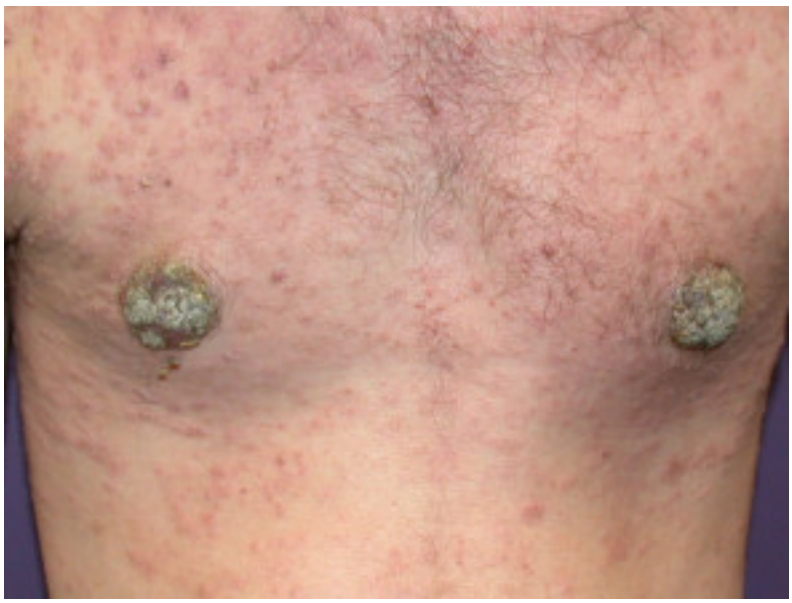
Persistent atopic dermatitis



Atopic eczema



Atopic eczema



Atopic eczema

Does atopic dermatitis persist forever?

Atopic dermatitis affects 15–20% of children but is much less common in adults. It is impossible to predict whether eczema will improve by itself or not in an individual. Sensitive skin persists life-long. A meta-analysis including over 110,000 subjects found that 20% of children with atopic dermatitis still had persistent disease 8 years later. Fewer than 5% had persistent disease 20 years later. Children who developed AD before the age of 2 had a much lower risk of persistent disease than those who developed AD later in childhood or during adolescence.

It is unusual for an infant to be affected with atopic dermatitis before the age of four months but they may suffer from infantile seborrhoeic dermatitis or other rashes prior to this. The onset of atopic dermatitis is usually before two years of age although it can manifest itself in older people for the first time. Atopic dermatitis is often worst between the ages of two and four but it generally improves after this and may clear altogether by the teens. Certain occupations such as farming, hairdressing, domestic and industrial cleaning, domestic duties and care-giving expose the skin to various irritants and, sometimes, allergens. This aggravates atopic dermatitis. It is wise to bear this in mind when considering career options — it is usually easier to choose a more suitable occupation from the outset than to change it later.

What is the treatment for atopic dermatitis?

Treatment of atopic dermatitis may be required for many months and possibly years.

It nearly always requires:

- Reduction of exposure to trigger factors (where possible)
- Regular emollients (moisturisers)
- Intermittent topical steroids

In some cases, management may also include one or more of the following:

- Topical calcineurin inhibitors, such as pimecrolimus cream or tacrolimus ointment
- Crisabarole ointment
- Antibiotics
- Antihistamines
- Phototherapy
- Oral corticosteroids
-

Longstanding and severe eczema may be treated with an immunosuppressive agent.

- Methotrexate
- Ciclosporin
- Azathioprine
-

New biologics are under investigation. The first to be approved for the treatment of atopic dermatitis is:

- Dupilumab

What is irritant contact dermatitis?

Irritant contact dermatitis is a form of contact dermatitis, in which the skin is injured by friction, environmental factors such as cold, over-exposure to water, or chemicals such as acids, alkalis, detergents and solvents.

Why does irritant contact dermatitis occur?

Irritant contact dermatitis occurs when chemicals or physical agents damage the surface of the skin faster than the skin can repair the damage. Irritants remove oils and moisture (natural moisturising factor) from its outer layer, allowing chemical irritants to penetrate more deeply and cause further damage by triggering inflammation.

The severity of the dermatitis is highly variable and depends on many factors including:

- Amount and strength of the irritant
- Length and frequency of exposure (eg, short heavy exposure or repeated/prolonged low exposure)
- Skin susceptibility (eg, thick, thin, oily, dry, very fair, previously damaged skin or pre-existing atopic tendency)
- Environmental factors (eg, high or low temperature or humidity)

What are the most common irritants?

Irritants include such everyday things as water, detergents, solvents, acids, alkalis, adhesives, metalworking fluids and friction. Often several of these act together to injure the skin.

Who gets irritant contact dermatitis?

Irritant contact dermatitis may affect anyone, given sufficient exposure to irritants, but those with atopic dermatitis are particularly susceptible. 80% of cases of occupational hand dermatitis are due to irritants, most often affecting cleaners, hairdressers and food handlers.

What is the differential diagnosis of irritant contact dermatitis?

Irritant contact dermatitis can appear similar to other forms of dermatitis, notably:

- All kinds of hand dermatitis
- Allergic contact dermatitis — this is caused by an immune response following skin contact with an allergenic substance. Small quantities may be sufficient to cause allergy, whereas a certain

minimum exposure is necessary for irritant contact dermatitis. Irritant and allergic contact dermatitis may coexist.

- Pompholyx eczema (dyshidrotic eczema) — in which there are itchy clusters of blisters along the sides of the fingers and on the palms, often triggered by sweating.

What are the clinical features of contact irritant dermatitis?

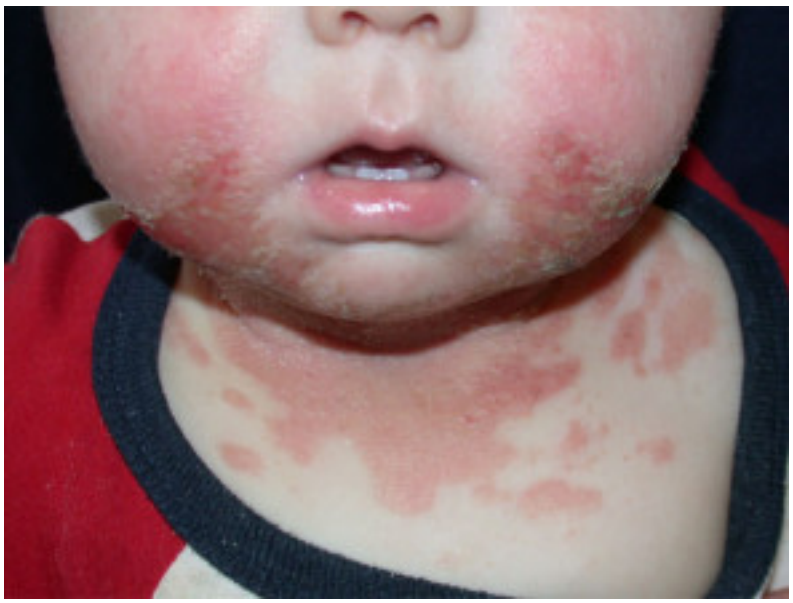
Irritant contact dermatitis is usually confined to the site of contact with the irritant, at least at first. If the dermatitis is prolonged or severe, it may spread later to previously unaffected areas, but it is less likely to do this than allergic contact dermatitis.

Dermatitis often appears as a well-demarcated red patch with a glazed surface, but there may be swelling, blistering and scaling of the damaged area, indistinguishable from other types of dermatitis. It can be very itchy.

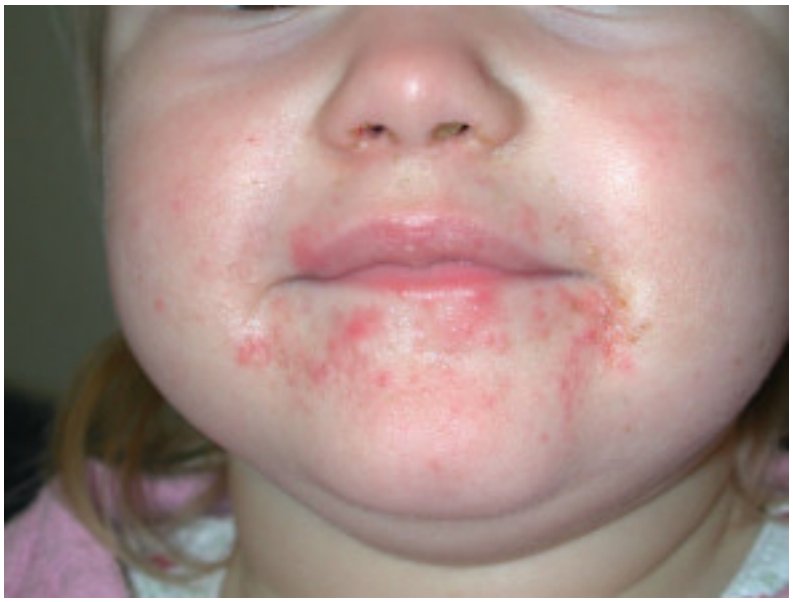
Contact irritant dermatitis can appear differently according to the conditions of exposure.

- Accidental exposure to a strong irritant such as a strong acid or alkali substance may cause an immediate skin reaction resulting in pain, swelling and blistering.
- Contact with mild irritants such as water and soap or detergent may over weeks cause dryness, itching and cracking of the skin. Eventually, sores may appear which form crusts and scales.

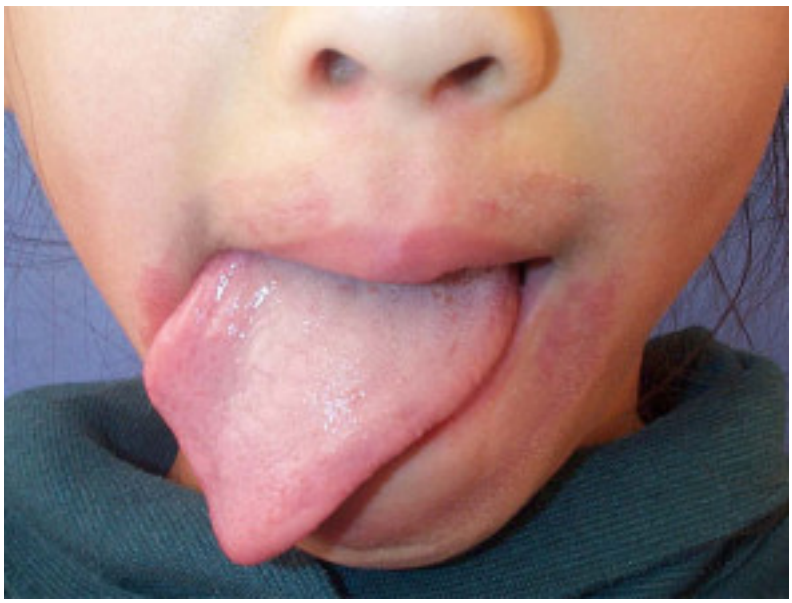
Contact irritant dermatitis due to saliva



Irritant dermatitis



Irritant dermatitis



Irritant dermatitis

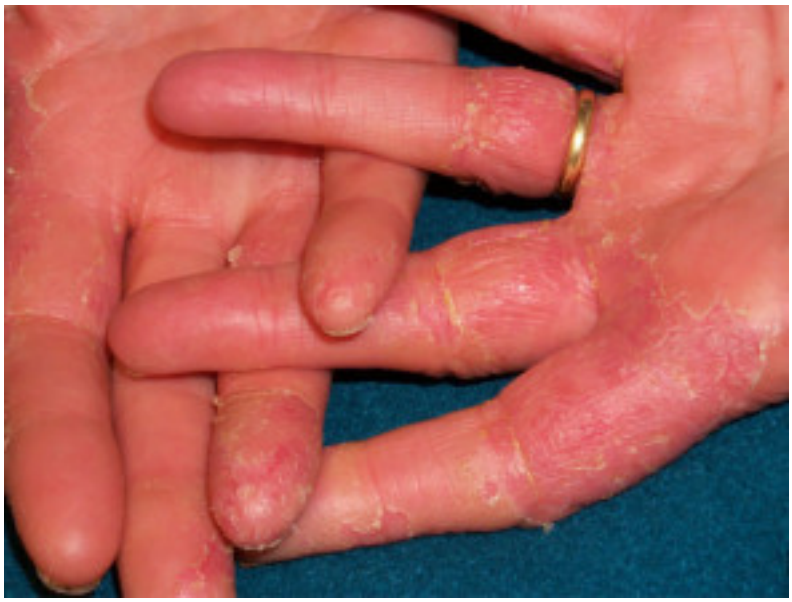
Some typical examples of irritant contact dermatitis include:

- Dribble rash around the mouth or on the chin in a baby, or older children due to lip-licking; the cause is saliva, which is alkaline. Skin bacteria may contribute to the clinical appearance.
- Napkin dermatitis due to urine and faeces can affect older incontinent patients as well as babies (incontinence-associated dermatitis).
- Chemical burns from strong acids (eg, hydrochloric acid) and particularly alkalis (eg, sodium or calcium hydroxide).
- Housewife's eczema is hand dermatitis caused by excessive exposure to water, soaps, detergents, bleaches and polishes.
- Dermatitis on a finger underneath a ring. Soaps, shampoos, detergents and hand creams may accumulate under the ring and cause irritant contact dermatitis.
- Gloves or glove powder or sweat or tiny quantities of chemicals that have been occluded inside the gloves may have a direct irritant action on hands (rubber may also result in latex or rubber antioxidant allergy).
- Fibreglass may cause direct mechanical/frictional damage.
- Dry cold air may cause dry, irritable skin (winter itch).
- Reactions to contact with beetles, for example, paedarus, lax beetles, and caterpillars.
- Cosmetics may irritate sensitive facial skin (especially in rosacea) resulting in immediate stinging, burning and redness followed by itching

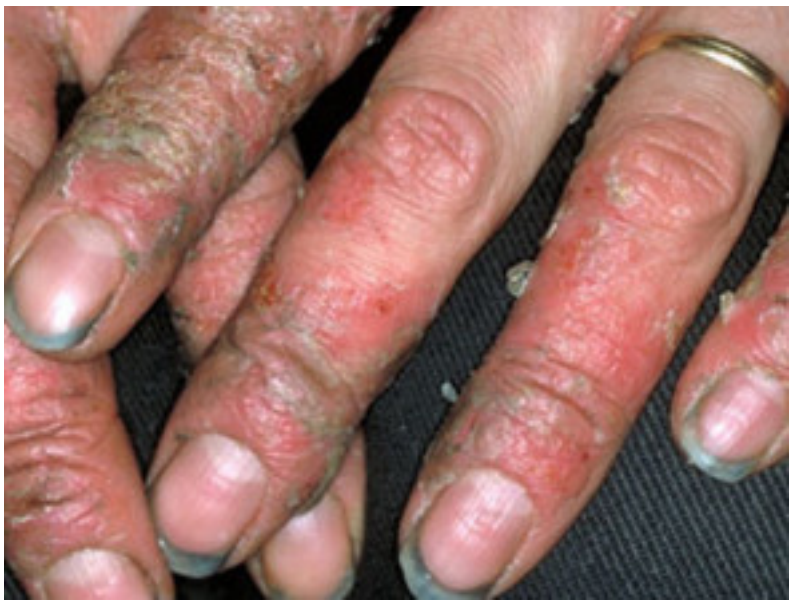
and dryness. Gels and solutions tend to be more irritating than creams and ointments.

In time, the skin may develop some tolerance to mild irritants.

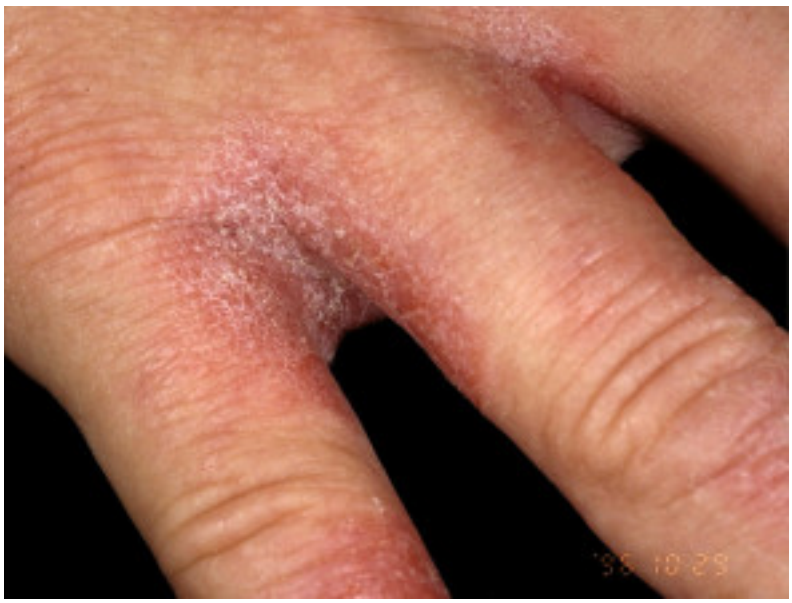
Irritant hand dermatitis



Irritant dermatitis



Irritant dermatitis



Irritant dermatitis

Testing for irritant contact dermatitis

Sometimes it is easy to recognise irritant contact dermatitis, and no specific tests are necessary. The rash usually heals once the irritant is removed and, if necessary, special treatment is applied. While some tests can indicate the irritant potential of substances, there are no specific tests that can reliably show what the effect of an irritant will be in each case. Irritant dermatitis is usually the result of the cumulative impact of multiple irritants.

Patch tests

Patch tests are used to confirm allergic contact dermatitis and identify the allergen(s). They do not exclude irritant contact dermatitis as the two may coexist.

What is the treatment of contact irritant dermatitis?

It is essential to recognise how you are in contact with the responsible substance(s) so that, where possible, you can avoid it (them) or at least reduce exposure. Wear suitable gloves to protect against irritants in your home and work environment.

Irritant contact dermatitis is usually treated with the following:

- Chemical burns are usually flushed with water followed by the use of antidote or specific remedy against the particular toxic chemical.
- Compresses, creams and ointments may assist healing
  - Emollient creams
  - Topical steroids
  - Antibiotics for secondary infection (usually flucloxacillin or erythromycin).

Allergic contact dermatitis

What is allergic contact dermatitis?

Allergic contact dermatitis is a form of dermatitis/eczema caused by an allergic reaction to a material, called an allergen, in contact with the skin. The allergen is harmless to people that are not allergic to it. Allergic contact dermatitis is also called contact allergy.

Who gets allergic contact dermatitis?

Allergic contact dermatitis is common in the general population and in specific employment groups.

- It is more common in women than men, mainly due to nickel allergy and, recently, to acrylate allergy associated with nail cosmetics.
- Many young children are also allergic to nickel.
- Contact allergy to topical antibiotics is common in patients over the age of 70 years old.
- Allergic contact dermatitis is especially common in metal workers, hairdressers, beauticians, health care workers, cleaners, painters and florists.

What causes allergic contact dermatitis?

Allergic contact dermatitis is a type 4 or delayed hypersensitivity reaction and occurs 48–72 hours after exposure to the allergen. The mechanism involves

CD4+ T-lymphocytes, which recognise an antigen on the skin surface, releasing cytokines that activate the immune system and cause the dermatitis.

Note:

- Contact allergy occurs predominantly from an allergen on the skin rather than from internal sources or food.
- Only a small number of people react to the specific allergen, which is harmless to those who are not allergic to it.
- They may have been in contact with the allergen for years without it causing dermatitis.
- Contact with tiny quantities of an allergen can induce dermatitis.
- Patients with impaired barrier function of the skin are more prone to allergic contact dermatitis, eg patients with leg ulcers, perianal dermatitis, or chronic irritant contact dermatitis.
- Patients with atopic dermatitis associated with defective filaggrin (a structural protein in the stratum corneum) have a high risk of also developing allergic contact dermatitis.
-

What are the clinical features of allergic contact dermatitis?

Allergic contact dermatitis arises some hours after contact with the responsible material. It settles down over some days providing the skin is no longer in contact with the allergen.

Allergic contact dermatitis is generally confined to the site of contact with the allergen, but it may extend outside the contact area or become generalised.

- Transmission from the fingers can lead to dermatitis on the eyelids and genitals.
- Dermatitis is unlikely to be due to a specific allergen if the area of skin most in contact with that allergen is unaffected.
- The affected skin may be red and itchy, swollen and blistered, or dry and bumpy.

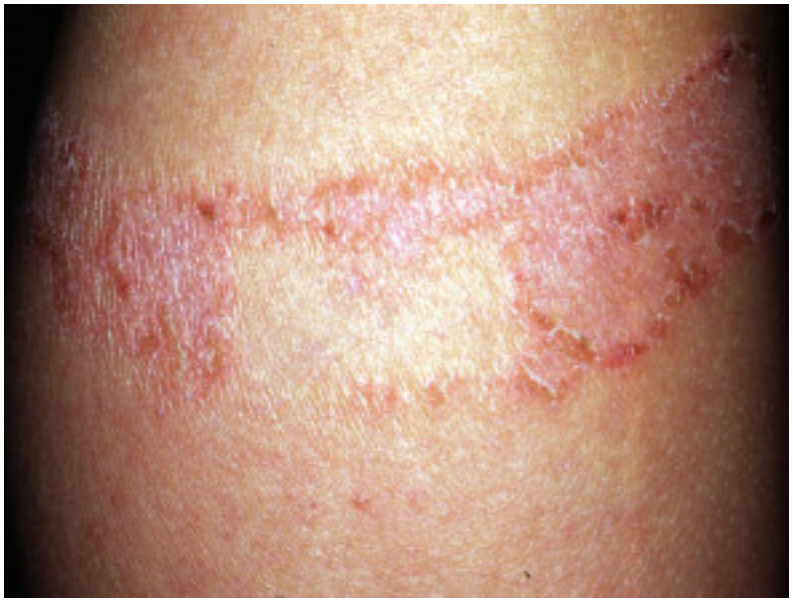
Some typical examples of allergic contact dermatitis include:

- Eczema in the skin in contact with jewellery items, due to contact allergy to nickel
- Reactions to fragrances in perfumes and household items
- Eczema under adhesive plaster, due to contact allergy to rosin

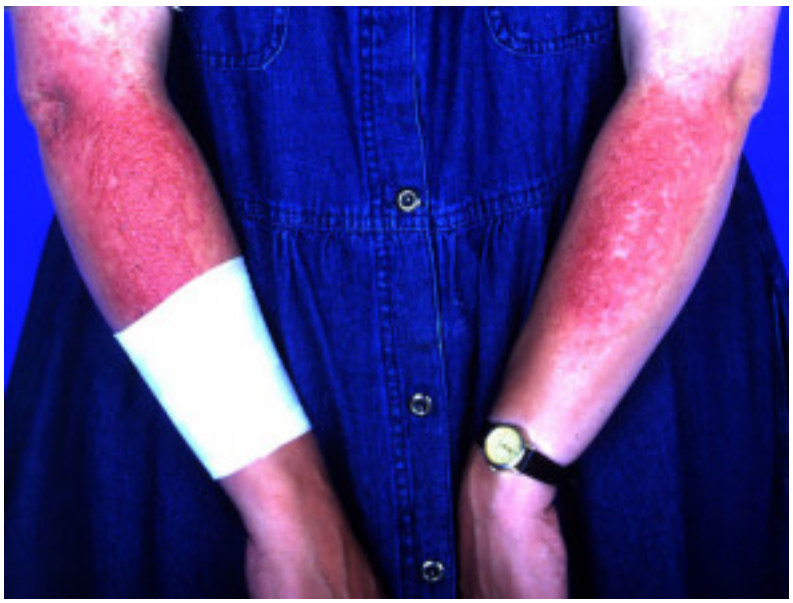
- Swelling and blistering of face and neck in reaction to permanent hair dye, due to allergy to paraphenylenediamine
- Hand dermatitis caused by rubber accelerator chemicals used in the manufacture of rubber gloves
- Itchy red face due to contact with methylisothiazolinone, a preservative in wash-off hair products and baby wipes
- Fingertip dermatitis due to acrylates used in hair extensions and nail cosmetics.
- Reactions after dental implants containing acrylates
- Localised blistering at the site of topical medications such as antibiotics
- Swelling and blistering on exposed sites (eg face and hands) due to contact with plants such as poison ivy or, in New Zealand, the Japanese wax tree Toxicodendron succedaneum

There is a very long list of materials that have caused contact allergy in a small number of individuals.

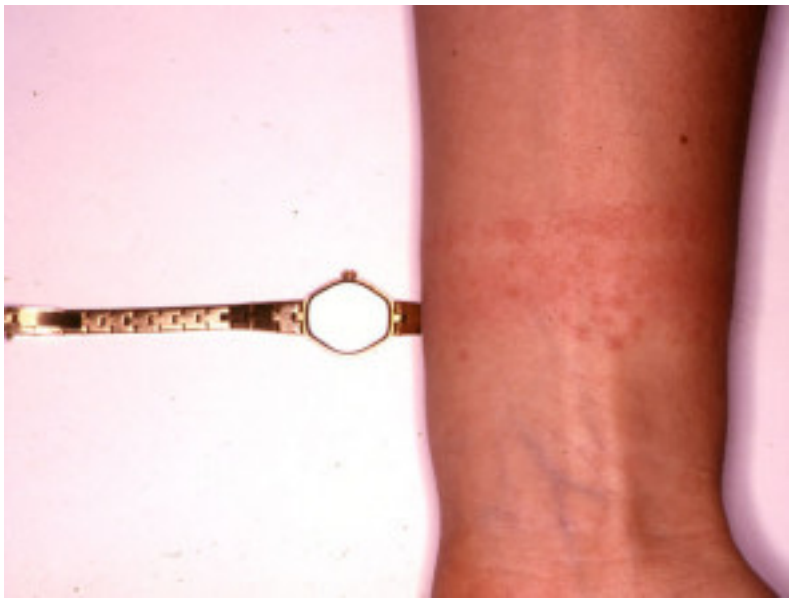
Allergic contact dermatitis



Adhesive plaster reaction



Sunscreen reaction



Watch strap reaction

What is the differential diagnosis of allergic contact dermatitis?

Allergic contact dermatitis should be distinguished from:

- Irritant contact dermatitis, which is due to irritation or repetitive injury to the skin. Irritants include water, soaps, detergents, solvents, acids, alkalis, and friction. Irritant contact dermatitis may affect anyone,

providing they have had enough exposure to the irritant, but those with atopic dermatitis are particularly sensitive. Most cases of hand dermatitis are due to contact with irritants. Irritant contact dermatitis can occur immediately after a single injury or develop slowly after repeated exposure to an irritant.

- Other forms of dermatitis, which may mimic allergic contact dermatitis.
- Contact urticaria, in which a rash appears within minutes of exposure and fades away within minutes to hours. The allergic reaction to latex is the best-known example of allergic contact urticaria.
- Fungal infections; tinea corporis may present as a unilateral rash.

What are the complications of allergic contact dermatitis?

Allergic contact dermatitis starts as a localised reaction to an allergen in contact with the skin, but severe reactions may generalise due to autoeczematisation and can lead to erythroderma.
Ingestion of a contact allergen may rarely lead to baboon syndrome or generalised systemic contact dermatitis.

Photoallergy

Sometimes contact allergy arises only after the skin has been exposed to ultraviolet light. The rash is confined to sun-exposed areas even though the allergen may have been in contact with covered areas. This is called photocontact dermatitis.

Examples of photoallergy include:

- Dermatitis due to a sunscreen chemical, affecting the top but not the under the surface of the arm
- Dermatitis of face, neck, arms and hands due to antibacterial soap.
-

How is allergic contact dermatitis diagnosed?

Sometimes it is easy to recognise contact allergy and no specific tests are necessary. Taking a very good history including information on the work environment, hobbies, products in use at home and work and sun exposure will enhance the chances of finding a diagnosis. The rash usually (but not always) completely clears up if the allergen is no longer in contact with the skin, but recurs even with slight contact with it again.

The open application test is used to confirm contact allergy to a cosmetic, such as a moisturiser. The product under suspicion is applied several times daily for several days to a small area of sensitive skin. The inner aspect of the upper arm is suitable. Contact allergy is likely if dermatitis arises in the treated area.

Dermatologists will perform patch tests in patients with suspected contact allergy, particularly if the reaction is severe, recurrent or chronic. The tests can identify the specific allergen causing the rash.
Fungal scrapings of skin for microscopy and culture can exclude fungal infection.

Dimethylgloxime test is available to 'spot test' if a product contains nickel.

What is the treatment for allergic contact dermatitis?

It is important to recognise how you are in contact with the responsible substance so that, where possible, you can avoid it.

- Find out precisely what you are allergic to by having comprehensive patch tests.
- Identify where the allergen is found, thus read labels of all products before use.
- Carefully study your environment to locate the allergen. Note: many chemicals have several names, and cross-reactions to similar chemicals with different names are common.
- Wear appropriate gloves to protect hands from touching materials to which you react and remove gloves in the appropriate way. Some chemicals will penetrate certain gloves; seek a safety expert's advice.
- Ask your dermatologist to help.

Active dermatitis is usually treated with the following:

- Emollient creams
- Topical steroids

- Topical or oral antibiotics for secondary infection
- Oral steroids, usually short courses, for severe cases
- Phototherapy or photochemotherapy.
- Azathioprine, ciclosporin or another immunosuppressive agent.
- Tacrolimus ointment and pimecrolimus cream are immune-modulating calcineurin inhibitors and may prove helpful for allergic contact dermatitis.
-

What is the outcome for allergic contact dermatitis?

Contact allergy often persists lifelong so it is essential to identify the allergen and avoid touching it. Dermatitis may recur on re-exposure to the allergen.

- Some allergens are more difficult to avoid than others, with airborne allergens being a particular problem (eg epoxy resin, compositae pollen).
- The longer a person suffers from severe allergic contact dermatitis, the longer it will take to clear after the diagnosis is made and the cause detected.
- Dermatitis may clear up on avoidance of contact with the allergen, but sometimes it persists indefinitely, for example, chromate allergy.

Prognosis depends on patient education and compliance in avoiding allergens and appropriate skin care.

Dry skin

What is dry skin?

Dry skin refers to skin that feels dry to touch. Dry skin is lacking moisture in the outer horny cell layer (stratum corneum) and this results in cracks in the skin surface.

Dry skin is also called xerosis, xeroderma or asteatosis (lack of fat).

Who gets dry skin?

Dry skin can affect males and females of all ages. There is some racial variability in water and lipid content of the skin.

- Dry skin that starts in early childhood may be one of about 20 types of ichthyosis (fish-scale skin). There is often a family history of dry skin.
- Dry skin is commonly seen in people with atopic dermatitis.
- Nearly everyone > 60 years has dry skin.

Dry skin that begins later may be seen in people with certain diseases and conditions.

- Postmenopausal females
- Hypothyroidism
- Chronic renal disease
- Malnutrition and weight loss
- Subclinical dermatitis
- Treatment with certain drugs such as oral retinoids, diuretics and epidermal growth factor receptor inhibitors

People exposed to a dry environment may experience dry skin.

- Low humidity: in desert climates or cool, windy conditions
- Excessive air conditioning
- Direct heat from a fire or fan heater
- Excessive bathing
- Contact with soap, detergents and solvents
- Inappropriate topical agents such as alcohol
- Frictional irritation from rough clothing or abrasives

What causes dry skin?

Dry skin is due to abnormalities in the integrity of the barrier function of the stratum corneum, which is made up of corneocytes.

- There is an overall reduction in the lipids in the stratum corneum.
- The ratio of ceramides, cholesterol and free fatty acids may be normal or altered.
- There may be a reduction in the proliferation of keratinocytes.
- Keratinocyte subtypes change in dry skin with a decrease in keratins K1, K10 and increase in K5, K14.

- Involucrin (a protein) may be expressed early, increasing cell stiffness.
- The result is the retention of corneocytes and reduced water-holding capacity.

The inherited forms of ichthyosis are due to loss of function mutations in various genes (listed in parentheses below).

- Ichthyosis vulgaris (FLG).
- Recessive X-linked ichthyosis (STS)
- Autosomal recessive congenital ichthyosis (ABCA12, TGM1, ALOXE3)
- Keratinopathic ichthyoses (KRT1, KRT10, KRT2)

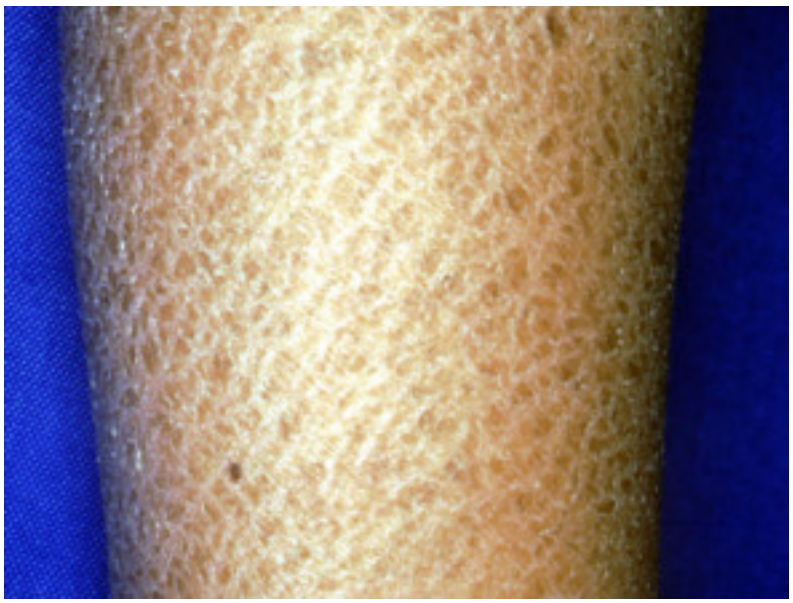
Acquired ichthyosis may be due to:

- Metabolic factors: thyroid deficiency
- Illness: lymphoma, internal malignancy, sarcoidosis, HIV infection
- Drugs: nicotinic acid, kava, protein kinase inhibitors (eg EGFR inhibitors), hydroxyurea.

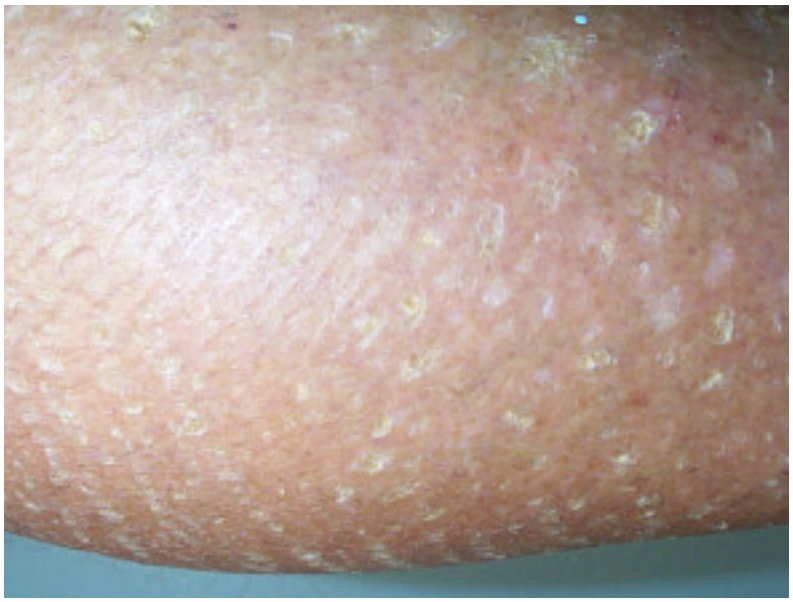
What are the clinical features of dry skin?

Dry skin has a dull surface with a rough, scaly quality. The skin is less pliable and cracked. When dryness is severe, the skin may become inflamed and fissured.
Although any site can be dry, dry skin tends to affect the shins more than any other site.
The clinical features of ichthyosis depend on the specific type of ichthyosis.

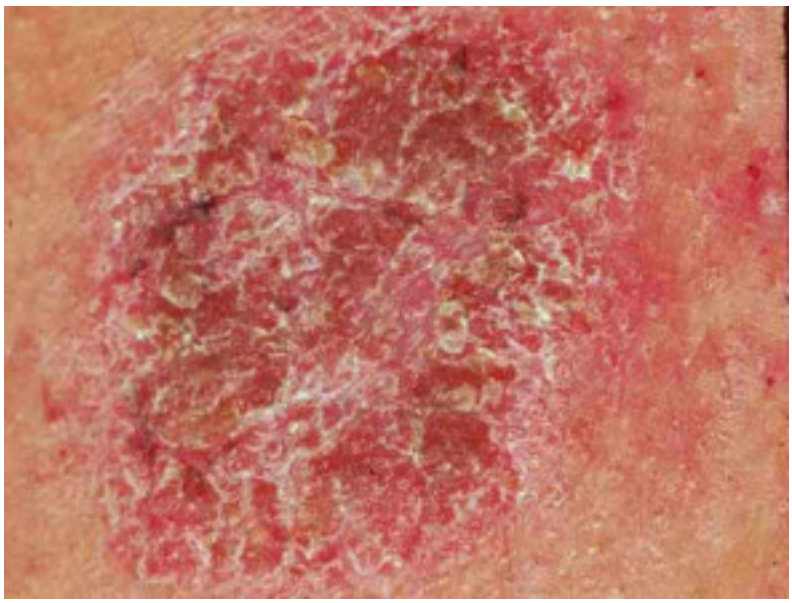
Dry skin



Ichthyosis



Close-up of ichthyosis



Dermatitis from dry skin

Complications of dry skin

Dry areas of skin may become itchy, indicating a form of eczema/dermatitis has developed.

- Atopic eczema — especially in people with ichthyosis vulgaris
- Eczema craquelé — especially in older people. Also called asteatotic eczema
- A dry form of nummular dermatitis/discoid eczema — especially in people that wash their skin excessively

When the dry skin of an older person is itchy without a visible rash, it is sometimes called winter itch, 7th age itch, senile pruritus or chronic pruritus of the elderly.

Other complications of dry skin may include:

- Skin infection when bacteria or viruses penetrate a break in the skin surface
- Overheating, especially in some forms of ichthyosis
- Food allergy, eg, to peanuts, has been associated with filaggrin mutations
- Contact allergy, eg, to nickel, has also been correlated with barrier function defects.

How is the type of dry skin diagnosed?

The type of dry skin is diagnosed by careful history and examination.

In children:

- Family history
- Age of onset
- Appearance at birth, if known
- Distribution of dry skin
- Other features, eg eczema, abnormal nails, hair, dentition, sight, hearing.

In adults:

- Medical history
- Medications and topical preparations
- Bathing frequency and use of soap
- Evaluation of environmental factors that may contribute to dry skin.

Sometimes, a skin biopsy may be requested. There may be additional tests requested to diagnose some types of ichthyosis.

What is the treatment for dry skin?

The mainstay of treatment of dry skin and ichthyosis is moisturisers/ emollients. They should be applied liberally and often enough to:

- Reduce itch
- Improve the barrier function
- Prevent entry of irritants, bacteria
- Reduce transepidermal water loss.

When considering which emollient is most suitable, consider:

- Severity of the dryness
- Tolerance
- Personal preference
- Cost and availability.
-

Emollients generally work best if applied to damp skin, if pH is below 7 (acidic), and if containing humectants such as urea or propylene glycol.

Additional treatments include:

- Topical steroid if itchy or there is dermatitis — choose an emollient base
- Topical calcineurin inhibitors if topical steroids are unsuitable.

How can dry skin be prevented?

Eliminate aggravating factors.

- Reduce the frequency of bathing.
- A humidifier in winter and air conditioner in summer
- Compare having a short shower with a prolonged soak in a bath.
- Use lukewarm, not hot, water.
- Replace standard soap with a substitute such as a synthetic detergent cleanser, water-miscible emollient, bath oil, anti-pruritic tar oil, colloidal oatmeal etc.
- Apply an emollient liberally and often, particularly shortly after bathing, and when itchy. The drier the skin, the thicker this should be, especially on the hands.

What is the outlook for dry skin?

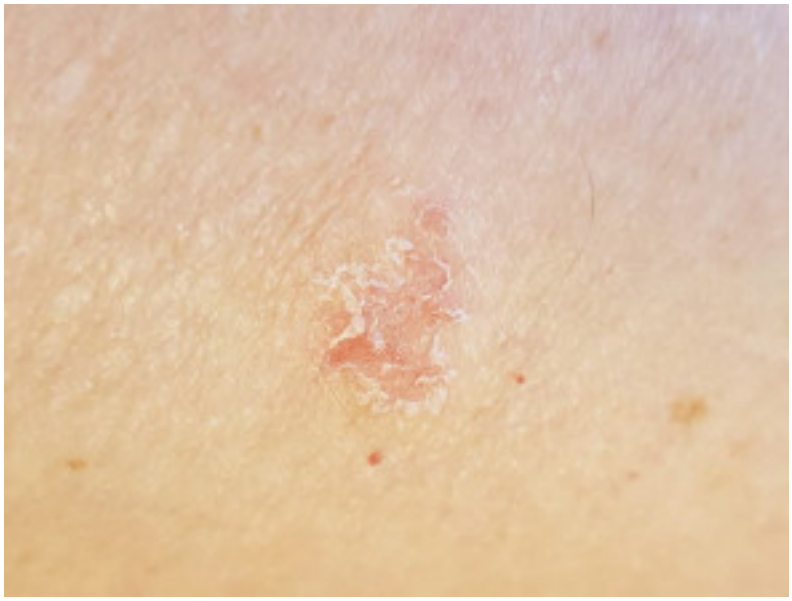
A tendency to dry skin may persist life-long, or it may improve once contributing factors are controlled.

Discoid eczema

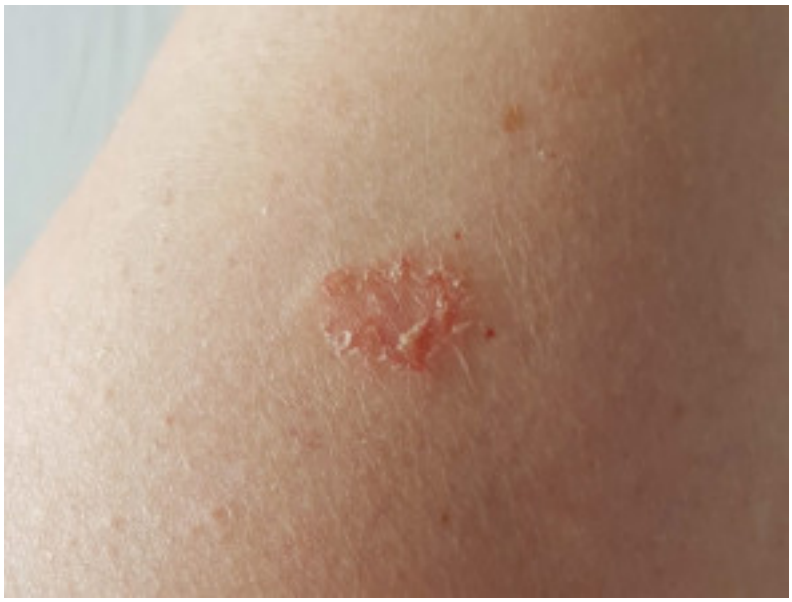
What is discoid eczema?

Discoid eczema is a common type of eczema/dermatitis defined by scattered, well-defined, coin-shaped and coin-sized plaques of eczema. Discoid eczema is also called nummular dermatitis.

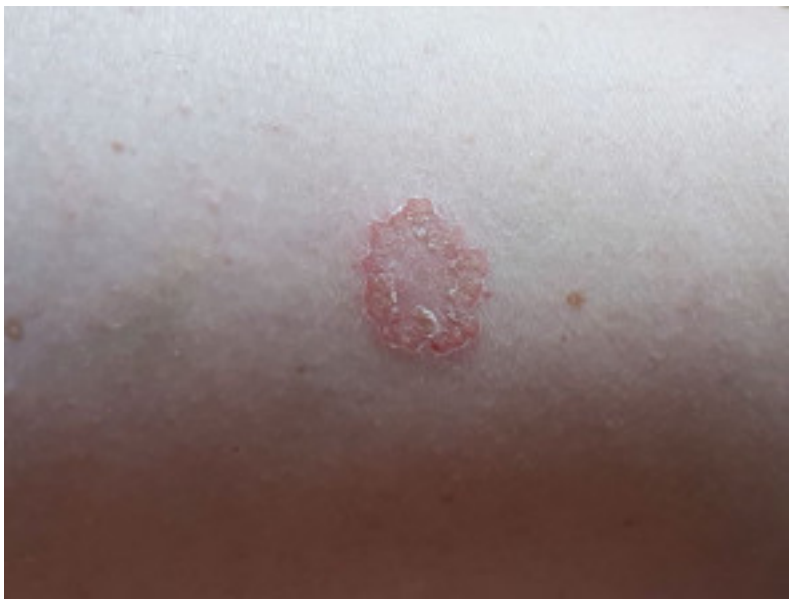
The progression of a dry discoid eczema plaque



Discoid eczema day 2



Discoid eczema day 6



Discoid eczema day 14

What is the cause of discoid eczema?

The cause of discoid eczema is unknown. Some cases are associated with Staphylococcus aureus infection.

The eruption can be precipitated by:

- A localised injury such as scratch, insect bite or thermal burn
- Impetigo or wound infection
- Contact dermatitis
- Dry skin
- Varicose veins (varicose eczema)

Who gets discoid eczema?

Discoid eczema can affect all age groups. It is slightly more common in older adult males and younger adult females. In males there is an association with chronic alcoholism. Drug-induced discoid eczema can be due to medications that cause skin dryness.

Discoid eczema can occur in association with atopic eczema, eczema craquelé, and secondary eczematisation.

What are the clinical features of discoid eczema?

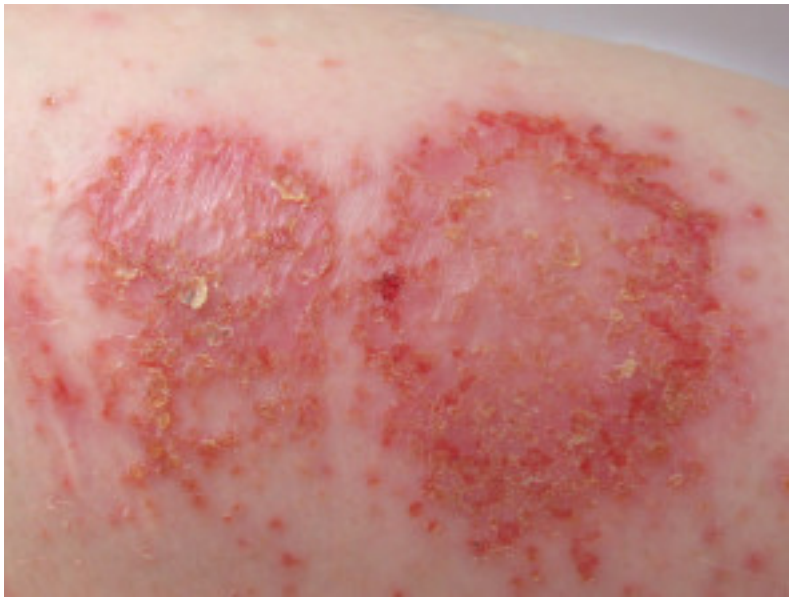
Discoid eczema usually affects the limbs, particularly the legs, but the rash may be widespread. Although often bilateral, the distribution can be asymmetrical especially if related to varicose veins.

There are two clinical forms of discoid eczema:

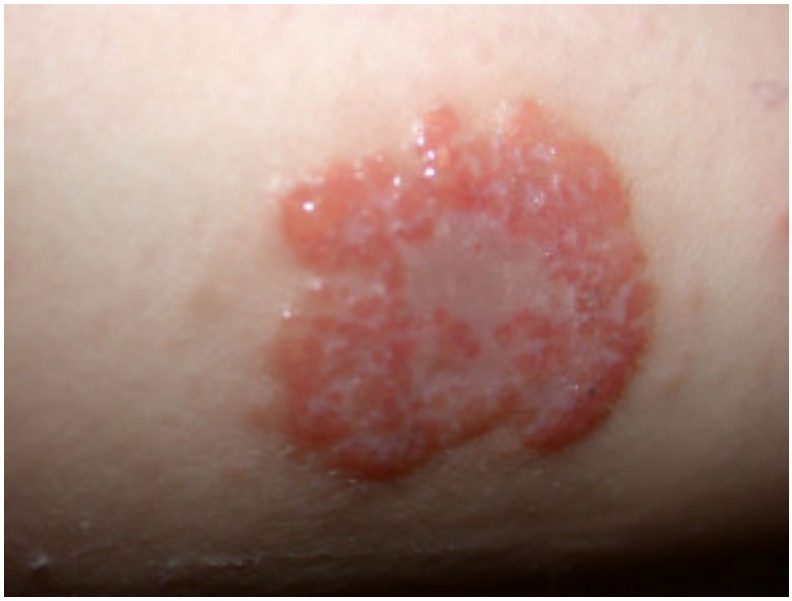
- Exudative acute discoid eczema: oozy papules, blisters, and plaques
- Dry discoid eczema: subacute or chronic erythematous, dry plaques

Individual plaques are well circumscribed, mostly 1–3 cm in diameter, and inflamed. The majority of patches are round or oval. The plaques are usually very itchy. The skin between the patches is usually dry and irritable. Severe discoid eczema may generalise, with numerous small to large itchy plaques appearing all over the body due to an autoeczematisation reaction. Patches may clear up without leaving a sign. In dark skin, marks may persist for months as dark brown postinflammatory hyperpigmentation or pale postinflammatory hypopigmentation.

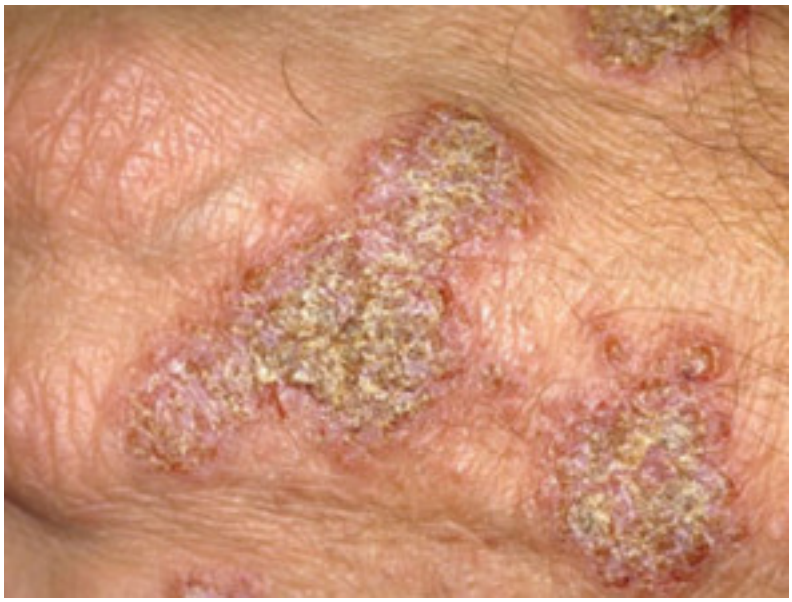
Exudative discoid eczema



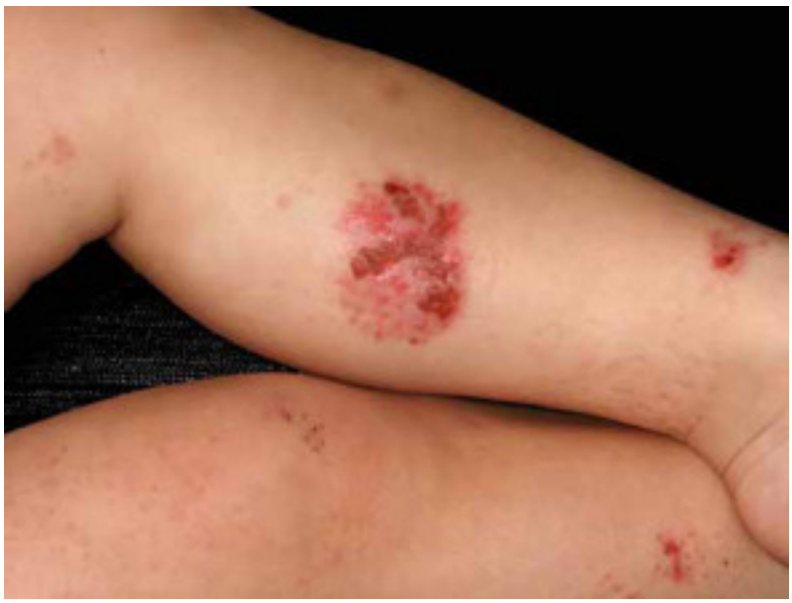
Discoid eczema



Discoid eczema



Discoid eczema



Infected nummular atopic dermatitis

How is discoid eczema diagnosed?

In most cases, the appearance of discoid eczema is quite characteristic.

- Bacterial swabs may reveal Staphylococcus aureus colonisation or infection.
- Scrapings are commonly taken for mycology, as discoid eczema can look very similar to tinea corporis (ringworm infection).
- Patch testing should be considered in chronic discoid eczema as contact allergy to metals, such as nickel and chromate, have been commonly reported.

What is the differential diagnosis of discoid eczema?

Discoid eczema may resemble other annular skin eruptions including tinea corporis, plaque psoriasis, and pityriasis rosea.

What is the treatment of discoid eczema?

As discoid eczema is associated with loss of skin barrier function, it is important to:

Protect the skin from injury.

This type of dermatitis often starts after minor skin injuries, so careful skin protection is required. If the hands are affected, use gloves and tools to make sure the skin is not irritated by friction, detergents, solvents, other chemicals, or excessive water.

Apply emollients frequently

Emollients include bath oils, soap substitutes and moisturising creams. They can be applied to dermatitis as frequently as required to relieve itching, scaling, and dryness. Emollients should also be used on the unaffected skin to reduce dryness. It may be necessary to try several different products to find one that suits. Many people find one or more of the following helpful: glycerine and cetomacrogol cream, white soft paraffin/liquid paraffin mixed, fatty cream, wool fat lotions, or urea cream.

Avoid allergens

If patch testing has identified contact allergy, exposure to the allergen should be avoided.

Anti-inflammatory treatments include:

Topical steroids

Topical steroids are anti-inflammatory creams or ointments available on prescription to apply just to the patches once or twice daily for 2–4 weeks. Topical steroids reduce symptoms and clear the dermatitis.

Antibiotics

Antibiotics (eg, erythromycin, flucloxacillin) are often prescribed if the dermatitis is blistered, sticky, or crusted. Sometimes discoid eczema clears completely on oral antibiotics, only to recur when they are discontinued. Other treatments sometimes prescribed for severe discoid eczema include:

Oral antihistamines

Antihistamine pills may reduce the itch in some patients with discoid eczema. They do not clear the rash.

Ultraviolet radiation (UV) treatment

Phototherapy several times weekly for 6–12 weeks for generalised or widespread discoid eczema can reduce itch and improve the rash.

Steroid injections

Intralesional steroids are sometimes injected into one or two particularly stubborn areas of discoid eczema. This treatment is unsuitable for multiple lesions.

Oral steroids

Systemic steroids are reserved for severe and extensive cases of discoid eczema. They are usually prescribed for a few weeks before continuing topical steroids and emollients on residual dermatitis.

Other oral treatments

Persistent and troublesome discoid eczema is occasionally treated with methotrexate, azathioprine or ciclosporin. These medicines require careful monitoring by a specialist dermatologist. They may be more suitable than long-term systemic steroids.

What is the outcome of discoid eczema?

Discoid eczema tends to be a chronic condition that often relapses especially in cold winter months. Many cases do eventually resolve.

Seborrhoeic dermatitis

What is seborrhoeic dermatitis?

Seborrhoeic dermatitis (American spelling is ‘seborrheic’) is a common, chronic or relapsing form of eczema/dermatitis that mainly affects the sebaceous, gland-rich regions of the scalp, face, and trunk .

There are infantile and adult forms of seborrhoeic dermatitis. It is sometimes associated with psoriasis (sebopsoriasis). Seborrhoeic dermatitis is also known as seborrhoeic eczema.

Dandruff (also called ‘pityriasis capitis’) is an uninflamed form of seborrhoeic dermatitis. Dandruff presents as bran-like scaly patches scattered within hair-bearing areas of the scalp.

What causes seborrhoeic dermatitis?

The cause of seborrhoeic dermatitis is not completely understood. It is associated with proliferation of various species of the skin commensal Malassezia, in its yeast (non-pathogenic) form. Its metabolites (such as the fatty acids oleic acid, malssezin, and indole-3-carbaldehyde) may cause an inflammatory reaction. Differences in skin barrier lipid content and function may account for individual presentations.

Who gets seborrhoeic dermatitis?

Infantile seborrhoeic dermatitis affects babies under the age of 3 months and usually resolves by 6–12 months of age.
Adult seborrhoeic dermatitis tends to begin in late adolescence. Prevalence is greatest in young adults and in older people. It is more common in males than in females.

The following factors are sometimes associated with severe adult seborrhoeic dermatitis:

- Oily skin (seborrhoea)
- Familial tendency to seborrhoeic dermatitis or a family history of psoriasis
- Immunosuppression: organ transplant recipient, human immunodeficiency virus (HIV) infection and patients with lymphoma
- Neurological and psychiatric diseases: Parkinson disease, tardive dyskinesia, depression, epilepsy, facial nerve palsy, spinal cord injury, and congenital disorders such as Down syndrome
- Treatment for psoriasis with psoralen and ultraviolet A (PUVA) therapy
- Lack of sleep, and stressful events.

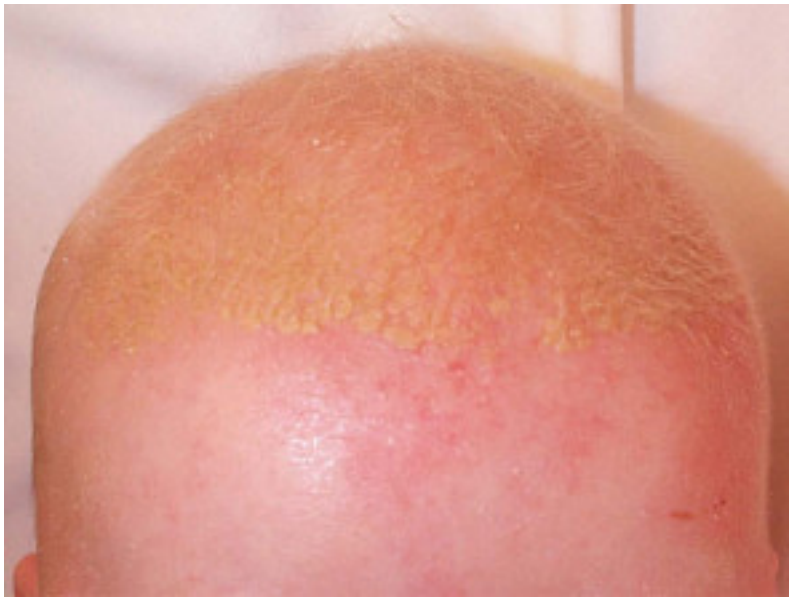
What are the clinical features of seborrhoeic dermatitis?

Infantile seborrhoeic dermatitis

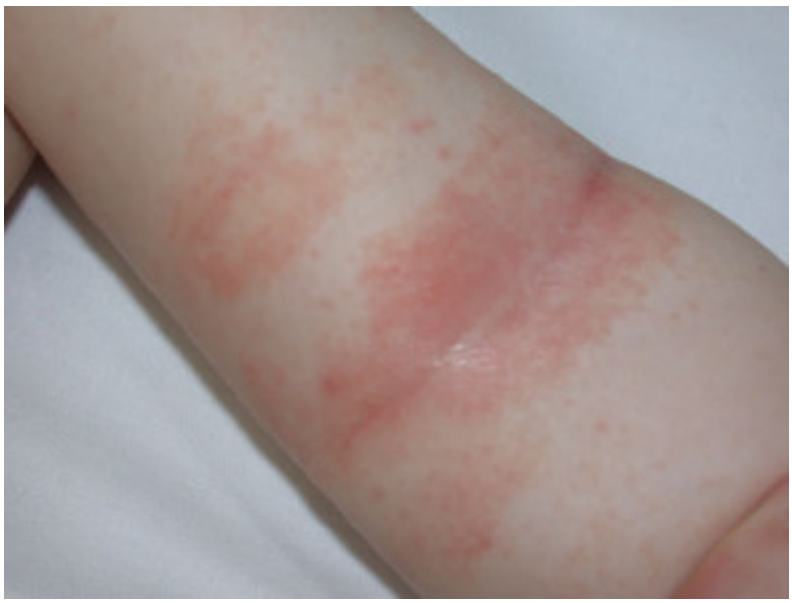
Infantile seborrhoeic dermatitis causes cradle cap (diffuse, greasy scaling on scalp). The rash may spread to affect armpit and groin folds (a type of napkin dermatitis).

- There are salmon-pink patches that may flake or peel.
- It is not especially itchy, so the baby often appears undisturbed by the rash, even when generalised.

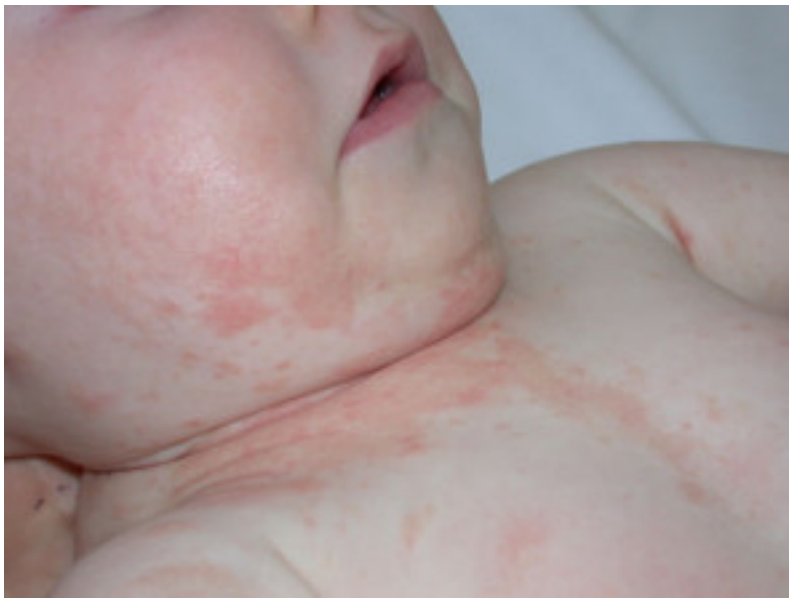
Infantile seborrhoeic dermatitis



Seborrhoeic dermatitis



Seborrhoeic dermatitis



Seborrhoeic dermatitis

Adult seborrhoeic dermatitis

Seborrhoeic dermatitis affects scalp, face (creases around the nose, behind ears, within eyebrows) and upper trunk.

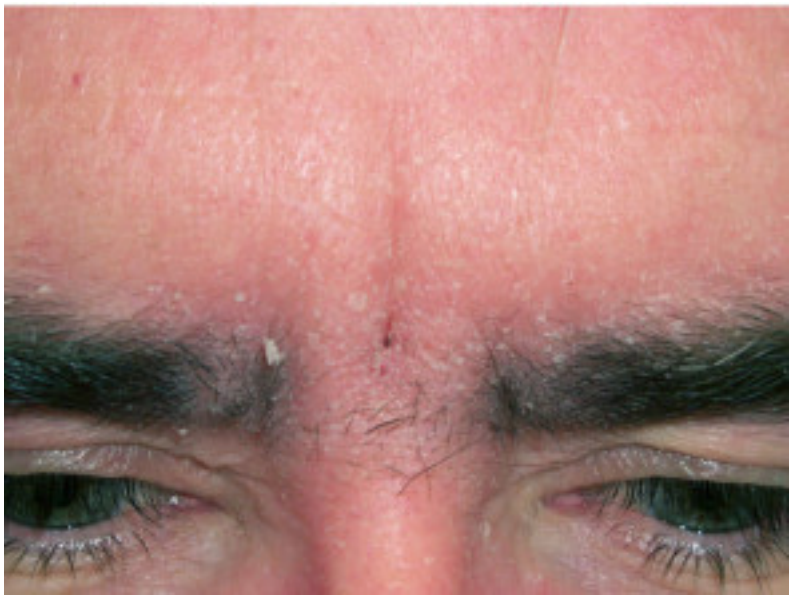
Typical features include:

- Winter flares, improving in summer following sun exposure
- Minimal itch most of the time
- Combination oily and dry mid-facial skin

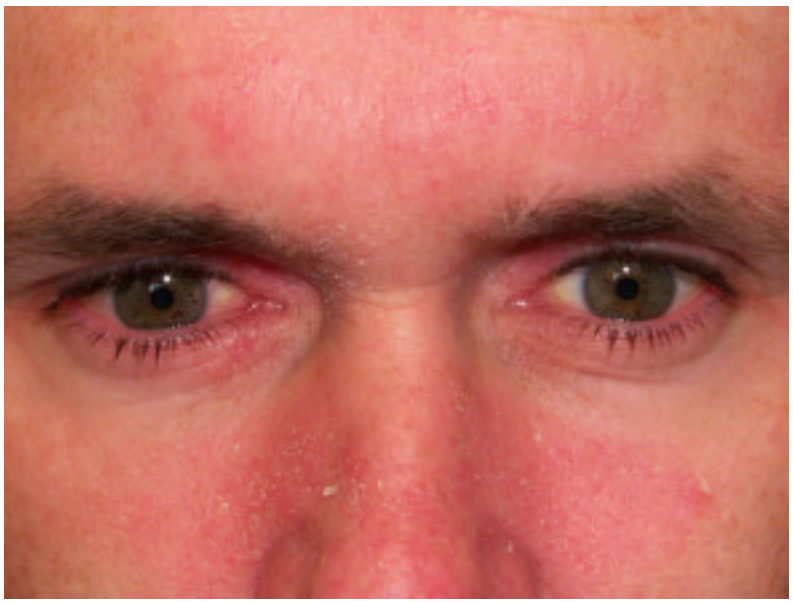
- Ill-defined localised scaly patches or diffuse scale in the scalp
- Blepharitis: scaly red eyelid margins
- Salmon-pink, thin, scaly, and ill-defined plaques in skin folds on both sides of the face
- Petal or ring-shaped flaky patches on hair-line and on anterior chest
- Rash in armpits, under the breasts, in the groin folds, and genital creases
- Superficial folliculitis (inflamed hair follicles) on cheeks and upper trunk.

Extensive seborrhoeic dermatitis affecting scalp, neck and trunk is sometimes called pityriasiform seborrhoeide.

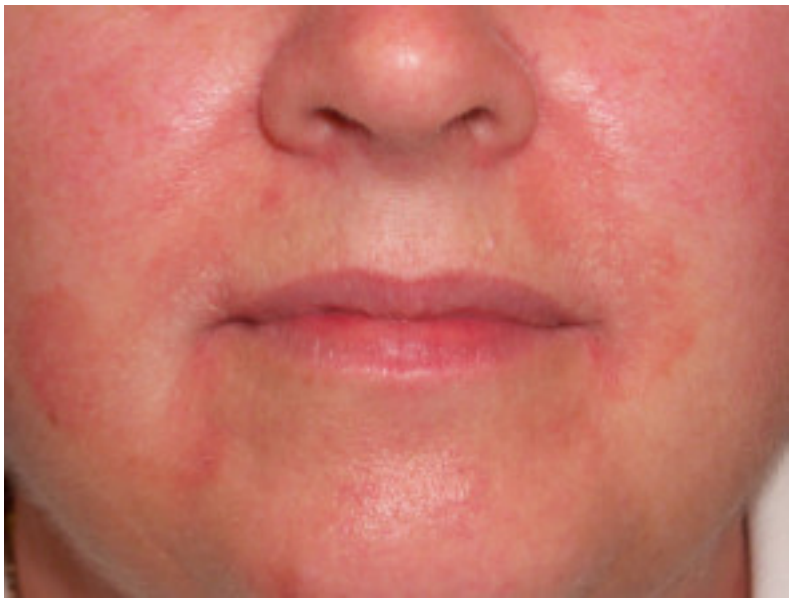
Seborrhoeic dermatitis



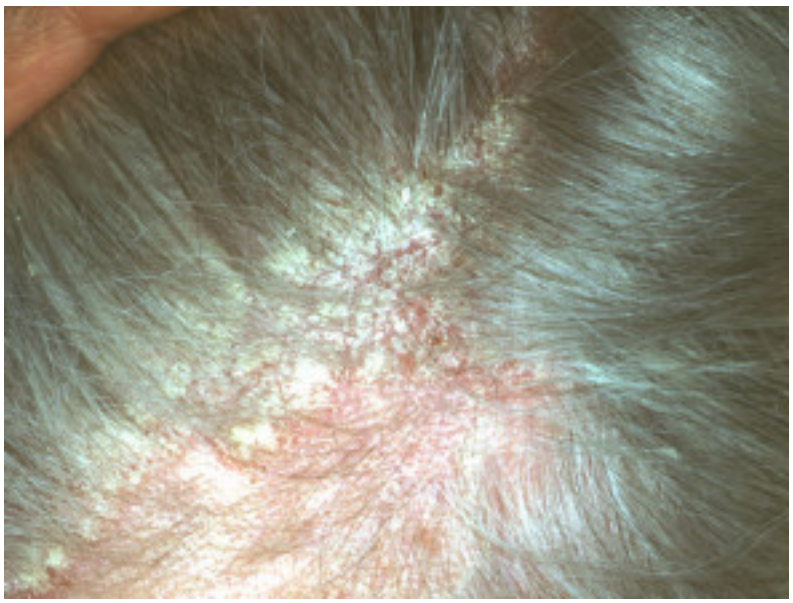
Seborrhoeic dermatitis



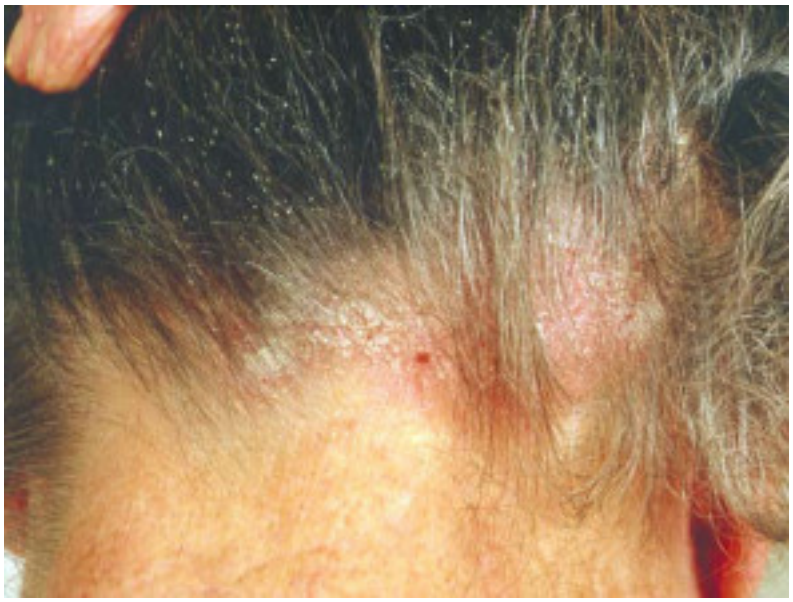
Seborrhoeic dermatitis



Seborrhoeic dermatitis



Seborrhoeic dermatitis



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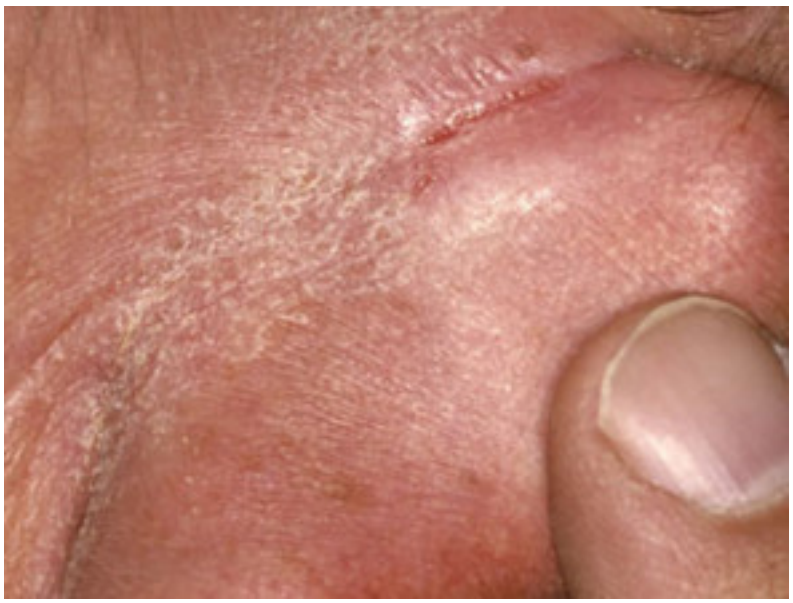

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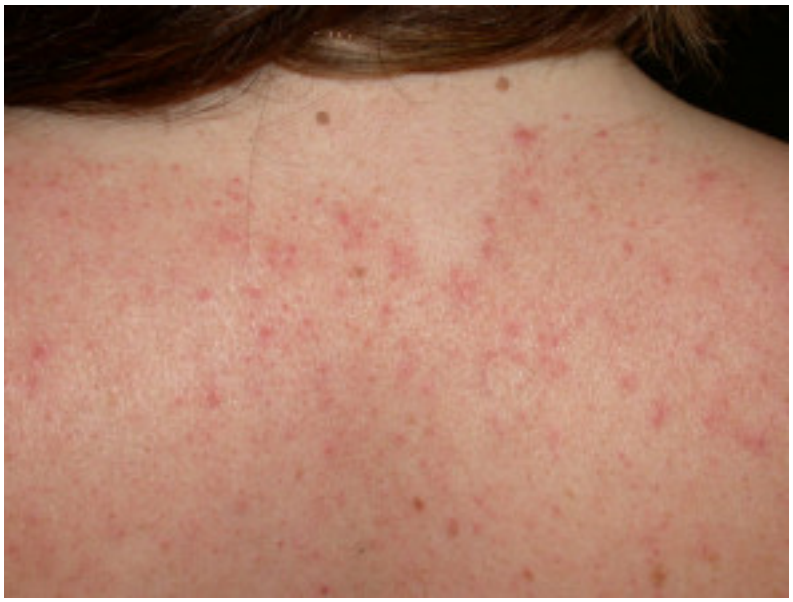

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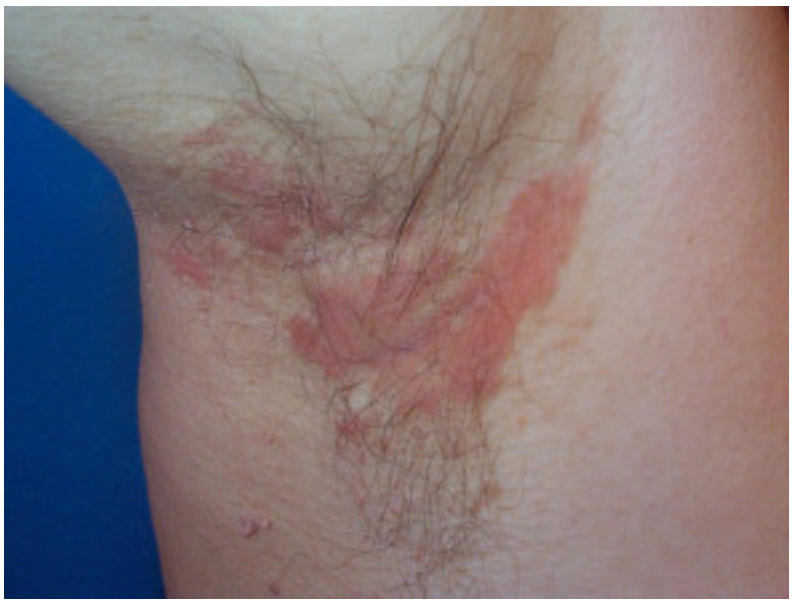
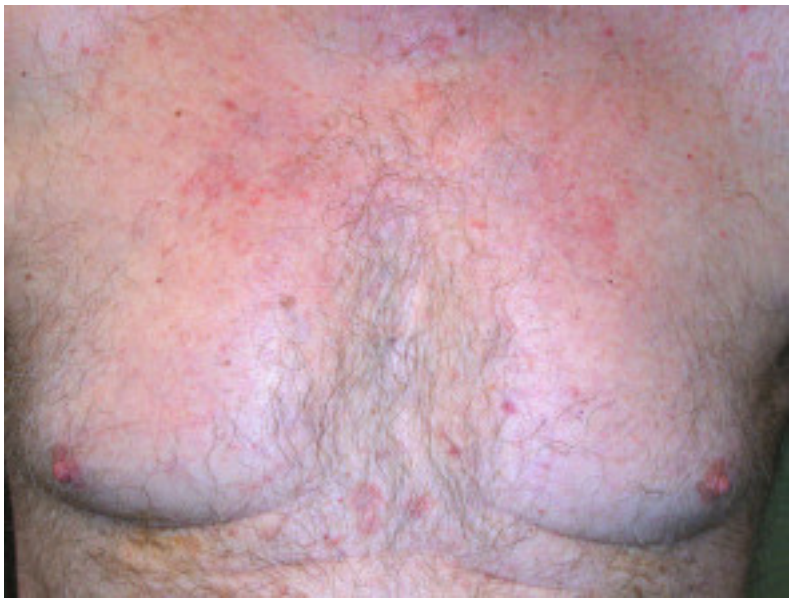


Seborrhoeic dermatitis



Seborrhoeic dermatitis

How is seborrhoeic dermatitis diagnosed?

Seborrhoeic dermatitis is diagnosed by its clinical appearance and behaviour. As malassezia are a normal component of skin flora, their presence on microscopy of skin scrapings is not diagnostic.

Skin biopsy may be helpful but is rarely indicated. Histological findings specific to seborrhoeic dermatitis are superficial perivascular and perifollicular

inflammatory infiltrates, psoriasiform hyperplasia, and parakeratosis around follicular openings.

What is the treatment for seborrhoeic dermatitis?

Treatment of seborrhoeic dermatitis often involves several of the following options.

- Keratolytics can be used to remove scale when necessary, eg salicylic acid, lactic acid, urea, propylene glycol
- Topical antifungal agents are applied to reduce malassezia eg ketoconazole, or ciclopirox shampoo and/or cream. Note, some strains of malassezia are resistant to azole antifungals. Try zinc pyrithione or selenium sulphide
- Mild topical corticosteroids are prescribed for 1–3 weeks to reduce the inflammation of an acute flare
- Topical calcineurin inhibitors (pimecrolimus cream, tacrolimus ointment) are indicated if topical corticosteroids are often needed, as they have fewer adverse effects on facial skin.

In resistant cases in adults, oral itraconazole, tetracycline antibiotics or phototherapy may be recommended. Low dose oral isotretinoin has also been shown to be effective for severe or moderate seborrhoeic dermatitis.

Scalp treatment

- Medicated shampoos containing ketoconazole, ciclopirox, selenium sulfide, zinc pyrithione, coal tar, and salicylic acid, used twice weekly for at least a month and if necessary, indefinitely.
- Steroid scalp applications reduce itching, and should be applied daily for a few days every so often.
- Calcineurin inhibitors such as tacrolimus can be used as steroid alternatives.
- Coal tar cream can be applied to scaling areas and removed several hours later by shampooing.
- Combination therapy is often advisable.

Face, ears, chest and back

- Cleanse the affected skin thoroughly once or twice each day using a non-soap cleanser.
- Apply ketoconazole or ciclopirox cream once daily for 2 to 4 weeks, repeated as necessary.
- Hydrocortisone cream can also be used, applied up to twice daily for 1 or 2 weeks. Occasionally a more potent topical steroid may be prescribed.
- Topical calcineurin inhibitors such as pimecrolimus cream or tacrolimus ointment may be used instead of topical steroids.
- A variety of herbal remedies are commonly used, but their efficacy is uncertain.

Management in infants

Regular washing of the scalp with baby shampoo or aqueous cream is followed by gentle brushing to clear the scales.

- White petrolatum may be useful.
- Topical antifungals are often prescribed, depending on the extent of the rash.

Venous eczema

What is venous eczema?

Venous eczema is a common form of eczema/dermatitis that affects one or both lower legs in association with venous insufficiency. It is also called gravitational dermatitis.

Who gets venous eczema?

Venous eczema is most often seen in middle-aged and older patients — it is reported to affect 20% of those over 70 years. It is associated with:

- History of deep venous thrombosis in an affected limb
- History of cellulitis in an affected limb
- Chronic swelling of the lower leg, aggravated by hot weather and prolonged standing

- Varicose veins
- Venous leg ulcers.

What causes venous eczema?

Venous eczema appears to be due to fluid collecting in the tissues and activation of the innate immune response.

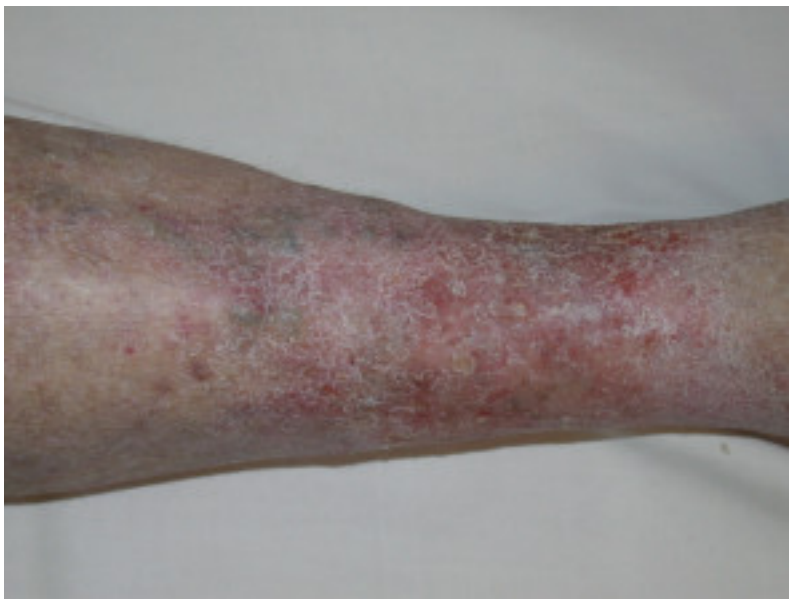
Normally during walking the leg muscles pump blood upwards and valves in the veins prevent pooling. A clot in the deep leg veins (deep venous thrombosis or DVT) or varicose veins may damage the valves. As a result back pressure develops and fluid collects in the tissues. An inflammatory reaction occurs.

What are the clinical features of venous eczema?

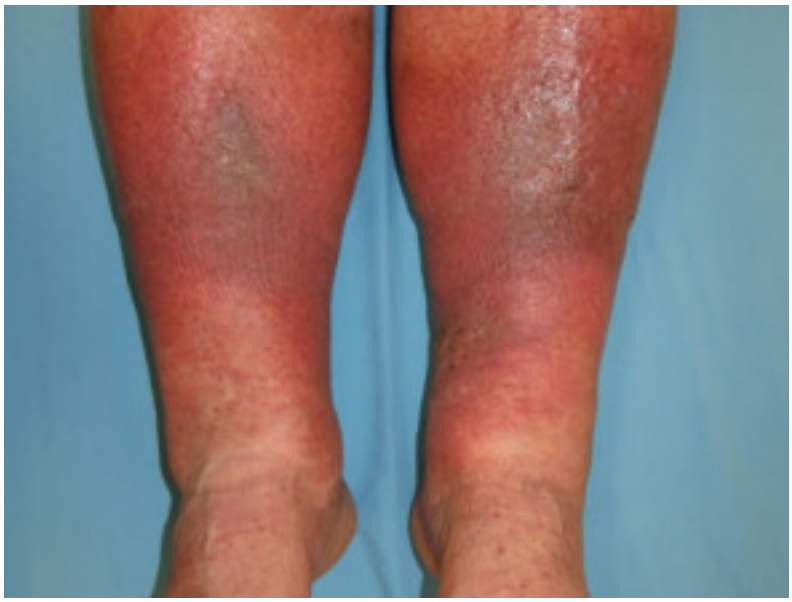
Venous eczema can form discrete patches or become confluent and circumferential. Features include:

- Itchy red, blistered and crusted plaques; or dry fissured and scaly plaques on one or both lower legs
- Orange-brown macular pigmentation due to haemosiderin deposition
- Atrophie blanche (white irregular scars surrounded by red spots)
- 'Champagne bottle' shape of the lower leg — narrowing at the ankles and induration (lipodermatosclerosis)

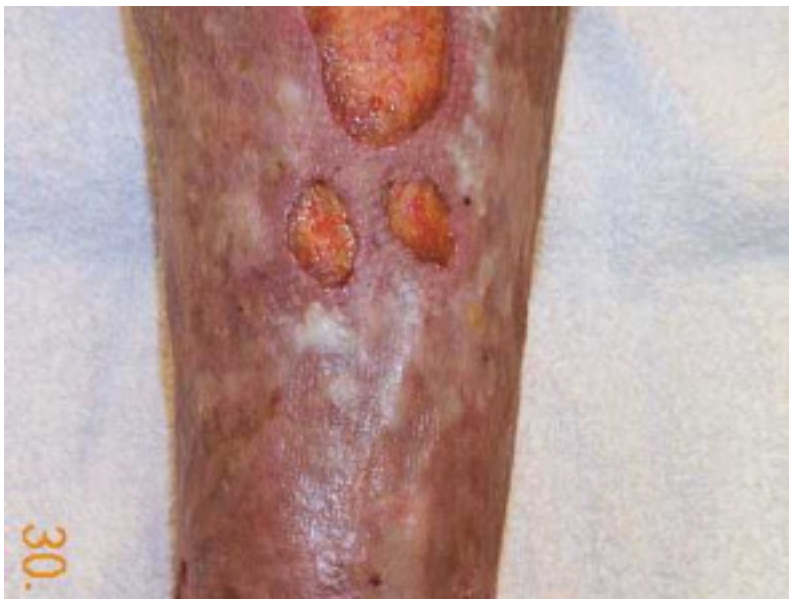
Venous disease



Venous eczema



Lipodermatosclerosis



Venous ulcer

What are the complications of venous eczema?

- Impetiginisation — secondary infection with Staphylococcus aureus resulting in yellowish crusts
- Cellulitis — infection with Streptococcus pyogenes: there may be redness, swelling, pain, fever, a red streak up the leg and swollen nodes in the groin
- Secondary eczema — eczema spreads to other areas on the body
- Contact allergy to one or more components of the ointments or creams used

How is venous eczema diagnosed?

The diagnosis of venous eczema is clinical.
Patch tests may be undertaken if there is suspicion of contact allergy.

What is the treatment for venous eczema?

Reduce swelling in the leg

- Don't stand for long periods.

- Take regular walks.
- Elevate your feet when sitting: if your legs are swollen they need to be above your hips to drain effectively.
- Elevate the foot of your bed overnight.
- During the acute phase of eczema, bandaging is important to reduce swelling.
- When eczema has settled, wear graduated compression socks or stockings long term. Fitted moderate to high compression socks can be obtained from a surgical supplies company. Light compression using travel socks may be adequate, and these are easy to put on. They can be bought at pharmacies, travel and sports stores. More compression is obtained by wearing two pairs.
- Horse chestnut extract appears to be of benefit for at least some patients with venous disease.

Treat the eczema

- Dry up oozing patches with Condy's solution (potassium permanganate) or dilute vinegar on gauze as compresses.
- Oral antibiotics such as flucloxacillin may be prescribed for a secondary infection.
- Apply a prescribed topical steroid: start with a potent steroid cream applied accurately daily to the patches until they have flattened out. After a few days, change to a milder steroid cream (eg. hydrocortisone) until the itchy patches have resolved (maintenance treatment). Check with your doctor if you are using steroid creams for more than a few weeks. Overuse can thin the skin, but short courses of stronger preparations can be used from time to time if necessary to control dermatitis. Coal tar ointment may also help.
- Use a moisturising cream frequently to keep the skin on the legs smooth and soft. If the skin is very scaly, urea cream may be especially effective.
- Protect your skin from injury: this can result in infection or ulceration that may be difficult to heal.
-

Treatment for varicose veins

- Seek the opinion of a vascular surgeon regarding varicose veins.
- These can be treated surgically, by endovenous laser, or sclerotherapy.

- Varicose veins may develop again after an apparently successful operation because venous disease is progressive.

How can venous eczema be prevented?

Venous eczema cannot be completely prevented but the number and severity of flare-ups can be reduced by the following measures.

- Avoid prolonged standing or sitting with legs down.
- Wear compression socks or stockings.
- Avoid and treat leg swelling.
- Apply emollients frequently and regularly to dry skin.
- Avoid soap; use water alone or non-soap cleansers when bathing.

What is the outlook for venous eczema?

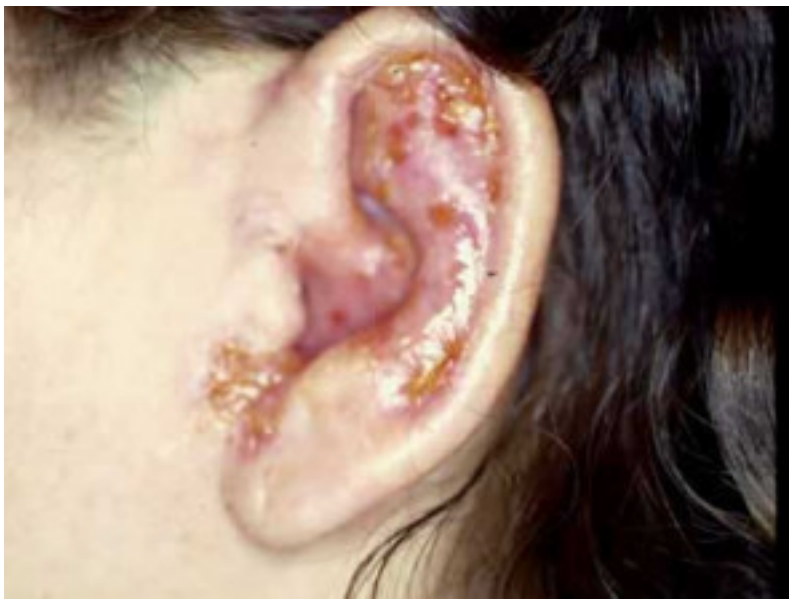
Venous eczema tends to be a recurring or chronic disorder lifelong. Treat recurrence promptly with topical steroids.

Otitis externa

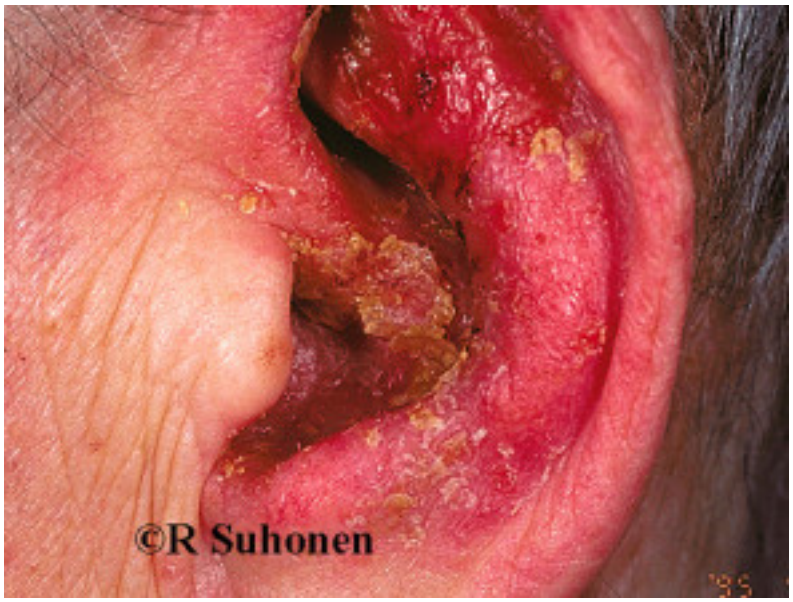
What is otitis externa?

Otitis externa is an inflammatory condition of the external auditory canal (the ear canal). It is characterised by redness, swelling, scaling and thickening of the canal skin lining and is accompanied by varying degrees of discomfort, itch, deafness and discharge.

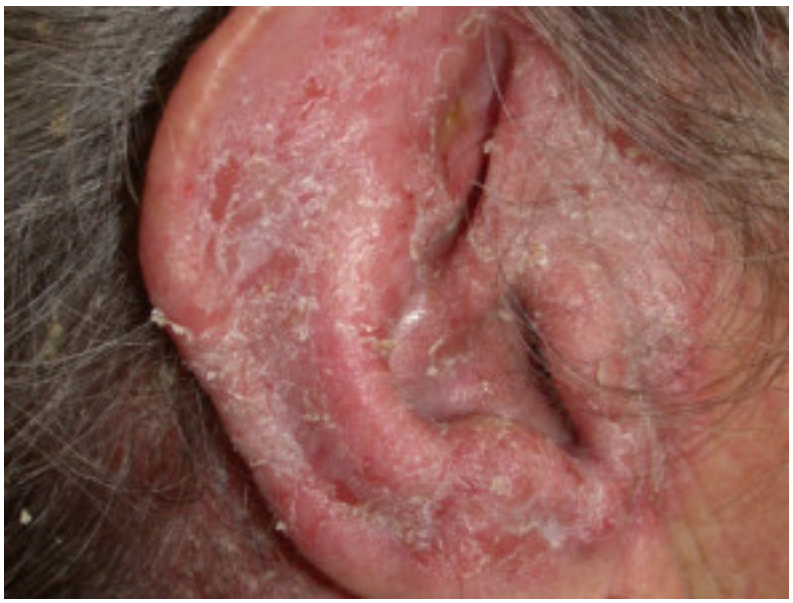
Otitis externa



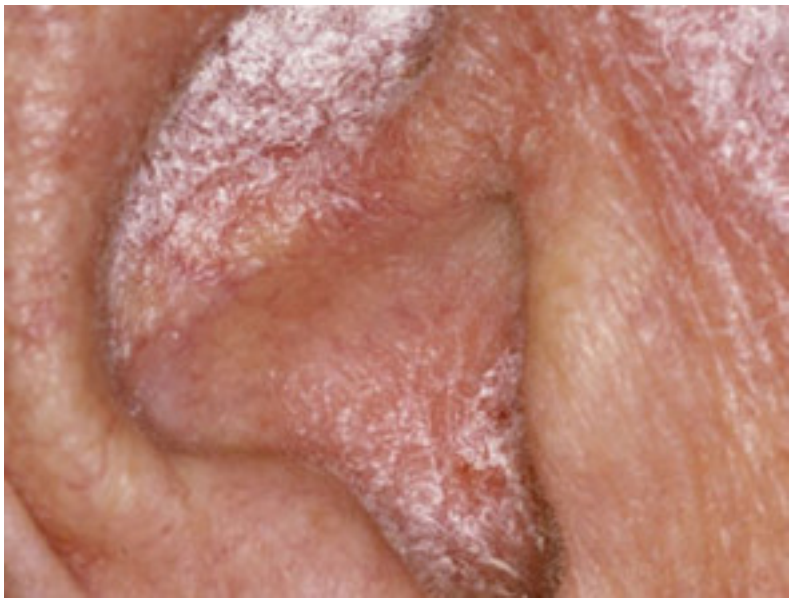
Staphylococcus



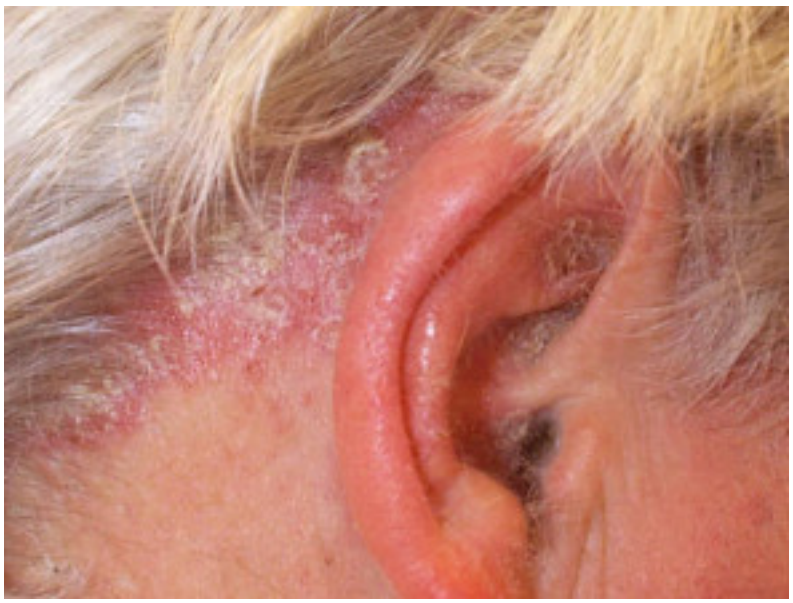
Dermatitis



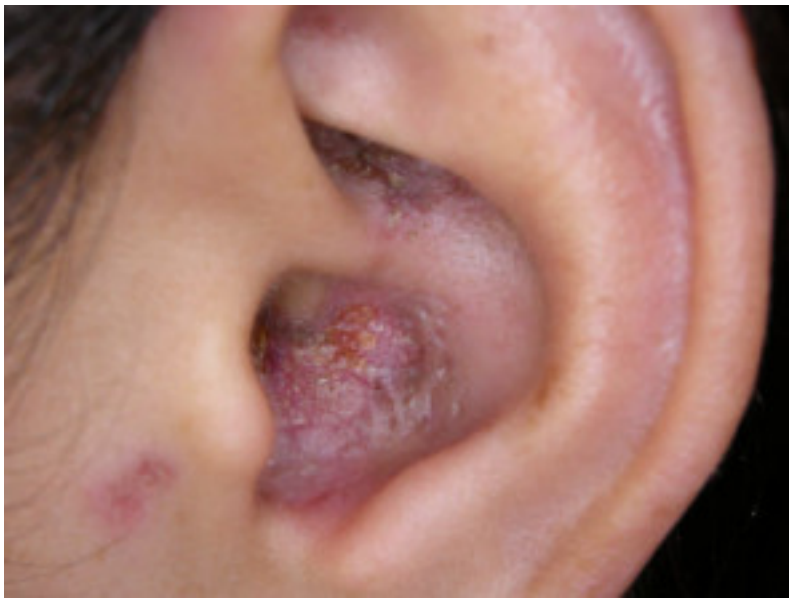
Dermatitis



Psoriasis



Psoriasis



Cutaneous lupus

What causes otitis externa?

The causes of otitis externa can be split into two main groups: those caused by bacterial or fungal infection and those by non-infectious dermatological conditions. Bacterial infections are the most common cause of otitis externa. Primary skin disorders are often precipitants of infectious otitis externa, but they can also be the sole cause of otitis externa.

Infectious otitis externa

As with all skin the external auditory canal has a normal bacterial flora that remains free from infection until skin defences fail or become damaged. Some common causes that allow the overgrowth of bacteria in the external ear include:

- Swimming, perspiration, high humidity – these create excessive moisture that carry bacteria into the cerumen (ear wax) of the ear canal, leading to maceration and inflammation
- Local trauma to the ear canal allowing bacteria to enter damaged skin, e.g. insertion of objects such as cotton buds, matchsticks and fingers to relieve itching or impacted earwax
-

Bacteria commonly implicated in otitis externa include Pseudomonas aeruginosa and Staphylococcus aureus. In about 10% of cases of infectious otitis externa, fungal infections are the cause. The most common fungal pathogen is Aspergillus (80-90%), followed by Candida. Mixed bacterial and fungal infections are common.

Non-infectious dermatological causes

Otitis externa caused by dermatological conditions are often referred to as “eczematous otitis externa”. Skin conditions that may cause otitis externa include:

- Atopic dermatitis (eczema)
- Psoriasis
- Seborrhoeic dermatitis
- Acne
- Cutaneous lupus erythematosus (rare)
- Irritant or allergic contact dermatitis – from local irritants, including topical preparations or use of hearing aids or ear plugs

Often the condition is complicated by secondary bacterial infections.

What are the signs and symptoms?

The most common symptoms of otitis externa are otalgia (ear discomfort) and otorrhoea (discharge from the external auditory canal). Ear discomfort can range from pruritus (itching) to severe pain that is worsened by motion of the ear, e.g. chewing. Discharge from the ear varies between patients and may give a clue to the cause of the condition. Swelling within the external auditory canal may cause feeling of fullness in the ear and loss of hearing. The clinical features of otitis externa may vary according to the cause.

Bacteria

- Significant swelling of canal is common
- Discomfort is often severe enough to require oral analgesics
- Fever may be present
- Lymphadenopathy (swollen lymph nodes) around the base of the ear
- Discharge is usually scant white mucus, but occasionally thick in acute infection
- Bloody discharge in the presence of granulation tissue in chronic infection

Fungi

- Often there are no symptoms apart from a discharge, this is typically a fluffy white to off-white discharge, but may be black, grey, bluish-green or yellow
- If symptoms are present, discomfort in the form of pruritus and a feeling of fullness in the ear is most common. Pruritus may be quite intense, resulting in scratching and further damage to the skin lining
- Tinnitus (ringing in the ears)

Atopic dermatitis

- Intensely itchy
- Typically part of a more generalised skin involvement, including the external ears, face and neck
- Skin may become red, thickened, crusty and hyperpgimented from scratching intense itch

Psoriasis

- Commonly associated with scalp involvement but rarely facial involvement
- Raised, red lesion with thick, silvery-white adherent scale
- Often itchy

Allergic contact dermatitis

- Occurs suddenly
- Red, swollen, itchy and exuding lesions
- External auditory canal may react to allergens that do not cause a reaction elsewhere

- May affect the outer ear and lobe

Irritant contact dermatitis

- Slower onset than allergic contact dermatitis
- Lesions are usually patches of thickened, hardened skin
- May affect the outer ear and lobe

How is the diagnosis made?

History taking and physical examination is often all that is required to make a diagnosis of otitis externa. If fever or signs of toxicity are present, perform standard laboratory testing. Gram staining and culture of the discharge may be helpful, particularly when a bacterial or fungal cause is suspected.

What treatment is available?

Initial treatment begins with cleaning debris and wax from the canal. Once the ear is cleaned specific treatment that is prescribed according to the cause of otitis externa should be administered. Occasionally if swelling in the ear is severe, a wick may be inserted before medication is applied, usually in the form of topical eardrops.

Bacteria

- 2% acetic acid solution – inexpensive and effective against most infections but can be irritating to inflamed canal
- Neomycin otic drops – effective but can cause contact dermatitis in 15% of patients
- Polymixin B drops – avoids potential neomycin sensitisation but is ineffective against Staphylococcus and other gram-positive organisms
- Aminoglycoside drops – less irritating than previous preparations but has potential ototoxicity
- Fluoroquinolone drops (ofloxacin, ciprofloxacin) – very effective without causing irritation or sensitisation, no risk of ototoxicity, but is expensive and overuse may cause antibiotic resistance in an important class of antibiotics
- Topical drops that combine antibiotic with steroids may help to reduce inflammation and help resolve symptoms more quickly

- Drops are usually administered 3-4 times daily (fluoroquinolones only require twice daily administration) for 5-7 days. More severe infections may require 10-14 days treatment. Drops should be continued for 3 days more after symptoms disappear.
- Oral antibiotics are rarely indicated except for in severe and persistent otitis externa

Fungi

- 2% acetic acid solution 3-4 times daily for 5-7 days
- If the infection does not respond to acidifying drops, antifungal drop such as clotrimazole can be used
- Aspergillus infections may be resistant to clotrimazole and require oral itraconazole

Systemic dermatological disease, e.g. psoriasis

- Goal of treatment should be to control the systemic dermatological disease
- Because these conditions are often inflammatory, topical steroid drops may be used but this can often lead to bacterial or fungal superinfection. Acidifying drop may be added to prevent secondary infection.

Contact dermatitis

- Remove the irritant or allergen
- Topical steroids may be useful
- Burow's otic solution (aqueous solution of aluminium acetate) with 2% acetic acid may be added to prevent secondary infections, reacidify the skin, dry weeping lesions and remove crusts.

Patients should be educated about how to prevent recurrences of otitis externa. Some simple general measures include:

- Wear a tight fitting swimming cap to prevent water entering the ear canal
- Attention to drying the ears after swimming or showering
- Patients prone to recurrences may use acidifying drops after swimming or water sports

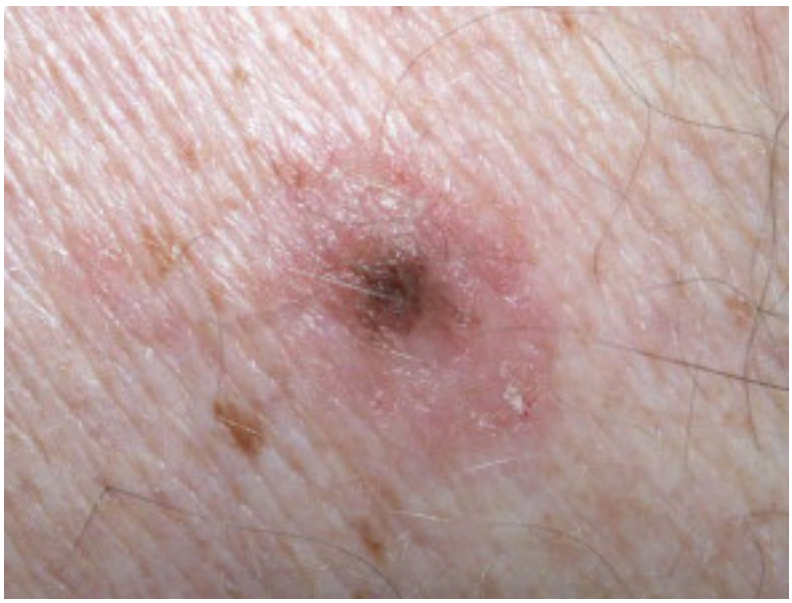
- Avoid poking and scratching the skin of the external auditory canal as damage to the skin and removal of earwax makes the canal more vulnerable to infection
- Patients with otitis externa should preferably abstain from water sports for at least 7-10 days.

Meyerson naevus

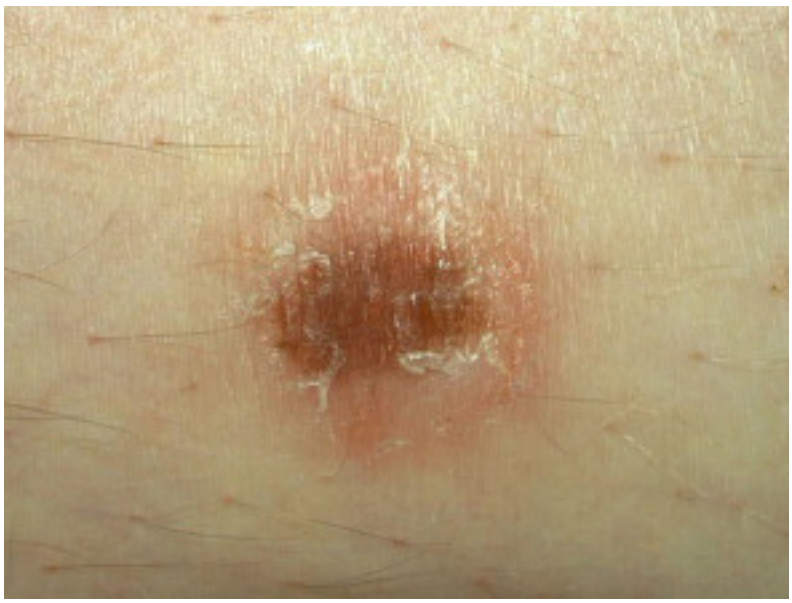
What is Meyerson naevus?

Meyerson naevus is a mole that is surrounded by eczema (an itchy rash also known as dermatitis). A mole is more properly called a benign melanocytic naevus.
Meyerson first described the condition in 1971 where he reported 2 men with benign melanocytic naevi surrounded by eczema. Meyerson naevus is also referred to as halo dermatitis, halo eczema and Meyerson's phenomenon.

Meyerson naevus



Meyerson naevus



Meyerson naevus

Who gets Meyerson naevus?

Meyerson naevus appears to occur more commonly in young males (average age 30 years). It is nearly three times as common in males compared to females. Meyerson naevi may co-exist with halo naevi. It may also occur in patients with eczema or other atopic conditions (these are asthma and hay fever). However, it most often occurs in healthy individuals without any pre-existing conditions.

What is the cause of Meyerson naevus?

The cause of Meyerson naevus is not known. Skin biopsy specimens show inflamed skin rather like that seen in psoriasis and eczema.
Excision of the central mole results in resolution of the eczematous reaction, which suggests the mole is the reason for it. It may be the result of immune cells (CD4+ lymphocytes) reacting against target antigens on the surface of the naevus cells (these are melanocytes).

Factors that have been found to trigger Meyerson naevus include:

- Exposure to ultraviolet radiation (in one case halo dermatitis lesions developed a few weeks after the patient experienced a severe sunburn)

- Interferon-alfa-2b (lesions developed in a patient with Behcet disease that was treated with this)
-

What are the signs and symptoms of Meyerson naevus?

Meyerson naevus usually develops as a single itchy patch, but multiple lesions appearing at the same time have also been reported. Meyerson naevus consists of a centrally positioned naevus that has regular borders and has a uniform colour. This is surrounded by an red, dry or blistered rash that may or may not be itchy. The naevus (mole) in the centre of the halo is rarely cancerous (melanoma).

Meyerson naevus is sometimes mistaken for a halo mole (Sutton naevus). The main difference between these two lesions is what happens to the central naevus. The lesion in the centre of a Meyerson naevus never changes or fades away even when the surrounding eczema has resolved. In contrast, the central mole of a halo mole usually fades in colour and eventually disappears altogether.

What is the treatment of Meyerson naevus?

The eczema around the Meyerson naevus usually resolves by itself within weeks. However, if necessary it can be treated with topical corticosteroid cream or ointment for a few days or weeks.

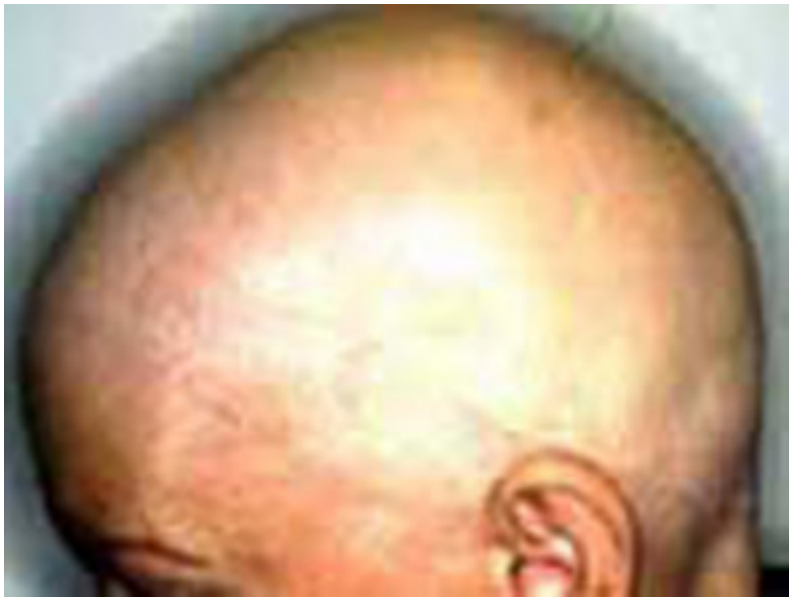
The central naevus remains unchanged. Because the naevus is harmless, it does not need to be removed. Surgical excision may be arranged if there is any concern that the lesion is melanoma, or because the symptoms are a nuisance.

Examination of hair and scalp

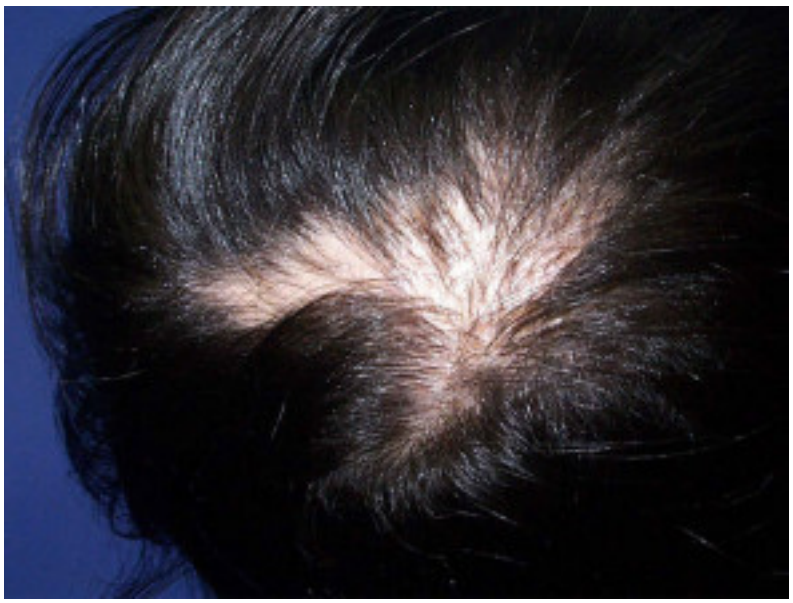
Examination of the hair and scalp, axillary and pubic hair and body hair is part of a full skin examination. Evaluate hair quantity and quality i.e. its length, density, colour and texture. Is it straight, wavy or curly? Look for associated skin conditions, especially those affecting the scalp.

Disorders affecting hair

Long hair



Total alopecia (from chemotherapy)



Patchy hair loss

Thinned or absent hair

Hair pull test

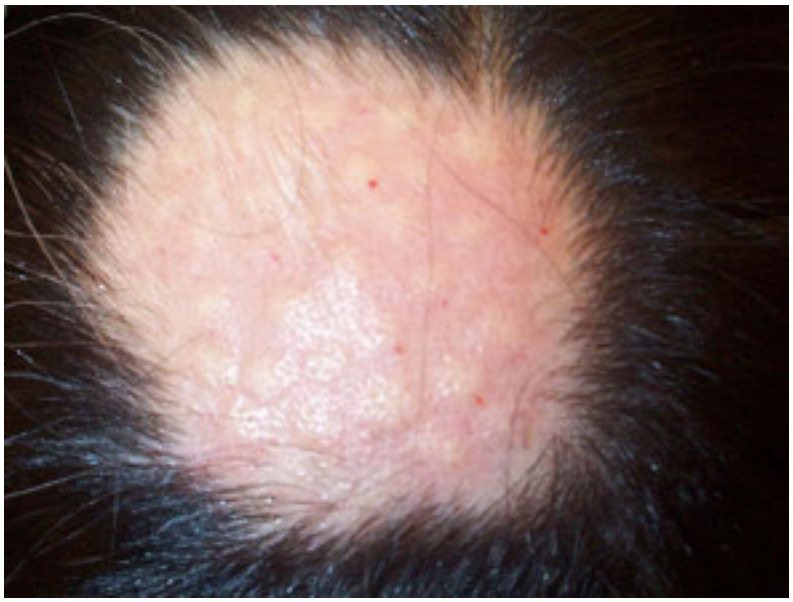
Hair loss associated with excessive shedding results in a positive “gentle hair pull” test. Grasp a lock of hairs to determine if any can be extracted with firm pull. Normally 0-2 telogen hairs can be extracted: these are hairs in the resting phase, identified using magnification by a rounded bulb at the proximal end. An elongated or tapered end indicates anagen hair (growing phase); anagen hairs extracted by the gentle hair pull test are pathologic.

Scalp hair

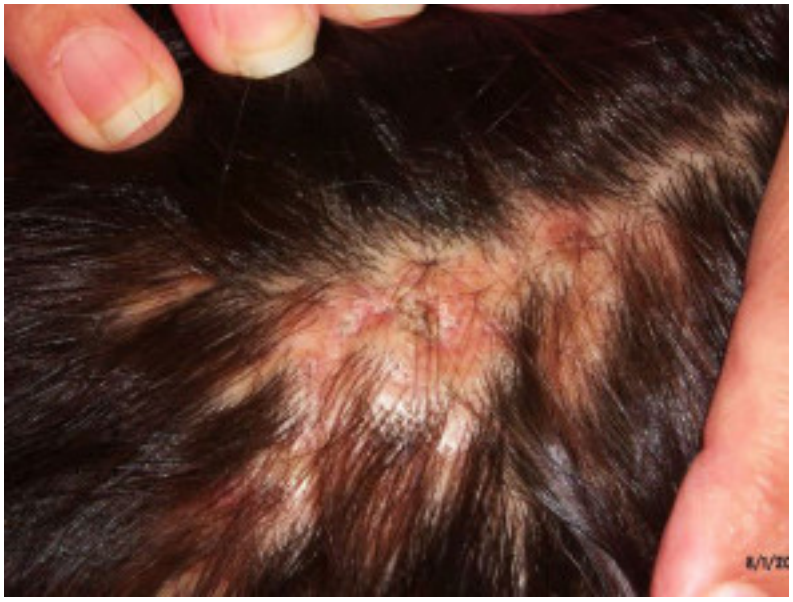
Thinning hair or balding (alopecia) may be localised or diffuse.
Localised alopecia may affect a single or multiple areas. Inspection may reveal:

- Round/oval, “moth-eaten” or linear bald patches
- Short hairs: these may be tapered at the tip (normal re-growth), “exclamation mark” (hairs tapered near scalp), broken-off, singed or cut
- Negative or positive hair pull test
- Scarring (cicatricial alopecia) i.e. no follicles, or non-scarring alopecia (follicular orifices present)
- Multiple hairs in single follicle (“tufted folliculitis”).

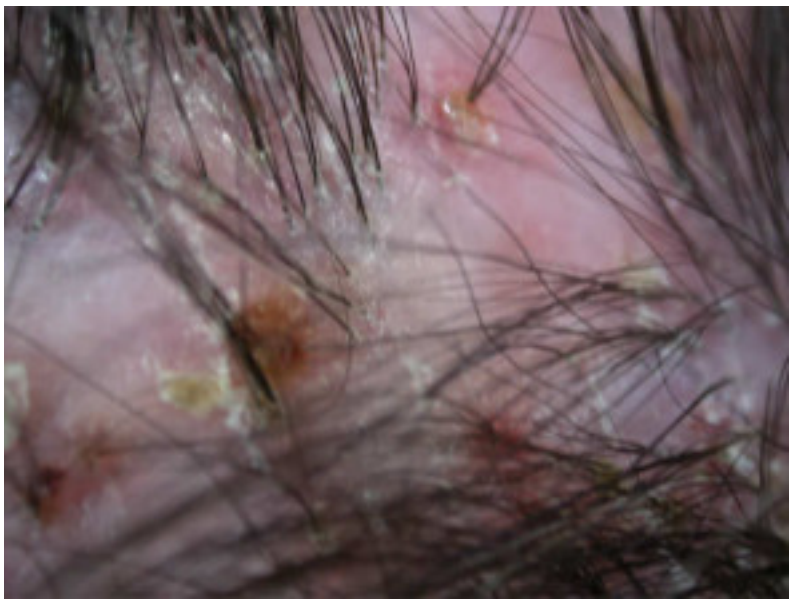
Localised alopecia



Localised alopecia areata



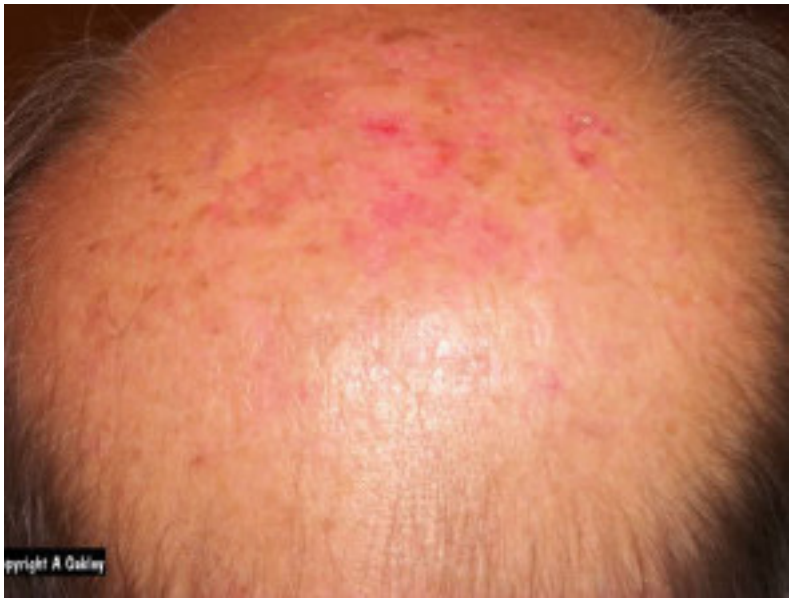
Scarring alopecia



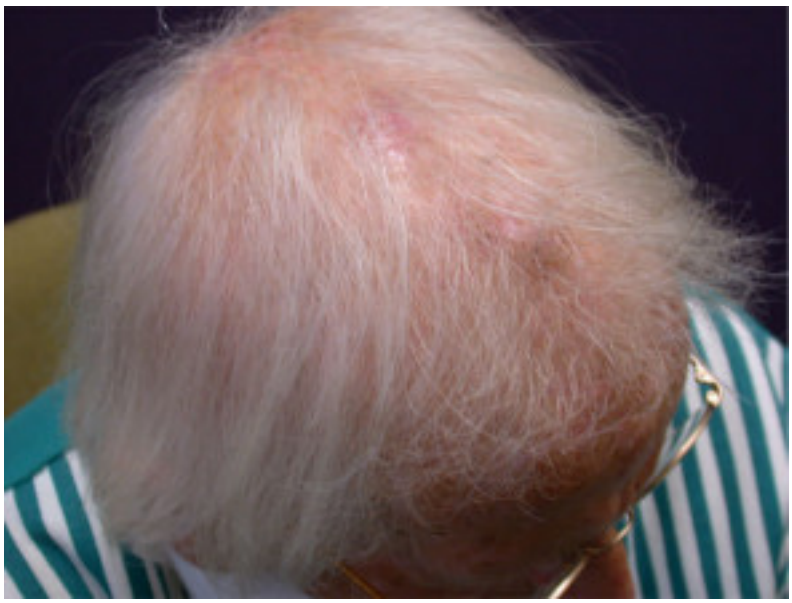
Tufted folliculitis

Diffuse alopecia is most often due to pattern balding, and more prominent over the vertex of the scalp. In males there is often frontal recession. Shedding is usually normal or mildly increased in pattern balding. The hair shaft is thinned.

Diffuse alopecia



Pattern balding (male)



Pattern balding (female)

Diffuse alopecia areata

Generalised diffuse alopecia is more likely to be associated with excessive shedding. A hair pull test may reveal numerous telogen and/or anagen hairs (telogen or anagen effluvium respectively).

Hair collected by patient with telogen effluvium

Hair ball

Rare genetic hair shaft abnormalities identified on microscopy are beyond the scope of this article.

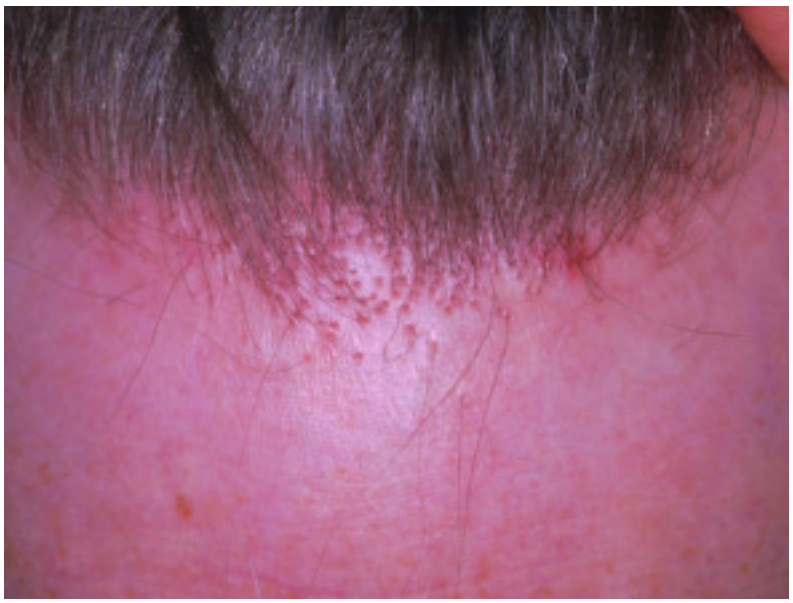
Scalp skin

Evaluate the appearance of the scalp, whether it is generally excessively oily or dry. Look for localised lesions and inflammatory skin diseases.
Evaluate:

- Diffuse, patchy or perifollicular erythema
- Diffuse, patchy or follicular flaking or scaling
- Follicular or non-follicular papules, erosions or pustules
- Hair shaft free or encased by a hair cast
- Nits (louse egg cases)
- Excoriations (an indication of severity of itching).

Disorders of the scalp

Scaling



Perifollicular erythema

Elsewhere

A complete examination should include inspection of terminal hair of the eyebrows, eyelashes, beard, axilla and pubic area as well as body hair generally.

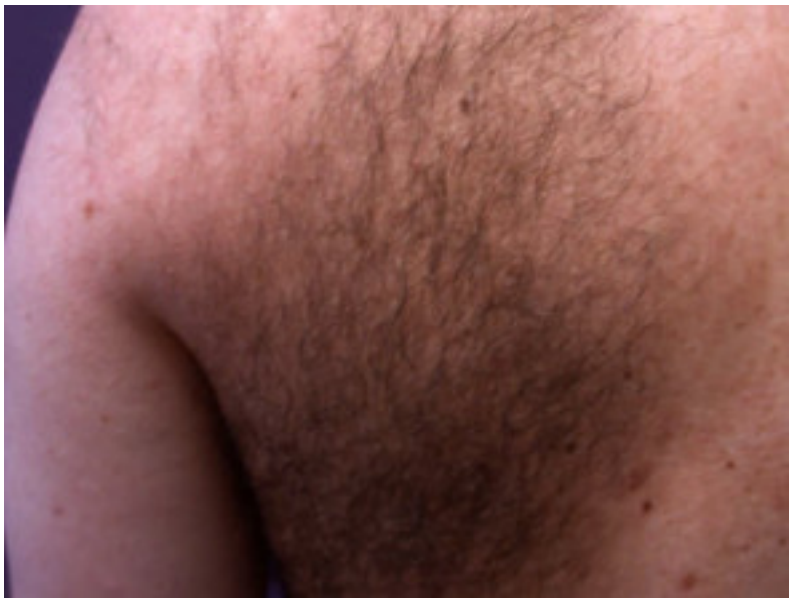
In adolescents where relevant, note stage of pubertal development using Tanner growth charts. Premature pubarche refers to appearance of pubic hair without other signs of puberty before age 9 years in boys, before age 7 years in white girls and before age 6 years in black girls.

Excessive hair

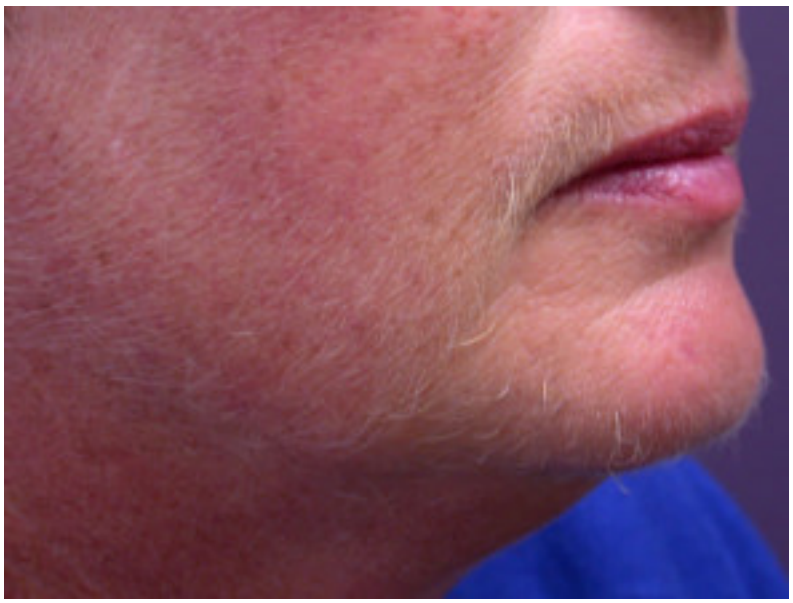
Excessive hair may be due to localised or diffuse hypertrichosis or in women, hirsutism, which refers to an adult male pattern of hair growth. Hypertrichosis describes localised or diffuse excessive hair on face, arms, legs or trunk. It may be due to increase in lanugo (soft, fine and blond) or terminal hair.

To assess hirsutism, evaluate presence and severity of terminal hair on the face in the beard areas and the lower abdomen. A diamond pattern for escutcheon indicates hirsutism, as the usual female pattern is a triangle. Hirsutism may also affect chest and back.

Excessive hair



Hair-bearing Becker's naevus



Facial hirsutes