Reference of TCM Dermatology

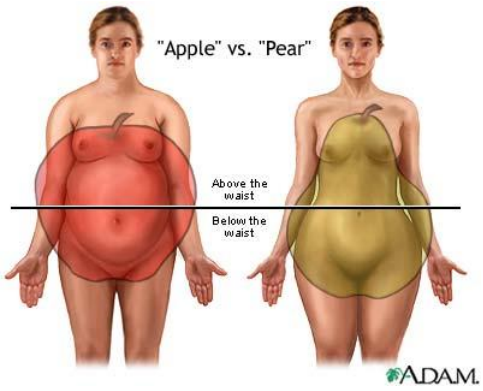
Yu Qi MD (China)

Contents

Chapter 1. TCM cosmetology 2
1. Obesity 2
2. Androgenetic Alopecia (Male pattern baldness) 3
3. Acne 痤疮 5
4. Melasma/Chloasma 黄褐斑 6
Chapter 2. Viral skin diseases 7
1. Herpes simplex 7
2. Herpes zoster 9
3. Genital herpes 生殖器疱疹 12
4. Wart 疣 15
Chapter 3. Fungi skin diseases 15
1. Tinea pedis 足癣 (Athlete's foot, Ringworm of feet) 15
2. Tinea capitis 头癣 16
3. Tinea cruris 股癣 17
Chapter 4. Bacterial skin diseases 19
1, Paronychia 甲沟炎 19
Chapter 5. Skin diseases due to physical factors 20
1. Decubitus Ulcer /Bed sore / pressure sore 褥疮 20
2. Miliaria (Prickly Heat) 痱子 22
3. Burn 23
Chapter 6. Dermatitis due to allergy 26
1. Eczema 湿疹 26
2. Urticaria 荨麻疹 27
3. Contact dermatitis 接触性皮炎 29
4. Neurodermatitis (lichen simplex) 神经性皮炎 31
5. Drug Rashes 药疹 32
Chapter 7. Dermatitis with erythema and squame 34
1. Psoriasis 牛皮癣,银屑病 34
Chapter 8. Connective tissue diseases 34
1. Discoid Lupus Erythematosus 34
2. Scleroderma 36
Appendix 39

Chapter 1. Body and skin beauty

1. Obesity



Obesity is a term used to describe body weight that is much greater than what is considered healthy. There are many ways to determine if a person is obese, but experts believe that a person's **body mass index (BMI)** is the most accurate measurement of body fat for children and adults.

The body mass index (BMI is a statistical measurement which compares a person's weight and height. Though it does not actually measure the percentage of body fat, it is a useful tool to estimate a healthy body weight based on how tall a person is.

A person with a BMI of 18.5 to 24.9 is considered to be at a healthy weight. A person with a BMI of 25-29.9 is considered to be overweight. A BMI over 30 is considered obese. A BMI of 40 or above indicates that a person is morbidly obese. This can increases a person's risk of death from any cause by 50%-150%. There are exceptions. For example, an athlete may have a higher BMI but not be overweight.

Body Mass Index (BMI), Kg/M 2

Weight (pounds)	Height (feet, inches)					
	5'0"	5'3"	5'6"	5'9"	6'0"	6'3"
140	**27**	**25**	23	21	19	18
150	**29**	**27**	24	22	20	19
160	**31**	**28**	**26**	24	22	20
170	**33**	**30**	**28**	**25**	23	21
180	**35**	**32**	**29**	**27**	**25**	23
190	**37**	**34**	**31**	**28**	**26**	24

200	**39**	**36**	**32**	**30**	**27**	**25**
210	**41**	**37**	**34**	**31**	**29**	**26**
220	**43**	**39**	**36**	**33**	**30**	**28**
230	**45**	**41**	**37**	**34**	**31**	**29**
240	**47**	**43**	**39**	**36**	**33**	**30**
250	**49**	**44**	**40**	**37**	**34**	**31**

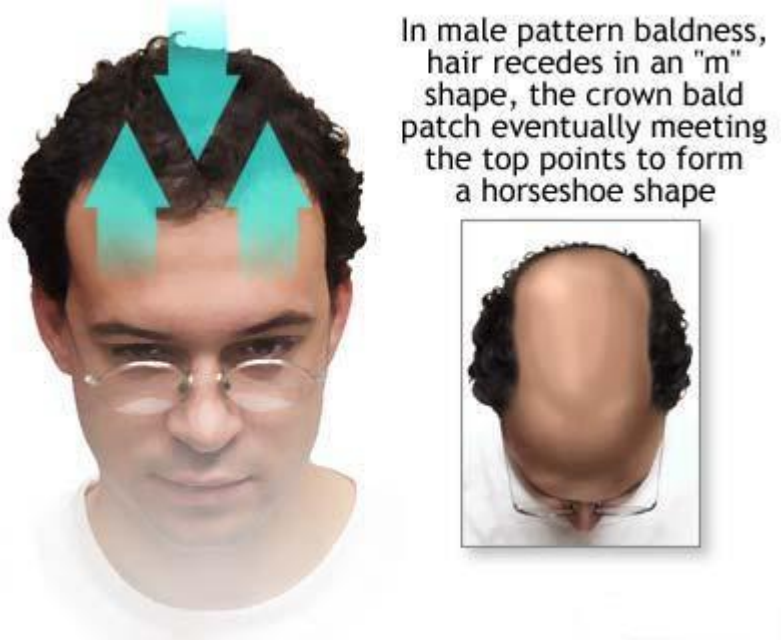
Nearly two-thirds of the United States population is overweight. Anyone more than 100 pounds overweight is considered morbidly obese.

Rates of obesity are climbing. The percentage of children who are overweight has doubled in the last 20 years. The percentage of adolescents who are obese has tripled in the last 20 years.

Obesity increases a person's risk of illness and death due to diabetes, stroke, heart disease, high blood pressure, high cholesterol, and kidney and gallbladder disease. Obesity may increase the risk for some types of cancer. It is also a risk factor for the development of osteoarthritis and sleep apnea.

Genetic factors play some part in the development of obesity -- children of obese parents are 10 times more likely to be obese than children with parents of normal weight.

2. Androgenetic Alopecia (Male pattern baldness)



On average, **there are 100,000 to 150,000 hairs on the human scalp**. The hairs grow from hair roots, **follicles**. Blood vessels at the base of each follicle provide the nourishment necessary for hair growth. Hair growth in each root occurs in a cycle independent of the other roots. At any time about 90 percent of the hairs on the scalp are in the growth phase, while the other 10 percent are in the resting phase. The growth phase lasts an average of four to five years, after which the follicle enters the resting phase, which lasts about two months to four months. At the end of the resting phase, the hair falls out naturally and is replaced by a new hair. Consequently, some hair loss is a normal part of the hair growth cycle. **In fact, on a typical day, about 50 to 150 scalp hairs are lost.** Baldness (or alopecia) results when hair loss occurs at an abnormally high rate; when hair replacement occurs at an abnormally slow rate; or when normal hairs are replaced by thinner, shorter ones.

About 95 percent of all cases of hair loss are the result of androgenetic alopecia (also known as male pattern baldness in men). Androgenetic alopecia occurs much more frequently in men than in women. It affects roughly 40 million men in the United States. Approximately 25 percent of men begin balding by age 30; two-thirds begin balding by age 60.

While some types of hair loss are easily reversible, male pattern baldness is more permanent. It occurs in a characteristic pattern on the scalp: hair loss usually begins at the temples and at the top of the head toward the back, causing a receding hairline and a bald spot. Hair loss may continue until the two sections become joined, leaving a horseshoe-shaped area of hair on the sides and back of the head. Balding may begin at any age after puberty, even in the middle teens, and can range from partial loss to complete baldness. Male pattern baldness progresses slowly and is not associated with redness, itching, or pain. Currently, there is no way to prevent male pattern baldness from occurring.

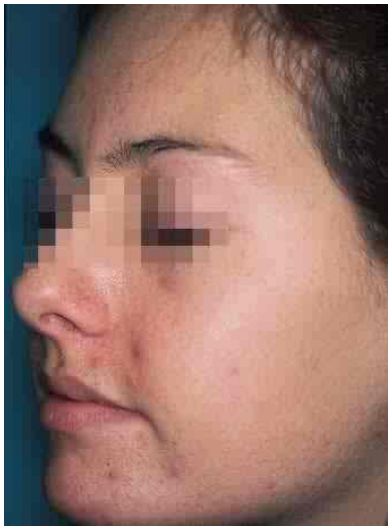
The causes of male pattern baldness are thought to be complex and are not completely understood. However, as suggested by its medical name (androgenetic alopecia), **male pattern baldness seems to involve both hormonal (androgen) and genetic factors.** Many different types of hormones play roles in the regulation of scalp hair, but the hormones with the largest effect are the androgens. **Testosterone and its more potent derivative dihydrotestosterone (DHT) are responsible for increasing the size of hair follicles in areas such as the beard and underarm during puberty, but can also cause hair follicles in the scalp to decrease in size later in life.** These contrasting responses to DHT might be explained by genetic differences in the individual hair follicles. Similarly, in men who are balding, genetically determined characteristics of hair follicles in the scalp may cause the follicles to be more likely to degrade in the presence of androgens. Hair follicles become miniaturized, and the hair consequently becomes thinner and shorter. The growing phase of the growth cycle becomes progressively shorter, which means more hairs are shed. Although the follicles still have an adequate blood supply, they continue to shrink, and some eventually die.

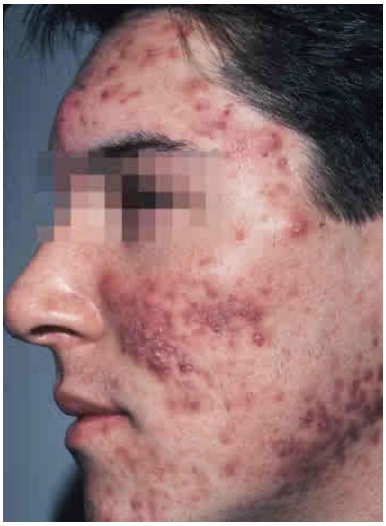
Whatever the exact causes of male pattern baldness may be, it is a hereditary trait. There are multiple genetic factors that influence male pattern baldness. A tendency toward baldness in the men on either the mother's or father's side of a man's family indicates a genetic predisposition to baldness. The speed, pattern, time of onset and degree of balding are all influenced by heredity. Generally, the earlier the onset of balding, the more extensive the degree of hair loss will eventually be.

Aside from male pattern baldness, some of the most common types of hair loss include:

- **Alopecia areata**, which leads to patchy hair loss on the scalp. While the causes of alopecia areata are not completely understood, the hair loss is thought to be the result of the body's immune system attacking the hair follicle. Alopecia areata often resolves without treatment.
- **Traction alopecia**, which results from long-term pulling on the hair. This type of hair loss can be caused by certain hairstyles, such as **tight braids**. The hair loss is usually reversible once the cause of this pulling is eliminated.

3. Acne 痤疮





A common inflammatory disease of the pilosebaceous glands characterized by comedones, papules, pustules, inflamed nodules, superficial pus-filled cysts, and (in extreme cases) canalizing and deep, inflamed, sometimes purulent sacs.

Pathogenesis

An interaction among hormones, keratin, sebum, and bacteria determines the course and severity. Acne usually begins at puberty, when an increase in androgens causes an increase in the size and activity of pilosebaceous glands. Inflammatory acne lesions include papules, pustules, and nodules or cysts. Noninflammatory lesions include open and closed comedones (ie, blackheads and whiteheads). First, intrafollicular hyperkeratosis leads to blockage of the pilosebaceous follicle; consequently, comedones form, composed of sebum, keratin, and microorganisms, particularly *Propionibacterium acnes.* Lipases from *P. acnes* break down triglycerides in the sebum to free fatty acids (FFA), which irritate the follicular wall. Retention of sebaceous secretions and dilation of the follicle may lead to cyst formation. Rupture of the follicle, with release into the tissues of FFA, bacterial products, and keratin, induces an inflammatory reaction that usually results in an abscess. These abscesses heal, with scarring in severe cases. Acne usually spontaneously remits, but the time of remittence cannot be predicted.

Symptoms and Signs

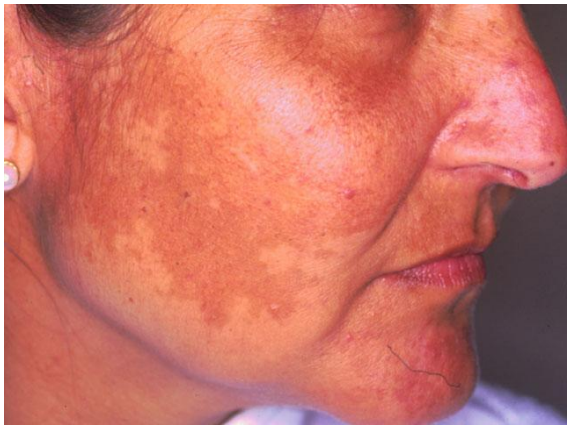
Acne is often worse in winter and improved in summer, probably because of the benefits of sunlight. Diet has little effect; however, if a food is suspected, it should be omitted for several weeks and then eaten in substantial quantities to determine if acne worsens. Acne may cycle with

the menses, and it may improve or worsen during pregnancy. Although cosmetics rarely aggravate acne, the traditional advice to avoid greasy preparations seems prudent.

Superficial acne: Blackheads (open comedones) or whiteheads (closed comedones), inflamed papules, pustules, and superficial cysts are characteristic. Large cysts occur occasionally, sometimes after manipulation or trauma to an otherwise uninflamed blackhead. The prognosis for healing without scars is good in superficial acne, but attempts to extrude blackheads or superficial cysts and scratching of ruptured lesions may increase scarring.

Deep acne: This form is characterized by the above findings with deep inflamed nodules and pus-filled cysts, which often rupture and become abscesses. Some of the abscesses open on the skin surface and discharge their contents. Lesions are most common on the face, but the neck, chest, upper back, and shoulders may also be affected. Scarring is frequent.

4. Melasma/Chloasma 黄褐斑



Chloasma, also known as melasma, appears as a blotchy, brownish pigmentation on the face that develops slowly and fades with time.

Chloasma usually affects women but occasionally is seen in young men who use after-shave lotions, scented soaps, and other toiletries.

Chloasma is especially common in women aged 20-40. It affects the forehead, cheeks and upper lips. It occurs frequently during pregnancy and is more common in dark skins than in fair skins. Often called "the mask of pregnancy", chloasma is more pronounced during the summer months as a result of sun exposure. It usually fades a few months after delivery. Repeated pregnancies, however, can intensify the pigmentation.

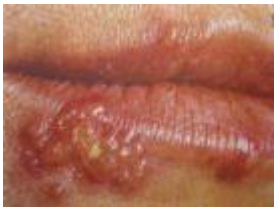
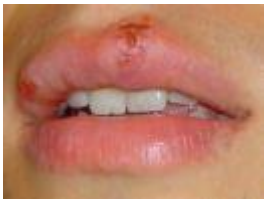
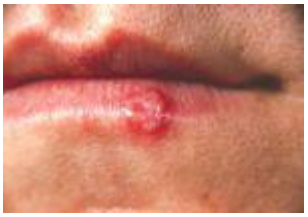
Chloasma also occurs as a side-effect of taking contraceptive pills and injected depot contraceptive preparations. It may also be noticed in apparently healthy, normal, non-pregnant women where it is presumed to be due to some mild and harmless hormonal imbalance.

Sun exposure, following the use of deodorant soaps, scented toiletries, and various cosmetics can also produce this mottled pigmentation. This is called a phototoxic reaction and is due to ultraviolet radiation being absorbed by the chemical substance (perfume, cologne and other types of fragrance) on the skin. This pigmentation often extends down to the sun-exposed areas of the neck and may be

more pronounced on the right side of the forehead, face and neck due to sun exposure while driving a car (or the left side - if you drive on the right).

Chapter 2. Viral skin diseases

1. Herpes simplex

Definition

Herpes simplex is an infection that primarily affects the mouth or genital area.

Causes, incidence, and risk factors

There are two different strains of herpes simplex viruses:

- **Herpes simplex virus type 1 (HSV-1)** is usually associated with infections of the lips, mouth, and face. It is the most common herpes simplex virus and is usually acquired in childhood. HSV-1 often causes lesions inside the mouth such as cold sores (fever blisters) and is transmitted by contact with infected saliva. By adulthood, up to 90% of individuals will have antibodies to HSV-1.
- **Herpes simplex virus 2 (HSV-2)** is sexually transmitted. Symptoms include genital ulcers or sores. In addition to oral and genital lesions, the virus can also lead to complications such as meningoencephalitis (infection of the lining of the brain and the brain itself) or cause infection of the eye -- in particular the conjunctiva, and cornea. However, some people have HSV-2 but do not display symptoms. Up to 30% of U.S. adults have antibodies against HSV-2. Cross-infection of type 1 and 2 viruses may occur from oral-genital contact.

A finger infection, called **herpetic whitlow**, is another form of herpes infection. It usually affects health care providers who are exposed to oral secretions during procedures. Sometimes, young children contract the disease.

A herpes virus can infect the fetus and cause congenital abnormalities. It may also be transmitted to a newborn during vaginal delivery in mothers infected with herpes viruses, particularly if the mother has active infection at the time.

However, the virus may be transmitted even in the absence of symptoms or visible lesions.

Symptoms

- mouth sores
- genital lesions (male) -- may be preceded by burning or tingling sensation

- genital lesions (female) -- may be preceded by burning or tingling sensation
- blisters or ulcers -- most frequent on the mouth, lips and gums or genitalia
- fever blisters
- fever -- may be present especially during the first episode
- enlargement of lymph nodes in the neck or groin

Signs and tests

In many instances, the physical appearance of the lesions is highly suggestive of the diagnosis of herpes-simplex infections. However, certain tests may be ordered to establish a definitive diagnosis. They include:

- Blood test
- Viral culture of lesion
- Tzanck test
- Direct fluorescent antibody (DFA) test

Treatment

Some cases are relatively mild and may not require treatment.

In severe or prolonged cases, or in individuals who are immunosuppressed or who have frequent recurrences, antiviral medications such as acyclovir may be used.

In individuals with more than 6 recurrences of genital herpes per year, chronic antiviral medications may be offered to reduce recurrences.

Expectations (prognosis)

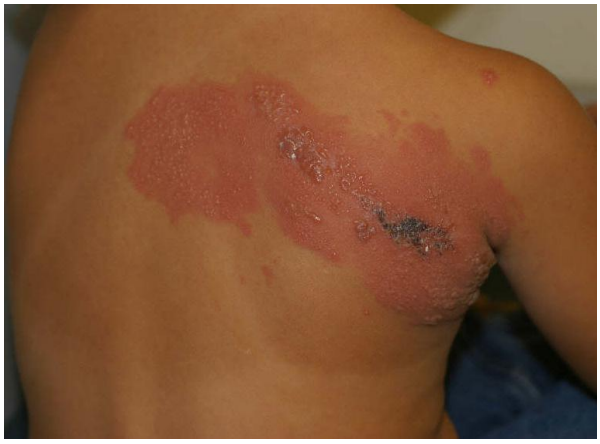
The oral or genital lesions usually heal on their own in 7 to 10 days unless an individual has an underlying condition that weakens the immune system, in which case the infection may be more severe and last longer.

Once infected, the virus spreads to nerve cells and stays in the body for the rest of a person's life . It may intermittently reactivate and cause symptoms, or flares. Recurrences may be precipitated by overexposure to sunlight, fever, stress, acute illness, and medications or conditions that weaken the immune system (such as cancer, HIV/AIDS, or use of corticosteroids).

Complications

- meningitis
- encephalitis
- eczema herpetiform (widespread herpes across the skin)
- infection of the eye -- keratoconjunctivitis
- prolonged, severe infection in immunosuppressed individuals
- pneumonia
- infection of the trachea

2. Herpes zoster



Alternative names

Shingles

Definition

Herpes zoster is an acute, localized infection with varicella-zoster virus, which causes a painful, blistering rash.

Causes, incidence, and risk factors

Herpes zoster, or shingles, is caused by the same virus that causes **chickenpox**. After an episode of chickenpox, the virus becomes dormant in the body. Herpes zoster occurs as a result of the virus re-emerging after many years.

The cause of the re-activation is usually unknown, but seems to be linked to aging, stress, or an impaired immune system. Often only one attack occurs, without recurrence.

If an adult or child is exposed to the herpes zoster virus and has not had chickenpox as a child or received the chickenpox vaccine, a severe case of chickenpox may develop, rather than shingles.

After infection with chickenpox, the virus resides in a non-active state in the nerve tracts that emerge from the spine. When it is re-activated, it spreads along the nerve tract, first causing pain or a burning sensation.

The typical rash appears in 2 to 3 days, after the virus has reached the skin. It consists of red patches of skin with small blisters (vesicles) that look very similar to early chickenpox. The rash often increases over the next 3 to 5 days. Then, the blisters break, forming small ulcers that begin to dry and form crusts. The crusts fall off in 2 to 3 weeks, leaving behind pink healing skin.

Lesions typically appear along a single dermatome (the body area served by a single spinal nerve) and are only on one side of the body (unilateral). The trunk is most often

affected, showing a rectangular belt of rash from the spine around one side of the chest to the breastbone (sternum).

Lesions may also occur on the neck or face, particularly along the trigeminal nerve in the face. The trigeminal has three branches that go to the forehead, the mid-face, and the lower face. Which branch is involved determines where on the face the skin lesions will be.

Trigeminal nerve involvement may include lesions in the mouth or eye. Eye lesions may lead to permanent blindness if not treated with emergency medical care.

Involvement of the facial nerve may cause Ramsay Hunt syndrome with facial paralysis, hearing loss, loss of taste in half of the tongue and skin lesions around the ear and ear canal. Shingles may, on occasion, involve the genitals or upper leg.

Shingles may be complicated by a condition known as post-herpetic neuralgia. This is persistence of pain in the area where the shingles occurred that may last from months to years following the initial episode. This pain can be severe enough to be incapacitating. The elderly are at higher risk for this complication.

Herpes zoster can be contagious through direct contact in an individual who has not had chickenpox, and therefore has no immunity. Herpes zoster may affect any age group, but it is much more common in adults over 60 years old, in children who had chickenpox before the age of one year, and in individuals whose immune system is weakened. The disorder is common, with about 600,000 to one million cases in the U.S. per year.

Most commonly, an outbreak of shingles is localized and involves only one dermatome. Widespread or recurrent shingles may indicate an underlying problem with the immune system such as leukemia, Hodgkin's disease and other cancers, atopic dermatitis, HIV infection, or AIDS. People with suppressed immune systems due to organ transplant or treatment for cancer are also at risk.

Symptoms

- Warning symptoms of unilateral (on one side) pain, tingling, or burning sensation limited to a specific part of the body -- pain and burning sensation may be intense
- Reddening of the skin (erythema) followed by the appearance of blisters (vesicles)
- Grouped, dense, deep, small blisters that ooze and crust

Additional symptoms that may be associated with this disease:

- Fever, chills
- General feeling of malaise
- Headache
- Lymph node swelling

- Vision abnormalities
- Taste abnormalities
- Drooping eyelid (ptosis)
- Loss of eye motion (ophthalmoplegia)
- Hearing loss
- Joint pain
- Genital lesions (female or male)
- Abdominal pain

Signs and tests

Diagnosis is suspected based on the appearance of the skin lesions, and strengthened by a prior history of chickenpox or shingles. It can be confused with herpes simplex.

Tests are rarely necessary, but may include:

- Viral culture of skin lesion
- Tzanck test of skin lesion
- Complete blood count (CBC) may show elevated white blood cells, a nonspecific sign of infection
- Specific antibody (immunoglobulin) measurement demonstrates elevation of varicella antibodies

Treatment

Herpes zoster usually disappears on its own, and may not require treatment except for symptom relief, such as pain medication.

Acyclovir is an antiviral medication that may be prescribed to shorten the course, reduce pain, reduce complications, or protect an immunocompromised individual. Desciclovir, famciclovir, valacyclovir, and penciclovir are similar to acyclovir and may be used to treat herpes zoster.

For the greatest effect, treatment with acyclovir-like medications should start within 24 hours of the appearance of pain or burning sensation, and preferably before the appearance of the characteristic blisters.

Typically, the drugs are given as pills, in doses four times greater than those recommended for herpes simplex or genital herpes. Severely immunocompromised individuals may require intravenous (IV) acyclovir therapy.

Corticosteroids, such as prednisone, may occasionally be used to reduce inflammation and risk of post-herpetic neuralgia. They have been shown to be most effective in the elderly population. Corticosteroids have certain risks that should be considered before using them.

Pain medicines (analgesics), mild to strong, may be needed to control pain. Antihistamines may be used topically (direct application to the body) or orally (by mouth) to reduce itching. Zostrix, a cream containing capsaicin (an extract of pepper), may prevent post-herpetic neuralgia.

Cool wet compresses can be used to reduce pain. Soothing baths and lotions, such as colloidal oatmeal bath, starch baths, or lotions and calamine lotion, may help to relieve itching and discomfort. Resting in bed until the fever goes down is recommended.

The skin should be kept clean, and contaminated items should not be re-used. Non-disposable items should be washed in boiling water or otherwise disinfected before re-use. The person may need to be isolated while lesions are oozing to prevent infection of others -- especially pregnant women.

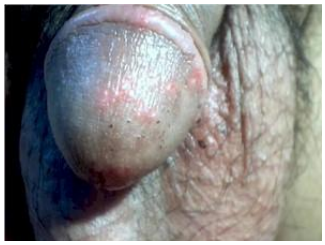
Expectations (prognosis)

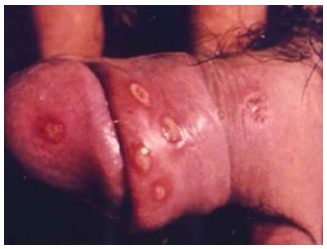
Herpes zoster usually clears in 2 to 3 weeks and rarely recurs. Involvement of motor nerves (nerves that control movement) may cause temporary or permanent nerve palsy (weakness or paralysis). Neuralgia (continued nerve pain) may persist for years in 50% of those over 60 years old who have shingles, particularly if the trigeminal nerve was affected. Eye lesions may lead to permanent blindness and require emergency medical care.

Complications

- Post herpetic neuralgia
- Secondary bacterial skin infections
- Recurrence (rare)
- Generalized infection, visceral organ lesions, encephalitis or sepsis in immunosuppressed persons
- Blindness (if lesions occur in the eye)
- Deafness
- Loss of taste
- Facial paralysis

3. **Genital herpes** 生殖器疱疹





What is genital herpes?

Genital herpes is an infection caused by the **herpes simplex virus** (HSV). There are two types of HSV, and both can cause genital herpes. HSV type 1 most commonly infects the lips, causing sores known as fever blisters or cold sores, but it also can infect the genital area and produce sores. HSV type 2 is the usual cause of genital herpes, but it also can infect the mouth. A person who has genital herpes infection can easily pass or transmit the virus to an uninfected person during sex.

Both HSV 1 and 2 can produce sores (also called lesions) in and around the vaginal area, on the penis, around the anal opening, and on the buttocks or thighs. Occasionally, sores also appear on other parts of the body where the virus has entered through broken skin.

HSV remains in certain nerve cells of the body for life, and can produce symptoms off and on in some infected people.

According to the U.S. Centers for Disease Control and Prevention, 45 million people in the United States ages 12 and older, or 1 out of 5 of the total adolescent and adult population, are infected with HSV-2.

Nationwide, since the late 1970s, the number of people with genital herpes infection has increased 30 percent. The largest increase is occurring in young teens. HSV-2 infection is more common in three of the youngest age groups which include people aged 12 to 39 years.

How does someone get genital herpes?
Most people get genital herpes by having sex with someone who is having a herpes "outbreak." This outbreak means that HSV is active. When active, the virus usually causes visible lesions in the genital area. The lesions shed (cast off) viruses that can infect another person. Sometimes, however, a person can have an outbreak and have no visible sores at all. People often get genital herpes by having sexual contact with others who don't know they are infected or who are having outbreaks of herpes without any sores.

A person with genital herpes also can infect a sexual partner during oral sex. The virus is spread only rarely, if at all, by touching objects such as a toilet seat or hot tub.

What are the symptoms?
Unfortunately, most people who have genital herpes don't know it because they never have any symptoms, or they do not recognize any symptoms they might have. When there are symptoms, they can be different in each person. Most often, when a person becomes infected with herpes for the first time, the symptoms will appear within 2 to 10 days. These first episodes of symptoms usually last 2 to 3 weeks.

Early symptoms of a genital herpes outbreak include

- Itching or burning feeling in the genital or anal area
- Pain in the legs, buttocks, or genital area
- Discharge of fluid from the vagina
- Feeling of pressure in the abdomen

Within a few days, sores appear near where the virus has entered the body, such as on the mouth, penis, or vagina. They also can occur inside the vagina and on the cervix in women, or in the urinary passage of women and men. Small red bumps appear first, develop into blisters, and then

become painful open sores. Over several days, the sores become crusty and then heal without leaving a scar.

Other symptoms that may go with the first episode of genital herpes are fever, headache, muscle aches, painful or difficult urination, vaginal discharge, and swollen glands in the groin area.

Can outbreaks recur?
If you have been infected by HSV 1 and/or 2, you will probably have symptoms or outbreaks from time to time. After finished being active, the virus then travels to the nerves at the end of the spine where it stays for a while. Even after the lesions are gone, the virus stays inside the nerve cells in a still and hidden state, which means that it's inactive.

In most people, the virus can become active several times a year. This is called a recurrence. But scientists do not yet know why this happens. When it becomes active again, it travels along the nerves to the skin, where it makes more viruses near the site of the very first infection. That is where new sores usually will appear.

Sometimes, the virus can become active but not cause any sores that can be seen. At these times, small amounts of the virus may be shed at or near places of the first infection, in fluids from the mouth, penis, or vagina, or from barely noticeable sores. You may not notice this shedding because it often does not cause any pain or feel uncomfortable. Even though you might not be aware of the shedding, you still can infect a sex partner during this time.

After the first outbreak, any future outbreaks are usually mild and last only about a week. An infected person may know that an outbreak is about to happen by a tingling feeling or itching in the genital area, or pain in the buttocks or down the leg. For some people, these early symptoms can be the most painful and annoying part of an episode. Sometimes, only the tingling and itching are present and no visible sores develop. At other times, blisters appear that may be very small and barely noticeable, or they may break into open sores that crust over and then disappear.

The frequency and severity of recurrent episodes vary greatly. While some people have only one or two outbreaks in a lifetime, others may have several outbreaks a year. The number and pattern of repeat outbreaks often change over time for a person. Scientists do not know what causes the virus to become active again. Although some people with herpes report that their outbreaks are brought on by another illness, stress, or having a menstrual period, outbreaks often are not predictable. In some cases, outbreaks may be connected to exposure to sunlight.

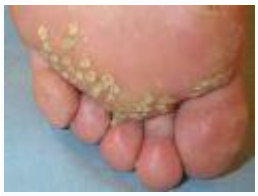
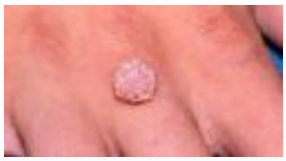
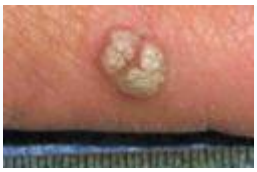
How is genital herpes diagnosed?
Because the genital herpes sores may not be visible to the naked eye, a doctor or other health care worker may have to do several laboratory tests to try to prove that symptoms are caused by the herpes virus. A person may still have genital herpes, however, even if the laboratory tests do not show the virus in the body.

A blood test cannot show whether a person can infect another with the herpes virus. A blood test, however, can show if a person has been infected at any time with HSV. There are also newer blood tests that can tell whether a person has been infected with HSV 1 and/or 2.

How can I protect myself or my sexual partner?

If you have early signs of a herpes outbreak or visible sores, you should not have sexual intercourse or oral sex until the signs are gone and/or the sores have healed completely. Between outbreaks, using male latex condoms during sexual intercourse may offer some protection from the virus. When used with these precautions, Valtrex can also help prevent infecting your partner during heterosexual sex.

4. Wart 疣

Warts are simply areas of skin that grow faster than normal due to the presence of the **wart virus**. Warts are skin-colored and feel rough to the touch. The technical name is verruca vulgaris. They are most common on the hands, feet and face but they can grow almost anywhere in the body. They are infectious and some people, especially children, are more susceptible than others.

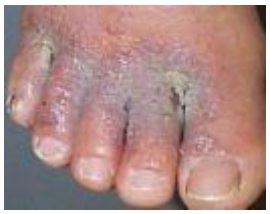
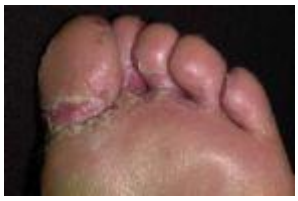
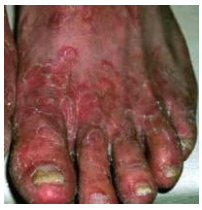
Flat warts are much smaller and are less rough than hand or foot warts. They tend to grow in great numbers -- 20 to 100 at any one time. They can occur anywhere, but in children they are most common on the face. In adults they are most often found in the beard area in men and on the legs in women. Skin irritation from shaving probably accounts for this.

A plantar wart is simply a wart growing on the weight-bearing surface of the foot that grows inward rather than outwards because it is pressed on when a person walks.

As warts are caused by a virus infection, the body will build up resistance over a period of time and eventually the body will cause the warts to disappear. This may take months or sometimes years but is the natural way the body deals with warts. If they are allowed to disappear in this way it is less likely that a person will get any further ones as one will then be immune to that virus.

Chapter 3. Fungi skin diseases

1. Tinea pedis 足癣 (Athlete's foot, Ringworm of feet)

Athlete's foot is a fungal infection of the foot caused by parasites on the skin called dermatophytes. Dermatophytes can be divided into three groups according to their favourite hosts:

- fungi preferring soil (geophile)
- fungi preferring animals (zoophile)
- and fungi preferring humans (anthropophile).

Athlete's foot is usually caused by anthropophile fungi. The most common species are Microsporum, Epidermophyton and Trichophyton. These account for 90 per cent of all skin fungal infections, commonly referred to as ringworm.

The medical terms for athlete's foot are tinea pedis or dermatophytosis palmaris, plantaris and interdigitalis - the latter indicates that, in addition to the soles and toes of the feet, the palms of the hands can also become infected.

What causes it?

We all have one or more of the fungi that can cause athlete's foot on our bodies. They feed on dead skin cells and are usually harmless.

Athlete's foot is a common condition in young people and adults. The fungi love warm, moist places with the result they are primarily a problem for people who wear tight-fitting trainers or don't dry their feet properly.

The condition is contagious. It can be spread by direct skin-to-skin contact and indirectly through towels, shoes, floors, etc.

What are the symptoms?

There are two variants of the condition.

Classic cases

The infection is caused by one of the most common fungi.

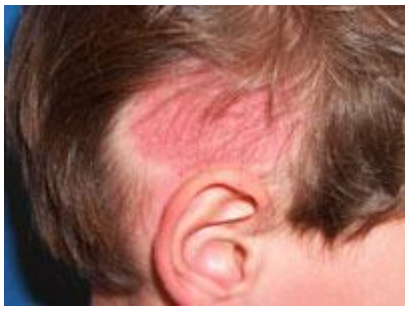
- A red itchy rash in the spaces between the toes (often between the 4th and 5th toes initially) and possibly small pustules.
- Often a small degree of scaling.
- The infection can spread to the rest of the foot and other parts of the body.

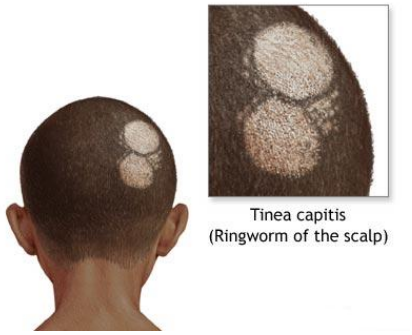
Rarer cases

Infection of the soles of both feet by Trichophyton rubrum.

- The skin reddens and its furrows become marked, resembling chalked lines.
- If the condition is not treated, a similar rash may appear on one or both palms.
- After a while, the rash becomes scaly, resembling eczema.

2. Tinea capitis 头癣





Tinea capitis is an infection of the scalp by mold-like fungi called dermatophytes.

Causes, incidence, and risk factors

The body normally hosts a variety of microorganisms, including bacteria, mold-like fungi (dermatophytes) and yeast-like fungi (such as Candida). Some of these are useful to the body. Others may multiply rapidly and cause symptoms.

Tinea capitis (also called ringworm of the scalp) is a skin disorder that affects children almost exclusively. It can be persistent and contagious, almost to the point of epidemic; however, it often disappears spontaneously at puberty.

The fungi that cause tinea infections thrive in warm, moist areas. Susceptibility to tinea infection is increased by poor hygiene, prolonged wetness of the skin (such as from sweating), and minor skin or scalp injuries.

Tinea infections are contagious and may be passed by direct contact with affected individuals or by contaminated items such as combs, hats, clothing, or similar surfaces. They can be transmitted by contact with pets that carry the fungus, for instance, cats.

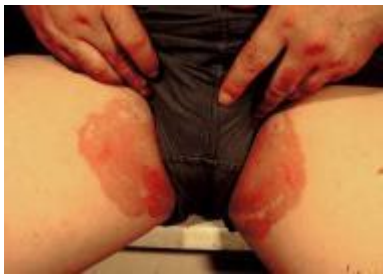
Symptoms

- Itching of the scalp, may be slight or absent
- Skin (scalp) lesions that are:
    - Round, scaly
    - Gray or reddened (skin redness or inflammation)
    - Bald-appearing patches (hair is broken off, not actually missing)
    - Possibly small black dots on the scalp
- Occasionally localized area of swelling, raw skin, or pus-filled lesion on the scalp (kerion)

Signs and tests

The diagnosis is suspected primarily based on the appearance of the scalp. A skin lesion biopsy with microscopic examination or culture may show dermatophytes. This test is often not necessary to diagnose tinea capitis. A Wood's lamp test may be performed to confirm the presence of a fungal scalp infection.

3. Tinea cruris 股癬



Jock itch, also called tinea cruris or ringworm of the groin, is an infection of the groin area caused by fungi.

Causes, incidence, and risk factors

The body normally hosts a variety of bacteria and fungi. Some of these are useful to the body. Others can multiply rapidly and form infections. Jock itch occurs when a particular type of fungus grows and multiplies in the groin area.

Jock itch occurs almost exclusively in adult men. It can sometimes accompany athlete's foot and ringworm. The fungus that causes jock itch thrives in warm, moist areas. Jock itch can be triggered by friction from clothes and prolonged wetness in the groin area (such as from sweating).

Jock itch may be contagious. It can be passed from one person to the next by direct skin-to-skin contact or contact with unwashed clothing. Jock itch usually stays around the creases in the upper thigh and does not involve the scrotum or penis. It is often less severe than other tinea infections, but may last a long time. Jock itch may spread to the anus, causing anal itching and discomfort.

Other causes of itching in the groin include:

- Lichen simplex chronicus
- Eczema
- Pubic lice
- Chemical irritation

For itching of the groin in women, see vaginal itching.

Symptoms

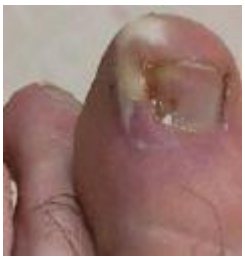
- Itching in groin, thigh skin folds, or anus.
- Red, raised, scaly patches that may blister and ooze. The patches often have sharply-defined edges. They are often redder around the outside with normal skin tone in the center. This may create the appearance of a ring.
- Abnormally dark or light skin.

Signs and tests

- Your doctor will usually diagnose jock itch based on the appearance of the skin. Tests are usually not necessary. If tests are needed to confirm the diagnosis, either a culture or a skin lesion biopsy

Chapter 4. Bacterial skin diseases

1, Paronychia 甲沟炎



Paronychia is a superficial infection of the skin around the nails, most commonly caused by staphylococcus bacteria or fungi.

Causes, incidence, and risk factors

Paronychia is a fairly common superficial infection of the skin around the nail. It usually results from injury to the area from biting off or picking a hangnail, or from manipulating, trimming, or pushing back the cuticle.

Disorders include:

- Bacterial paronychia (caused by bacteria)
- Candidal paronychia (caused by a specific type of yeast)
- Fungal paronychia (caused by a fungus other than Candida)

Paronychia may include a combined bacterial and fungal infection. Fungal paronychia may accompany fungal nail infection.

Fungal paronychia is common among people with diabetes and among people who have their hands in water for long periods of time.

Symptoms

- Skin lesion located in the skin around the nail, often at the cuticle or at the site of a hangnail or other injury
- Painful
- Onset sudden (bacterial) or gradual (fungal, mixed infection)
- May persist (fungal, mixed infection)
- May be acute or chronic
- Localized redness
- Localized swelling
- Pus-filled blisters (especially with bacterial infection)
- Swelling of the finger or the cuticle
- Nail changes
- Discoloration
- Distorted shape

- Detached

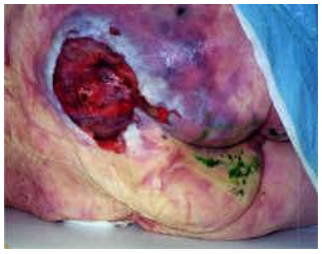
Signs and tests

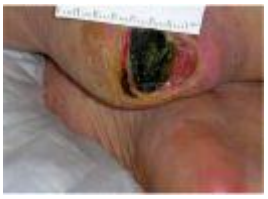
The health care provider primarily bases the diagnosis on the appearance of the skin lesion.

Aspiration and culture of pus or fluid from the area may reveal the organism causing the infection.

Chapter 5. Skin diseases due to physical factors

1. Decubitus Ulcer /Bed sore / pressure sore 褥疮





Pressure sores (bedsores; decubitus ulcers; trophic ulcers): Ischemic necrosis and ulceration of tissues overlying a bony prominence that has been subjected to prolonged pressure against an external object (eg, bed, wheelchair, cast, splint).

Pressure sores occur most often in patients with diminished or absent sensation or who are debilitated, emaciated, paralyzed, or long bedridden. Tissues over the sacrum, ischia, greater trochanters, external malleoli, and heels are especially susceptible; other sites may be involved depending on the patient's position. Pressure sores can also affect muscle and bone.

Etiology

Intrinsic factors include loss of pain and pressure sensations (which ordinarily prompt the patient to shift position and relieve the pressure) and minimal fat and muscle padding between bony weight-bearing prominences and skin. Disuse atrophy, malnutrition, anemia, and infection contribute. In a paralyzed patient, loss of vasomotor control leads to lowered tone in the vascular bed and lowered circulatory rate. Spasticity, especially in patients with spinal cord injuries, can place a shearing force on the blood vessels to further compromise circulation.

Of **extrinsic factors,** the most important is pressure due to infrequent shifting of the patient's position; friction, irritation, and pulling of skin from ill-adjusted supports or wrinkled bedding or clothing contribute. The force and duration of pressure directly determine the extent of the ulcer. In an immobilized patient, severe pressure can impair local circulation in < 3 h, causing local tissue anoxia that, if unrelieved, progresses to necrosis of the skin and subcutaneous tissues. Moisture (eg, from perspiration or incontinence) leads to tissue maceration and predisposes to pressure sores.

The stages of pressure sore formation correspond to tissue layers

Stage	Characteristics
1	Nonblanchable erythema of intact skin
2	Partial-thickness epidermal or dermal loss, presenting superficially as an abrasion, blister, or shallow crater
3	Full-thickness damage or necrosis down to underlying fascia, presenting as a deep crater with or without undermining of adjacent tissue
4	Full-thickness destruction, necrosis, or damage to muscle, bone, or supporting structures; undermining, sinus tracts, osteomyelitis, or septic arthritis may occur

Prophylaxis

The best treatment is prevention by relief of pressure on sensitive areas.

In a bedridden patient, position must be changed at least q 2 h until tolerance for longer periods can be demonstrated (by the absence of redness). Air-filled alternating-pressure mattresses, sponge-rubber eggcrate mattresses, and silicone gel or water mattresses help decrease pressure on sensitive areas but do not negate the need for position changes q 2 h. When maximal relief of pressure is needed, other systems, including air flotation mattresses, must be used. A Stryker frame facilitates turning patients with spinal cord injuries. Protective padding (eg, sheepskin or a synthetic equivalent) at bony prominences should be used, especially under braces or plaster casts; a window should be cut out of the cast at potential pressure sites.

Wheelchair-bound patients may develop pressure sores. Thus, they must shift position or be shifted q 10 to 15 min, even if a pressure-relieving pillow is used.

Inspection under adequate light is important. Pressure points should be checked for erythema or trauma at least once/day. Patients and families must be taught a routine of daily visual inspection and palpation of sites for potential ulcer formation.

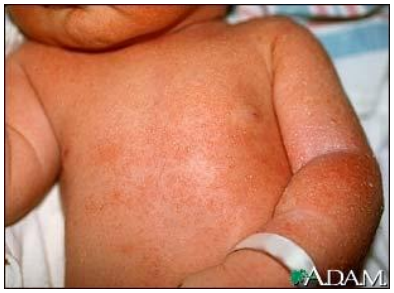
Meticulous care is necessary to prevent maceration and secondary infection. Lying on a sheepskin helps keep the patient's skin in good condition and minimize pressure sores. Protective padding, pillows, or a sheepskin can be used to separate body surfaces.

Maintaining **cleanliness and dryness** helps prevent maceration. Bedding and clothing should be changed frequently; sheets should be soft, clean, and free from wrinkles and particulate matter. Sponging the skin in hot weather and thorough drying after baths are essential. Special efforts are required for incontinent patients. Most areas may be powdered with plain talc.

Oversedation should be avoided and activity encouraged. Physiotherapy, when practicable, may be carried out by means of **passive and active exercises.** Hydrotherapy is also valuable.

A **well-balanced diet,** high in protein, is important. There is some evidence that supplemental vitamin C and zinc help healing.

2. Miliaria (Prickly Heat) 痱子



Miliaria is an itchy rash which arises from obstruction of the sweat ducts. Miliaria is commonest in hot, humid conditions but may occur in desert regions. It affects up to 30% of people exposed to these climatic conditions. It may begin within a few days of arrival in a tropical climate but is maximal after 2-5 months. There is a striking variation in individual susceptibility. Infants are especially prone.

Causes

Miliaria may be produced experimentally in susceptible subjects by injury to the epidermis (ie. surface layers of the skin). It can be reproduced regularly by occlusion of the skin under polythene for 3-4 days, following which the sweat ducts remain blocked for about 3 weeks. Prolonged exposure of the skin to sweat achieves the same effect. It is believed the first event in the production of miliaria is an increase in certain normal *Staphylococcus epidermidis* bacteria which live on the skin. These produce a sticky substance which blocks the sweat ducts. Leakage of sweat through the walls of the duct behind the block is responsible for production of the miliaria spots and for further aggravation.

Clinical Features

The typical spots develop in skin folds and on the body, especially in areas of friction from clothing. The lesions are minute red papules which may be present in very large numbers. There is characteristically intense discomfort; not so much itching as an unbearable pricking sensation. In infants lesions commonly appear on the neck, groins and armpits, but also on the face and elsewhere.

Once triggered off, an attack of miliaria commonly lasts 5-6 weeks despite the best treatment that can be offered. This is because the plugs which form in the sweat duct openings can only be cast off by the outward growth of the sweat duct cells. This takes several weeks. Treatment cannot influence this process.

In New Zealand, miliaria is most common during humid summer weather. Activities which encourage sweating and the wearing of synthetic clothing against the skin are important precipitating factors.

In winter, miliaria can result from swaddling up in too much clothing, sitting too close to the fire or heater and being hot in bed with a duvet and/or electric blanket.

In hospital, miliaria is typically seen on the backs of people who are lying for prolonged periods in bed, particularly when they are sweating from an infection or heart attack, or have been immobilized by a stroke, head injury or orthopaedic operation.

Tropical holidays such as visits to northern Australia and the Pacific islands, can result in miliaria.

3. Burn

In medicine, a **burn** is a type of injury to the skin caused by heat, electricity, chemicals, or radiation (an example of the latter is *sunburn*).

Classification

In classical medical literature, there were six degrees, the first three of which are still commonly used by the public:

- **First-degree burns** are usually limited to redness (erythema), a white plaque and minor pain at the site of injury. These burns usually extend only into the epidermis.
- **Second-degree burns** additionally fill with clear fluid, have superficial blistering of the skin, and can involve more or less pain depending on the level of nerve involvement. Second-degree burns involve the superficial (papillary) dermis and may also involve the deep (reticular) dermis layer.
- **Third-degree burns** additionally have charring of the skin, and produce hard, leather-like eschars. An eschar is a scab that has separated from the unaffected part of the body. Frequently, there is also purple fluid. These types of burns are often painless (insensate) because nerve endings have been destroyed in the involved areas.

A newer classification of "Superficial Thickness", "Partial Thickness" (which is divided into superficial and deep categories) and "Full Thickness" relates more precisely to the epidermis, dermis and subcutaneous layers of skin and is used to guide treatment and predict outcome.

Table 1. **A description of the traditional and current classifications of burns.**

Nomenclature	Traditional nomenclature	Depth	Clinical findings
Superficial thickness	First-degree	Epidermis involvement	Erythema, minor pain, lack of blisters
Partial	Second-degre	Superficial (papillary)	Blisters, clear fluid, and

thickness — superficial	e	dermis	pain
Partial thickness — deep	Second-degre e	Deep (reticular) dermis	Whiter appearance, with decreased pain. Difficult to distinguish from full thickness
Full thickness	Third- or fourth-degree	Dermis and underlying tissue and possibly fascia, bone, or muscle	Hard, leather-like eschar, purple fluid, no sensation (insensate)

Serious burns, especially if they cover large areas of the body, can cause death; any hint of burn injury to the lungs, for example through smoke inhalation, is a medical emergency.

Chemical burns are usually caused by chemical compounds, such as sodium hydroxide (lye), silver nitrate, and more serious compounds (such as sulfuric acid). Note that most chemicals (but not all) that can cause moderate to severe chemical burns are strong acids or bases. Nitric acid is possibly one of the worst burn-causing chemicals, as an oxidizer. Hydrofluoric acid can eat down to the bone and its burns are often not immediately evident. Most chemicals that can cause moderate to severe chemical burns are called caustic.

Electrical burns are generally symptoms of electrocution, being struck by lightning, being defibrillated or cardioverted without conductive gel, etc. The internal injuries sustained may be disproportionate to the size of the "burns" seen - as these are only the entry and exit wounds of the electrical current.

Survival and outcome (scars, contractures, complications) of severe burn injuries is remarkably improved if the patient is treated in a specialized burn center/unit rather than a hospital.

Scald

Scalding is a specific type of burning that is caused by hot fluids. Examples of common liquids that cause scalds are water and cooking oil. Steam is a common gas that causes scalds. The injury is usually regional and usually does not cause death. More damage can be caused if hot liquids can enter an orifice. However, deaths have occurred in more unusual circumstances, such as when people have accidentally broken a steam pipe.

Cold burn

A cold burn is a kind of burn which arises when the skin is in contact with a low-temperature body. They can be caused by prolonged contact with moderately cold bodies (snow for instance) or brief contact with very cold bodies such as dry ice, liquid helium or liquid nitrogen, which are used in the process of wart removal. In such a case, the heat transfers from the skin and organs to

the external cold body (as opposed to most other situations where the body causing the burn is hotter, and transfers the heat into the skin and organs). The effects are very similar to a "regular" burn. The remedy is also the same as for any burn: for a small wound keep the injured organ under a flow of comfortably temperatured water; the heat will then transfer slowly from the water to the organs and help the wound. Further treatment or treatment of more extended wound also as usual.

Assessing burns

Burns are assessed in terms of total body surface area (TBSA), which is the percentage affected by partial thickness or full thickness burns (superficial thickness burns are not counted). The rule of nines is used as a quick and useful way to estimate the affected TBSA.

***Table 2*. Rule of nines for assessment of total body surface area affected by a burn**

Anatomic structure	Surface area
Head	9%
Anterior Torso	18%
Posterior Torso	18%
Each Leg	18%
Each Arm	9%
Genitalia/perineum	1%

Management

The first step in managing a person with a burn is to stop the burning process. With dry powder burns, the powder should be brushed off first. With other burns, the affected area should be rinsed with a large amount of clean water to remove foreign bodies and help stop the burning process. Cold water should never be applied to any person with extensive burns, as it may severely compromise the burn victim's temperature status.

At this stage of management, it is also critical to assess airway status. If the patient was involved in a fire, then he or she has inhalation injury until proven otherwise, and should be managed accordingly.

Once the burning process has been stopped, and airway status is ensured, the patient should be volume resuscitated according to the Parkland formula. This formula dictates that the amount of Lactated Ringer's solution to deliver in the first twenty four hours after time of injury is:

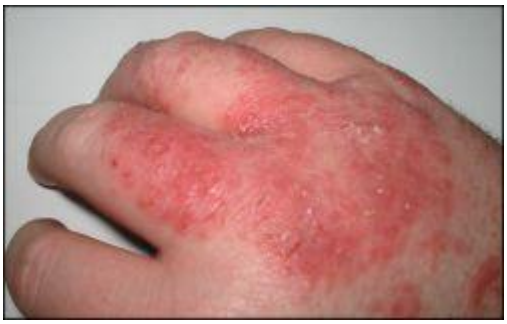
Fluid = 4cc *x* %TBSA *x* weight in kg
%TBSA excludes any first degree burn

Half of this fluid should be given in the first eight hours post injury and the rest in the subsequent sixteen hours. The formula is a guide only and infusions must be tailored to urine output and central venous pressure. Inadequate fluid resuscitation causes renal failure and death.

Chapter 6. Dermatitis due to allergy

1. Eczema 湿疹

Eczema is a general term encompassing various inflamed skin conditions. One of the most common forms of eczema is atopic dermatitis (or "atopic eczema"). Approximately 10 percent to 20 percent of the world population is affected by this chronic, relapsing, and very itchy rash at some point during childhood. Fortunately, many children with eczema find that the disease clears and often disappears with age.



In general, atopic dermatitis will come and go, often based on external factors. Although its cause is unknown, the condition appears to be an abnormal response of the body’s immune system. In people with eczema, the inflammatory response to irritating substances overacts, causing itching and scratching. Eczema is not contagious and, like many diseases, currently cannot be cured. However, for most patients the condition may be managed well with treatment and avoidance of triggers.

Although eczema may look different from person to person, it is most often characterized by dry, red, extremely itchy patches on the skin. Eczema is sometimes referred to as "the itch that rashes," since the itch, when scratched, results in the appearance of the rash.

Eczema can occur on just about any part of the body; however, in infants, eczema typically occurs on the forehead, cheeks, forearms, legs, scalp, and neck. In children and adults, eczema typically occurs on the face, neck, and the insides of the elbows, knees, and ankles. In some people, eczema may "bubble up" and ooze. In others, the condition may appear more scaly, dry, and red. Chronic scratching causes the skin to take on a leathery texture because the skin thickens (lichenification).

Many substances have been identified as itch "triggers" in patients with eczema, and triggers are not the same for every person. Many times it is difficult to identify the exact trigger that causes a flare-up. For some, it seems that rough or coarse materials coming into contact with the skin causes itchiness. For others, feeling too hot and/or sweating will cause an outbreak. Other people find that certain soaps, detergents, disinfectants, contact with juices from fresh fruits and meats, dust mites, and animal saliva and danders may trigger itching. Upper respiratory infections (caused by viruses) may also be triggers. Stress can also sometimes aggravate an existing flare-up.

Eczema occurs in both children and adults, but usually appears during infancy. Although there is no known cause for the disease, it often affects people with a family history of allergies.

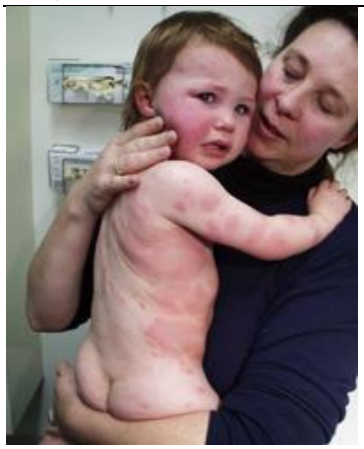
Those who are genetically predisposed and then exposed to environmental triggers may develop eczema. Many people who have eczema also suffer from allergic rhinitis and asthma, or have family members who do.

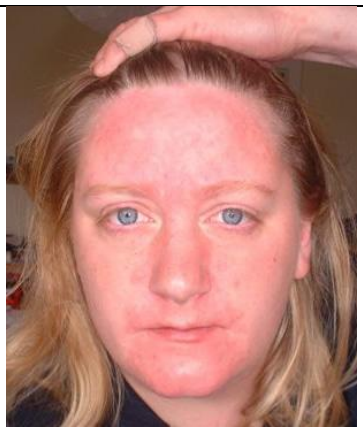
The National Institutes of Health estimates that 15 million people in the United States have some form of eczema. About 10 percent to 20 percent of all infants have eczema; however, in nearly half of these children, the disease will improve greatly by the time they are between five and 15 years of age. Others will have some form of the disease throughout their lives.

Eczema outbreaks can usually be avoided with some simple precautions. The following suggestions may help to reduce the severity and frequency of flare-ups:

- Moisturize frequently
- Avoid sudden changes in temperature or humidity
- Avoid sweating or overheating
- Reduce stress
- Avoid scratchy materials (e.g., wool or other irritants)
- Avoid harsh soaps, detergents, and solvents
- Avoid environmental factors that trigger allergies (e.g., pollens, molds, mites, and animal dander)
- Be aware of any foods that may cause an outbreak and avoid those foods

2. Urticaria 荨麻疹





Urticaria, commonly known as hives, usually strikes suddenly. First the skin itches, then it erupts into red welts. The itching may be severe, keeping people from working or sleeping. It's a distressing disorder which affects an estimated 20 percent of the population at one time or another in their lives.

Most cases of urticaria are acute, lasting from a few hours to less than six weeks. Some cases are chronic, lasting more than six weeks. The welts may appear in one place, disappear after a short time, then erupt at another spot, then another. They are made worse by scratching. Each individual hive lasts no more than 24 hours.

Bouts of urticaria have been traced to such triggers as infections, drugs (including aspirin), certain foods and additives, cold, sun exposure, insect stings, alcohol, exercise, endocrine disorders and emotional stress. In some people, pressure caused by belts and constricting clothing causes eruption. Urticaria may be a response to infection including the common cold, strep throat and infectious mononucleosis.

In the urticaria-prone person, these triggers cause the body to release chemical mediators, including histamine, from cells. Histamine (which causes itchy, runny noses and watery eyes in hay fever sufferers) dilates the walls of blood vessels, allowing fluids to leak out into the surrounding tissues. Swelling and itching are the result.

In some cases, the trigger is obvious - a person eats strawberries or shrimp, then develops urticaria within a short time. But because there are so many possible causes for urticaria, other cases require determined detective work on the part of the patient and physician. In some cases, the cause is never identified.

A single episode of uncomplicated acute urticaria probably does not need formal evaluation. Patients with recurrent episodes of acute urticaria, with chronic urticaria, or with urticaria complicated by swelling, trouble breathing or other potentially serious problems, an evaluation is recommended. See your regular physician first, in order to evaluate for non-allergic causes of urticaria. If allergy is suspected, keep a diary of foods eaten, any unusual exposures, and when you have hives. Bring the diary with you to the allergist's office. To unravel the urticaria puzzle, your allergist-immunologist will take a detailed history, looking for clues in your lifestyle that will help pinpoint the cause of your symptoms. You'll be asked about the frequency and severity of your symptoms, your family's medical history, medications you're taking, your work and home

environment, and miscellaneous matters. The allergist will want to review your diary for further clues.

In some cases you may require tests to analyze blood and urine, and other procedures such as x-rays. Skin testing may provide useful information in some cases. Your allergist-immunologist will decide which tests to order based on the different types of urticaria and the suspected cause.

Urticaria can be classified into two categories: allergic and non-allergic.

Allergic urticaria is the least common form, although it is somewhat more common in children than in adults. It is caused by the immune system's overreaction to foods, drugs, infection, insect stings, blood transfusions or other substances. Foods such as eggs, nuts and shellfish, and drugs such as penicillin and sulfa are common causes of allergic or immunologic urticaria. Recent studies also suggest that some cases of chronic urticaria are caused by autoimmune mechanisms, when the patient develops immune reactions to components of his or her skin.

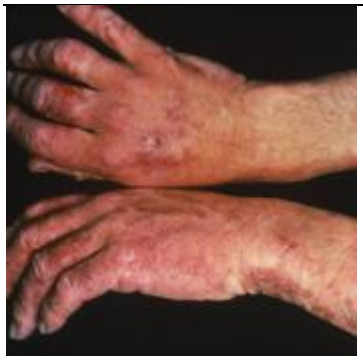
Non-allergic urticaria are those types of urticaria where a clear-cut allergic basis cannot be proven. These take many forms:

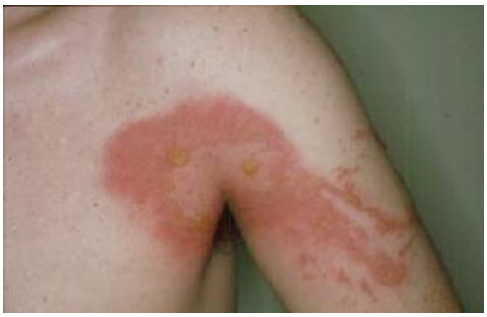
- Dermographism is urticaria that develops when the skin is stroked with a firm object.
- Cold-induced urticaria appears after a person is exposed to low temperatures for example, after a plunge into a swimming pool or when an ice cube is placed against the skin.
- Cholinergic urticaria, which is associated with exercise, hot showers and/or anxiety, is a form of hives that is related to release of certain chemicals from parts of the nervous system that controls such body functions as blood pressure and heart rate.
- Pressure urticaria develops from the constant pressure of constricting clothing such as sock bands, bra straps, belts or other tight clothing.
- Solar urticaria arises on parts of the body exposed to the sun; this may occur within a few minutes after exposure.

Some cases of non-allergic urticaria may be caused by reactions to aspirin and, possibly, certain food dyes, sulfites, and other food additives. In many cases, particularly in chronic urticaria, the trigger for the problem can't be found; in this instance it is called idiopathic urticaria.

Certain types of urticaria are more painful than itchy, may go away leaving a bruise on the skin, and individual hives may last more than 24 hours. In such cases, and selected other situations, a biopsy of the skin may be necessary for diagnosis.

3. Contact dermatitis 接触性皮炎





Contact dermatitis is an inflammation of the skin caused by direct contact with an irritating substance.

Causes, incidence, and risk factors

Contact dermatitis is an inflammation of the skin caused by direct contact with an irritating or allergy-causing substance (irritant or allergen) vary in the same individual over time. A history of any type of allergies increases the risk for this condition.

Irritant dermatitis, the most common type of contact dermatitis, involves inflammation resulting from contact with acids, alkaline materials such as soaps and detergents, solvents, or other chemicals. The reaction usually resembles a burn.

The second most common type of contact dermatitis is caused by exposure to a material to which the person has become hypersensitive or allergic. The skin inflammation varies from mild irritation and redness to open sores, depending on the type of irritant, the body part affected, and the sensitivity of the individual.

Over treatment dermatitis is a form of contact dermatitis that occurs when treatment for another skin disorder causes irritation.

Common allergens associated with contact dermatitis include:

- Poison ivy, poison oak, poison sumac
- Other plants
- Nickel or other metals
- Medications
    - Antibiotics, especially those applied to the surface of the skin (topical)
    - Topical anesthetics
    - Other medications
- Rubber
- Cosmetics
- Fabrics and clothing
- Detergents
- Solvents
- Adhesives
- Fragrances, perfumes
- Other chemicals and substances

Contact dermatitis may involve a reaction to a substance that the person is exposed to or uses repeatedly. Although there may be no initial reaction, repeated use (for example, nail polish remover, preservatives in contact lens solutions, or repeated contact with metals in earring posts and the metal backs of watches) can cause eventual sensitization and reaction to the product.

Some products cause a reaction only when they contact the skin and are exposed to sunlight (photosensitivity). These include shaving lotions, sunscreens, sulfa ointments, some perfumes, coal tar products, and oil from the skin of a lime. A few airborne allergens, such as ragweed or insecticide spray, can cause contact dermatitis.

Symptoms

- Itching (pruritus) of the skin in exposed areas
- Skin redness or inflammation in the exposed area
- Tenderness of the skin in the exposed area
- Localized swelling of the skin
- Warmth of the exposed area (may occur)
- Skin lesion or rash at the site of exposure
    - Lesions of any type: redness, rash, papules (pimple-like), vesicles, and bullae (blisters)
    - May involve oozing, draining, or crusting
    - May become scaly, raw, or thickened

Signs and tests

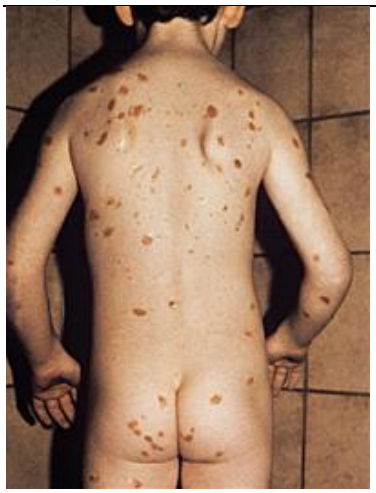
The diagnosis is primarily based on the skin appearance and a history of exposure to an irritant or an allergen.

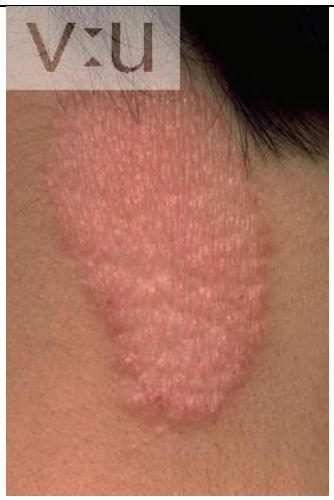
According to the American Academy of Allergy, Asthma, and Immunology, "Patch testing is the gold standard for contact allergen identification." Allergy testing with skin patches may isolate the suspected allergen that is causing the reaction.

Patch testing is used for patients who have chronic, recurring contact dermatitis. It requires three office visits and must be done by a clinician with detailed experience in the procedures and interpretation of results. Patients should bring suspected materials with them, especially if they have already tested those materials on a small area of their skin and noticed a reaction.

Other tests may be used to rule out other possible causes, including skin lesion biopsy or culture of the skin lesion.

4. Neurodermatitis (lichen simplex) 神经性皮炎





Neurodermatitis, also known as lichen simplex, is a chronic form of dermatitis. Dermatitis is a skin problem where inflammation causes the skin to become scaly and sometimes itchy. Chronic itching and scratching can cause the skin to thicken and have a leather texture. Neurodermatitis results in a constant scratch-itch cycle. Although signs increase in times of stress, changes in the nerve fibers are also present. Women more often than men have this skin problem. The problem happens most often between the ages of 20 and 50.

What causes neurodermatitis?

A scratch-itch cycle happens when a small itch causes scratching which increases the itch, leading to more scratching. The cause of the itch can be anything - an insect bite, tight clothing or dry skin. The skin then can become very irritated when scratched over and over. Stress and tension increase the itching. This results in the scratching process turning into a habit that we do without thinking. Things in your environment can also cause itching. There may be a family history of asthma, hay fever, eczema, psoriasis or other skin disorders. The back of the neck, arms, legs and ankles are the most common sites of an outbreak.

How is neurodermatitis diagnosed?

Neurodermatitis can be hard to tell from other forms of dermatitis. You should also see your doctor if you are losing sleep or your normal routine is upset because of the itching. Your doctor can also treat you if you have an infection, your skin is very painful or if you at-home treatment methods are not working.

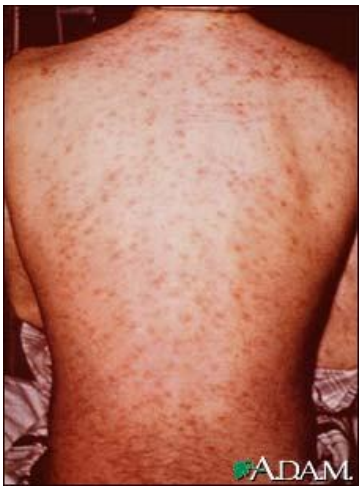
Diagnosis is based on the appearance of the skin. The doctor should look at all skin lesions to rule out other causes. A skin lesion test, called a biopsy, may be done to help decide the cause of skin problem.

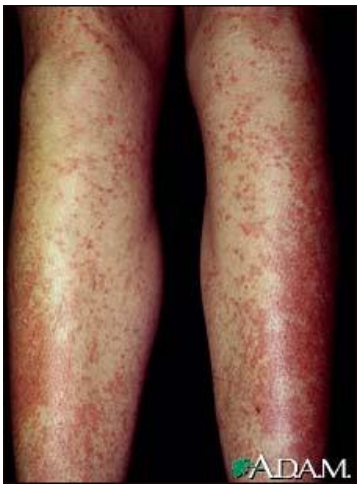
5. Drug Rashes 药疹

Drug rashes are a side effect of a drug that manifests as a skin reaction.

Most drug rashes result from an allergic reaction to the drug. The drug does not have to be applied to the skin to cause a drug rash. Sometimes a person can be sensitized to a drug by one exposure,

and other times sensitization occurs only after many exposures to a substance. Later exposure to the drug may trigger an allergic reaction, such as a rash.





Sometimes a rash develops directly without involving an allergic reaction. For example, corticosteroids and lithium produce a rash that looks like acne, and anticoagulants (blood thinners) may cause bruising when blood leaks under the skin. Other important nonallergic rashes that may result from drugs are those that occur in Stevens-Johnson syndrome, toxic epidermal necrolysis, and erythema nodosum.

Certain drugs make the skin particularly sensitive to the effects of sunlight (photosensitivity). These drugs include certain antipsychotics, tetracycline, sulfa antibiotics, chlorothiazide, and some artificial sweeteners. No rash appears when the drug is taken, but later exposure to the sun produces a reddened area of skin that is sometimes itchy or that appears grayish blue.

Symptoms

Drug rashes vary in severity from mild redness with tiny bumps over a small area to peeling of the entire skin. Rashes may appear suddenly within minutes after a person takes a drug, or they may be delayed for hours or days. People with an allergic rash often have other allergic symptoms—runny nose, watery eyes, wheezing, and even collapse from dangerously low blood pressure. Hives are very itchy, whereas other drug rashes itch little, if at all.

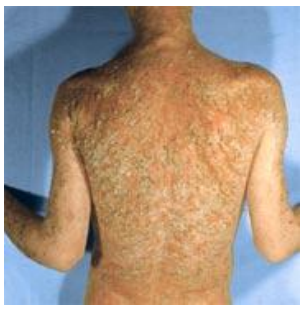
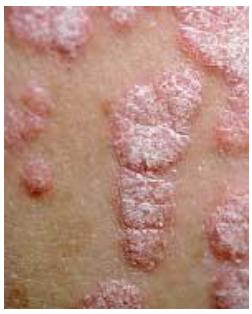
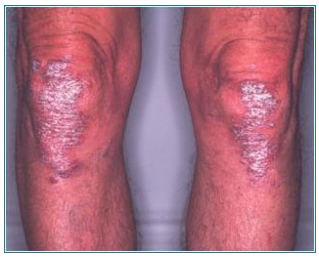
Diagnosis and Treatment

Figuring out whether a drug is responsible may be difficult because a rash can result from only a minute amount of a drug, it can erupt long after a person has taken a drug, and it can persist for weeks or months after a person has discontinued a drug. Every drug a person has taken is suspect, including those bought without a prescription; even eye drops, nose drops, and suppositories are possible causes. Sometimes the only way to determine which drug is causing a rash is to have the person discontinue all but life-sustaining drugs. Whenever possible, chemically unrelated drugs are substituted. If there are no such substitutes, the person starts taking the drugs again one at a time to see which one causes the reaction. However, this method can be hazardous if the person

had a severe allergic reaction to the drug. Skin testing is not helpful, except when penicillin is the suspect drug.

Chapter 7. Dermatitis with erythema and squame

1. Psoriasis 牛皮癣,银屑病

Psoriasis (say "sor-eye-ah-sus") is a condition that affects your skin and causes thick red marks that look like scales to form. The thick scaling is due to an increase in the number of skin cells. Sometimes pus-filled blisters form. Most of the time, the skin on the elbows and knees is affected, but psoriasis can occur anywhere on the body, including the scalp, fingernails and mouth, and even the skin over the joints. Psoriasis tends to run in families and it usually appears between the ages of 15 and 35.

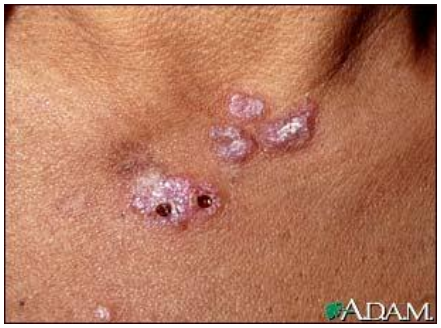
Doctors now believe that psoriasis starts with the immune system. T cells, a type of white blood cell, usually protect the body against infection and disease by attacking bacteria and viruses. However, when you have psoriasis, your T cells mistakenly attack your skin cells instead. Your body then produces other immune system responses, leading to swelling and rapid production of of skin cells.

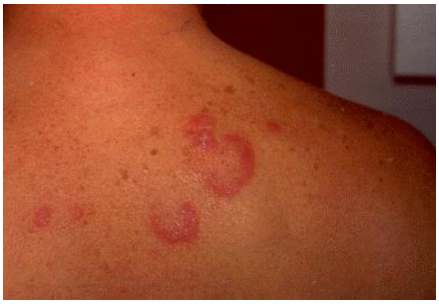
Certain things that can cause the psoriasis to get worse include:

- Infections
- Disease that weaken the immune system
- Stress
- Certain medications

Chapter 8. Connective tissue diseases

1. Discoid Lupus Erythematosus





Discoid lupus erythematosus (DLE) is a disease in which coin-shaped (discoid) red bumps appear on the skin.

Discoid lupus erythematosus only affects the skin, although similar discoid skin lesions can occur in the serious disease called systemic lupus erythematosus (SLE). Only about 10% of all patients with DLE will go on to develop the multi-organ disease SLE.

The tendency to develop DLE seems to run in families. Although men or women of any age can develop DLE, it occurs in women three times more frequently than in men. The typical DLE patient is a woman in her 30s.

■ **Causes and symptoms:** The cause of DLE is unknown. It is thought that DLE (like SLE) may be an autoimmune disorder. Autoimmune disorders are those that occur when cells of the immune system are misdirected against the body. Normally, immune cells work to recognize and help destroy foreign invaders like bacteria, viruses, and fungi. In autoimmune disorders, these cells mistakenly recognize various tissues of the body as foreign invaders, and attack and destroy these tissues. In SLE, the misdirected immune cells are antibodies. In DLE, the damaging cells are believed to be a type of white blood cell called a T lymphocyte. The injury to the skin results in inflammation and the characteristic discoid lesions.

In DLE, the characteristic skin lesion is circular and raised. The reddish rash is about 5-10 mm in diameter, with the center often somewhat scaly and lighter in color than the darker outer ring. The surface of these lesions is sometimes described as "warty." There is rarely any itching or pain associated with discoid lesions. They tend to appear on the face, ears, neck, scalp, chest, back, and arms. As DLE lesions heal, they leave thickened, scarred areas of skin. When the scalp is severely affected, there may be associated hair loss (alopecia).

People with DLE tend to be quite sensitive to the sun. They are more likely to get a sunburn, and the sun is likely to worsen their discoid lesions.

■ **Diagnosis:** Diagnosis of DLE usually requires a skin biopsy. A small sample of a discoid lesion is removed, specially prepared, and examined under a microscope. Usually, the lesion has certain microscopic characteristics that allow it to be identified as a DLE lesion. Blood tests will not reveal the type of antibodies present in SLE, and physical examination usually does not reveal anything other than the skin lesions. If antibodies exist in the blood, or if other symptoms or physical signs are found, it is possible that the discoid lesions are a sign of SLE rather than DLE.

2. Scleroderma

Scleroderma is an autoimmune disease of the connective tissue. Scleroderma is characterized by the formation of scar tissue (fibrosis) in the skin and organs of the body. This leads to thickness and firmness of involved areas. Scleroderma is also referred to as systemic sclerosis.

The cause of scleroderma is not known. Researchers have found some evidence that genes are important factors, but the environment seems to also play a role. The result is activation of the immune system causing injury to tissues that result in injury similar to scar tissue formation. The fact that genes seem to cause a predisposition to developing scleroderma means that inheritance at least play a partial role. It is not unusual to find other autoimmune diseases in families of

scleroderma patients. Some evidence for the role genes may play in leading to the development of scleroderma comes from the study of Choctaw Native Americans who are the group with the highest reported prevalence of the disease. The disease is more frequent in females than in males.

Scleroderma can be classified in terms of the degree and location of the skin involvement. Accordingly, scleroderma has been categorized into two major groups, diffuse and limited.

The diffuse form of scleroderma is involves symmetric thickening of skin of the extremities, face, trunk (chest, back, abdomen, or flanks) which can rapidly progress to hardening after an early inflammatory phase. Organ disease can occur early on and be serious. Organs affected include the esophagus, bowels, lungs with scarring (fibrosis), heart, and kidneys. High blood pressure can be a troublesome side effect.

The limited form of scleroderma tends to be confined to the skin of the fingers and face. The skin changes and other features of disease tend to occur more slowly than in the diffuse form. Because a characteristic clinical pattern can occur in patients with the limited form of scleroderma, this form has taken another name which is composed of the first initials of the common components. Thus, this form is also called the **CREST** variant of scleroderma. This name represents the following features:

C.....Calcinosis, refers to the formation of tiny deposits of calcium in the skin. This is seen as hard whitish areas in the superficial skin, commonly overlying the elbows, knees, or fingers. These firm deposits can be tender, can become infected, and can fall off spontaneously or require surgical removal. This is the least common of the CREST scleroderma variant features.

R.....Raynaud's phenomenon refers to the spasm of the tiny artery vessels supplying blood to the fingers, toes, nose, tongue, or ears. These areas turns blue, white, then red after exposure to extremes of cold, or even sometimes with extremes of heat or emotional upset. For more information, please read the Raynaud's Phenomenon article.

E.....Esophagus disease in scleroderma is characterized by poorly functioning muscle of the lower 2/3 of the esophagus. This can lead to an abnormally wide esophagus which allows stomach acid to backflow into the esophagus to cause heartburn, inflammation, and potentially scarring. This can eventually lead to difficulty in passing food from the mouth through the esophagus into the stomach. Symptoms of heartburn are treated aggressively in patients with scleroderma in order to prevent injury to the esophagus.

S.....Sclerodactyly refers to the localized thickening and tightness of the skin of the fingers or toes. This can give them a "shiny" and slightly puffy appearance. The tightness can cause severe limitation of motion of the fingers and toes. These skin changes generally progress much slower that those of patients with the diffuse form of scleroderma.

T.....Telangiectasias are tiny red areas, frequently on the face, hands and in the mouth behind the lips. These areas blanch when they are pressed upon and represent dilated capillaries.

Patients can have variations of CREST, for example, CRST, REST, ST, etc. Patients can also have "overlap" illness with features of both CREST and the diffuse form of scleroderma. Some patients have overlaps of scleroderma and other connective tissue diseases, such as rheumatoid arthritis,

systemic lupus erythematosus, and polymyositis. When features of scleroderma are present along with features of polymyositis and systemic lupus erythematosus, the condition is referred to as mixed connective tissue disease (MCTD).

Finally, scleroderma skin changes can be very localized. Morphea is scleroderma skin that is localized to a patchy area of the skin that becomes hardened and slightly pigmented. Sometimes morphea can cause multiple lesions in the skin. Linear scleroderma is scleroderma that is localized usually to a lower extremity, frequently presenting as a strip of hardening skin down the leg of a child. Linear scleroderma in children can stunt bone growth of the affected limb. Sometimes linear scleroderma is associated with a "satellite" area of a patch of localized scleroderma skin, such as on the abdomen.

Symptoms of scleroderma

The symptoms of scleroderma depend on the type of scleroderma present and the extent of external and internal involvement in the individual affected. Because scleroderma can affect the skin, esophagus, blood vessels, kidneys, lungs, blood pressure and bowels, the symptoms it causes can involve many areas of the body.

Scleroderma affects the skin to cause local or widespread signs of inflammation (redness, swelling, tenderness, itching, and pain) that can lead to skin tightness or hardening. These skin changes can be widespread, but most common affect the fingers, feet, face, and neck. This can lead to decreased range of motion of the fingers, toes, and jaw. Tiny areas of calcification (calcinosis), while not common, can sometimes be noticed as hard nodules at the tips of the elbows or in the fingers.

Scleroderma affecting the esophagus leads to heartburn. This is directly a result of stomach acid backflowing up into the esophagus. Sometimes this can lead to scarring of the esophagus with difficulty swallowing and/or localized pain in the central chest.

Blood vessels that can be affected include the tiny arterioles of the finger tips, toes, and elsewhere. These vessels can have a tendency to spasm when the areas are exposed to cold, leading to blueness, whiteness, and redness of involved fingers, toes, and sometimes nose or ears. These color changes are referred to as Raynaud's phenomenon. Raynaud's phenomenon can cause inadequate supply of oxygen to the involve tips of fingers or toes, causing tiny ulcers or blackened (dead) skin. Sometimes Raynaud's phenomenon is also associated with tingling. Other blood vessels that can be involved in scleroderma are the tiny capillaries of the face, lips, mouth, or fingers. These capillaries widen (dilate) forming tiny, red blanching spots, called telangiectasias.

Elevated blood pressure is potentially serious and can lead to kidney damage. Symptoms include headache, fatigue, and in severe cases, stroke.

Inflammation of the lungs in scleroderma can cause scarring, resulting in shortness of breath, especially with physical exertion. Elevated pressure in the arteries to the lungs (pulmonary hypertension) can also cause shortness of breath and difficulty getting an adequate breath with activity.

Scleroderma affecting the large bowel (colon) most often causes constipation, but can also lead to cramping and diarrhea. When this is severe, it complete stool blockage (fecal impaction) can result.

Diagnoses of scleroderma

The diagnosis of the scleroderma syndrome is based on the finding of the clinical features of the illnesses. Nearly all patients with scleroderma have blood tests which suggest autoimmunity, antinuclear antibodies (ANAs). A particular antibody, the anticentromere antibody, is found almost exclusively in the limited, or CREST, form of scleroderma. Anti-Scl 70 antibody (antitopoisomerase I antibody) is most often seen in patients with the diffuse form of scleroderma.

Other tests are used to evaluate the presence or extent of any internal disease. These may include upper and lower gastrointestinal tests to evaluate the bowels, chest x-rays, and lung function tests to examine the lungs, EKG and echocardiograms to evaluate the heart and lung arteries.

Appendix

Dermatological diagnosis of Western medicine

1. History

A complete history should be obtained to emphasize the following features:

1. Evolution of lesions
    a. Site of onset
    b. Manner in which eruption progressed or spread
    c. Duration
    d. Periods of resolution or improvement in chronic eruptions
2. Symptoms associated with the eruption
    a. Itching, burning, pain, numbness
    b. What, if anything, has relieved symptoms?
    c. Time of day when symptoms are most severe
3. Current or recent medications (prescribed as well as over-the-counter)
4. Associated systemic symptoms (e.g., malaise, fever, arthralgia)
5. Ongoing or previous illnesses
6. History of allergies
7. Presence of photosensitivity

2. DIAGNOSTIC TECHNIQUES

Many skin diseases can be diagnosed on gross clinical appearance, but sometimes relatively simple diagnostic procedures can yield valuable information. In most instances, they can be performed at the bedside with a minimum of equipment.

Skin Biopsy: A skin biopsy is a straightforward minor surgical procedure; however, it is important to biopsy the anatomic site most likely to yield diagnostic findings. In this procedure, a small area of skin is anesthetized with 1% lidocaine with or without epinephrine. The skin lesion in question can be excised with a scalpel or removed by punch biopsy. In the latter technique, a punch is pressed against the surface of the skin and rotated with downward pressure until it penetrates to the subcutaneous tissue. The circular biopsy is then lifted with forceps, and the bottom is cut with iris scissors. Biopsy sites may or may not need suture closure depending on size and location.

Koh Preparation: A potassium hydroxide (KOH) preparation is performed on scaling skin lesions when a fungal etiology is suspected. The edge of such a lesion is scraped gently with a scalpel blade, and the removed scale is collected on a glass microscope slide and treated with 1 to 2 drops of a solution of 10 to 20% KOH. KOH dissolves keratin and allows easier visualization of fungal elements. Brief heating of the slide accelerates dissolution of keratin. When the preparation is viewed under the microscope, the refractile hyphae will be seen more easily when the light intensity is reduced. This technique can be utilized to identify hyphae in dermatophyte infections, pseudohyphae and budding yeast in Candida infections, and fragmented hyphae and spores in tinea versicolor. The same sampling technique can be used to obtain scale for culture of selected pathogenic organisms.

Tzanck Smear: A Tzanck smear, named after Arnault Tzanck, is a cytologic technique most often used in the diagnosis of herpesvirus infections (simplex or varicella zoster). An early vesicle, not a pustule or crusted lesion, is unroofed, and the base of the lesion is scraped gently with a scalpel blade. The material is then placed on a glass slide, air-dried, and stained with Giemsa or Wright's stain. Multinucleated giant cells suggest the presence of herpes, but culture or immunofluorescence testing must be performed to identify the specific virus.

Dias copy: Diascopy is designed to assess whether a skin lesion will blanch with pressure as, for example, in determining whether a red lesion is hemorrhagic or simply blood-filled. For instance, a hemangioma will blanch with pressure, whereas a purpuric lesion caused by necrotizing vasculitis will not. Diascopy is performed by pressing a microscope slide or magnifying lens against a specified lesion and noting the amount of blanching that occurs. Granulomas often have an "apple jelly" appearance on diascopy.

Wood's Light: A Wood's lamp generates 360-nm ultraviolet (or "black") light that can be used to aid the evaluation of certain skin disorders. For example, a Wood's lamp will cause erythrasma (a superficial, intertriginous infection caused by Corynebacterium minutissimum) to show a characteristic coral red color, and wounds colonized by Pseudomonas to appear pale blue. Tinea capitis caused by certain dermatophytes such as Microsporum canis or M. audouini exhibits a yellow fluorescence. Pigmented lesions of the epidermis such as freckles are accentuated, while dermal pigment such as postinflammatory hyperpigmentation fades under a Wood's light. Vitiligo appears totally white under a Wood's lamp, and previously unsuspected areas of involvement often become apparent. A Wood's lamp also may aid in the demonstration of tinea versicolor and in recognition of ash leaf spots in patients with tuberous sclerosis.

Patch Tests: Patch testing is designed to document sensitivity to a specific antigen. In this procedure, a battery of suspected allergens is applied to the patient's back nder occlusive dressings and allowed to remain in contact with the skin for 48 h. The dressings are then removed, and the area is examined for evidence of delayed hypersensitivity reactions (e.g., erythema, edema, or papulovesicles). This test is best performed by physicians with special expertise in patch testing and is often helpful in the evaluation of patients with chronic dermatitis.