Methodic elaboration for practical lesson in

# Dermatovenerology

for students of Medicine Faculty nr.2

## Topic N3

**Pyodermas.**
**Infestations.**

## BACTERIAL INFECTIONS (PYODERMAS)

### Skin Infections

The two gram-positive cocci, *Staphylococcus aureus* and the group A beta-hemolytic streptococci, account for the majority of skin and soft tissue infections. The streptococci are secondary invaders of traumatic skin lesions and cause impetigo, erysipelas, cellulitis, and lymphangitis. *S. aureus* causes impetigo, folliculitis, cellulitis, and furuncles. Elaboration of toxins by *S. aureus* causes the lesions of bullous impetigo and staphylococcal scalded skin syndrome.

### IMPETIGO

Impetigo is common, contagious, superficial skin infection that is produced by streptococci, staphylococci, or a combination of both bacteria. There are two different clinical presentations: bullous impetigo and non-bullous impetigo.

Both begin as vesicles with a very thin, fragile roof consisting only stratum corneum. Bullous impetigo is primarily a staphylococcal disease.

Nonbullous impetigo was once thought to be primarily a streptococcal disease, but staphylococci are isolated from the majority of lesions in both bullous and nonbullous impetigo. *S. aureus* is now known to be the primary pathogen in both bullous and nonbullous impetigo.

Children in close physical contact with each other have a higher rate of infection than do adults.

Symptoms of itching and soreness are mild; systemic symptoms are infrequent. The disease is self-limiting, but when untreated it may last for weeks or moonths.

- **Bullous impetigo.** It typically occurs on the face, but it may infect any body surface. One or more vesicles enlarge rapidly to form bullae in which the contents turn from clear to cloudy. The center of the thin-roofed bulla collapses, but the peripheral area may retain fluid for many days in an inner tube-shaped rim. A thin, flat, honey-colored, 'varnishlike' crust may appear in the center and, if removed, discloses bright red, inflamed, moist that oozes serum. The center may dry without forming a crust, leaving a rad base with a rim the scale. In most case, a tinea-like scaling border replaces the fluid-filled rim as the round lesions enlarge and become contiguous with the others. The border dries and forms a crust. The lesions have little or no surrounding erythema. In some untreated cases, lesions may extend radially and retain a narrow, bullous, inner tube rim. Lesions heal with hyperpigmentation in black patients. Regional lymphadenitis is uncommon with pure staphylococcal impetigo.
- **Nonbullous impetigo** originates as a small vesicle or pustule that ruptures to expose a red moist base. A honey-yellou to white-brown, firmly adherent crust accumulates as the lesion extends radially. There is little surrounding erythema. Satellite lesions appear beyond the periphery. The lesions are generally asymptomatic. The skin around the nose and mouth and the limbs are the sites most commonly affected. The palms and soles are not affected. Most cases without scarring. Regional lymphadenopathy is common. Children ages 2 to 5 years commonly have streptococcal impetigo.

#### Laboratory findings

Cultures of the pharynx and any skin lesion should be made and the serotype of the group A streptococcus that is responsible should be determined by typing with M-group and T- types antiserum. M-T serotypes associated with acute nephritis.

#### Prevention of impetigo

Mupirocin (Bactroban) or Triple antibiotic ointment, containing Bacitracin, Polysporin, and Neomycin, applied three times daily to sites of minor skin trauma can be efficacious as a preventative treatment.

**Recurrent impetigo**

Patients with recurrent impetigo should be evaluated for carriage of *S. aureus*. The nares are the most common sites of carriage, but the perineum, axillae, and toe webs may also be colonized. Bactroban applied to the nares twice each day for 5 days signi
ficantly reduced *S. aureus* carriage in the nose and hands at 3 days and in the nasal carriage for as long as 1 year.

**Treatment of impetigo**

Impetigo may resolve spontaneously or become chronic and widespread.

**Topical treatment**

Local treatment does not treat lesions that evolve in other areas. Infected children should be briefly isolated until treatment is under way. If topical treatment is elected, then it might be worthwhile to wash the involved areas once or twice a day with an antibacterial soap such as Hibiclens or Betadine. Crusts should be removed because they block the penetration of antibacterial creams. To facilitate removal, soaking with a wet cloth compress softens crusts.

- Bactroban ointment is the first topical antibiotic approved for the treatment of impetigo.
- Studies show that 2% Mupirocin ointment is as safe and effective as oral erythromycin in the treatment of patients with impetigo.

**Sistemic treatment**

- Oral antibiotics -A 5- to 10-day course of an oral antibiotic such as Cloxacillin, Dicloxacillin, or Cephalexin induced rapid healing. Erythromycin may not be as effective because strains of resistant staphylococci are appearing in some areas. Azithromycin given over 5 days, as a once-a-day regimen is as effective and better tolerated than either Erythromycin or Cloxacillin.

**FOLLICULITIS.**

Folliculitis is inflammation of the hair follicle caused by infection, chemical irritation, or physical injury. Inflammation may be superficial or deep in the hair follicle. Folliculitis is very common and is seen as a component of a variety of inflammatory skin diseases, which are listed in the box below.

In superficial folliculitis, the inflammation is confined to the upper part of the hair follicle. Clinically, it is manifested as a painless or tender pustule that eventually heals without scarring. In many instances, the hair shaft in the center of the pustule cannot be seen. Inflammation of the entire follicle or the deeper portion of the hair follicle initially appears as a swollen, red mass, which eventually may point toward the surface becoming a somewhat larger pustule than that seen in superficial folliculitis. Deeper lesions are painful and may heal with scarring.

- **Staphylococcal folliculitis.** Staphylococcal folliculitis is most common form of infectious folliculitis. One pustule or a group of pustules may appear, usually without fever or other systemic symptoms on any body surface. Staphylococcal folliculitis may occur because of injury, abrasion, or nearby surgical wounds or draining abscesses. It may also be a complication of occlusive topical steroid therapy.
- **Sycosis barbae.** Sycosis implies follicular inflammation of entire depth of the hair follicle and may be caused by infection with Staphylococcus aureus or dermatophite fungi. The disease occurs only in men who have commenced shaving. It begins with the appearance of small follicular papules or pustules and rapidly becomes more diffuse as shaving continues. Infiltration about the follicle may be slight or extensive. The more infiltrated cases heal with scarring. In chronic cases the pustules may remain confined to one area, such as the upper lip or neck. The hairs are epilated with difficulty in staphylococcal sycosis and relative ease in fungal sycosis. Hairs should be removed and examined for fungi and the purulent material should be cultured. Localized inflammation is treated topically with

Bactroban. Extensive disease is treated with oral antibiotics. Shaving should be performed with a clean razor.

- **Furuncles and Carbuncles**

o **A furuncle** is a walled-of collection of pus that is a painful, firm, and fluctuant mass. An abscess is not hollow sphere, but a cavity formed by fingerlike locutions of granulation tissue and pus that extends outward along planes of least resistance. They occur at any side, but they appear particularly in areas prone to friction or minor trauma, such as underneath a belt, the anterior thighs, buttocks, groin, axillae, and waist. Furuncles are uncommon in children, but increase in frequency after puberty. *S. aureus* is the most common pathogen (other organisms either aerobic – *E. coli, P. aeruginosa* or unaerobic – *Bacteroides, Lactobacillus*).

o **Furunculosis** occurs as a self-limited infection in which one or several lesions are present or as a chronic, recurrent disease that lasts for months or years, effecting one or several family members. Most patients with sporadic or recurrent furunculosis appear to be otherwise normal and have an intact immune system.

o **Predisposing conditions.** A number of factors predispose the hair follicle to infections. Occlusion of the groin and buttocks by clothing, especially in-patients with hyperhidrosis, encourages bacterial colonization. Follicular abnormalities, evident by the presence of comedones and acneiform papules are often found on the buttocks and axillae of patients with recurrent furunculosis of those areas; these findings suggest the diagnosis of hidradenitis suppurativa. Bacteria colonize the skin in-patients with atopic dermatitis, eczema, and scabies

o .**Clinical manifestations.** The lesion begins as a deep, tender, firm, red papule that enlarges rapidly into a tender, deep-seated nodule that remains stable and painful for days and then becomes fluctuant. The temperature is normal and there are no systemic symptoms. Pain becomes moderate to severe as purulent material accumulates. Pain is most intense in areas where expansion is restricted, such as the neck and external auditory canal. The abscess both remains deep and reabsorbs or points and ruptures through the surface. The abscess cavity contains a surprisingly large quantity of pus and white chunks of necrotic tissue. The point of rupture heals with scarring.

o **Carbuncles.** Carbuncles are aggregates of infected follicles. The infection originates deep in the dermis and the subcutaneous tissue, forming a broad, red, swollen, slowly evolving, deep, painful mass that points and drains through multiple openings. Malaise, chills, and fever precede or occur during the active phase. Sloughing and extensive scarring may follow deep extension into the subcutaneous tissue. Areas with thick dermis are the preferred sites. In the preantibiotic era, there were some fatalities.

**Treatment. Antibiotics.** Patients with recurrent furunculosis learn that they can sometimes stop the progression of an abscess by starting antistaphylococcal antibiotics at the first sign of the typical localized swelling and erythema. They continue to use antibiotics for 5 to 10 days. Antibiotics should be started immediately in order to attenuate the evolving abscess. Antibiotics have little effect once the abscess has become fluctuant. The primary management of cutaneous abscesses should be incision and drainage.

**STAPHYLOCOCCAL SCALDED SKIN SYNDROME (SSSS) –**

Red blistering skin that looks like a burn or scald, hence its name staphylococcal scalded skin syndrome.

- SSSS is caused by the release of two exotoxins (epidermolytic toxins A and B) from toxigenic strains of the bacteria *Staphylococcus aureus*.
- The toxins bind to a molecule within the desmosome called Desmoglein 1 and break it up so the skin cells become unstuck.
- SSSS has also been called Ritter's disease or Lyell's disease when it appears in newborns or young infants.

- SSSS usually starts with fever, irritability and widespread redness of the skin. Within 24-48 hours fluid-filled blisters form. These rupture easily, leaving an area that looks like a burn.

Characteristics of the rash include:

- Tissue paper-like wrinkling of the skin is followed by the appearance of large fluid-filled blisters (bullae) in the armpits, groin and body orifices such as the nose and ears.
- Rash spreads to other parts of the body including the arms, legs and trunk. In newborns, lesions are often found in the diaper area or around the umbilical cord.
- Top layer of skin begins peeling off in sheets, leaving exposed a moist, red and tender area.
- Other symptoms may include tender and painful areas around the infection site, weakness, and dehydration.

**ECTHYMA**.

Ecthyma is characterized by ulceration that is covered by adherent crusts. Poor hygiene is a predisposing factor. Ecthyma has many features similar to those of impetigo. The lesions begin as vesicles and bulla. They then rupture to form an adherent crust that covers an ulcer rather than the erosion of impetigo. This type of lesion occurs most commonly on the legs, where there are usually less than 10 lesions. Another more diffuse form occurs on the buttocks and legs of children who excoriate. Except for the thick crusts and underlying ulcers, the picture is approximately identical to diffuse streptococcal impetigo. Lesions heal with scarring.

Ecthyma is initiated by group A beta-hemolytic streptococci, but quickly becomes contaminated with staphylococci. This should be treated with a 10-day course of the oral antibiotic such as Dicloxacillin or a Cephalosporin such as Cephalexin.

**ERYSIPELAS**

Erysipelas is a superficial form of cellulitis, a potentially serious bacterial infection affecting the skin. Erysipelas most often affects infants and the elderly, but can affect any age group. Risk factors are similar to those for other forms of cellulitis. However, unlike cellulitis, almost all erysipelas is caused by Group A beta haemolytic streptococci (*Streptococcus pyogenes*)

Clinical features:

- Erysipelas predominantly affects the skin of **the lower limbs,** but when it involves **the face** it can have a characteristic butterfly distribution on the cheeks and bridge of the nose.
- Symptoms and signs of erysipelas are usually **abrupt in onset** and often accompanied by **general illness** in the form of fevers, chills and shivering.
- Affected skin **is distinguished** from other forms of cellulitis **by a well-defined, raised border**. The affected skin is red, swollen and may be finely dimpled (like an orange skin). It **may be blistered**. Bleeding into the skin may cause purpura (purple patches).
- **Cellulitis does not usually exhibit such marked swelling** but shares other features with erysipelas such as pain and increased warmth of affected skin.

**CELLULITIS**

Is a common bacterial infection of the hypodermis, which can affect all ages. It usually affects a **limb** but can occur anywhere on the body.

- Symptoms and signs are usually **localised to the affected area** but patients can become generally unwell with fevers, chills and shakes (bacteraemia).
- Severe or rapidly progressive cellulitis may lead to **septicaemia** (blood poisoning), **necrotising fasciitis** (a more serious soft tissue infection), or **endocarditis** (heart valve infection).

Some or all of the following features may be seen over the affected skin.

- Redness
- Swelling
- Increased warmth

- Tenderness
- Blistering
- Abscess
- Erosions and ulceration
- If there is no increased warmth over the skin it is unlikely to be cellulitis.
- Lymphangitis is a red line originating from the cellulitis and leading to tender swollen lymph glands draining the affected area (e.g. in the groin with a leg cellulitis). It is caused by infection within the lymph vessels.
- After successful treatment, the skin may flake or peel off as it heals.

**NECROTISING FASCIITIS**

Is a very serious bacterial **infection of the soft tissue and fascia** (a sheath of tissue covering the muscle). The bacteria multiply and release toxins and enzymes that result in thrombosis (clotting) in the blood vessels. The result is destruction of the soft tissues and fascia. There are **three main types** of necrotising fasciitis:

  - Type I (polymicrobial i.e. more than one bacteria involved)
  - Type II (due to haemolytic group A streptococcus)
  - Type III (gas gangrene)

- Bacteria causing type 1 necrotising fasciitis include ***Staphylococcus aureus***, *Haemophilus*, *Vibrio* and several other aerobic and anaerobic strains. It usually follows significant injury or surgery.
- Type II necrotising fasciitis has recently been sensationalised in the media and is commonly referred to **as flesh-eating disease**.
- Type III is caused by ***Clostridia perfringens*** or less commonly *Clostridia septicum*. It usually follows significant injury or surgery and results in gas under the skin: this makes a crackling sound called **crepitus.**

**Signs and symptoms:**

- Symptoms appearing usually within 24 hours of a minor injury
- Pain in the general area of the injury and worsening over time
- Flu-like symptoms such as nausea, fever, diarrhoea, dizziness and general malaise
- Intense thirst as body becomes dehydrated
- Within 3-4 days of the initial symptoms the following may occur:
  - Affected area starts to swell and may show a purplish rash
  - Large dark marks form that turn into blisters filled with dark fluid
  - Wound starts to die and area becomes blackened (necrosis)
  - Severe pain
- By about days 4-5, the patient is very ill with dangerously low blood pressure and high temperature. The infection has spread into the bloodstream and the body goes into toxic shock. The patient may have altered levels of consciousness or become totally unconscious.

**SCABIES**.

Human scabies is a contagious disease caused by the mite *Sarcoptes scabiei* var. *hominis.* Dogs and cats may be infested by almost identical organisms; these sometimes may be a source for human manifestation. In the past, scabies was attributed to poor hygiene. Most contemporary cases, however, appear in individuals with adequate hygiene who are in close contact with numbers of individuals, such as schoolchildren. Blacks rarely acquire scabies; the reason is unknown.

Anatomic features, life cycle, and immunology.The adult mite is 1/3 mm long and has a flattened, oval body with wrinklelike, transverse corrugations and eight legs. The front two pairs of

legs bear claw-shaped suckers and two rear pairs end in long, trailing bristles. The digestive tract fills a major portion of the body and is readily observed when the mite is seen in cross-section of histologic specimens.

Infestation begins when a fertilized female mite arrives on the skin surface. Within an hour, the female excavates a burrow in the stratum corneum. During the mite's 30-days life cycle, the burrow extends from several millimeters to a few centimeters in length. The burrow does not enter the underlying epidermis except in the case of hyperkeratotic Norwegian scabies, a condition in which retarded, immunosupressed, or elderly patients develop scaly, thick skin in the presence of thousands of mites. Eggs laid at the rate of 2 or 3 day and fecal pellets are deposited in the burrow behind the advancing female. Scybala are dark, oval masses that are seen easily with the eggs when burrow scrapings are examined under a microscope. Scybala may act as an irritant and may be responsible for some of the itching. The larvae hatch, leaving the egg casings in the burrow, and reach maturity in 14 to 17 days. The adult mites copulate and repeat the cycle. Therefore, 3 to 5 weeks after infestation, there are only a few mites present. This life cycle explains why patients experience few if any symptoms during the first month after contact with an infested individual. After a number of mites have reached maturity and have spread by migration or the patient's scratching, the initial, minor, localized itch evolves into intense, generalized pruritis.

A hypersensitivity reaction rather than a foreign-body response may be responsible for the lesions, which may delay recognition of symptoms of scabies. Some patients infested with scabies develop elevated IgE titers, eosinophilia, and an immediate-type hypersensitivity reaction to an extract prepared from female mites. IgE levels fall within a year after infestation. Eosinophilia returns to normal shortly after treatment. The fact that patients develop symptoms much more rapidly when reinvested supports the claim the symptoms and lesions of scabies are the result of a hypersensitivity reaction.

**Clinical manifestations.** Transmission of scabies occurs during direct skin contact with an infected person. Whether or not the mite can be acquired from infested clothing or bed lines is not known. A mite can possibly survive for days in normal home surroundings after leaving human skin. Mites survive up to 7 days in mineral oil microscopic slide month.

**Symptoms.** The disease begins insidiously. Symptoms are minor at first and are attributed to a bite or dry skin. Scratching destroys burrows and removes mites, providing initial relief. The patient remains comfortable during the day but itches at night. Nocturnal pruritis is highly characteristic of scabies. Scratching spreads mites to other areas and after 6 to 8 weeks the once localized area of minor irritation has become a widespread, intensely pruritic eruption.

The most characteristic features of the lesions are pleomorphism and a tendency to remain discrete and small. Scratching soon destroys primary lesions.

**Primary lesions .** Mites are found in burrows and at the edge of vesicles, but rarely in papules.

**Burrow.** The linear, curved, or S-shaped burrows are approximately as wide as #2 suture material and are 2 to 15 mm long. They are pink-white and slightly elevated. A vesicle or the mite, which may look like a black dot at one end of the burrow, often may be been. Scratching destroys burrows, therefore they do not appear in some patients. Burrows are most likely to be found in the finger webs, wrists, sides of the hands and feet, penis, buttocks, scrotum, and the palms and soles of infants.

**Vesicles and papules.** Vesicles are isolated, pinpoint, and filled with serous rather than purulent fluid. The fact that they remain discreet is a key point in differentiating scabies from other vesicular diseases such as poison ivy. The finger webs are the most likely areas to find intact vesicles. Infants may have vesicles or pustules on the palms and soles. Small, discrete papules may represent a hypersensitivity reaction and rarely contain mites.

**Secondary lesions.** Scratching causes secondary lesions from infection. They often dominate the clinical picture. Pinpoint erosions are the most common secondary lesions. Pustules are a sign of secondary infection. Scaling, erythema, and all stages of eczematous inflammation occur as a response to excoriation or to irritation caused by overzealous attempts at self-medication.

Nodules occur in covered areas such as the buttocks, groin, scrotum, penis, and axillae. The 2- to 10-mm indolent, red papules and nodules sometimes have slightly eroded surfaces, especially on the glans penis. Nodules may persist for weeks or months after the mites have been eradicated. They may result from persisting antigens of mite parts.

**Distribution.** Lesions of scabies are typically found in the finger webs, wrists, extensor surfaces of the elbows and knees, sides of the hands and feet, axillary areas, buttocks, waist area, and ankle area. In men, the penis and scrotum are usually involved; in women, the breast, including the areola and nipple, may be infested. Lesions, often vesicular or pustular, may be most numerous on the palms and soles of infants. The scalp and face, rarely involved in adults, occasionally are infested in infants.

**Diagnosis.**

The diagnosis is suspected when burrows are found or when a patient has typical symptoms with characteristic lesions and distribution. The definite diagnosis is made when any of the following products are obtained from burrows or vesicles and identified microscopically: mites, eggs, egg casing, or feces.

**Treatment.** 5% Permethrin cream (adults wash 12 hours after application and infants should be washed 8 to 12 hours after application);
1% for adults, 0,1% for children of Lindane lotion, shampoo, cream (also);
Ivermectine oral;
Sulfur 5% to 10% in petrolatum or a cod cream base;
Sulfur ointment 10-15% - for children, 33% - for adults (5 days application);
Benzyl-benzoate lotion, 20% for adults and 10% for children (2 days application).

**PEDICULOSIS**

Infestation with lice is called *pediculosis*. Like are transmitted by close personal contact and contact with objects such as combs, hats, clothing, and bed linen. Seeing the like or their eggs makes diagnosis. Treatment with lindane, permithrin is effective.

Biology and life cycle. Lice are obligate human parasites that cannot survive off their host for more than 10 days (adults) to 3 weeks (fertile eggs). Actual survival rates may be shorter than this. Lice are called *ectoparasites* because they live on, rather than in, the body. They are classified as insects because they have six legs. Three kinds of lice infest humans: *Pediculosis humanus* var. *capitis* (head louse), *Pediculosis humanus* var. *corporis* (body louse), and *Pthirus pubis* (pubic or crab louse). All three have similar anatomic characteristics. Each is a small (less than 2 mm), flat, wingless insect with three pairs of legs located on the anterior part of the body directly behind the head. The legs terminate in sharp claws that are adapted for feeding and permit the louse to grasp and hold firmly on to hair or clothing. The body louse is the largest and is similar in shape to the head louse. The crab louse is the smallest, with a short, oval body and prominent claws resembling sea crabs.

Lice feed approximately five times each day by piercing the skin with their claws, injecting irritating saliva, and sucking blood. They do not become engorged like ticks, but, after feeding, they become rust colored from the ingestion of blood; their color is an identifying characteristic.

Lice feces can be seen on the skin as small, rust-colored flecks. Saliva and, possibly, fecal material can induce a hypersensitivity reaction and inflammation. Lice are active and can travel quickly, which explains why they can be transmitted so easily. The life cycle from egg to egg is approximately 1 month. The female lays approximately six eggs, or nits, each day. The louse incubates, hatches in 8 to 10 days, and reaches maturity in approximately 18 days. Nits are 0,8 mm long and are

firmly cemented to the bases of hair shafts close to the skin to acquire adequate heat for incubation. Nits are very difficult to remove from the hair shaft.

**Clinical manifestations**.

- Pediculosis capitis. Lice infestation of the scalp is most common in children. More girls than boys are afflicted and American blacks rarely have head lice. Head lice can be found anywhere on the scalp, but are most commonly seen on the back of the head and neck and behind the ears. Scratching causes inflammation and secondary bacterial infection, with pustules, crusting, and cervical adenopathy. Posterior cervical adenopathy without obvious disease is characteristic of lice. The eyelashes may be involved, causing redness and swelling. Examination of the posterior scalp shows few adult organisms, but many nits. Nits are cemented to the hair, whereas dandruff scale is easily moved along the hair shaft.
- **Pediculosis corporis**. Infestation by body lice is uncommon. Typhus, relapsing fever, and trench fever are spread by body lice during wartime and in underdeveloped countries. Pediculosis corporis is a disease of the unclean. Body lice live and lay their nits in the seams of clothing and return to the skin surface only to feed. They run and hide when disturbed and are rarely seen. Body lice induce pruritus that leads to scratching and secondary infection.
- **Eyelash infestation.** Infestation of the eyelashes is seen almost exclusively in children. The lice are acquired from other children or from an infested adult with pubic lice. Eyelash infestation may induce blepharitis with lid pruritius, scaling, crusting, and/or purulent discharge. Eyelash infestation may be a sign of childhood sexual abuse.
- **Pediculosis pubis.** Pubic lice are the most contagious sexually transmitted problem known. Up to 30% of patients infested with pubic lice have at least one other sexually transmitted disease. The chance of acquiring pubic lice from one sexual exposure with an infested partner is more than 90%, whereas the chance of acquiring syphilis or gonorrhea from one sexual exposure with an infected partner is approximately 30%. Blacks are affected with the same frequency as whites. The pubic hair is the most common site of infestation, but lice frequently spread to the hair around the anus. On hairy persons, lice may spread to the upper thighs, abdominal area, axillae, chest, and beard. Infested adults may spread pubic lice to the eyelashes of children.

  The majority of patients complain of pruritus. Many patients are aware that something is crawling on the groin, but are not familiar with the disease and have never seen lice. Approximately 50% of patients have little inflammation, but those who delay seeking help may develop widespread inflammation and infection of the groin with regionally adenopathy. Occasionally, gray-blue macules (maculae ceruleae). Varying in size from 1 to 2 cm are seen in the groin and at sites distant from the infestation. Their cause is not known, but they may represent altered blood pigment.

  Diagnosis. Lice are suspected when a patient complains of itching in a localized area without an apparent rash. Scalp and pubic lice will be apparent to those who carefully examine individual hairs; they are not apparent with only a cursory examination. Lice and nits can be seen easily under a microscope. Live nits fluoresce and can be detected easily by Wood's light examination, a technique that is especially useful for rapid examination of a large group of children. Nits that contain an unborn louse fluoresce white. Nits that are empty fluoresce gray.

  Treatment.

  1. Lindane (Kwell) – for 5 min (shampoos or lotions);
  2. Permethrin (Nix Creme Rinse) – 25 to 50 ml for 10 min.