Parasite, Worms and Protozoa -

Assistant Prof. Dr. Khudair Kh. Al-Kayalli .

College of Medicine, Diyala University.

<u>Parasite</u>: is an organism that depends upon a living host for one or more of its essential metabolic requirement. It is of two types :

***Endoparasites : are** parasites which lives within the body , like some intestinal worms , may not invade the tissue .

***Ectoparasite :** are those living on the body surface , like the scabies mite , may in fact burrow into it .

Definitive (final) host – is the host in which the parasite reaches sexual maturity , e.g. human is the definitive host for *Tinea solium*.

Intermediate host – is the host in which the parasite undergoes larval development or an asexual phase , e.g. human may act as intermediate host for the bladder worm (Cysticercus stage) .

The Cutaneous lesions may result from direct damage by or presence of the parasite, or occurs in sites that are not themselves infected, e.g. scabies, the inflammatory lesions present at the sites and away from the presence of *Sarcoptes scabieii*.

<u>Leishmaniasis :</u>

Definition- are a group of diseases caused by several species of the genus **leishmania**. It has been estimated that 1.5million new cases of Cutaneous leishmaniasis occurs annually and more than 80% of the total cases affect individuals in developing countries .

Leishmania spp. under go a cycle of development in the gut of female sand flies , of the genera Phlebotomus in the Old World , and Lutzomyia and Psychodopygns in the New world . In its vertebrate host , the amastigote form of the parasite is found in cells of the reticuloendothelial system or in the dermis following severe parasite load and mononuclear cell necrosis , it is round or oval , 2-3um in diameter , with no protruding flagellum , with nucleus and kinetoplast , stain deeply with Geimsa or Romannovsky stain . In the sand fly and in artificial culture media , leishmania spp. are the elongated promastigote stage , motile with an anterior flagellum .

Infection is transmitted by the bite of the fly, usually at night and outdoors, however infected vectors can take a blood meal during the day if disturbed and also are responsible for inoculating parasites indoors within the household environment. **Commonly the infection** is **zoonotic**, one species of leishmania may be associated with one, or many, natural vertebrate hosts, which provide the reservoir of infection. Humans are commonly accidental hosts, although there are situations in which they may be the reservoir in anthroponotic cycle.

Human leishmaniasis is usually classified as Cutaneous and visceral, but the species that cause visceral disease may also cause skin lesions.

<u>Old world Cutaneous leishmaniasis (</u>Oriental sore , Baghdad boil , Delhi boil , Aleppo boil) .

Aetiology & epidemiology – it is caused by :Leishmania major, L. tropica, L. Ethiopiaca, and L.donovoani infantum. In endemic areas where transmission is stable, children are usually especially 100% afected, in less stable situation for example around oases, epidemics occurs affecting all ages and sexes.

Pathogenesis – sandfllies bite the skin , inoculate the infective metacyclic **promastigotes** when taking a blood meal from the superficial vascular network in the human dermis . Inoculated promastigotes are taken up by histiocytes and newly immigrated monocytes , in which they multiply , most inoculations are killed by phagocytosis and complement – mediated functions , only a minority of successful parasite inoculations results in localized or disseminated clinical Cutaneous leishmaniasis . After a period of time (incubation) , which depends on *parasite species , *size of inoculum , *and the host's cellular immune response , a clinical lesion appears .

Clinical features – all previously uninfected individuals are susceptible, the **incubation period** is usually measured in months, but ranges from a few days to over a year. **One or more lesions** occurs on the unclothed (exposed) parts of the body, particularly on acral skin (face, neck, and arms), easily bitten by phlebotomus, usually in child. In endemic areas more than one children in the same family are affected (infected fly bites them). The sequence of lesions in all species are nodule, crusting, ulceration and healing with scare formation is common in all self healing sores. The clinical forms are :

1. Cutaneous leishmaniasis due to L. major (Wet, rural, or zoonotic):-

After a short incubation period of less than 2months, a red furuncle-like nodule, appears at the site of inoculation, 2weeks later on a central crust forms, which may persist or fall a way revealing the underlying ulcer, the **ulcer** and the raised, red margin enlarge over the next 2-3months, and reaches a diameter of 3-6cm. **Multiple**, small secondary nodules (2-4mm) some times occurs around the lesion in lymphatics, **healing** takes place in 2-6months and leaves a scare. This type of **C.L.** is acquired in a rural area, where the infecting organisms are also rodent parasites (Zoonosis).

2. Cutaneous leihmaniasis due to L. tropica (Dry, urban or anthroponotic):-

After an incubation period of more than 2months, a small brownish –red nodule appears, which extend slowly to form a plaque of 1-2cm in diameter in about 6months, at this stage a shallow ulcer appears in the center, which develops a closely adherent crust. Multiple secondary nodules occurs much less frequently than in the wet form, after 8-12months, the lesion starts to regress and ulcer heals, leaving a scare. The average time from nodule to scare is about 1year, approximately twice as long as in the wet-form. Rarely *L. tropica*

causes **viscerotropic infections in war-veterans** in the middle East and in cases of Indian kala-azar .

3. Cutaneous leihmaniasis due to L. aethiopica :-

The lesions are most commonly central on the face and single, satellite papules acuminate into large, spreading nodules, that may note crust or ulcerate, lesions are seldom much inflamed and heal over 2-5 years. If the sand fly bite has been on the mucosal border of the nose and mouth, primary muco-cutaneous leishmaniasis (MCL), may develop, producing swelling of the lips, nose and persist for many years, but without the gross destruction seen in Latin America caused by *L. brasiliensis*.

4. Cutaneous leishmaniasis due to L. donovani infantum :-

Infants infected with this parasite tend to get **visceral leishmaniasis**, adults are more likely to develop simple self-healing cutaneous disease, with out concurrent or subsequent visceral involvement. The appearance and evolution of the lesions is typically slow and mild, when compared with that of *L. major*, with which it coexists in North Africa. **Solitary mucosal** lesions have occasionally been reported.

5. Other types of cutasneous leishmaniasis :-

a. Leishmaniasis recidivans (*Chronic*, *lupoid leishmaniasis*) :- Approximately 4% of *L. tropica* infections from Iran and Afghanistan will develop this chronic form of the disease . Brown –red or brown-yellow papules appears , usually close to a scar of an old lesion of cutaneous leishmaniasis or actually in the scar , they coalesce and form a plaque closely resembling lupus vulgaris , even to the formation of **apple-gelly** nodules . The lesions frequently worsen in the summer and may ulcerate to form concentric rings , rarely keloidal and verrucous or psoriasiform forms occurs on the limps . The recidivans lesion is the result of the failure of cellular immunity to sterilize the lesion . Investigations to demonstrate the parasite or leishmanial DNA in the infected skin are commonly negative .

b. Diffuse cutaneous leishmaniasis (*Disseminated Cutaneous leishmaniasis*, *leishmaniasis cutis diffusa*) :- In the Old-World this form of the disease id due to *L*. *aethiopica*, and has certain characteristic features, which include :-

1. There is an initial lesion , which spreads locally and from which the disease disseminates to other parts of the skin often involving large area .

- 2. The lesions are nodules that do not ulcerate .
- 3. There is a superabundance of parasites in the lesions .
- 4. The histology is characteristic in that the macrophage full of amastigotes .
- 5. Internal organs are not invaded and there is no history of kala-azar.
- 6. The leishmanin test and other tests of specific cellular immunity are negative .
- 7. The disease progresses slowly and become chronic .
- 8. Treatment produce only gradual improvement and relapse is the rule .

c. Post-kala-azar dermal leishmaniasis (*Dermal leishmanoid*, *PKDL*):- In 5% of East African patients , and 20% of Indian patients , a rash develops after the visceral disease has healed , either spontaneously or following treatment . A small proportion of patients with PKDL give no previous history of visceral disease . The rash in Africa begins during convalescence , appearing on the cheeks , chin , ears , extensor aspects of forearms , buttocks and lower legs , which consists of discrete papules , with tuberculoid histology and scanty parasites , leishmanin test is positive . The rash heals spontaneously over a few months . In Indian , by contrast the rash appears 1-2 years after recovery of kala-azar , as hypopigmented macules , similar in appearance and distribution to those of lepromatous leprosy . After a variable period of years or months , diffuse nodulation begins to develop in these macules , the rash is progressive over many years and seldom heals spontaneously , tongue , palate , and genitalia may be involved , lymphadenopathy , leishmanin test is negative .

Diagnosis :- A positive diagnosis of Cutaneous leishmaniasis (Old and New World types) can be suggested, and in most cases **confirmed** by the presence of one or more of the following criteria :

- 1. History of exposure to an endemic area in the previous weeks or months .
- 2. History of sand fly bites in the previous weeks or months .
- 3. History of high risk activities such as sleeping outdoors, desert trek.
- 4. Non-healing chronic nodular violaceous ulcer for 4-6weeks or longer .
- 5. Demonstration of amastigotes in Giemsa-stained smears from infected skin.
- 6. Demonstration of intracellular amastigotes in dermis of H&E section of skin .
- 7. Presence of leishmanial granulomas in the dermis in H&E specimens .
- 8. Growth of promastigotes in Nicole –Novy-MacNeal (NNN) culture medium from lesional specimens.

Confirmation of diagnosis is through the : **a. Demonstration of the parasite in** <u>: either</u> a smear of material from the sore and staining it with **Wright's**, **Giemsa or Leishman's stains** (by needle aspirate , dental broach or slit-skin smear) , <u>or</u> smear material should be cultured on NNN or similar medium .

b. Lesional skin biopsy for culture and histological examination (granuloma with plasma cells).

c. Leishmanin test will be positive in all forms of cutaneous leishmaniasis once the stage of crusting has been reached, it is negative in diffuse anergic forms. It is called **Montenegro test**. *L. major* is commonly used as culture containing 5000000promastigotes /ml of 0.5% phenol saline, dose of 0.1ml is injected, intradermally in the volar surface of forearm, read it after 48-72hours, induration >5mm, means positive test.

d. PCR to detect leishmanial DNA .

Treatment- although most sores will heal **spontaneously**, but the role of treatment is to reduce the duration of the lesions and to minimize the scars formation. There are 2types of therapy :-

1. Local therapy- which includes :-

a. Heating the sore to40-42degree C. for several hours each day by infrared light to promote healing .

b. Freezing by CO2 snow .

c. Local infiltration of the lesions by 1-2ml Na. stibogluconate (pentostam) or meglumine antimoniate (one or two injections of few days apart), also Nacl solution 7%, or zinc sulphat solution 2%(Iraqi).

2. Systemic therapy – by Na. stibogluconate or meglumine antimonite by IV or IM injection in a single daily dose of 20mg/kg body weight for 15-21days . **L. aethiopica is** not sensitive to antimony so should be treated with **pentamidine isethionate in dose of 4mg salt/kg**, once weekly for as long as necessary .

For patients with diffuse cutaneous leishmaniasis, treatment for many months beyond clinical and demonstrable parasitic cure was required.

Leishmaniasis recidivans (lupoid), may respond to local infiltration after nodulectomy or systemic antimonies.

Sever scaring may require plastic surgery.

Diseases caused by Arthropods :-

<u>Mechanisms of skin injury by arthropods</u>: Arthropods produces their effects on the skin by a variety of mechanisms, more than one of which may be implicated simultaneously, which includes :

- Mechanical trauma the puncture wound or laceration produced by the penetration of the skin seldom causes serous disturbance to the host. There are two methods of feeding on blood : *vessels feeders ; which insert tip of their mouthparts into capillary , e.g. lice , mosquito , *pool feeders ; which lacerate the skin and damaging the blood vessels and feed on the extravasated blood e.g. flies .
- 2. Injection of irritant, cytotoxic or pharmacologically active substances salivary secretions and sting venoms may contain various enzymes such as hyaluronidase, proteases, peptidases, phospholipases, kinins, histamine liberating agents, histamine, 5-hydroxytryptamine or acetylcholine, which produce local or if sufficient quantity, systemic effects.
- **3. Injection of potential allergens** the vast majority of reactions to arthropods bites or stings depend upon the presence of specific antibodies to antigenic substances in the

saliva or venom, manifested clinically as an itchy papule, weal reaction, or in some patients as anaphylaxis.

- 4. Secondary infections e.g. bacteria infections like impetigo, strep. cellulites.
- 5. Invasion of the host's tissues certain flies causes myiasis (larvae invasion).
- 6. Contact reactions ACD or ICD develops by contact with the secretions, living or dead bodies of certain arthropods.
- 7. Reactions to retained mouthparts as forgin body granuloma.
- **8. Transmission of diseases** arthropods as vectors for many diseases e.g. malaria (mosquitoes), leishmaniasis (sand fly) and typhus (lice).

Myiasis:

Definition- is the infestation of the body tissues of humans and animals by the larvae of *Diptera (flies)*. From **entomological** point of view **myiasis** is classified into 3types: **a. Obligatory-** which always pass their larval stage parasitically in the body of an animal . **b. Facultative which usually** developed on decaying flesh or vegetable matter , but may infest wounds . **c. Accidental- in which** the eggs or larvae of *Diptera are ingested* in food or drink , producing intestinal myiasis . **While clinically** myiasis is classified according to the part of the body affected , there are :- **a. Cutaneous myiasis (wound myiasis and furuncular myiasis)** . **b. Nasopharyngeal myiasis . c. Ophthalmomyiasis . d. Intestinal myiasis . e. Urogenital myiasis .**

Clinical features- the habits of the flies and their larvae determined the variations in the clinical manifestations . **Traumatic or wound** myiasis has been a serious complication of War wounds in tropical areas , and is some times seen in neglected ulcers or wounds in most part of the World .

Obligatory Cutaneous myiasis – occurs in two main clinical forms, in both, there may be mild constitutional symptoms and eosinophilia. **Both** occurs mainly on exposed skin, often **the face**, **scalp**, **arms and legs**. **1. Furuncular form**, **boil-like** lesions developed gradually over a few days, each lesion has a central punctum, which discharges serosanguineous fluid. The posterior end of the larvae, equipped with a group of spiracles, is usually visible in the punctum and its movement may be noticed by the patient. The lesions are often extremely painful, and may be accompanied by lymphangitis and regional lymphadenopathy. Once the larvae has emerged, or has been removed, the lesions rapidly resolve, the flies causing furuncular myiasis in humans are Dermatobia hominis, Cuterebra, Cordylobia, and Hypo derma species.

2. Creeping eruption – in which a tortuous, thread –like red line with a terminal vesicle marks the passage of the larvae through the skin. The larvae lies ahead of the vesicle in apparently normal skin, this form is produced by Gasterophilus larvae.

Treatment – by removal of larvae by expression, surgically or application of mineral oil, petrolatum or butter (Vaseline).

Lice (Phthiraptera) :

Lice are members of the order *Phthiraptera and suborder Anoplura*, they are wingless, dorso-ventrally flattened insects, which are obligate blood-sucking ectoparasites of birds and mammals. The *Phthiraptera* are highly host-specific and spend their entire lives on the host .The Anoplura are vessels feeders (solenophages), introducing their mouthparts directly in to a blood vessels to withdraw blood.

Humans are parasitized by three species of **Anoplura :** *Pediculus capitis (head louse)*, *Pediculus humanus (clothing or body louse)*, *and Pthirus pubis (pubic or crablouse)*. Head lice and clothing lice are morphologically almost identical, and are capable of interbreeding, pthirus pubis is morphologically quite distinct from.

Pediculus (Pediculosis) capitis :

The adult female is a grayish –white insect 3-4mm long, the male is slightly smaller, the claws on the legs are adapted for clinging to hair. During her lifespan of approximately 40days, the female is capable of laying about 300eggs at the rate of 7-10daily, the eggs are cemented to hair shafts with a chitinous cement material secreted by the female's accessory glands, close to the surface of the scalp, they are oval, flash cloured, hatch in about 8days, and the louse nymph reaches maturity in approximately 10days, the empty egg (nit) appears white and easily seen.

Prevalence- it has a worldwide distribution, common both in developed and developing countries, more common in rural areas, and in children between 3-11years age, frequently girls, with long hair. The majority of head louse infections are acquired by direct head to head contact, spread of lice is encouraged by poverty, poor hygiene and overcrowding.

Clinical features- scalp pruritis is the characteristic feature of head louse infection (may be asymptomatic), secondary bacterial infection (impetigo) may occur as a result of scratching. **Pruritic papular** lesions may occur on the nape of the neck, and occasionally a generalized non-specific pruritic eruption develops. The empty egg cases (nits) are easily identified, and occur in greatest density on the parietal and occipital regions, they may be confused with peripilar keratin casts. **Detection** of adult lice and nymphs provides evidence of an active infection, whereas the presence of eggs and egg cases alone merely indicates that infection has occurred at some time.

Treatment :

a. Chemical treatment – which include :

1. Malathion and carbaryl (carbaril) – are acetyl cholinesterase inhibiting insecticides , both are efficient pediculicides and have good , but not complete , ovicidal activity , it is recommended that lotion (alcoholic basis) , and liquid (aqueous basis) formulation of both

should remain on the scalp for 12ho., before being washed off. Hot-air-hair dryer should not be used after their application, because both are degraded by heat, treatment should be repeated after 10dayes, to deal with any nymphs which emerge from the eggs.

2. Pyrethrins synergized with piperonyl butoxide and the synthetic pyrethroids permethrin and phenothrin – lotion and liquid formulation are preferable to shampoos (latter one exposed the insects to relatively low concentrations, which lead to development of resistance). Aqueous basis are less likely to irritate an excoriated scalp, than alcoholic solutions, also do not irritate asthmatics, and are not flammable.

3. USA head louse treatment – a combination therapy containing 1% lindane , 1% permethrin , 0.5% malathion and pyrethrin synergized with piperonyl butoxide , carried out on lice and eggs .

b. Physical treatment : is an alternative to the use of chemical agents :-

1. Bug Busting (wet-combing method) – this technique involves ordinary shampooing of the hair , followed by application of generous amounts of conditioner , and combing using a fine –tooth comb to remove lice , is repeated every 4days for 2weeks . malathion lotion was twice as effective as this method .

2. A battery –powered device (Robi-comb), which kills lice as it used to comb through the hair . Notes- family members should be examined, development of resistance changed to an other insecticides .

c. Other therapies :- 1. Topical crotamiton (escabule) lotion .

2. Topical and oral ivermectin (orally 200ug/kg body weight), as single dose, repeated after 10days, but not ovicidal.

3. Oral co-trimaxazol – which is ingested by the louse and affects its symbiotic bacteria , which are essential for vitamins B synthesis , so the louse cannot survive .

Pediculus (pediculosis) humanus (clothing , body lice , pediculosis corporis) :

It is almost identical in appearance and development to the head louse, it's natural habitant is the clothing of it's host, and it only visits the skin to feed, its eggs are cemented to clothing fibers, with a preference for clothing close to the skin. It is the parasite of individuals whose clothing is rarely changed or washed, because the louse and it's eggs will not survive high temperature washing and ironing, and it is intolerant of temperature changes in it's environment.

Prevalence- it occurs through out the world, although it is now uncommon in developed countries, it is the louse of poverty and neglect, flourished in over crowded, who rarely remove their clothing. In most infected individuals the population is small, but in some there may be thousands of lice. This louse is the vector of epidemic typhus, trench fever and louse –borne relapsing fever.

Clinical features- itching is the principle complaint, which is the result of sensitization to louse salivary antigens, other acquired tolerance to the bites are asymptomatic. Itching causes excoriations and there may be secondary bacterial infections, later on the skin often become hyperpigmented (so called 'Vagabonds' disease, morbus errorum). Lice and eggs should be sought in the clothing.

Treatment – the clothing , not the patient requires treatment . ***Tumble-drying** is the most effective means of killing both lice and eggs , high temperature **laundering of undergarments and dry** cleaning of outer clothing are also effective .

*Malathion dusting powder, and more recently permethrin treated clothing has been shown to be toxic to clothing lice.

***Previously DDT** (dichlorodiphenyltrichloroethane) and **lindane** are used , but lice resistance was developed .

Phthirus pubis (Crab lice , phthiriasis pubis) :

It is quite distinctive, it's body is squat and the second and third pairs of legs carry heavy, pincer-like claws, to grip adjacent hairs close to the skin surface. The eggs are light brown in colour and cemented to the hair of the host, it colonize axillary hair, eyebrows, eyelashes, beard hair, scalp margins and hair on the trunk and limbs, in addition to pubic hair. The crab louse become active at night, when the host is sleeping, has difficulty in moving when taken from its host, where as the head and body lice are quite mobile out off the host. **Phthirus pubis** is a specific parasite of humans, but it's transfer to a **dog** has been recorded. *Prevalence-* crab lice are transmitted by close physical contact, usually sexual, it appears to be a common disorder among sexually active young adults, who are found to be suffering from other sexually transmitted infections (STDS).

Clinical features- itching mainly in the evening and at night, is the principal symptom, close inspection of affected areas will reveal lice grasping hair close to the skin surface, and louse eggs attached to the hair shafts, louse feces are often visible as rust-coloured speckles on the skin and hair, and the under clothes may be spotted with altered blood. **Blue-grey macules (maculae caeruleae)** are occasionally seen on the skin. In children crab lice may colonize the eyelashes and scalp, which is usually acquired by close physical contact with infected parents and occasionally as a result of sexual abuse.

Treatment- malathion, pyrethrins with piperonyl butoxide, pyrethroids and carbaryl, may be used to eradicate crab lice, it is preferable to treat the whole of the trunk and limbs, and the scalp may also require treatment, an aqueous base is preferable. Treatment should be repeated after an interval of 7-10days, all sexual contacts should also be treated.

Eyelashes infection (phthiriasis palpebrarum) is treated by either mechanical removal of lice and eggs with fine forceps or epilation of the lashes with their attached eggs and lice ,

but petrolatum ointment used as a thick application twice/day for 2-3weeks is the treatment of choice , other methods are criotherapy , argon laser .

Bugs (Hemiptera) including bedbugs :

Bedbugs are blood sucking, temporary ectoparasites of birds and mammals, it is 4-5mm in length, with dorsoventrally flattened, oval bodies, the four wings reduced to scale-like pads, and the hind wings are absent, each female lays about 300eggs in her life-time, in the crevices of floors and walls, furniture, bed frames and mattresses, which hatched after about 10days to nymphal stage, which last approximately 6weeks. Bedbugs normally feed at night, usually about an hour before dawn, but they may feed during the day, in the absence of a suitable food supply, however, adult bedbugs can survive starvation, in ideal circumstances for a year or more.

Clinical features- the bites of the bedbugs are painless, but it produces a reaction on the sites, commonly affected areas are the **face**, **neck**, **hands and arms**, but occasionally be generalized. In the first exposure, only a purpuric macule indicates the site of the bite, but in previously exposed, sensitized subjects, intensely irritating weals or papules surmounted by haemorrhagic puncta, and in some cases in which the reaction is sever, bullae predominate. Bedbugs act as a possible vectors for transmission of hepatitis B and HIV

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Bedbugs are one of the causes of papular urticaria .

<u> Papular urticaria :</u>

It is an immediate IgE –mediated reaction, results from injection of forgin protein by biting of insects into skin of the most sensitive subjects, these insects includes : **bedbugs**, **fleas**, **mosquito**, **sand fly and some time sarcoptus scabiaii mite**.

Clinical features- there is a punctum visible on the weal which may blister. Some times the reaction evolves into delayed hypersensitivity reaction leading to intensely itchy, indurated papules lasting weeks or months, with signs of excoriation and some times secondary bacterial infection. It is most commonly seen on the legs of children, also on the forearms, sometimes on the face, during early summer and autium times, usually recurrent for several years.

Treatment- eradication of insects by insecticide (DDT, malathion), **topical steroid mixed** with antibiotic if infection are present, oral antihistamine to control the itching.

Scabies :

Definition- it is parasitic contagious disease of humans and other animals, caused by mites of the family *Sarcoptidae*, which includes *Sarcoptes scabiei* (scabies mite human), and *Notoedres cati* (*a mange mite of cats*, *which cause scabies in other animals*).

Human scabies : it is of two types ordinary and Norwegian scabies .

1. Ordinary scabies :

Morphology – *Sarcoptes scabiei var-hominis* has ovoid body, flattened dorsoventrally, the adult female measures approximately 0.4mm long by 0.3mm broad, and the smaller male 0.2mm long by 0.15mm broad. The body is creamy white and is marked by transverse corrugations, and on its dorsal surface by bristles and spines (denticles), has four pairs of short legs. Copulation occurs in a small burrow excavated by the female, the fertilized female enlarges the burrow and begins egg laying. Eggs and mite feces are deposited behind the female in burrow (seballa), approximately 40-50eggs are laid by each female during lifespan of 4-6weeks, during which time she dose not leave the burrow. After 3-4days the egg hatched, and a six –legged larvae emerge and escape from the burrow, in which they transform into naphs, than maturation to adult males and females. The mites avoid areas with high density of pilosebaceous follicles, the average number of adult female mites on an individual suffering from the common form of scabies is about 12, while in crusted (Norwegian) scabies, a large number of mites are present.

Prevalence and epidemiology- scabies affects all races and social classes worldwide, both sex affected equally, most common in children and young adults and in winter than summer. In developed countries scabies shows cyclic fluctuation, with an interval of approximately 10-15 years, between the end of one epidemic and the beginning of another, this fluctuation suggests that an epidemic of scabies confier a degree of immunity, so that a further epidemic will not occur until a new susceptible population has arisen. **Overcrowding , poverty , poor hygiene and War** encourages the spread of scabies , which is usually transmitted by close physical contact, such as prolonged –hand –holding or the sharing of bed. A way from the host, **scabies mite survive for 24-36hours**, in room conditions (21degree centegrat and 40-80% relative humidity).

Immunology- evidence suggests that both immediate and delayed type hypersensitivity are involved, IgE level may be normal or elevated in many individuals. **Delayed –type hypersensitivity** in the production of inflammatory papules and nodules is suggested by the histological changes and predominance of T-lymphocytes in the Cutaneous infiltrate, other immunological findings includes high serum IgG and IgM and low IgA, with levels returning to normal after treatment, HLA-A11 is higher among patients with scabies, than in normal population.

Clinical features – itching is usually the most obvious manifestation of scabies , generally worst at night and when the patient is warm . The onset occurs 3-4weeks after the infection is acquired , and coincides with a widespread eruption of inflammatory papules , reification of previously cured individuals , however provokes immediate symptoms . The **pathognomonic** lesions of scabies are burrows , which appears as slightly raised , brownish , tortous lesions . The point of entry of the mite , is the most superficial part of the burrow , has slightly scaly appearance , and the distal end there may be a tiny vesicle adjacent to

which is the female mite . There may be few or many burrows and difficult to find in patients with a good standard of hygiene , burrows occurs on the **wrists** , **borders of the hands** , **the sides of the fingers and the finger web spaces** , **the feet** , **particularly the instep** , **and males genitalia** . They are often present on the **palms and soles of the infants** , **elderly and in adult women** , **but less frequently found on the palms in men** , the trunk may be involved in the elderly and in infants , **head and neck in babies** , **scalp was however** involved in an adult who apply a topical steroid for seborrhoeic dermatitis and scalp involvement in ordinary scabies may be reason for relapse . The reason for this distribution of burrows is not understood , but the mites appears to prefer , non-hairy skin and areas of low sebum production .

The pruritic papules that accompany the development of hypersensitivity occurs predominantly around the axillae , periareolar regions , abdomen , particularly the periumbilical region , buttocks and thighs . **Indurated inflammatory nodules** some times occurs , particularly on the axillae , groins , scrotum and penis , which are intensely itchy , and may persist for weeks or months after the scabies has been effectively treated . Inflammatory papules or nodules on the male genitalla are characteristic of scabies and may provide an important diagnostic clue if burrows are difficult to find .

Nail involvement is uncommon in ordinary scabies , but frequent in crusted (Norwegian) scabies , it's presence may be a reason for relapse . In addition to these primary lesions , secondary features may frequently confuse the clinical picture , eczema , secondary infection (impetigo , folliculitis , glomerulonephritis) , bullous pemphigoid –like eruption .

Scabies in infants and young children, in addition to the more extensive distribution of burrows mentioned above, vesicular and vesiculopustular lesions on the hands and feet are frequent with extensive eczematization is often present.

Diagnosis- the typical history of pruritis , with nocturnal exacerbation , and distribution of the eruption , genital lesions in males are pathognomonic , **The absolute confirmation** can only be made by scraped material placed in a drop of 10%KOH , or mineral oil on a microscopic slide , the **presence of mites , eggs , or fragments of egg shells** confirms the diagnosis , **PCR** is the diagnostic tool in difficult situations .

Treatment- there have been many suggested remedies for scabies :

- **1.** Sulpher ointment 10% sulpher in yellow soft paraffin , is in general , safe and effective for adults , daily application for 3-5days , after path or without , which may cause irritant dermatitis (controlled by mixing with steroid) , in infants and young children , a concentration of 2.5-5% may be used .
- 2. Benzyl benzoate it occurs naturally in balsams of Peru , and now is synthesized , employed as a 25% emulsion , it should remain on the skin for 24% hours , two or three applications , either within 24 hours , on successive days or separated by intervals of a week , it is irritant and patients should be warned about overuse .

- **3. Monosulfiram** a 25% solution in industrial methylated spirit is diluted with 2or3parts of water to form emulsion immediately prior to application , as the suspension is unstable , applied once daily for 2-3days , may cause flashing , sweating and tachycardia , monosulfiram soap has been used as a prophylactic , where scabies is endemic .
- 4. Malathion -0.5% in aqueous base, for 24hours, repeated after a week.
- **5. Permethrin-** 5% dermal cream is an effective , scabicidal , washed of after 8-12hours , repeated after an interval of a week .
- **6. Gamma benzene hexachloride (lindane)-** a single application of 1% washed off after 12-24 hours is usually recommended, it is absorbed through the skin, especially if the barrier function of the epidermis is compromised, which may be result in adverse neurological effects, principally seizures.
- **7. Other topical treatments-** includes thiabendazole and crotamiton , both have limited scabicidal activity , and several applications on consecutive days are required .
- 8. Ivermectin is structurally similar to the macrolide antibiotics, but dose not have antibacterial activity, it is active against a number of ecto-andoparasites. Can be used topically or orally and is safe in both children and adult, a single dose of 200ug/kg of body weight will be effective in many cases of ordinary scabies, but presumably because of a lack of ovicidal activity, higher cure rates are obtained with two doses separated by an interval of a week, it is indicated in institutional outbreaks of scabies e.g. in prisoners.

It was suggested that permethrin 5%, is the preferred treatment for scabies in the present time .

Liquid scabicides are most conveniently applied with 2-inch (5cm) pain brush, to the whole body except the head and neck, although the latter should be included if there is clinical evidence of involvement and a non-irritant agent employed. **Advice that itching** will persist for a few days up to 2weeks, **All members** of the family and close physical contacts should be treated, whether symptomatic or not, and ordinary laundering of clothing and bedding, with treatment of secondary infection and eczematization.

Treatment of infants and young children –benzyl benzoate should be diluted with 2or3 parts of water , permethrin cream is the treatment of choice .All scabicides must have been used on **pregnant women , in breast feeding women** , it would appear preferable to stop breast feeding during and for few days after treatment , to allow plasma levels of any percutaneously absorbed scabicide to fall .

<u>Crusted scabies</u> (Norwegian scabies) :

It is firstly described in Norway, so called Norwegian scabies, in which huge number of mites were presented, it's name was replaced by crusted scabies.

Lecture

Parasite

Aetiology and pathogenesis – in common or ordinary scabies there are few mites, probably because scratching destroys the burrows and a good standard of hygiene may also help to control the mite population . In crusted scabies, the host's response to the mites is modified , allowing them to multiply , skin anesthesia secondary to sensory neuropathy or spinal injury, mental retardation, dementia and down's syndrome is frequent association, immunosuppression is also important factor (either due to disease or therapy).

Clinical features- large, warty, crusts form on the hands and feet, the palms and soles may be irregularly thickened and fissured , masses of horny debris accumulate beneath thickened and discoloured nail. Erythema and scaling occurs on the face, neck, scalp and trunk and may be generalized, the extent of the erythroderma and the warty plaques varies greatly and either may predominant, itching is often absent or slight, but may occasionally be sever. Generalized lymphadenopathy and blood eosinophilia are common.

Diagnosis – DD. from hyperkeratotic eczema, psoriasis, Dariers's disease and contact dermatitis, the clinical diagnosis is readily confirmed by examination of scraping for mites and eggs.

Treatment- admission to hospital, treated by topical scabicides, but several applications for prolonged period is often required and incomplete response is not uncommon. Recently ivermectin has become the treatment of choice, either alone or in combination with topical agent, two doses of (200ug/kg), separated by an interval of week and cut the nails shortly.