Skin diseases among schoolchildren in Africa

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Chapter 8

Skin diseases among children in Africa

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This chapter of the thesis reflects the wish of putting acquired knowledge and information into a format which is useful in daily practice in Africa. It describes the epidemiology, etiology and pathogenesis, clinical symptoms and the management and treatment of common and typically tropical skin diseases among children in Africa. The described management of the diseases is experience based. There is little evidence based pharmacotherapy in children.
This chapter is complementary to the book 'Common Skin Diseases in Africa. An illustrated Guide' by Colette van Hees and Ben Naafs (ISBN/EAN: 978-90-808016-2). There is some overlap in text and illustrations. However "Skin diseases among children in Africa" focuses specifically on children, and, in line with the rest of the thesis, on epidemiology. Illustrations were provided by Arjan Hogewoning, Sjan Lavrijsen, Colette van Hees, Ben Naafs, Johan van der Stek and Rosemarie Moser.

This chapter is meant to be a practical guide for general practitioners, health care workers, students and all others who are working in the medical field. The list of skin diseases described is far from complete and will benefit from continuous improvements and additions. Modern communication tools like websites provide these functionalities. We created a freely accessible website named www.africanskindiseases.org to cater for this. "Skin diseases among children in Africa" and "Common skin diseases in Africa" are the first of hopefully many publications accessible through this website.

Skin diseases among children in Africa

Epidemiology-Etiology and Pathogenesis-Clinical Findings-Differential Diagnosis-Management-Clinical Pictures-References

Skin infections

BACTERIAL

Pyoderma
(Impetigo, ecthyma, folliculitis, furuncle)

The majority of the skin diseases found among schoolchildren in Africa are dominated by fungal infections and pyoderma.[1-4] Factors like overcrowding, malnutrition and climatic conditions such as heat and humidity lead to an increase in bacterial infections in tropical and semi-tropical countries.[5;6]

The term pyoderma is used to describe bacterial skin infections; ***impetigo***, ***ecthyma***, ***folliculitis***, ***furuncle*** or ***carbuncle***. It is usually caused by *staphylococci* and/ or *pyogenic streptococci* which may penetrate the skin primarily or secondary to trauma or other infections. Invasive infections may spread from superficial infections or enter through a defect in the skin such as interdigital tinea pedis.[7;8] A problem is the misuse of antibiotics available without prescriptions.

Reasons for concern are recent reports of growing incidences of *S.aureus* bacteraemia coupled with high prevalences of methicillin resistance (MRSA), particularly in HIV-infected children.[9] This growing rate of resistance to currently recommended antibiotics for skin and soft tissue infections could pose a significant health threat in sub-Sahara Africa, especially in regions with limited access to microbiological laboratory facilities and to adequate antimicrobial agents.[10;11]

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Impetigo

Epidemiology

Impetigo is a frequently observed superficial, very contagious, bacterial infection which can be divided in a non-bullous and a bullous form. Non-bullous impetigo accounts for more than 70% of cases of impetigo. It is frequently diagnosed in regions with a warm humid climate. Overcrowding, malnutrition and lack of hygiene also play an important role.

Etiology and pathogenesis

The predominant cause of non-bullous impetigo is *Staphylococcus aureus* although also *Streptococcus pyogenes* can be involved, especially in tropical countries. Bullous impetigo is nearly always caused by a coagulase positive *S. aureus*. These bacteria belong to a specific group (phage group 2) which produces an exfoliative toxin responsible for the blister formation. Phage group 2 *S. aureus* are also responsible for the development of the staphylococcal scalded skin syndrome (SSSS) which occurs mainly in neonates and infants.

Clinical findings

Impetigo usually occurs on exposed areas like the face and extremities. Non-bullous impetigo starts often with a pustule which can develop rapidly and lead to the formation of yellow or brown colored crusts. Usually there is no pain but the lesions may be itchy. In the majority of cases regional lymphadenopathy can be found. Bullous impetigo presents with large blisters which rupture easily. They are usually localized on the face, extremities and the diaper area and they heal without scarring.

Differential diagnosis

- Herpes simplex
- Varicella
- Candidiasis
- Insect bites (hypersensitivity response)
- Pemphigus
- Trauma (thermal)

Management

- Impetigo is highly contagious, spreading needs to be prevented. Do not share the same towels and change clothes and towels frequently.
- In limited cases local therapy is usually sufficient. Wash with betadine shampoo daily and apply gentian violet paint 0.5%, mupirocin ointment, fusidic acid cream, sulphur 5% in zinc oxide cream or betadine ointment twice daily on the lesions.
- In moderate /severe cases an oral antibiotic, active against both *streptococci* and *staphylococci* (also beta-lactamase producing strains) like dicloxacillin is the drug of first choice. In case of penicillin-allergic patients, erythromycin can be given. When MRSA is suspected, cefalexin is an option.

* **Flucloxacillin** (British National Formulary)

- Child under 2 years: quarter of the adult dose: 62.5-125 mg every 6 hours.
  Oral solution (Syrup, flucloxacillin, 25 mg/1mL) 2.5 mL-5 mL 4 times daily.
- Child 2-10 years : half of the adult dose: 125mg-250mg every 6 hours.
  Oral solution (Syrup, flucloxacillin, 25 mg/mL) 5 mL 4 times daily or
  Capsules (flucloxacillin, 250 mg) 1 capsule 4 times daily.
- Child above 10 years: adult dose: 250-500 mg 4 times daily.
  Capsules (flucloxacillin, 250 mg or 500 mg) 1 capsule 4 times daily.

* **Erythromycin** (British National Formulary)

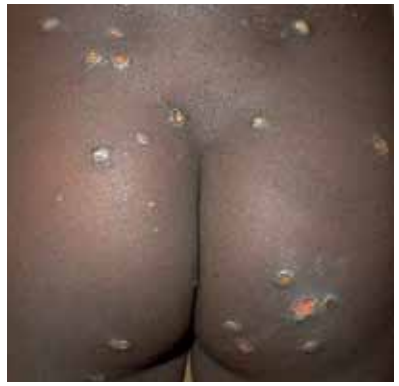
- Child up to 2 years: 125 mg 4 times daily.
  Oral solution (Syrup, erythromycin, **25** mg/1mL) 5mL 4 times daily.
- Child 2-8 years: 250 mg 4 times daily.
  Oral solution (Syrup, **50** mg/1mL) 5 mL 4 times daily or
  Capsules (erythromycin, 250mg) 1 capsule 4 times daily.
- Child above 8 years: adult dose: 250-500 mg 4 times daily.
  Capsules (erythromycin, 250 mg or 500 mg) 1 capsule 4 times daily.

* **Cefalexin** (British National Formulary)

- Child under 1 year: 125 mg every 12 hours.
  Oral solution (Syrup, cefalexin **25** mg/1mL) 5 mL 2 times daily.
- Child 1-5 years: 125 mg every 8 hours.
  Oral solution (Syrup, cefalexin 25 mg/1mL) 5 mL 3 times daily.
- Child 6-12 years: 250 mg every 8 hours.
  Oral solution (Syrup, cefalexin **50** mg/1mL) 5 mL 3 times daily or
  Capsules (cephalexin 250 mg) 1 capsule 3 times daily.
- Above 12 years: adult dose: 250 mg every 6 hours or 500mg every 12 hours.
  Capsules (cefalexin 250 mg) 1 capsule 4 times daily or 2 capsules 2 times daily or 1 capsule (cefalexin 500mg) 2 times daily.

Clinical pictures

Non-bullous impetigo: Small erosions with brown-yellow crusts



Bullous impetigo: Superficial blisters/ erosions

Reference list see Pyoderma

Bacterial folliculitis

Epidemiology

Bacterial folliculitis is mainly diagnosed among children and caused by *S.aureus*.

Etiology and pathogenesis

Folliculitis is an inflammation of hair follicles which, when bacterial, is usually caused by *staphylococci*. Sometimes also other causative agents like *streptococci* or *Pseudomonas aeruginosa* are involved. Minor trauma caused by scratching, physical or chemical injury and the use of topical steroids can induce folliculitis.

Clinical findings

Follicular dome shaped yellow papulopustules are surrounded by a red areola. Lesions develop in crops; the most affected areas are the scalp, thighs and buttocks.

Differential diagnosis

- Insect bites
- *Pityrosporum* folliculitis
- Acne vulgaris
- Folliculitis caused by oily or tar products
- Follicular pustules can also occur in or around a mycotic infection

Management

- Local therapy is usually sufficient. As local treatment wash with betadine shampoo daily and apply mupirocin ointment, fusidic acid cream, sulphur 5% in zinc oxide cream, gentian violet paint 0.5% or betadine ointment twice daily on the lesions.
- Avoid oil and vaseline based topical products.
- Severe or recurrent infections may be treated systemically with oral antibiotics. like flucloxacillin. In case of penicillin-allergic patients, erythromycin can be given. For the dosages see impetigo.

Reference list see Pyoderma

Clinical picture

Folliculitis on a leg

Ecthyma

Epidemiology

Ecthyma describes deeper punched out lesions which can be complicated by lymphangitis and cellulitis. Overcrowding, poor hygiene and malnutrition are important factors in its development. Ecthyma often occurs as a secondary lesion after scratching itchy lesions such as insect bites or after local trauma.

Etiology and pathogenesis

Ecthyma is usually caused by *Streptococcus pyogenes* but may be caused by *Staphylococcus aureus* as well. It occurs mostly on the legs where infection extends into the sub-cutaneous tissue.

Clinical findings

The initial lesion is a blister, surrounded by redness and edema. In the beginning it can resemble impetigo but ecthyma extends into the sub-cutaneous tissue and causes a painful ulcer. It usually heals with the formation of scars.

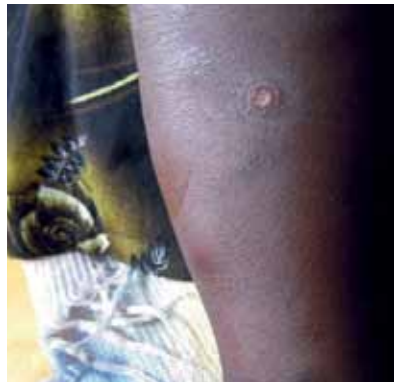
Differential diagnosis

- Impetigo
- Burns
- Ecthyma gangrenosum (caused by *Pseudomonas aeruginosa)*. This usually occurs in patients with immunedeficiency
- Anthrax

Management

- Removal of the crust.
- As local treatment wash with betadine shampoo daily and apply Gentian violet paint, mupirocin ointment, fusidic acid cream, sulphur 5% in zinc oxide cream or betadine ointment twice daily on the lesions.
- A small spectrum antibiotic therapy against *Strep.pyogenes* and *Staph.aureus* is recommended. Phenoxymethylpenicillin can be given. In case of penicillin-allergic patients, erythromycin is a good alternative. For the dosage see impetigo.

Clinical picture



Painful ulcer on the leg

*__Phenoxymethylpenicillin__ (British National Formulary)

- Child up to 1 year: 62.5 mg 4 times daily.
  Oral solution (Syrup, phenoxymethylpenicillin, 25 mg/1mL)
  2.5 mL 4 times daily.
- Child 1- 5 years: 125 mg 4 times daily.
  Oral solution (Syrup, phenoxymethylpenicillin, 25 mg/1mL) 5mL 4 times daily.
- Child 6-12 years: 250 mg 4 times daily.
  Tablets (phenoxymethylpenicillin, 250 mg) 1 tablet 4 times daily.
- Above 12 years: 500 mg 4 times daily.
  Tablets (phenoxymethylpenicillin, 250 mg) 2 tablets 4 times daily.

Reference list see Pyoderma

Furuncle

Epidemiology

A furuncle is a painful abscess around the hair shaft and in the perifollicular skin. Furuncles are more common in boys than in girls.

Etiology and pathogenesis

Furuncles occur in hair-bearing skin. The causative agent is nearly always *Staphylococcus aureus*. Risk factors for the development of furuncles are: a humid environment, obesity or malnutrition, HIV infection and *S. aureus* carriage.

Clinical findings

A furuncle presents as a painful, deep-seated well circumscribed papulopustule which develops into a nodule with central necrosis and pus. Sites of predilection are: the neck, buttocks, groin and armpits. When there is a group of furuncles which form one nodular lesion with multiple drainage points it is called a carbuncle.

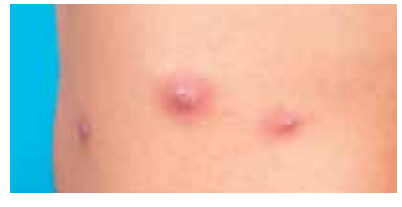
Differential diagnosis

- Hidradenitis suppurativa
- Folliculitis
- Acne vulgaris
- Sinus pilonidalis
- Myiasis

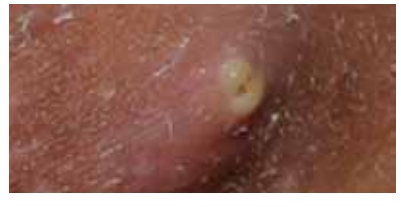
Management

- Frequent application of a moist compress to stimulate drainage.
- Do not share the same towels and change clothes and towels frequently.
- In uncomplicated lesions local therapy is usually sufficient. As local treatment wash with betadine shampoo daily and apply fusidic acid cream, sulphur 5% in zinc oxide cream or betadine ointment twice daily on the lesions.
- Lesions with surrounding cellulitis, or furuncles located on the face demand systemic antibiotic treatment. Flucloxacillin (which is active against beta-lactamase producing strains) is the drug of first choice. In case of penicillinallergic-patients, erythromycin or cefalexin can be given. For dosages see impetigo.
- When furuncles recur and S. aureus carriage is suspected, the patient can be treated with mupirocin nasal ointment, to apply three times daily to the inner surface of each nostril for the first 5 days of each month. In poor resource countries the use of gentian violet paint 0.5% is an option.

Clinical pictures



Caucasian boy 11 years, some furuncles on the adomen



Furuncle, detail

Reference list see Pyoderma

Buruli ulcer

Epidemiology

Buruli ulcer is caused by Mycobacterium ulcerans. It is the third most common mycobacterial disease among humans, after tuberculosis and leprosy. The incidence is highest in children up to 15 years old. Among the younger children males are more infected. Buruli ulcer is endemic in Africa and most patients live in West Africa. In Ghana seasonal variation has been described.[1-3] Environmental factors like deforestation, increased manual agriculture of wetlands, illegal diamond or gold digging etcetera seem to play an important role.[3-7]

Etiology and pathogenesis

Most probably the mode of transmission is by skin trauma at sites contaminated by *M. ulcerans*. The pathway of transmission remains unknown, despite many years of research. The primary risk factor associated with Buruli ulcer is proximity to slow moving water and direct water contact. In arid regions Buruli ulcer is usually absent. *M.ulcerans* contains a plasmid that produces a diffusible necrotizing toxin in tissues, mycolactone, which gives the ulcers the typical undermining aspect.

Clinical findings

Buruli ulcer is a necrotizing skin disease that can leave patients with prominent scars and lifelong disability. After infection a painless nodule is formed which eventually ulcerates. This process evolves very slowly, and large body areas may eventually be affected. Despite their impressive appearance the lesions are strikingly painless and patients are usually otherwise healthy. There are several types of lesions:

- I Small early lesion (eg, nodules, papules, plaques, ulcers < 5 cm in diameter)
- II Non ulcerative and ulcerative plaque and edematous forms
- III Large ulcerative lesions (>5 cm in diameter)

Besides the skin and the subcutis deeper structures may be affected, leading to osteomyelitis and bone destruction. [8-10] [11]

Differential diagnosis

- Other tropical ulcers[12]
- Leishmaniasis
- Cutaneous tuberculosis
- Onchocerciasis nodules
- Fungal skin infections.

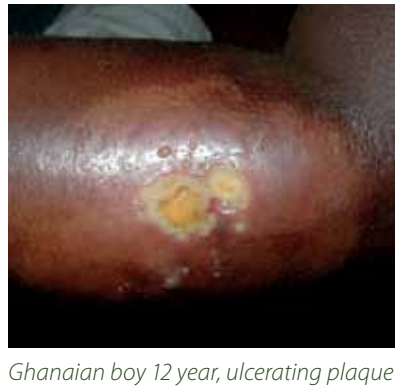
Management

- In the recent past excision was the treatment of choice but now serves more as an adjunct to antibiotic treatment.[13-15]
- A combination of rifampicin and streptomycin for 8 weeks should be given. Rifampicin, 10 mg/kg body weight by mouth daily for 8 weeks and streptomycin, 15 mg/kg body weight by intramuscular injection daily for 8 weeks. Because its side effects (ototoxicity and nephrotoxicity) streptomycin is more and more replaced by clarithromycine, ciprofloxacin, moxifloxacin or amikasin.
- If surgery is combined with antibiotic therapy only minimal surgery to excise necrotic tissue is required when antibiotics have arrested progression of the disease.
- Interventions to minimize or prevent disabilities.
- BCG Vaccination programmes, though the protective effect is short-term and according to some studies non existing[13]
- The treatment depends on the different clinical categories.

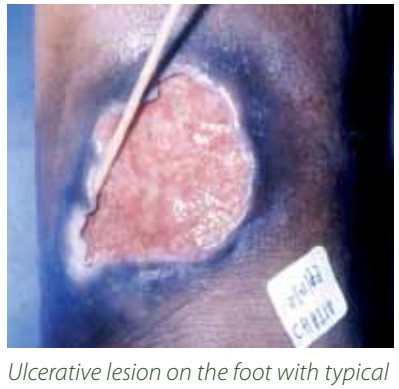
* Category I (small early lesion), if possible a direct excision and suturing is recommended. Antibiotics should be started at least 24 hours before surgery and continue for 4 weeks. If surgery is not possible all lesions in this category can be treated with antibiotics for 8 weeks. Category I can be treated in smaller clinics / primary health care centers and referral hospitals.

* Categories II and III should be treated with antibiotics for at least 4 weeks, then surgery (if necessary), followed by another 4 weeks of antibiotics. Both categories should be treated in a district or tertiary health care facility. (see: http://www.who.int/buruli/information/antibiotics/en/.)

Clinical pictures



Ghanaian boy 12 year, ulcerating plaque



Ulcerative lesion on the foot with typical "undermining"

Clinical picture

Ghanaian boy, 12 years old
Large ulcerative lesion with undermining

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Leprosy

Epidemiology

The newly detected number of patients (NCD) with leprosy in 2010 was 228.474, which is about 50% of the NCD in 1985. Up to 10% of new leprosy cases occur in children under 15 years.[1;2] This means that even though elimination strategies have had a positive effect, leprosy is still endemic in South East Asia, South America and Africa, India and Brazil being the most affected. An explanation may be that contagious patients are not discovered in time.

Etiology and pathogenesis[3]

Leprosy is an infectious and immunological disease caused by *Mycobacterium leprae*. It is transmitted by leprosy patients who may carry many bacilli, particularly multibacillary patients, usually by sneezing or coughing. Of those infected only few develop leprosy. Leprosy is a generalized disease which especially affects skin and nerves. The clinical presentation and damage done depend on host immunity. Skin and nerve involvement and damage may occur by infiltration with *M. leprae,* or in particular during leprosy reactions, which may occur before, during or after treatment.

Clinical findings[4-6]

In paucibacillary (PB) leprosy there is strong cellular immunity. Five or less well demarcated hypopigmented or slightly erythematous skin patches with loss of sensation are seen on the skin and no bacilli are found in the patches. One or more local or regional nerves may be enlarged. In multibacillary (MB) leprosy there are more than five skin lesions which may be flat, popular, nodular or plaques. In total absence of a cell mediated immune response the whole skin may be infiltrated (Lepra bonita). MB patients have positive skin smears and are contagious.

Leprosy reactions may cause severe nerve damage if not recognized and treated properly. Symptoms of reversal reactions (RR) are erythema and swelling of previous lesions, appearance of new lesions or enlargement, tenderness and loss of function of nerves.[7] Sometimes there is acral edema. In erythema nodosum leprosum (ENL) tender erythematous nodules appear, nerves may become tender and the patient usually feels sick. Other organs may be affected too, causing for example arthritis, lymphadenitis, orchitis and iridocyclitis. Ulceration is secondary to the loss of protective sensation and may lead to cellulitis, deep infections, osteomyelitis and consequently loss of digits, causing deformity.

Differential diagnosis

- Tinea corporis
- Lupus vulgaris
- Atypical mycobacterial infection
- Leishmaniasis

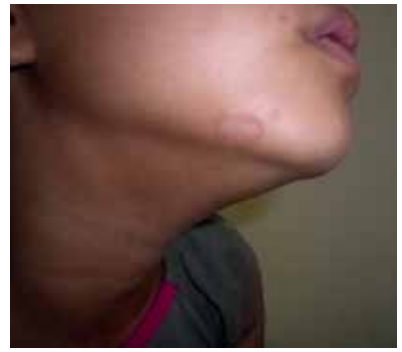
- Neurofibromatosis
- Sarcoidosis
- Pityriasis versicolor
- Granuloma annulare
- Vitiligo
- Erythema nododum
- Yaws
- Kaposi sarcoma

Management of uncomplicated leprosy as advised by the WHO

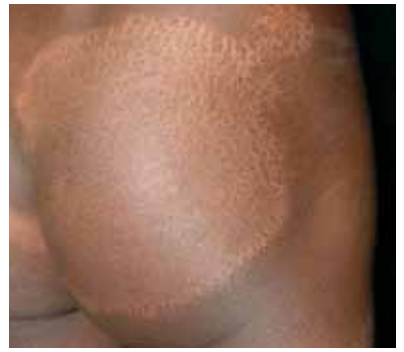
- PB leprosy, children under 10 years: Rifampicine 300 mg once a month under supervision plus dapsone 25 mg daily unsupervised for 6 months (sometimes 12 months treatment may be needed), 6 monthly doses in 9 months are considered enough.
- PB leprosy, children 10-14 years: Rifampicine 450 mg once a month under supervison plus dapsone 50 mg daily unsupervised for 6 months (sometimes 12 months treatment may be needed), 6 monthly doses in 9 months are considered enough.
- PB leprosy above 14 years: Rifampicine 600 mg under supervison plus dapsone 100 mg daily unsupervised for 6 months (sometimes 12 months treatment may be needed), 6 monthly doses in 9 months are considered enough.
- MB leprosy children under 10 years: Rifampicine 300 mg and clofazimine (lampren) 100 mg under supervision monthly plus dapsone 25 mg daily and clofazimine 50 mg twice a week unsupervised for 12 months. (sometimes 24 months treatment may be needed), 12 monthly doses in 18 months are considered enough.
- MB leprosy children 10-14 years: Rifampicine 450 mg and clofazimine (lampren) 150 mg under supervision plus dapsone 50 mg daily and clofazimine 50 mg every other day unsupervised for 12 months. (sometimes 24 months treatment may be needed), 12 monthly doses in 18 months are considered enough.
- MB leprosy above 14 years (50-80 kg): Rifampicine 600 mg and clofazimine (lampren) 300 mg plus dapsone 100 mg plus dapsone 100 mg daily and clofazimine 50 mg daily unsupervised for 12 months (sometimes 24 months treatment may be needed), 12 monthly doses in 18 months are considered enough.
- Single lesion PB children 5-14 years: Rifampicin 300 mg, Ofloxacin 200 mg, Single lesion PB adults: Rifampicin 600 mg, Ofloxacin 400 mg, Minocyclin 100 mg
- Minocyclin 50 mg (not recommended under age 5).
- In younger children treatment regimens should be adjusted according to age and weight.
- Always check for reactions and complications, particularly haemolysis in Northern and Western Europeans.
- RR: prednisolon 0,5 mg/kg daily, tapering down slowly but remaining above 0,25 mg/kg/day for 3-6 months according to clinical signs and symptoms, then taper down to zero in 2 months.

- ENL: Mild ENL: acetylsalicylic acid 1000 mg 3 times daily (or less according to age) for 1-2 weeks. Severe ENL: prednisolone high dose eg 1-1.5 mg kg for two days, tapering off in two weeks. To be repeated if necessary.

Clinical pictures



Girl with TT leprosy, hypopigmented patch with loss of sensation



11 year old boy with BT leprosy

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Skin infections

FUNGAL

Tinea capitis

Epidemiology

Fungal infections of the scalp (tinea capitis) are endemic among schoolchildren in tropical Africa and they can cause significant public health problems. The prevalence of tinea capitis is higher among schoolchildren in rural schools and schools with a lower socioeconomic status.[1-5]

Etiology and pathogenesis

It is an infection of the hair shaft on the scalp, which may be caused by *Trichophyton* and *Microsporum* species. It is predominantly a disease of prepubertal children and the incidence of *Microsporum* species is higher in boys than in girls. The causative agent of tinea capitis varies with geography, socioeconomic status and time.[6]

Antropophilic infections like *Trichophyton tonsurans, violaceum*, *sudanense and Microsporum audouinii*, are most prominent in Africa.

Clinical findings

The clinical appearance can vary from scaling (diffuse scaling with discrete patches of hair loss), hair loss, black dots and sometimes pustules, nodules to massive purulent secretion (Kerion).

Late detection and lack of treatment can result in widespread infections and, in rare cases, permanent alopecia. Because the fungus has grown into the hair follicle, systemic treatment is necessary.[7;8]

Differential diagnosis

- Seborrheic dermatitis
- Atopic dermatitis
- Tinea amiantacea
- Psoriasis
- Alopecia areata
- Trichotillomania
- CDLE
- Pyodermia

Management

- Oral antifungal treatment is always indicated

*** Griseofulvin**

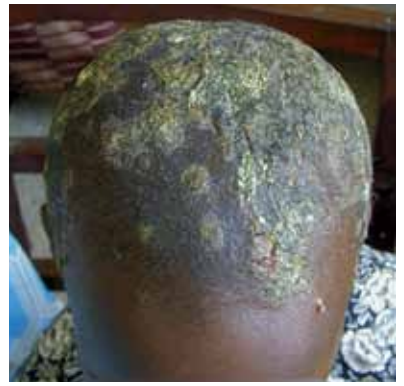
- The dose is based on body weight and is usually 20 mg/kg of body weight once a day for 6-8 weeks. Oral solution (Syrup, griseofulvin microcrystalline, 25 mg/ml) or tablets (griseofulvin 125 mg or 500mg).
- Dosing recommendations have not been established for children < 2 years of age.
- Children above 30 kg 500 mg daily for 6-8 weeks.
- For tinea capitis Griseofulvin is the treatment of choice.

***Terbinafin**

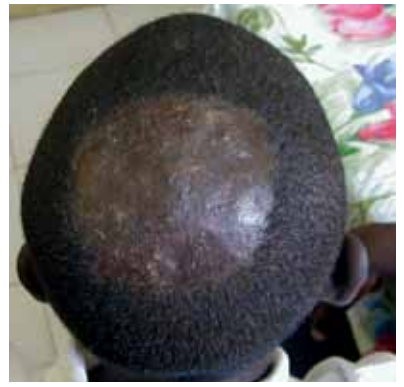
- In certain countries not approved for children below 2 years of age.
- The dose is based on body weight.
- Children below 20 kg 62.5 mg daily for 6 weeks. (Syrup, terbinafin, 25 mg/ml), 2.5 ml Syrup daily or tablet (terbinafin 250 mg), ¼ tablet daily.
- Children between 20 and 40 kg 125mg daily for 6 weeks. Oral solution (Syrup, terbinafin, 25 mg/ml) 5 ml daily or tablets (terbinafin 250 mg) ½ tablet daily.
- Children above 40kg and adults 250 mg daily for 6weeks. Tablets (terbinafin 250 mg), 1 tablet daily.
- To prevent shedding, apply Whitfield's cream or miconazole cream twice daily topically, preferably after shaving or use anti-fungal shampoo (like 2% ketoconazole or 2.5% selenium sulfide).

Clinical pictures

Scaling and hair loss



Secondary infection



Kerion and possible permanent alopecia

- Infected siblings and friends of affected children should also be treated.
- Appropriate adjunctive treatment for household contacts includes daily use of an antifungal shampoo.
- In case of a secondary bacterial infection oral antibiotics like cloxacilline or erythromycine can be given. For dosages see impetigo.

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Tinea corporis

Epidemiology

Superficial fungal infections ("ringworm") of the skin are common in sub-Sahara Africa, especially on exposed skin surfaces, though tinea corporis is less common than tinea capitis and pedis. It is found mostly in rural areas.[1;2]

Etiology and pathogenesis

Zoophilic fungal infections like *Microsporum canis* and *Trichophyton* verrucosum normally present on the exposed surfaces of the body like the face, arms and shoulders. On the trunk and the legs antropophilic infections like *Trichophyton tonsurans, violaceum, sudanense and Microsporum audouinii*, which are most prominent in Africa, are more frequently found.[3;4]

Clinical findings

Tinea corporis presents as typical round lesions with central healing, hair loss and scaling on the edges. They can be large and widespread, due to lack of treatment or in case of immunosuppression. The clinical and social impact of fungal infections on individuals varies with local conditions.[5-7]

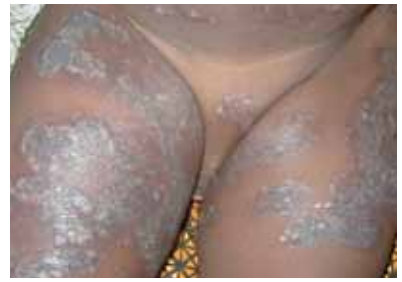
Differential diagnosis

- Eczema
- Pityriasis versicolor / rosea
- Granuloma annulare
- Psoriasis
- Acne vulgaris
- Leprosy

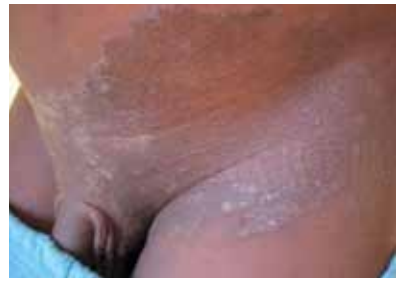
Management

- Application of an imidazole cream or Whitfield's ointment twice daily for 6 weeks.
- In case of large and multiple lesions oral treatment with griseofulvin or terbinafin is preferred during 2-4 weeks. See for the dosages tinea capitis.

Clinical pictures



Widespread round lesions



Localized lesion, central healing and scaling on the edges

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Tinea pedis (Interdigital type)

Epidemiology

Hot, humid climate and a changing, more western lifestyle of wearing closed shoes makes tinea pedis an increasing problem among African schoolchildren, especially in urban areas. The prevalence rate is still low.[1;2]

Etiology and pathogenesis

Trichophyton rubrum, mentagrophytes and *Epidermophyton floccosum* account for most cases of tinea pedis. In a tropical environment *Hendersonula toruloidea* is also frequently involved. Interdigital infections are often mixed infections of the above mentioned fungi and bacteria (*Nocardia minutissima*) which can cause erythrasma. Dermatophyte infection provides a portal of entry which may lead to bacterial infection with *Streptococci or S.aureus*.[3;4]

Clinical findings

Tinea pedis or athlete's foot causes cracking, maceration and inflammation with itching between the toes, most commonly between the 4th and 5th toe.[5]

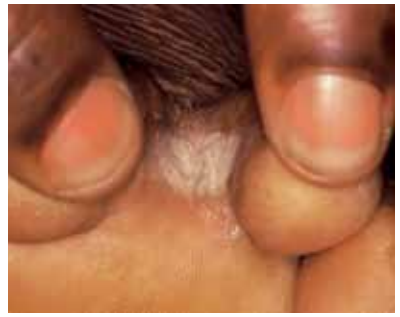
Differential diagnosis

- Erythrasma
- Bacterial infection
- Eczema (dyshidrotic or contact allergic)

Management

- Topical treatment is always necessary.
- An imidazole containing cream, ciclopirox cream twice daily or terbinafine cream once daily for 6 weeks or longer, until a week after the symptoms subside. The web spaces between the toes should be kept dry, especially after washing. Also cotton socks should be used and changed daily.
- When the complaints are often recurring, the inderdigital spaces can be treated twice weekly with the above mentioned creams as prophylaxis.
- Oral antifungals alone are usually ineffective because topical treatment is essential and infections are often mixed.
- Children should be advised to wear well ventilated shoes.
- If there is a superimposed bacterial infection, topical antibiotic treatment can be applied like Gentian violet paint 0.5%, mupirocin ointment or betadine ointment twice daily on the lesions. In severe cases oral antibiotics can be given like cloxacillin or erythromycin. For dosages see impetigo.

Clinical picture



Typical white macerated lesions of Athlete's foot

Reference List

1. Hogewoning A.A., et all. Skindiseases among schoolchildren in Ghana,Gabon and Rwanda. August 2012. Accepted for publication in the *International Journal of Dermatology*
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Pityriasis versicolor

Epidemiology

Pityriasis versicolor is a chronic benign fungal infection frequently seen among young adults, more commonly in a tropical environment.[1;2]

Etiology and pathogenesis

Pityriasis verscicolor is caused by the yeast *Malassezia* which is a normal resident of the skin and is usually asymptomatic.[3] In favorable circumstances such as a hot and humid climate, and / or sweating the infection becomes symptomatic.

Clinical findings

Clinically it is characterized by well-defined scaly hypo-or hyper pigmented patches primarily affecting the upper trunk, neck or upper arms, in areas with active sebaceous glands.[4] In longstanding disease the patches become confluent and may cover large

areas. After treatment hypopigmented macules without scaling may persist but these will disappear after sun exposure.

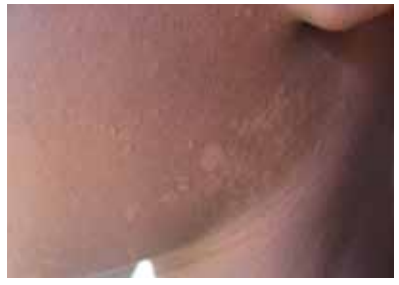
Differential diagnosis

- Vitiligo
- Pityriasis alba
- Sarcoidosis
- Epidermodysplasia verruciformis
- Verrucae planae (in a HIV+ patient)

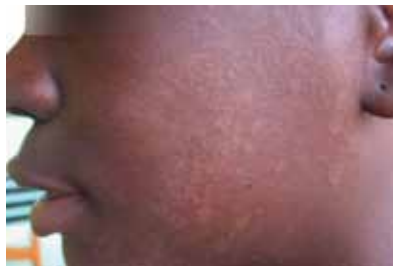
Management

- Avoid the use of vaseline, olive oil and other greasy products.
- Ketoconazol, miconazol or terbinafin cream twice daily on the lesions for 3 weeks.
- Apply selenium sulphide shampoo as a lotion on the whole body overnight, wash off in the morning and wash the scalp extra.
- Selenium sulphide shampoo or ketoconazole 2% shampoo daily for 7 days or twice weekly for 4 weeks. The shampoo should be left on the skin for at least 15 minutes before being rinsed off.
- Salicylic acid 5% + sulphur 5% ointment during the night for 4 weeks.
- Recurrences can be prevented by once monthly preventive treatment with any of the above mentioned medications.
- Because of the risk of hepatotoxicity and the high recurrence rate in the tropics oral treatment should be avoided among children.

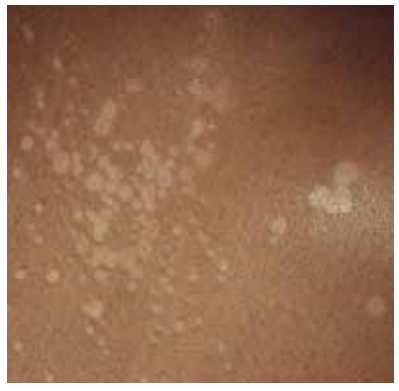
Clinical pictures



Hypopigmented macules



Hypopigmented patches with fine scaling



Hypopigmented patches with fine scaling

Reference List

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Skin infections

VIRAL

Verrucae vulgares

Epidemiology

Common warts are caused by a small group of Human Papilloma Virus types. They penetrate the skin after skin to skin contact or through contaminated surfaces and objects (e.g. at home, public showers, swimming pools). Prevalences of 20% are reported among schoolchildren in industrialized countries although the prevalences found in most community based studies in sub-Sahara Africa are much lower.[1-5]

Etiology and pathogenesis

Papillomavirus infect squamous epithelia of the skin and mucous membranes in most vertebrate species. Many types of HPV have been identified and are associated with various clinical lesions. HPV types 1, 2 and 4 infect the skin and induce common warts. They are found at any age but are most common in teenagers. The extent of lesions is determined by the immune status of the host.[6]

Clinical findings

The lesions are discrete, round papules and nodules with verrucous surface. They can be small papules (1-10mm) or large plaques. Sometimes the lesions become confluent and form a mosaic. In the majority of patients with a normal immune system warts will disappear spontaneously within several months to years. Treatment is sought for when lesions are painful (eg on the soles) or unsightly but is not always necessary. Treatment results are unpredictable and often disappointing. Warts may spread fulminantly and persist indefinitely.[7;8]

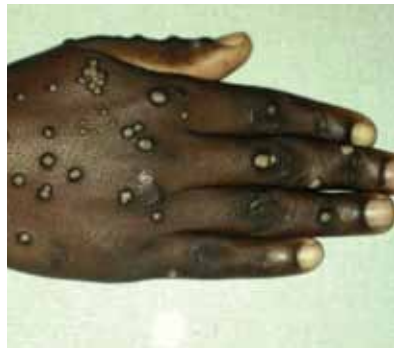
Differential diagnosis

- Lichen planus
- Psoriasis
- Plantar callus (corns)
- Mollusca contagiosa
- Verrucous tuberculosis

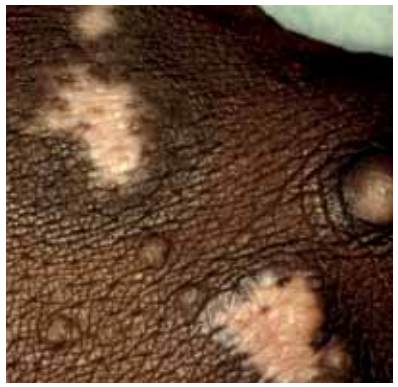
Management

- Apply salicylic acid 25% ointment daily (possibly under occlusion) and cut the warts with a razorblade. Repeat this for weeks to months.
- Apply trichloro or monochloro acetic acid.
- Freeze with liquid nitrogen. Warn the patient for post-treatment hypo or de pigmentation which is usually temporary. (see picture 2).
- Curettage (after local analgesia with Emla cream).
- Laser only in specialized centers.

Clinical pictures



Hyperkeratotic papules and nodules



Depigmentation after cryotherapy

The differential diagnose with Lichen planus is not always easy. ...

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Mollusca contagiosa

Epidemiology

Mollusca contagiosa are frequently seen in children under the age of 5 years which can be the reason of a low prevalence found among schoolchildren in sub-Sahara Africa.[1-3] They can be localized anywhere on the body but are often seen in areas of warmth, moisture and friction such as the armpits and groins. In cooler climates the infection seems to be more common at a later age. The use of public swimming pools has been correlated with childhood infections.[4]

Etiology and pathogenesis

It is a common cutaneous infection caused by a pox virus and can affect both children and adults. The virus can be transmitted directly from person to person or by autoinoculation, the incubation time can vary from weeks to months. In adults it is regarded as a sexually transmitted infection and one should consider the possibility of co-existent HIV infection. Therapy is not always necessary but may be beneficial in preventing transmission or autoinoculation.[5-7]

Clinical findings

Pearl-like, dome shaped nodules with a dimple on top can be seen, the diameter varies from 5 to 10 mm. If squeezed a white/yellow greasy mass comes out of it. Sometimes a single lesion can be seen but normally there are several and sometimes hundreds. Most cases are self-limiting within 6-9 months.

Differential diagnosis

- Verruca vulgaris
- Milium
- Histiocytoma
- Keloid
- Adenoma sebaceum
- Cryptococcosis
- Tricholemmoma

Management

- Most treatment options are mechanical, sometimes causing discomfort but in the majority of cases therapy is not necessary and natural resolution can be awaited.
- Curettage with a sharp curette after applying 1% iodide tincture. Local anesthesia can be accomplished after application of Emla cream during 30 minutes.
- Cryotherapy with liquid nitrogen to be repeated every 3 weeks.
- Prick the center with a toothpick and press out the contents.
- Apply 50-88% trichloro acidic acid.
- Apply retinoid cream / tincture 0.05-0.1% 2 times daily.

Clinical picture

Little boy with "pearl" like nodules

Reference List

1. Hogewoning A.A., et all. Skindiseases among schoolchildren in Ghana,Gabon and Rwanda. August 2012: Accepted for publication in the *International Journal for Dermatology*
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Varicella / Chickenpox

Epidemiology

Varicella zoster virus (VZV) has a worldwide distribution, 98% of the adult population is seropositive. The first manifestation of a VZV infection is varicella (chickenpox). Varicella affects 90% of unvaccinated children under 10 years of age and less than 5 % over 15 years. Several point prevalence studies in Africa showed low percentages but epidemics occur seasonally.[1-3] It predisposes to the development of herpes zoster later in life. Immunization reduces the incidence of herpes zoster markedly.[4-6]

Etiology and pathogenesis

Varicella is very contagious and is spread by airborne droplets or direct contact with vesicular fluid. After primary infection it moves from cutaneous and mucosal lesions to dorsal root ganglion cells. From there it can be reactivated in a later stage.[7]

Clinical findings

Prodromes of primary varicella vary from mild fever to general malaise and are followed by multiple pruritic, erythematous papules and vesicles which become pustules and hemorrhagic crusts. From the scalp and face they spread to the trunk and extremities. Any numbers of vesicles varying from a few to several hundreds are seen in all stages of development at the same time. Itch is the major complaint and scratching may lead to secondary infection. The disease is normally self- limiting and lesions heal in 7 to 10 days. Common complications are secondary infection with scarring and pneumonia. In immunocompromised patients varicella can lead to severe morbidity and even death (see picture 3).[8]

Differential diagnosis

- Disseminated herpes simplex infection
- Disseminated herpes zoster infection
- Hand, foot and mouth disease
- Insect bites and scabies
- Bullous impetigo
- Pityriasis lichenoides et varioliformis acuta (PLEVA)

Management

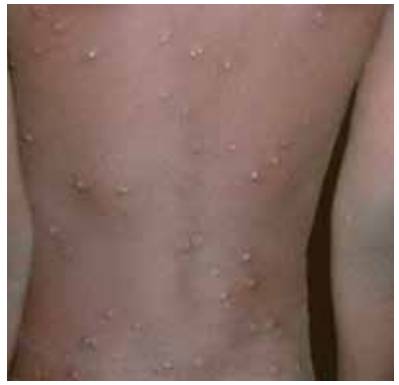
- Calamine lotion or phenol-zinc lotion as necessary for itch and drying in.
- Sedating oral antihistamines like piriton and phenergan. For the dosages see urticaria.
- Limited secondary infection: use betadine scrub, apply betadine ointment, fucidin cream or ointment, mupirocin ointment, sulphur 5% in zinc oxide cream or gentian violet paint 0.5% 2 times daily.
- Severe or widespread bacterial secondary infections can be treated with systemic antibiotics like cloxacillin or erythromycin. For the dosages see impetigo.

- Immunization has 80% effectivity. Recommended for HIV+ children or children on HAART.[9]
- In immunocompetent children oral medication with aciclovir is only indicated in severe infections.
- Immunocompromised children should be referred to a specialist.

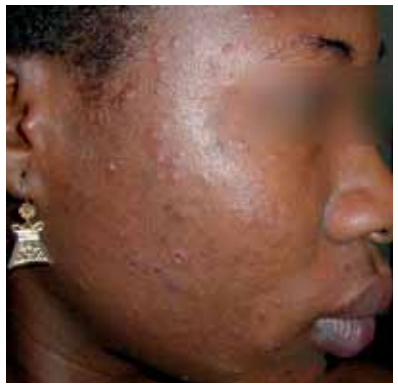
***Aciclovir**

- Child under 2 years: 200 mg 5 times daily for 5 days. Oral suspension (Syrup 40mg/mL or 80mg/mL) 5 ml (40mg/ml) 5 times daily for 5 days.
- Children between 2-5 years: 400mg 5 times daily for 5 days. Oral suspension (Syrup 40mg/mL or 80mg/mL) 5 ml (80mg/ml) 5 times daily for 5 days or tablets 400 mg 5 times daily for 5 days.
- Children above 6 years of age 800 mg 5 times daily for 5 days. Tablets 400mg 2 tablets 5 times daily for 5 days.

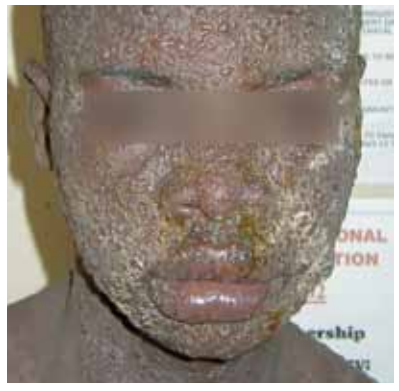
Clinical pictures



Widespread lesions on the back. The different stages of lesions, arising over 7 to 10 days, is typical of varicella.



Multiple, pruritic, papules, vesicles and pustules on the face of a young female. The different stages of lesions, arising over 7 to 10 days, is typical of varicella.



This HIV positive boy died two days later due to a generalized infection.

***Valaciclovir**: The dosage which is recommended in pediatric patients who are at least 2 years old to less than 18 years is 20 mg/kg administered 3 times daily for 5 days. The total dose should not exceed 1 gram 3 times daily.

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Herpes zoster

Epidemiology

Varicella Zoster Virus (VZV) can produce two different clinical manifestations, varicella (chickenpox) and herpes zoster (shingles). The point-prevalence of both skin diseases found among schoolchildren in sub-Sahara Africa was low,[1;2] most probably because affected children tend to stay at home. Chickenpox is primarily a disease of children and shingles a disease of adults but they may both occur at any age. VZV is distributed worldwide and 98 % of the adult population is seropositive. These figures become lower after vaccination campaigns.[3;4]

Each person with a history of varicella has approximately 20% chance of acquiring shingles in his/her lifetime. These figures are much higher in those infected with HIV.[5-7]

Etiology and pathogenesis

During the course of a primary varicella infection the VZV spreads from the skin and mucosal lesions into the sensory nerve endings. Reactivation of the VZV may occur spontaneously or may be triggered by fever, trauma, stress or immunosuppression. It can spontaneously lead to a clinical herpes zoster which is usually more severe in young children than in adults. Herpes zoster is more severe in the immune suppressed. [6;7]

Clinical findings

Herpes zoster can be preceded by a severe itchy, burning pain sensation in the involved dermatome. This prodrome may also consist of fever, headache and general malaise. The rash which develops within a sensory dermatome starts with erythematous macules and papules which later progresses to vesicles, pustules and crusts. There may be secondary bacterial infection. Especially in Africans the infection can lead to the formation of keloids but this is uncommon among immunocompetent children. Herpes zoster can be complicated by post herpetic neuralgia (rare among children). Herpes zoster of the ophthalmic branch of the facial nerve may be complicated by keratoconjunctivitis and lead to blindness, therefore ophthalmologic care should be sought.[5;6;8]

Differential diagnosis

- Insect bites
- Papular urticaria
- Herpes simplex (especially in the genital area)
- Contact dermatitis
- Localized bacterial or viral infections

Management

In a healthy child usually analgesics and local therapy are sufficient.

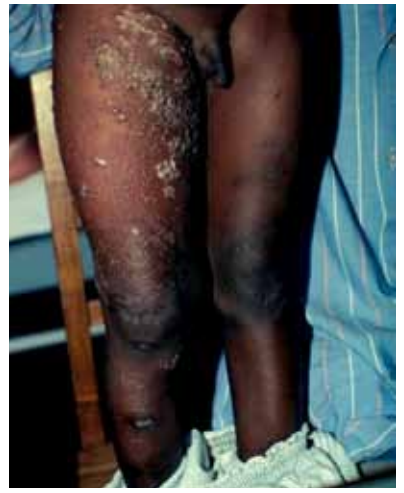
- Calamine or phenol-zinc lotion for vesicular stage.Gentian violet paint 0.5% may be used as well.
- Use betadine scrub/shampoo as a soap to prevent secondary infection. Do not use vaseline.
- In case of a secondary infection oral antibiotics like cloxacillin or erythromycin can be given. For the dosages see impetigo.
- Refer to the eye specialist when the eye is involved.
- In immunocompetent children oral medication is only indicated in severe infections.
- Immunocompromised children can better be referred for treatment with aciclovir or valaciclovir.

*Aciclovir

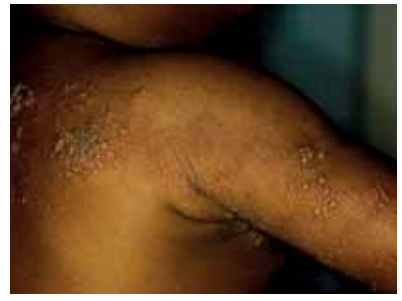
- Child under 2 years: 200 mg 5 times daily for 5 days. Oral suspension (Syrup 40mg/mL or 80mg/mL) 5 ml (40mg/ml) 5 times daily for 5 days.
- Children between 2-5 years: 400mg 5 times daily for 5 days. Oral suspension (Syrup 40mg/mL or 80mg/mL) 5 ml (80mg/ml) 5 times daily for 5 days or tablets 400 mg 5 times daily for 5 days.
- Children above 6 years of age 800 mg 5 times daily for 5 days. Tablets 400mg 2 tablets 5 times daily for 5 days.
- *Valaciclovir: The dosage which is recommended in pediatric patients who are at least 2 years old to less than 18 years is 20 mg/kg administered 3 times daily for 5 days. The total dose should not exceed 1 gram 3 times daily.

Clinical pictures

Multiple vesicles and open lesions on a leg of a 3 year old boy



Multiple vesicles following a dermatome



Herpes zoster of the shoulder

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Herpes simplex

Epidemiology

Herpes simplex infections commonly take place in early childhood and are often asymptomatic. The clinical prevalence among schoolchildren is low. More than 60% of infected children remain carriers of the virus. Percentages of 90% antibodies against herpes simplex virus 1 (HSV1) have been found among young adults worldwide. The percentage of antibodies against HSV2 is much lower before adolescence as it is usually sexually transmitted. Minor epidemics of HSV1 may occur among schoolchildren or in nurseries.[1;2]

Etiology and pathogenesis

HSV1 is usually transmitted by direct contact with saliva. After an often asymptomatic infection viral elements persist as a latent infection for life. Reactivation occurs during periods of immunosuppression like fever, stress, exposure to UV light, HIV infection, menstrual period or infectious diseases like malaria.[3] Recurrent herpes simplex is common and may be the result of an endogenous reactivation or an exogenous reinfection.[1;4]

Clinical findings

Most infections are asymptomatic. Symptomatic primary infections in children often present as a gingivostomatitis together with an increased salivation and difficulty in eating. On the lips multiple painful, burning vesicles appear and develop into pustules and ulcerations. In recurrent infections the clinical picture is usually much milder.[1]
In HIV positive children the symptoms are more severe and the attack rates are higher but this improves after initiation of HAART.[5;6]
In cases of genital herpes in young children sexual abuse should be considered.

Differential diagnosis

- Angina
- Aphthous stomatitis
- Oral candidiasis
- Fixed drug eruption

Management [7]

In case of a primary infection analgesics are indicated.

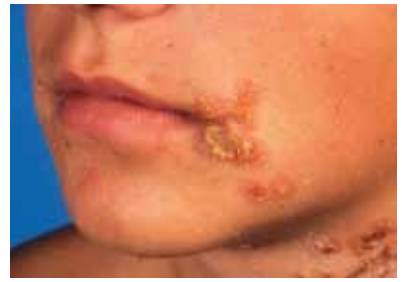
- In recurrent infections: A lip cream / stick with a sun blocker daily to prevent recurrences.
- In immunocompetent persons oral medication with aciclovir is only indicated in severe primary infections or very frequent recurrences. For dosages see varicella/chicken pox.
- In the immunesuppressed oral (val)aciclovir is recommended.
- Warn for meningitis.

***Aciclovir** (British National Formulary)

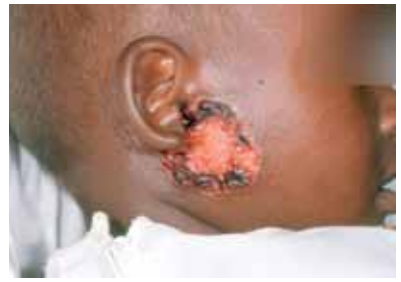
- Child under 2 years: 200 mg 5 times daily for 5 days. Oral suspension (Syrup 40mg/mL or 80mg/mL) 5 ml (40mg/ml) or 2.5 ml (80mg/mL) 5 times daily for 5 days.
- Child over 2 years adult dose: 400mg 5 times daily for 5 days.
  Oral suspension (Syrup 40mg/mL or 80mg/mL) 10 ml (40mg/ml) or 5ml (80mg/mL) 5 times daily or tablets (200mg) 5 times daily for 5 days.

***Valaciclovir**: The dosage which is recommended in pediatric patients who are at least 2 years old to less than 18 years is 20 mg/kg administered 3 times daily for 5 days. The total dose should not exceed 1 gram 3 times daily.

Clinical pictures



Herpes simplex with secondary impetigenisation in a caucasian boy



Secundary infected Herpes simplex lesion in a HIV+ child

Herpes simplex infection of index finger in a small child

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Skin infections

PARASITIC

Cutaneous Leishmaniasis

Epidemiology

Leishmaniasis in its various forms is present on all continents except Australia and Antartica.[1] It is a widespread disease with 350 million people at risk. There are around 2 million new cases a year of which 500,000 cause visceral and 1,500000 cause (muco) cutaneous disease.[2;3] The epidemiology and age range affected depend on the characteristics of the parasite species and exposure history. In areas with high transmission rates the adult population will generally have acquired immunity and more children will be affected.[4] Most CL cases occur in the Middle East and in South and Central America but it is also endemic around the Mediterranean basin and in South Sudan, Kenya and especially Northern Ethiopia.[5;6]

Etiology and pathogenesis

The leishmaniases are a complex of diseases caused by the intracellular protozoaLeishmania. Disease transmission occurs through the bite of an infected sandfly. CL in Africa is mainly caused by *Leishmania major*. There are smaller foci of *L. tropica, L. infantum and L. Aethiopica*.

CL usually affects the exposed skin, of the face, neck and arms. Poorly functioning health care facilities, poverty and lack of knowledge all play a role in the spread of leishmaniasis.[3;4;6;7]

Clinical findings

One week to three months after an infected bite or bites solitary or multiple lesions appear. A red or skincoloured papule develops into a non-healing plaque or nodule which often shows central ulceration with a well demarcated with a violaceous border. It is usually painless unless superinfected. Regional lymphatic tissue can be involved, leading to a lymphadenitis. Untreated it usually leaves an ugly atrophic scar. Continuous ulceration and diffuse cutaneous infection may occur.

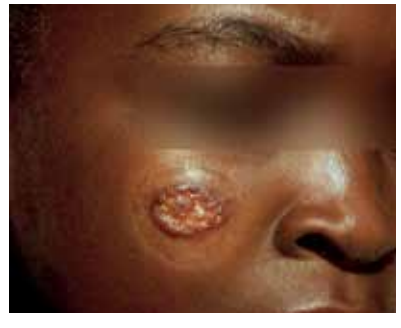
Differential diagnosis

- Leprosy
- Impetigo / ecthyma
- Insectbite
- Cutaneous tuberculosis
- Atypical mycobacterial infections
- Syphilis

Management [4]

- The choice of treatment depends on the type of leishmania and the number of lesions.
- Preventive measures like protective clothing and avoidance of bites.
- When there are single or limited number of localized lesions cryotherapy, electrocoagulation and surgery are treatment options.
- Single or limited number of lesions Pentostam or sodium stibogluconate (SSG): 6 to 10 times once or twice weekly intralesional injections (inject 0.5 to 1.5 ml of 100 mg/ml).
- Glucantine or Pentostam 20 mg/kg/day for 20-30 days i.v or i.m.
- Pentamidine 4mg/kg/weekly i.m. as long as necessary in cases of diffuse cutaneous leishmaniasis by *L. aethiopica*.
- Miltefosine, for children from 3 years and older: 1.5-2.5 mg/kg/daily orally for 28 days.
- Itra-, keto-, fluconazole depending on species.
- Amphotericine B (Amphotericin B and liposomal Amphotericin B are especially effective in visceral leishmaniasis and PKDL and should be administered in a specialized centre).

Clinical picture



Typical lesion on the cheek of a young east African boy

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Scabies

Epidemiology

Scabies is a common ectoparasitic infestation caused by *Sarcoptes scabiei,* a human-specific mite that is highly prevalent in some areas of the developing world, though the prevalence of infection in communities may be cyclical.[1-3]

Etiology and pathogenesis

It is mostly spread by close personal contact but can also be spread by clothing, sheets and towels. Secondary bacterial infection of scabies is common and might increase the risk for glomerulonephritis.[4;5]

Clinical findings

Typical sites of involvement are the interdigital spaces of the hands, the flexural parts of the wrists, the armpits, the feet and the genitals. One of the clinical signs, though not always present in warm climates, is the burrow (S- shaped ridge) caused by the excavation of the female mite for her eggs. Small erythematous papules can be present together with excoriations. Itching, especially at night, is the main complaint which often results in scratch marks and secondary infection.[6-8]

Differential diagnosis

- Eczema
- Contact dermatitis
- Pyoderma
- Bullous pemphigoid
- Insect bites
- Papular urticaria

Management

- Treat all individuals living in same household at the same time.
- Wash sheets and clothes or hang them outdoors for at least 24 hours.
- Sulphur ointment 5-10% to apply twice daily for at least one week.
- Benzyl benzoate emulsion (10 to 25%) is applied over the entire body and left on the skin for up to 24 hours before washing off. Treat during 3 nights and repeat after one week.
- Epidemics in institutions like prisons and boarding schools may be treated with Ivermectin on day 1 and day 10 . Not suitable for children below 5 years of age. See for the dosages cutaneous larva migrans.
- In case of secondary infection oral antibiotics like cloxacillin or erythromycin can be given. For the dosages see impetigo.
- For severe itchiness sedating oral antihistamines like Piriton or promethazine can be used. For the dosages see urticaria.
- After treatment complaints of itch may persist for weeks. This can be treated with mild topical steroids like hydrocortisone cream or ointment two times daily.

Clinical pictures

Interdigital papules

Papules, pustules and scratchmarks

Reference List

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Skin infections

HELMINTH

Cutaneous larva migrans/creeping eruption

Epidemiology

Cutaneous larva migrans (CLM) is endemic in resource-poor communities in the developing world and occurs sporadically in high-income countries, where it is commonly seen as an imported skin disease in travelers[1-4]

Etiology and pathogenesis

Cutaneous larva migrans (CLM) in humans is usually caused by the penetration of cat or dog hookworm larvae into human skin. The presence of animal reservoirs like cats and dogs ensures ongoing transmission. Anyone walking barefoot or sitting on a contaminated beach is at risk. Transmission occurs when skin is in direct contact with soil, contaminated by dog or cat faeces and/or urine or indirect via towel and underware. Humans become a dead-end host because the migrating parasite cannot penetrate into the dermis and eventually dies in the epidermis. Its cutaneous manifestations usually resolve within weeks or months. [2;5]

Clinical findings

The lesions are characteristically urticarial, raised and vesicular. The diagnosis is made clinically in the presence of a linear serpiginous track moving forward in the skin at a speed of 1 to 5 cm per day. The lesions can be intensely pruritic and bacterial super infection often occurs as a result of scratching. Most lesions are located on the trunk, legs, and feet.[6;7]

Differential diagnosis

- Larva currens (Strongyloides stercoralis infection)
- Folliculitis
- Scabies and other ectoparasites
- Insect bites
- Urticaria

Management

- Cryotherapy with liquid nitrogen may be tried for limited lesions. Treat the skin at 1 cm ahead of the visible trail; this is where the larva is found.
- For oral treatment the use of ivermectin or albendazole can be considered.

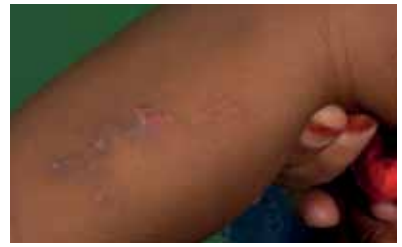
Ivermectin

- Preferably not for children below 5 years of age.
- Dosage depends on bodyweight and is usually given in a single dose.
- Child between 15-25 kg: 1 tablet of 3 mg ; Child between 25-35 kg: 2 tablets of 3 mg; Child between 35-50 kg: 3 tablets of 3 mg; Child between 50-65 kg 4 tablets of 3 mg; Child above 65 kg: adult dose 5 tablets of 3 mg

***Albendazole**

- Children below 2 years of age: 200 mg once or twice daily for 1 to 3 days. Oral suspension (40 mg/mL) 5 ml syrup once or twice daily for 1 to 3 days. Children of 2 years and above: 400 mg once or twice daily for 1 to 3 days. Oral suspension (40mg/mL), 10 ml syrup once or twice daily for 1 to 3 days or tablets (200mg), 2 tablets once or twice daily for 1 to 3 days.
  - Secundary infection can be treated with betadine scrub, potassium permanganate solution or Gentian violet paint.

Clinical pictures



Cutaneous larva migrans on the leg of a Kenian toddler

Cutaneous larva migrans detail

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Lymphatic Filariasis

Epidemiology

Lymphoedema in the tropics can have several causes, but is usually caused by inflammation and consequently adenitis due to bacteria, fungi or minerals. Secondary lymphoedema due to filariasis has a high prevalence and is considered by many the most prevalent cause. In Africa *Wuchereria bancrofti,* a parasitic worm infection transmitted by mosquitoes, is responsible for the majority of cases of lymphatic filariasis. Out of the 120 million infected patients worldwide 40 million develop clinical symptoms.[1-3]

Etiology and pathogenesis

Lymphatic filariasis is a helminth disease that causes chronic and long-term infection with host inflammation due to the antigenic determinants of the parasite. Adult worms are present in the lymphatics and the resulting inflammatory response can cause obstruction. This obstruction is often acquired in childhood and leads to acute attacks of dermato-lymphangio-adenitis and elephantiasis, lymphoedema of limbs and genitals. Like in other filarial infections the symbiosis with the Wolbachia bacteria is likely to be essential for the multiplication and development of the parasite.[2;4]

Clinical findings

After an incubation period of 5 to 15 months the presence of adult worms can lead to lymphangitis and lymphadenitis with localized pain and pitting oedema starting in the upper legs. These attacks can become chronic and cause lymphatic vessel dysfunction and damage.[2;4-6]

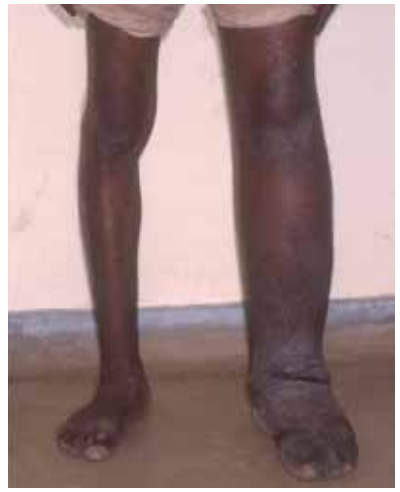
Differential diagnosis

- Kaposi sarcoma
- Lymphoedema caused by bacterial or fungal lymphangitis.
- Lymphoedema caused by podoconiosis (silicates in red volcanic soil which enter the soles and block the lymphnodes after an inflammatory reaction).[6]

Management

- Prevent secondary infections.
- Lymph massage, elastic compression bandages and or stockings.
- (Breathing) exercise. [7]
- Ivermectin plus albendazol in a single dose. To be repeated yearly during 5 years.[2;8;9] For the dosages see cutaneous larva migrans.
- Together with the ivermectin and albendazol treatment, a 6-week course of doxycycline (100–200 mg per day) has been recommended (not in young children). This treatment serves to reduce the Wolbachia bacteria but is still under discussion.[2;8;10]

Clinical picture



A late consequence of filariasis

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Onchocerciasis

Epidemiology

Onchocerciasis is a chronic tropical parasitic disease, caused by the nematode *Onchocerca volvulus*, most widely known for causing "river blindness" and severe dermatological problems.[1] It is found in 28 African countries with the highest prevalences in sub-Saharan West African nations like Ghana, Nigeria, Liberia and Mali.[2] Around 17 million people are affected worldwide.[3] In Africa, where the the burden of onchocerciasis is greatest, years of treatment and eradication programmes have led to a dramatic decrease of transmission.[4;5]

Etiology and pathogenesis

The vector of *Onchocerca volvulus* is the *Simulium* or black fly which lives close to fast moving, oxygen rich water. After infection it takes 12 to 18 months before the first

clinical signs present. The female larvae develop to adulthood and form fibrous capsules, the so called onchocercomata. During adulthood, the female worm sheds hundreds of thousands of microfilaria which migrate through the skin of the human host and cause severe itch, and, after repeated infections, in some regions blindness. Like in other filarial infections symbiosis with the Wolbachia bacteria is essential for multiplication and development of the parasite. A biopsy or skin snip test may show microfilaria.

Clinical findings

The most common skin problem in the first stage is troublesome itching with some erythematous hyper pigmented papules and patchy lichenification. In the chronic stage there can be pruritic generalized lichenification and depigmentation ("Leopard skin") later on. Sub dermal nodules ("onchocercomata,") are mostly seen over bony prominences like the hips but can be present anywhere. The loss of elasticity may cause so-calles hanging groins and lymph edema.[7;8]

Differential diagnosis

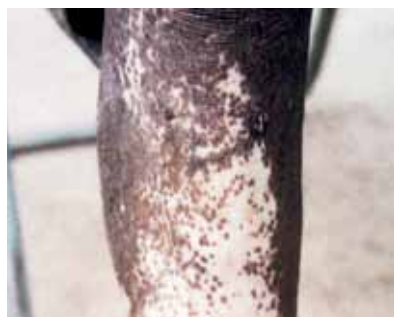
- Food allergy
- Other parasitic infestations
- Leprosy
- Syphilis

Management

- The standard treatment is ivermectin orally every 6 to 12 months). For the dosages see cutaneous larva migrans. Single-dose ivermectin effectively kills microfilariae but has little effect on adult worms; therefore, it controls but does not cure the disease.
- A patient staying in an endemic area needs treatment every 3 to 12 months, not only to kill new microfilaria but also for the treatment of reinfection.

Clinical pictures

Itch and lichenification



"Leopard" skin

- Together with the ivermectin treatment a 6-week course of doxycycline (100–200 mg per day) given to eliminate the Wolbachia bacteria. Because of the deposition of tetracyclines in growing bone and teeth it should not be given to children under 12 years or to pregnant or breast-feeding women. [2]

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Inflammatory skin diseases

Eczema/Atopic Dermatitis

Epidemiology

Eczema is widespread in the industrialized world and a growing clinical problem in sub-Sahara Africa.[1;2] In West Africa, the prevalence of eczema was considered to be < 5% although recent studies in West Africa and other parts of Africa have shown an increase, particularly amongst infants.[3;4] However, recent point-prevalence rates among schoolchildren in West and Central Africa, derived after physical examination by a dermatologist, were considerably lower.[5]

Etiology and pathogenesis

Atopic dermatitis or eczema is a chronic relapsing, pruritic inflammatory skin disorder. Although termed atopic, up to 60% of the children with the clinical phenotype do not

have demonstrable IgE-mediated sensitivity to allergens. Therefore it is preferable to use the term 'eczema'.[6;7]

Eczema is a multifactorial skin disease. Some risk factors for eczema are; genetic predisposition (like asthma and hay fever which may run in the family), emotional stress and change in lifestyle (such as changes in food patterns, contact with irritants or frequent washing).[8-11] Mutations in the filaggrin gene (FLG) are a major predisposing factor for ichthyosis vulgaris and eczema in individuals of European and Asian descent. These genetic findings provide an important support for the well known impairment of the epidermal barrier observed in eczema and could also deliver further clues to the natural history of the disease. Recent research indicates that FLG loss-of-function variants are less common in Africa.[12]

Clinical findings

Clinically eczema in the acute stage is characterized by itching, redness, oozing, crusts and often secondary infection with *Staphylococcus aureus*. The chronic stage is characterized by lichenification, excoriations and a very dry skin. Especially elbow-and knee folds, wrists, ankles, face and neck are affected.[13;14]

Three distinct clinical phases of eczema can be observed according to the age. In the infantile phase the eruption characteristically starts on the cheeks and scalp but the whole body can be affected. In the childhood phase especially the flexural areas of the knee and elbows are affected but also the wrists, ankles and buttocks can be involved. In the adult phase especially the neck and face are affected with a more diffuse scaling and erythema.Xerosis and lichenification are important characteristics.

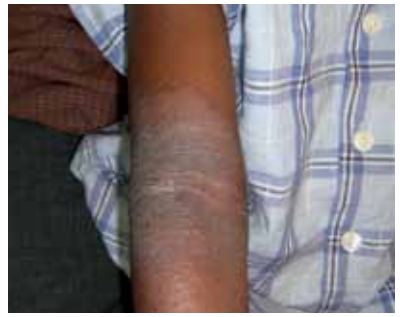
Differential diagnosis

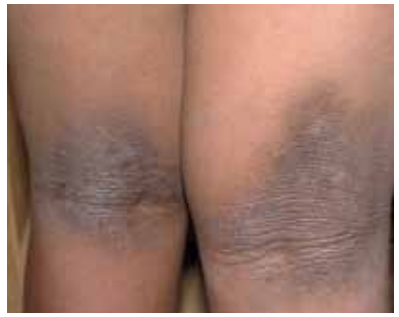
- Seborrheic dermatitis
- Contact dermatitis
- Psoriasis
- Scabies
- Dermatophytosis
- HIV related dermatoses

Management

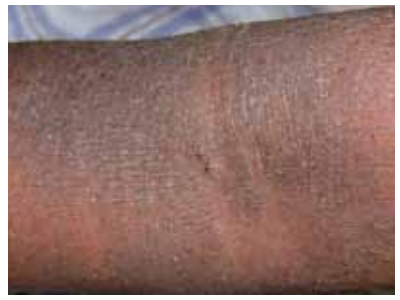
- Explain the multifactorial and chronic character of the disease to the patients, parents and / or care takers.
- The use of soap and the frequency of washing should be reduced. Cotton clothing is preferred to wool or synthetics. Children should not be dressed too warm.
- Moisturize the skin regularly with an emollient cream or ointment like aqueous cream, coco butter or shea butter.
- In severe cases a potent steroid ointment like betamethasone can be applied once daily on the lesions.[15] Potent steroids should be used during a limited time and intermittently because of the risk of atrophy and bleaching. The use of potent corticosteroids should be avoided for use on the face or intertriginous sites like the groin or armpits.
- If available topical calcineurin inhibitors (TCI) like tacrolimus (0.03% and 0.1% ointment) or pimecrolimus (1% cream) can be used as maintenance treatment. The TCI don't cause skin atrophy. They may however cause a burning sensation upon application especially in the beginning of the treatment.
- In case of secondary infection oral antibiotics like cloxacillin or erythromycin can be given. For the dosages see impetigo.
- For severe itchiness sedating oral antihistamines like Piriton or promethazine can be used. For the dosages see urticaria.
- In more severe cases, when phototherapy or systemic therapy might be needed, patients should be sent to a referral / university hospital.

Clinical pictures

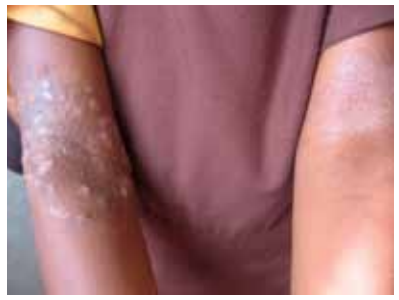




Eczema: elbow and knee folds, typical localizations



Eczema: detail: lichenification, hyper-pigmentation and scratch marks



Eczema: secondary infection

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Acne vulgaris

Epidemiology

Acne vulgaris is common in children and adolescents from age 10 and commonly persists up to age 25. In industrialized countries it affects between 30% and 100% of the adolescent population.[1-3] The prevalence of acne is considerably lower in developing countries though Westernization in urban areas in developing countries has been shown to lead to higher prevalence.[4-7]

Etiology and pathogenesis

Several factors play an important role in the etiology of acne: follicular epidermal hyper proliferation, excess of sebum production, activity of *Propionibacterium acnes* and inflammation. Androgenic hormones stimulate the hyperproliferation of the follicular keratinocytes and lead to an increased sebum production which is followed by the formation of comedones. Especially *P. acnes* is an important factor in the process of inflammation. A genetic predisposition, especially with the nodulocystic form has been

suggested. Recently some studies described a relationship between the development of acne and Body Mass Index.[4;8;9] The use of oil, bleaching and cosmetic creams is another important factor.

Clinical findings

The sites most affected are the face, back, chest and shoulders. Non inflammatory acne may consist of open comedos (blackheads) or closed comedos (whiteheads). In inflammatory acne the comedos expand to form erythematous papules, pustules, nodules or cysts. Pomade acne is very frequently seen in Africa due to the use of petrolatum or petroleum jelly (e.g. Vaseline as brand name). The nodulocystic form of acne can lead to severe scarring.

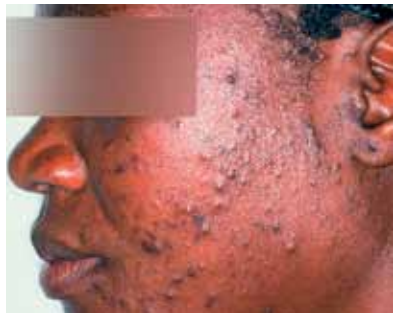
Differential diagnosis

- Folliculitis due to yeasts (Pityrosporum) or bacteria
- Perioral dermatitis
- Milia

Management

- Stop the use of oily cosmetics or petrolatum on the skin and hair.
- Apply benzoyl peroxide 5-10% preparations at night (because of its photosensitive effect) and warn the patient that it can bleach the pillows and pyjamas. Benzoyl peroxide preparations are available in creams, gels, lotions and washes.
- Apply topical retinoids at night (because of their photosensitive effect).Options are tretinoin (0.05% -0.1% solution or 0.02%-0.05% cream), adapalene and tazarotene (0.1% gel). Start at low concentrations to prevent irritation and hyperpigmentation.[10] Greater tolerability can be achieved by applying it the first two weeks of treatment on alternate nights.
- Apply topical clindamycin 1% lotion or erythromycin 2% lotion or gel in the morning.
- In case of moderate/ severe acne, use oral tetracyclines like tetracycline 250 mg twice or four times daily, doxycycline 100 mg once daily or erythromycin 4 times daily 250 mg and after one month 2 times daily 250 mg. The treatment has to be continued for several months and repeated when the acne comes back. Oral tetracyclines should not be given to young children.
- In case of moderate acne in women oral contraceptives may be given like Diane- 35 (cyproteronacetaat).
- Postinflammatory hyperpigmentation commonly occurs in a dark skin. Acne treatment should be started in an early phase in order to prevent this occurring.
- In case of severe acne or nodulocystic acne oral isotretinoin may be considered. The patient should be referred to a dermatologist for this treatment because of its potentially severe side effects among which teratogenicity.

Clinical pictures



A greasy skin and multiple papulopustules and comedones and postinflammatory hyperpigmentation

Pomade acne

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Psoriasis

Epidemiology

Psoriasis is a common skin disease in children although the prevalence is much lower than in adults. The total rate of psoriasis in children younger than 18 years found in Germany was 0.71% while this was 1.4% in Great Britain.[1;2] In the Netherlands and the US even lower figures were found and juvenile psoriasis seemed to be less common in the US among African Americans than among Hispanics and Caucasians.[3-5] The prevalence among girls is normally higher than in boys. Hospital based studies from Africa show prevalences of 1.5% in Egypt, 0.9% in Nigeria, 0.05% in Mali and 3.5% in Kenya.[6-9] Population based studies among schoolchildren in West and Eastern Africa showed very low prevalences.[10]

Etiology and pathogenesis

Psoriasis is characterized by the proliferation of keratinocytes and inflammatory cell infiltration of the dermis and epidermis. This reaction is caused by dermal infiltration of T lymphocytes and macrophages and leads to a fast turnover and hyperplasia of the epidermis. This results in a chronic inflammatory condition affecting the skin, nails, and joints.[11] Patients are genetically predisposed to psoriasis. Psoriasis in adults is associated with comorbidities such as obesity, hyperlipidemia, diabetes mellitus (metabolic syndrome), rheumatoid arthritis and Crohn's disease.[12] Physical trauma may trigger psoriatic lesions at sites of injury (Koebner's phenomenon).[4;11] Other triggers are antimalarials, lithium, beta blockers, stress, infections such as streptococcal angina and a cooler climate.

Clinical findings

The plaque type; this is the most frequently observed variant of psoriasis. It is characterized by sharply demarcated erythematous plaques covered by silvery white scales which shows the typical candle wax phenomenon after scratching. Lesions commonly appear on the elbows, knees, scalp, umbilicus, and lumbar area. The scalp is the most frequently affected site of involvement in pediatric psoriasis. Facial and intertriginous lesions may be difficult to differentiate from seborrheic eczema if there are no other typical psoriasis lesions.

Guttate psoriasis; is more frequently seen in children and consists of numerous papules and plaques (like "drops") all over the body. Guttate psoriasis is often preceded by a streptococcal throat infection.[11;13] The prognosis is good, with spontaneous remissions in weeks to months.

The inverse type of psoriasis; in this type of psoriasis the lesions appear as sharply defined erythematous plaques which show no or minimal scaling in intertriginous areas like the groin and armpits.

Erythrodermic psoriasis; nearly the whole body surface can be involved but this is rare in children.

Nail involvement (especially the fingernails) is uncommon in children with psoriasis. If it occurs nail-pitting is the common manifestation. Onycholysis and the "oil drop" sign are rare.[13]

Psoriatic arthritis; is an extracutaneous manifestation which is rare among children in Africa. A recent African review suggested an association between psoriatic arthritis and HIV infection.[9]

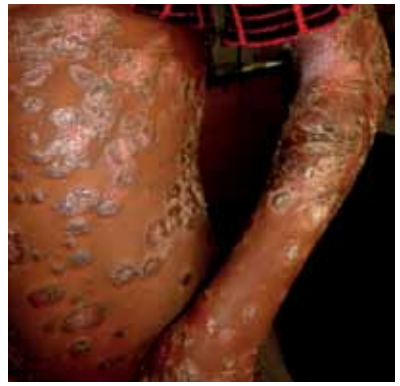
Differential diagnosis

- Tinea capitis and corporis
- Seborrheic dermatitis
- Eczema
- Lichen planus
- Pityriasis rosea / secondary syphilis (d.d. psoriasis guttata)

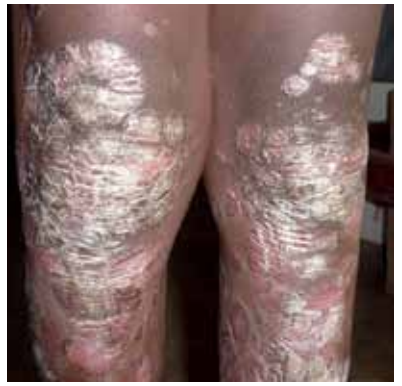
Management

- Discuss the chronic character ("come and go") of the disease with the patients and the parents / caretakers. Explain that psoriasis is not contagious but can be triggered by an infection. Natural sunlight has a beneficial effect.
- Approximately 70 to 80 percent of all patients with psoriasis can be treated adequately with use of topical therapy!
- Salicylic acid 5-10% in an oil, lotion, cream or ointment base 2 times daily to reduce the scaling.
- A moderate to strong topical steroid like betamethason ointment can be applied daily on the lesions. Cannot be used continuously for a long time because of side effects like atrophy and bleaching. Can be used in combination with salicylic acid 2-10% ointment.
- Coal tar 5-10% ointment or sulphur 5% in coal tar 5-10% at night.
- Vitamin D3 analogue like calcipotriol 2 times daily on the lesions, especially with plaque psoriasis. Can be used in combination with corticosteroids.
- Anthralin 0.1-1% cream or ointment. Especially for plaque psoriasis. Has to be wiped or washed off after 10-60 minutes. Not always suitable for children because of the irritative reaction.
- Find the possible bacterial sources of streptococcal infection (pharyngeal and perianal) and treat with antibiotics like erythromycin, penicillin or cephalosporines. For dosages see impetigo and ecthyma.
- If possible refer the patient to a dermatologist for phototherapy in the case of guttate psoriasis (UVB is the preferred form of phototherapy for pre-adolescent pediatric psoriasis).
- Systemic therapy with *Methotrexate, Ciclosporin, Retinoids* and *Biologicals* may be used for severe cases of chronic plaque psoriasis, guttate psoriasis in children who are unresponsive to antibiotics, topical treatment and UV therapy and to children with severe arthropathic psoriasis. These cases are rare and need to be referred to an university hospital or specialized centre.

Clinical pictures



Multiple hyperkeratotic plaques on the trunk and arms



Multiple hyperkeratotic, well circumscribed plaques on both knees

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Seborrheic dermatitis

Epidemiology

Seborrheic dermatitis is one of the most frequent skin disorders, especially the infantile form which affects as many as 70% of the newborns but disappears by the age of 1 year.[1;2] The prevalence in immunocompetent adults is between 1% and 3%, and is more common in men than in women. The prevalence is low in children over one and under 12 years of age[3;4] However in children of all ages, as in adults, it is frequently seen in combination with a HIV infection.[5]

Etiology and pathogenesis

The cause of seborrheic dermatitis is not completely understood. It occurs most often during periods of active sebum production (e.g., the neonatal period) and in areas of the skin where sebum is produced. There is no clear genetic predisposition but climate, stress and immunologic factors play an important role.[2;6]

Malassezia yeasts may play a role in the pathogenesis of seborrheic dermatitis since they are present on affected skin, and antifungal agents are useful in the treatment.[7] Especially in HIV-infection they appear to play a role.

Clinical findings

Seborrheic dermatitis is characterized by scaling and poorly demarcated erythematous patches that vary from pink yellow to red brown in color. In the African skin they are often hypopigmented. There is a predilection for places which are rich in sebaceous glands like the scalp, the nasolabial folds, glabella and the hairline, the sternum, the armpits and the groins.[2;6;8] The morphologic characteristics depend on the area of the skin involved. In healthy people the face and scalp are commonly affected, in the HIV-infected, armpits and groins also show lesions and they easily become superinfected. The lesions cause normally mild itching. Seborrheic dermatitis can give reason to social problems, especially with severe / moderate scaling of the scalp.[9;10]

Differential diagnosis

- Psoriasis
- Atopic dermatitis
- Tinea capitis

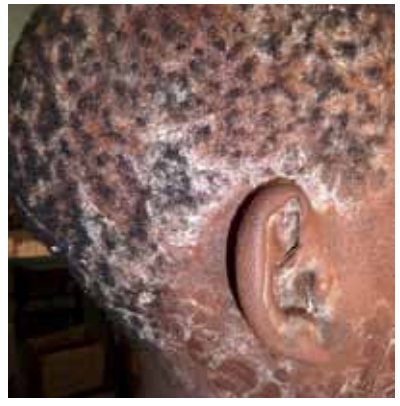
Management [2;11]

- In infantile seborrheic dermatitis the application of an emollient cream can be useful.
- Sulphur 3-5% cream to apply 2 times daily.
- Ketoconazole 2% cream or cicloporox 0.77% cream to apply 2 times daily.
- Ketoconazole or cicloporox shampoo. Low potency topical corticosteroids like hydrocortisone 1% cream 2 times daily, used intermittently.

- Topical calcineurin inhibitors like 0.1% tacrolimus ointment may be useful for facial lesions; it is too greasy for the armpits and groins.
- In severe and widespread lesions oral ketoconazole can be used. Usually it is given only to children above 2 years of age. The dosage in a child is 3 mg/kg daily for 2 weeks from 15 kg body weight onwards.

Clinical pictures

Bov 7 years old. HIV+ Seborrheic dermatitis of the scalp and the groin



Girl 6 years old, the differential diagnose with psoriasis capitis can be difficult.

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Lichen planus

Epidemiology

Lichen planus is frequently encountered in hospital based studies in sub-Sahara Africa.[1;2] In childhood it is unusual and pediatric patients comprise only 2% to 3% of all those affected.[3;4] There is no consistent gender predilection for childhood. In the USA it has been reported to be more prevalent among African American children.[5]

Etiology and pathogenesis

Lichen planus is an inflammatory dermatosis of unknown origin. Several reports have shown an association between lichen planus and liver disease such as chronic active hepatitis and as a complication of hepatitis B vaccination.[6] Also a positive history of auto immune diseases like myasthenia gravis, alopecia areata and lupus erythematosus has been described. In several studies there was a positive correlation with atopic dermatitis.[5] Quinine has also been described as initiating or worsening lichen planus.

Clinical findings

Lichen planus often presents with pruritic violaceous, polygonal, flat-topped papules and plaques most frequently seen on the flexor surfaces of the wrists and forearms (see picture 1) but the anterior side of the lower legs, the lumbo sacral region (see picture 2) and the neck are also common sites. Papules can develop at sites of trauma which represents the Koebner phenomenon. In the African skin, the lesions have a more grey aspect. With a drop of oil the striae of Wickham become visible. It can also affect the skin of the genitals and mucous membranes although this is very uncommon in young people. Lichen planus may resolve spontaneously with time ranging from a few months to years and often leaves residual areas of hyper pigmentation.[4;7]

Differential diagnosis

- Lupus erythematosus
- Lichen sclerosus and striatus
- Pityriasis rosea
- Secondary syphilis

Management [5;7;8]

- Parents and patients should be reassured that lichen planus is a benign non- infectious, self limiting disease.
- Moderate to strong topical corticosteroids like betamethasone 2 times daily, preferably combined with salicylic acid 5 % are the treatment of choice.
- Topical calcineurin inhibitors like tacrolimus 0.1% 2 times daily.
- In severe cases oral corticosteroids can be given (0.5-1 mg/kg daily) as a tapering dose over a 2-6 week period. Long term maintenance therapy with systemic corticosteroids should be avoided.
- Dapson 1mg/kg daily has been reported to be very helpful in severe cases.[9]
- Severe cases, unresponsive to treatment, should be referred to a dermatologist for phototherapy, intralesional therapy with triamcinolone 5-10 mg/ml, or systemic therapy with methotrexate or cyclosporine.

Clinical pictures

Multiple polygonal flat-topped papules and plaques especially on the wrists (differential diagnose verrucae vulgares!) and the lumbo sacral region.

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Alopecia areata

Epidemiology

Alopecia areata generally concerns pupils or students although the prevalence among schoolchildren in Africa was less than 1%.[1-4] The life-time risk of alopecia areata in the general population is approximately 1.7%. and in as many as 60% of patients the disease starts before the age of 20 years.[5] In patients with alopecia areata a considerable amount had episodes before or has a positive family history.[4]

Etiology and pathogenesis

Alopecia areata is an autoimmune disease that presents with nonscarring hairloss.[4] The pathogenesis is not completely clear.[6;7] Atopy, autoimmune thyroid disease, a positive family history and vitiligo are commonly associated. The course of the disease is unpredictable. Early and severe cases which last long have a less favorable prognosis.[8]

Clinical findings

Alopecia areata most commonly manifests as sudden loss of hair in a well demarcated, localized area in the scalp. The hair loss is usually limited to a single patch. The lesion is usually round or oval. “Exclamation point hairs” are frequently seen at the periphery of the lesion.[5]

The majority of patients present with limited alopecia. Approximately 80% present with one patch, about 12% with multiple patches on the scalp and possibly also in the eyebrows, lashes, and beard area, and about 7 % develop total baldness of the scalp (alopecia totalis), some even of all body hair (alopecia universalis).[9] The clinical diagnosis is made by the aspect of hairless patches with a normal skin.

Differential diagnosis [3;8;10]

- Androgenetic alopecia
- Traction alopecia
- Tinea capitis
- Trichotillomania
- Syphilis
- Atopic dermatitis
- Vitiligo

Management [5;11]

- Because of the high rate of spontaneous recovery a "watch-and-see" approach is often recommended.
- Psychological support may be offered if necessary.
- For patients who actively desire treatment, topical or intralesional corticosteroids are the treatments of choice. Betamethasone dipropionate lotion 0.05% can be applied 2 times daily for 12 weeks or betamethason cream 2 times daily for 1-2 months. If there is no improvement after 12 weeks the treatment should be stopped. Intralesional corticosteroids are appropriate for older children. Triamcinolon acetonide 10 mg/ml diluted with 2% lidocaine with epinephrine (to reduce the pain with the injections) can be injected intradermal once monthly and not longer than 6 months.
- Topical sensitizers like Anthralin (Dithranol) can be used in concentrations of 0.25-1% cream. Anthralin cream may be applied overnight, initially for 30 minutes and gradually to 1 hour. If there is no result it can be stopped after 3 months. Another possibility is the treatment with diphenylcyclopropenone (DPCP) but this is usually done under the supervision of a dermatologist.

Clinical pictures

Alopecia areata in a young child. A round well circumscribed area with hairloss...

Traction alopecia in a young girl...

- Ultraviolet A phototherapy (PUVA) is another option for which the patient has to be referred to a dermatologist.

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Pityriasis rosea

Epidemiology

Pityriasis rosea is a common, acute, self-limiting papulosquamous eruption. It typically affects children and young adults. There is a worldwide distribution and no ethnic predilection has been found. The prevalence among females seems to be slightly higher than males.[1] The point prevalence found among schoolchildren in Africa in several studies was low but in other, hospital based studies the period prevalences were higher.[1-3]

Etiology and pathogenesis

Pityriasis rosea is possibly of viral etiology ("flu of the skin"), it has been linked to human herpes virus 6 (HHV6). About a quarter of the patients have a history of a viral infection with upper respiratory symptoms shortly before or during the occurrence of the rash. Several medications can cause a rash similar to pityriasis rosea. It is self-limited and normally the eruptions last for 6 to 8 weeks.[4-6]

Clinical findings

Pityriasis rosea is characterized by an initial "herald patch" ("plaque mère"), followed by the development of a diffuse papulosquamous rash on trunk and arms. It can be difficult to identify until the appearance of characteristic smaller oval shaped secondary lesions that follow the cleavage lines. These lesions can form a so called "Christmas tree pattern" on the back. Pityriasis rosea is usually asymptomatic but can sometimes itch.[7;8]

Differential diagnosis

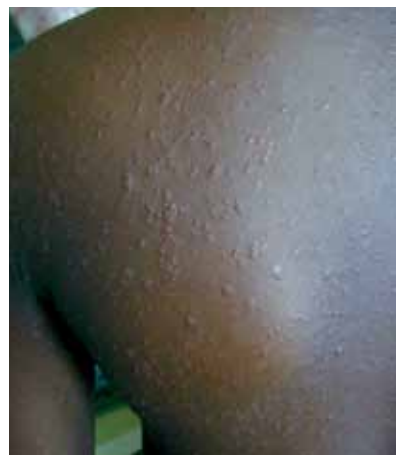
- Secondary syphilis
- Eczema (especially the herald patch)
- Psoriasis
- Tinea corporis

Management

- To differentiate between pityriasis rosea and secondary syphilis serologic testing for syphilis (VDRL or FTA-ABS) is necessary.
- Because in most cases it is self-limited and asymptomatic, a good explanation and reassurance of the patient is very important.
- In case of pruritus Calamine lotion or low to medium potent topical corticosteroids like hydrocortison1% or triamcinolon acetonide 0.1% cream can be applied 2 times daily.
- For severe itchiness oral sedating antihistamines like piriton or promethazine can be used. For the dosages see urticaria.
- Natural sunlight exposure can be beneficial.

Clinical pictures

Typical Christmas tree pattern on the back



More papulous pattern

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Benign skin tumors and nevi

Infantile Hemangioma (IH)

Epidemiology

Infantile hemangiomas are common, benign tumors of blood vessels, observed in 1-4 % of infants during the first year of life. Although most cases progress without problems, a small proportion can experience life-threatening complications.[1] They are more prevalent in female, caucasian infants and related with prematurity, advanced maternal age and multiple gestations.[2]

Etiology and pathogenesis

IHs are primarily composed of endothelial cells and can grow rapidly in the first 6 months of life, the proliferation phase. This phase can cause great concern to the parents. Normally it is followed by slow involution, leading to complete regression in about 70% of the patients in 5 to 10 years. The etiology of both stages is still not completely understood.[3;4]

Clinical findings

Most infantile hemangiomas occur within the first weeks of life. They vary in size from less than 1 cm to more than 10 cm. They can occur anywhere on the skin and mucosal surfaces though the preferred site is the face. Hemangiomas which are located in the superficial dermis are bright red in color ("strawberry ").Deep hemangiomas can be located in the deep dermis or subcutis and present as blue purple tumors. They may pose a problem during the growth phase when they can cause obstruction of vision or of the larynx or mouth.[5]

During the regression phase they can bleed easily or become necrotic. Ulceration is the most common complication occurring in approximately 15% of the patients. Regression can be complete and leaves no residual change at the site in most lesions (80%).In some areas it can leave atrophy, depigmentation, teleangiectasis and scarring.[6]

Differential diagnosis

- Capillary malformations or teleangiectasias
- Pyogenic granuloma
- Vascular malformations

Management

- Most cases need no treatment and have an excellent functional and cosmetic prognosis. Active intervention has to be avoided. Explanation to the parents / caretakers is essential.
- Proper follow up and management of ulceration. Local wound care with topical antibiotics like mupirocin or bacitracin ointments and occlusive dressings.
- When vital organs and functions like vision, hearing and breathing are impaired, the patient should be referred to a dermatologist and/or pediatrician for treatment with intralesional or systemic corticosteroids or interferon. Recent publications show good results with the treatment with local or systemic propanolol and systemic atenolol.[7;8]

Clinical pictures

A hemanioma on the lower lip in a Zimbabwean infant...

No problems feeding...

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Miscellaneous skin diseases

Oculocutaneous albinism (OCA)

Epidemiology

The prevalence of OCA is relatively low at general schools in sub-Sahara Africa.[1;2] Children affected with OCA are more commonly found in schools for the blind. Several prevalence studies in South Africa, Tanzania, Nigeria and Zimbabwe show figures of 1/5000-1/15000 but prevalences as high as 1 in 1000 were reported for selected populations.[1-6] The medical and social issues facing children with OCA are enormous and life expectancy is decreased compared with the general population.[7]

Etiology and pathogenesis

Oculocutaneous albinism (OCA) is an inherited functional disorder of melanin production which results in hypo or depigmentation of the skin, hair and eyes and extreme sensitivity to UV-damage. There are different types of OCA all of which have an autosomal recessive inheritance pattern.[7]

Clinical findings

People with OCA have a hypopigmented retina and fovea which leads to photophobia, nystagmus and lower vision. Exposure of the yellowish or white skin to the sun leads to sunburn, blisters, freckling and the formation of solar keratoses. Without sun protection measures basal and squamous cell carcinomas appear from the second or third decade[6;8;9]

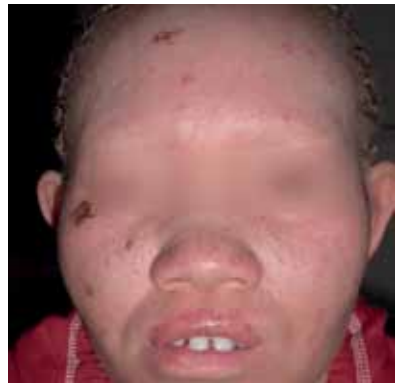
Differential diagnosis

- Vitiligo
- Nutritional deficiencies

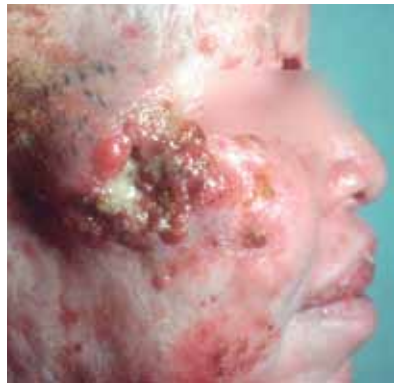
Management

- Protect the skin and eyes from sun damage: avoid the midday sun, wear a wide rimmed hat, protective clothing (long sleeves, long skirts and trousers) and sunglasses.
- Always use a sun block or a sun screen with a high sun protection factor (SPF) (PABA, zinc oxide, titanium dioxide).
- Use a sun block (e.g. zinc oxide or titanium dioxide) for the lips.
- Regular ophthalmological and dermatological check-ups.
- Treat solar keratoses with liquid nitrogen, curettage or topical 5% 5-fluoro-uracil.
- Malignancies should be excised, preferably in a specialized clinic.

Clinical pictures



Multiple lentigenes due to ultra violet damage



Without sun protection basal and squamous cell carcinomas appear in an early age...

...a vulnerable group of children

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Vitiligo

Epidemiology

Vitiligo may appear at any age. It affects around 0.5% of the world population. In hospital based studies from West Africa percentages between 2.8 and 6% have been presented.[1,2] In several community based studies among schoolchildren in Africa the prevalences were rather low.[3-6] The average age of onset found in a Nigerian study [7] was approximately 20 years.

Etiology and pathogenesis

The etiology of vitiligo is not exactly known though several studies point towards an autoimmune base, indicating the importance of a positive family history and the presence of other autoimmune diseases, such as diabetes mellitus and hyper-thyroidism.[7] There is an absence of melanocytes in the affected skin. Vitiligo is usually slowly progressive and seldom regresses spontaneously; sometimes the involved skin is pruritic.

Clinical findings

Vitiligo is characterized by sharply demarcated white macules surrounded by normal skin. It can be present on any part of the body but it is frequently localized on the face, the dorsal side of the fingers, the anogenital region and on sites of stretch and pressure. In an affected person it also occurs in traumatized skin, the Koebner phenomenon.
On darkly pigmented skin it is more obvious than on light skin. It can lead to a high level of social stigmatization due to confusion with leprosy.[1]

Differential diagnosis

- Leprosy
- Pityriasis versicolor
- Pityriasis alba
- Onchocerciasis
- "Bleaching" practices like the misuse of potent topical corticosteroids as adjuncts with hydroquinone
- Lichen sclerosus

Management

- Therapeutic treatments are not yet available. Proper explanation and reassurance of the patient is important. Good and practical advice about sun protection and local camouflage can often decrease the psychological burden of the disease a lot.
- Topical treatment (for small localized areas) with intermittent potent corticosteroids during a set period of time, eg four days a week for 6 months, in combination with controlled UV exposure.
- If available topical calcineurin inhibitors (TCI) like tacrolimus (0.03% and 0.1% ointment) or pimecrolimus (1% cream) may be used. The advantage is that they don't cause cutaneous atrophy.[8]
- Sometimes dapson can be useful. Dosage: 1-2mg/kg daily.
- UVB 311 nm phototherapy can be given in a specialized centre.

Clinical picture

Round, oval shaped white macula on the face

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Fixed drug eruption

Epidemiology

Fixed drug eruption (FDE) is one of the most common types of drug eruption among children presenting in (dermatology) clinics.[1;2] The incidence of fixed drug eruptions among both children and adults in several studies from Asia varied between 1 to 9%.[3;4] In recent studies from Nigeria, the hospital based prevalence of drug eruptions was 1% of which half was caused by FDE. [5;6]

Etiology and pathogenesis

Lesions develop up to several weeks after first exposure to the causative drug but may develop within 24 hours after subsequent exposures. Although the pathogenesis remains unclear positive patch test results suggest type IV hypersensitivity. Patch test results vary greatly, depending on the causative drugs.[1;2;7] The drugs most frequently associated with FDE are barbiturates, paracetamol, sulphonamides (e.g. trimethoprim-sulfamethoxazole), anti malarials and various other antibiotics, especially tetracyclines. FDE caused by Sulfa-based antimalarials frequently affect the face, lips, and limbs, whereas co-trimoxazole frequently causes genital and oral lesions. [1;6;8]

Clinical findings

The characteristic finding in FDE is recurrence of the lesions at the same sites. Lesions are sharply demarcated round or oval erythematous to violaceous / black plaques 2 to 10 cm in diameter. Usually they present as a single lesion or in limited numbers and are localized. Any cutaneous or mucosal surface can be involved including lips and genitals. With repeated episodes, the lesions may increase in size and/or number and present with more profound hyperpigmentation.[1;2;6;8]

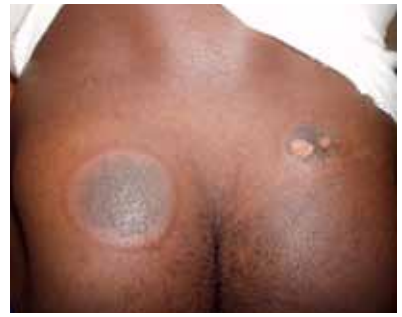
Differential diagnosis

- Insect bites
- Urticaria
- Erythema multiforme

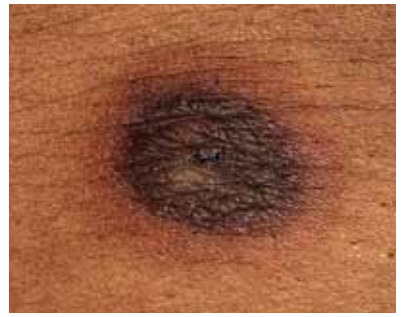
Management

- Prevent recurrence by identification of the responsible drug.
- Counsel the parents / caretakers about proper drug use and avoidance of responsible drugs.
- When itchy : Calamine lotion to apply 2 times daily.
- For severe itchiness oral antihistamines like piriton or promethazine can be used. For the dosages see urticaria.

Clinical pictures



Sharply demarcated, round lesions with a hyper pigmented centre and an erythematous edge



Detail, round lesion with a hyperpigmented centre

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Keloids

Epidemiology

For centuries, keloids have been a well known clinical problem and despite considerable research to unravel this phenomenon there is still no universally accepted or effective treatment.[1] Keloids and hypertrophic scars occur worldwide in all skin types but they are more common in people of African descent. Incidence rates of 16% among adult Africans have been reported while these percentages were considerably lower among schoolchildren.[2;3] In severe forms they can become disabling.

Etiology and pathogenesis

Hypertrophic scars and keloids are formed from excessive scar tissue formation at the site of prior skin injury. There is often a familial tendency for developing hypertrophic

scars and keloids but the pathogenesis remains unknown. Most probably nutritional, biochemical, immunological, and genetic factors play a role in the abnormal wound healing.[1;4;5] Another hypothesis is the influence of change in hormonal status. This might be the reason that in children before puberty there is no keloid formation after piercing the earlobes. Unfortunately prevention is often not successful.

Clinical findings

Keloids are fibrous tumors caused by overgrowth of connective tissue. They occur as a result of skin injury, such as burns, surgical or tribal cuts and ear piercing but also after inflammatory skin diseases like acne and herpes zoster.

Sites of predilection are shoulders, upper back, chest and earlobes. At first lesions are pink-to purple and often pruritic and painful. Hypertrophic scarring is restricted to the area of the original lesion and has a tendency of gradual resolution over time. Keloids can migrate into adjacent tissue to form hard, irregular shiny ridges or plaques and are persistent. [4;6]

Differential diagnosis

- Differentiating between a hypertrophic scar and keloid can be difficult.
- Scleroderma
- Dermatofibroma
- Kaposi sarcoma

Management

- Keloids and hypertrophic scars are chronic skin conditions, their treatment also takes time!
- One of the most important things that one can do to prevent the formation of keloids is to avoid trauma to the skin, attend to cuts or abrasions immediately to minimize inflammation and infection, avoid ear piercing and refrain from elective surgery unless medically indicated.
- Intralesional steroid injections: eg kenacort (1:40) on a 1:1 dilution with lidocain 2% once every 3 weeks.
- The following treatments should be preferably carried out in a specialized or university hospital:
  - ➢ Surgical excision of keloids leads to recurrence and more deformity. In severe cases debulking may be needed, and should be followed by regular intralesional steroid injections (7)
  - ➢ Cryosurgery in combination with intralesional corticosteroids can be used for small lesions.
  - ➢ Pressure with silastic gel sheets or pressure garments at night for several months.
  - ➢ Radiotherapy is highly successful but the use is limited due to its damaging long term side effects

Clinical picture

Keloid formation in a 14 year old girl after ear piercing

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Urticaria

Epidemiology

Urticaria is seen in 1-5% of the population and may present at any age. There are no known racial differences. It is more common among women with a female: male ratio of 2:1.[1-3]

Etiology and pathogenesis

Urticaria is a vascular reaction of the skin characterized by mast cell degranulation In children they caused by several factors like allergic or hypersensitivity reactions food (fish, milk, tomatoes, citrus fruits, cocoa, strawberries), drugs (aspirin, pethidine, hydralazine, ibuprofen) insect bites (bee, wasp, mosquito). [4]Also viral infections, mycotic infections, helminthic infections and skin contact with allergens can be a cause. Physical urticaria may be induced by cold, heat, pressure and exercise. In the majority of the cases, the cause remains unidentified.[5]

Clinical findings

Urticaria are well demarcated small (< 1 cm) to large (> 8cm) smooth, slightly elevated patches (wheals) which can itch severely. Individual lesions are self-limiting and resolve

in several hours but may be recurrent over weeks. Chronic urticaria is defined as urticaria with episodes lasting longer than 6 weeks.[6]
They are erythematous or white with an erythematous rim. The erythema which may be prominent in a light skin is not visible on a dark skin. Lesions can be oval, annular or serpiginous. They can appear anywhere and at any interval on the body and as angioedema in the face. The lesions are prutitic. Some patients also show dermographism. Normally urticaria in children is an isolated event, a massive reaction may occur which can lead to an anaphylactic shock.[5;7;8]

Differential diagnosis

- Contact dermatitis
- Maculopapular drug eruptions
- Insect bites
- Pityriasis rosea
- Leprosy reactions

Management [6;8;9]

- Avoid or treat the cause if possible. A thorough history is essential.The treatment depends on the severity, the duration and the type of hives.
- Further investigations, unless aimed at a specific suspected cause, are usually negative and not helpful. Only in debilitating chronic urticaria the following may be considered: Blood count, liver function test, kidney function test, infection parameters, allergy test and tests for autoimmune diseases.
- The most common treatment is oral antihistamines which controls the itching. Wheals may still be visible.

Sedating antihistaminica

*__Piriton (chlorphenamine maleate)__ (British National Formulary)

- Child under 1 year not recommended.
- Child 1-2 years: 1 mg twice daily.
  Oral solution (Syrup, chlorphenamine, 2mg/5mL) 2.5ml twice daily.
- Child 2-5 years: 1 mg 4 to 6 times daily, maximum 6 mg daily.
  Oral solution (Syrup, chlorphenamine, 2mg/5mL) 2.5ml 4 times daily.
- Child 6-12 years: 2 mg 4 to 6 times daily, maximum 12 mg daily.
  Tablets (chlorphenamine, 4 mg) ½ tablet 4 times daily.
- Above 12 years: 4 mg 4 to 6 times daily, maximum 24 mg daily.
  Tablets (chlorphenamine), 4 mg) 1 tablet 4 times daily.

*__Phenergan (promethazine)__ (British National Formulary)

- Child under 2 years not recommended.
- Child 2-5 years : 5-15 mg daily in 1-2 divided doses.
  Oral solution (Syrup, promethazine, 5mg/5mL) 5-15ml daily in 1-2 divided doses.
- Child 5-10 years: 10-25 mg daily in 1-2½ divided doses.
  Tablets (promethazine 10 mg) 1- 2½ tablet in 1-2 divided doses.
- Above 10 years: 25 mg at night, increased to 25 mg twice daily if necessary.
  Tablets (promethazine 25 mg) 1 tablet 2 times daily.

Non-sedating antihistaminica

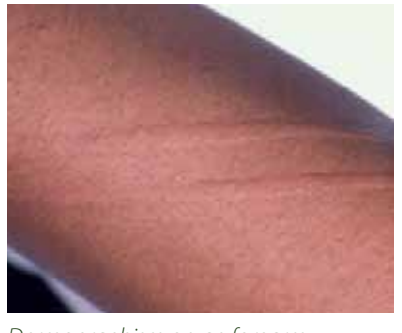
*__Cetirizine (cetirizine)__ (British National Formulary)

- Child under 2 years not recommended.
- Child 2-6 years: 5 mg daily or 2.5 mg twice daily.
  Oral solution (Syrup, cetirizine hydrochloride, 5mg/5mL) 5 mL daily or 2.5ml twice daily.
- Child over 6 years: 10 mg daily or 5 mg twice daily.
  Tablets (cetirizine hydrochloride 10mg) 1 tablet daily or ½ tablet twice daily.

- If the first antihistamine is not effective, it might be necessary to increase the dose, or use a different antihistamine. Sometimes a combination of antihistamines is effective.
- In case of dermographism a combination of H1 (like Piriton) and H2 (like cimetidine) antihistamines is advisable.
- Oral steroids (prednisolon) in moderate dose for a few days can be helpful in severe cases of acute hives. They are not recommended long term because of adverse effects. Topical steroids like betamethason cream might be used twice daily for a short period in the case of severe itching.
- Avoid the use of aspirin, codeine and nonsteroidal anti-inflammatory drugs like ibuprofen.

Clinical pictures

Urticarial wheals on the back of an 18 year old girl



Dermographism on an forearm

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Papular urticaria

Epidemiology

Papular urticaria is regularly seen among schoolchildren in sub-Sahara Africa, especially in countries with a hot and humid climate.[1-3] The prevalence rate in Europe and the USA is unknown but it tends to be more evident during spring and summer months.[4] Papular urticaria are mainly seen among children between the age of 2 and 12.

Etiology and pathogenesis

Papular urticaria is a hypersensitive reaction to contact with arthropods, especially insects such as mosquitoes, fleas, mites, flies and bedbugs.[4;5] A type I hypersensitivity reaction plays a role in the pathogenesis of papular urticaria but delayed type (type IV) reactions are more important. Children eventually outgrow this disease, probably through desensitization. There may be a relation with atopy and poverty.

Clinical findings

The classic presentation of papular urticaria includes crops recurrent pruritic papules and papulovesicles and varying degrees of local edema. Individual papules may surround a wheal and display a central point. Scratching causes erosions and ulcerations, so secondary pyoderma is common.[8;9]

Differential diagnosis

- Insect bites
- Impetigo
- Scabies

- Dermatitis herpetiformis
- Papular pruritic rash (in HIV infection)

Management

Prevention: use insect repellents and impregnated bed nets.

- Mild topical steroids like hydrocortisone 1% two times daily.
- Topical antipruritics such as calamine lotion. Gels or lotions containing menthol or camphor may also be used sparingly in children. Do not use in infants.
- Systemic sedating antihistamines like piriton or promethazine can be tried for relief of the itching. For dosages see urticaria.
- In case of a secondary infection oral antibiotics like cloxacillin or erythromycin can be given.For dosages see impetigo.

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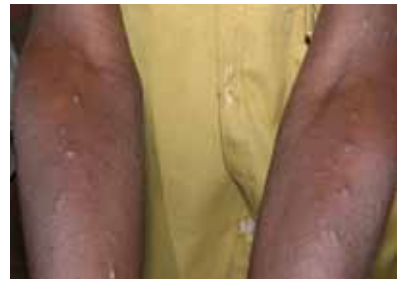
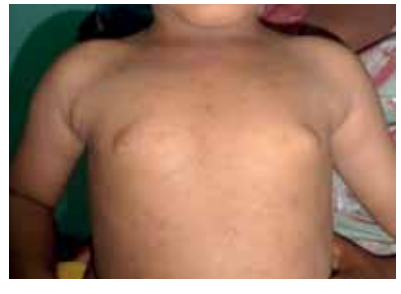
Clinical pictures



Severe itching papules in a Ghanaian schoolboy…



Papular urticaria in a young child

Skin conditions

Keratosis pilaris

Epidemiology

Keratosis pilaris is a common and harmless condition of keratinized hair follicles, especially among children.[1;2] It can also be seen as a symptom of the skin disease ichthyosis vulgaris and considered a symptom of atopy. It is more common in people who have a dry skin, or who have eczema.[3]In the USA between 50-80% of children, the majority of them female, are affected. Among schoolchildren in sub-Sahara Africa and other parts of the world these numbers are much lower.[2;4-6]

Etiology and pathogenesis

This disorder is characterized by grouped, horny, keratotic follicular papules predominantly located on the extensor surfaces of the proximal limbs, the posterolateral upper arms and anterior thighs. It is usually asymptomatic, sometimes slightly itchy especially when the skin is dry, and it may be disturbing cosmetically. Treatment is marginally effective and only provides temporary relief. The cause is unknown but there is hyperkeratinization which is partly inherited. This skin condition seems to run in families, which is consistent with autosomal dominant transmission. Ichthyosis vulgaris is caused by a mutation in the filaggrin gen and there is a close relationship with dry skin, allergies and eczema.[7-

Clinical findings

Numerous small, rough papules around hair follicles on the upper arms, legs, and buttocks can be seen, leading to a "chicken skin" appearance. Inflammation can be present and scratching can cause secondary infection. In the dark skin it often leads to hyperpigmentation around the follicle. Keratosis pilaris tends to fade slowly with age.[1;8]

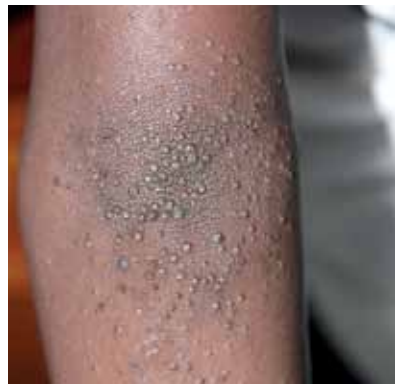
Differential diagnosis

- Acne
- Milia
- Folliculitis
- Xerosis cutis
- Pityriasis rubra pilaris
- Lichen planopilaris

Management

- Explain to the patient that it is a chronic skin condition and it can be a part of other skin diseases like ichthyosis vulgaris. Improvement often takes months and the bumps are likely to come back.
- To prevent excessive dryness the skin should be treated regularly with an emollient cream or ointment like aqueous cream, emulsifying ointment, creams or ointments containing lactic acid 5%, coco butter or shea butter.
- Topical treatment with keratolytic ointments 3%-5% salicylic acid or ureum in the same dosage. In case of inflammation topical mild / moderate steroid ointments like hydrocortisone 1% or triamcinolon acetonide 0.1% can be used two times daily.
- Scrubbing the skin, eg. with a pumice stone.

Clinical picture



Hyperkeratotic papules…..."Chicken skin"

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Xerosis Cutis / Dry skin

Epidemiology

A very dry skin xerosis cutis or asteatosis cutis has been seen in several studies among schoolchildren in sub-Sahara Africa. Frequent washing with soap due the hot, humid climate and subsequent sweating, could explain the high prevalence.[1-4]

Etiology and pathogenesis

The etiology of xerosis cutis is multifactorial. The role of the barrier function of the stratum corneum is important. When the barrier is impaired the skin will be dry because of trans-epidermal water loss and will be more vulnerable for both infectious and inflammatory skin diseases.[5] Several studies suggest that black skin has a higher trans-epidermal water loss than light skin types.[6]

Clinical findings

Dry skin is characterized by a dull color, rough texture and elevated number of ridges.[7;8] Dry skin often itches and could lead to prurigo simplex and eventually to secondary infection; it can also trigger or worsen eczema.

Differential diagnosis

- Allergic contact dermatitis
- Nummular dermatitis
- Scabies

Management

- The use of soap and the frequency of washing should be reduced. The skin should be treated twice daily with an emollient cream or ointment like aqueous cream, emulsifying ointment, coco butter or shea butter.

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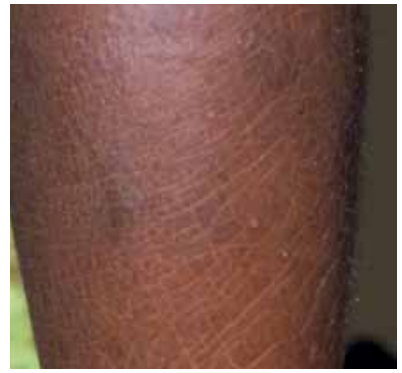
Clinical picture



Dry, rough skin with some superficial cracking (elevated ridges)

Pityriasis alba

Epidemiology

Pityriasis alba occurs mainly in infants, children and adolescents and is more often diagnosed among children with a darker complexion but may occur in individuals of all skin types.[1] It is seen more frequently among male than female and among eczema patients.[2] Prevalences of 8.4 % in India, 5,4 % in Ethiopia and 13.1 % (among children with eczema) in Nigeria have been published.[3-5]

Etiology and pathogenesis

The etiology and pathogenesis are still poorly understood. Recent studies have found direct correlations between the incidence of pityriasis alba and atopy, the amount of sun exposure, and the frequency of bathing. Because it is usually asymptomatic, findings are often incidental. Without intervention, the lesions can persist for months to years and the hypo pigmentation usually does not clear with steroids but will clear in time.[6;7] There is no difference in the number of melanocytes between lesional and normal skin which can be of help when diagnosing and differentiating pityriasis alba from other skin disorders with hypo pigmentation.[1]

Clinical findings

Pityriasis alba is a skin disorder characterized by asymptomatic, hypo pigmented, slightly scaling patches with unclear margins. It is one of the minor features of eczema and is primarily seen on the face and the trunk. Although treatment with emollients and mild topical corticosteroids may accelerate the repigmentation, they have limited efficacy.

Differential diagnosis

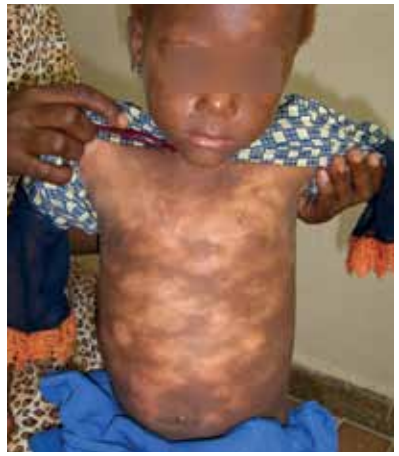
- Leprosy
- Vitiligo
- Pityriasis versicolor

Management

- Explain that the condition is not serious and will disappear in time.
- The skin can be treated regularly with an emollient cream or ointment like aqueous cream, coco butter or shea butter.
- Apply a mild topical corticosteroid cream like hydrocortisone 1% in case of inflammation.
- If available topical calcineurin inhibitors (TCI) like tacrolimus (0.03% and 0.1% ointment) or pimecrolimus (1% cream) may be used. The advantage is that they don't cause cutaneous atrophy.[2]

Clinical pictures

Hypopigmented macules on the trunk



Hypopigmented macules in a patient with eczema

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