Dermatophytosis encompasses several distinct clinical entities, namely *tinea capitis* (scalp ringworm), *tinea corporis* (ringworm of glabrous skin), *tinea cruris* (ringworm of the groin), *tinea unguium* or onychomycosis (ringworm of the nail), *tinea pedis* (ringworm of the feet), *tinea barbae* (ringworm of the beard), and *tinea manuum* (ringworm of the hand).

Dermatophyte fungi causing *tinea capitis* can be divided into anthropophilic and zoophilic organisms. Anthropophilic fungi grow preferentially on humans, and the most common type forms large conidia of approximately 3-4 mm in diameter within the hair shaft (endothrix). Common causes of endothrix infection include *Trichophyton tonsurans*, *T. schoenleinii* and *T. violaceum*. Zoophilic fungi are acquired through direct contact with infected animals (e.g. puppy, kitten). Smaller conidia of approximately 1-3 mm in diameter extend around the exterior of the hair shaft (ectothrix). Ectothrix infection is caused by *T. verrucosum*, *T. mentagrophytes*, and all *Microsporum* species.

*Tinea capitis* occurs occasionally in other age groups. The fungistatic properties of post-pubertal sebum may repel dermatophytes.

The following morphological presentations have been described: black dot type, seborrhoeic dermatitis type, gray patch type, yellow patch type, yellow patch type, pustular type, annular type, kerion, favus and dermatophytic mycetoma (Figures 1-6). Combinations of these lesions are not uncommon. The common denominator of all types is the presence of hair loss. Pruritus is usually minimal. Infected hairs are brittle, and by the third week, broken hairs are evident. Hair loss is subtle in the seborrhoeic type. Secondary impetigo may complicate any form of *tinea capitis*. Regional occipital lymphadenopathy is not uncommon. Impetigo of the scalp should always arouse suspicion of underlying *tinea capitis*. Other causes of scalp impetigo in this age group include *pediculosis capitis*, scabies, atopic dermatitis and primary impetigo.

Dermatophyte infection of the scalp can usually be diagnosed clinically. Additional diagnostic methods include a potassium hydroxide (KOH) preparation and microscopy, culture on Sabouraud dextrose agar (SDA), Wood’s lamp examination and skin biopsy.

Infection may resolve spontaneously at puberty. Treatment using X-ray epilation was reported in 1904. Griseofulvin became available in the
1950s and is the treatment of choice at a dosage of 10-25 mg/kg/day for 6-8 weeks. Drug absorption is enhanced when fatty food is taken simultaneously (e.g., glass of milk). There are three reasons for treatment failure with griseofulvin: dosage too low, treatment duration too short, and, failure to take the drug with a meal. The drug accumulates in keratin of the horny layer, hair, and nails, rendering them resistant to invasion by the fungus. Treatment must continue long enough for infected keratin to be replaced by resistant keratin, usually 4-6 weeks. Resistant strains of dermatophytes are rare.

Contraindications include documented hypersensitivity, porphyria and hepatic cellular failure. Griseofulvin may decrease the hypoprothrombinemic activity of warfarin (adjust dose); coadministration decreases contraceptive effects, resulting in breakthrough bleeding, amenorrhea, or unintended pregnancy; may reduce effects of cyclosporine and salicylates; barbiturates may decrease griseofulvin effects. The oral form is embryotoxic and teratogenic to pregnant rats; therefore, do not prescribe for women contemplating pregnancy. Adverse effects are reported occasionally, including headache, nausea, fatigue, abdominal discomfort, or transient rash; less common adverse reactions include urticaria, diarrhoea, and photosensitivity; may precipitate acute intermittent porphyria and systemic lupus erythematosus in predisposed individuals.

Alternative treatment methods include itraconazole (Sporanox®), terbinafine (Lamisil®) and fluconazole (Diflucan®). Although oral ketoconazole (Nizoral®) is an acceptable alternative, the risk of hepatotoxicity is significant. Topical treatment is usually ineffective. Oral steroids may help reduce the risk for and extent of permanent alopecia in the treatment of kerion. Topical corticosteroids should be avoided. Povidone-iodine (Betadine®) or selenium sulphide shampoo may diminish the spread of spores. The shampoo is applied twice weekly for 15 minutes for 4 consecutive weeks.

Clinical presentation, diagnosis and treatment

Black dot *tinea capitis* refers to an infection with fractures of the hair leaving the infected dark stubs visible in the follicular orifices. Closely shaven hair may simulate black dot *tinea capitis* (personal observation). Black dot *tinea capitis* is caused by *T. tonsurans* and *T. schoenleinii*.

A kerion is an abscess-like lesion studded with pustules. Lesions heal with scarring and permanent alopecia. Kerion is commonly caused by *T. tonsurans*.

Favus is caused most commonly by *T. schoenleinii* and occasionally by *T. violaceum* or *Microsporum gypsum*. Yellow, saucer-shaped crusts termed scutulae surround infected hair follicles. Favus heals with scarring.

Dermatophytic mycetoma is uncommon and is characterised by the presence of one or more reddish nodules on the scalp. Histology shows granules composed of masses of dermatophytes, usually *T. rubrum*, in a background of inflammation.

Yellow patch *tinea capitis* is characterised by one, a few or several patches of hair loss covered fully or partially by yellowish scale-crust.
Secondary infection (= impetigo) by Staphylococcus aureus or beta haemolytic streptococci causes this appearance. Regional lymphadenopathy is common.

Gray patch tinea capitis is an ectothrix infection and by far the commonest presentation seen at the Dermatology Clinic at Tygerberg Hospital. This form is characterised by one, a few or several patches of hair loss, of variable shape and size with lack of inflammation. Fine, grayish scales cover the surface of these lesions. Hairs in the involved area are dull, grayish, discoloured and broken off.

Seborrhoeic dermatitis-like tinea capitis shows more or less diffuse scaling of the scalp. Hair loss is often subtle. Seborrhoeic dermatitis is extremely uncommon in this age group.

The pustular type of tinea capitis refers to the presence of one or more areas of hair loss, of variable shape and size, peppered with pustules, scaling and scale-crust.

The annular type is characterised by papules or pustules forming a ring that may coalesce with other infected areas. Combinations of these morphological presentations are not uncommon.

Iod reactions are manifestations of the immune response to dermatophytes. These reactions occur at a distant site and are devoid of organisms. Iod reactions may be triggered by antifungal treatment. These patients have a strong delayed-type hypersensitivity reaction to intradermal dermatophyte antigens. These eruptions may be vesicular, especially of the hands and feet, dermatitis-like, annular (e.g. erythema annulare centrifugum) or nodular (e.g. erythema nodosum).

The causative fungal organisms of tinea capitis destroy hair and pilosebaceous structures, resulting in severe hair loss and scarring alopecia. The disease is detrimental, both physically and mentally, to children who are affected. Young patients with itchy scalp and patchy or total hair loss frequently are ridiculed, isolated and bullied by classmates or playmates. In some cases the disease can cause severe emotional impairment in vulnerable children and can destabilise family relationships.

Four diagnostic methods are utilised in the diagnosis of tinea capitis

Potassium hydroxide and microscopy

The belly of a number 15 blade is applied to the surface of a scaly patch. Scales are gently scraped off onto a clean glass slide. A drop of 20% KOH is placed next to the material and thereafter thoroughly mixed. The preparation is left for 15-20 minutes. Gentle heat may be applied. A coverslip is placed on the glass slide and viewed with a microscope (x 40 magnification). Rubblings (with a moist gauze pad or toothbrush), pluckings or clippings from lesions may be prepared similarly. The presence of fungal hyphae and spores within (endothrix) or around (ectothrix) hair shafts is diagnostic.

Culture on Sabouraud's dextrose agar

Material is collected as for microscopy. Scales, crusts and/or hair are placed between two glass sides, taped at the ends and despatched to the laboratory. This material is incubated on SDA + cycloheximide (suppresses the growth of environmental contaminant fungi) and SDA + chloramphenicol (to prevent bacterial overgrowth). Most dermatophytes can be identified within 2 weeks. Identification depends on gross colony and microscopic morphology.

Wood's lamp examination

Wood's lamp is a source of long wave (365 nm) ultraviolet light. Hairs infected by M canis, M audouinii and M verrucosum fluoresce a bright green to yellow-green color. Hairs infected by T schoenleini may show a dull green or blue-white color. T verrucosum exhibits a green fluorescence in cow hairs, but infected human hairs do not fluoresce. T violaceum, the causative fungus of grey patch tinea capitis, is Wood's light negative. Wood's light is not used in the Department of Dermatology.

Scalp biopsy

This procedure is seldom employed in the diagnosis of tinea capitis. Hair loss, not responding to treatment, may be an indication for skin biopsy. Dermatophytes may be visualised on routine staining, but a PAS&D stain facilitates identification of fungi. In endothrix infection, spheric-to-box-like spores are found within the hair shaft. In ectothrix infection, organisms form a sheath around the hair shaft. The cuticle is destroyed. Fungal elements may also be present in the epidermis and dermis.

In the differential diagnosis of tinea capitis one should consider alopecia areata, primary impetigo, lupus erythematosus, psoriasis, seborrhoeic dermatitis, secondary syphilis and trichotillomania.

Alopecia areata is characterised by patches of complete hair loss, of variable number, shape and size. Inflammatory changes are absent. The examination mark hairs seen in alopecia areata, in which broken hairs taper from the fractured end towards the skin surface, are pathognomonic. Alopecia areata is an autoimmune disease. Regrowing hairs are usually thin and non-pigmented.

Primary impetigo is caused by S aureus or group A beta haemolytic streptococci. Lesions show pustulation and honey-coloured crusts. Hairs tend to be firmly seated in impetigo. Regional lymphadenopathy is common. Skin lesions of lupus erythematosus show atrophy, scaling and follicular plugging. Hair loss is permanent.

Psoriasis shows patches of erythema with silvery scaling. Hair loss is uncommon. Hairs are not broken. Lesions may be present elsewhere, such as the elbows, knees and lower back.

Seborrhoeic dermatitis, characterised by greasy scaling and variable hair loss, is uncommon in childhood. Seborrhoeic dermatitis may also involve the ears, eyebrows, paranasal area, chin, anterior chest, back and intertriginous areas. In seborrhoeic dermatitis hairs are not broken.

Patchy hair loss occurs in secondary
The drug is safe in pregnancy but safety for use during pregnancy has not been established. Safety for use during pregnancy may occur when administered concurrently. Safety for use during pregnancy has not been established.

Patients should be followed up clinically, by KOH-preparation and microscopy, by Wood's light examination, and mycologically (= culture). Children receiving treatment may attend school. Haircuts, shaving of the head, and wearing a cap during treatment are not necessary.

Deterrence/prevention

Asymptomatic carriers should be detected and treated, since they are the continuous source of infection. Siblings and playmates of patients should avoid close physical contact and sharing of toys or other personal objects, such as combs and hairbrushes, since organisms can spread from one person to another and infectious agents can be transported to different classrooms within the same or different schools. Shared facilities and objects also may promote spread of disease, both within the home and classroom.

**THE ROLE OF PHARMACIST COUNSELLING IN PREVENTING ADVERSE DRUG EVENTS AFTER HOSPITALISATION**

In March 2006, the Archives of Internal Medicine published a paper reporting on a recent randomised trial conducted at a large teaching hospital.

The authors of the paper recognised that hospitalisation and subsequent discharge home often involve discontinuity of care, multiple changes in medication regimens, and inadequate patient education, which can lead to adverse drug events (ADEs) and avoidable health care utilisation. Their objectives were to identify drug-related problems during and after hospitalisation and to determine the effect of patient counselling and follow-up by pharmacists on preventable ADEs.

Patients in the intervention group received pharmacist counselling when they were discharged from hospital, as well as a follow-up telephone call 3 to 5 days later. Inter-ventions focused on clarifying medication regimens, reviewing indications, directions and potential side effects of medications, screening for barriers to adherence and early side effects, and providing patient counselling and/or physician feedback when appropriate.

Pharmacists observed the following drug-related problems in the intervention group:

- unexplained discrepancies between patients' pre-admission medication regimens and discharge medication orders in 49% of patients
- unexplained discrepancies between discharge medication lists and post-discharge regimens in 99% of patients
- medication nonadherence in 23%.

Comparing trial outcomes 30 days after discharge, preventable ADEs were detected in 11% of patients in the control group and 1% of patients in the intervention group (P = .01). No differences were found between groups in total ADEs or total health care utilisation.

The authors concluded that pharmacist medication review, patient counselling, and telephone follow-up were associated with a lower rate of preventable ADEs 30 days after hospital discharge.

REFERENCES