The itchy scalp

Many dermatological conditions can cause scalp pruritus of varying degrees, but it can be

difficult to make an accurate diagnosis as signs are often nonspecific.

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Scalp pruritus is a common presentation to GPs. This article outlines the clinical features and management of some common dermatological conditions that cause scalp pruritus, as well as other important but more rare causes of this problem.

Common causes of scalp pruritus Seborrhoeic dermatitis and pityriasis capitis Clinical features

Seborrhoeic dermatitis is an inflammatory dermatitis of the scalp, face, chest and upper back. In its less severe form it is known as pityriasis capitis or 'dandruff'. Both conditions are associated with an increase in the numbers of the yeast *Malassezia furfur* (previously named *Pityrosporum ovale*) on the skin and in hair follicles.

Seborrhoeic dermatitis commonly occurs in infancy and usually resolves spontaneously. Cases are rare after infancy and before puberty but become more common during adolescence. Seborrhoeic dermatitis may persist into adulthood or even first appear during this time. Prevalence in the general population is about 1 to 3%. However, it is more common and more severe in individuals who have HIV infection or a neurological disorder, especially parkinsonism or paraplegia or quadriplegia.

Clinically, seborrhoeic dermatitis is characterised by fine, loosely adherent scales on the scalp and retroauricular regions (Figure 1a). There may be associated blepharitis and facial rash (Figure 1b). Other areas, such as the axillae and groin, may show a glazed erythema with little scaling. Seborrhoeic dermatitis is often associated with secondary impetiginisation of the scalp.

Treatment

Both seborrhoeic dermatitis and pityriasis capitis are chronically relapsing conditions. It is important to remind the patient that treatment is only suppressive and that the condition is likely to return once treatment is stopped.

In mild cases, regular use of a shampoo containing one of the following is usually effective: selenium sulfide (Selsun Preparations), pyrithione zinc (Fongitar, Head and Shoulders Intensive Solutions, Neutrogena T/Gel Daily Control), 1 to 2% ketoconazole (Hexal Konazol 2% Shampoo, Nizoral Treatment, Sebizole Shampoo), ciclopirox olamine (Stieprox Liquid), 2% miconazole (HairScience

- Scalp pruritus may be a feature of many common dermatological conditions including seborrhoeic dermatitis, psoriasis, pediculosis, eczema and lichen simplex chronicus. In some patients, androgenetic alopecia is associated with itching and irritation of the scalp.
 - Scalp pruritus can also be a prominent feature of contact dermatitis and rare conditions such as dermatitis herpetiformis, lichen planopilaris and trigeminal trophic syndrome.
 - Psychological issues, particularly anxiety, may exacerbate or manifest as scalp pruritus.
 - Identifying distinguishing features in the patient's history and examination to make an accurate diagnosis is important. Sometimes a therapeutic trial is required before the diagnosis is known.
 - A definitive cause for scalp pruritus may not be found in some cases, but empirical topical treatments may still help to alleviate symptoms.

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Anti-Dandruff Shampoo) or tar (Fongitar, Ionil T, Neutrogena T/Gel Therapeutic Shampoo, Polytar Liquid, Sebitar). The shampoo should be massaged into the scalp and left for approximately five minutes before rinsing out. Washing the shampoo out too soon or not using it often enough are the usual causes of treatment 'failure'.

For more difficult cases, a topical antifungal preparation such as a ketoconazole cream (Dakta-Gold, Nizoral Cream) may be used in addition to a shampoo. The cream is mixed with water and massaged into the scalp at night and washed out the next morning. Topical corticosteroid lotions such as those containing mometasone furoate (Elocon, Novasone) or methylprednisolone aceponate (Advantan) can be useful if there is significant inflammation. Other topical treatments include cade and coconut (cocois) oil pomades.

In patients who also have seborrhoeic dermatitis of the face and chest, treatment may be supplemented with a mild topical corticosteroid (e.g. 1% hydrocortisone), 2.5 to 5% benzoyl peroxide, topical imidazole antifungals (clotrimazole, miconazole [Daktarin, Eulactol Antifungal Spray, Resolve Solution], ketoconazole, bifonazole [Canesten Once Daily Bifonazole Cream 1%, Mycospor]), pimecrolimus cream (Elidel) or lithium succinate ointment, extemporaneously compounded. A short course of oral ketoconazole (Nizoral, 200 mg daily for 10 days) may be considered for stubborn cases. Caution is recommended when prescribing oral ketoconazole due to the risk of hepatotoxicity; prolonged courses should be avoided.

Psoriasis

Clinical features

Psoriasis is a chronic relapsing, inflammatory papulosquamous dermatosis. The pathogenesis involves abnormal hyperproliferation, rapid cell turnover and loss of normal differentiation of the skin. Scalp involvement occurs in at least 50% of affected individuals and may occasionally be the sole manifestation. Generally, psoriasis is not thought to be a particularly pruritic condition but when the scalp is involved, itch is often a prominent feature.

The clinical presentation of scalp psoriasis can take several forms. In milder cases there may only be patchy or diffuse scaling without any specific features and it may resemble pityriasis capitis. Some patients will have classical psoriatic plaques





on the scalp, which are often hyperkeratotic plaques covered with thick scale that develop just outside the hairline, especially in the retroauricular and occipital zones.

In severe cases, pityriasis amiantacea may occur. This consists of single or overlapping asbestos-like scale crusts encasing and binding down tufts of hair. Forcible peeling of the scales removes the hair, revealing an erythematous patch of alopecia. While pityriasis amiantacea can be indicative of psoriasis, it may also occur in seborrhoeic dermatitis, eczema, or lichen simplex. Scalp psoriasis may be differentiated from these conditions by the presence of thick, large silvery scales on the scalp as well as the presence of psoriatic plaques on other areas of the body (Figures 2a to c). Psoriasis of the scalp seldom results in alopecia. Figures 1a and b. a (above). Seborrhoeic dermatitis, characterised by fine, loosely adherent scales on the scalp. b (left). Facial rash associated with seborrhoeic dermatitis.

continued









Many triggers may exacerbate the severity of psoriasis. They include stress, streptococcal pharyngitis and certain medications such as lithium, beta blockers and NSAIDs. The 'Koebner phenomenon' is the development of psoriasis at sites of trauma. Indeed, vigorous scratching or combing of the hair may induce scalp psoriasis and perpetuate the cycle of pruritus.

Treatment

Treatment with tar shampoos may be effective for mild cases. If itching is very problematic, a topical corticosteroid lotion such as mometasone furoate lotion or methylprednisolone aceponate lotion can be applied to the scalp after shampooing and left on overnight. A keratolytic preparation is required for patients with thicker scales. This is usually left on overnight and washed out the next morning. A typical preparation may include a combination of a tar (e.g. 3 to 10% liquor picis carbonis) and 3 to 5% salicylic acid in aqueous cream, Figures 2a to c. Large silvery scales of psoriasis developing just around the hairline in the occipital (a, above left) and retroauricular (b, above right). zones. c (left). Patients with scalp psoriasis often also have psoriatic plaques on other areas of the body.

extemporaneously compounded. Dithranol at a concentration of 0.1 or 0.2% can be added to the extemporaneously compounded mixture for overnight application to the scalp in more severe cases. Higher concentrations of dithranol (0.5 to 1%; (DithraSal, Micanol) can also be used as a short-term contact treatment as long as it is washed off within 30 minutes of application. For patients who have pityriasis amiantacea, an extemporaneously compounded mineral oil solution containing up to 15% salicylic acid can be prescribed.

Preparations containing dithranol and tars should be used with caution in fairhaired individuals as these can stain the hair. Adult patients can further enhance the treatment's overnight efficacy by wearing a plastic shower cap. The use of shower caps is not suitable for children due to the risk of suffocation. Once the scales have been removed, usually after a few nights' treatment, tar shampoos and topical corticosteroid lotions can be used to reduce the likelihood of the scales reappearing. Systemic treatments such as methotrexate (Methoblastin) can also help with scalp psoriasis but are rarely indicated for scalp lesions alone. Ultraviolet B light and psoralen plus ultraviolet A light (PUVA) treatments are of little benefit as the scalp is shielded by the patient's hair.

Lichen simplex chronicus Clinical features

Lichen simplex chronicus (LSC) is a chronic, pruritic inflammation of a localised area of skin. An initial trigger, such as eczema or stress, initiates an itch–rub cycle and the chronic manifestation is LSC. The condition can occur anywhere on the body, but when it is in the scalp the almost pathognomonic locations are the occipital area or the nape of the neck.

Clinically, the skin is thickened and scaly, and the scale is often very adherent. There may be secondary broken hairs or hair loss adjacent to the affected area due to rubbing. Secondary infections are common.

The important differential diagnosis for LSC is scalp psoriasis. However, a distinguishing feature of LSC is the tendency of the plaque to extend some distance beyond the hair margin – for example, onto the neck before reaching normal skin (Figure 3). In addition, in patients with psoriasis, other skin lesions may be present elsewhere on the body.

Treatment

The primary objective in treating LSC is to stop the itch–rub cycle. Potent topical corticosteroids such as betamethasone dipropionate ointment (Diprosone OV, Eleuphrat) are required. Occasionally, intralesional triamcinolone (Kenacort) can be helpful. Managing any underlying stress or psychological issues is also important in preventing recurrence of the condition.

Pediculosis capitis (head lice) Clinical features

Head lice can be easy to miss if the numbers of egg cases or lice are not great;

continued

indeed, the number of adult lice is fewer than 10 in most infestations. Clinically, egg cases, or nits, may be seen with the naked eye or, more easily, with a Wood's lamp. They are differentiated from dandruff and peripilar hair casts by their immobile nature on the hair shaft. Pruritus is often worse in the occipital area, where infestations may be heaviest. There may be secondary impetiginisation, and hairs may be matted down by exudates.

Treatment

Topical malathion (Maldison) and permethrin (Pyrifoam Lice Breaker, Quellada Head Lice Treatment) are the mainstays of therapy. Two applications – initially and then seven to 10 days later – are usually required because not all the eggs will be killed by the first treatment. Malathion is absorbed in the hair shaft, making it resistant to re-infection for six weeks. Blowdrying should be avoided as heat degrades some pediculocides. Residual nits should be removed by combing with a finetoothed comb. The entire family should be treated at the same time.

A 'nonchemical' option involves the application of hair conditioner and use of a lice comb. Head lice breathe through small openings along their abdomens called spiracles. Coating the hair, and therefore the louse, in a thick and occlusive preparation such as hair conditioner obstructs these openings and consequently shuts down the louse's breathing for about 20 minutes. This method does not kill the lice but it does slow them down sufficiently so that they can be caught in a comb. To be successful, the process needs to be repeated on a nightly basis for at least two weeks but often for much longer. Many lice will be missed if a head lice comb is used on a dry scalp as the lice are able to move at great speeds across the scalp, thereby avoiding capture in the comb. The conditioner method will not kill or remove the eggs, but some good quality lice combs will be able to remove these.

In the last few years, the emergence of lice that are resistant to conventional treatments has led to the expansion of treatment options to include oral trimethoprim 80 mg/sulfamethoxazole 400 mg (Bactrim, Resprim, Septrin). This is given twice daily for three days and repeated 10 days later for a further three days. More recently, oral



ivermectin (Stromectol) $200 \mu g/kg$ has also been prescribed off-label, with a repeated dose given 10 days later.

Other less common causes of scalp pruritus Contact dermatitis

Scalp pruritus can be caused by a contact dermatitis, either due to an irritant or an allergen. The scalp is relatively resistant to contact dermatitis due to a rapid epidermal turnover and a thick epidermis and stratum corneum. It is also well protected by hair. Often the ears, forehead, neck or face are affected first.

Potential irritants include bleaching agents (the most common irritant), agents containing thioglycolates for permanently waving hair, and blow-drying the hair. Major allergens are found in hair dyes, bleaches, permanent wave solutions and hair creams.

Clinically the scalp may be erythema-

tous, vesicular and weeping. Patch testing is generally useful for diagnosing the culprit. In addition to avoiding the 'trigger', topical corticosteroid treatment is usually all that is needed. Oral prednisolone can be used for severe cases.

Endogenous eczema/atopic eczema

Scalp involvement in eczema usually occurs in the setting of a generalised flare rather than as an isolated presentation. The scalp may become diffusely red, vesicular and weeping. The hair may become matted, and temporary alopecia may occur.

Treatment with topical corticosteroids can be considered. An alcohol-based lotion is useful for the thickened, matted hairs but can be strongly irritating. A potent corticosteroid cream (betamethasone dipropionate, methylprednisolone aceponate or mometasone furoate) diluted with water is a more acceptable alternative.

In eczematous flares there is usually increased colonisation or infection with *Staphylococcus aureus*, and successful treatment often requires concurrent oral antibiotics.

Tinea capitis

Fungal infections of the hair are common in children but seldom seen in adults. In



Figure 3. Lichen simplex chronicus. Plaques tend to extend some distance from the hair margin.

continued





Figure 4. Tinea capitis caused by *Trichophyton soudanense*.

Australia *Microsporum canis* is the pathogen most commonly isolated, followed by *Tricophyton tonsurans*. There has also been a recent increase in cases of tinea capitis caused by *Trichophyton violaceum*, *Trichophyton soudanense* (Figure 4) and *Microsporum audouini*, as this condition is endemic among children from Africa.

Clinical diagnosis of tinea capitis can be difficult as symptoms are varied. It may manifest as minimal pruritus and fine scaling, or as annular lesions or grey patches. Patients with severe disease may have alopecia or show inflammatory kerion formation. Wood's lamp examination is not always helpful as only certain fungi (*M. canis*) fluoresce. In suspicious cases, it is crucial to confirm the diagnosis with skin scrapings and plucked hair for fungal microscopy and culture. A clean toothbrush brushed on different sites of the scalp can also be cultured. Family members should be screened for infection as well.

Treatment of tinea capitis requires oral antifungals as topical therapy alone rarely works. Traditionally, oral griseofulvin (Grisovin) is given for a minimum of six to eight weeks and sometimes for as long as 16 weeks, until a clinical and mycological cure is achieved. The recommended

Figure 5. Follicular papules and pustules of Malassezia folliculitis.

dose is 10 to15 mg/kg/day for ultra-micronised formulations (not currently available in Australia) or 20 to 25 mg/kg/day for micronised formulations (Grisovin). Common side effects include gastrointestinal upset and headaches, while sun sensitivity and even mild hepatitis can occur on occasions. Compliance is an issue given the long duration of therapy required to ensure eradication. This problem is often overcome in young children by combining the crushed tablet with a spoonful of ice-cream. The concurrent use of ketoconazole or selenium sulfide shampoo is also helpful as these agents reduce the infectivity of the dermatophyte.

Newer agents such as oral itraconazole (Sporanox), fluconazole and terbinafine (Lamisil, Tamsil, Terbihexal, Zabel) may be used for a shorter duration (e.g. two to four weeks) with reasonable efficacy. However, a prescription issued under the Pharmaceutical Benefits Scheme requires initial treatment failure with griseofulvin.

Urticaria

The scalp may become involved in urticaria, although typical urticarial wheals are usually also present in other skin areas. The pruritus can be intense, but excoriations are uncommon as the patient tends to rub rather than scratch. Investigation for any underlying triggers – for example, foods, parasites or drugs – is important.

Symptomatic treatment is primarily with antihistamines. The traditional sedating antihistamines are useful for night-time sedation, while second generation antihistamines (e.g. cetirizine [Alzene, Zilarex, Zyrtec], loratadine, desloratadine [Claramax], fexofenadine) are as effective with minimal cholinergic side effects. Occasionally, a course of oral corticosteroids for no more than four weeks' duration may be prescribed.

Malassezia folliculitis

A pruritic scalp associated with follicular papules and pustules (Figure 5) raises the possibility of Malassezia folliculitis (previously known as Pityrosporum folliculitis), a benign disorder caused most often by the *Malassezia globosa* yeast. It is more common in tropical than temperate regions.

The severity of the pruritus can be out of keeping with the physical signs. Microscopy and culture may yield *Malassezia*, but as colonisation of hair follicles by the yeast is not abnormal, the best way to diagnose the condition is by a trial of antifungal medication.

Topical therapy with antifungal shampoo is usually effective, although on occasions a short course of oral ketoconazole, fluconazole or itraconazole may be required.

Acne necrotica

Acne necrotica presents as intensely pruritic papules and pustules with central necrosis or ulceration, typically followed by crusting and subsequent varioliform scarring. It most often occurs around the frontal hairline. Alopecia secondary to the scarring may result.

Tetracyclines are generally effective in inducing remission of acne necrotica, but long-term maintenance therapy is often required.

The itchy scalp

continued



Figure 6. Lichen planopilaris causing an area of scarring alopecia.

Dermatitis herpetiformis

Dermatitis herpetiformis is a rare, chronic, recurrent autoimmune papulovesicular disease that occurs in patients of all ages. It is intensely pruritic. The lesions consist of small erythematous papules, vesicles or wheals in crops that are easily excoriated. The lesions most commonly present on the extensor aspects of the limbs and the scalp is frequently involved. All patients have an underlying gluten-sensitive enteropathy, although this may be asymptomatic. Diagnosis is confirmed with a skin biopsy for histological analysis and direct immunofluorescence. Patients also need to be assessed by a gastroenterologist.

Dermatitis herpetiformis is usually treated with dapsone, topical corticosteroids, and a gluten-free diet.

Lichen planopilaris

Lichen planopilaris is a rare, benign inflammatory disorder affecting the scalp. Early lesions consist of perifollicular erythema, desquamation and rarely violaceous papules. The area of scalp involved is often pruritic. Later there may be follicular keratosis, as well as scarring causing alopecia (Figure 6).

In patients with lichen planopilaris, response to topical corticosteroids is often satisfactory, although some may require systemic therapy.



Figure 7. Trigeminal trophic syndrome.

Scabies

Scalp involvement in scabies only occurs among infants and young children. Other sites that are involved are the palms of the hand, soles of the feet, neck and flexural areas. The lesions may consist of papules, vesicles, pustules, nodules and the pathognomonic scabetic 'burrow'. Pruritus associated with scabies is often intense and is due to the host's immune response to the scabies products.

Treatment consists of two applications, one week apart, of topical permethrin cream (Lyclear, Quellada Scabies Treatment) and careful hot washing and tumble drying of bedclothes. Oral ivermectin may also be used off-label. It is essential to treat both the infected individual and all those who have been in contact with him or her.

Trigeminal trophic syndrome

Previous herpes zoster, cerebrovascular disease or other conditions may cause an area of altered sensation, including pruritus, within the area of skin innervated by the trigeminal nerve. This is frequently followed by repeated iatrogenic trauma, ulceration and trophic changes (Figure 7). Trigeminal trophic syndrome can be significant in patients with dementia and in elderly patients who sometimes have the compulsion to pick at their skin.

The mainstay of treatment is protection

of the damaged area, although plastic surgery may be required in severely affected patients.

Psychosomatic disorders

Psychosocial stressors, anxiety or endogenous depression may manifest as various dermatological complaints, including pruritus of the scalp. Direct questioning may reveal such precipitants in the absence of clear organic disease.

Idiopathic pruritus capitis

In patients in whom pruritus in the scalp is not explained, empirical treatment may nevertheless be helpful. Tar-based shampoos can be used, and over-shampooing should be avoided.

Conclusion

Scalp pruritus may be a feature of many dermatologic conditions. It is important to identify distinguishing features in the patient's history and examination to make an accurate diagnosis. Sometimes, a therapeutic trial is required before the diagnosis is known. MT

COMPETING INTERESTS: Dr Ng and Dr Chong: None. Dr Foley is a member of Medical Advisory Boards and has been an investigator in clinical trials for therapeutic agents for Stiefel, Galderma and Schering-Plough.

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