

## Differential Diagnoses of Acneiform Eruptions

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“Pimple” is one of the commonest complaints of dermatology patients. However, there are a number of differential diagnoses of acneiform eruptions other than acne vulgaris. Acneiform eruptions refer to the presence of one or more of the classical features of acne vulgaris. Those are comedones, papules, pustules and nodular cysts. Acne-like disorders can be due to a wide variety of diseases such as infections, drug reactions and growth anomalies. Therefore, history and physical examination are important to help narrowing down the list of differential diagnoses. Occasionally, one needs to perform a skin biopsy in order to get a firm diagnosis. The following entities will be discussed:

### Acne Vulgaris

Acne vulgaris is one of the commonest entities seen in a dermatology clinic. It usually affects adolescents and young adults. However, it is not rare to appear first at late twenties in some patients. Hormonal factor, *Propionibacterium acnes*, follicular hyperkeratinization and sebum secretion are all contributing factors in pathogenesis. Other factors include genetic factor, exposure to substances such as oil, crude tar, chlorinated hydrocarbons (chloracne), comedogenic cosmetics (acne cosmetica); and physical factors such as repetitive occlusion, friction and pressure (acne mechanica). The classic feature of acne is pleomorphic; it includes papulopustules, comedones (open and closed), scarring, and infrequently nodules and cysts in case of severe disease (acne conglobata). Acne excoriee is another variant that is more common in females. The excoriated lesions are the results of a compulsive behaviour, that is, an excessive picking by patients. Acne is not exclusive in children. Nearly 1 in 5 neonates exhibit mild neonatal acne. It is characterized by multiple erythematous closed comedones on the nose, forehead and cheeks with an onset frequently between 0-6 weeks. The pathogenesis is believed to be due to the stimulation of neonatal sebaceous glands from

maternal and infant androgens. The condition usually resolves within 1-3 months without scarring. Infantile acne is seen later in infancy with onset beginning in 3-6 months of age, less common than neonatal acne, characterized by more numerous inflammatory lesions. Scarring is a risk. It affects more male infants, postulated to be associated with precocious secretion of gonadal androgens. There is increased risk of development of severe acne vulgaris later in teenage years. In severe cases, one should investigate for conditions with hyperandrogenism. The main concern in acne is the possibility of scarring and it can be disfiguring in cases of acne conglobata and acne fulminans (the latter condition almost exclusively occurs in teenage boys that is due to an immunological response to *P. acnes*, characterized by fever, arthralgia, myalgia, leukocytosis, painful and ulcerative lesions on the trunk). Diagnosis of acne is usually made clinically. However, in a hirsute female with or without irregular menses, an evaluation for hypersecretion of adrenal and ovarian androgens is needed: total testosterone, free testosterone, and/or dehydro-epiandrosterone sulphate, so as to rule out the diseases such as congenital adrenal hyperplasia and polycystic ovarian syndrome. Treatment of acne vulgaris can be topical, systemic or combined according to the severity of the condition. Topical agents include anti-inflammatory such as clindamycin, erythromycin and benzoyl peroxide. Combined use of topical antibiotic with benzoyl peroxide can reduce the emergence of bacterial resistance. Topical retinoids such as tretinoin and adapalene mainly work as comedolytics and carry mild anti-inflammatory effect. Not only useful in comedonal acne, they are used as maintenance therapy since they can inhibit the formation of microcomedones, the precursor lesions of acne. Therefore, combination therapy (topical antibiotic and/or benzoyl peroxide plus topical retinoids) gives the best result. Azelaic acid is another useful topical agent though it takes longer time to work. It has low level of side effects and carries both

anti-inflammatory and comedolytic effects. For more severe form of acne especially those result in scarring, systemic therapy should be adopted. It includes oral antibiotics, hormonal treatment and systemic retinoid. For oral antibiotics, the options are the tetracycline group (tetracycline, doxycycline, minocycline) and erythromycin. Treatment period should be at least 4-6 months. In general, tetracyclines work better than erythromycin and with lesser frequency of developing bacterial resistance. Hormonal treatment includes oral contraceptive that contains ethinyl estradiol and norgestimate, anti-androgens such as cyproterone acetate and spironolactone. Hormonal therapy can be offered to females only and usually requires even longer period of treatment. Oral isotretinoin is often reserved for severe acne because of the cost. It is a powerful agent because it acts on all the pathogenetic factors of acne, so it offers a cure rate of about 60% after a single course (1 mg/kg/day until the target cumulative dose of 120 mg/kg reached, usually started at a lower dose and then gradually increased). Even if there is relapse, the condition is usually much milder and more easily managed. Repeated courses can be given in refractory cases after an interval of 2 months. However, the side effects of the drug should be well acquainted such as raised lipid levels and teratogenicity. Pregnancy should be ruled out, lipid profile and liver function should be checked before therapy is started and should be monitored intermittently during therapy. Isotretinoin should not be used together with tetracyclines since both medications can induce pseudotumour cerebri.

### **Rosacea**

Rosacea appears similarly to acne vulgaris with papulopustules on the face, but in addition, patients usually have facial flushing and telangiectases. Unlike acne vulgaris, the lesions are mainly located in the central part of the face and there is no comedone. Rosacea is more common in women in their third and fourth decades. Men, however, are affected more commonly with sebaceous and connective tissue hyperplasia of the nose (rhinophyma) or other parts of the face. Associated eye findings may be present. The disease is likely related to a vasomotor instability and therefore, temperature change, sunlight, hot or spicy foods, alcohol or hot beverages can exacerbate the condition. Biopsy is usually not required for making a diagnosis, however, one should always bear in mind the differential diagnoses of flushing disorders such as

carcinoid syndrome and photosensitive diseases such as lupus erythematosus. Erroneous use of topical steroids for prolonged period on the face may also give rise to steroid rosacea. Treatment of rosacea includes sunscreens, avoiding factors provoking facial erythema, topical antibiotics such as metronidazole and oral tetracyclines or erythromycin. The dose of the tetracycline can be started at 1 gm/day, tailed down to the lowest effective dose gradually and then maintained for a few months. Telangiectasia and all "phymas" usually do not respond to topical/oral antibiotics and require laser and cosmetic surgery. Rosacea fulminans, also called pyoderma faciale, is a conglobate, nodular disease with draining sinuses that develops abruptly and almost exclusively in post adolescent women. In this condition, just as in acne fulminans, oral corticosteroid should be started before the introduction of oral isotretinoin.

### **Iatrogenic Acneiform Drug Eruptions**

Iatrogenic acneiform drug eruptions can happen in patients taking oral steroids, androgens, oral contraceptives, isoniazid, lithium, phenytoin, bromides or iodides. The eruption is seen as monomorphous papulopustules located predominantly on the trunk and extremities. It can also happen in anywhere of the skin after prolonged topical use. The eruption usually resolves after the discontinuation of the drug.

### **Perioral Dermatitis**

This is also a disorder of unknown etiology. It often appears in young female population as asymptomatic micropapulopustules and micropapulovesicles with erythematous bases predominantly located around the mouth, characteristically sparing the vermilion border of the lip. It may also affect the perinasal and periorbital areas (periorificial dermatitis). Biopsy is rarely necessary. The etiology is unknown and the suggested causative agents include topical or inhaled corticosteroids, moisturizers, fluorinated compounds, and contact irritants or allergens. Therapy includes cessation of halogenated topical steroids, and initiation of topical antibiotic such as metronidazole plus oral tetracycline or erythromycin for 6 weeks.

### **Infectious Folliculitis**

Infectious folliculitis is an infection of the upper portion of the hair follicle, characterized by a follicular papule, pustule, erosion or crust. The commonest causative

agent is *Staphylococcus aureus*. It commonly infects the beard area (sycosis barbae), trunk and buttock. Diagnosis can be confirmed by Gram-stain and bacterial culture. Treatment includes anti-bacterial soap; topical anti-inflammatory agent/antibiotic; with or without a course of oral antibiotic with coverage of *S. aureus*. Gram-negative folliculitis can complicate patients with acne vulgaris on prolonged antibiotics that presents as sudden deterioration of the acne. In that case, discontinue the current antibiotic and give a course of ampicillin; trimethoprim-sulphamethoxazole or according the result of culture and sensitivity. Fungal infection can also give rise to folliculitis such as in tinea barbae and candidal folliculitis. The latter is not uncommonly seen on the back of hospitalized patients who are feverish and bedbound. Pityrosporum folliculitis is caused by a host reaction to the yeast *Pityrosporum ovale*, a normal human skin commensal. It appears as pruritic follicular papulopustules primarily on the trunk and upper extremities. Treatment with topical antifungals or ketoconazole shampoo usually suffices, if not, itraconazole 100 mg bd can be given for a week. Hot-tub folliculitis is caused by *Pseudomonas aeruginosa*. It happens in healthy individuals after aqueous exposure in hot tubs or physiotherapy pools, presenting as multiple follicular pustules on the trunk. The disease is self-limiting or a course of quinolone can be given.

### **Eosinophilic Pustular Folliculitis**

This is another disease of unknown etiology that usually presents as a recurrent pruritic follicular papules and pustules on the face, neck, trunk, and proximal extremities. Diagnosis can be confirmed by skin biopsy. The disease has been described in immunocompromised patients with HIV and in healthy individuals (known as Ofuji disease). Patients may also demonstrate blood eosinophilia and leukocytosis. Treatment options include topical steroid, systemic corticosteroids, antihistamine, antifungal, isotretinoin and phototherapy.

### **Pseudofolliculitis Barbae**

This is more commonly found in black population. Because of the tight curls in beard, hair often grows back into the skin, causing an inflammatory response, a pseudofolliculitis. *Staphylococcus aureus* secondary infection is common.

The list is not exhausted. Other rare conditions that can be mistaken as acne are lupus miliaris disseminatus faciei (a granulomatous variant of rosacea), papular sarcoidosis, adenoma sebaceum in tuberous sclerosis, benign adnexal tumors such as syringoma and multiple trichepithelioma. In conclusion, an accurate diagnosis of the acneiform eruption should be established before one can give appropriate management to the patient.

## The Non-venereal Skin Conditions in Genital Area

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There are many normal and abnormal skin conditions in the genitalia. Not all the dermatoses seen in the genital region are venereal in origin. Many patients in our venereal clinics actually do not have any venereal exposure. The only reason for referring them to us is the dermatosis occurred in the genital area. In the following discussion, some of the common genital skin conditions will be highlighted in a symptomatic approach. However, some of the dermatoses may present in different forms at different stages, so do not limit the differential diagnosis in this approach as the lesion may change its appearance later on. Dermatitis involving the other part of body may manifest differently in the genital region. There may be less scaling, more maceration or secondary infected; depending on many factors such as humidity and friction.

### Genital Growth

There are many genital growths that may mimic genital wart (GW) and molluscum contagiosum. Pearly penile papules in male and vulval papillomatosis in female are commonly seen. These congenital tiny swellings are arranged in rows regularly around the coronal sulcus or over the labium minorus and introitus. They are only hypertrophic papillae with normal epidermal covering. 5% acetic acid test (acetowhitening) may help to identify the GW but there may be false positive and negative results. The opening of Tyson's glands, located on either side of the fraenum, may look like GW. These are secretory glands of no clinical significance. Fordyce Spots are ectopic sebaceous glands. They may appear on the shaft of penis and scrotum or the labium minorus as multiple small white or yellow spots in submucosa. Syringoma and epidermolytic acanthoma are some examples of benign genital tumours that can be misdiagnosed as GW in the genitalia. Angiokeratoma of Fordyce is a benign vascular neoplasm with hyperkeratotic surface, usually associated with varicose vein of the genital region. They can be found on the scrotum and labia majora. Occasionally bleeding may occur.

Electrocauterisation can usually cure them. Sclerosing lymphangitis occurs as asymptomatic worm-like translucent masses of cartilage-like hardness in or near the coronal sulcus. It is thought that the lymphatics are temporarily blocked. Some cases may follow prolonged or frequent intercourse. However, in the largest series reported, the majority was unexplained although the patients had coitus. The condition resolves within a few weeks and no treatment is necessary. Bowenoid papulosis, which may be preceded by GW, consists of some asymptomatic fleshy-pigmented papules. Histologically, they are squamous cell carcinoma in situ. In female, cervical neoplasia should be screened. Other skin tumours, benign or malignant, can be seen in the genital region. These include squamous cell carcinoma, basal cell carcinoma, malignant melanoma etc. They are usually irregular in shape, color, surface and edge. In case of doubt, a punch biopsy may help to establish the diagnosis. For those normal variants, reassurance is all that required. For pre-malignant or malignant conditions, more radical treatments such as surgery may be needed.

### Inflammatory Dermatitis

Balanitis is the inflammation of glans penis, which can be secondary to traumatic, allergic, irritant or infective causes. Similar condition can occur in vulva, which causes vulvitis. Friction during intercourse, zipfastener injuries is some examples of traumatic balanoposthitis. Fixed drug eruption is an example of allergic reaction that may present as a well-defined inflammatory patch healed with post inflammatory hyperpigmentation in the genitalia. Poor hygiene, deodorant and perfume, detergents and contraceptive medication may cause irritant contact dermatitis in adult while napkin erythema is the example in infant and elderly. For infective causes, candidal infection is more common in diabetic patients and patients taking oral contraceptive pill or systematic antibiotic. The characteristic features include a glazed non-purulent surface with slightly scaly edge and satellite lesions

at the periphery. Microscopy and culture confirm the diagnosis. Scraping should be taken from both the anus as well as the genitalia. Correction of the underlying causes and application of topical anticandidal agents of the polyene or azole group are the usual treatments. Intestinal or urethral reservoir and re-infection account for the 10% failure rate of refractory cases. Other fungal infections can also appear in the genitalia. Treatment is similar to that in candidal infection. The clinical features of anaerobic balanitis include superficial erosions, oedema of the prepuce, foul smelling discharge and inguinal lymphadenopathy. *Bacteroides* species are the commonest anaerobes seen. It responds to metronidazole rapidly. Plasma cell balanitis of Zoon in an uncircumcised man appears as indolent well-defined shiny red to brown plaques on the glans penis with central stippling reminiscent of 'cayenne pepper'. The corresponding vulvitis is rare in female. It is a persisted chronic form of balanitis, which may be mistaken as erythroplasia of Queyrat (a premalignant condition). Diagnosis is established by biopsy, which shows plasma cell infiltration. It responds to gentamycin rather than topical steroid. Circumcision is often curative. Extrammary-Paget's disease can present as some itchy eczematous plaques over the genitalia. If the dermatosis persisted for a long time even after prolonged treatment, a biopsy should be taken.

### **Genital Ulcer (GU) and Necrosis**

Non-venereal causes of GU include trauma, neoplasm, infection and allergy. For pruritic conditions, scratching can induce ulceration and application of inappropriate topical treatment may cause contact dermatitis with ulceration. Tightly attached tapes for the condom catheter drainage systems in geriatric and paraplegic patients may cause painless necrosis or gangrene of the penis. Other traumatic causes of GU are similar to those mentioned in balanitis. When a neoplasm necroses, it may ulcerate with irregular edge and infiltrative base. It usually does not healed spontaneously or with conservative treatment. Behcet's syndrome can present as recurrent genital and oral ulceration. It frequently associated with ophthalmic complications and pyoderma. In late stages, neurological, gastrointestinal, pulmonary and cardiac complications may appear. The diagnosis is

based on history, physical examination, exclusion of other conditions and long-term follow-up with high index of suspicion. Bullae rupture easily to produce erosions, so the differential diagnosis of blistering disorders can be applied in GU as well. Aphthosis is a common idiopathic cause of GU, which may mimic genital herpes. But it usually associates with oral lesions.

### **Blister**

Herpes zoster can rarely be confused with herpes simplex on the genitalia. The vesicles usually locate unilaterally and rarely cross the midline. They should follow the dermatome. Fixed drug eruption and erythema multiforme major may cause blisters on the genitalia. In the latter case, the other part of the body should also be involved. Juvenile dermatitis herpetiformis, pemphigus vulgaris and vegetans have a predilection for the genitalia. Familial benign chronic pemphigus is easily induced by irritants, infection and friction, and may escape recognition. Other immunoblistering disorders also can involve the genitalia but it is seldom the only site.

### **Dystrophic Conditions**

Lichen sclerosus et atrophicus (LSA) occurs more in female genitalia which may be initially inflammatory with blisters, then progresses to chronic ivory white plaques. A "figure-of-eight" may appear to involve the perianal skin. Stenosis and atrophy may lead to dyspareunia in female and phimosis in male. A small proportion may develop carcinoma preceded by leukoplakia. So long-term follow-up is recommended, with potent topical steroid for symptomatic cases. Vulval atrophy, occurs in postmenopausal women, is usually asymptomatic. In symptomatic cases, there may be dyspareunia, which may be relieved by topical estrogen cream and bland emollients.

A useful classification based on clinical and histological criteria of the reactive and neoplastic disorders of vulval epithelium is as follow:

1. Benign dermatoses:
  - a. lichenification;
  - b. psoriasis;
  - c. lichen planus;
  - d. seborrhoeic dermatitis;
  - e. eczematous dermatitis (chronic).

2. Vulvar epithelial hyperplasia:
  - a. without atypia;
  - b. with atypia (leukoplakia).
3. Lichen sclerosus.
4. Lichen sclerosus with foci of epithelial hyperplasia:
  - a. without atypia;
  - b. atypia (VIN).
5. Squamous cell carcinoma in situ/invasive VIN.
6. Paget's disease of the vulva.
7. Plasma-cell vulvitis.

### **Pigmentary Anomalies**

Vitiligo should be distinguished from LSA. There is only depigmentation without atrophy (apart from side effect of topical steroid) or pruritus. A history of dermatosis over the genitalia is usually preceded the post-inflammatory hyper/hypopigmentation. Pseudoacanthosis nigraicans is often seen in obesity, pregnancy or puberty. It is asymptomatic with slight lichenification and hyperpigmentation over the vulva, perianal and groin region. Acanthosis nigraicans is a similar condition, associated with underlying malignancy or diabetes.

### **Granulomatous Lesion**

Superinfections and abscess formation arising from trivial infections may cause granulomas. The rare causes of granulomatous lesions in the genitalia are tuberculosis, schistosomiasis, cutaneous leishmaniasis, amoebiasis, Crohn's disease and hidradenitis suppurativa. They should be distinguished from the usual venereal causes such as nodular gummatous form of tertiary syphilis, lymphogranuloma venereum, and granuloma inguinale by patient's history, physical examination of other regions, culture and histological examination of the biopsy tissue.

### **Conclusion**

By recognizing the normal anatomical variation of the genitalia, one can save many unwarranted investigations and most importantly prevent the patient from unnecessary apprehension. For those persistent dermatoses with bizarre outlook and progressive course, a skin biopsy may help to rule out pre-malignant or malignant conditions.

# Papulosquamous Dermatoses and Diagnostic Pitfalls

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## Introduction

Papulosquamous dermatoses is a heterogeneous group of disorders which aetiology is primarily unknown. The name of this group of disorders is based on a descriptive morphology of clinical lesions characterized by scaly papules and plaques. The major entities are listed in Table 1. Adverse reactions to many drugs may also produce papulosquamous eruptions.

In clinical practice, diseases are however by no means obliged to respect classifying criteria, and papulosquamous diseases can present in non-papular and non-scaling forms. This is often seen in dermatoses partially treated with over-the-counter topical agents. Moreover, it must be remembered that papulosquamous diseases, which are mainly inflammatory skin diseases, can be simulated by non-inflammatory skin entities. Thus, the potentially life-threatening Langerhans' cell histiocytosis may mimic diaper dermatitis while scabies infestation may mimic severe atopic eczema. It is therefore important to consider all possible dermatoses in the differential diagnosis of a papulosquamous eruption. One of the pitfalls in diagnosis of any cases of papulosquamous diseases is secondary syphilis.

**Table 1. Major Papulosquamous Dermatoses**

Papulosquamous Dermatoses	
Include:	Psoriasis
	Eczema
	Seborrhoeic dermatitis
	Dermatophytosis
	Pityriasis rosea
	Drug Eruption
	Lichen Planus
	Pityriasis rubra pilaris
	Pityriasis lichenoides
	Bowen's Disease
	Mycosis Fungoides
	Parapsoriasis
	Secondary Syphilis
	Discoid Lupus Erythematosus
	Graft Versus Host Diseases

Borderline leprosy is as much of an imposter and its relative rarity facilitates misdiagnosis.

## Making an Accurate Diagnosis

Successful diagnosis of papulosquamous and other skin diseases relies on sound knowledge and a systemic approach. There is no replacement for focused systemic history and good physical examination. An initial inspection may help to identify the type of disease process and is often helpful to direct the thought process during history taking. Important factors to consider include symptoms, time-scale, evolution, body site distribution, close up morphology and changes at specific sites such as nails and mucosae. Appropriate investigations including fungal microscopy and culture, skin biopsy and blood tests are of value in diagnosis and identification of associated disorders.

## Making a Complete Diagnosis

It must be realized that diagnosis should not only be *right* but also *complete*. Every clinical condition can be regarded as an entity in some cases, while in others as a syndromic constellation representing a sign of another underlying condition. Guided by a high index of suspicion, severe tinea corporis may suggest underlying undiagnosed diabetes mellitus, while seborrhoeic dermatitis may be the first presentation of HIV infection. Sporadic associations, such as co-localization of psoriasis and vitiligo, do not seem to affect treatment options but may suggest a common pathogenetic pathway, which could be targeted for in clinical research.

## Misdiagnosis in Papulosquamous Eruptions

The scope of this article does not allow an extensive review of each papulosquamous dermatosis. Three selected common conditions that often present as scaly papules and plaques are discussed with emphasis on avoiding misdiagnosis.

### **Secondary Syphilis**

The diagnosis of secondary syphilis should always be considered in any eruption that did not fit a recognized pattern. This inflammatory response to disseminated

*Treponema pallidum* spirochaete invariably resolves spontaneously without treatment in one to three months. Thus, misdiagnosis will forfeit the chance of treating a curable condition and subject the untreated to potential serious cardiovascular, ocular and neurological complications.

The secondary phase of syphilis starts four to twelve weeks after the appearance of the primary chancre. The chancre may be asymptomatic or unrecognized. Secondary syphilis consists of an eruption, lymphadenopathy and variable malaise and constitutional upset. Pink or copper-coloured macules, which later develop into papules, squamous and nodular types erupt in a symmetrical distribution on the trunk and limbs. Characteristically, it is non-itchy and involves the palms and soles. Annular patterns are not uncommon. Other signs are moist warty condylomata lata in the anogenital area, buccal snail-tract ulcers and moth-eaten alopecia.

The differential diagnostic considerations include pityriasis rosea, psoriasis, drug eruption, lichen planus, parapsoriasis and infective exanthem. Any of these conditions may co-exist with syphilis. A thorough clinical history including sexual exposure and history of ulcer should be explored. Darkground microscopy from abraded muco-cutaneous lesions or lymph node aspirates may demonstrate characteristic motile spirochaete. Serological tests for syphilis, including non-specific reagin tests and specific treponemal antibody tests, must be performed for confirmation of diagnosis. Treatment is with intramuscular procaine penicillin.

### **Scabies**

Often presented as an itchy papulosquamous eruption, scabies is commonly misdiagnosed as recalcitrant eczema. Caused by the mite *Sarcoptes scabiei* var. *hominis*, this common skin infestation is transmitted by close physical contact, usually by sleeping in the same bed. In adults, it is commonly sexually acquired and some patients may deny venereal exposure.

At the initial phase of infestation, the host is asymptomatic. A hypersensitivity response to mite antigens occurs in four to six weeks coinciding with the eruption of itchy inflammatory papules. Itchiness is generally worse at night and scratching may lead to secondary infection and eczematization, which can

obscure the primary features. Burrows often occur at interdigital spaces, flexural aspects of wrists and elbows, axillary folds, the nipples, the umbilicus and external genitalia. In males, inflammatory papules on the penis and scrotum are pathognomonic of scabies and may be the only presentation. In infants and the elderly, burrows may occur on the head and neck. In babies, burrows may occur on their faces and pustular papules on their palms and soles. Crusted or Norwegian scabies is a variant in which thick-crust lesions form. It occurs when itching is reduced or absent or when the host is unable to perceive itching because of sensory impairment. This is usually seen in institutionalized, retarded, debilitated or immunocompromised people.

The typical history of pruritus with nocturnal exacerbations, and the distribution of the eruption of inflammatory papules should suggest the diagnosis. Absolute confirmation can only be made by the discovery of burrows and microscopic examination. A burrow is gently scraped off the skin with a blunt scalpel, and the material placed in a drop of 10% potassium hydroxide or mineral oil on a microscope slide. The presence of mites, eggs or fragments of eggshells confirms the diagnosis. Occasionally, burrows are difficult or impossible to find, and the diagnosis can then only be presumptive, based on the history, distribution of the papular eruption and the presence of contact cases within the family. Skin biopsy is a last resort but is diagnostic only when biopsy of a burrow reveals a mite within a subcorneal vesicle. Topical regimen with a scabicide such as malathion or benzyl benzoate treating all patients, their family and sexual partners is usually sufficient. Oral ivermectin is reserved for recalcitrant cases.

### **Tinea Incognito**

Misuse of topical corticosteroids for dermatophytosis can profoundly modify its clinical appearance. This is seen all too often due to misdiagnosis or self-treatment with over the counter steroid preparations. Occasionally, patients may have received topical or systemic steroid for other pre-existing pathology. Aptly termed tinea incognito, the well-defined annular plaques with raised edges and central clearing typically seen in tinea corporis are often absent. The edges may no longer be distinct or raised, scaling is reduced, the eruption becomes more widespread and follicular nodules and pustules become prominent. As the inflammation and itchiness are

suppressed by steroid action, patients and clinicians may be mistaken that steroid is indeed the correct treatment and more may be administered when the eruption relapses.

Diagnosis of tinea incognito requires a high index of suspicion. A ready awareness that the face, groins and the hands are sites of diagnostic error is important in alerting the clinician. Patients may not volunteer history of self-medication and direct questioning is often necessary. When the corticosteroid is stopped, the typical configuration and scaling of dermatophytosis returns quickly facilitating the diagnosis. Skin scraping should be taken from scaly lesions for fungal microscopy and fungal culture. Visualization of septate hyphae and positive culture confirms the diagnosis and species respectively. If both fail to yield a diagnosis, consider skin biopsy at lesional site for histology and special

stains. Treatment with a topical anti-fungal agent such as imidazole cream usually clears localized lesions. Oral treatment such as griseofluvin, terbinafine or itraconazole may be required for more widespread infection.

### **When Should I Refer?**

The general principles in making an accurate and complete diagnosis of papulosquamous eruptions are discussed and three commonly misdiagnosed conditions are presented. Specialist referral should be considered under the following circumstances: 1) diagnosis is uncertain, 2) failure to respond despite adequate treatment, 3) specialist investigations are indicated, and 4) specialist treatment is required and unavailable in the community. With the cooperation between the primary help care and specialist settings, the quality of the management for the patients can be greatly enhanced.