Number **3** *in a series of* **7**

CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

SKIN INFECTION AND INFESTATION

AERIAL VIEW OF CORAL REEF IN THE MALDIVES

UPDATED CHAPTER
SEPTEMBER 2025

‘Active edge’ border of lesion raised or increased scaling with relative clearing in centre (characteristic of ringworm)

CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

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CHAPTER ONE: THE ECZEMAS

CHAPTER TWO: PSORIASIS

CHAPTER THREE: SKIN INFECTION AND INFESTATION

CHAPTER FOUR: SKIN MALIGNANCY

CHAPTER FIVE: LEG ULCERS

CHAPTER SIX: ACNE

CHAPTER SEVEN:
URTICARIA AND RELATED ALLERGIC DISORDERS

A SYSTEMATIC APPROACH TO DIAGNOSING SKIN CONDITIONS
A PREQUEL TO THE CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

CUTANEOUS MANIFESTATIONS OF SYSTEMIC DISEASES IN DERMATOLOGY
A SUPPLEMENT TO THE CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

TESTS AND TECHNIQUES – WHICH, WHEN AND WHY?
A SEQUEL TO THE CORE TUTORIALS IN DERMATOLOGY FOR PRIMARY CARE

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CONTENTS

CHAPTER THREE: SKIN INFECTION AND INFESTATION

Bacterial infections 1

Fungal infections 4

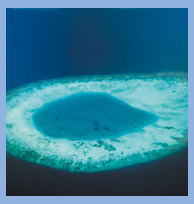
Yeast infections 8

Viral infections 9

Infestation 13

TEACHING POINTS 16

REFERENCES 16

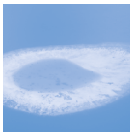


It was with some trepidation that I approached the third article of the 'Core' series. Infections and infestations in dermatology encompass a huge panoply of subjects which would require volumes to cover comprehensively. However, I shall adhere to the philosophy of these articles and focus on key issues in common presentations.

BACTERIAL INFECTIONS

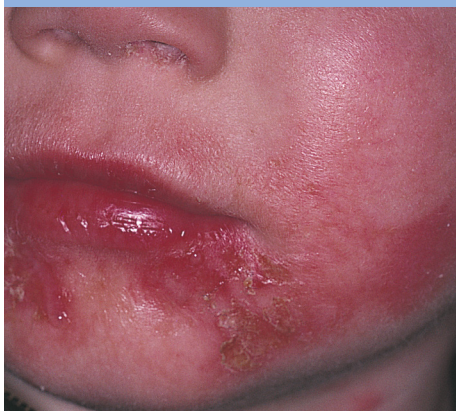
Let us first establish some general principles about skin flora. The normal commensals we would expect to find are coagulase negative *Staphylococcus*, diphtheroids and propionibacterium. *Staph. aureus* is not a normal commensal of human skin, although it is not infrequently found in the nasal vestibule and the perineum of healthy subjects.

There is established community resistance of *Staphylococcus* to erythromycin and almost universally to penicillin. More worryingly, there are increasing concerns recently about developing resistance to fusidic acid (Fucidin) which delivers its effect by inhibiting bacterial protein synthesis. Beta haemolytic *Streptococcus* are also pathogenic – Groups A, C & G remain almost universally sensitive to penicillin in the community. The other topical antibiotic in common usage is mupirocin (Bactroban) with its unique bactericidal action via RNA inhibition. It is active against both *Staphylococcus*, including MRSA, and *Streptococcus*. Resistance presently remains low but long term usage can induce irreversible *Staph. aureus* resistance and is therefore to be discouraged.



IMPETIGO – This is a very common presentation in primary care; it can be primary or secondary, simple or bullous, single or multiple. The characteristic presentation of rapidly spreading, but ultimately, self-limiting painless lesions with classical 'honey crust' in an otherwise well child usually presents little diagnostic challenge. The implicated organisms are *Staphylococcus* or, less commonly, Group A haemolytic *Streptococcus* (10% of cases). The bullous variation is invariably staphylococcal mediated. Infestation/eczema are commonly secondarily impetiginised. Swabs are useful to differentiate the causal organism although erythromycin may cover both likely culprits. Topical fusidic acid and mupirocin are useful for limited involvement although there are concerns regarding the development of bacterial resistance with more widespread use. Local antisepsis and soaks are a useful adjunct or stand alone treatment for limited disease. Topical 1% hydrogen peroxide cream is an option but is relatively expensive. The balance between cost effectiveness and the risk of inducing bacterial resistance with topical antibiotics should be carefully considered. School exclusion is only necessary if there is involvement of uncovered sites prior to treatment commencing. The lesions resolve without scarring and the only complication is streptococcal mediated glomerulo-nephritis. An incidence rate of 0.5 – 2.0% is stated for this in the textbooks but I suspect this to be much lower in primary care.

IMPETIGO



CELLULITIS – This is another common presentation manifesting as a rapidly progressive infection through the deeper subcutaneous planes, usually due to the ingress of bacteria, most commonly *Strep. pyogenes* but sometimes *Staphylococcus aureus*. There is often some pre-existing minor breach in the epidermis.

THERE ARE TWO KEY CARDINAL POINTS IN THE MANAGEMENT OF CELLULITIS THAT REQUIRE EMPHASIS:

a) Search diligently for the portal of entry of the offending organism.
So often in my experience the cellulitis is treated with great energy while the coexistent tinea pedis or a fissured corn is overlooked or ignored increasing the risk of recurrent infections unless addressed.

b) Understand the likely pathogenicity.
Cellulitis is often treated with 'exotic' combinations of expensive second and third line antibiotics, especially quinolones when penicillin V or amoxicillin and flucloxacillin will provide perfectly adequate coverage in most cases.

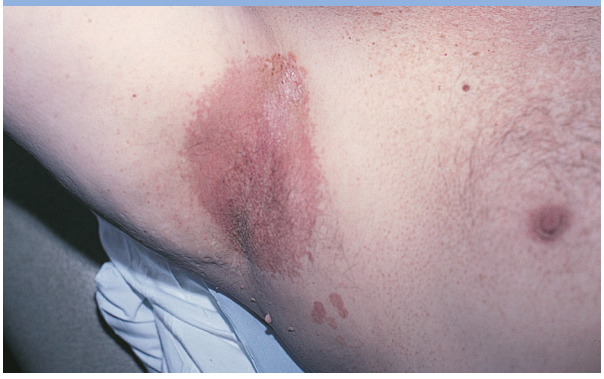
Each episode of cellulitis can cause damage to the lymph drainage system, leaving the patient more prone to further episodes. The use of long term low dose prophylactic penicillin V 250mg od-bd is an accepted and effective approach in patients with recurrent cellulitis.

ERYSIPELAS – This is a rarer but potentially more serious infection of the epidermal tissues. It is characteristically a more superficial infection than cellulitis with a sharply demarcated area of involvement more often in elderly patients. It is mediated by Group A haemolytic *Streptococcus* and most commonly presents in the lower leg, with the arm, buttock and face less commonly involved. There is often coexistent prominent lymphadenitis. The patient is often constitutionally unwell with pyrexia and rigors and needs urgent treatment. Flucloxacillin is the first-line choice with erythromycin, clarithromycin and doxycycline as alternatives. Again, remember to search for a portal of entry!

CORYNEBACTERIUM INFECTIONS – There are two common but poorly recognised infections caused by the humble *Corynebacterium minutissimum*.

The first is **erythrasma**, often masquerading as 'fungal infection' with a predilection for the toe webs, axillae and genitocrural region. Clinically, there is often a subtle confluent, slow spreading, well demarcated scaly erythema which fluoresces 'coral pink' under Wood's light (filtered long wave UVL), if available, as a consequence of porphyrin production as a metabolic by-product.

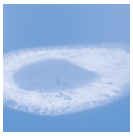
ERYTHRASMA 'CORAL PINK' UNDER WOOD'S LIGHT



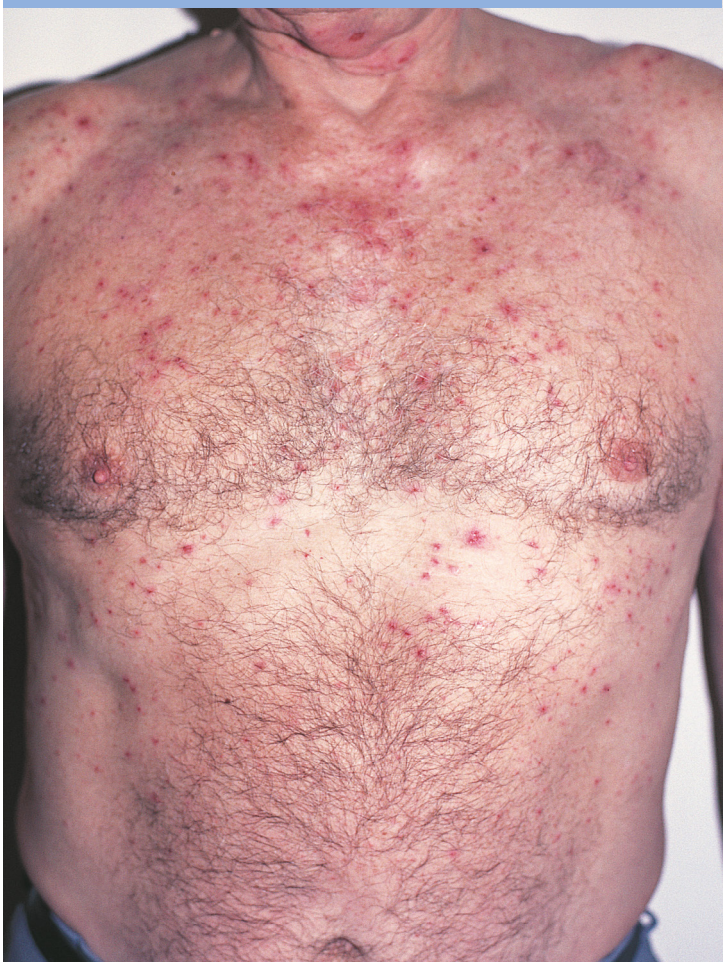
The second is **pitted keratolysis** which is a consequence of *Corynebacterium* overgrowth in the soles of feet of hyperhidrotic individuals, giving a very characteristic punctate (pitted) appearance in association with macerated and malodorous skin on the plantar aspects. As well as treating the *Corynebacterium*, the underlying hyperhidrosis needs addressing or recurrence is likely.

Corynebacterium is sensitive topically to imidazole creams e.g. clotrimazole (Canesten), fusidic acid and clindamycin. In more severe resistant cases systemic erythromycin is an option.

SYCOSIS BARBAE/FOLLICULITIS – This is another bacterial infection worthy of mention. It is defined as a pyoderma which is usually *Staph. aureus* mediated and localised to the hair follicles. This can often prove therapeutically very stubborn, often requiring extended combined regimes of topical and systemic treatment. More recently, the phenomenon of *Pseudomonas* folliculitis has been recognised in association with poorly maintained jacuzzis.



FOLLICULITIS



FUNGAL INFECTIONS

Fungi are primitive plant like organisms – they live as parasites or saprophytes and can be zoophilic, geophilic or anthropophilic. Numerically, this group of infections are the most prevalent of all and incidence appears to be increasing. Studies demonstrate over a million cases[3] of established fungal nail infection in the UK adult population although I not infrequently encounter this problem in children as well, with a slight bias towards the male. 15%[4] of the population also have evidence of tinea pedis and there have been upsurges of almost epidemic proportions in the incidence of tinea capitis, especially in urban populations particularly London and Birmingham.

The zoonotic fungal infections largely comprise of 6 species:

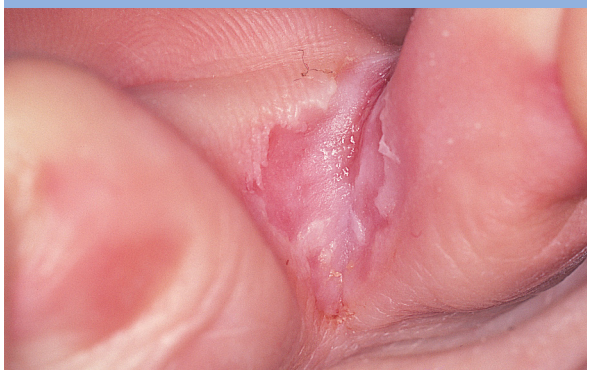
- *T. rubrum,*
- *T. interdigitale,*
- *T. mentagrophytes,*
- *T. verrucosum,*
- *M. canis,*
- *E. floccosum,*

with a 7th, • *T. tonsurans,* largely responsible for the uplift of tinea capitis in the capital.

They are spread both by direct and indirect contact. Let us now consider common patterns of infection.

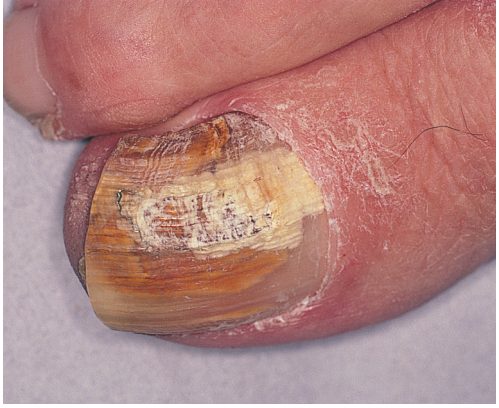
TINEA PEDIS – this is a very common problem, as previously stated. Its spread has a strong association with communal bathing facilities. One survey revealed a 22% incidence in school boarders, but only an 8.5% incidence in the coexistence day pupil population.[5] A third have coexistent nail involvement. The fungus has a predilection for the 4th and 5th toe web spaces and is often initially asymptomatic. Tinea pedis can progress to a more widespread pedal involvement, so called 'moccasin foot'. The organism usually implicated is *T. rubrum.*

ATHLETES FOOT



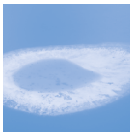
TINEA UNGUIUM – every distorted or dystrophic nail appears to be labelled 'fungal'. However, it is important to establish a diagnosis on clinical and also mycological grounds, if possible, if treatment is proposed. A history should reveal a slow onset of nail dystrophy involving 1 or 2 nails initially with a gradual spread to other healthy nails with asymmetric involvement and often complete sparing of other nails. There is often evidence of coexistent skin involvement. This is in contrast to another common form of nail dystrophy, namely psoriasis, which is typically symmetrical. The two conditions of course commonly co-exist. The infection is usually dermatophytic with involvement of the distal portion of the nail most common.

FUNGAL NAIL INFECTION



There is an increased awareness of fungal nail infection in the population. The vast majority of people, especially with the more common toenail infection, are either happy to 'live with it' or, most commonly, are unaware they have an infection in the first place. Indeed I tend to use the terminology "fungal contamination" rather than "fungal infection" to reduce any alarm to the patient. The appearances of chronic tinea unguium are often put down to 'ageing'. A cost/benefit analysis of treating tinea unguium, which is evident in over 2% of the population, with synthetic antifungal agents, especially given the documented high recurrence rates, would have no difficulty in concluding that the limited NHS funds would be better spent elsewhere. This argument is perhaps less persuasive now that terbinafine is relatively cheap although other synthetic antifungals remain expensive. Tinea unguium rarely causes complications however occasional drug reactions, particularly liver toxicity, to oral antifungals can be serious. However, individual careful case selection for treatment on an individual basis is required. Some patients are immunocompromised or their dystrophic nails can be symptomatically painful. Foot care can be of particular importance in the diabetic sub population. More commonly, however, there is often significant patient pressure for cosmetic reasons.

Having made an argument for **not** treating a large proportion of fungal nail infection, it certainly is indefensible to treat nail dystrophy for prolonged periods with expensive antifungals if the aetiology is **not** fungal! More proximal involvement, especially if coexistent nail bed and cuticle disease may implicate *Candida*. If mycological investigation is undertaken, then nail clippings and scrapings should be collected by someone practised in the art, not the patient themselves! Mycological analysis is challenging and very variable from lab to lab with positive microscopy in only 40% of cases and positive culture in only a meagre 18%.[6,7] However, these rather dismal statistics I am sure are influenced by the poor collection of samples. **Remember** too that the presence of fungus may be a secondary rather than a primary phenomenon due to a pre-existing nail pathology such as psoriasis.



TIPS FOR A GOOD SPECIMEN COLLECTION INCLUDE	
Nails	Clip as proximally as possible with good quality, 'heavy duty' nail clippers. Scrape subungual debris as this can be a rich source of harvest.
Hair	Obtain plucked hairs or brushings from the area – cut hair is of little value.
Skin	Scrapings should be obtained from the active edge outwards. Vesicles should be de-roofed and the skin submitted. Involved skin can be peeled from the interdigital spaces.

Rarely, lab reports will identify non dermatophyte moulds and saprophytic fungi e.g. *Scytalidium* and *Scopulariopsis*. The relevance of these should be discussed with a local microbiologist/dermatologist as often there is no reliable treatment available.

TINEA CAPITIS – historically *M. canis* was the most implicated organism with *T. verrucosum* transmitted from infected cattle in rural areas, but as previously stated *T. tonsurans* and, to a lesser extent, *T. violaceum* are on a steep increase in the urban population, especially in the ethnic Asian and Afro-Caribbean groups. Tinea capitis can produce a dramatic immune response almost exclusive to young children, presenting with a rapidly growing, boggy mass on the scalp called a 'kerion'.

DISCUSSION POINTS

There are two major topical discussion points regarding kerions

1. Recognition

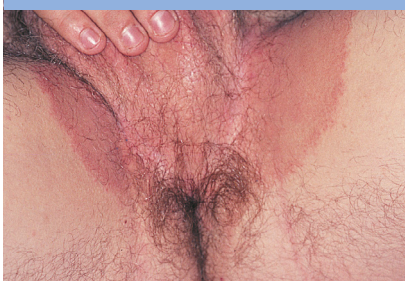
The child is usually completely well but prompt recognition of the presentation is important as a kerion may result in significant areas of scarring alopecia. If suspected, prompt referral to the local Dermatology department should be considered. The reality is, however, that the condition is often misdiagnosed as some form of scalp abscess and directed to casualty or surgical departments, and inappropriate surgical interventions carried out. So commonly does this happen, that it prompted a dedicated article in the BMJ.[8] I personally have experience of 3 such cases.

2. Treatment

A kerion requires systemic plus or minus topical treatment, but the only systemic antifungal agent with a paediatric licence is the relatively ineffective griseofulvin, which is a fungistatic agent available since 1952. Available liquid formulations are very expensive. It is common practice in dermatology to discuss the risk/benefit of using synthetic antifungals off licence in these cases. Itraconazole is available in liquid preparation; it again is fungistatic rather than its main competitor, terbinafine, which has a more effective fungicidal action. This debate is likely to run, as there is not a large commercial opportunity for the manufacturers of terbinafine or itraconazole to seek a paediatric licence. It is thought useful to combine systemic treatment for scalp ringworm with a topical therapy e.g. ketoconazole, as well to reduce fungal shedding and infectivity. It is important to note that although "off licence", the synthetic antifungals, terbinafine and intraconazole, are well established and in common usage in paediatric practice and there are dosing regimens available in the paediatric BNF.

Diagnosis of tinea capitis can be aided with Wood's light. This will demonstrate green fluorescence in the presence of *M. canis* which is an ectothrix infection, but not in *T. tonsurans* which is an endothrix infection.

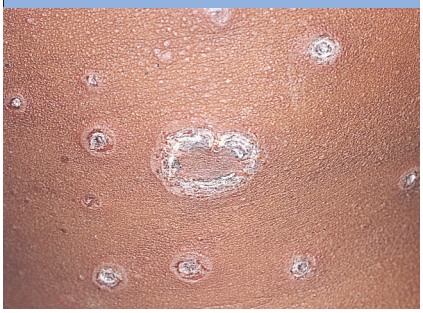
JOCK STRAP ITCH



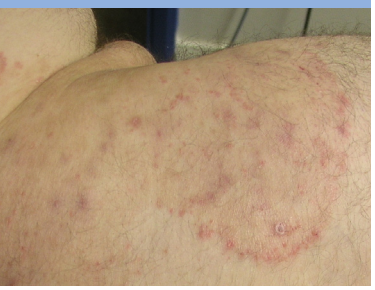
TINEA CRURIS – affectionately known among the sporting genre as 'jock strap itch'. This is a dermatophyte infection which characteristically affects the groins but spares the scrotal skin. Remember to look for coexistent tinea pedis!

TINEA CORPORIS – this manifests as the classical 'ringworm', a slow, radially enlarging dermatosis, single or multiple, and often relatively asymptomatic with an inflammatory scaly edge and central clearing. Three common genera of dermatophyte are implicated: *Microsporum, Epidermophyton* and *Trichophyton*. The incubation period is 1 – 3 weeks. The diagnosis can be confused with discoid eczema and can often be very difficult to distinguish initially, although discoid eczema is often intensely itchy and settles into a symmetrical distribution. Less forgivable is confusion with the other commonly occurring annular lesion, granuloma annulare, which is a dermal inflammatory process with no epidermal involvement i.e. an absence of scale.

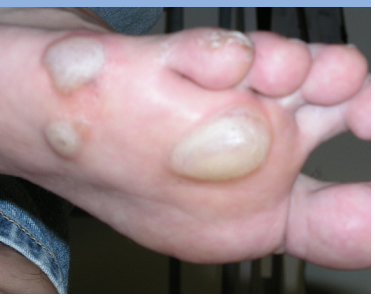
RINGWORM – *M. canis*

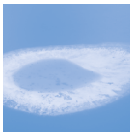


TINEA INCOGNITO



BULLOUS TINEA





TREATMENT OPTIONS

OTHER COMMON DERMATOPHYTE PRESENTATIONS WORTHY OF MENTION ARE

a) tinea barbae – usually confined to 'at risk' agricultural workers presenting with a pustular folliculitis or a frank kerion in the beard area. This is often painless, unlike bacterially mediated infections which enter the differential diagnosis.

b) tinea incognito – this is a common consequence of the misapplication of topical steroids to fungally mediated dermatoses. There may appear to be initial improvement due to a non specific reduction in the inflammatory reaction, and also a reduction in symptomatic itch. However, the resultant local steroid mediated immunosuppression will 'fuel' the fungal infection, but often alter the morphology with widespread and often bizarre geometric patterns on the skin (see illustration) – hence the term 'incognito'. Always be suspicious of the presentation of unilateral 'eczema'!

An interesting phenomenon with fungal infections is the 'id' reaction. This is where a fungal infection can sometimes produce an allergic reaction on a distant site which presents either as an eczema or a pompholyx type eruption. Localised vesico-bullous reactions can also cause diagnostic confusion.

The management of these infections has been revolutionised by the advent of the synthetic antifungal agents, available both topically and systemically, which augmented the limited pre-existing options of topical imidazole creams and systemic griseofulvin.

The classification of antifungal drugs available is as follows

- Azole group – including imidazoles e.g. ketoconazole and tioconazole and triazoles e.g. fluconazole and itraconazole
- Allylamines – e.g. terbinafine

The triazoles are fungistatic but are active against yeast infections as well. Terbinafine has had rare reports of hepatotoxicity but is 'cleaner' in terms of potential drug interactions. It has been demonstrated to be more effective than pulsed itraconazole in the treatment of tinea unguium.[9] Another study demonstrated 44% disease free nails at 1 year as opposed to itraconazole with a 34% outcome.[10] Topical terbinafine cream has also been demonstrated to have a higher eradication rate of tinea pedis after 1 week, than clotrimazole after 4 weeks, making an argument for its cost effective use. To treat tinea unguium of toenails, the minimum duration for recommended treatment is 3 months. The huge price reduction in generic terbinafine means this is no longer expensive but there are significant recurrence rates unless preventative measures are practised and a small risk of drug side effects. Clinical decision making about whether to treat or not to treat remains a balance.

There was a vogue for using itraconazole as a 'pulsed' treatment. There seems little practical advantage to this. The use of the nail paints, tioconazole and amorolfine, should be confined to distal nail disease affecting 1 or 2 nails only. These preparations are relatively ineffective. Even after 6 months of continuous treatment, outcomes are disappointing with cure rates of only 30 – 50% quoted. With the advent of synthetic antifungals, griseofulvin, with the exception of tinea capitis mentioned above, is no longer commonly indicated due to very prolonged treatment times (up to 18 months for toe infection) and poor outcome data.

Common yeast infections include

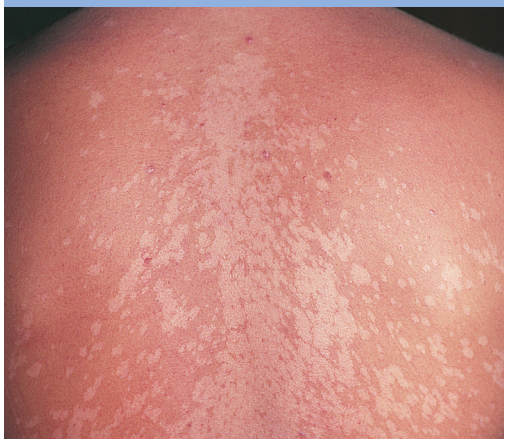
1. CANDIDIASIS – this is more likely in the presence of a number of well recognised underlying factors

- local maceration e.g. angular cheilitis
- mechanical trauma
- physiological states e.g. infancy/pregnancy
- endocrine disturbance e.g. diabetes mellitus
- malignancy/immunodeficiency N.B inhaled steroids
- iatrogenic factors e.g. steroids/oral contraceptive pill

Cutaneous candidiasis characteristically produces satellite lesions +/- pustulation. Treatments include topical imidazole creams or oral fluconazole/itraconazole. Chronic candidal paronychia can be particularly stubborn to treat and a pharmacological approach should be combined with physical efforts to keep the affected area dry and effectively 'desiccate' the organisms. Distal nail involvement is rare.

2. PITYRIASIS VERSICOLOR – this common fungal infection literally translated means 'scaly and of various colours'. This typically affects young adults especially if there is a history of foreign travel in hot and humid climes. The normal commensal organism, *P. orbiculare* undergoes a change to a more aggressive variation, *Malassezia furfur*. This yeast produces azelaic acid which temporarily damages the melanocyte resulting in a fine, scaly, 'moth-eaten' pigmentary disturbance, characteristically affecting the trunk and limbs. Mild itch is the only common associated symptom. Treatment is with topical agents, such as selenium sulphide shampoo 2.5% daily for 14 days, or ketoconazole shampoo for 5 days, both as 'wash off' treatments, or topical terbinafine (oral terbinafine is ineffective). For more widespread cases, oral itraconazole 200 mg daily for 1 week is very effective. The patient must be warned that repigmentation, however, requires post treatment exposure to UVB/ sunlight. Recurrence is common.

PITYRIASIS VERSICOLOR

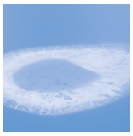


Other presentations of overgrowth of *Pityrosporum* yeasts are seborrhoeic dermatitis/ capitis and *Pityrosporum* folliculitis which produces an itchy monomorphic acne-type rash. This should be suspected if 'acne' is not responding to conventional treatments, especially in an age cohort rather older than the usual adolescent acne sufferer.

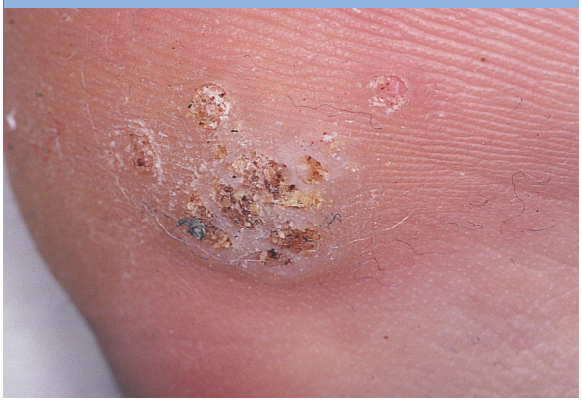
VIRAL INFECTIONS

WARTS – the humble wart still occasions a disproportionate response in terms of both anxiety generated and the energy channelled at rather ineffective and often wholly inappropriate range of treatments. However, society does seem to be becoming more at ease with its coexistence with the wart virus. Gone are the days, at least, of school inspections where the slightest suspicion of a verruca would result in an extended ban from the swimming pool! Warts are 'complained of' rather than 'suffered from'. They are ubiquitous in the young population with a prevalence of 5 –10% with no social or sexual bias. Although there are 69 different types of human papillomavirus (HPV), only 4 characteristically infect man: HPV 1 – 4. There is rarely diagnostic confusion. They can be distinguished from corns by paring down to reveal tiny capillary thromboses.

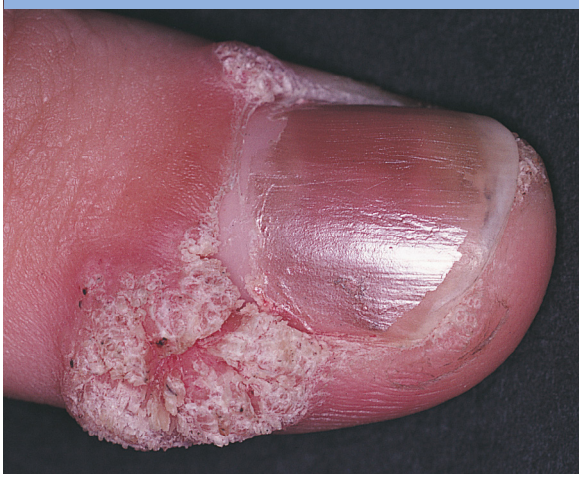
My personal opinion is **do not treat** unless there are persuasive arguments to do so. This is generally the currently adopted NHS policy. There are certainly no public health grounds for treatment and warts cause no risk in food handling. Two thirds will self resolve within 2 years and plantar warts demonstrate 30 – 50% regression within 6 months. Persuasive arguments to treat include painful, symptomatic warts, usually verrucae on pressure points of the foot, and significant cosmetic morbidity e.g. prominent hand warts in young adults or filiform facial warts in children where ridicule at school can be a factor. Purely parental pressure without informed commitment from a child is not a good argument to treat, and in my practice treating children under the age of 12 is the exception rather than the rule due to the pain involved with physical procedures. Even historically in dedicated wart clinics, whether they were hospital or community based, outcomes of audit usually demonstrated no more than 50% and at the very best 70% resolution after 3 months of treatment. The most optimistic return from topical treatments alone demonstrated a 67-84% clearance.[11] Particular difficulties with treatment are encountered when dealing with multiple warts, plane warts, mosaic warts, periungual warts and facial warts.



MOSAIC WART



PERIUNGUAL WART



TREATMENT OPTIONS

A wide range of treatments are advocated, extending from rubbing with banana skins to 'charming'! Sceptically, the 'success' of such treatments are likely based on the fact that when warts regress they can do so very quickly, often confusing cause and effect. We shall confine ourselves to the more conventional treatments which mainly rely not on necessarily killing the virus itself, but effectively physically disrupting its environment, either with cold, heat or chemical injury. This produces a non specific inflammatory reaction that is thought to trigger an appropriate immune response.

1. TOPICAL – The first option is usually empowering motivated parents/patients to treat their warts with a range of topical compounds containing either salicylic acid with concentrations ranging from 11 – 50% or formaldehyde. The latter chemical is virucidal. They are made up in a wide range of film forming agents, paints and gels. Some of these contain collodion with the risk of contact sensitivity. However, this may be indirectly therapeutic! Treatment should be for up to 3 months, usually on a daily basis. Unless there is effective paring or abrading of the warts to remove the overlying 'armour plate' of dead skin before the topicals are applied, the treatment is likely to be ineffective. Paring alone will render a wart pain free as it is the build up of skin overlying the wart that produces symptoms. Paring should be continued either until there is pain or bleeding i.e. down to 'living' tissue. Topical chemicals should not be applied to the face. The treatment of genital warts will not be covered here.

2. CRYOTHERAPY – This treatment modality is increasingly unavailable outside of hospitals due to expense, technical logistic and lack of both efficacy and revenue opportunities. There are a number of misconceptions regarding this modality; one is that it is a more potent treatment than topicals, but parallel treatment studies show no statistically significant outcome measures between these two. Treatment with cryo, however, can be effective where topicals have failed. As a consequence, historically, this resulted in high levels of secondary care referrals for wart treatments. One study from Southampton demonstrated between 15 – 25% of out patient workload[12] to revolve around wart treatment! This was clearly an inappropriate use of an expensive secondary resource. However, in my own locality now referral to hospital for wart treatment is the exception rather than the rule and such "low priority" referrals are now widely discouraged as policy. Another misconception with cryotherapy is its mode of action. Viruses are relatively resistant to cold injury – indeed viruses are stored in liquid nitrogen to maintain their viability! Cryotherapy appears to work, again by disrupting the cellular environment and the production of an inflammatory/immune response. There are a number of both cryotherapy agents and devices, ranging from the humble cotton wool ball to the more sophisticated, but rather overpriced, cryoguns and cryoprobes. Cotton wool application, which is the least effective, requires a loose wound ball. If multiple treatments are made from the same flask of liquid nitrogen, there is a theoretical risk of virus transmission.

Cryotherapy, where still available, should be practised according to agreed guidelines and protocols as it is a treatment modality not without risk in unfamiliar hands. Particular care should be taken with pigmented skin due to the risk of post inflammatory hypopigmentation. The evidence suggests an interval between treatments of 2 – 3 weeks. There is an argument for using interim topical treatments if tolerated. I would not treat an individual wart on more than 6 occasions.

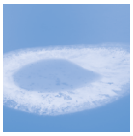
The application of a potent topical steroid immediately post cryotherapy is said to reduce the risk of nasty inflammatory or blistering responses, especially prominent in Type I skins.

3. OTHER – There are a number of other medical treatments which although not widely practised or available are worthy of mention for special situations; these include

- topical 5 fluorouracil
- intralesional bleomycin
- podophyllotoxin (1st line treatment for genital warts)
- photodynamic therapy
- topical/systemic retinoids
- topical imiquimod

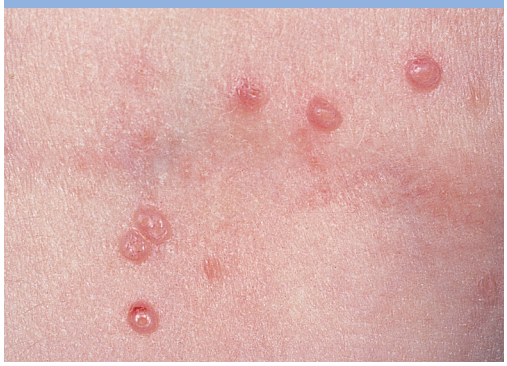
CRITERIA FOR REFERRAL

Ideally, referral to secondary care should be confined to 'atypical' warts, especially in older patients which can be confused with certain neoplasms, and patients with multiple warts possibly associated with immunosuppression.



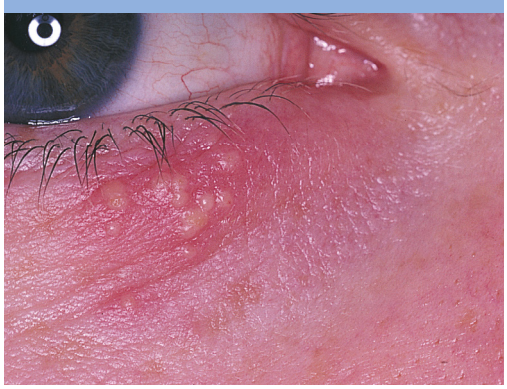
MOLLUSCUM CONTAGIOSUM – this is another very common viral condition of young children, although it can on occasion infect adults. Since the demise of smallpox, molluscum is the only pox virus that habitually infects man. It is most common on the face neck and genitalia and can be more florid both with coexistent eczema and immunosuppression. The lesions are characteristic with central umbilication and natural resolution occurs in a mean of 6 – 9 months. A range of treatment options are available including cryotherapy, topical application of potassium hydroxide 5% solution (Molludab), podophyllotoxin, benzoyl peroxide, imiquimod and retinoids; phenolisation or physical methods but these are rarely justifiable and risk conflict with the aspiration of Hippocrates to "above all do no harm!" and masterly inactivity remains my preferred option!

MOLLUSCUM CONTAGIOSUM



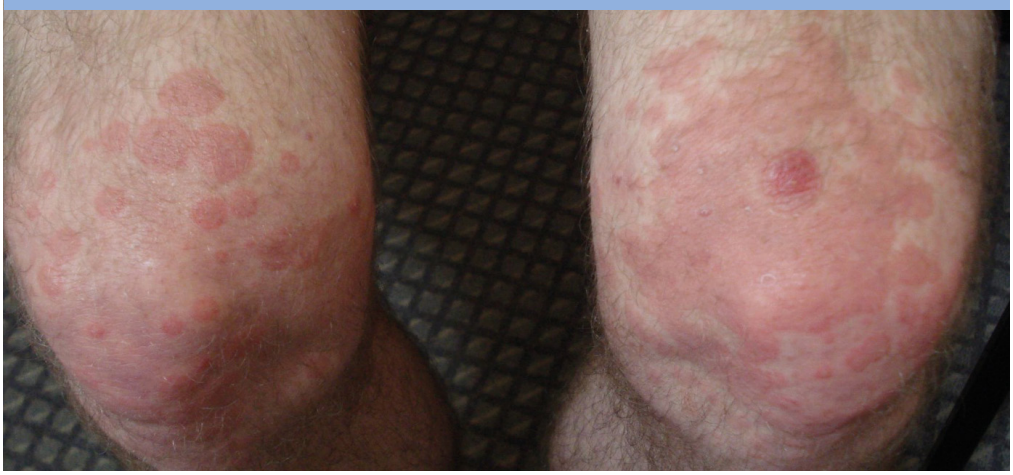
OTHER VIRAL INFECTIONS – Other viruses worthy of mention include herpes simplex which usually manifests itself as an initial infection of a young child with a trivial stomatitis, thereafter recurring at intervals as a characteristic group of vesicles, commonly on the lip although it can affect virtually any part of the body.

HERPES SIMPLEX



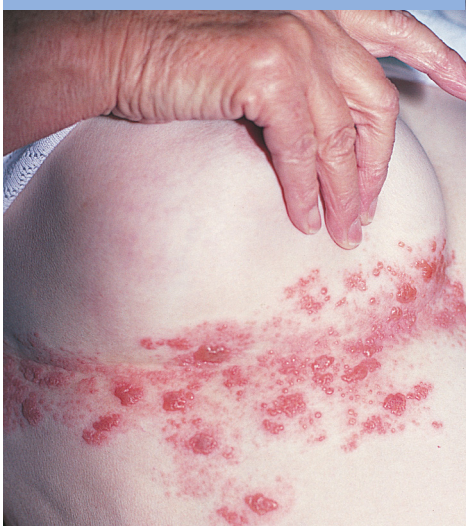
Direct viral culture from a swab can confirm the diagnosis and subtype the virus. The prevalence in the general population is 50%. Viral DNA persists in the neural tissue. There can be serious or even life threatening herpes infection, either simplex or zoster, in patients with pre-existing significant eczema or immunosuppression which can constitute a true dermatological emergency – so called eczema herpeticum or Kaposi's varicelliform eruption. Herpes virus is also often implicated in the cause of recurrent erythema multiforme.

ERYTHEMA MULTIFORME



If antiviral treatment for herpes virus is contemplated, it must be commenced promptly, ideally within 48 – 72 hours of onset of signs or symptoms. There is no evidence of benefit from initiating treatment after 5 days. There is now good evidence that prompt treatment with systemic antivirals can reduce the long term morbidity of shingles with postherpetic neuralgia.

HERPES ZOSTER



There are a number of other common virally mediated characteristic exanthemata that should be recognised

- herpes zoster – chicken pox or secondary infections with shingles
- hand, foot and mouth disease caused by Coxsackie A virus
- erythema infectiosum aka "slapped cheek" or 5th disease caused by parvovirus

Also worthy of mention, although largely confined to rural areas is the paravaccinia virus transmitted from infected sheep causing orf which can be complicated by erythema multiforme.

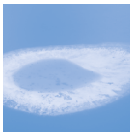
More rarely, there is the very striking and characteristic rash of Gianotti-Crosti disease (Acrodermatitis). This has been associated with a number of different viruses including hepatitis B, Epstein-Barr, cytomegalovirus, respiratory syncytial virus, enteroviruses, and post vaccination on occasion. The rash characteristically affects buttocks, limbs and face and can take several weeks to resolve and there is often co-existent lymphadenopathy. Constitutional upset is usually relatively minor.

INFESTATION

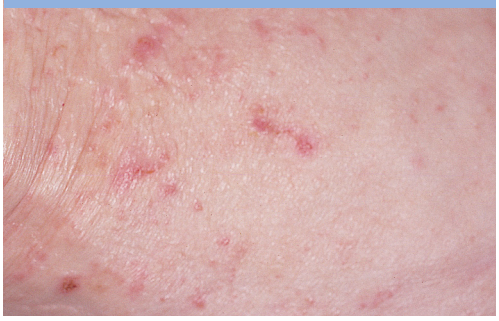
Our two most frequent uninvited visitors are the scabies mite and the head louse. They are no respecters of socio-economic status!

SCABIES

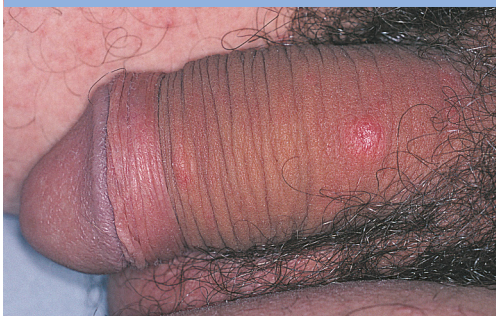
The scabies mite – *Sarcoptes scabiei* – produces an itchy papular eruption with an initial predilection for the wrists, hands, finger webs, feet and genitalia, but sparing the head and neck. The rash of scabies extends inexorably to become more generalised and most commonly affects children and young adults with no previous history of skin problems. There is often a supportive history of close contact with a similarly affected child, partner or relative. Contact must be close, even intimate, to allow transmission of the mite that can survive only for very limited periods off the human body. Characteristic burrows can be identified unless masked by excess excoriation or secondary eczema, and penile and scrotal nodules are almost pathognomonic, so it is important to inspect 'down below' as this will often not be volunteered.



SCABIES BURROW



PENILE NODULE



The incubation period is 4 to 6 weeks. Isolating a mite for dermoscopy or microscopy at the end of a sewing needle (not a venepuncture needle as the mite readily disappears down the bore shaft!) can be professionally very satisfying to the doctor, and impressively persuasive, if somewhat horrifying, to the patient!

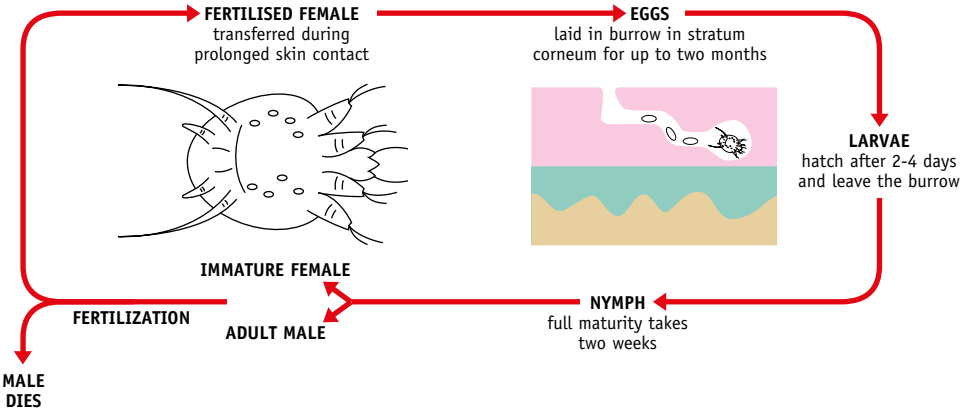
Doctors who claim they never miss scabies are no friends of the truth however. Presentation is not always textbook as in all things medical and can vary in different age groups. Babies and young children often have head and neck involvement and the elderly debilitated either physically, mentally or both can develop a form of overwhelming infestation called **'crusted or Norwegian' scabies** as they have either lost the sensation of itch or are unable to effectively scratch. Such patients literally carry millions of mites as opposed to the normal numbers of 20 – 30 adult mites involved in 'common or garden' scabies. It can be a source of epidemics, especially if in institutional care requiring the shutting of wards and residential/nursing homes and requiring hundreds of contacts to be screened or treated. Truncal scabies and involvement of the axillary folds and inframammary areas are also more typical in infestation in the elderly. There may be more burrows present due to a modified host response.

Having addressed the 'missed' diagnosis, another common phenomenon I see is the correct diagnosis made but when the problem fails to resolve, courage fails the doctor and the diagnosis is cast into doubt and a referral to secondary care made.

MANAGEMENT PRINCIPLES

There are a number of reasons why scabies appears not to respond to first line treatments. Poor compliance is often due to poor education. Instruction should be precise and an adequate amount of treatment prescribed, and treatment repeated at a 5 – 7 day interval to maximise results. All immediate household contacts should ideally be treated. 50 – 75 ml of topical medication is required for a single adult application. The interval between treatments relates to an understanding of the life cycle of the mite, designed to exterminate any emerging adults from eggs missed by the initial treatment.

LIFE CYCLE OF SCABIES MITE – *Sarcoptes scabiei*



The established concept of scabicide resistance also appears not to be widely recognised. Although difficult to examine reliably, scabies resistance has been measured in many parts of the country and has been found to be significant. However, so often patients come with a story of having had repeated treatments from their GP but invariably with the same scabicide, and yet if an infection didn't respond to a certain antibiotic there would be no hesitation in switching to an alternative. The success of treatment also is often not recognised as even after adequate eradication of the mite, the patient can remain symptomatic for several weeks and post scabetic nodules can persist sometimes for months. This is often misinterpreted as evidence of ongoing infestation.

TREATMENT OPTIONS

Let us briefly review our antiscabetic armamentarium. Historically 0.5% **malathion** was first line treatment with an 80% cure rate[1] and licensed for paediatric use but this has been superseded by the synthetic pyrethroid, **permethrin**, marketed as 5% Lyclear Dermal Cream.[13] Although more expensive, this has the highest "cure rate" and is licensed down to 2 months of age. **Crotamiton** is still sometimes used in infants but is a weak antiscabetic or to manage post scabetic itch. There is no established evidence of teratogenicity with antiscabetics but the risk/benefit ratio of treatment in pregnancy must be addressed in the usual way. In the future, a more simple and effective treatment may lie with the more widespread use of oral ivermectin.[2] This is a drug widely available in farming and veterinary practice which is similar in structure to the macrolide antibiotics. It is very active against both ecto and endoparasites and is given as a stat dose of 200 mcg/kg body weight, in combination with topical drugs, for the treatment of hyperkeratotic (crusted or 'Norwegian') scabies, that does not respond to topical treatment alone. It appears to be safe and effective, and already has an established role in treating outbreaks of scabies in institutional settings such as nursing homes which would normally be the remit of the local Public Health Department.

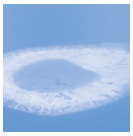
HEAD LICE (PEDICULOSIS CAPITIS) – This problem appears truly endemic particularly in the age group of 4 – 11. There is an estimated half a million cases per annum in the UK. Emotion still runs high, often fuelled by ill informed scare-mongering, particularly within schools. My anxieties revolve more around children constantly having their heads marinated in organophosphates with often little or no justification!

TREATMENT OPTIONS

The arguments still range between the physical and pharmacological schools of thought. These generate more heat than light. One properly constructed trial demonstrated treatment with topical malathion to be twice as effective as 'bug busting' measures. There are numerous agencies involved in regards to head lice; pharmacists, practice nurses, health visitors, GPs, teachers and community physicians. There needs to be a consistent and coherent message, particularly in regard to who actually needs treatment! This requires objective evidence of the presence of live, viable adult lice.

Treatment ideally should combine both physical and chemical methods with ongoing physical methods employed for prevention in those affected and their immediate close household contacts.

Chemical methods involve the use of synthetic pyrethroids (Lyclear/Full Marks) and malathion. There is established resistance to both therapeutic groups. Malathion is available in an aqueous preparation for those suffering from atopy. The previous cyclical policy with these agents has now been suspended and has been superseded by the present 'mosaic' policy.

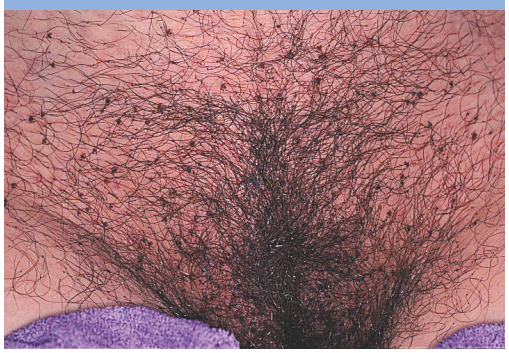


Lotions are superior to shampoos as treatment is often too transient in the latter, possibly encouraging resistance. Treatment, when indicated, should be twice at a 7 – 10 day interval. Treatment failure is as for scabies – lack of concordance with instructions, resistance or re-infection.

Mechanical nit wet combing has little evidence either to support or refute its efficacy but is increasingly popular as it avoids an increase in resistance. Other physical methods using various preparations containing agents such as dimeticone and isopropyl myristate work on the principle of 'suffocation'.

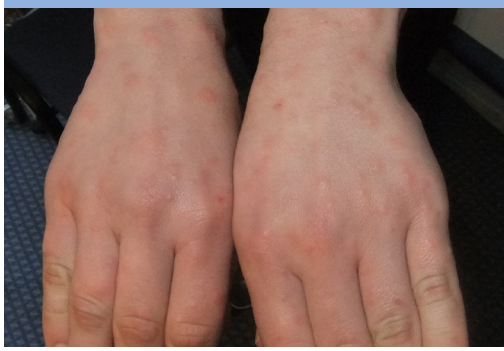
CRAB LICE (*Phthirus pubis*) – Although more in the realms of genitourinary medicine, the crab louse can stray, as the morphology of pubic hair is very similar to that found in the axilla and eyelashes. Infestation of the eyelashes in a child is not necessarily evidence of child sexual abuse, just of close physical contact. The crab louse will respond to topical application of malathion. Eyelash involvement can be treated with soft paraffin applied 3 times daily for 2 – 3 weeks.

CRAB LICE

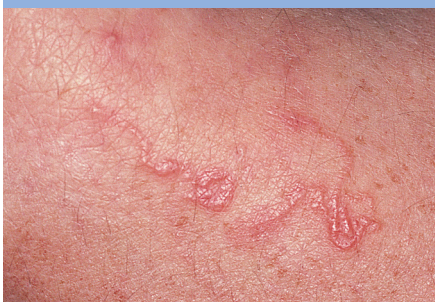


BED BUGS (CIMICIDAE) – there have been major outbreaks reported of infestation reaching epidemic proportions especially in the US. Infestation can often go undetected as eggs are laid in wall crevices, bed frames and furniture. The bites are painless but can produce florid sensitising reactions in the skin. Clinical suspicion as with scabies is the key to diagnosis.

BED BUGS



LARVA MIGRANS



TROPICAL DISEASES – Finally, the World continues to shrink due to the proportionately cheap opportunity to travel. There is likely to be an increasing risk of 'illegal immigrants'. In the past I have had cases of both cutaneous leishmaniasis and cutaneous larva migrans. A high degree of suspicion should be maintained for 'atypical' presentations in returning travellers.

TEACHING POINTS

1) In cases of cellulitis look for a pre-existing pathology

2) Penile nodules are almost pathognomonic of scabies

3) Be prepared to use permethrin if other anti scabies treatment fails

4) Re-pigmentation after infection with pityriasis versicolor will only occur with subsequent exposure to ultraviolet light

5) Do not embark on treatment of presumed fungal nail infection without a high degree of diagnostic probability or mycological confirmation

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