

23.10 Infections of the skin

5695 Roderick J. Hay

23.10 Infections of the skin

5695 Roderick J. Hay

ESSENTIALS A huge variety of different organisms exist on healthy skin, the normal microbiome. Under certain circumstances, microbes can actively infect the skin as a primary or secondary event in cutaneous or systemic disease. The most common causes of bacterial infection of the skin are *Staphylococcus aureus* or Group A streptococci. There are increasing reports of both hospital- and community-acquired infection by methicillin-resistant *S. aureus*. Superficial infection that spreads laterally in the upper dermis or along the subcutaneous fascia is known as cellulitis. Complications of cellulitis include septicaemia or lymphoedema, hence it requires immediate antibiotic therapy. *Mycobacterium tuberculosis* remains a common skin infection in many tropical areas, and syphilis, leprosy, and leishmaniasis are important skin infections in some parts of the world (see Section 8). Dermatophytosis, or ringworm, is caused by mould fungi that can digest keratin (keratinophilic). The diagnosis can be confirmed in the laboratory by examining scrapings or clippings of skin, hair, or nails mounted in potassium hydroxide. Pityriasis versicolor is mainly caused by *Malassezia* species: it is a chronic scaly condition in which the skin becomes either hyper or hypopigmented in the affected area; macules and scales join and may become confluent over the back and chest, less commonly elsewhere. Herpes simplex virus causes both acute as well as recurrent infections, such as cold sores. Occasionally, in the immunocompromized patient these and other herpesvirus infections can disseminate, both to other parts of the skin as well as internally.

Introduction The structure of the epidermis and its appendages such as hair and nails has been described elsewhere (see Chapters 23.1 and 23.13). The outer epidermis and the openings of the pilosebaceous units play host to a large diverse microbiome, the majority comprises bacteria such as micrococci, coryneform bacteria, and staphylococci such as *Staphylococcus epidermidis* and *Staphylococcus saprophyticus*. These can achieve very high densities in excess of $4 \log/\text{cm}^2$. However, in occluded areas and other parts of the body, anaerobic bacteria such as brevibacteria or microaerophilic organisms including *Propionibacterium acnes* are also present. Generally, these do not cause disease unless their local environment is disturbed, for example, by occlusion or blockage of the sebaceous follicle, or if they find entrance to the bloodstream through an internal appliance such as a cannula. Also, these might cause disease in the presence of immunosuppression. *S. epidermidis*, for instance, is a

common pathogen in neutropenic patients. Other factors that play a role in determining surface populations include ultraviolet light exposure and the presence of skin disease. Fungi, apart from members of a single genus of lipophilic (fat metabolizing) yeasts called the *Malassezia* species, are not commonly carried on the skin surface. These are mainly found in the openings of sebaceous follicles. A mite species, *Demodex folliculorum*, is also found on the skin surface within hair follicles. Again, these organisms are normally in balance with the host and unless other conditions prevail, they do not cause disease. In addition to these microbes that are members of the resident skin microbiome, some bacteria can be temporarily carried as transient colonists of the skin. Examples include *Staphylococcus aureus* which colonizes the anterior nares, the axillae, or perineum in about 8–25% of normal healthy individuals, depending on site. In addition, these bacteria or fungi can also colonize abnormal skin surfaces (e.g. patients with psoriasis or atopic dermatitis often carry *S. aureus* on their skin lesions). Similarly, fungal species such as *Candida albicans* are occasionally carried on the skin surface (e.g. on the hands). The skin is therefore an ecological niche for a variety of microorganisms that, in turn, affect local environmental factors, such as pH. The skin can also be the focus for infections which are usually, but not invariably, caused by external pathogens. These are discussed under the heading of bacterial, fungal, viral, and ectoparasitic infections. The skin can also be affected by infections originating from other internal sites and carried via the bloodstream. Detailed accounts of diseases associated with particular organisms are also presented elsewhere in this book (e.g. for *Mycobacterium leprae* see Chapter 8.6.28 and for *Treponema pallidum* see Chapter 8.6.37). Cutaneous bacterial infections

Bacteria commonly cause infection of the skin either as a primary event or secondary to some pre-existing skin condition. The term 23.10 Infections of the skin Roderick J. Hay

section 23 Disorders of the skin 5696 'pyoderma' is used to describe purulent infection of the skin caused by bacteria. The most common of these infections is the epidermal infection impetigo, but small abscesses such as boils or furunculosis can also result. The most common causes of pyoderma are *S. aureus* or group A streptococci. Uncommonly, but increasingly, both hospital- and community-acquired infection by methicillin-resistant *S. aureus* (MRSA) are being reported. Infections with community-acquired methicillin-resistant staphylococci are more virulent; for example, they are more likely to require surgical intervention and many of these strains carry the Pantón-Valentine leukocidin (PVL) gene which enhances virulence; PVL-positive staphylococci without resistant genes are also present with difficult to treat infections. Other forms of bacterial infection affecting deeper planes of the skin (e.g. dermis or subcutaneous tissue), leading to cellulitis, are also seen regularly in clinical practice. Examples of secondary bacterial infection include secondary infection of atopic dermatitis, Darier's disease, or psoriasis. Impetigo This is a superficial infection confined to the epidermis caused by *S. aureus* (see Chapter 8.6.4) or group A streptococci (see Chapter 8.6.2), the cause depending, to some extent, on geography and climate. In temperate zones, the normal cause is *S. aureus* but in the tropics streptococci are more common; although in about 20% of cases these organisms can both be isolated from lesions, indicating a mixed infection. In most cases this is a primary infection but it might also be secondary to other skin conditions such as insect bites or scabies. Impetigo is common in primary care in temperate climates, its incidence accounting for about 20 per 1000 person years in the under 18 population, and it is among the top 50 most common of human diseases. However, it can also be found in other groups where close contact is likely (e.g. military recruits). It is more common in summer than winter. Pyoderma is more common in the tropics where it is often secondary to another skin disease such as scabies (see next). In most European countries and the United States

of America, where *S. aureus* predominates, some cases present with blistering lesions (bullous impetigo—see next). *S. aureus* produces exfoliotoxins that cleave the human cell adhesion molecule desmoglein 1 (Dsg1). This results in the formation of an epidermal bulla similar to the blister formation in pemphigus foliaceus where Dsg1 is the target of an autoantibody. Very extensive blistering occurs in the childhood infection staphylococcal scalded skin syndrome (SSSS). Production of exfoliotoxin A is more associated with bullous impetigo and exfoliotoxin B with SSSS. Impetigo usually presents with itching or discomfort in the affected area, which becomes red and the surface glazed, with a golden-coloured serous ooze. As stated previously, *S. aureus* infections can also result in bullous lesions (bullous impetigo) (Chapter 8.6.4). Impetigo is most common in children and often develops on the face or trunk, but lesions can be found elsewhere. Infected lesions can also develop from localized wounds, or insect bites, or around areas where the carriage rate is high (e.g. near eczematous plaques, or close to the nose). Patients are generally well and usually show no systemic signs of infection, such as fever. Treatment consists of flucloxacillin or clindamycin, although, if the lesion is very localized, a topical antibiotic such as fusidic acid or mupirocin can be used (short term). Impetigo is contagious, particularly among children at school or in families, and therefore close contacts should be screened for infection. There is little evidence to support the use of antiseptics in primary treatment, however logical. Hand washing, though, has been shown to reduce the risk of impetigo. If the lesions penetrate deeper into the dermis they appear as well defined but localized ulcers known as ecthyma. These occur, particularly, in hot climates or where the site of infection is occluded by tight clothing. Ecthyma can also complicate other skin infections (e.g. chickenpox). It is treated with systemic antibiotics.

Folliculitis and furunculosis *S. aureus* is the usual cause of a boil or furuncle which develops around an infected hair follicle. If the infection is restricted to the superficial part of a follicle the lesion is called folliculitis, whereas if it is deeper, a cutaneous abscess, boil, or furuncle develops. Folliculitis commonly presents in hair bearing areas such as in the beard area. Lesions are small, multiple, and pustular; with the earliest symptom often being itching. Folliculitis-like lesions can also be caused by inflammatory tinea corporis and might also accompany ingrowth of curling hairs in the beard area (pseudofolliculitis) that traps skin commensal bacteria causing a papular or pustular response. A furuncle or boil usually starts as a solitary, tender, and inflamed nodule which, as it develops, points to form a pustular head. The patient might be febrile. Treatment is by incision to release pus followed by antibiotics (flucloxacillin or clindamycin). The most common sites for the formation of boils are the axillae or trunk areas, or even the face. Boils can develop at any age but there might be a cluster of cases within the same household over several months, suggesting transmission within the family. Although it is frequently stated that recurrent boils are indicative of underlying disease such as diabetes or immunosuppression, this is, in reality, unusual; it is more common that the individual is a bacterial carrier. Carriage of *S. aureus* is common in atopsics and history of atopy is therefore quite frequent in those with recurrent furunculosis. Severe and recalcitrant boils are often associated with a virulence determinant, Panton-Valentine leucocidin, carrying strains of *S. aureus*. Lesions that resemble furuncles might appear in the axillae or groin associated with entrapment of commensals. This condition, known as hidradenitis suppurativa, is not associated with *S. aureus*, a useful clue to the diagnosis. Secondary scarring and recurrent attacks are common. There is little effective treatment for this condition (see Chapter 23.11). If several boils amalgamate, the large pustular mass is called a carbuncle. Sites of predilection include the neck.

Cellulitis Superficial infection that spreads laterally in the upper dermis or along the subcutaneous fascia is known as cellulitis. The main cause is infection with Group A streptococci (also see Chapter 8.6.2), but other common causes include *S. aureus*. Cellulitis starts

as a zone of spreading erythema and tenderness with other signs of inflammation such as increased surface temperature. With streptococcal infections the patient is often systemically unwell, with fever and chills. The main sites for infection are the face or the lower legs. Cellulitic infections affecting the dermis and upper subcutaneous tissue are sometimes known as erysipelas. Nongroup A streptococci are also sometimes responsible.

23.10 Infections of the skin 5697 Complications include the development of septicaemia or encroachment on adjacent structures such as the orbital cavernous sinus leading to thrombosis. Rare causes of the same syndrome include zygomycete fungi. Cellulitis requires immediate antibiotic therapy with an oral penicillin such as flucloxacillin or a macrolide antibiotic, and in systemically unwell patients with an intravenous regime. Recurrent attacks are also seen, particularly on the limbs and are often associated with lymphoedema. Management is difficult as local antiseptics and improved drainage by themselves do not appear to prevent recurrences and long-term oral penicillin V (phenoxymethylpenicillin) is often necessary for recurrent disease. However, in lower limb cellulitis there is an association with skin lesions such as the cracks caused by athlete's foot and treatment aimed to heal local skin defects (e.g. antifungals), is indicated. In the early phases, these infections are easily confused with early necrotizing fasciitis (see Chapter 8.6.2) where the surrounding erythema spreads slowly but the patient remains unwell and the overlying skin becomes hypoaesthetic. Necrotizing fasciitis can be caused by group A streptococci but also by mixed bacterial infections after surgery or a compound fracture. The spreading erythema, dull ache, systemic reaction, and the reduction in overlying sensation should alert the physician. Surgical exploration is warranted, which then reveals a necrotic and oedematous reaction in the underlying dermis and deeper fascial planes. Treatment is by early surgical debridement, which is an essential part of management in addition to systemic broad spectrum antibiotics. Other bacterial skin infections Gram-negative bacteria, such as a *Pseudomonas* species, can cause skin lesions. Most commonly these present as interdigital infections of the feet. It occurs often in those whose occupations involve wearing heavy footwear or wet working conditions. The skin becomes eroded and cracked; the toe web is also painful. If *pseudomonas* is present, the edge of the lesions appears greenish. Often, Gram-negative interdigital infection follows fungal infection (tinea pedis). Treatment with topical povidone-iodine or 1-2% acetic acid is useful, but sometimes the lesion flares up after treatment and this heralds the return of a dermatophyte infection. Gram-negative folliculitis is a painful form of folliculitis presenting with small tender pustules on the trunk and limbs, sometimes associated with fever. It is associated with bathing in whirlpool bathtubs contaminated with Gram-negative bacteria ('hot tub folliculitis'). Ecthyma gangrenosum is a necrotic ulcerative condition which is a manifestation of disseminated *Pseudomonas* infection in patients with Gram-negative septicaemia, usually in the context of neutropenia. The lesions develop as nodules that evolve into raised plaques with a black necrotic centre. Tuberculosis of the skin and other

mycobacterial infections *Mycobacterium tuberculosis* is a rare cause of skin infection in most industrialized societies although it remains a regular infection seen in many tropical areas (see Chapter 8.6.26). With the increase in HIV and drug-resistant tuberculosis, there have been increasing numbers of cases seen in recent years. Once common, cutaneous tuberculosis presents with several different clinical forms, all of which reflect the state of the host's immunity and the route of infection to reach the skin, either through haematogenous spread, by direct spread from an underlying infected structure (e.g. lymph node or bone), or by direct inoculation into the skin. In immune individuals there are usually few organisms, which is described as paucibacillary. In other

forms of infection, particularly in those who are immunocompromised, there are many organisms, which is described as multibacillary disease. The term tuberculid is used to describe skin lesions that do not contain viable organisms but that are associated with tuberculosis elsewhere. Usually, but not always, the pathogenesis is through an antigen-mediated reaction (e.g. vasculitis). Examples include erythema nodosum and erythema induratum (Bazin's disease), both of which are forms of vasculitis and which can, in some countries, be caused by tuberculosis among other causes. In papulonecrotic tuberculid, the rash is a reaction to disseminated and particulate antigen and, in some cases, viable bacteria are present. This can progress to infective forms of tuberculosis such as lupus vulgaris. The main cutaneous forms of infection seen are tuberculosis verrucosa cutis, scrofuloderma, lupus vulgaris, and papulonecrotic tuberculid. The clinical and immunological features are seen in Table 23.10.1. Cutaneous tuberculosis is seen more frequently now with the spread of HIV/AIDS. Nontuberculous mycobacteria can also infect the skin. These include *Mycobacterium chelonae* and *Mycobacterium fortuitum* which can cause cold abscesses, often as a result of local skin injury, including needlestick injury or surgical or cosmetic procedures. *Mycobacterium ulcerans* is the cause of rapidly spreading skin ulceration or Buruli ulcer seen in tropical and semitropical areas, ranging from West Africa to south Australia. It is associated with

Form	Immune status/organisms	Clinical features
Evidence of TB elsewhere	Tuberculosis verrucosa cutis TB +ve	No organisms
Warty lesions on peripheries	Slow spread	No
Scrofuloderma	TB +ve or -ve	Organisms
Purplish plaques over lymph node, often in neck	Lymphadenopathy	Lupus vulgaris TB +ve
No organisms	Plaques on face or trunk	Heals with central scarring
Risk of squamous cell carcinoma	Uncommon	Papulonecrotic tuberculid
Usually TB -ve. Seen in healthy and HIV +ve patients.	Organisms	Multiple papules, with central area of necrosis, limbs more than trunk
Often other sites (e.g. lungs)	TB, tuberculin test.	

section 23 Disorders of the skin 5698 exposure to fresh water and a potential link with aquatic insects has been proposed. It is difficult to arrest the process with antibiotics and the most successful form of management is rifampicin and streptomycin plus surgical excision. *Mycobacterium marinum* is an infection usually contracted from tropical fish kept in a fish tank (fish tank granuloma); however, naturally acquired infections are seen in the tropics, for example, in association with commercial fish-farming (Chapter 8.6.27). It presents with an area of localized pustular swelling, and granulomatous infiltration or ulceration, usually on a peripheral site such as a finger. In some cases there is local lymphadenitis (sporotrichoid spread). The main differential diagnosis is sporotrichosis or leishmaniasis. Treatment with a range of different antibiotics from minocycline to rifampicin has been used. Cutaneous fungal infections With the exception of infections due to *Malassezia* species, most fungal infections of the skin are extrinsic. The most common of these, dermatophytosis, is seen in all countries, whereas *Malassezia* infections are more common in the tropics. *Candida* infections of the skin are less common, but are seen regularly as secondary skin infections. Cutaneous viral infections Certain viruses are capable of establishing infections of the epidermis and dermis, but, strictly speaking, there are no viral commensals of the skin. The main infections are the human papillomavirus (HPV) infections (warts) and herpes zoster and simplex. These infections are discussed in detail in Section 8. Table 23.10.2 outlines common HPV types and associated clinical patterns. Ectoparasitic infestations *Demodex folliculorum*, an acarid mite, is a commensal on normal skin, but there are also infestations due to ectoparasites—scabies and pediculosis (lice). These are described in detail in Chapter 8.12. FURTHER READING Beyt BE Jr, et al. (1981). Cutaneous mycobacteriosis: analysis of 34 cases with a new classification of the disease. *Medicine (Baltimore)*, 60, 95–109. Daikos GL, et al. (1998). Disseminated miliary tuberculosis of the skin in patients with AIDS: report of four cases. *Clin Infect*

Dis, 27, 205–8. Fogo A, Kemp N, Morris-Jones R (2011). PVL positive *Staphylococcus aureus* skin infections. *Br Med J*, 343, d5343. Grice EA, Segre JA (2011). The skin microbiome. *Nat Rev Microbiol*, 9, 244–53. Koning S, et al. (2012). Interventions for impetigo. *Cochrane Database Syst Rev*, 1, CD003261. Schachner LA (2005). Treatment of uncomplicated skin and skin infections in the pediatric and adolescent patient populations. *J Drugs Dermatol*, 4 Suppl 6, S30–3. Seal DV, Hay RJ, Middleton K (2000). *Skin and wound infection: investigation and treatment in practice*. Martin Dunitz Ltd, London. Stanley JR, Amagai M (2006). Pemphigus, bullous impetigo, and the staphylococcal scalded-skin syndrome. *N Engl J Med*, 355, 1800–10. Table 23.10.2 Clinical patterns and common types of human papillomavirus Skin or mucosal infection HPV typea Common, plantar, mosaic warts 1, 2, 4 Plane warts 3, 10 Butcher’s warts 7 Bowen’s disease (some cases) 16 Epidermodysplasia verruciformis 3, 5, 8, 12, 36–38, and others Condyloma acuminata (genital warts) 6, 11 Intraepithelial neoplasia (e.g. cervical dysplasia, bowenoid papulosis) 16 HPV, human papillomavirus. a Only commonly associated forms are shown.

Revision #1

Created 2026-01-22 16:42:54 UTC by Omar Ayman

Updated 2026-01-22 16:42:54 UTC by Omar Ayman