CHAPTER

1

Bacterial Infections

FOLLICULITIS

INTRODUCTION

Superficial folliculitis also called as follicular impetigo of Bockhart, is inflammation which is confined to the ostium of the hair follicle or extends only very slightly below it, and heals without scar formation. Deeper follicular extension of infection below the infundibulum is called furuncle or sycosis.

Superficial folliculitis is caused by:

- **Infection:** With *Staphylococcus aureus*, coagulase-negative staphylococci, *Pseudomonas aeruginosa*, pityrosporum yeast and dermatophytes.
- **Non-infective causes due to physical or chemical irritation of hair follicle:** e.g. due to occupational contact with mineral oils, tar products used for therapeutic or occupational purpose, due to use of adhesive dressings and plasters and traumatic folliculitis following epilation and waxing of hair (Fig. 1).

It commonly affects scalp and limbs in children and beard area in adults.

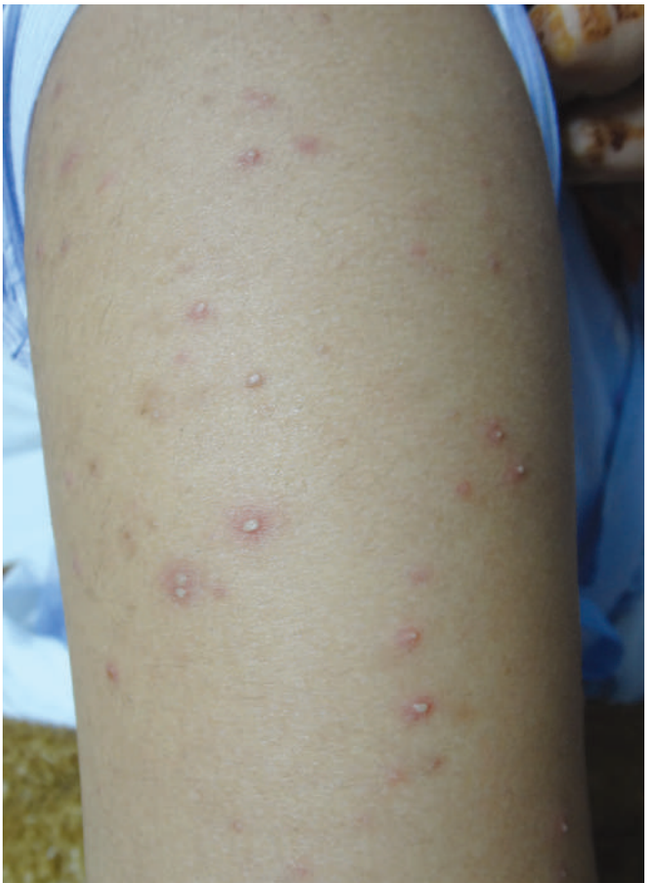


Fig. 1: Folliculitis following epilation of hairs by waxing with multiple follicular pustules surrounded by rim of erythema

CLINICAL FEATURES

The lesions present as small, follicular papules, tiny pustules or domed yellow pustules surrounded by a red narrow rim of erythema (Figs 2 and 3). Sometimes crust is seen covering the red pouting follicular

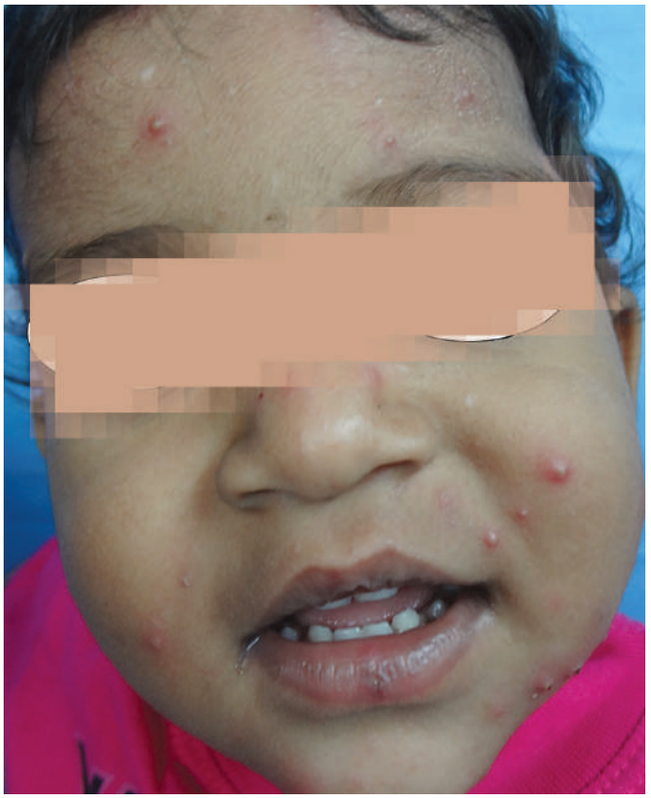


Fig. 2: Multiple follicular papulopustules on face

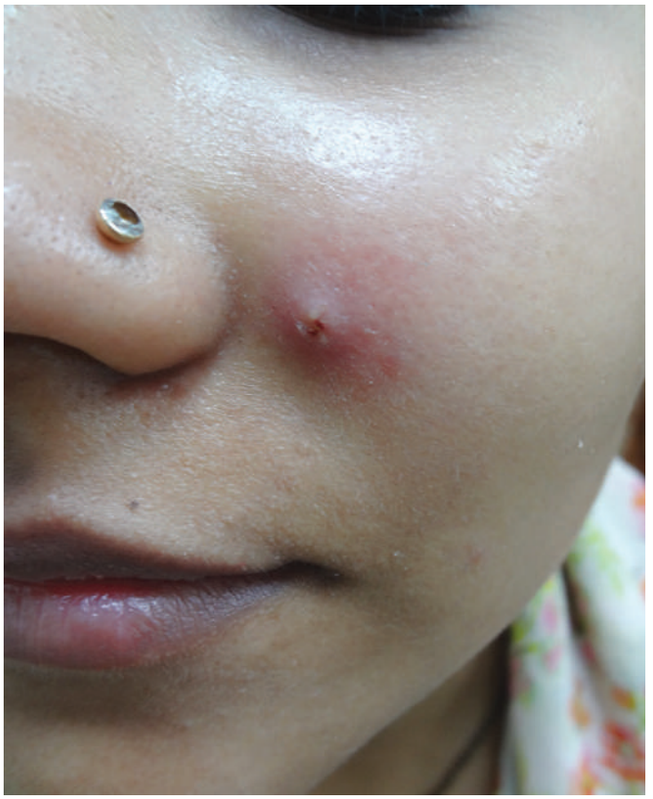


Fig. 4: Crust covering the red pouting follicular opening

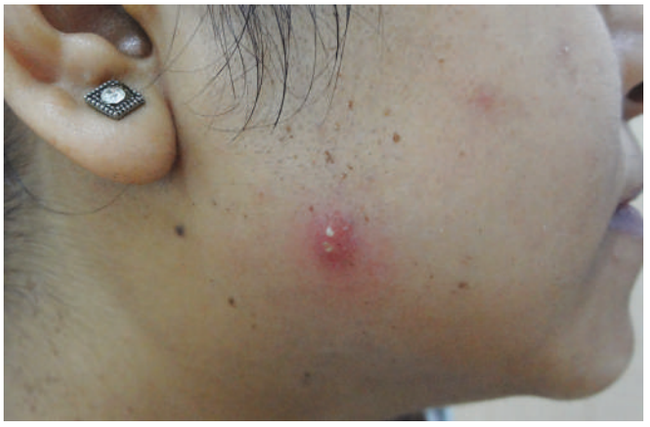


Fig. 3: Pustule surrounded by a rim of erythema

opening (Fig. 4). Lesions are rarely painful. Folliculitis is usually self-limiting and heals in 7 to 10 days, but sometimes may become chronic.

TREATMENT

For superficial folliculitis caused by physical or chemical irritants, irritant should be removed. Mild folliculitis caused by staphylococcus can be treated with topical antiseptics, and severe folliculitis with topical or systemic antibiotics. Topical antibiotics include fusidic acid, mupirocin, clindamycin and retapamulin. Systemic antibiotics include flucloxacillin, clarithromycin, clindamycin, roxithromycin, penicillin and erythromycin. In patients with persistent or recurrent infection, the usual sites of staphylococcal carriage like nares, axilla, perineum and perianal area should be sought in patient and their contacts and treated with twice daily application of topical mupirocin or fusidic acid for 10 days. Since infection is contagious, hand washing is essential for prevention of spread.

FURUNCLE

INTRODUCTION

Furuncle, also called as boil, is an acute, deep necrotic infection of hair follicle, usually the vellus hair. Abscesses are also deep necrotic infection of soft tissue but may not necessarily be centered on the hair follicle. When multiple hair follicles get infected, it is called carbuncle, which presents with very tender, large suppurative lesions with multiple draining sinuses. Furuncle is mostly caused by infection with *Staphylococcus aureus* and methicillin resistant *Staphylococcus aureus* (MRSA). However, sterile furuncles can follow injection of oil-based drugs into the skin.

Predisposing factors include patients who are chronic carrier of staphylococcus aureus in their nares, axilla and perineum from where the infection is disseminated through fingers and by clothing; malnutrition; diabetes and HIV.

CLINICAL FEATURES

In contrast to superficial staphylococcal folliculitis, furuncles are deep-seated nodules. A furuncle starts as a small, inflammatory, follicular nodule, which soon becomes pustular and then turns necrotic with discharge of purulent material. Furuncles heal with a violaceous macule eventually with a permanent scar. Tenderness is invariable and larger lesions may cause throbbing pain. It may be accompanied with fever and constitutional symptoms. Furuncle present on upper lip (Fig. 5) and cheek can cause cavernous sinus thrombosis. Furuncles are commonly seen involving the face, neck, arms, fingers, wrists, buttocks and the anogenital region. Staphylococcus carrier sites like nares, axilla, perineum and perianal area are responsible for recurrent and persistent infection.

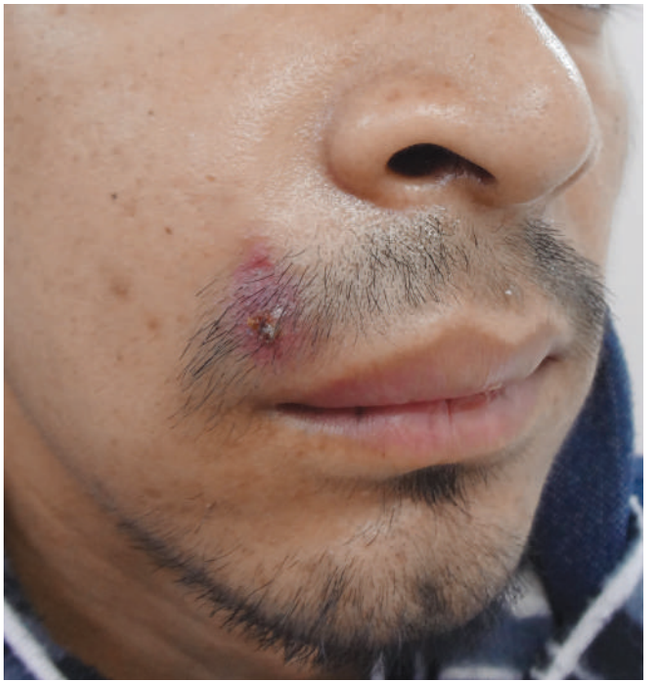


Fig. 5: Furuncle on upper lip can cause cavernous sinus thrombosis

TREATMENT

Surgical incision and drainage are the first line of therapy and should be done for isolated lesions. Oral antibiotics are indicated in patients with severe disease, multiple lesions, associated cellulitis, presence of systemic illness, diabetes, HIV and sites at which lesions are difficult to drain by incision like face, genitalia and hands. Oral antibiotics of choice include doxycycline, minocycline, clindamycin, linezolid and trimethoprim/sulfamethoxazole. Treatment of MRSA include linezolid, ceftaroline, teicoplanin, vancomycin, rifampicin and tigecycline. For nasal carriage of staphylococcus, topical fusidic acid or mupirocin is applied 2 times a day for 10 days on all carrier sites.

CELLULITIS AND ERYSIPELAS

INTRODUCTION

Cellulitis is painful diffuse deep inflammation involving the loose subcutaneous tissue, which may be acute, subacute or chronic. Erysipelas is more superficial as compared to cellulitis and is infection of the dermis and superficial subcutaneous tissue. Due to its superficial dermal involvement, erysipelas has well-defined raised edge as compared to cellulitis.

Causative organisms include infection with streptococcal diseases, group A more than group B and staphylococcus infection including methicillin resistant staphylococcus infection. Streptococcus is more likely to cause non-purulent cellulitis. Purulent cellulitis characterized by presence of pustules and purulent discharge is likely to be caused by *Haemophilus influenzae* type B which is seen causing facial cellulitis in children less than 2 years of age. In cellulitis seen in patients having venous or lymphatic compromise, groups B and G streptococci predominate. Periorbital cellulitis secondary to sinusitis is caused by *Streptococcus pneumoniae* in addition to group A streptococci and *S. aureus*. Excoriations in eczema, trauma, tinea pedis of web spaces of toes and lymphedema offer sites for entry of infection.

CLINICAL FEATURES

Patients complain of redness, heat, swelling, pain and tenderness.

Edge of the lesion is well demarcated, raised and palpable (Fig. 6) in erysipelas due to superficial involvement as compared to cellulitis which is diffuse (Fig. 7), although intermediate and overlapping picture may be present. Blistering is commonly seen in erysipelas (Figs 8A and B). Severe cellulitis may progress to necrosis, fasciitis or myositis. Lymphangitis and regional lymphadenopathy are common with fever and malaise. Systemic features of sepsis may be present. The leg and face are the commonest sites involved.

TREATMENT

Uncomplicated cellulitis and erysipelas with no signs of systemic toxicity and no uncontrolled

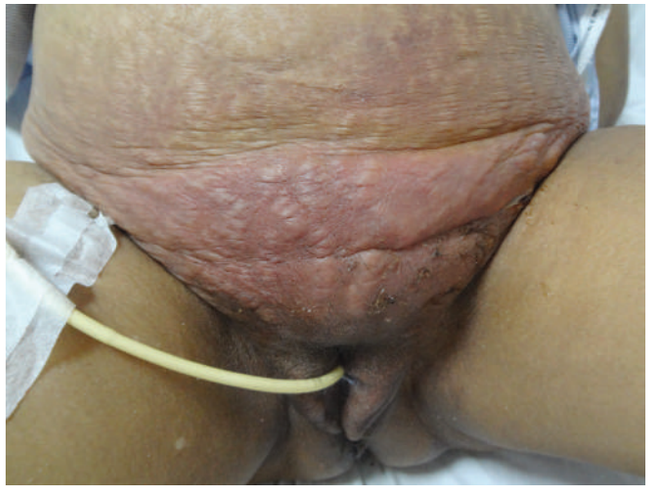


Fig. 6: Erysipelas; note the well demarcated, raised and palpable edge of lesion involving lower abdomen and perineum

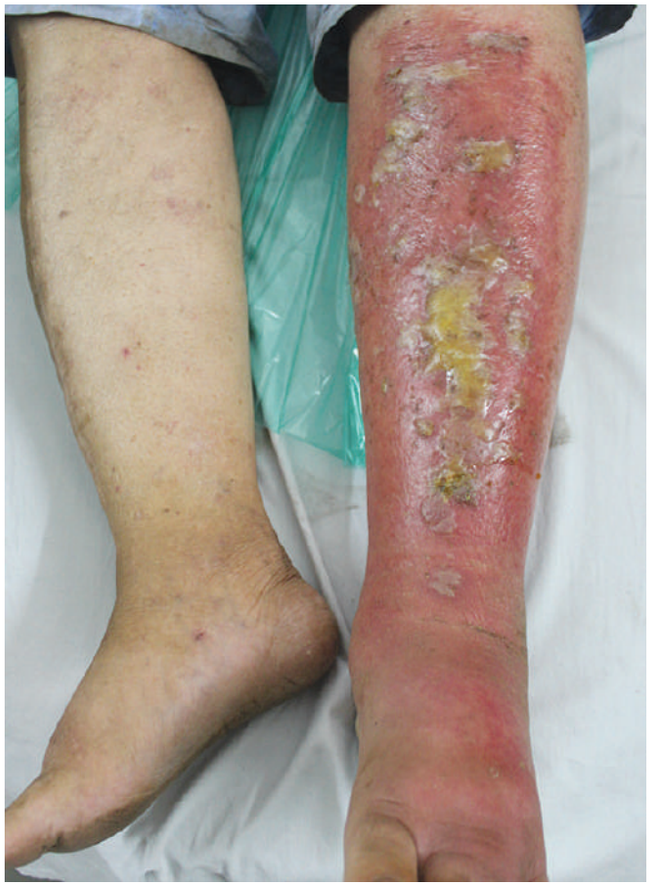
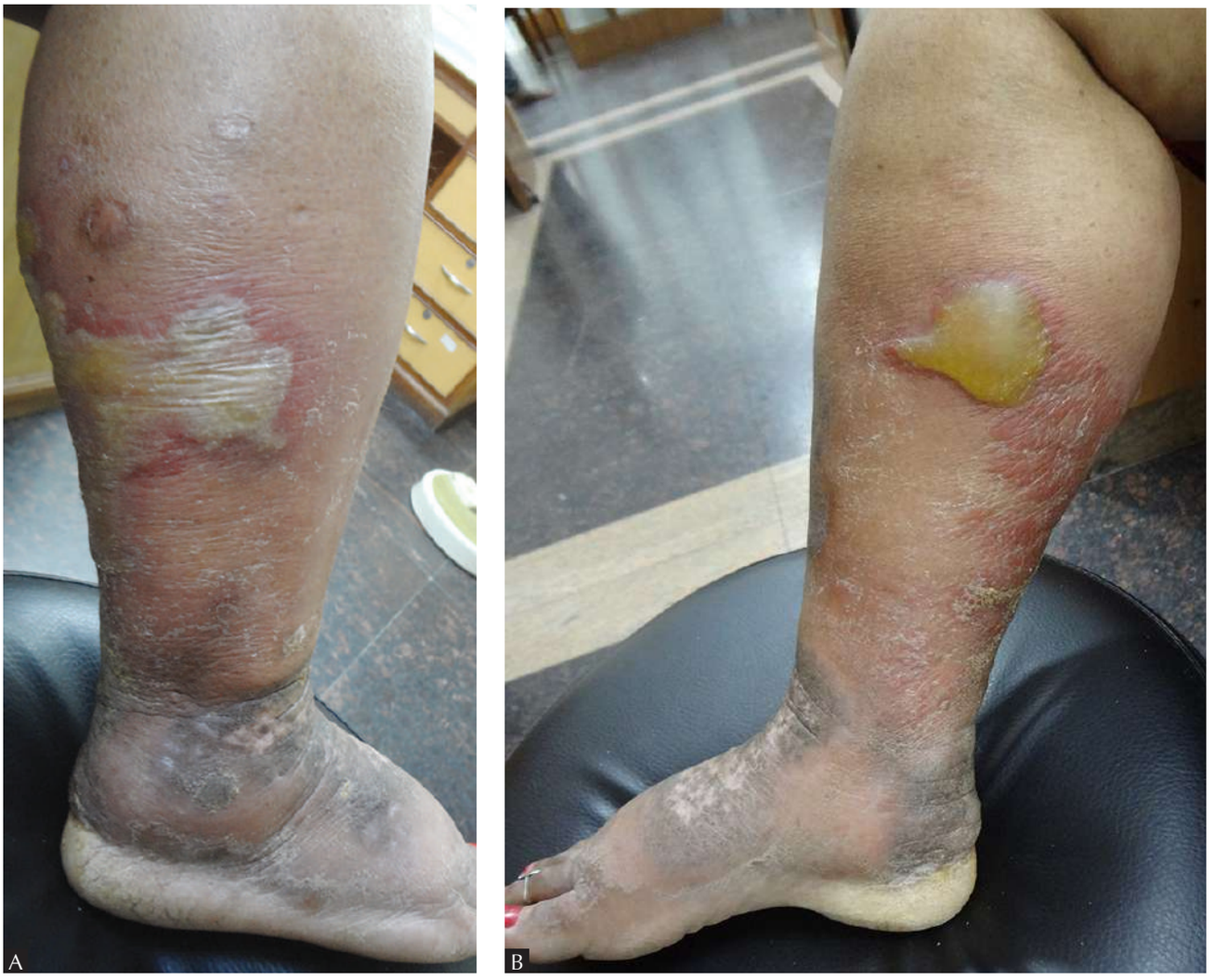


Fig. 7: Cellulitis affecting lower leg with diffuse erythema, tenderness and blistering



Figs 8A and B: Erysipelas with erythema and blistering caused most probably due to scratching in case of eczema in lower leg

comorbidities like diabetes are managed with systemic broad-spectrum antibiotics covering streptococcus and staphylococcus infection. These include oral penicillin, penicillin and flucloxacillin, flucloxacillin, clarithromycin, clindamycin, ceftriaxone, roxithromycin and amoxicillin with clavulanic acid.

Intravenous antibiotics and admission are indicated in patients with systemic toxicity such as tachycardia, acute confusion, hypotension, tachypnea; immunosuppressed patients; patients with comorbidities like chronic venous insufficiency, peripheral arterial disease, morbid obesity; and debilitated patients. Antibiotics of choice are penicillin G, vancomycin, ciprofloxacin, linezolid, teicoplanin, daptomycin and oritavancin.

IMPETIGO

INTRODUCTION

Impetigo is a contagious, superficial bacterial infection of the skin. Causative organism includes staphylococcal and less frequently streptococcal infections. Impetigo is more commonly seen in children as compared to adults.

CLINICAL FEATURES

Two main clinical forms of impetigo are recognized: non-bullous and bullous impetigo.

In non-bullous impetigo, very thin-walled vesicle, which is seldom seen, is present on an erythematous base. The vesicle ruptures rapidly to exude serum, which dries to form a yellowish brown crusts (Figs 9 and 10). This crust is thicker and 'dirtier' in infections caused by streptococcus. Lesions gradually increase by peripheral extension without any central healing. Dried crust exfoliates to leave erythema, which fades without scarring. Severe cases may be associated with regional adenitis and fever. Lesions most commonly involve areas around the nose and mouth.

In bullous impetigo, the bullae are larger in size, around 1–2 cm in diameter, and do not rupture rapidly, persisting for 2 or 3 days. The bullae are initially filled with clear fluid, which later turns cloudy, ruptures and dries to form a thin, flat, brownish crust (Figs 11 and 12). Circinate lesions are formed due to central clearing and peripheral extension. Face is the most commonly affected site. Regional adenitis is uncommon.

Impetigo is usually a self-limiting infection which resolves within days to weeks with antibiotics. Since it is contagious, close contact can be infected.

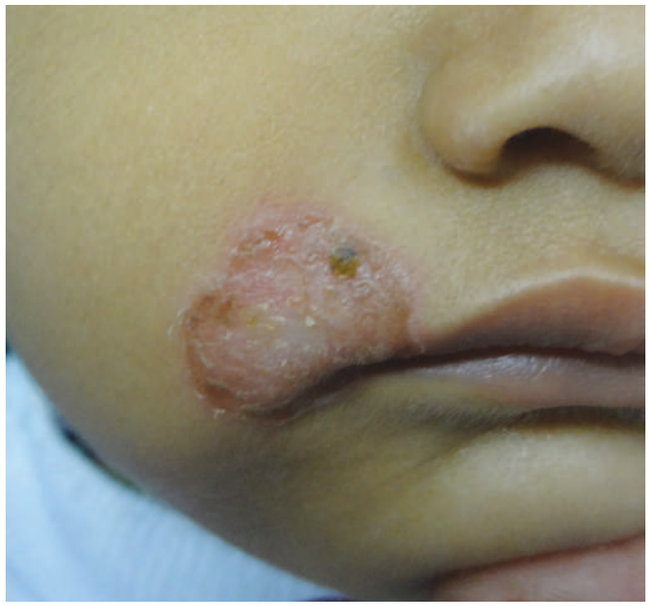


Fig. 9: Non-bullous impetigo: honey-colored crusted lesion present near the angle of mouth in a child

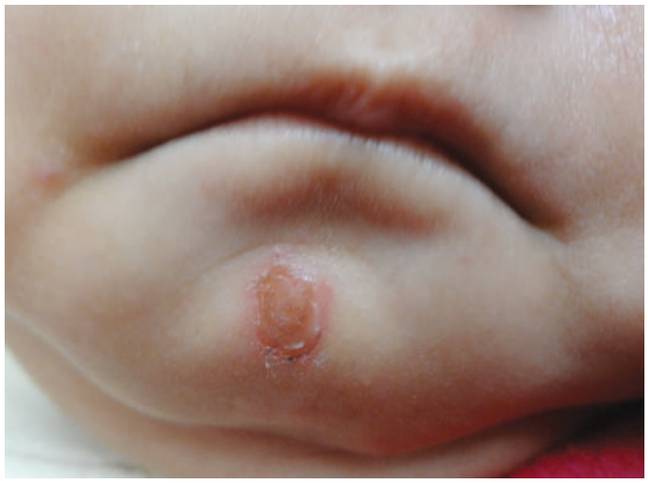


Fig. 10: Non-bullous impetigo: honey colored crusted lesion on the chin in an infant

TREATMENT

The affected skin should be disinfected daily with chlorhexidine, povidone–iodine or sodium hypochlorite. All close contacts should use antibacterial soap to wash hands to reduce onward transmission. Topical antibiotics, like mupirocin, fusidic acid, retapamulin and clindamycin should be applied on the affected skin twice a day for 5–7 days. Systemic antibiotic given for 1 week is indicated in extensive impetigo, in patients with marked bullous component and in those with palpable lymphadenopathy. First-line antibiotics include flucloxacillin, cephalexin, clindamycin, co-amoxiclav and cloxacillin. Second-line antibiotics include erythromycin, clarithromycin and co-trimoxazole. Trimethoprim and tetracyclines are the third-line antibiotics.

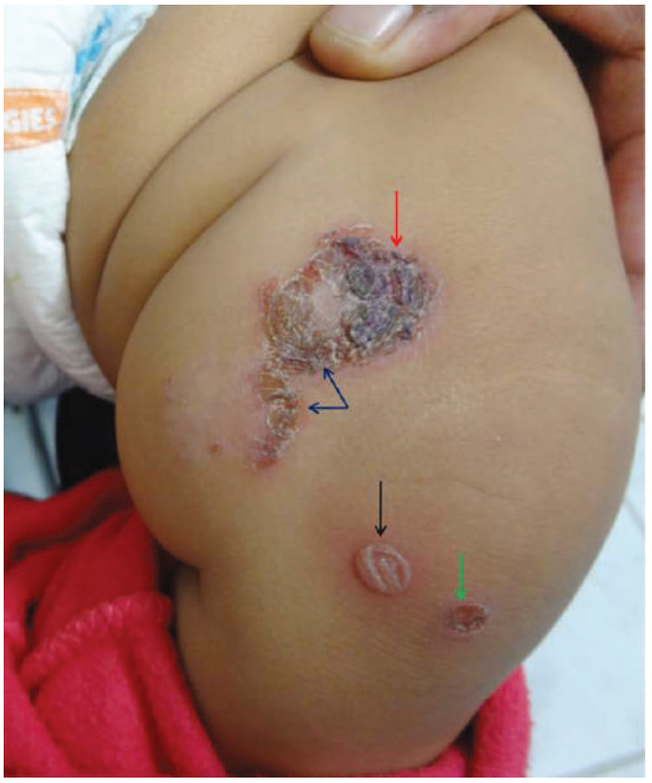


Fig. 11: Bullous impetigo: Flaccid bulla (black arrow) filled with clear fluid with erythema of surrounding skin, erosion (green arrow) and crusted [both hemorrhagic (red arrow) and honey colored (blue arrow) crust] lesions on the infant's thigh. Lesions show peripheral extension with central healing forming both annular and circinate lesions

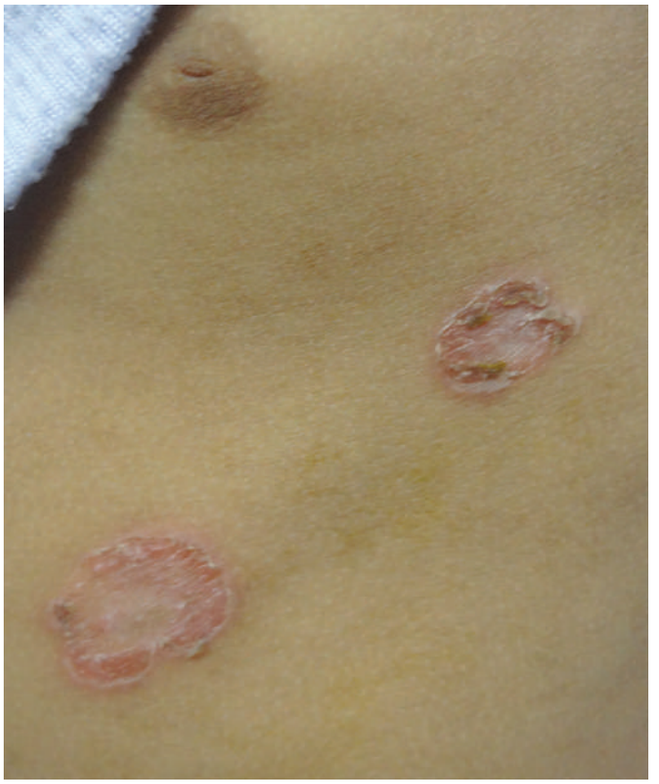


Fig. 12: Bullous impetigo: Crusted lesions forming annular pattern with peripheral extension and central healing on trunk of a child

ERYTHRASMA

INTRODUCTION

Erythrasma is a superficial infection of the skin caused by *Corynebacterium minutissimum*. Erythrasma is associated with diabetes mellitus.

CLINICAL FEATURES

It commonly affects axillae, groins, intergluteal region, submammary area and toe webs. It presents with irregular, sharply marginated, red to brown patches (Figs 13 and 14). New lesions appear smooth, while older lesions are finely creased and scaly. Lesions are mostly symptomless, but may cause irritation leading to scratching and lichenification. Wood's lamp examination shows coral-red fluorescence.

TREATMENT

Without treatment, erythrasma tends to persist indefinitely. Treatment options include topical azole antifungals, like clotrimazole and miconazole, erythromycin and fusidic acid for 2 weeks. Oral agents include single dose of 1 g clarithromycin.

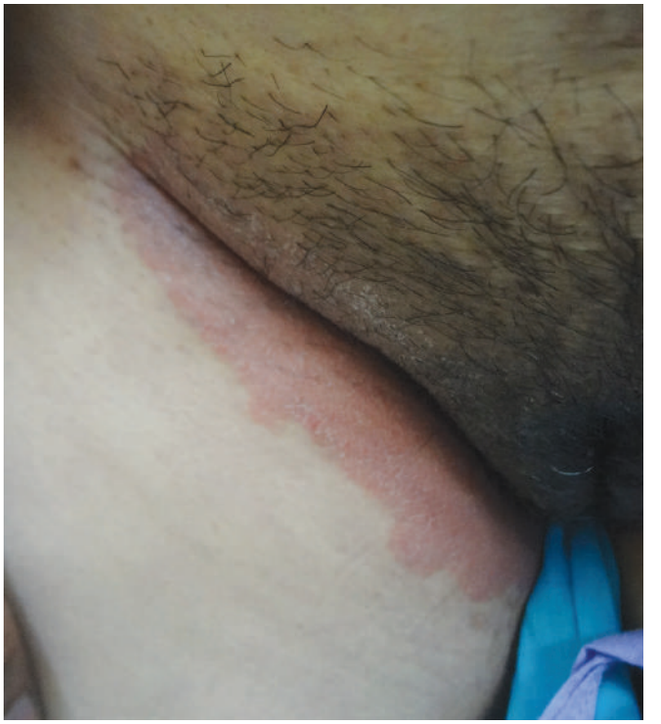
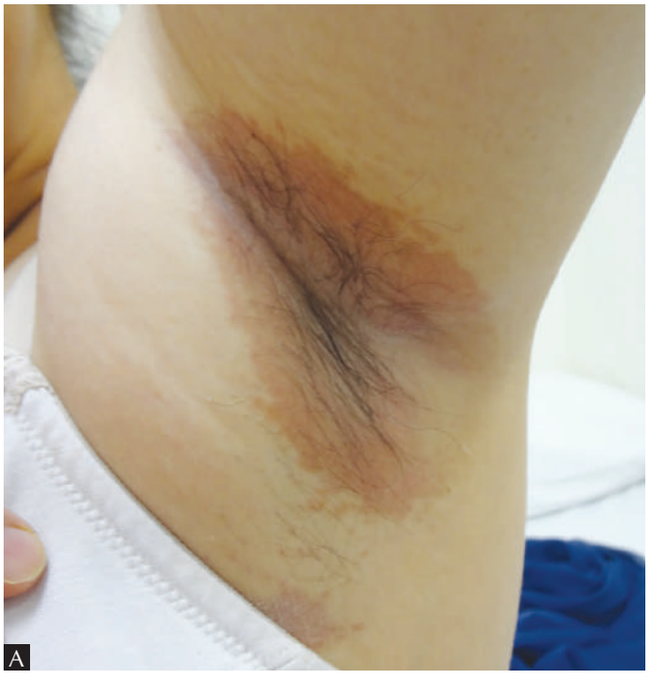
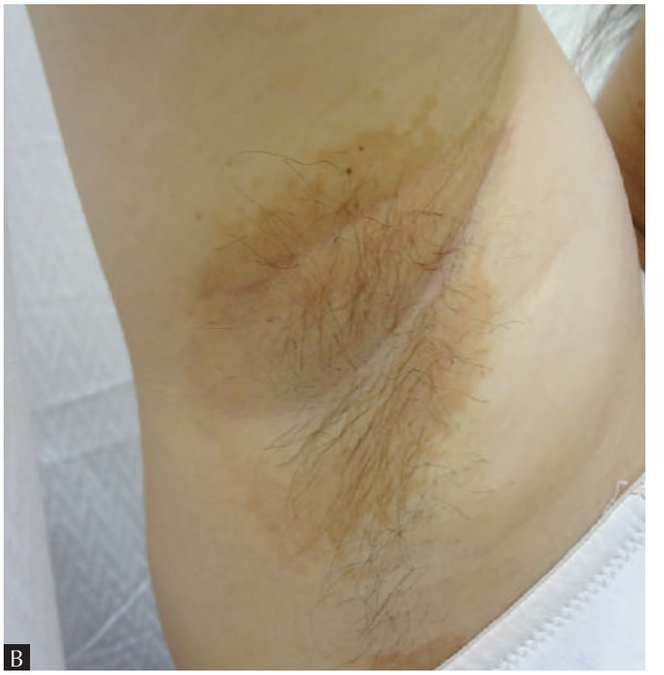


Fig. 13: Erythrasma with erythematous well defined plaque with fine scaling and no central clearing





Figs 14A and B: Erythrasma of axilla

PITTED KERATOLYSIS

INTRODUCTION

It is a superficial skin infection caused by *Corynebacterium, Dermatophilus congolensis, Kytococcus sedentarius* and *Streptomyces*.

CLINICAL FEATURES

It can affect any part of the sole, especially the pressure-bearing and friction areas. It presents with discrete, shallow, circular, punched-out lesions, which may coalesce to produce irregular erosions (Figs 15–18). Green or brown discoloration of the involved skin may be seen. It is often associated with hyperhidrosis, maceration, stickiness and a foul smell. Lesions are usually asymptomatic.

TREATMENT

Treatment includes topical fucidin ointment and other topical antibiotics, imidazole antifungals such as clotrimazole and benzoyl peroxide. Treatment of hyperhidrosis includes potassium permanganate soaks, aluminum chloride, iontophoresis and botulinum toxin injection.

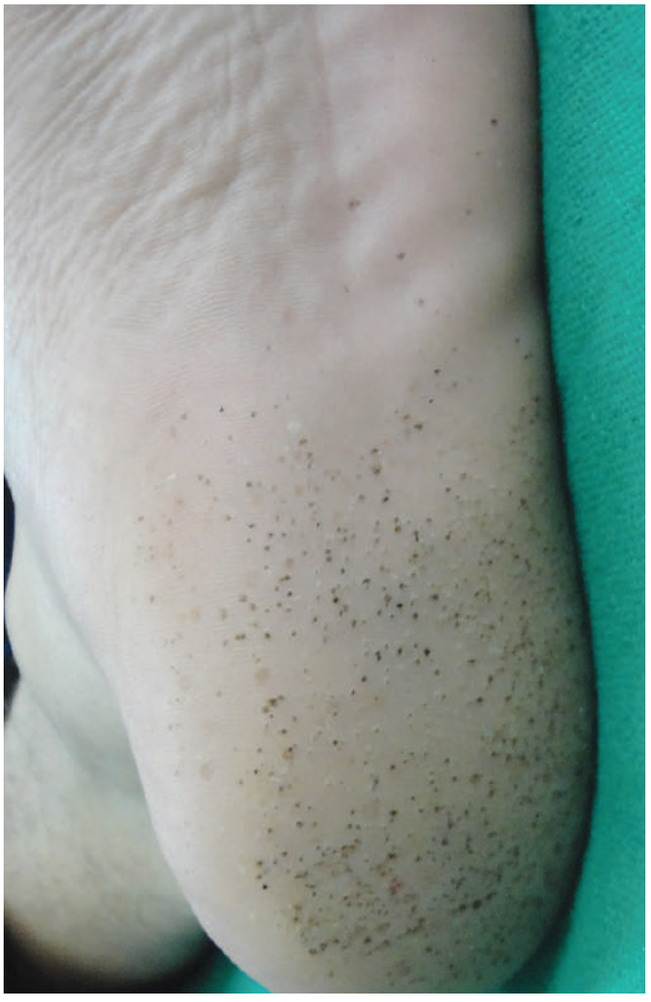


Fig. 15: Pitted keratolysis: Multiple micro pits (1 mm) on the heels (pressure bearing areas)

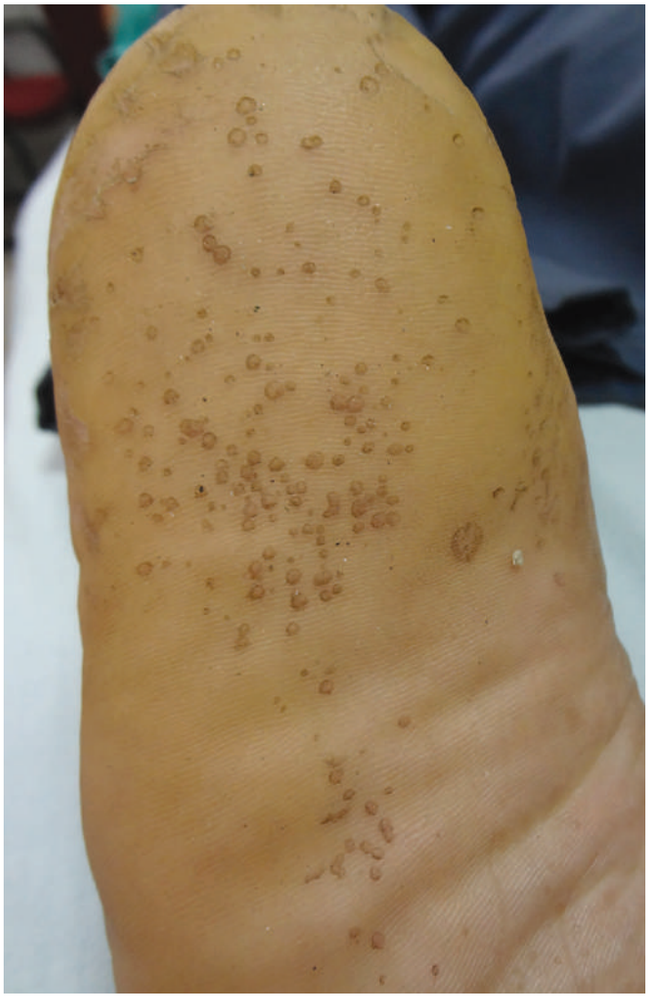


Fig. 16: Pitted keratolysis: Multiple pits on the sole

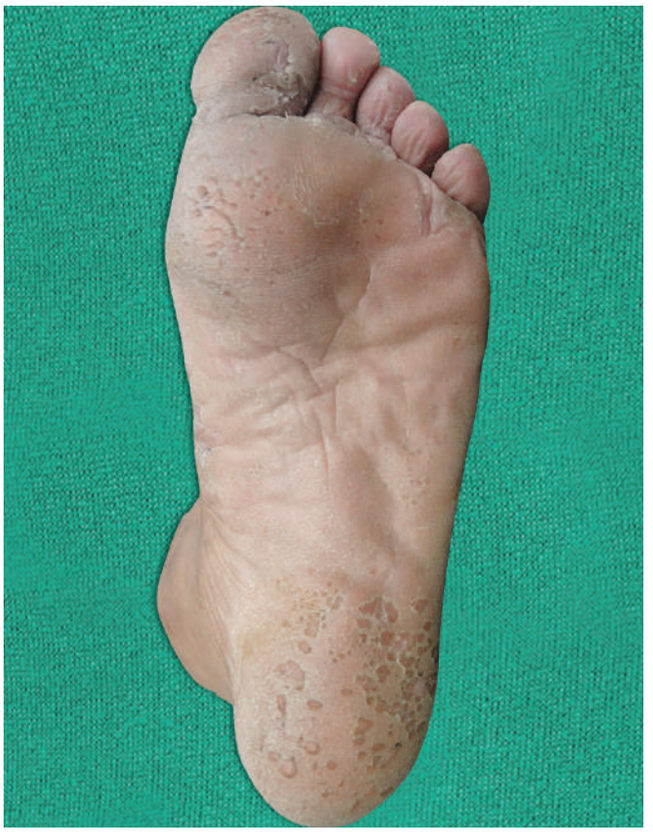


Fig. 17: Pitted keratolysis: Pits coalescing to form larger craters on soles

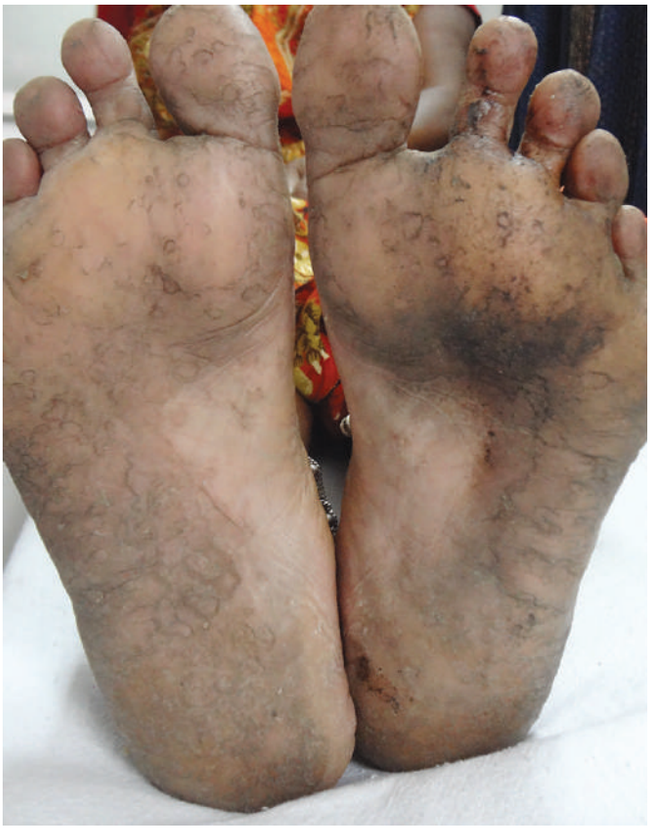


Fig. 18: Pitted keratolysis: Pits coalescing to form larger craters on soles on friction and pressure areas