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Bacterial Skin Infections in the Elderly
Diagnosis and Treatment

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Contents

Abstract

Skin and soft tissue infections are quite common in elderly people. A number of special conditions and circumstances need to be considered in the diagnosis and therapy. It is important to try to establish the causative organism, exclude other cutaneous disorders and identify precipitating factors. Treatment modalities include antiseptics, topical and systemic antibacterials, dressings and biotherapy.

Skin infections presenting with erythema, blisters, pustules, and ulcerations or in body folds are described in detail. Cellulitis and infected ulcers are the most commonly encountered cutaneous infections in the elderly. Accurate and quick diagnosis and treatment are imperative to prevent significant morbidity and mortality. Appropriate antibacterials, antiseptics and dressings are necessary depend-

ing on the severity of the clinical presentation and resistance patterns. Laboratory tests, such as skin swabs, to establish the exact pathogen take time and the results might represent colonisation rather than infection of the skin. Cellulitis should be clinically distinguished from erysipelas and necrotising fasciitis. The latter is a life-threatening condition, which in the majority of cases requires surgical debridement of the infected tissue. Blisters and honey-coloured crusts are typical features of impetigo. It is very contagious and close contacts should be examined. Folliculitis is a commonly seen skin infection, which often responds to the use of antiseptics and topical antibacterials. More severe pustular skin eruptions, such as furunculosis and carbunculosis, usually require treatment with systemic antibacterials. Intertrigo and erythrasma have a predilection for the body folds, especially the axillae and groin, and topical therapy is usually sufficient. Secondary skin infections are often the result of persistent pruritus associated with increasing dryness of the aging skin. Emollients and antihistamines are useful measures. Primary cutaneous disorders and systemic diseases should be excluded with the aid of appropriate investigations, such as blood tests and skin biopsy. *Staphylococcus aureus* and β-haemolytic streptococci are the most common causative organisms of cutaneous infections.

1. General Considerations of Bacterial Skin Infections in the Elderly

1.1 Aetiology

For any given medical problem, morbidity and mortality are increased by 2- to 3-fold in the elderly. The elderly population has an increased incidence of pneumonia, urinary tract infections, and skin and soft-tissue infections.[1]

This increased susceptibility to infection has been attributed to a dysregulation and decline of immune function with age, particularly in cell-mediated immunity.[2] Gram-negative bacterial colonisation of the oropharynx is common in elderly patients, probably because of a reduced production of adherence proteins for Gram-positive bacteria.[3] The prevalence of skin colonisation by *Proteus mirabilis* and *Pseudomonas aeruginosa* in the over 65-year-old population is increased by about 25% compared with those younger.[4]

Aging is associated with thinning and increased dryness of the skin and a reduced blood flow, therefore increasing the chance of injury and reducing the speed of healing. Both malnutrition and obesity are further risk factors for infection and poor wound healing.

In addition, the elderly patient may not be able to accurately report any symptoms and has to rely on relatives or carers, which may delay presentation.

1.2 Clinical and Laboratory Diagnosis

Infections in the elderly may present with atypical signs and symptoms. Fever is often low grade or absent. On the other hand, because of delay in presentation, patients may present with septic shock or confusion. Skin and soft tissue infections may complicate an underlying chronic skin disorder, causing further diagnostic difficulties.

Traditionally, skin infection is confirmed by taking a swab of the wound surface, which is then sent for bacterial analysis. *Staphylococcus aureus* and β-haemolytic streptococci are the most common cutaneous pathogens, and their presence is usually of clinical significance. Coagulase-negative staphylococci, coryneform bacteria, micrococci and Gram-negative bacilli are mostly skin contaminants and non-pathogenic.

The amount or type of bacteria identified by wound swab does not necessarily relate to the presence or absence of wound infection. In particular, chronic wounds are contaminated or colonised

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with bacteria without an associated host reaction.[5,6] Wound swabs, therefore, can provide some information about the type of bacteria present, but diagnosis of infection is usually based on clinical features. An infected wound will either not start to heal or will stop healing. Other useful signs are the presence of pus, malodour, worsening exudate, increase in pain, presence of a neutrophilia and change in appearance of the wound, including erythema.

Alternatively, tissue for culture should be obtained by needle aspiration or biopsy, although the yield of significant positive culture results is low.[7] Viral cultures may be necessary if there is the clinical suspicion of herpes zoster or herpes simplex infection. Blood cultures are useful if the patient is septicaemic.

1.3 Differential Diagnosis

For the differential diagnosis of common skin problems presenting with erythema, blisters, pustules, ulcerations or in body folds, see table I.

1.4 Treatment

The principles of treatment of bacterial skin infections in the elderly are the same as for any age group, but there are some important differences.

Absorption, distribution, metabolism and elimination of drugs are altered in the elderly because of a variety of reasons.[1] Increased drug interactions resulting from polypharmacy, comorbidity and reduced renal function are some of the factors that influence pharmacokinetics. Compliance with treatment may be impaired. The practicalities of some therapies need to be considered, for instance if the patient experiences arthritis or dementia, or impaired hearing or eyesight. Social circumstances also play an important role, for example if a patient lives alone or is cared for in a long-term residential or nursing home.

1.4.1 Antibacterials

Infections require effective treatment with an antibacterial. The choice of drug depends on the

Table I. Differential diagnosis

Erythema	Pustular eruptions	Blisters	Body folds	Ulceration
Infections	**Infections**	**Infections**	**Infections**	**Infections**
Bacterial	Folliculitis	Impetigo	*Candida*	Lupus vulgaris/tuberculosis
Fungal	Furunculosis	Herpes simplex/shingles	Dermatophyte	Syphilis
Viral	Herpes simplex	**Inflammatory disorders**	Erythrasma	**Vascular**
Physical	Tinea incognito	Bullous/cicatricial pemphigoid	**Others**	Vasculitis
Burns	*Candida*	Pemphigus vulgaris	Psoriasis	Venous disease
Erythema ab igne	**Inflammatory disorders**	Dermatitis herpetiformis	Seborrhoeic dermatitis	Arterial insufficiency
Inflammatory disorders	Rosacea	Erythema multiforme	Hidradenitis suppurativa	Rheumatoid arthritis
Eczema	Subcorneal pustular dermatosis	**Others**	Pemphigus vulgaris/vegetans	Pyoderma gangrenosum
Psoriasis	Pustular psoriasis	Oedema		**Neoplastic**
Dermatomyositis	Pyoderma gangrenosum	Insect bites		Basal cell carcinoma
Bullous pemphigoid	**Other**	Drug reactions		Squamous cell carcinoma
Erythema multiforme	Drug reactions	Eczema		Keratoacanthoma
Erythema nodosum		Porphyria cutanea tarda		Melanoma
Panniculitis		Epidermolysis bullosa acquisita		**Trauma**
Others		Toxic epidermal necrolysis		Neuropathic
Lymphoproliferative disorders		Diabetes mellitus		Pressure sore
Drug reactions				Pretibial injury
Urticaria				Dermatitis artefacta
Erythromelalgia				

pathogen, local resistance pattern and the patient's history of allergy and comedication.

The use of topical antibacterials is controversial and should be restricted to superficial infections of limited extent and short duration. Prolonged or frequent application can cause resistance and allergic contact dermatitis. Well-established examples of this are fusidic acid, which is often used on patients with infected eczema, and neomycin, a common ingredient in eye, nose and ear preparations. Both of these can cause allergic contact dermatitis, and the incidence is increased in elderly patients who have long-standing dermatoses or ulcers previously treated with many different topical agents including these antibacterials. Mupirocin is indicated for primary and secondary skin infections, and for the eradication of nasal colonisation of *S. aureus,* particularly methicillin-resistant strains (MRSA). Metronidazole is sometimes used topically on infected non-healing leg ulcers. It is strongly recommended to limit the application of topical antibacterials to a maximum of 2 weeks because of the risk of resistance and sensitisation.

Flucloxacillin and penicillin are commonly used systemic antibacterials. Amoxicillin-clavulanic acid covers most of the common pathogens in cellulitis. Patients with penicillin allergy can be given a first- or second-generation cephalosporin, but there is a 10% risk of cross-allergy. Other alternatives include macrolides and tetracyclines. Erythromycin and clarithromycin have been used extensively in the treatment of skin infections, and some countries have seen a significant increase of up to 30% in strains of *Streptococcus pyogenes* and *S. aureus* resistant to these antibacterials.[8] Clindamycin inhibits synthesis of bacterial proteins and should be given in addition to β-lactam antibacterials in severe streptococcal infections.[9] Metronidazole is a good choice for anaerobic infections, and ciprofloxacin is effective against *P. aeruginosa*, although resistant strains have been reported.[10] Invasive MRSA infection requires treatment with rifampicin (rifampin), fusidic acid, ciprofloxacin or vancomycin depending on the sensitivity profile.

1.4.2 Antiseptics

Antiseptics were designed to reduce bacterial load and prevent infection on intact skin. They do not appear to have a significant effect on the microbial load of chronic wounds such as venous leg ulcers.[11] Research evidence suggests that antiseptics are both toxic and beneficial in the treatment of wound infections. Laboratory and animal studies have demonstrated toxic effects on cells such as fibroblasts and keratinocytes, and interference with formation of new vasculature.[12] However, conditions in a human wound may be different, and experimental and clinical evidence suggests that antiseptics do not affect endpoint wound healing.[13,14] Some authors consider arguments about toxicity as irrelevant, as there is little or no fibroblast activity, keratinocyte production or angiogenesis in infected and inflamed wounds.

The most commonly used antiseptics are povidone-iodine, potassium permanganate and chlorhexidine. Skin paints such as carbol-fuchsin solution (magenta blue or Castellani's paint) also have bactericidal and astringent activities, but may be difficult to obtain because of the possible carcinogenic risk of its manufacturing. Some topical preparations can cause allergic reactions.

Over the last few years, special brands of honey have been reported to be useful in wound healing because of antimicrobial properties.[15]

1.4.3 Dressings

The correct dressing depends on the type of wound and on the stage of the healing process. Different dressings are required for different stages: cleansing and removal of slough, granulation and vascularisation, and epithelialisation. If necessary, wound debridement of necrotic tissue has to be carried out before an appropriate dressing can be applied. A moist wound environment appears to be associated with decreased wound infection rate. Alginate and foam dressings are highly absorbent and therefore suitable for exudating wounds. Hydrogel

dressings can be used for debridement, and hydrocolloids are occlusive dressings suitable for softening and promoting granulation. Controversy exists over the use of other desloughing agents such as sugar paste and Edinburgh University Solution of Lime (EUSOL) in paraffin.[16-18] Activated charcoal dressings are beneficial in absorbing the malodour of infected wounds. No product appears to have a particular advantage and the choice of an individual dressing depends on one's personal experience, patient's preference and availability of the dressing.[19]

1.4.4 Biotherapy (Maggot Therapy)

The larvae of the greenbottle fly (*Phaenicia sericata*) are used for debridement of sloughy, infected or non-healing wounds. They are specially bred so that they do not carry any bacteria.[20] Each treatment is left in place for 2 to 3 days. The maggots break down dead tissue via the secretion of enzymes and then ingest this material along with wound bacteria. They may also stimulate the production of granulation tissue.

2. Erythema

2.1 Erysipelas

Erysipelas is an acute infection of the dermis and upper subcutaneous tissue, caused in the majority of cases by β-haemolytic *Streptococcus pyogenes* and less commonly *S. aureus*. The face and legs are the most commonly affected sites, and a search for a portal of entry is important. The skin is bright red, oedematous and often very tender in a unilateral distribution. Characteristically, there is a well-demarcated advancing border and vesicles and bullae may be present in the advancing margin. A classic sign is the orange-peel (peau d'orange) appearance (figure 1). Malaise and fever are often accompanying symptoms. In the elderly, erysipelas can present abruptly as toxic confusional state. It may result in severe tissue necrosis, cavernous sinus thrombosis and can also recur, especially in patients with underlying lymphoedema.

The drug of choice is penicillin, and the response is usually quite dramatic. Alternatively, erythromycin or a cephalosporin can be given. In cases of recurrent erysipelas, prophylactic antibacterials need to be given to prevent the development or worsening of lymphoedema, although this is not always successful.

In patients with diabetes mellitus, immunocompromised patients or patients with pre-existing renal and hepatic disease, erysipelas may be caused by a mixed bacterial infection consisting of Gram-positive organisms as well as enterococci and enterobacteria. These patients usually require an intravenous cephalosporin.

2.2 Cellulitis

Cellulitis is a bacterial infection of the lower dermis and subcutaneous soft tissue. It is a com-

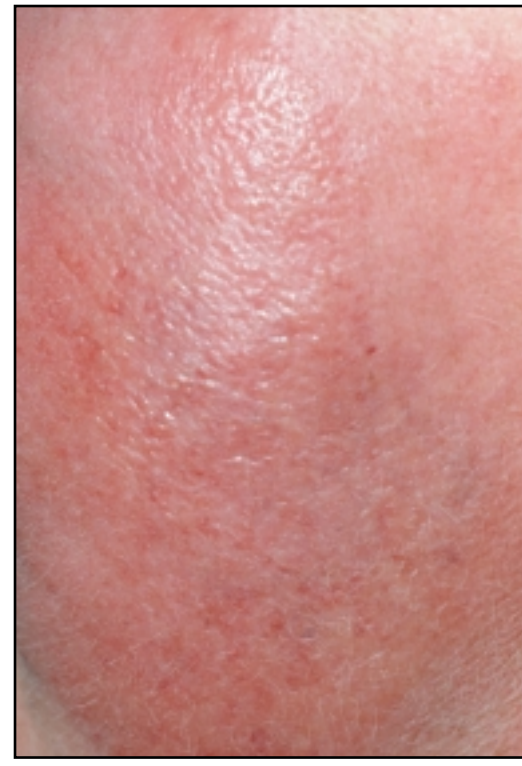

Fig. 1. Erysipelas.

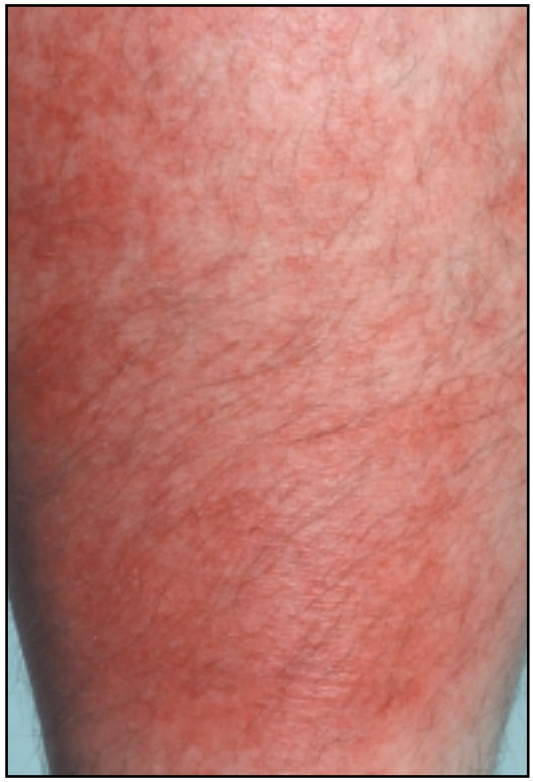

Fig. 2. Cellulitis of the lower leg

mon condition in the elderly and usually affects the legs (figure 2). The major causative organisms are β-haemolytic streptococci, especially groups G and A, and *S. aureus*. Both are highly virulent organisms. Streptococci produce potent proteolytic exotoxic enzymes causing rapid progression and extensive necrosis. Other organisms found are *Pseudomonas* spp., *Serratia* spp., *P. mirabilis, Escherichia coli* and *Klebsiella* spp.[1]

The organisms often gain entry through broken areas, such as cuts, abrasions, ulcers, fissures caused by dermatophytes, or animal bites (figure 3). Cat bites result more often than dog bites in wound infection, and *Pasteurella multocida* is most commonly isolated.[21] (Cat-scratch disease, which is caused by *Bartonella henselae*, starts with a red papule at the site of trauma and is associated with regional lymphadenopathy.)

Cellulitis presents with ill-defined erythema, pain and oedema. The pain is often worse on palpation and there may be blister formation and exudation. The draining lymph nodes can be palpable and tender. The skin signs can be accompanied by systemic symptoms including fever, malaise and confusion. It is not always possible to distinguish clinically between cellulitis and erysipelas.

If inspection of the wound reveals dark and mottled areas and a putrid malodorous exudate, infection with the gas bacillus *Clostridium perfringens/welchii* should be suspected, and fewer than half of such patients will have initially detectable gaseous crepitation.

Skin swabs should be taken to try to confirm the causative organism and test for antibacterial sensitivities. Needle aspiration, skin biopsy and blood cultures should be considered in severe or complicated cases.[7]

The main differential diagnosis is deep vein thrombosis. Doppler ultrasound or other imaging tests should be carried out if there is any doubt about the diagnosis. Other differential diagnoses to consider are allergic contact dermatitis and stasis dermatitis. Allergic contact dermatitis can present with a red, itchy, sometimes blistering, area where the allergen has been in contact with the skin, but unlike cellulitis is usually less tender and warm

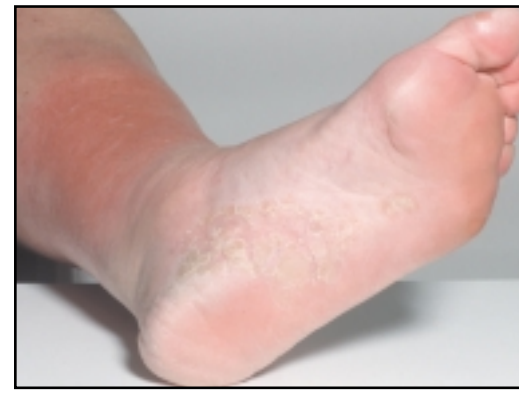

Fig. 3. Cellulitis. Fungal infection with fissuring acting as a portal of entry for organisms.

and there are no associated systemic symptoms. Stasis dermatitis typically involves the skin of the lower third of the lower legs, but can also spread on to the thighs, upper arms and trunk. Brown haemosiderin pigmentation is commonly found.

Initial management of cellulitis should cover both *Staphylococcus* and *Streptococcus* with flucloxacillin and penicillin or ampicillin for 10 days. Patients with penicillin allergy should be given erythromycin, a first-generation cephalosporin, or tetracycline. Depending on the severity of the cellulitis and accompanying symptoms, treatment can either be given orally or parenterally. Most patients can be managed as outpatients, especially when antibacterials are started quickly. A response to the treatment should be seen within 48 hours.

One retrospective study showed that streptococci were the most frequently found organisms, and that almost a third of patients had either recurrent episodes of cellulitis or other continuing morbidity.[22] Patients with chronic leg swelling or lymphoedema are especially prone to recurrent infections and risk worsening of their leg problems. Patients with two or more episodes of cellulitis should be treated with long-term or even life-long low-dose penicillin.[22] Other measures to prevent recurrences involve the treatment of fungal infections such as tinea pedis, support stockings or bandages, and general good skin care especially in the toe webs. In the case of recurrent infections providing a portal of entry between the toe webs, skin paints such as magenta blue or gentian violet have an antiseptic and astringent effect and can be applied between the toes.[23]

2.3 Necrotising Fasciitis

Necrotising fasciitis is an especially destructive infection causing rapidly advancing deep tissue necrosis. It may be caused by a number of organisms, either in isolation or more commonly as polymicrobial infection, including facultative bacteria and anaerobes such as *Klebsiella* spp. and *P. aeruginosa*. About 10% of cases are caused by streptococci, mostly group A, and were previously called streptococcal gangrene. Predisposing factors include prior injury, surgery, irradiation, cancer, diabetes mellitus, alcoholism, obesity and malnutrition. Although in the very early stages fever may be mild, the patient is usually septic and may have toxic shock-like syndrome. The skin is dusky red and there may be associated gaseous crepitation and putrid discharge. However, pain is often absent or minimal since cutaneous sensation is lost.

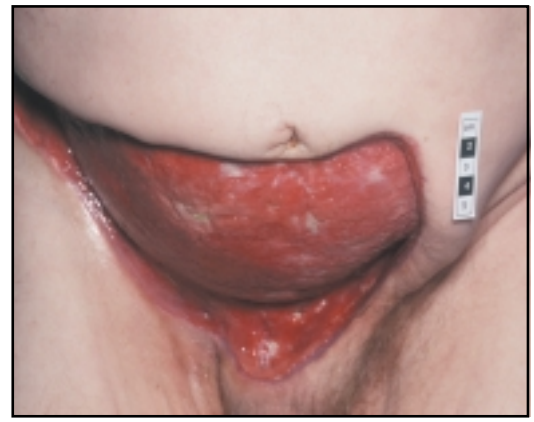

Fig. 4. Necrotising fasciitis.

Rapid diagnosis and treatment is essential in this potentially fatal condition. Fine-needle aspiration for Gram stain and frozen-section tissue biopsy can help the early diagnosis. Magnetic resonance imaging (MRI) has been shown to be very useful in defining the presence and extent of necrosis in patients with fasciitis.[24]

Intravenous broad-spectrum antibacterials to cover both aerobes and anaerobes should be given, but the most important measure is rapid and vigorous debridement of all necrotic tissue, which usually requires general anaesthesia (figure 4).[25,26] If streptococcal necrotising fasciitis is suspected, antibacterial therapy should include high-dose penicillin and clindamycin.[9] Hyperbaric oxygen therapy appears to be of benefit in some patients.

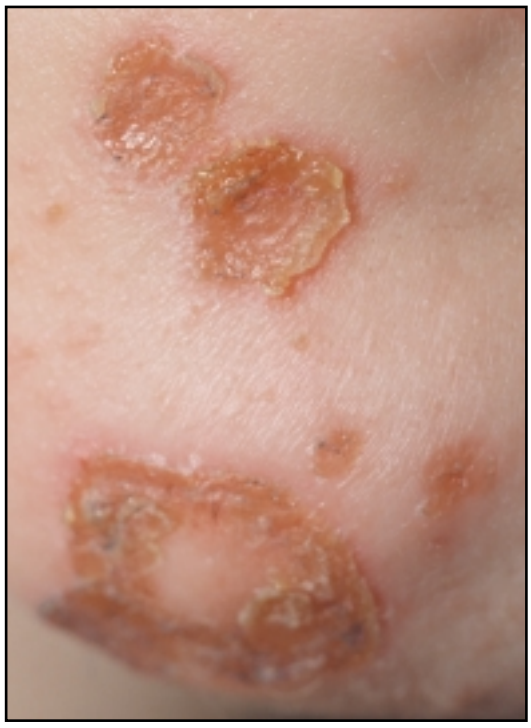

Fig. 5. Impetigo.

3. Blisters

3.1 Impetigo

This superficial cutaneous infection is caused by either *S. aureus* or β-haemolytic streptococci group A. There are two classic forms of impetigo: a non-bullous variant, which accounts for approximately 70% of cases, and a bullous variant. Patients develop pustules and blisters, which then form honey-coloured yellowish crusts (figure 5). Lymphadenopathy and leucocytosis are often associated findings. The areas most often affected are the face, especially around the nose and mouth, and wounds, even as small as a shaving nick or a scraped knee. Impetigo is very contagious and close contacts should be examined.

A small and localised area of impetigo can be treated with topical application of fusidic acid or mupirocin for 7 to 10 days.[27] A widespread infection requires oral flucloxacillin, erythromycin or a cephalosporin for 10 days. If there is no improvement with flucloxacillin, or if bullae are present, penicillin or ampicillin should be added for better coverage of a streptococcal infection. Crusts can be removed with warm saline or antibacterial cleansers. Bathing in antiseptics such as chlorhexidine will help to control skin flora.

4. Pustular Eruptions

4.1 Folliculitis

Folliculitis is a superficial infection of the pilosebaceous follicles, usually in areas with short coarse hair such as the neck, beard area, buttocks and thighs. It is most commonly caused by *S. aureus*. Crops of multiple small erythematous papules and pustules develop, characteristically

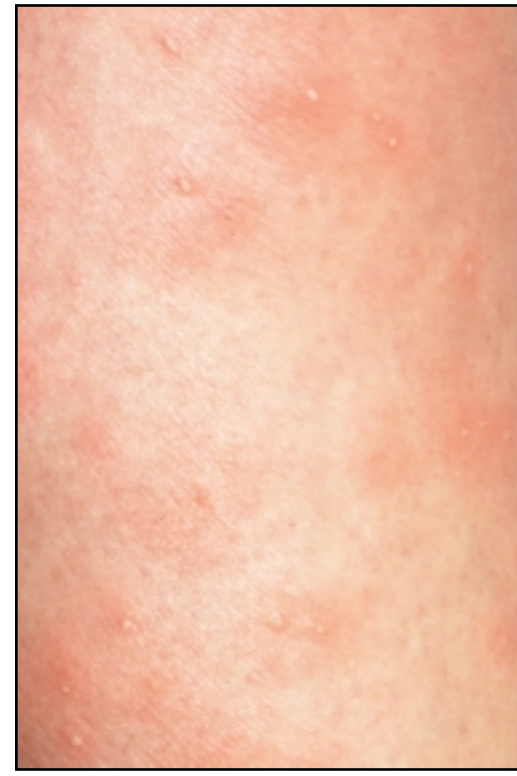

Fig. 6. Folliculitis.

surrounding a hair (figure 6). There may be some pain or itching.

Gram-negative organisms such as *Klebsiella* or *Proteus* can also cause folliculitis. This occurs usually in patients taking prolonged courses of antibacterials. 'Hot tub' or 'spa pool' folliculitis is due to *P. aeruginosa*. It occurs when the tub or spa is used by a large group of people and the water is poorly chlorinated. Swimming pools, contaminated natural or synthetic sponges or poorly cleaned bathtubs have also been identified as sources.

Folliculitis can also be a feature of infections with *Pityrosporum*, *Candida* or dermatophytes, as well as of irritant reactions to mineral oils, wax epilation or padding. Extensive folliculitis is sometimes seen as a complication of topical corticosteroid therapy or as a reaction to coal tar products. Recurrent infections should trigger a search for conditions such as diabetes mellitus.

Mild localised folliculitis may resolve without treatment; otherwise, a topical antibacterial such as fusidic acid, erythromycin, clindamycin or mupirocin is usually sufficient.[27] Oral antibacterials are necessary for more extensive and severe cases. Most commonly used are flucloxacillin, ampicillin-clavulanic acid or erythromycin, but antibacterial sensitivity should be confirmed with a skin swab.

Avoiding friction from tight-fitting and restrictive clothes against the skin helps prevent folliculitis. Shaving in the direction of hair growth rather than against it is advisable.

4.2 Furunculosis and Carbunculosis

Furuncles or boils develop when a deeper infection of the hair follicle occurs with *S. aureus*. They are usually tender and heal with scarring. Predisposing factors include bacterial carriage in the nostrils, scabies, excoriations, diabetes mellitus, obesity, malnutrition, and the administration of corticosteroids and immunosuppressive drugs.

Carbuncles are the result of neglected, manipulated or mistreated furuncles. The result is a lateral extension and destruction of the tissue and the formation of interconnecting abscesses (figure 7).

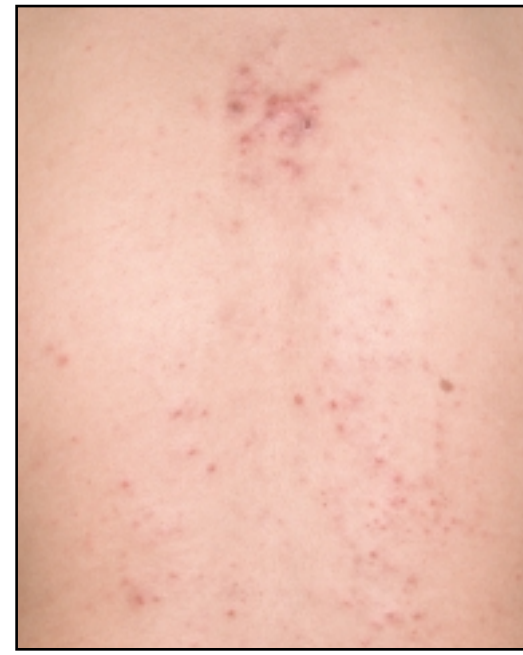

Fig. 7. Furuncles.

Complications include septicaemia, thrombophlebitis and cellulitis.

Oral flucloxacillin is the drug of choice for these infections. A protective dressing may be helpful to prevent further trauma to the area. Incision and drainage may be needed if the nodule is very large and tender.

5. Body Folds

5.1 Intertrigo

This condition affects the submammary, axillary, inguinal and perineal folds. It is most commonly a problem in obese people. Friction causes the skin creases to become macerated and sore, and subsequently a superinfection with yeasts or bacteria develops. In a dermatophyte infection the edge of the rash advances, leaving clearer skin behind. However, if topical corticosteroids have been applied this appearance may be altered, and the eruption becomes more diffuse with studded fol-

licular lesions (tinea incognito). Skin swabs and scrapings for mycology are recommended.

The affected area should be cleaned and protected from further maceration with either a greasy emollient or a nonadhesive dressing. Depending on the microbiological results, topical treatment is usually sufficient. It is often helpful to use a product containing a topical corticosteroid to reduce erythema and discomfort.

5.2 Erythrasma

This superficial cutaneous infection is caused by *Corynebacterium minutissimum*. The characteristic rash is of reddish-light brown, slightly scaly, appearance, typically in the groin and axillae. It spreads slowly with a well-demarcated advancing edge. It is generally asymptomatic, but in the tropics irritation can lead to scratching and lichenification. The diagnosis is usually confirmed by pink/coral-red fluorescence when examined under a Wood's lamp. Alternatively, cultivation of *Corynebacterium* from skin scrapings corroborates the diagnosis.

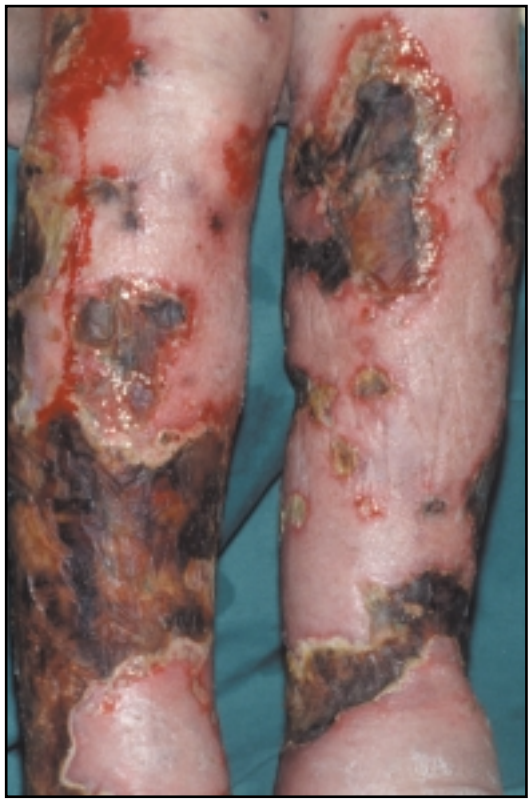

Fig. 8. Infected leg ulcers.

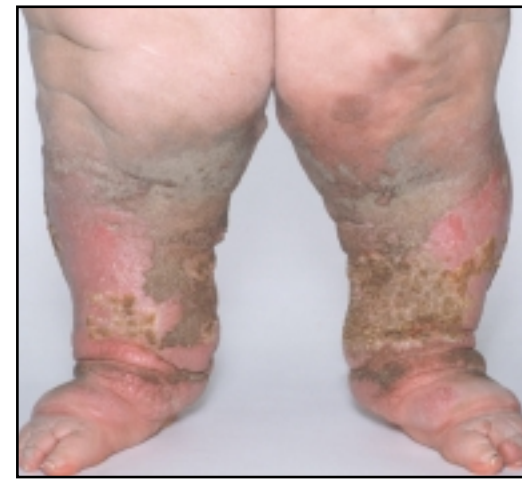

Fig. 9. Chronic lymphoedema/elephantiasis.

Topical imidazoles such as clotrimazole or miconazole, or topical erythromycin or clindamycin, for 2 to 3 weeks are usually sufficient. If necessary, a 14-day course of oral erythromycin can be prescribed.

6. Ulcerations

Venous ulcerations are usually painless. Pain can be a sign of associated arterial insufficiency, peripheral neuropathy, lipodermatosclerosis or the development of an infection. Ischaemic pain gets more pronounced when the leg is elevated, such as overnight.

Infected ulcers stop healing or break down further (figure 8). There is often slough, increased exudate and malodour. The area around the ulcer usually shows the typical signs of cellulitis, with erythema, swelling, increased warmth and tenderness, and there is usually pyrexia and malaise. A similar picture occurs in infected pressure sores.

Oral or parenteral antibacterials are required (section 2.2). Cellulitis associated with leg ulcers or pressure sores usually requires longer courses of treatment for 3 to 4 weeks.

Patients with chronic lymphoedema/elephantiasis have a high risk of developing leg ulcers and cellulitis (figure 9).

Routine administration of antibacterials in patients with ulcers (without infection or cellulitis) does not result in significant changes of bacterial flora or in increased healing rates of ulcers, and is therefore not recommended.[5,28] Patients should also be assessed for the need for analgesics, antiseptic and astringent soaks (for example potassium permanganate solution), change of dressings, biotherapy and bandages.

7. Secondary Infections

Excoriations or scratch marks can be caused by itching arising from a variety of dermatological problems, including eczema, psoriasis, autoimmune blistering diseases, lichen planus, infestations or drug eruptions. Sudden aggravation of atopic eczema, contact dermatitis and some cases of psoriasis may be caused by *S. aureus* and some streptococci producing toxins and proteins acting as superantigens. Pruritus can also be a symptom of systemic conditions such as diabetes mellitus, renal and hepatic impairment, haematological diseases, thyroid disorders and neoplasms.

Relevant investigations to establish the underlying diagnosis include blood tests, radiographs and skin biopsies for histology and immunofluorescence. However, in the elderly, tests for the abovementioned conditions are often unremarkable and itchy dry skin is attributed to aging and environmental factors.

Scratch marks often show signs of exudate and crusting in keeping with secondary infection, most commonly caused by *S. aureus*. A skin swab should be taken for confirmation and testing of sensitivities. Depending on the extent and severity of the infected excoriations, either topical or oral antibacterials can be used.

The frequent application of emollients and mild-to-moderate potency topical corticosteroids is beneficial. It is important to prevent further scratching, and patients may benefit from antihistamines. A sedating antihistamine is preferable, especially if the patient complains of interrupted sleep and/or subconscious scratching.

8. Conclusion

Skin and soft tissue infections in the elderly are frequently encountered both in hospitals and in the community. The most common causative organisms are *S. aureus* and β-haemolytic streptococci.

An accurate diagnosis should be made, taking into account the presentation and clinical picture as well as laboratory investigations, mainly skin swabs. Treatment should be commenced immediately and not delayed until laboratory results are available. Appropriate antibacterials, which are known to be effective against the relevant organism causing a particular skin infection and taking into consideration local resistance patterns, need to be prescribed.

Predisposing factors need to be identified and managed to prevent recurrences.

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