LOCAL WOUND INFECTION FACT SHEETS

A EWMA resource for clinical practice

The European Wound Management Association
EWMA

LOCAL WOUND INFECTION FACT SHEETS
INTRODUCTION

The EWMA Antimicrobial Stewardship (AMS) Programme aims to promote the responsible and effective use of antimicrobials in wound management. As part of this programme, **a series of local infection factsheets** have been developed for clinical practice to better identify early signs and symptoms of local wound infections.

The factsheets:

- provide key insights into aetiology, including common diagnostic clues and pitfalls;
- highlight “red flags” that indicate when escalation or specialist assessment is required.

The seven fact sheets cover the following topics:

1. Recognising infection in atypical wounds
2. Recognising infection in diabetic foot ulcers
3. Recognising infection pressure ulcers
4. Recognising infection in venous leg ulcers
5. Recognising infection in skin tears
6. Chronic limb-threatening ischaemia
7. Surgical site infections: signs, symptoms and management

The authors responsible for the fact sheets are listed in the top of the factsheets.

The EWMA Antimicrobial Stewardship Programme is supported by BBraun, DebX, Essity, Flen Health, Smith+Nephew, Solventum.

Antimicrobial stewardship in wound care is not about withholding treatment, it is about preventing infections and using the right treatment, at the right time, for the right reason. Clinicians play a central role in balancing infection control with responsible antimicrobial use. Applying AMS principles daily will help preserve antimicrobial effectiveness, improve healing outcomes, and reduce the global threat of AMR.

Read more about the EWMA AMS Programme and access additional resources supporting education and clinical decision making at https://ewma.org/antimicrobial-stewardship/.

FACTSHEET 1:

RECOGNISING INFECTION IN ATYPICAL WOUNDS

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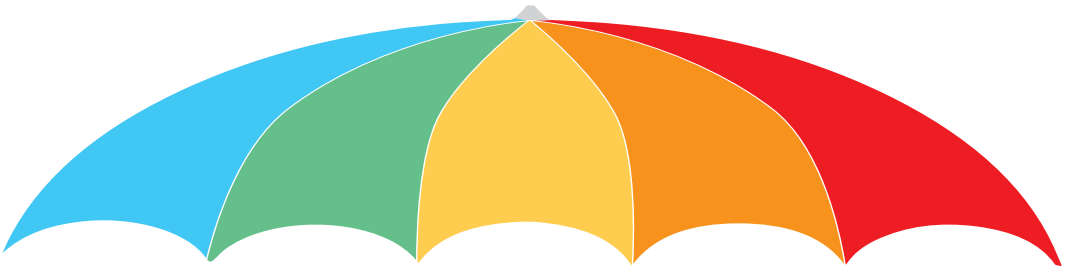
What are atypical wounds?

Atypical wounds is an umbrella term for a heterogenous group of wounds, i.e. inflammatory, vasculopathy, malignant, infectious, iatrogenic and self-inflicted. The diagnosis is typically delayed. Dermatologists and dermatopathologists are key people in diagnosing atypical wounds, but in the treatment an interprofessional team, including nurses , is needed. Due to the large variety of causes, atypical wounds as a wound entity are not so rare and may account up to 10% of chronic wounds treated at tertiary wound clinics. The diagnostic delay with atypical wounds can worsen significantly the prognosis of the patients, and therefore prompt diagnosis is essential.

Figure 1: Types of atypical wounds.



Inflammatory (Pyoderma gangrenosum, vasculitis, hidradenitis suppurativa)

Vasculopathy (Calciphylaxis, Martorell, livedoid vasculopathy)

Malignant (basal cell cancer, melanoma, squamous cell cancer

Infectious (ecthyma gangrenosum, leishmaniasis, tuberculosis)

Iatrogenic and psychogenic

RECOGNISING INFECTION IN ATYPICAL WOUNDS

Infection characteristics specific to atypical wounds

Exact diagnosis of the atypical wound and knowledge of their specific clinical features is essential to avoid over and underdiagnosis of infection.

Inflammatory wounds, such as Pyoderma gangrenosum (PG) and vasculitis, resemble clinically infectious wounds. Wound edges are violaceous, wounds are painful and especially PG ulcers contain a purulent discharge which does not contain bacteria but is neutrophilic (Figures 2 and 3).

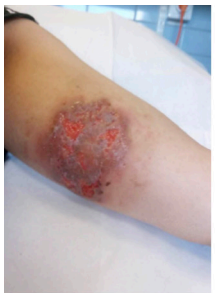


Figure 2

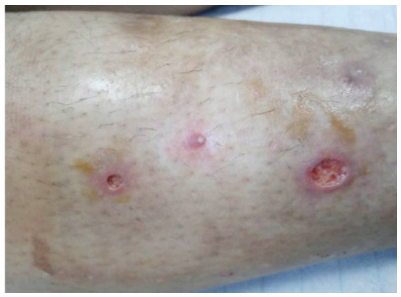


Figure 3

Many atypical wounds contain necrosis, which is superficial in PG and vasculitis, and deeper in Martorell ulcers and calciphylaxis.

Patients suffering from Martorell or calciphylaxis ulcers have an increased risk of wound infection and sepsis (Figures 4 and 5).

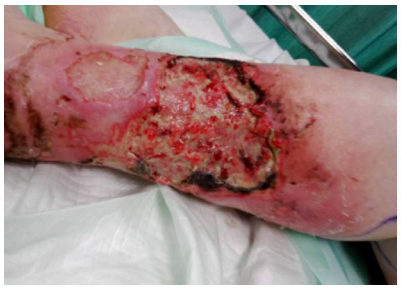


Figure 4

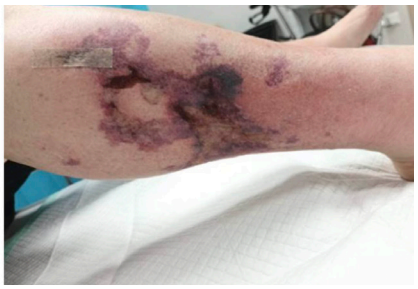


Figure 5

Key clinical indicators of infection

The diagnosis of clinical infection in atypical wounds is based on the same criteria as in other types of wounds, but based on the inflammatory features, diagnosis of infection might be challenging. Main infection indicators are erythema or discoloration expanding 0.5 - 2 centimetres from the wound edge, swelling, heat, loss of function, pain, odour and the presence of purulent discharge. There might be also systemic signs such as fever, chills and general fatigue. C-reactive protein, neutrophil count and leucocyte count might be elevated.

Atypical infections

There are several micro-organisms that can cause ulcers. The amount of these ulcers is likely to rise in Western countries due to travelling and immigration. Treatment of these infectious ulcers should be planned with an internist specialised in infectious diseases.

Cutaneous leishmaniasis presents as an atypical ulcer that can heal by itself over 3-18 months but can also lead to severe scarring. Up to 10% of cutaneous leishmaniasis can progress to a more severe manifestation/disseminated disease in immunocompromised patients (Figure 6).

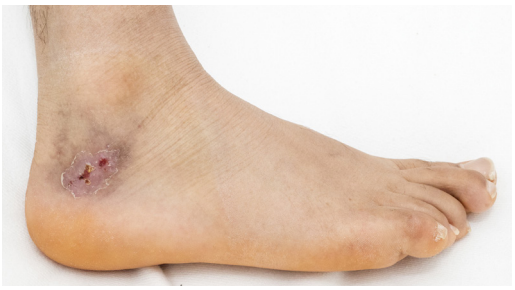


Figure 6

In **Acanthamoeba**, ulcerated nodules can be the first sign of a protozoal infection that can cause clinically evident disease after months or years following exposure.

Amebiasis is a protozoal infection that can cause clinically evident disease after months or years following exposure. Cutaneous ulcerations due to amebiasis typically occur in the perianal region.

Mycobacteria are also capable of causing a wide range of cutaneous manifestations, and **Buruli ulcer is caused by Mycobacterium ulcerans** and other related slowly growing mycobacteria. Buruli ulcer occurs mainly in the lower extremities and presents first as a painless nodule or a large, indurated plaque that evolves in a period of approximately four weeks into an ulcer with undermined borders.

Ecthyma and ecthyma gangrenosum

Ecthyma is an ulcerative infectious disease and may be caused by different bacteria, mainly group A beta-haemolytic streptococci, such as Streptococcus pyogenes, or Staphylococcus aureus, often following minor trauma like insect bites or scratches.

It presents painful ulcers with thick adherent necrotic scabs and an inflammatory erythematous halo.

Ecthyma gangrenosum mainly affects immunocompromised patients, and Pseudomonas aeruginosa is a common pathogen. When a clinical suspicion of ecthyma gangrenosum raises, Pseudomonas should be covered empirically in the antibiotic treatment. The clinical picture consists of rapidly evolving macules that form central necrosis (Figure 7-8).

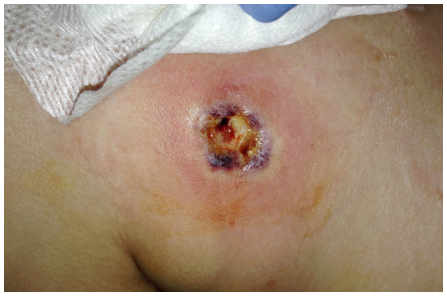


Figure 7

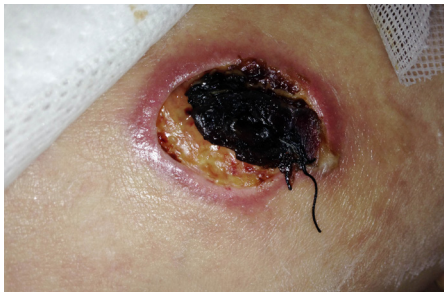


Figure 8

Clinical considerations

Some practical reminders for a clinician when considering an atypical wound with an infection or an infectious atypical wound:

- The primary **PG ulcer** contains neutrophilic pus, but biofilm development and superinfection are common as wounds progress.

RECOGNISING INFECTION IN ATYPICAL WOUNDS

- When treating **Martorell or calciphylaxis ulcers** the clinician must be aware of the higher risk of wound infection and sepsis.
- Many **inflammatory ulcers** resemble infectious wounds because of the inflammatory signs. It is wise to ask a second opinion before prescribing systemic antibiotics. Often, corticosteroid cream and antimicrobial dressings are a better choice.
- **Inflammatory ulcers** typically respond well to immunosuppressive therapy, such as systemic or topical corticosteroids, and not to antibiotics unless there is secondary infection. On the other hand, infectious ulcers improve with targeted antimicrobial treatment.
- Underlying autoimmune or systemic conditions are commonly associated with **inflammatory ulcers**, whereas infectious ulcers are more frequently seen in patients who are immunocompromised, have poor wound care, or have environmental exposures.
- When a patient has travel or immigration history, it is important to think about the atypical infectious diseases and consult infectious disease specialist when needed.

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FACTSHEET 2:

RECOGNISING INFECTION IN DIABETIC FOOT ULCERS

Author: José Luis Lázaro-Martínez, Full Time Professor, Diabetic Foot Unit, Complutense University of Madrid, Madrid, Spain.

What is a diabetic foot ulcer?

The International Working Group on the Diabetic Foot (IWGDF) defines a Diabetic Foot Ulcer (DFU) as an ulceration of the foot occurring in a person with current or previously diagnosed diabetes mellitus, typically associated with peripheral neuropathy and/or peripheral arterial disease in the lower extremities (1). While DFU prevalence varies across different regions of the world, it is widely accepted that the lifetime incidence among individuals with diabetes ranges from 19% to 34% globally (2).

One of the most serious complications of DFU is infection, which affects approximately 60% of cases. In around 50% of these, infection is accompanied by ischemia. Due to their typical location near bony prominences or beneath foot deformities, 30% to 40% of diabetic foot infections involve bone, leading to Diabetic Foot Osteomyelitis (DFO) (3).

Diabetic foot ulcer colonisation and diabetic foot infections

Bacterial colonisation of foot ulcers is a constant and expected phenomenon, characterised by the presence of microorganisms on the wound surface without evidence of tissue invasion.

In contrast, wound infection is a pathological condition caused by the invasion and proliferation of microorganisms within host tissues, triggering an inflammatory response that often results in tissue damage. Since all chronic wounds are colonised (frequently by potentially pathogenic microorganisms) the diagnosis of infection cannot rely solely on the results of wound cultures.

Risk of developing a diabetic foot infection

Several factors may predispose a diabetic foot ulcer (DFU) to become infected, including (4):

- a positive probe-to-bone test,
- ulcer duration longer than 30 days,
- history of previous ulceration or amputation,
- traumatic aetiology,
- presence of peripheral arterial disease,
- loss of protective sensation,
- poor metabolic control,
- diabetes-related immune dysfunction.

Diagnosis of diabetic foot ulcer infection

The diagnosis of diabetic foot infection (DFI) is based on the presence of local and/or systemic signs and symptoms of inflammation (3).

A diabetic foot ulcer is considered infected when at least two of the following criteria are present:

- local swelling or induration,
- erythema extending >0.5 cm but <2 cm around the wound
- local tenderness or pain
- increased local temperature
- purulent discharge.

RECOGNISING INFECTION IN DIABETIC FOOT ULCERS

Following the diagnosis of a diabetic foot infection (DFI), it is essential to classify its severity according to the IWGDF/IDSA (3) guidelines to guide antibiotic selection and determine the appropriate setting for management, including the need for hospitalisation or emergency surgical intervention.

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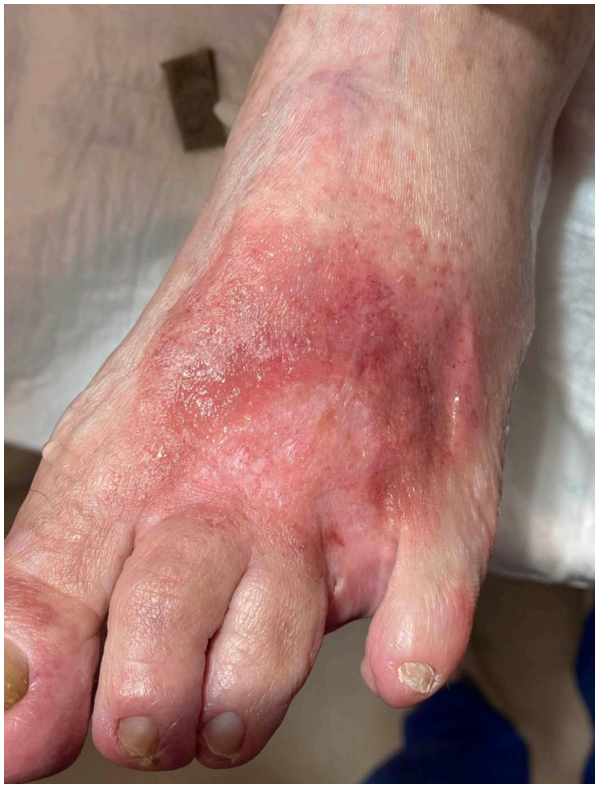

Figure 1. DFU showing moderate infection due to an interdigital ulcer

Diagnosis of diabetic foot osteomyelitis

Osteomyelitis can underlie any foot ulcer, particularly those of long duration or with specific high-risk characteristics such as being extensive, deep, situated over a bony prominence, showing visible bone, or associated with a red, swollen ‘sausage-like’ toe (5).

RECOGNISING INFECTION IN DIABETIC FOOT ULCERS

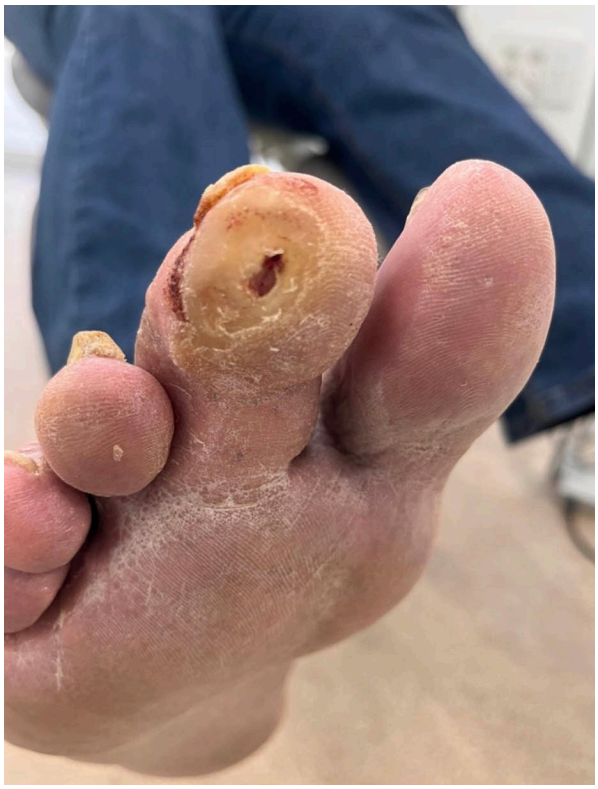

Figure 2. Sausage toe in a patient with DFO at the distal phalanx of the second toe.

When diabetic foot osteomyelitis (DFO) is suspected, diagnosis should be based on a combination of a positive probe-to-bone (PTB) test, plain radiographs, and inflammatory markers such as erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), or procalcitonin (3).

If the diagnosis of DFO remains uncertain despite clinical assessment, plain X-rays, and laboratory results, magnetic resonance imaging (MRI) is recommended to further evaluate the extent and presence of bone infection (3).

DFU at risk of infection - clinical presentation

There is limited evidence regarding the clinical features of DFUs at risk of infection. However, beyond the accumulation of previously described risk factors, it is widely accepted that a DFU which worsens or deteriorates despite appropriate standard of care should be considered at risk of infection.

A DFU may be considered at risk of infection when the following features are present:

- pale wound bed,
- hypergranulation or unhealthy granulation tissue,
- increased exudate and perilesional maceration,
- friable tissue,
- thickened or undermined wound edges,
- presence of tunnelling or fistulous tracts,
- exposure of bone, joint, or tendon.

RECOGNISING INFECTION IN DIABETIC FOOT ULCERS

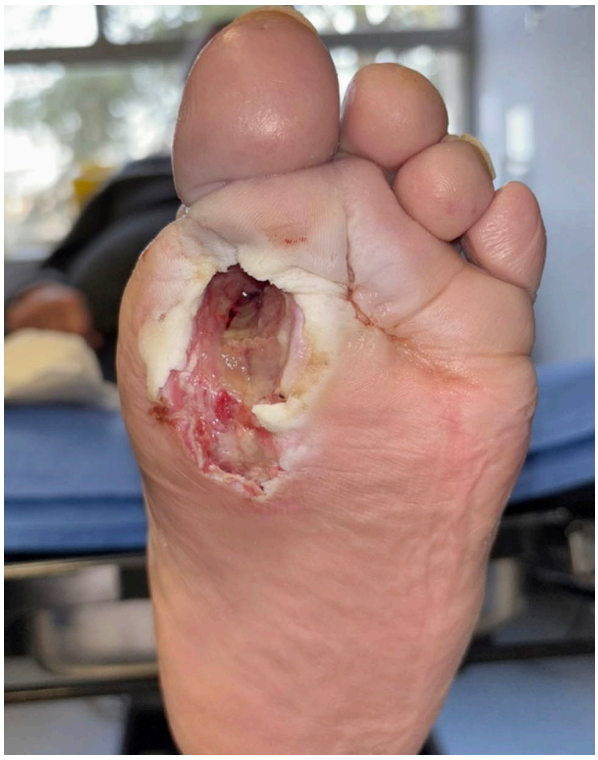

Figure 3. DFU at risk of infection showing perilesional maceration, pale wound bed with friable granulation tissue, thickened edges and fistulous track.

Close monitoring of these patients, including imaging studies and assessment of inflammatory markers, is highly recommended in such cases. In these cases, the implementation of anti-biofilm strategies and strict wound hygiene protocols is strongly recommended to prevent the development of diabetic foot infection (DFI).

DFU culturing

A sample for culture should be taken only if a diagnosis of clinically infected DFU is made. Routine or repeated cultures of colonised DFUs may lead to overdiagnosis of diabetic foot infection and contribute to the inappropriate use of antibiotics, potentially resulting in the emergence of multidrug-resistant organisms at the wound site (3).
When infection is suspected, it is recommended to obtain a sample for culture to identify the causative pathogens, ideally by aseptically collecting a tissue specimen (via curettage or biopsy) from the base of the wound. If bone involvement is suspected, the sample should include bone tissue, obtained either intraoperatively or percutaneously (3).

Conventional culture methods are preferred over molecular microbiological techniques for the initial identification of pathogens from soft tissue or bone samples in patients with DFU (3).

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FACTSHEET 3:

RECOGNISING INFECTION IN PRESSURE ULCERS

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About pressure ulcers

A pressure ulcer (PU) is an injury to the skin and underlying tissue that can be caused by persisted pressure or pressure in combination with shear.

An infected pressure ulcer (IPU) is a challenging situation, involving complications which jeopardise the successful outcome of the therapeutic process.

Clinical diagnosis of an IPU

Phase 1: from inflammation to infection

Inflammation is part of both an acute and chronic wound, although it is present in different ways. In both cases it expresses a reaction of the tissues, useful in trying to heal the lesion itself. When the delicate and often vague signs, which may be difficult to interpret or traitorous, veer from signs of inflammation to an infection, it is necessary to recognise them quickly to prevent a local infection from taking hold and spreading rapidly.

The clinical signs requiring more attention are:

- **Erythema (redness)** becomes diffusing, its colour is more intense and can rapidly turn purple;
- **Exudate** production increases, it may becomecloudier and it is necessary to dress the lesion more frequently (night dribbling);
- **Unpleasant/foul smelling** becomes evident, indicating a more intense bacterial metabolism because of malodorous chemicals (volatile fatty acids);
- **Fragility of neo granulating tissues** and propensity for spontaneous bleeding or just changing the dressing (soft-touch and bleeding from a vanishing tissue);
- **Moving from an uncomfortable feeling to a pain** sensation to an intense pain not only localised but distant from the PU site. This worsening chain of sensations indicates a negative evolution of the process from inflammation to infection and its spread.

Phase 2: the clinical diagnosis of local IPU is becoming obvious:

1. **Erythema (redness).** This is one of the first pieces of evidence, firstly discovered as a non-uniform, reddish-pink halo on the surface of the skin, most often a bone-prominence, particularly exposed to pressure/friction mechanisms. IPU in dark skin tones may present with subtle or different visual cues compared to lighter skin tones, making early detection crucial for effective treatment. Instead of the typical redness (erythema), areas of discoloration, such as darkening, purple or shiny, may be the first sign.
2. **Hypertermia.** This is an elevated skin temperature associated with increased metabolic activity in the underlying deep tissues.
3. As the infection appears the **Swelling** becomes clearly visible, often accompanied by an increasing uniform redness, oedema and pain. The colour tends to veer towards violet-purple the more the skin takes on a dark tone, considering the various shades (1).
4. **Pain** is indicating an IPU, and its appearance is not only perceived on the dome of the IPU but also in the immediate periwound, indicating the extension of the infection to the surrounding tissues and into the deepness. During the evolution of the infectious process, the central area of the IPU shows hardness, stiffness and often discoloration,

5. Now the tissues could show a fracture with a widespread loss of thickness, in a diffuse lumpy and malodorous situation. **Bad smelling** is an important criterion for defining an infected pressure ulcer. Severe pain or pain that's getting worse indicates a well-structured infection.

6. **Limited impaired function** is permeated by the full-thickness IPU, with the extension of the infectious process to the surrounding tissues, beyond the immediate (or proxi-periwound) area and therefore at a distance from the main lesion.

7. **Stagnation** in the wound healing process is evident (stalled or recalcitrant IPU, or Hard-to-Heal IPU are the most frequently coined) and a biofilm is shown in 80% of chronic IPU. Serological signs of systemic infection, e.g. leukocytosis and fever are associated with this situation. In these cases, a microbiological examination is indicated.

8. Depending on the pressure ulcer regenerating phase, discoloration of **granulation tissue**, friable granulation tissue, surface usually bleeding and pocketing at the base of the wound are part of the clinical signs well evident when there's an infection (2).

The Periwound in IPU: How does it behave if an ulcer is infected?

If colonisation proceeds uncontrolled and pressure ulcer infection occurs, the periwound is constantly at risk of participating and contributing to infection, especially due to changes in microclimate, maceration and changes in local pH.

The infection causes damage not only to the wound bed but also to the edges and the immediately surrounding skin. The periwound becomes red, oedematous, easily breakable and fragile, macerates and must be carefully irrigated, cleansed and subjected to local disinfection like the rest of the pressure ulcer(2,3).

PU complications

Although non-infectious complications of pressure ulcers occur, systemic infections are the most prevalent. Non-infectious complications include amyloidosis, heterotopic bone formation, perineal-urethral fistula, pseudoaneurysm, Marjolin ulcer, and systemic complications of topical treatment.

Infectious complications include bacteraemia and sepsis, cellulitis, endocarditis, meningitis, osteomyelitis, septic arthritis, and sinus tracts or abscesses. Osteomyelitis has been reported in 17 to 32 percent of infected ulcers and may lead to nonhealing ulcers with or without systemic manifestations. Plain radiographs and bone scans are often unreliable. Magnetic resonance imaging has a 98 percent sensitivity and 89 percent specificity for osteomyelitis in patients with pressure ulcers; however, needle biopsy of the bone is recommended and can guide antibiotic therapy.

Complications of an IPU

Complications of Infected Pressure Ulcers (IPU)

Assessment of an IPU should include ulcer history, location, size (length, width, depth), and the condition of surrounding skin. Check for redness, warmth, swelling, hardness, pain, or drainage—these may indicate infection. The wound bed should be inspected for necrotic tissue (slough or eschar), granulation tissue, or persistent fibrinous coating.

RECOGNISING INFECTION IN PRESSURE ULCERS

Pressure ulcers are often **critically colonised** with mixed bacteria forming biofilms - structured communities of microorganisms encased in a protective matrix. Biofilms are present in up to 90% of chronic wounds and are a major cause of delayed healing and recurrent infection (4).

Signs suggesting infection or complications include:

- increased pain, redness, warmth, swelling, or exudate.
- change in wound odour or colour,
- delayed healing or deterioration of the wound,
- systemic symptoms (fever, tachycardia, confusion).

Common infectious complications

Cellulitis:
Acute infection of skin and subcutaneous tissue around the ulcer. Presents with spreading redness, warmth, swelling, tenderness, and sometimes fever or confusion. If untreated, it can spread to bone (osteomyelitis), joints (septic arthritis), or bloodstream (sepsis) (5,6) (Table 1).

Necrotising fasciitis:
A rare but severe infection of deeper tissue causing intense pain, redness, swelling, and sometimes gas under the skin (crepitus). Requires **immediate surgical treatment**—do not delay in conducting imaging studies.

Osteomyelitis:
Deep infection of bone beneath stage 4 pressure ulcers. Suspect when healing is poor, bone is visible or palpable, or systemic symptoms persist. Diagnosis relies on clinical signs, imaging, and culture of bone biopsy samples after debridement (7, 8, 9).

Preliminary signs and symptoms indicative for an Osteomyelitis:

- fever,
- malaise, tiredness, fatigue, lethargy,
- irritability or restlessness in paediatric patients,
- resolute refusal to move the affected area (e.g. Limb): in children, a limp or reluctance to bear weight on the affected leg,
- few soft tissues residual (us or 3dus imaging acquired with a sterile probe),
- intense redness of the skin immediately above the bone,
- local acute pain, well localised and persistent,
- sudden persistent swelling,
- rapid warm-up in pressure in wound bed and periwound (feeling of warmth is reported from pts),
- possible pus drainage and/or appearance of draining sinus or tunnel or fistula.

Stage III:

- huge necrosis,non-healing ulcer (stalled, recalcitrant),
- visible swelling around the bone and surrounding soft tissues.

Stage IV:

- visible and palpable bone at the ulcer base,
- local and systemic signs for infection.

Septic arthritis in IPU, or an infected joint, can involve any joint but typically involves the knee joint. Patients present with joint swelling, warmth, pain, and decreased mobility of the joint. Septic arthritis treatment is by joint aspiration and antibiotics directed at the most common pathogens.

Bacteraemia

Bacteraemia is defined as the presence of viable bacteria in the blood, documented by a positive blood culture (10). PUs can be reservoirs for resistant microorganisms and may evolve into local infections, also becoming a source of bacteraemia in hospitalised patients. When the signs of an IPU are clear, the bacteraemia may intervene. In hospitalised patients, the relationship between bacteraemia and IPU has been associated with a 50% mortality rate (11).

Signs and symptoms indicative for Bacteraemia:

- one or more IPUs,
- one or more positive blood cultures,
- ulcer positive culture/s,
- no other sources of bloodstream infections,
- fever,
- chills,
- elevated heart rate or hypotension,
- cognitive impairment and confusion,
- gastrointestinal issues (abdominal pain, nausea, vomiting, diarrhoea).

Please note that: The above listed infectious complications may become life-threatening.

Infection control: The two most important points

1) Pressure injuries should be continually assessed for signs of bacterial infection such as increased erythema, foul odour, warmth, drainage, fever, and elevated white blood cell count.

2) Impaired wound healing should also raise concern of infection. These abnormal findings indicate a wound culture should be done. However, because all pressure ulcers are colonised, results should be interpreted with caution. Bacterial count, pathogenicity and virulence rather than bacterial presence should guide treatment. As a further indication, the antibiogram will define the type of antibiotic molecules to be used: the modern types of antibiograms are the MIC (Minimum Inhibitory Concentration), which measures the lowest concentration of antibiotic that inhibits bacterial growth, and the MBC (Minimum Bactericidal Concentration), the concentration that kills the bacterium.

RECOGNISING INFECTION IN PRESSURE ULCERS

Table 1.
Cellulitis in IPU: the evolution of local signs

1. Clear to straw-coloured thin and watery exudates as well as purulent exudates are increasing;
2. Worsening erythema and spreading erythematous inflammation into deep dermal tissues;
3. Swelling and oedema are involving the subcutaneous tissue;
4. If gas is produced by anaerobic bacteria, crackling and crepitus (popping sound) are present;
5. IPU is enlarging, becoming warmer, friable granulations begin to bleed, pain is getting worse;
6. The IPU is non-healing and a foul smell is part of the signs affecting cellulitic tissues.

Figure 1

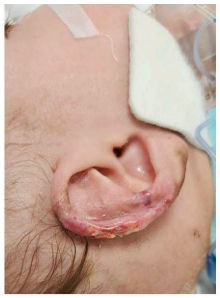

Figure 2

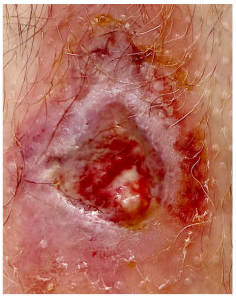

Figure 3

Fig. 1 Grade II pressure ulcer, right buttock. Perilesional erythema and superficial base with thin layer of fibrin. No exudation, no pain. Critical contamination is microbiologically assessed.

Fig. 2 Grade II pressure ulcer from forced decubitus on the right lateral side, involving the helix and antihelix in a 25-day-old neonate. Non-viable tissues are evident (light brown), small foci of epidermal necrosis and slight cloudy exudation.

Fig. 3 Grade II infected pressure ulcer, right ischiatic location. Proxy periwound erythema, circumferential maceration (white halo), bleeding from a friable granulating central tissue.

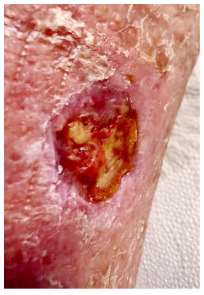

Figure 4

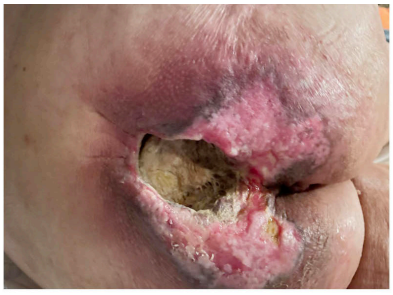

Figure 5

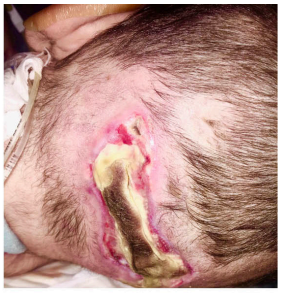

Figure 6

Fig. 4 Grade III infected pressure ulcer, with eroded muscular fascia, purulent exudate, slough (deep yellow), intense redness at the proxy and distant periwound and scaling (scaly skin) of the skin's outer layer which clearly appears as dry skin.

Fig. 5 Grade IV infected pressure ulcer, with bone-exposure, intense necrosis in the wound-bed, swelling borders, periwound intense purple erythema, purulent exudate, osteomyelitis assessed with RMI.

Fig. 6 Unstageable pressure ulcer lesion, occipital, 6 months old boy. Infection, dark and yellow eschar, very hard and different from the slough (lighter in colour).

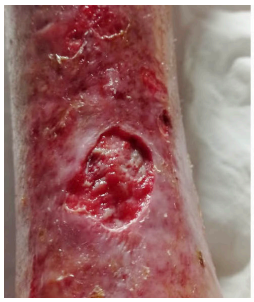

Figure 7

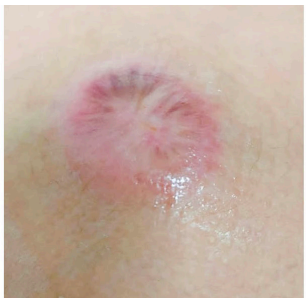

Figure 8a

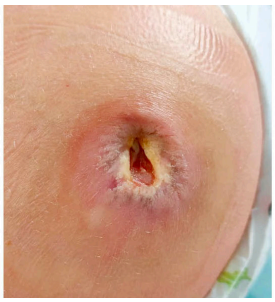

Figure 8b

Fig. 7 Cellulitis because of a third-degree pressure ulcer. The soft tissue infection surrounds the entire limb like a sleeve, demonstrating how quickly an infectious process can spread to the subcutaneous tissue. The skin colour is violet and, in some areas, purple. The infected pressure ulcer is located centrally.

Fig. 8a and 8b Trochanteric covered pressure ulcer of grade II, with perilesional erythema. Central fluctuation and suspected abscess. B: spontaneous opening of the abscess collection, evacuation to the outside, through fistula and positive MRI for osteomyelitis.

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FACTSHEET 4:

RECOGNISING INFECTION IN VENOUS LEG ULCERS

Author: Sebastian Probst, Professor of Tissue Viability and Wound Care, HES-SO University of Applied Sciences and Arts Western Switzerland, School of Health Sciences, Geneva, Switzerland.

What are venous leg ulcers?

Venous leg ulcers (VLUs) are the most common form of chronic leg ulcer, accounting for 60–80% of cases (1). Their global prevalence is estimated at 0.32%, with an incidence of 0.17%, and recurrence rates can reach up to 80% within months of healing (1). Due to their chronicity and exposure, VLUs are frequently colonised by bacteria; however, true infection is less common. Distinguishing colonisation from infection is important, as overuse of antibiotics contributes to antimicrobial resistance, while under-recognition of infection increases risks of delayed healing, cellulitis, or sepsis (2). Accurate identification and timely management of infection are therefore essential to improve outcomes and reduce complications in individuals with VLUs (3).

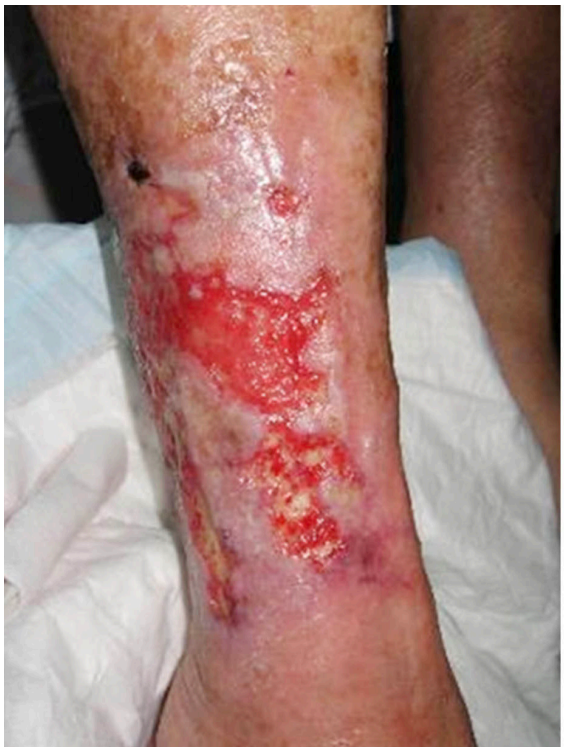

Figure 1: VLU with slightly macerated surrounding peri-wound skin.

Infection characteristics specific to VLU

Understanding how VLUs differ from other wounds is essential. VLUs do not typically present with necrosis. The presence of necrosis suggests an arterial or mixed aetiology and warrants vascular assessment (4). Infection signs may present subtly due to the chronic inflammatory state of VLUs.

RECOGNISING INFECTION IN VENOUS LEG ULCERS

Key clinical indicators of infection

Infection in VLUs often present subtly and can be overlooked. Clinicians should assess for the following key indicators during dressing changes or follow-ups (4):

- Increased exudate: a notable rise in wound drainage, especially if purulent or thick;
- Change in exudate character: transition from clear to coloured, malodorous fluid;
- Pain: sudden onset or escalation in pain is a red flag, especially in chronic, previously painless ulcers;
- Friable or discoloured granulation tissue: bleeds easily, may appear dull or dark;
- Delayed healing: ulcer stalls or deteriorates after prior improvement;
- Malodour: foul smell that persists after cleaning.

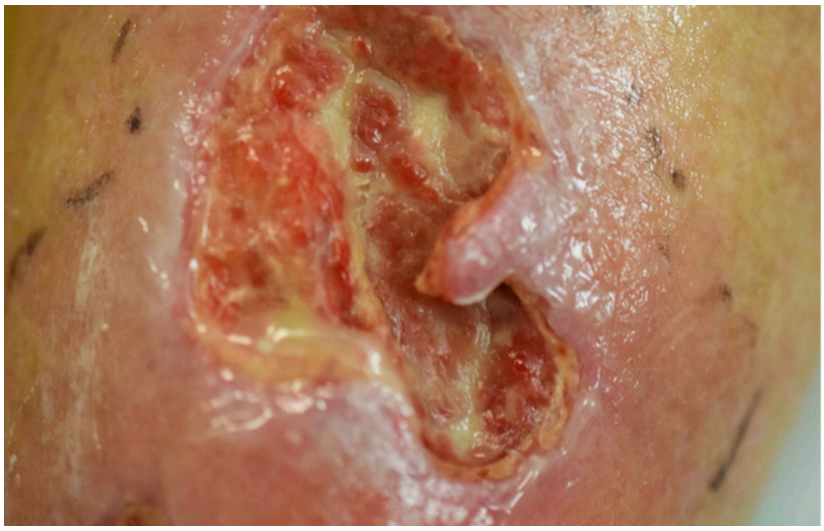

Figure 2: VLU in lighter skin tone (see redness around the wound).

Periwound erythema and skin tone considerations

Recognising erythema (redness) in various skin tones is vital. Clinical signs of infection like warmth or redness may be less visible in patients with melanin rich skin (5). Palpation becomes a more reliable diagnostic aid.

Lighter skin tones: Erythema may appear bright pink or red and is often more noticeable.
Melanin rich skin: Erythema may appear purplish, greyish, or violaceous discoloration. Feel for increased warmth or swelling as visual cues may be less apparent.

In all skin tones, use the back of the hand to detect temperature changes and compare them bilaterally when possible.

RECOGNISING INFECTION IN VENOUS LEG ULCERS

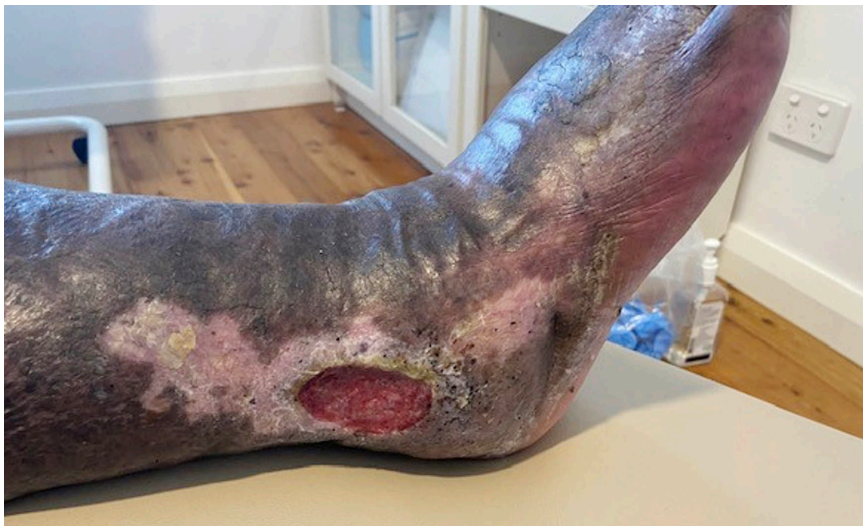

Figure 3: VLU in melanin rich skin with signs of infection (see grey color).

Atypical signs requiring attention

Some systemic or evolving signs of infection may not be immediately attributed to the ulcer but are important early clues. Recognising these 'silent' indicators helps prevent delays in care (4).

These indicators include:

- low-grade fever or systemic malaise,
- lymphangitis or swollen regional nodes,
- new localized oedema,
- increased periwound warmth,
- slough or fibrin returning after previous debridement.

Role of swabbing and testing

Swabs should not be routine. They are diagnostic tools to be used only when clinical signs of infection are evident. Proper swabbing techniques improve diagnostic accuracy and antimicrobial stewardship (6):

- Swab only when infection is clinically suspected;
- Use Levine technique: cleanse wound, then rotate swab with pressure over viable tissue;
- Avoid sampling from pus or slough alone.

Systemic or spreading infection – when to act

Sometimes, a VLU infection progresses beyond the wound, resulting in systemic symptoms. Recognising this escalation is key to timely intervention and potential hospital referral (4). The systemic symptoms to look out for include:

- spreading erythema or cellulitis,
- fever, elevated leukocyte or C-reactive protein (CRP),
- visible red lines along lymphatic vessels (lymphangitis),
- rapid wound deterioration with systemic symptoms.

RECOGNISING INFECTION IN VENOUS LEG ULCERS

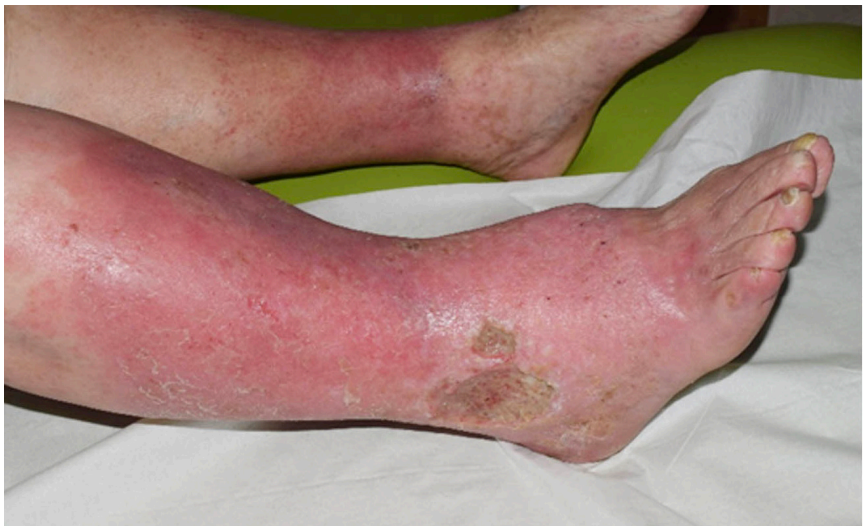

Figure 4: VLU with PAD component with signs of inflammation.

Clinical considerations

Here are practical reminders to help clinicians detect infection in VLUs, especially when traditional signs may be muted due to skin tone, age, or comorbidities.

- VLUs infected signs may differ by skin tone; assess visually and by palpation.
- Trust evolving clinical changes (pain, exudate, odour) over static lab results.
- Do not ignore pain or delayed healing in the absence of redness.
- Refer if worsening despite topical management or if systemic signs develop.

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FACTSHEET 5:

RECOGNISING INFECTION IN SKIN TEARS

Author: Karen Ousey, Clinical Manager at Omniamed (Wounds division), Emeritus Professor of Skin Integrity, University of Huddersfield & Adjunct Professor of Wound Management, Monash University, Melbourne, Australia.

What is a skin tear?

The International Skin Tear Advisory Panel (ISTAP) (1) define a skin tear as "a traumatic wound caused by mechanical forces, including removal of adhesives and patient handling, the depth of which may vary (not extending through the subcutaneous layer)". An uncomplicated skin tear should achieve full epithelisation or heal within approximately 4 weeks, if not healed within four weeks it may become a chronic wound and be classified as complicated. Skin tears on lower limbs or in individuals with multiple comorbidities, such as peripheral arterial disease or diabetes, are generally classified as a complicated skin tear with older patients being at risk of developing a wound infection (2).

Non infected skin tear

Below is an image of a non-infected skin tear, taken from an 82-year-old man who had fallen on a carpeted floor. He crawled across the carpet attempting to raise the alarm of having a fall, causing shear and friction to the skin resulting in a skin tear. He was apyrexial, had minimal exudate and no evidence of wound infection. This case is illustrated in images 1-2. Examples of skin tears on darker shin tones can be found in images 3-5.

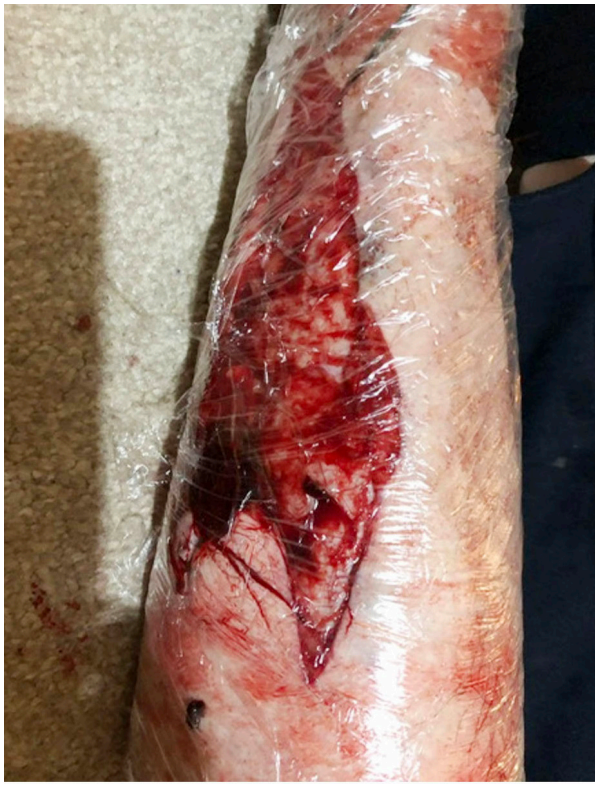

Figure 1: Immediate care from paramedics

RECOGNISING INFECTION IN SKIN TEARS

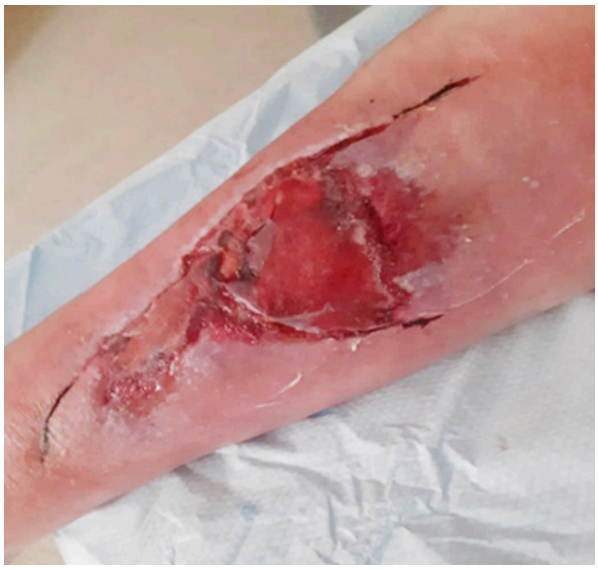

Figure 2: Day 6 - following cleansing and approximation of skin tear

Prevention

Prevention of wound infection is key and should be focused on implementing strategies to reduce individual patients risk factors. These should include a history of the cause of the wound, identification of co-morbidities, assessment of nutritional status, factors that influence the inflammatory and immune response, local tissue healing and psychosocial assessment (3).

Is the skin tear infected?

A wound infection occurs when microorganisms invade a wound and multiply to an extent that triggers a local, spreading, or systemic response in the host. These microorganisms reproduce within the wound, producing various virulence factors to bypass the host's defences, causing local tissue damage and impeding the healing process (3, 4). The International Wound Infection Institute (IWII) (3) identify clinical signs of wound infection See: IWII-CD-2022-web.pdf. If not identified in a timely manner infection can spread and become systemic.

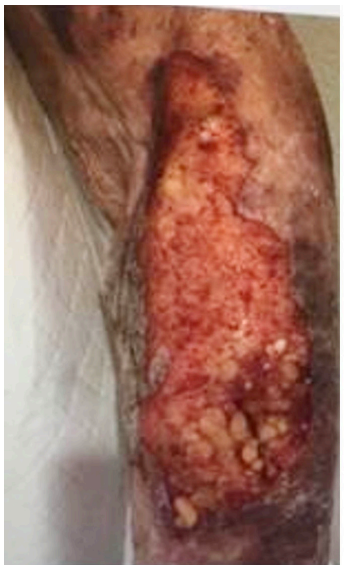

Figure 3: Full thickness skin tear (Darker skin tone)

RECOGNISING INFECTION IN SKIN TEARS

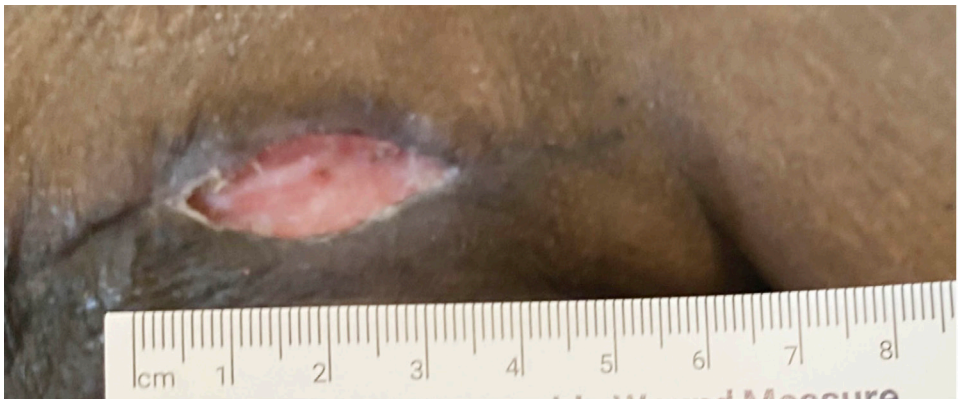

Figure 4: Skin tear (Darker skin tone)

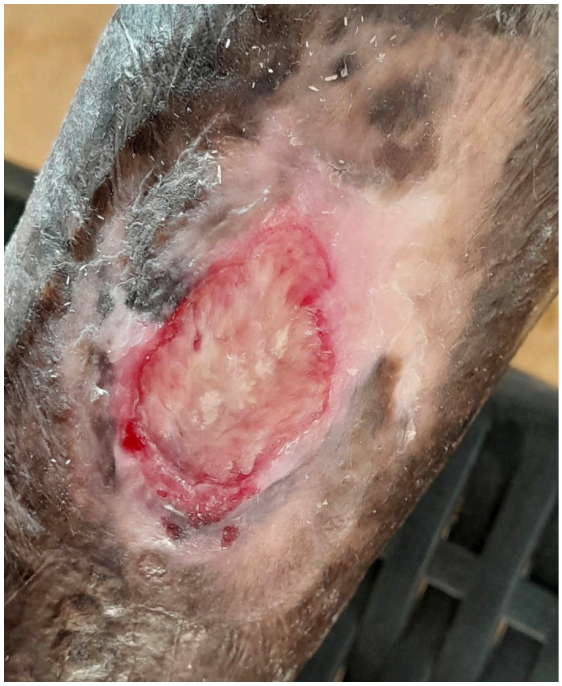

Figure 5: Skin tear (Darker skin tone)

Holistic assessment

Identifying a skin tear in a timely manner is essential to prevent an infection. Many skin tears are often under reported and misdiagnosed. Attention should be paid to skin tone with assessment of baseline skin tone being critical (5). This is especially important when identifying early signs of infection as erythema does not appear as redness in many dark skin tones (6). Consideration should be given to those people diagnosed with dermatoporosis, defined as a chronic syndrome of excessive skin insufficiency/fragility (7) where complications include skin lacerations/skin tears and delayed wound healing. On the lower limb deep dissecting haematomas can be a complication and may be misdiagnosed as cellulitis (8). They should not be confused with infection due to the absence of clinical signs of infection.

RECOGNISING INFECTION IN SKIN TEARS

Treatment to prevent infection

To reduce the risk of infection and further injury, while considering any comorbidities, clinicians should attempt to preserve the skin flap and maintain surrounding tissue by reapproximating the flap (without stretching the skin) (9). People with skin tears often have fragile and frail skin therefore non-adherent dressings should be used to avoid compromising the skin flap (1, 10). When accessible, silicone dressings are recommended, as evidence suggests they enhance skin tear healing more effectively than non-silicone, non-adherent alternatives (11, 12). Dry gauze or any adherent dressing should be avoided. Documentation of the skin tear should include cause of the tear, measurement of the skin tear including size, condition of the peri wound area, any signs of erythema, exudate, pyrexia, local wound pain, delayed healing – if any of these signs are present be aware of potential wound infection. Antimicrobial wound dressings can be applied and antibiotics prescribed if a wound infection is diagnosed. It is important that choice of antimicrobials is guided by the principles of antimicrobial stewardship to ensure appropriate use, minimise resistance, and preserve the effectiveness of treatments.

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FACTSHEET 6:

CHRONIC LIMB-THREATENING ISCHAEMIA (CLTI)

Author: Leanne Atkin, PhD, MHSc, RGN, Research Fellow/Vascular Nurse Consultant, School of Human and Health Sciences, University of Huddersfield and Mid Yorkshire NHS Teaching Trust.

What is arterial ulceration?

Lower limb arterial ulceration is a consequence of reduced blood supply to the area. This incident can be as a result of acute thrombosis (embolic disease) but is more likely to be related to atherosclerosis. The build-up of cholesterol-laden fatty plaque inside the artery causes narrowing of the lumen of the artery, therefore resulting in reduced blood flow to the limbs, most commonly the legs. Common risk factors for vascular disease are genetics, modifiable factors (e.g., smoking, high cholesterol, high blood glucose, high blood pressure), trauma, or infections.

Atherosclerosis is a systematic disease that is also the causative factor in coronary and cerebrovascular disease. Patients with peripheral arterial disease (PAD have a higher risk of having a stroke or a myocardial infarction, with a 2–3 times higher risk of cardiovascular mortality compared with the control population (1). Optimising cardiovascular risk factors is therefore paramount for patients with PAD to reduce cardiovascular morbidity and mortality. Many patients with PAD are asymptomatic; however, others progress to having symptoms such as intermittent claudication (exercise-induced muscle pain), and around 1% progress to 'end stage' PAD when patients experience arterial rest pain and tissue loss – this is termed CLTI (Chronic Limb Threatening Ischaemia).

Chronic Limb-Threatening Ischaemia and key indicators of infection

Chronic Limb Threatening Ischaemia (CLTI) is the advanced stage of PAD. It occurs due to the presence of severe PAD, whereby the blood supply to the foot is insufficient for the needs of the tissues. This results in a combination of rest pain, gangrene or lower limb ulceration which is present for a duration of 2 or more weeks and associated with one or more haemodynamic abnormalities. According to the Society for Vascular Surgery (2) CLTI is defined as "a clinical syndrome defined by the presence of peripheral artery disease (PAD) in combination with rest pain, gangrene, or a lower limb ulceration of more than 2 weeks' duration". Rest pain is discomfort in the forefoot, which typically occurs at night when the foot is elevated in the bed. Hanging the affected limb off the bed relieves the pain, which often wakes the patient from their sleep. This is because gravity helps blood flow return to the ischaemic leg.

Initially, the tissue loss presents as discoloured (purple/red/black) areas, often without a break in the epithelial surface (Figure 2). However, without timely revascularisation, deterioration is often seen (Figure 3). In both these cases there is evidence of colour change which is often mistaken for infection but is in fact due to reactive hyperaemia. A helpful way to assess whether the colour change to the surrounding tissue is related to infection or as a result of end-stage PAD is the use of the 'Buergers test' – looking for limb pallor on elevation of limb to above hip height (due to arterial pressure not being sufficient to overcome the effects of gravity), compared to the colour in the patient's limb when their limb is in a dependent position; due to vasodilation, a critically ischaemic limb will slowly turn pink and soon after red (so-called ischaemic rubor or sunset foot).

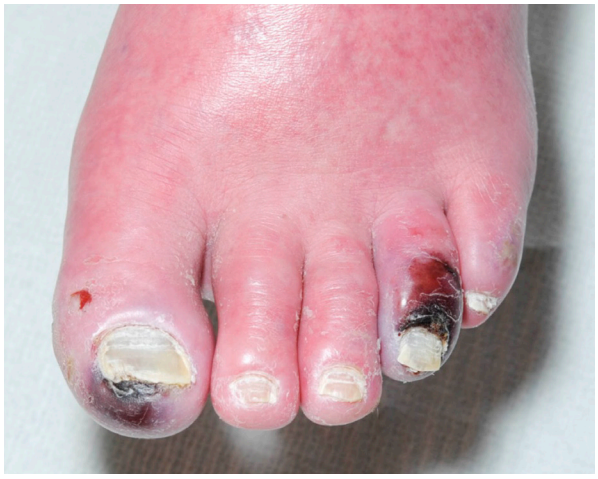

Figure 1: WIFI score: Wound 1: Ischaemia 2: Infection :0 – WIFI score = 3.

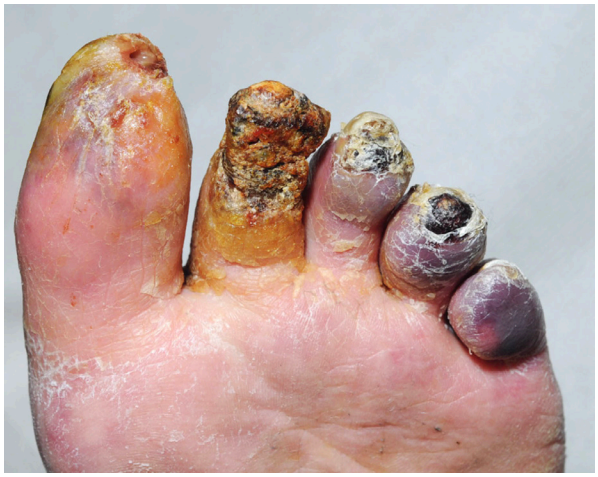

Figure 2: WIFI Wounds 1: Ischaemia 3: Infection 0 – WIFI = 4.

Severity assessment

The Wound Ischaemia, foot Infection (WIfI) score is a valuable method of assessing patients' severity and requirement for revascularisation. The WIFI score is based on three key factors: wound, ischaemia, and foot infection (Figure 3). The composite WIFI score, which is found by adding the wound, ischaemia, and infection sub-scores, helps predict the chances of amputation, saving the limb, and healing the wound, and can point out patients who might gain from revascularisation (Table 1).

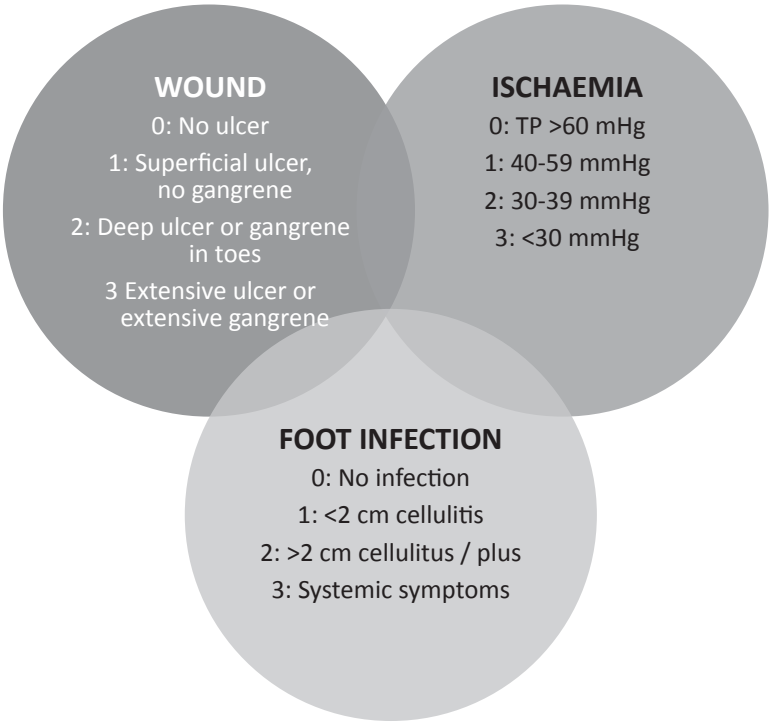

Figure 3: The WIfi score; based on three key factors: wound, ischaemia, and foot infection.

Table 1: Wound, Ischaemia, foot Infectionclinical stage associated with amputation risk and revascularisation benefit.

Stage	Major amputation risk at 1 year (estimated %)	Revascularisation benefit score
1	2-3	Very low
2	8-9	Low
3	25	Moderate
4	50	High

This table shows the clinical stages calculated from the WIfi system and how the stages are associated with amputation risk and revascularisation benefit.

Treatment of CLI

Optimising the treatment plan for patients with CLI is complex and multifaceted and is always taken on an individual patient basis; all patients will be considered for limb salvage and revascularisation if WIfI scores indicates. However, in some cases the patients may be better served with primary amputation or palliation depending on the severity of tissue loss, the estimated survival time of the patient and the complexity/ability to perform revascularisation (2).

Wound Management

If the areas of ulceration are dry, then the management is focused on atraumatic dressing renewal and protection from infection; debridement is only considered when adequate revascularisation has been achieved. However, it's important to stay alert, as these areas often get secondary infections (Figure 4), which means we may need to change treatment regime and consider using topical antimicrobials and possibly systemic antibiotics.

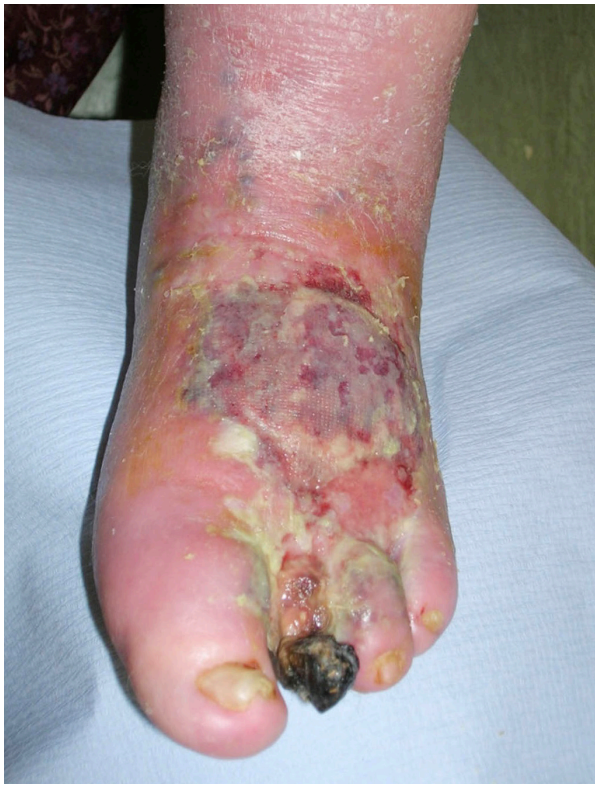

Figure 4: Secondary infection

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FACTSHEET 7:

SURGICAL SITE INFECTIONS - SIGNS, SYMPTOMS AND MANAGEMENT

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What are surgical site infections (SSIs)?

Annually more than 300 million surgeries are performed globally. In 2020, the document published by the European Wound Management Association (1) defines surgical site infection as an infectious process that develops on a surgical incision. It can occur up to 30 days after surgery (or 90 days if there is an implant) and can affect the superficial skin layers (superficial SSI), the fascial layers (deep SSI), or nearby organs and other sites, such as joints, abdomen, or thoracic space, which are manipulated during the procedure (organ/space SSI). These infections have also been associated with an increased risk of mortality by about 11%, as well as the length of stay (on average 10 plus days) and the associated direct cost, which can amount to several thousand, with the associated minimum average cost being around €20,000 (2). SSI is the main cause of morbidity after surgery, with an associated mortality rate of about 4% (3).

Signs and symptoms of infection

Detecting signs and symptoms of infection early is vital to prevent the development of a wide-spread infection and even sepsis. Early intervention for an infected wound is necessary, but it should not be neglected that the diagnosis of infection is purely clinical. It is important to point out that the wound may exhibit signs and symptoms that are compatible with the development of an infection, to initiate complementary diagnostic tests that can validate the diagnosis (1). It is important to realise that SSI is a surgical site complication (SSC) that must be diagnosed and treated as early as possible.

SSI has the potential to cause damage to the superficial skin, the fascial layers, or nearby organs and sites such as joints or the abdomen that are manipulated during the procedure, known as organ/space SSI (1) (Figure 1, 2 and 3).

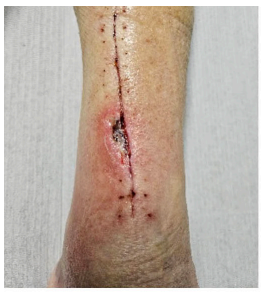

Figure 1

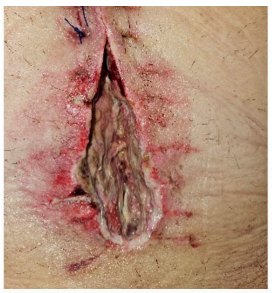

Figure 2

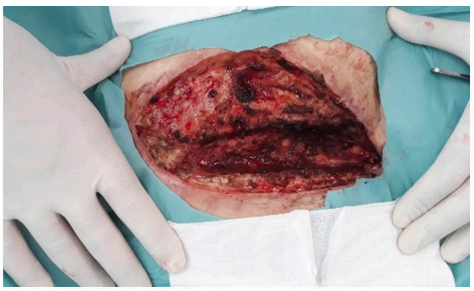

Figure 3

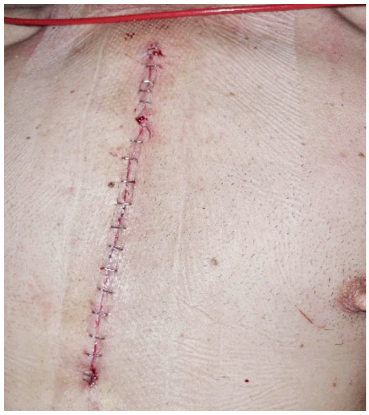

Figure 4

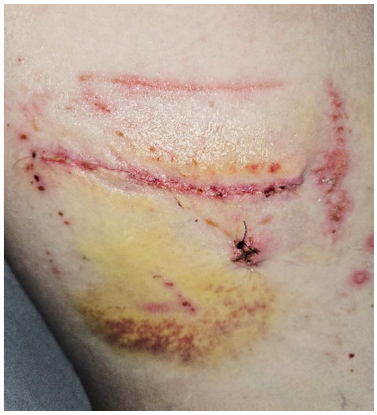

Figure 5

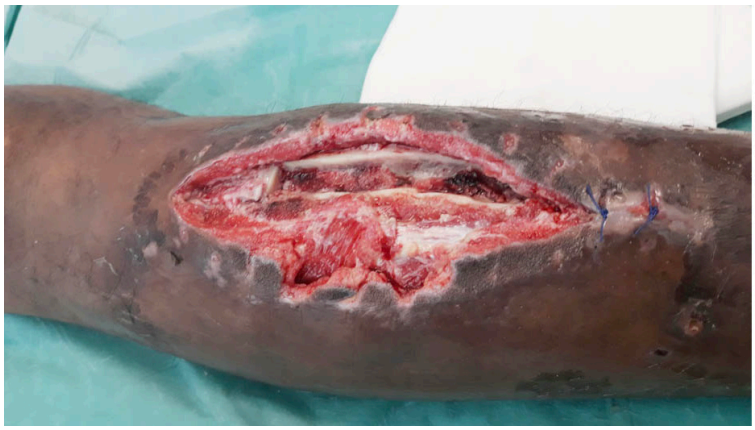

Figure 6

Fig. 4 Surgical wound with no signs of inflammation/infection.

Fig. 5 Surgical wound with inflammatory signs.

Fig. 6 Surgical wound with local infection.

Purulent drainage, pain or tenderness, localised swelling, redness, or heat are clinical signs and symptoms of SSI, which usually appear by the end of the first week after surgery. The presence of local infection signs can help identify superficial infection. SSI can also appear as cellulitis of soft tissue at the site of surgery or wound abscess. Deep and organ/space SSIs may have fewer local signs of infection, which may present themselves as purulent drainage from the wound, unexplained fever, pathological patient blood test results, or unusual systemic inflammatory responses of the body (1).

Prevention of SSI

Surgical site infections are a significant healthcare issue that can potentially be prevented in some instances. They have a significant impact on patient morbidity, mortality, and quality of life. To decrease the risk of developing SSI after surgery, it is essential to follow evidence-based guidelines and recommendations to prevent SSI. Clinical and surgical practices that aim to minimize the amount of microorganisms introduced into the surgical site prevent their multiplication, improve the patient's ability to fight infection, and prevent microorganisms from entering the incision postoperatively. The prevention of surgical site infection is fundamental in the process of controlling readmissions with surgical site cause, in the control of direct and indirect costs associated with health care, as well as in the quality of life of the patient and family(1, 4).

The prevention of complications, that often lead to an infection of the surgical site, begins at the same time as when it is decided to proceed with surgery. Identify co-morbidities, modifiable and non-modifiable factors, develop an intervention plan and choose the most appropriate treatment according to the identified risk (5). The patient related factors are age; body mass index (BMI) (≥40Kg/m2 or ≤18Kg/m2); diabetes mellitus; chronic kidney disease; chronic obstructive pulmonary disease (COPD); peripheral vascular disease; congestive heart failure; smoking; alcoholism; nutritional status; immunosuppression; corticosteroids; chemotherapy; ASA ≥ II (3, 6, 7, 8, 9, 10). After the identification of risk factors, it is necessary to understand which ones can be modified or not in order to reduce the likelihood of developing complications after surgery.

Clinical considerations

Prevention:

- Identify the modifiable and non-modifiable factors in the patient;
- Evaluate the patient in a holistic way, involving social, economic and family factors;
- Assess the patient's risk of developing infection in the surgical site;
- Use of appropriate dressing for the associated risk.

Management:

- Identify early signs and symptoms of infection (Redness and discoloration if seeing in dark skin tones, heat, oedema, drainage);
- Confirm possible development of infection with blood tests (C-reactive protein, procalcitonin, leukocyte count);
- Controle hemodynamic instability (tachycardia and low blood pressure);
- Microbiological identification;
- Early intervention, in order to control the development of the bacterial load and prevent a systemic infection;
- Multidisciplinary evaluation.

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