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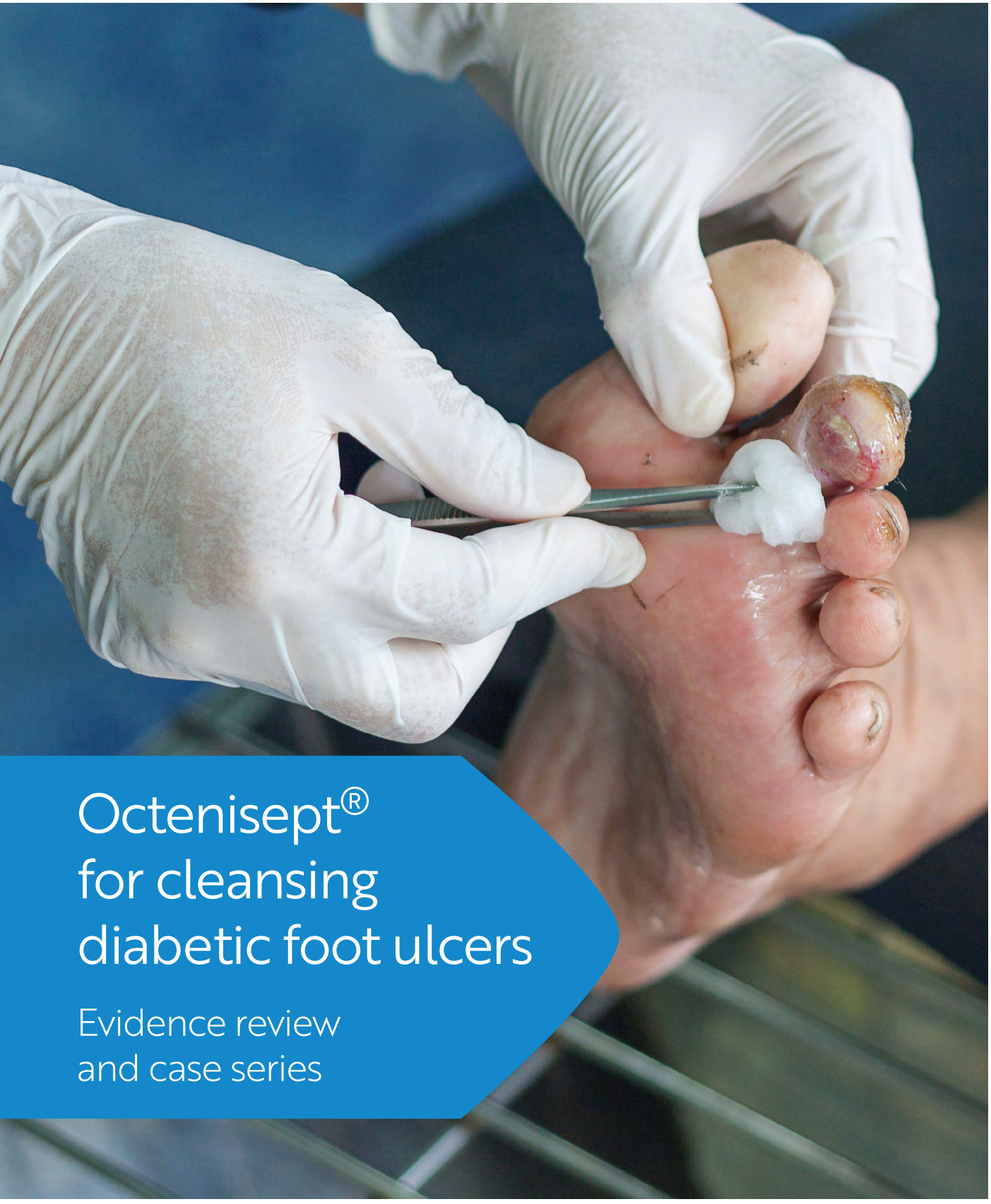

Octenisept® for cleansing diabetic foot ulcers

Evidence review and case series

Contents

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Foreword

Zhiwen Joseph Lo

'Dilution is the solution to pollution'. This was a surgical principle taught by my mentor, Professor Low Cheng Hock, while training us to drain a surgical abscess. He emphasised that the washout process was often more important than the actual solution used. Although some senior colleagues have advocated for a triple-washout regimen based on hydrogen peroxide, chlorhexidine and saline, evidence increasingly demonstrates the need to balance antimicrobial efficacy with tolerability and cytotoxicity. This balance is critical in wound care and particularly in the management of diabetic foot ulcers (DFUs), where impaired healing and a high risk of infection present constant challenges.

This supplement to the *Journal of Wound Care* explores the evolving role of antiseptic solutions in wound management, with a particular focus on octenidine-based preparations. Octenidine-based antiseptic solutions have emerged as valuable antiseptic agents, with broad-spectrum antimicrobial activity, low cytotoxicity and a favourable safety profile, making them important adjuncts in the management of hard-to-heal wounds.

The supplement includes a comprehensive review article that synthesises the current evidence on the role of antiseptic solutions in the care of DFUs. This is followed by 10 case studies from five different centres. These cases provide practical, real-world examples of octenidine's role in managing complex wounds, particularly in patients with DFUs, post-amputation wounds, necrotising fasciitis and arterial leg ulcers. These cases, contributed by multidisciplinary teams across various healthcare settings, illustrate how octenidine-based antiseptic solutions are being integrated into modern wound-care protocols. The case outcomes demonstrate not only octenidine's antimicrobial benefits, but also its contribution to improved wound-bed preparation, reduced infection burden and progression toward healing.

As the global burden of hard-to-heal wounds continues to rise, especially in people with diabetes, innovations in wound cleansing and antisepsis are more crucial than ever. This supplement aims to provide clinicians with both evidence-based guidance and practical insights into the use of antiseptic solutions, reinforcing the principle that effective wound hygiene remains the cornerstone of wound healing.

I hope this supplement serves as both a valuable resource and an inspiration for clinicians to continue striving for excellence in wound care.

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Role of therapeutic treatment with antiseptic solutions in the care of diabetic foot ulcers

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Background: Diabetic foot ulcers (DFUs) are a prevalent and severe complication of diabetes, leading to significant morbidity, impaired health-related quality of life (HRQoL) and economic burden on healthcare systems. The complexity of DFUs often results in prolonged healing and high recurrence rates. Effective management strategies are crucial for improving outcomes and reducing complications.
Aim: This study aimed to review the efficacy of antiseptic solutions in the treatment and care of DFUs.
Method: A literature analysis was conducted to review clinical studies and guidelines on the use and efficacy of antiseptic solutions, particularly Octenisept® (0.1% octenidine dihydrochloride and 2% 2-phenoxyethanol). The review focused on the antimicrobial properties, biofilm-disruption capabilities and wound healing outcomes associated with the use of antiseptic solutions in DFU management.
Results: Antiseptic solutions have potential to reduce bioburden, disrupt biofilm and modulate healing. There is a need to balance antimicrobial clinical efficacy with tolerability and cytotoxicity. The use and choice of adjunctive antiseptic solutions must be tailored to the patient, as antimicrobial efficacy can vary for antiseptic solutions, particularly for hypochlorous solutions. It is important to use products according to their instructions, with consideration of minimum contact time to maximise clinical efficacy. Low-pressure irrigation is adjunctive, and concurrent wound-bed preparation, including debridement, frequent inspection, infection and moisture control, remain important.
Conclusions: The therapeutic application of antiseptic solutions in DFU care presents a promising approach to enhancing wound healing and reducing infection risks. Integrating these solutions into standard wound care protocols could lower the incidence of complications, improve HRQoL and decrease the economic burden associated with diabetic foot disease. Further large-scale studies are recommended to validate these findings and refine guidelines for antiseptic use in DFU management.
Keywords: Antiseptic solutions, biofilm, diabetic foot ulcers, octenidine, Octenisept®, wound healing

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Diabetes is a serious threat to global health. Diabetic foot ulcers (DFUs) remain one of the most common complications of diabetes, affecting around 20 million people annually.[1,2] DFUs are classified into three types: neuropathic, ischaemic and neuro-ischaemic, based on the presence or absence of peripheral neuropathy, peripheral artery disease (PAD) or both (neuro-ischaemic).[3] DFUs are complex to treat, taking months or years to heal, with high recurrence rates of up to 40% within 1 year of healing.[4] Infected DFUs are associated with prolonged healing processes, leading to morbidity and lower health-related quality of life (HRQoL).[5,6] Daily wound care, prolonged hospitalisation and repeated surgical debridement also place a socio-economic burden on hospitals.[11]

Epidemiology and health economics

Over 50% of DFUs become infected, even in resource-rich settings.[6–9] Up to 20% of patients with moderate-to-severe infections eventually require minor (i.e., toes or part of the foot or distal to the ankle) or major (i.e., above the ankle)

lower-extremity amputations (LEAs).[10] Among Singaporean DFU patients, 36.4% underwent toe LEAs, 16.9% transmetatarsal LEAs and 6.5% major LEAs.[11] Approximately 20% of patients with a DFU require hospitalisation,[4,9] because the existence of a DFU and their associated infections constitute a significant risk factor for emergency department visits and hospital admissions among patients with diabetes, according to US data.[8] Likewise, in the UK, diabetic foot problems are the most frequent cause of diabetes-related hospital admissions.[1] Globally, an estimated 1.6 million LEAs are attributed to DFUs annually,[4] with diabetes-related LEA incidences ranging from 78–704 per 100 000 person years.[12] However, owing to disparities in healthcare access worldwide and across socioeconomic strata, there is a resultant inequity in diabetes-related lower extremity complications (DRLECs) across geography, communities and ethnicities.[1,13]

In addition to the increased use of healthcare services and mortality risks for patients with DFUs, a significant decrease is observed in their physical function and HRQoL,[14] with almost 50% of patients reporting symptoms of depression.[15] In the Eurodiale study, involving 1232 patients presenting with a new DFU, patients reported poor overall HRQoL, with problems primarily in the mobility and pain/discomfort domains.[16] Patients with an active DFU reported poorer HRQoL than those who underwent successful minor LEA. However, there is a paucity of high-quality data on HRQoL outcomes for diabetes-related LEA.[17]

Consequently, diabetic foot disease carries a heavy economic burden. In the US, $176 billion USD is spent annually on direct costs for diabetes care, with approximately one-third of this expenditure related to DRLECs.[18] The direct annual healthcare cost of DFU treatment in the US from 2007 to 2010 was roughly $9–13 billion USD.[19] In the UK, the estimated economic burden of diabetic foot disease amounted to $1.4–1.6 billion USD, almost 1% of the health service budget.[20] This rising economic burden is also noted in numerous other health economies worldwide, with substantial indirect societal costs, such as the loss of individual earnings, burden to carers and the effects of absenteeism on employers.[21,22] In Singapore, the incidence of DFUs is significant, at 170 per 100 000 people, with an incidence of LEA in DFU that exceeds all Organisation for Economic Co-operation and Development (OECD) countries, with the exception of Israel and Mexico.[23] The cost burden of hard-to-heal wounds is estimated at 3.14% of Singapore's gross domestic product, of which the value of lost health in terms of quality of life years (QALYs) is approximately 47% of the total cost of DFUs. DFUs have the highest QALY burden of all hard-to-heal wounds and are therefore the most researched wound type.[24] With the rise in DFU incidence in an aging population, there is a pressing need to address its prevention and provide cost-effective interventions.[23] Globally, this situation is worsened by the reported DFU recurrence rates of 40% within 1 year, 60% within 3 years and 65% within 5 years after healing.[2] Therefore, it may be more helpful to consider a state of remission rather than healing for patients with DFU wound closure. Similarly to cancer, the concept of remission offers a more effective framework for allocating resources, organising care and communicating information about risks.[25]

Diabetic foot infection

Diabetic foot infection (DFI) is a pathological state caused by the invasion and multiplication of microorganisms in host tissues that induce an inflammatory response, usually followed by tissue damage.[7] Immunological deficiencies related to diabetes further contribute to the prevalence of infection in DFUs.[26,27] While most DFIs are relatively superficial at presentation, microorganisms can spread contiguously to subcutaneous tissues, including fascia, tendons, muscles, joints and bones. The anatomy of the foot, which is divided into several separate but intercommunicating compartments, fosters the proximal spread of infection.[28] Bacterial virulence factors may also play a role in these complex infections.[29,30] Among the large panel of virulence factors, bacterial proteases (serine-, cysteine- and metallo-proteases), produced by a range of pathogenic bacteria, play a major role in the pathogenesis of wound healing.[31]

Risk factors for DFI (*Table 1*) include PAD, peripheral neuropathy and impaired immune function.[32] PAD is more prevalent in those with diabetes, and it is associated with deeper and larger ulcers, delayed healing in DFUs[6,33] and higher morbidity and mortality.[23] Such wounds, especially wounds that penetrate to bone, non-healing wounds (>30 days) and recurrent wounds, place patients at increased risk of infection.[24,34] In peripheral neuropathy, the loss of a protective sensation in diabetic feet predisposes the foot to ulcer formation and infection.[22] In a study by

Table 1. Classification of risk factors associated with diabetic foot infection

Factor classification	Risk factors
Patient factors	▪ Peripheral arterial disease ▪ Peripheral neuropathy ▪ Impaired immune function ▪ Neuro-arthropathy
Wound factors	▪ Penetration to bone ▪ Non-healing wounds ▪ Recurrent wounds
Social factors	▪ Smoking ▪ Access to healthcare ▪ Foot hygiene ▪ Social determinants of health

Lavery et al., all but one of the 1229 DFUs had a traumatic aetiology precipitating DFI.[25] Poorly controlled diabetes, signified by a raised glycated haemoglobin (HbA1c) above 7%, is associated with increased risks of DFI,[26,35] with studies showing that intensive diabetic control reduces the long-term risk of developing a DFU,[36] improves wound healing[37] and reduces LEA rates.[38] In addition, higher rates of DFI are seen where there is poor foot-care literacy, foot hygiene and healthcare resources.[34,39] Smoking is a risk factor for both PAD and independently for DFI,[34] and it results in oxidative stress, limits angiogenesis and reduces rates of wound healing.[40] In evaluating morbidity of poor wound healing in DFUs, the rates of LEA in people who currently smoke were higher than those who never or used to smoke, despite younger age,[41] with smoking cessation associated with decreased risks of LEA.[42]

Multiple assessment tools have been developed to assess DFU infection, with a focus on local and systemic signs and symptoms of inflammation, including the Texas University Classification, the International Working Group for Diabetic Foot Guidelines (IWGDF)/Infectious Diseases Society of America (IDSA) diabetic Foot Infection Clinical Classification, the Wagner Classification and the Diabetic Foot Risk Assessment (DIAFORA) score.[39] The first-line classification tool is the IWGDF/IDSA diabetic foot infection clinical classification for assessment of presence and severity of diabetic foot infection (*Box 1*), on which the Wound, Ischemia, and foot Infection (WIfI) and Site, Ischaemia, Neuropathy, Bacterial Infection and Depth (SINBAD) classifications are based. These have been shown to predict outcomes such as healing, ulcer-free duration, risk of hospitalisation and LEA.[43]

Clinical assessment includes assessing for signs and symptoms of local infection (the probe-to-bone test is highly specific and sensitive for osteomyelitis),[44] as well as assessment of peripheral pulses for concurrent PAD.[45] To guide treatment, deep tissue cultures should be sent, with caution against superficial cultures that are associated with high commensal bacteria growth.[39]

Evidence of systemic involvement includes raised inflammatory markers and deranged vital signs, which would render the infection severe (*Box 1*). Radiological imaging may be useful in assessing for abscesses and osteomyelitis, with common imaging tools being X-rays and magnetic resonance imaging (MRI) for osteomyelitis and/or abscesses – or computed tomography (CT) when MRI imaging is not suitable.[46]

Assessment for concurrent PAD is essential, and it is included in WIfI and SINBAD, especially in hard-to-heal wounds.[47] Screening modalities include bedside Doppler assessment of pulses, measurement of toe pressure,[48] toe brachial pressure index (TBPI) and ankle brachial pressure index(ABPI).[49] Further imaging Doppler ultrasonography of the lower limb is recommended for diagnosed PAD, in order to evaluate disease burden, with alternative imaging options including digital subtraction angiography, CT angiography and contrast-enhanced magnetic resonance angiography, in order of recommendation.[39]

Box 1. IWGDF/IDSA classification of diabetic foot infection

Grade 1 (uninfected)
No systemic or local symptoms or signs of infection

Grade 2 (mild)
Infected, where at least two of the following items are present:
- Local swelling or induration
- Erythema >0.5 but <2 cm in any direction around the wound
- Local tenderness or pain
- Local increased warmth
- Purulent discharge

and no other cause of an inflammatory response of the skin (e.g., trauma, gout, acute Charcot neuro-arthropathy, fracture, thrombosis or venous stasis)

Grade 3 (moderate)
Infection with no systemic manifestations and involving:
- Erythema extending ≥2 cm in any direction from the wound margin, and/or
- Tissue deeper than skin and subcutaneous tissues (e.g., tendon, muscle, joint and bone)

In infection involving bone (osteomyelitis), add '(O)'

Grade 4 (severe)
Any foot infection with associated systemic manifestations (of the systemic inflammatory response syndrome), as manifested by two or more of the following:
- Temperature >38 °C or <36 °C
- Heart rate >90 beats/min
- Respiratory rate >20 breaths/min or $PaCO_2$ <4.3 kPa (32 mmHg)
- White blood cell count >12000/mm^3 or <4 g/L or >10% immature (band) forms

In infection involving bone (osteomyelitis), add '(O)'

Note: Infection refers to any part of the foot, not just of a wound or an ulcer. If osteomyelitis is demonstrated in the absence of ≥2 signs/symptoms of local or systemic inflammation, classify the foot as either grade 3(O) (if <2 SIRS criteria) or grade 4(O) if ≥2 SIRS criteria)

IWGDF/IDSA=International Working Group for Diabetic Foot/Infectious Diseases Society of America; $PaCO_2$=partial pressure of carbon dioxide; SIRS=systemic inflammatory response syndrome

Delayed healing of diabetic foot ulcers

Wounds fail to heal due to molecular and cellular abnormalities that inhibit the healing process. This can result from disrupted cellular activities that result in elevated inflammatory cytokines and metallomatrix proteases (MMPs), decreased growth factor activities, abnormal matrix and senescent wound cells.[50] The wound microbiome plays an important function in wound healing,

with key factors being bioburden (microbial load), diversity of microbial population and presence of pathogenic organisms.[51,52] DFUs in particular are at increased risk of poor healing due to impaired host immunity, compromised oxygenation, poor glycaemic control and presence of biofilm. DFU depth has been associated with abundance of anaerobic bacteria and negatively correlated with abundance of *Staphylococcus*, while ulcer duration is positively correlated with bacterial diversity, species richness and relative abundance of proteobacteria.[53]

The formation of biofilms significantly contribute to the delayed healing of DFUs.[54,55] Biofilms are complex polymicrobial communities, where pathogenic and commensal bacteria coaggregate symbiotically to maintain a chronic infection.[56,57] The presence of biofilms has been found in approximately 60–80% of hard-to-heal wounds and 6% of acute wounds, indicating involvement in delayed wound healing.[58] Bacteria in biofilms are embedded in a self-produced polymeric matrix, which confers protection from the host's immune system and antibiotics.[59] The heterogeneity of biofilms enables them to adapt under various circumstances via enhanced metabolic cooperation and gene regulation between sessile cells. This contributes to the chronicity of wounds despite systemic antibiotic therapy and host defence.[60,60,621] The presence of fungal communities in the polymicrobial biofilms of hard-to-heal wounds is also associated with poor prognosis and delayed healing.[63]

Necrotic tissue in DFUs also attracts immune cells, such as neutrophils and macrophages, that generate chronic inflammatory response and secrete high levels of reactive oxygen species (ROS) and proteases, such as MMPs.[64] While MMPs play a major role in wound healing and can help to break down the attachment between biofilm and tissue, excessive amounts of MMPs in hard-to-heal wounds can impair healing by damaging normal or regenerating tissues and degrading the extracellular matrix and proteins involved in the healing cascade, leading to delayed wound healing.[65,66] This process perpetuates the cycle of wound chronicity and extracellular matrix destruction, which prolongs the inflammatory response, fueling more biofilm formation and MMP release from immune cells. Evidence suggests that 'trapping' MMPs may promote wound healing and new tissue growth.[67] Hence, effective wound-bed preparation (WBP) is the key step in diminishing MMP and biofilm production.

Wound bed preparation

Recognition around the factors contributing to delayed wound healing gave rise to the concept of WBP, as first described by Sibbald in 2000.[68] WBP is defined as the promotion of wound closure through diagnosis of the cause, attention to patient-centred concerns, and correction of systemic and local factors that may delay healing.[69] This concept originally focused on three components of local wound care: debridement, wound-friendly moist interactive dressings and bacterial balance, which aimed to reduce bacterial burden once systemic factors that inhibit wound healing have been addressed.[68]

Since then, various algorithmic approaches have been described to aid assessment and treatment of hard-to-heal wounds. These include Tissue, Infection/Inflammation, Moisture balance and Edges of wound non-advancing (TIME) and Moisture balance, Oxygen balance, Infection control, Supporting strategies, Tissue management (MOIST).[50,69] For DFU assessment and treatment, clinicians are advised to conduct radical and repeated debridement, frequent inspection, bacterial control and careful moisture balance to prevent maceration.[70]

Antiseptic solutions in wound cleansing as adjuncts to wound bed preparation

Wound cleansers are solutions that move foreign materials on wound surfaces and their surrounding skin. These include water, saline, wound irrigation solutions that have antimicrobial properties and antiseptic solutions. Possible modes of wound cleansing may include irrigation (flushing a wound to remove necrotic tissue and debris), rinsing (using a gentle stream of solution to remove contaminants) or soaking (immersing the wound in solution or using an overhydrated gauze to loosen debris and cleanse the wound).

Antiseptic solutions are defined by the International Wound Infection Institute (IWII) as topical agents with broad-spectrum activity that inhibit multiplication of, or sometimes kill, microorganisms.[71] Antimicrobial activity must be balanced against cytotoxicity, spectrum of antimicrobial activity, residual effect and potential for tolerance.[71] Antiseptic solutions have additional antimicrobial properties compared with other wound cleansers, and they are useful adjuncts to WBP besides debridement. They can treat wound infection by therapeutically reducing bioburden, ultimately disrupting biofilm while limiting its regrowth. For non-infected, high-risk wounds, antiseptic solutions can also potentially reduce the risk of infection.

Common antiseptic agents for wound cleansing are octenidine, polyhexamethylene biguanide, povidone iodine (PVP-I), hypochlorous acid (HOCl) and chlorhexidine (*Table 2*).

Evidence on antiseptic solutions in diabetic foot ulcers

There are limited in vivo studies investigating the use of antiseptic solutions as a wound cleanser for DFUs. Most have a non-uniform design, with small sample sizes and varying clinical end points.[62,63] The variability of these

Table 2. Antiseptic agents for wound cleansing

Antiseptic agent	Mechanism of action	Recommended use	Contraindications	Comments
Octenidine (OCT)[117] ▪ Octenisept® 0.1% OCT +2% 2-phenoxyethanol ▪ Octenilin® 0.05% OCT + ethylhexylglycerin	▪ Cellular death by binding to and disrupting cell membranes and inducing loss in the packing order of bacterial phospholipids with a broad spectrum of antimicrobial activity[71,72] ▪ Binds to and denatures enzymes ▪ Anti-inflammatory, immunomodulatory properties and ability to modulate MMPs ▪ Active against Gram-positive bacteria, Gram-negative bacteria, including multi-drug resistant variants, fungi, yeast and enveloped viruses ▪ Able to penetrate and disrupt biofilm ▪ Not effective against spores	▪ First-line antiseptic for acute, contaminated, and traumatic wounds, including MRSA-colonised wounds ▪ Suitable as a prophylaxis to protect wounds at risk of infection ▪ Non-allergic ▪ Suitable for neonates and pregnant women ▪ 0.05% OCT preferable for hard-to-heal wounds	▪ Not suitable for hyaline cartilage, central nervous system, peritoneal lavage and eye ▪ Not suitable for deep wounds, puncture wounds, bite wounds or abscess cavities with poor drainage due to risk of tissue oedema ▪ Avoid flushing into deep wounds	▪ Fast onset of action with a short contact time (1 minute), even in presence of a high amount of exudate or blood ▪ High biocompatibility index ▪ Antibiofilm ▪ Remanent effect: has residual efficacy of 48 hours ▪ Only superficial application by means of swabs or spray is recommended
Polyhexanide[118] ▪ Prontosan 0.1% PHMB + Betaine ▪ Actolind 0.1% PHMB + Poloxamer 188	▪ Cation that interacts with negatively charged phospholipids causing domain formation in the bacterial membrane ▪ Low surface tension of surfactant allows physical removal of debris and bacteria from the wound bed ▪ Broad spectrum of activity against Gram-positive and Gram-negative bacteria, fungi and biofilms	▪ Suitable for infected hard-to-heal wounds and burn wounds (gel, dressing) ▪ Efficiently decolonises MRSA in hard-to-heal wounds	▪ Not suitable for hyaline cartilage, aseptic joint surgeries, central nervous system, peritoneal lavage, eye, middle and internal ear ▪ Only suitable for use on cartilage when ≤0.005%	▪ Recommended 10–15 minutes of contact for efficacy due to slow onset of action ▪ Antibiofilm ▪ Remanent effect can be combined with surfactants to target biofilm
Hypochlorous acid (HOCl)[119] ▪ NaOCl 0.004%, HOCl 0.003%, (NaCl) 0.023% ▪ Granudacyn solution 0.005% HOCl and 0.005% NaOCl ▪ Hydrocyn Aqua 0.003% HOCl, 0.1% NaOCl and NaCl	▪ Oxidising agent with antimicrobial activity ▪ Anti-inflammatory properties that may improve wound healing ▪ Prevents proliferation of Gram-positive and Gram-negative bacteria including MRSA, ORSA, VRSA, VRE, viruses, fungi and spores	▪ Antiseptic cleaning of acute and hard-to-heal wounds ▪ Suitable for rinsing of peritoneum or cavities with a lack of drainage potential	▪ Few contraindications, avoid use in hypersensitivity	▪ Recommended 10–15 minutes of contact for efficacy; needs time for oxidation to be effective ▪ Low cytotoxicity ▪ In vitro studies have shown HOCl to be affected by organic material and high protein load[73]
Povidone iodine[120] ▪ Betadine 10% povidone iodine ▪ Aqueous solution	▪ Causes in vivo protein denaturation, precipitation of bacteria ▪ Antimicrobial against Gram-positive and Gram-negative bacteria, mycobacteria, yeasts and viruses ▪ Active against bacterial spores when used with a longer exposure time (2–24 hours)	▪ First choice for bite, stab/puncture and gunshot wounds	▪ Use with caution in patients with thyroid disease	▪ Easily available ▪ Decreased effectiveness in blood and organic material
Chlorhexidine[121] ▪ Chlorhexidine digluconate 0.05% (when used as wound cleanser) ▪ Aqueous solution	▪ Cation that binds to negatively-charged sites on the cell wall disrupt cell membranes ▪ Broad-spectrum antimicrobial with demonstrated activity against both Gram-positive and Gram-negative bacteria, yeasts and viruses ▪ Antimicrobial activity is dose-dependent: bacteriostatic at lower concentrations (0.02–0.06%) and bactericidal at higher concentrations (>0.12%)[73]	▪ Commonly used for pre-operative skin antisepsis ▪ Can be used for wound irrigation and cleansing	▪ Contraindicated in eyes, ears, mucous membranes and central nervous tissue ▪ Rare but theoretical risk of anaphylaxis	▪ Easily available ▪ May inhibit wound healing ▪ Higher risk of cytotoxicity when compared to OCT and PHMB

MRSA=methicillin-resistant *Staphylococcus aureus*; NaOCl=sodium hypochlorite; NaCl=sodium chloride; ORSA=oxacillin-resistant *Staphylococcus aureus*; VRSA=vancomycin resistant *Staphylococcus aureus*; VRE=vancomycin-resistant enterococci

studies, with investigations of numerous hard-to-heal wounds (e.g., venous leg ulcers, hard-to-heal burns and DFUs) presents a further limitation.

Piaggesi et al. studied the use of HOCl in postoperative DFU, showing a significant reduction in bacterial count after 1 month of treatment in the intervention group at 88% (HOCl) v 11% (povidone iodine) (P<0.05), as well as a higher rate of healing at 6 months at 90% (HOCl) versus 55% PVI (P=0.002).[62] Other studies have also shown that HOCl can reduce wound infection, improve wound healing and reduce periwound issues.[63]

There are no studies exploring the use of octenidine irrigation or washes in DFUs, although there are studies on its use in venous leg ulcers and malignant ulcers. A study of 126 patients with venous leg ulcers explored the effectiveness and tissue compatibility of octenidine dihydrochloride/phenoxyethanol (OHP) compared with Ringer's lactate.[75] This showed high tissue compatibility and tolerability for OHP, even over a 12-week period with several applications a week. The authors concluded that OHP is well suited for the treatment of hard-to-heal wounds, has no relevant side effects, has fewer adverse effects than Ringer's lactate and does not impair wound healing in chronic venous ulcers.[75] Another study of 30 patients with malignant ulcers used saline to rinse the wound, followed by octenidine dihydrochloride-saturated gauzes and absorbent dressings to cover the wound three times a day over a 3-week period.[76] This study showed that octenidine had high levels of activity against Gram-positive and Gram-negative bacteria. The use of octenidine correlated with a progressing eradication of multiresistant strains and alarm pathogens (P<0.001).[76] A reduction of necrotic tissue and decrease in the level of exudate, pain and malodour were observed in all patients.[76]

There are no randomised controlled trials (RCTs) exploring the use of povidone iodine as an irrigation or soaking solutions for DFUs. A retrospective study of 42 wounds (mainly DFUs) showed full closure in 29% and partial closure in 45% with regular topical povidone iodine application.[77] Another study investigating the use of wound soaking using 1% povidone-iodine solution in 153 patients with necrotising fasciitis secondary to DFUs showed no statistical difference in outcomes between the soaking and non-soaking arms.[78] The authors concluded that soaking diabetic wounds with severe infection in 1% dilute povidone-iodine solution may not reduce the hospital length of stay, risk of below-knee amputation or readmission rate.[78]

While there were no RCTs on polyhexamethylene biguanide (PHMB) solution in DFUs, PHMB wetted gauze has been shown to result in faster bacterial elimination and reduction of inflammation in acute contaminated wounds. In venous leg ulcers and pressure injuries, an RCT of 289 wounds comparing PHMB and betaine compared with normal saline showed better wound size reduction and granulation-tissue improvement in the intervention arm.[79] The use of PHMB and betain compared with normal saline as an instillation solution in negative pressure wound therapy with instillation and dwell time for DFUs has also been investigated.[80] The authors concluded that both treatments showed promise and effectiveness, but there was no clinical distinction observed between the two solutions.[80]

Properties of an ideal antiseptic solution

A 2023 consensus document on the use of wound antiseptics in practice described the following properties of an ideal antiseptic solution:[81]

- Ability to penetrate biofilm
- Antimicrobial activity against a broad spectrum of organisms
- Cost-effectiveness
- Ease and safety of use
- Fast action in acute wounds
- Non-carcinogenicity or mutagenicity
- Non-toxicity
- Non-traumatic nature
- Not causing allergy or pain
- Not causing resistance or cross-resistance
- Similarly tolerability to Ringer's solution
- Suitable chemical and physical properties with regards to colour, smell and consistency.

The same document also cited criteria for the selection of a wound cleansing solution (*Box 2*).[81] Key decisions include consideration of the trade-off between efficacy (antimicrobial effect), tolerability and cytotoxicity. In addition, in vitro data on antimicrobial efficacy may not always translate to real world results, due to differing

Box 2. Criteria for selection of wound cleansing solution

Patient factors
- Type of wound
- Location of wound (certain wound antiseptics are contraindicated on cartilage or tendons)
- Likelihood and risk of wound infection
- Allergies
- Patient's pain tolerance
- Type of organisms cultured
- Nature of usage (rinsing, soaking and instillation with negative pressure)

Technical factors
- Clinical efficacy of the solution
- Contact time required for antimicrobial effect (surfactants take effect almost immediately, while oxidising solution require time for oxidation)
- Compatibility with dressings
- Risk of cytotoxicity

Box 3. Non-direct and direct indication for antiseptic wound therapy (TILI score)

Non-direct indication (at least five)
- Erythema to surrounding skin
- Heat
- Oedema, induration or swelling
- Spontaneous pain or pressure pain
- Stalled wound healing
- Increased and/or changed colour or smell of exudate

Direct indication (one or more)
- Presence of wound pathogens
- Surgical septic wound
- Presence of free pus

effects at varying protein loads, with hypochlorous solutions having a lowered bactericidal activity in high-protein environments compared with octenidine dihydrochloride- and polyhexamethylene biguanide-based solutions.[81]

Best practices for wound cleansing

Before initiating therapeutic wound cleansing of a DFU, the patient should be assessed holistically with consideration of the aetiology and wound healing phase.[82] Systemic and local factors that may inhibit wound healing should be assessed and addressed. Systemic factors may include glycaemic control, alcoholism or smoking, while local factors can consist of vascularity and the presence of local infection in the wound. The Therapeutic Index for Local Infections (TILI) score can be used as a clinical guide for the probability of local infection and support decision making. At least five of six non-direct indicator criteria or at least one direct indicator criteria would indicate that antiseptic wound therapy be initiated (*Box 3*).[83]

The wound should be cleansed and aseptic technique followed if debridement is going to be performed. Antiseptics are not a substitute for adequate wound debridement in WBP. Slough, necrotic tissue and surrounding callus of a DFU should be removed with sharp debridement in preference to other methods, in accordance with IWGDF guidelines.[84] Deep wound swabs or tissue cultures should be taken when there is concern of systemic or spreading wound infection to guide subsequent culture-directed antibiotic therapy.

Following debridement, the wound should be rinsed, irrigated or soaked with the antiseptic agent of choice. Debris should be removed to improve efficacy and antimicrobial effect of the antiseptic. The antiseptic agent is chosen with consideration of the type and location of wound, exudate load, presumptive or cultured organism, contraindications (e.g., exposed cartilage), patient's allergies and pain tolerance, product cost and availability. The product's mechanism of action and recommended application frequency should be adhered to, and the onset of action and exposure time should be considered.[85]

Antiseptic mechanism of action

Octenisept® (Schülke, Germany) is a robust antiseptic solution consisting of 0.1% octenidine dihydrochloride and 2% 2-phenoxyethanol that exerts potent antimicrobial properties through two active ingredients: phenoxyethanol and octenidine dihydrochloride.

Phenoxyethanol is an aromatic ether alcohol that binds to and disrupts microbial cell membranes, causing intracellular leaking and cell death. It also competes with the active site of microbial enzymes, thereby halting enzymatic processes essential for microbe survival. In addition, phenoxyethanol has a direct inhibitory effect on microbial DNA and protein synthesis.[86–91] It has a broad spectrum of anti-microbial and anti-fungal activity, particularly against Gram-negative organisms and *Pseudomonas* species.[92,93]

Octenidine works primarily by targeting the cell membranes of microorganisms. It is a cation that binds to negatively charged proteins and phospholipids on cell membranes. Likewise, this disrupts the cell membrane integrity, causing intracellular leakage and ultimately cell death. In addition, octenidine binds to and denatures enzymes essential for microbial survival. Octenidine is also able to penetrate and disrupt biofilms that protect microbes, making them more susceptible to antibiotic action.[94,95]

The multifold advantages of octenidine can be summarised as follows:
- Fast onset of action: octenidine shows the fastest onset of action within a short contact time (15–30 seconds in vitro, although at least 1 minute wound contact time is recommended), compared to other common antiseptic solutions, such as povidone-iodine, chlorhexidine and polyhexanide, even in the presence of a high amount of wound exudate[102–104]
- Broad spectrum anti-microbial and anti-fungal activity: according to European standard tests, octenidine shows a broad spectrum of activity, covering all relevant pathogens in wound care, from Gram-positive and Gram-negative microbes to yeast and fungi species;[105–107] in-depth molecular and biochemical studies have also shown that the possibility of bacteria developing resistance towards octenidine is low[72,73]
- High biocompatibility: numerous studies have shown octenidine to have a favourable biocompability index with low potential for cytotoxicity and allergic reactions.[108–111]

The clinical advantages presented by Octenisept® render it an effective antiseptic solution in the setting of chronic diabetic wounds. The antiseptic is capable of preventing infection, disrupting wound biofilm, promoting rapid wound healing and reducing the ulcer surface area of DDFUs. The clinical efficacy of Octenisept® has been well documented in numerous clinical studies.[112–115]

Conclusions

The therapeutic use of antiseptic solutions, particularly Octenisept®, in the management of DFUs demonstrates promising results in enhancing wound healing and reducing microbial burden. This manuscript highlights the multifaceted benefits of Octenisept®, including its fast onset of action, broad-spectrum antimicrobial properties and high biocompatibility. These properties are critical in addressing the complex and chronic nature of DFUs, which are often compounded by biofilm formation and impaired immune responses in patients with diabetes. Clinical studies indicate that Octenisept® effectively disrupts biofilms and promotes faster wound healing compared to traditional antiseptics. Its application in DFU care could potentially lower the rates of infection, hospitalisation and LAEs, thereby improving patients' HRQoL. Moreover, the use of antiseptic solutions similar to Octenisept® can play a significant role in WBP, ensuring an optimal environment for wound healing.

Given the substantial healthcare and socioeconomic burden of DFUs, integrating antiseptic solutions into standard wound-care protocols offers a valuable strategy for improving clinical outcomes. Future research should focus on large-scale RCTs to further validate these findings and establish comprehensive guidelines for the use of antiseptics in DFU treatment.

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Case series 1

Rhyan Hitalla

These two case studies describe the use of Octenisept® in hard-to-heal wounds in patients with diabetes in a hospital network in the Philippines.

Case study 1.1

Background

A 70-year-old man presented with a hard-to heal wound on the upper left leg, which had been present for 3 months. He smoked (30 pack years), lived a sedentary lifestyle and had a medical history of type 2 diabetes, hypertension, peripheral arterial occlusive disease and hyperlipidaemia.

Presentation

The wound measured 5×7 cm, with a depth of 1.5 cm. Pale slough covered 40% of the wound bed. The wound appeared to have an appropriate moisture level, with no visible pooling of exudate. The periwound skin showed some erythema and induration.

The patient's left leg showed redness, warmth and swelling extending from the ankle to mid-calf. He also reported intermittent fever and chills over the previous week, with increased exudate and foul odour over the previous few days. All of this was indicative of local infection.

Vital signs and blood tests were undertaken (*Table 1*). His left leg had a capillary refill over 5 seconds; an ankle brachial pressure index (ABPI) of 0.5; and absent dorsalis pedis and posterior tibial pulses (diminished on the right), confirmed with a Doppler scan. He also reported worsening pain in his left leg, described as a burning sensation, which was aggravated by walking (claudication) but also present at rest. These findings were indicative of arterial insufficiency.

Intervention

The patient was started on the intravenous antibiotic clindamycin for 7 days, after which no more antibiotics were given. The wound and periwound were cleansed daily with Octenisept® antiseptic solution. Hydrogel was applied to the wound to soften the devitalised tissue, which then underwent sharp debridement (*Figure 1*). The wound was dressed daily with a regular foam dressing OR regular foam dressings to manage exudate.

Rhyan Hitalla, Nurse, Department Head, Stoma and Complex Wound Care Center, Medical City Hospital, Manila, Philippines

Outcomes

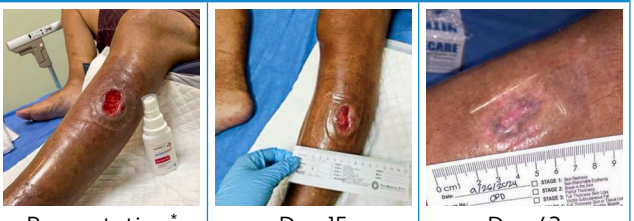
At day 15, the wound measured 3×1.5 cm, and the wound bed was covered in healthy-looking granulation tissue with a red and moist appearance, with no visible necrotic tissue or slough, suggesting that some healing was occurring. The immediate periwound area did not exhibit significant redness or swelling, suggesting no active local infection. However, systemic signs, such as cellulitis or secondary infection, must still be assessed through patient symptoms and lab values. The wound edges were well-defined, but there were few signs of potential epithelialisation.

At day 43, the wound bed showed epithelialisation, with a 1.5×0.5 cm area of pink tissue forming at the centre, and less visible granulation tissue, indicating transition to the proliferative phase and significant progress toward healing. There were no overt signs of active infection, and the surrounding skin looked healthier and less erythematous. The patient continued to use Octenisept® and the dressing regimen.

Table 1. Vital signs and blood tests

Reading	Result
Blood pressure	140/90 mmHg
Heart rate	92 beats per minute
Respiratory rate	20 breaths per minute
Temperature	38.2 °C
White blood cell count	14500 per mm^3
C-reactive protein	60 mg/L
Glycated haemoglobin	9.0%, 75 mmol/mol
Low-density lipoprotein	150 mg/dL

Figure 1. Case study 1.1



Presentation* | Day 15 | Day 43

*After debridement

Conclusions

This case involved a longstanding wound in a complex patient with diabetes. Healing progress accelerated after introduction of a multimodal regimen of Octenisept® cleansing, sharp debridement and a foam dressing.

Case study 1.2

Background

A 50-year-old man presented with a diabetic foot ulcer (DFU) on the dorsum of the foot. He had diabetes and uncontrolled blood glucose levels.

Presentation

The wound measured 4×5 cm, with no visible depth, and the wound bed showed a thin layer of pale slough (*Figure 2*). The wound surface appeared moist but not overly exudative. The wound edges were irregular and appeared inflamed, with no visible epithelialisation or contraction.

The periwound skin exhibited intense erythema, swelling and warmth, suggestive of inflammation and probable infection (e.g., cellulitis), although a culture was not taken. The patient's history of diabetes raised the possibility of underlying osteomyelitis or deep infection. His poor glycaemic control likely contributed to impaired wound healing, increased infection risk and delayed epithelialisation. He had a high risk for worsening cellulitis and potential sepsis, so immediate intervention was critical.

Intervention

The patient began intensive glycaemic management with insulin therapy under the supervision of an endocrinologist. He was given broad-spectrum antibiotics based on recent culture results and adjusted based on sensitivity.

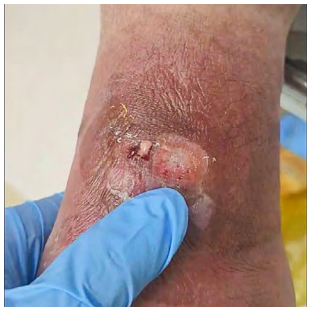
At each dressing change, the wound was cleansed with Octenisept®. The thin slough was then debrided to reveal red granulation tissue with active healing potential. This was followed by the application of an antimicrobial foam dressing to help manage bacterial load, along with a transparent secondary dressing to maintain moisture balance and hold the foam in place. Non-adhesive dressings were used to prevent further trauma to the inflamed skin.

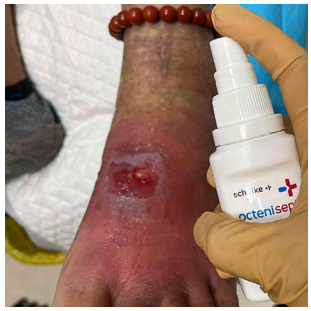
Outcomes

At day 7, the wound was slightly smaller. The wound bed showed two patches of thin, pale slough, surrounded by healthy red granulation tissue at the edges. The edges were more defined and appeared to be advancing, suggestive of tissue repair and progression to the proliferative stage. The surrounding skin appeared less inflamed, and there was no visible discharge. The patient mentioned that the secondary dressing would often come off while walking, so it was replaced with a self-adhesive cohesive bandage. The management focus remained on maintaining a clean environment, ensuring moisture balance and monitoring for any signs of infection.

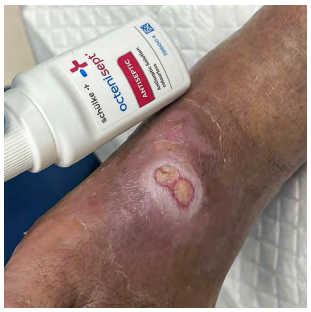
By day 45, the wound had healed completely, with no visible open lesions or active inflammation. However, the foot remained discoloured and swollen. These are not known adverse effects of Octenisept®, and the discolouration was likely a temporary result of the use of povidone iodine. However, the patient was referred for further investigations for potential vascular issues.

Figure 2. Case study 1.2

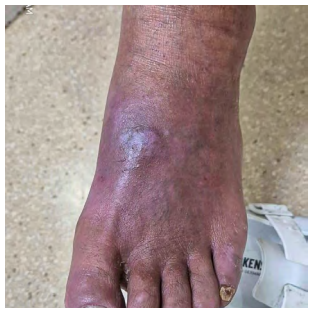

Presentation



After debridement



Day 7


Day 45

Conclusions

In this case, a DFU was treated multimodally with glycaemic management and antibiotics, as well as Octenisept®, debridement and an antimicrobial foam dressing, resulting in full healing in 45 days.

Case series 2

Luin Tongson

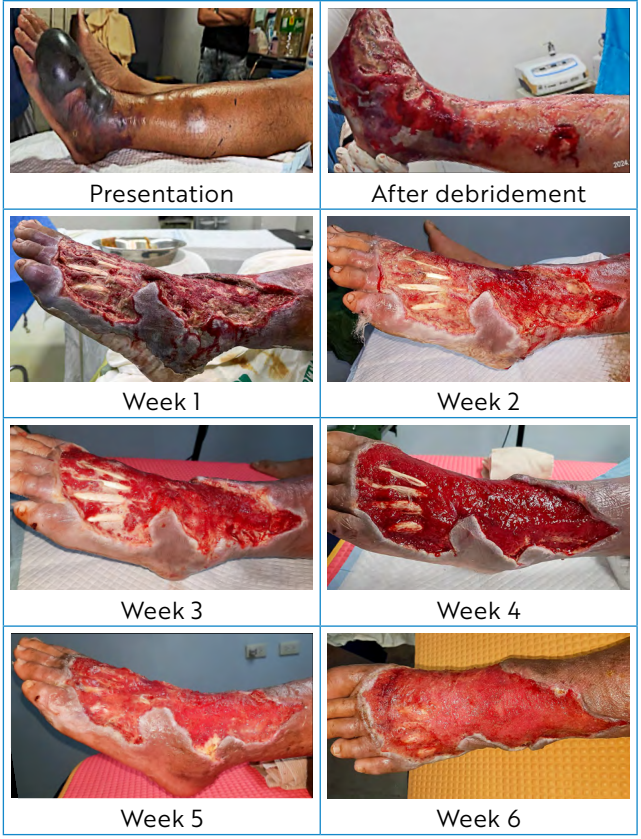
These two case studies describe the use of Octenisept® in diabetic foot ulcers at a medical centre in the Philippines.

Case study 2.1

Background

A 73-year-old man presented with a blistered (bullous) lesion on the foot, which had developed following a scratch 5 days earlier. The patient also had type 2 diabetes, with glycated haemoglobin (HbA1c) 9% (75 mmol/mol), as well as hypertension, stage 2 renal insufficiency and dyslipidemia. He had been treating the lesion with an analgesic oil (camphor 4%, menthol 1.45%, methyl salicylate 18.3%) applied 5 days prior to admission.

Figure 1. Case study 2.1



Luin Tongson, Head, Wound Care Centre, St Luke's Medical Center, Philippines

Presentation

The top of the patient's foot was covered with a necrotic bullous lesion (*Figure 1*). The blistering was surgically removed to reveal necrotic tissue up to the leg area, indicative of necrotising fasciitis, extensive infection and the need for aggressive debridement. He had pain, moderate fever, tenderness and erythema up to the middle third of the leg, which were signs moderate-grade infection.[1] The duration and recurrence of infection were suggestive of biofilm presence.[2] A culture was taken, showing bacterial growth. On palpation, the patient had grade 1 dorsalis pedis and posterior tibialis pulses and grade 2 popliteal and femoral pulses, as well as an ankle brachial pressure index (ABPI) of 0.8, indicating moderate-grade peripheral arterial disease (PAD).[3,4] He had a monofilament test score of 7/10, indicating peripheral sensory neuropathy. A Doppler scan was not performed.

Intervention

On admission, the patient was started on intravenous antibiotics, with tazobactam 4.5 g and clindamycin 600 mg every 8 hours.

At each of the following 6 weeks, the wound was assessed using the TIME tool[5] and managed according to the Wound Hygiene protocol of care:[6]

- The wound and periwound skin were cleansed with Octenisept® antiseptic solution
- Devitalised tissue was debrided with a curette (initially every 2 days in the operating room and later every 3–4 days in the wound clinic), followed by mechanical debridement with an Octenisept®-soaked gauze
- The wound edges were refashioned with a scalpel
- The wound was dressed with a foam dressing, cotton padding and compression bandages, along with zinc oxide at the periwound.

The foot was stabilised and immobilised with ankle foot orthoses and kept from bearing weight with use of a cane.

Outcomes

At week 1, the wound measured 30×15 cm, with self-reported pain (visual analogue score) of 7/10, high exudate and unhealthy rolled edges. The wound bed showed

50% granulation tissue, 40% slough and 10% necrotic tissue, with exposed tendons.

At week 3, the wound bed showed 60% granulation tissue and 40% slough, and the pain had reduced to 5/10. The foam dressing was replaced with negative pressure wound therapy (NPWT), which had not previously been available.

Significant signs of infection ceased to be present from week 4. From week 5, the wound showed notable improvements, including 80% granulation tissue and 20% slough, absence of devitalised tissue, moderate exudate, high viscosity, pain of 2/10 and healthy wound edges.

The size of the wound did not change over the 6 weeks. However, by week 6, the wound was 90% granulated, and a wound culture was negative for bacteria. Therefore, a plan was made for split-thickness skin grafting to reconstruct the soft tissue.

Conclusions

The case demonstrated effective treatment of a severely infected foot wound in the presence of necrotising fasciitis, type 2 diabetes and PAD, with the likely contribution of Octenisept® as part of a multimodal approach alongside antibiotics and aggressive debridement.

Case study 2.2

Background

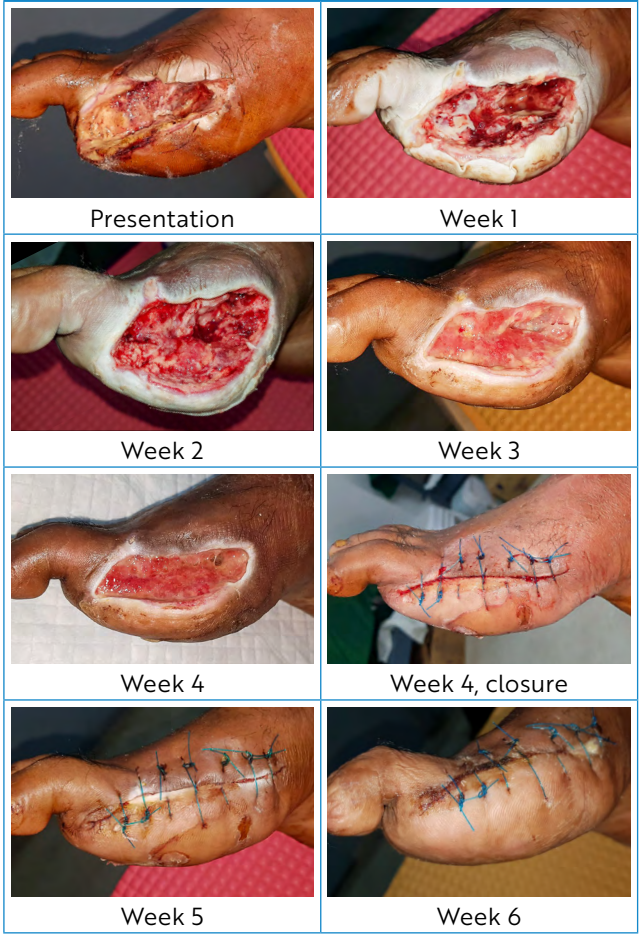
A 60-year-old man presented with two wounds on his right foot: a diabetic foot ulcer (DFU) on the heel and a surgical wound following an open ray amputation of a DFU. He was subsequently referred to the wound care centre for outpatient wound management. The patient also had type 2 diabetes (HbA1c 9.5%, 80 mmol/mol), hypertension and hyslipidaemia.

Presentation

The amputation wound measured 8×4 cm, with 2 cm depth, and the wound bed showed 20% granulation tissue, 70% slough and 10% necrotic tissue, with exposed muscle and tendon (*Figure 2*). The heel wound measured 5×5 cm, and the wound bed showed 50% granulation tissue, 40% slough and 10% necrotic tissue, with periwound maceration (*Figure 3*). Both wounds were heavily exuding, with high viscosity, had unhealthy-looking edges and showed signs of moderate-grade infection.[1] There was no sign of osteomyelitis.

The patient reported a pain score of 5/10. He had grade 1 dorsalis pedis and posterior tibialis pulses and grade 2 popliteal and femoral pulses. He had an ABPI of 0.8 on the right and left, indicative of moderate PAD.[3,4] He had a monofilament test score of 5/10, indicating peripheral sensory neuropathy. A Doppler scan was not performed.

Figure 2. Case study 2.2, amputation wound



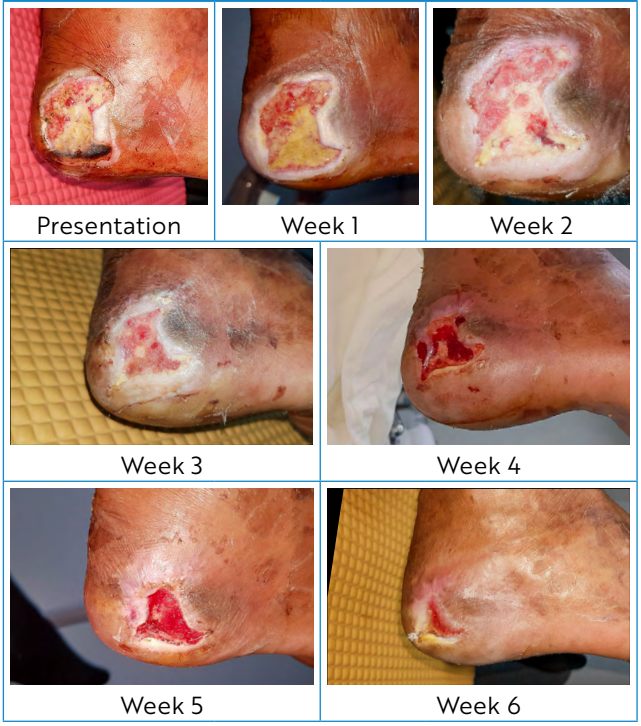
Intervention

The patient was started on intravenous antibiotics, beginning with broad-spectrum piperacillin-tazobactam 4.5 g every 8 hours and, following wound-culture results showing *Enterobacter*, shifted to ciprofloxacin. At each of the following 6 weeks, the wound was assessed using the TIME tool[5] and managed according to the Wound Hygiene protocol of care:[6]

- The wound and periwound skin were cleansed with Octenisept® antiseptic solution
- Devitalised tissue was debrided with a curette (initially every 2 days in the operating room and later every 3–4 days in the wound clinic), followed by mechanical debridement with an Octenisept®-soaked gauze
- The wound edges were refashioned with a scalpel
- The wound was dressed with a foam dressing to control exudate, cotton padding and compression bandages, along with zinc oxide at the periwound.

The foot was offloaded with use of an aircast walker and cane.

Figure 3. Case study 2.2, heel wound



Outcomes

The amputation wound gradually improved over 6 weeks. At week 2, it was 50% granulated and moderately exuding. At week 4, it was 90% granulated and no longer showed signs of infection, despite staying the same size. The wound was partially approximated with sutures. Through weeks 5 and 6, the exudate was light and the edges healthy, and the wound stayed closed.

The heel DFU also gradually improve over the same period. By week 3, it showed 70% granulation tissue, with no signs of infection. At week 4, the edges looked healthy and were advancing. From week 5, the exudate was moderate and periwound maceration was minimal. At week 6, the wound measured 3×2 cm, and the wound bed was 95% granulation tissue.

Conclusions

The case showed the effectiveness of a structured, multimodal approach to wound care, involving antibiotics, Octenisept® and debridement, in treating DFUs and amputation wounds. It also demonstrated the value of this approach in a resource-limited context, as both NPWT and split-thickness skin grafting had been considered but declined by the patient for financial reasons.

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Case series 3

Kavitha Sanmugam

These two case studies describe the use of Octenisept® in diabetic foot ulcers at a community hospital in Singapore.

Case study 3.1

Background

A 73-year-old man presented with a surgical wound on the right foot following amputations of the third right toe due to gangrene 35 days priors and the second right toe due to cellulitis 7 days prior. The amputations were necessary despite efforts by the vascular team at an acute hospital to manage and revascularise the patient, who underwent duplex scans (*Box 1*) and a right lower-limb angiogram and failed recanalisation of the anterior tibial artery and dorsalis pedis arteries. He had a history of hypertension, peripheral arterial disease and type 2 diabetes, which was poorly controlled, with glycated haemoglobin (HbA1c) of 10.6% (92 mmol/mol). Post-surgery, he was transferred to a community clinic for wound care management.

Presentation

Upon initial assessment using the Triangle of Wound Assessment (TOWA) tool, the wound was located over the right second and third digits post-amputation. The wound measured 6.3×3.8 cm and was 2.5 cm deep (*Figure 1*). The wound bed consisted of 80% adherent slough and 20% necrotic tissue. The wound edge was predominantly covered with dry necrotic tissue, and the periwound area showed necrosis on the plantar aspect. Moderate amounts of haemoserous exudate were present. The patient's pain was assessed using the visual analogue scale (VAS) with a score of 6. There were clinical signs of infection, including erythema, local warmth, delayed healing and malodour. Pain, chronic inflammation, recurrent infection, slough and friable tissue indicated a high likelihood of biofilm presence.

Intervention

The patient was prescribed antibiotics, comprising intravenous (IV) augmentin for 14 days, followed by IV pipetazo for 7 days, before being stepped down to oral augmentin. The wound management protocol involved dressing changes every other day. This began with cleansing and soaking the wound for 1 minute with Octenisept® antiseptic solution, chosen for its broad-spectrum activity and remanence (long-term antimicrobial effect). Cleansing was followed by application of an antimicrobial hydrofiber dressing, gauze and crepe bandage, with the dressing regimen initially including negative pressure wound therapy (NPWT). Initially, the wound was not debrided, due to the patient's intolerance to mechanical and sharp debridement.

Kavitha Sanmugam, Advanced Practice Nurse, SLCWC & SLA, St Luke's Hospital, Singapore

Box 1. Duplex scan results in the right leg

Pre-amputation

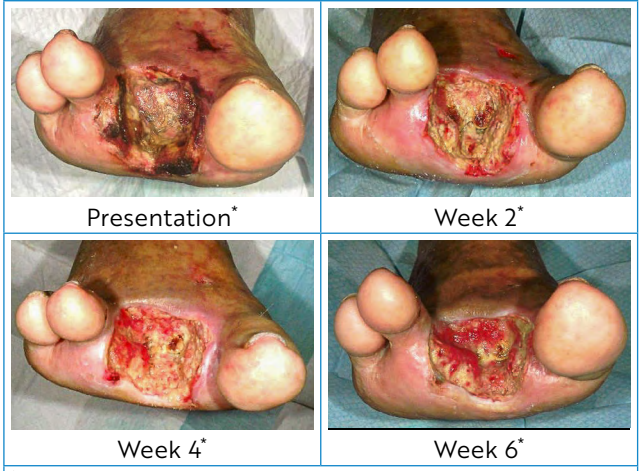
- Atherosclerotic peripheral arterial disease
- Significant stenosis in the mid-popliteal artery and origin of the PTA
- Occlusion in the ATA and DPA
- Post-endovascular thrombectomy and recanalisation
- Brisk flow to the foot via the PTA and plantar artery
- Sluggish flow to ATA and DPA
- Clinically strong DPA pulse

Post-amputation

- 50% stenosis in the distal femoral artery
- 60% stenosis in the mid-popliteal artery
- 50–69% stenosis in the origin of the PTA
- Narrowing of the distal peronal artery
- Occlusion of the ATA and stent

ATA=anterior tibial artery, DPA=dorsalis pedis artery, PTA=posterior tibial artery

Figure 1. Case study 3.1



Presentation* | Week 2* | Week 4* | Week 6*

*After debridement

Outcomes

Following the first application of Octenisept® antiseptic solution, the adherent slough and necrotic tissue softened, facilitating easier debridement, which meant that, from week 2 onwards, the patient consented to conservative sharp debridement.

At week 2, the wound measured 4.5×4 cm (25% area reduction), with 1.9 cm depth (24% reduction). The wound bed showed 10% necrotic tissue, 80% slough and 10% granulation tissue. The wound edges and periwound skin were mildly erythematous.

At week 4, the wound measured 4.3×3.8 cm (32% area reduction), with 1.8 cm depth (28% reduction). The wound bed showed 60% slough and 40% granulation tissue. The wound edges and periwound skin were less erythematous, with less erosion.

At week 5, photobiomodulation blue-light therapy became locally available as an adjuvant treatment for hard-to-heal wounds, and it was initiated and prescribed for 10 weeks.

At week 6, the wound measured 4.2×3.4 cm (40% area reduction), with 1.7 cm depth (32% reduction). The wound bed showed 30% slough and 70% granulation tissue. The wound edges were slightly macerated, and the periwound skin was intact. The moderate haemoserous exudate persisted, but the wound bed appeared healthier, and the clinical signs of infection were diminished.

The patient was maintained on a weight-bearing heel.

Conclusions

The patient reported no discomfort during the application of the Octenisept® antiseptic solution. Clinicians found Octenisept® user-friendly and effective in reducing infection in hard-to-heal wounds. The solution significantly decreased devitalised tissue, suggesting its potential to improve outcomes for chronic wounds.

A multimodal intervention, including Octenisept®, for managing a neuro-ischaemic diabetic foot ulcer demonstrated significant improvements in wound condition and infection control. This case highlights the solution's efficacy in reducing bacterial load and enhancing wound healing.

Case study 3.2

Background

A 63-year-old man presented at a community clinic with a worsening surgical wound on the medial foot. The wound was created following first ray amputation of the first right toe due to *Streptococcus pyogenes* bacteraemia, accompanied by pain, swelling and pus. Amputation was not preceded by vascular imaging or revascularisation. The patient had a history of poorly controlled type 2 diabetes (HbA1c 10.5%, 91 mmol/mol), hypertension with likely hypertensive cardiomyopathy, stage 5 chronic kidney disease approaching end-stage renal failure (not keen for renal replacement therapy), capillaritis/small vessel vasculitis, non-ST-elevation myocardial infarction (NSTEMI) and normocytic normochromic anaemia.

Presentation

The wound was assessed using the TOWA. The lateral and medial parts of the wound were closed with sutures, but the distal part was open and measured 4×2.4 cm. The distal wound bed was covered with 30% slough and 70% granulation tissue. The wound yielded a moderate amount of serous exudate. The wound edge was predominantly macerated, with haematoma and tissue necrosis on the plantar aspect, while the periwound skin was dry. The patient's pain was assessed using the VAS, with a score of 5. There were clinical signs of infection, including erythema, delayed healing and malodour (*Figure 2*).

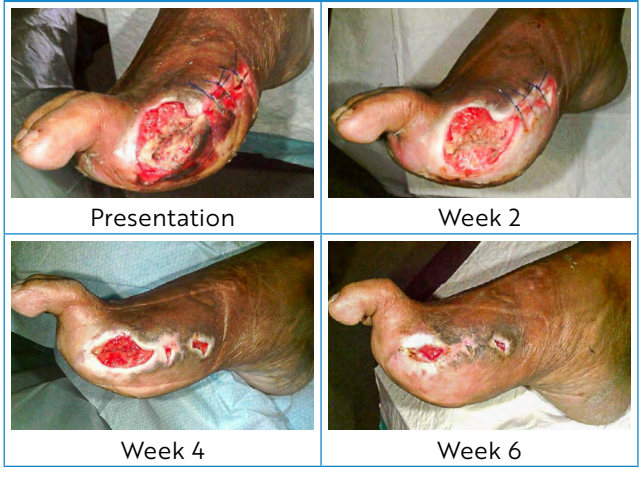
A Doppler scan revealed biphasic waveforms and an ankle brachial pressure index (ABPI) of 0.98 in the posterior tibial artery and 0.88 in the dorsalis pedis artery. Consequently, a degree of peripheral arterial disease was suspected, and further imaging would be considered if any deterioration in symptoms or the wound occurred.

Intervention

The patient was treated with antibiotics, comprising IV piptazocin for 3 days, followed by IV ceftriaxone for 2 weeks, after which he was stepped down to oral clindamycin.

The wound management protocol was carried out every other day. This included cleansing and soaking the wound for 1 minute with Octenisept® antiseptic solution, chosen for its broad-spectrum activity and long-term remanence effect. This was followed by conservative sharp wound debridement and application of a silver Hydrofiber

Figure 2. Case study 3.2



antimicrobial dressing and a foam dressing. For offloading, he was maintained on a forefoot offloading shoe with a weight-bearing heel.

Photobiomodulation blue-light therapy was provided over seven sessions. Blue-light therapy is a standard local adjuvant treatment for hard-to-heal wounds that directly probes key inflammatory cells relevant to complex healing, while promoting known inflammatory markers to switch from a pro-inflammation (M1) to pro-healing (M2) status.

Outcomes

At week 2, the distal wound measured 3.8×3.3 cm (31% area increase). The wound bed showed 20% slough and 80% granulation tissue. The wound edges and periwound skin were macerated. There was an improvement in clinical signs of infection such as erythema and malodour. Pain scores improved over the weeks of treatment.

At week 3, the sutures were removed, and the edges remained closed.

At week 4, the distal wound measured 3.1×1.7 cm (45% reduction since presentation). The wound bed had fully granulated, and the periwound skin was no longer macerated.

At week 6, the distal wound measured 2.3×0.9 cm (78% reduction). The wound edges were no longer macerated, and the level of exudate was low.

Conclusions

The reductions in 100% of slough and 78% wound area over 6 weeks of treatment represented significant healing progress. The progress was especially notable in a complex patient with multiple comorbidities. It is probable that these outcomes were the result of a decrease in wound infection brought about by a multimodal treatment strategy, including the use of Octenisept®.

The patient reported no discomfort during the application of Octenisept®. Clinicians found it easy to use and noted its effectiveness in reducing bacterial bioburden in hard-to-heal wounds.

Case series 4

Sheryl Phua, Cherry Cheong, Tiffany Chew and Enming Yong

These two case studies describe the use of Octenisept® in diabetic foot ulcers at a tertiary referral hospital in Singapore.

Case study 4.1

Background

A 71-year-old man presented with a plantar neuroischaemic diabetic foot ulcer (DFU) over the fifth left metatarsophalangeal joint, present for 9 months.[1] He also had a secondary infection, left fifth metatarsal osteomyelitis and a background of left first-to-fifth metatarsal fractures secondary to trauma, along with a complex medical history (*Box 1*).

The patient was known to the outpatient multidisciplinary foot clinic. Previous treatments had included advanced wound care therapies, including a course of macrophage-regulating cream. The patient was initially fitted with a knee-high offloading boot, but he declined using it due to feeling unstable in the boot. Offloading was then optimised with orthopaedic footwear and semi-compressed felt padding, with a cutout at the fifth metatarsophalangeal joint to offload the wound site.[2]

Presentation

On presentation, the wound measured 1.3×1.0 cm, with a sloughy wound bed and high level of serosanguinous exudate (*Figure 1*). The periwound skin was callused and macerated, as well as erythematous, swollen and warm, indicative of infection. The wound was debrided with a scalpel and then probed with a Black's file to reveal a depth of 1.3 cm. A positive probe-to-bone test palpated a hard or gritty substance that was presumed to be bone or joint space.[4] A deep wound swab confirmed methicillin-susceptible *Staphylococcus aureus*, group B *Streptococcus agalactiae* and mixed anaerobes, while previous cultures had also shown *Pseudomonas* and *Escherichia coli*. The left dorsalis pedis and posterior tibial pulses were both palpable (1+).

A duplex arterial scan of the left leg showed vessel runoff via the peroneal and posterior tibial artery, as well as a few areas of high-grade stenosis in the anterior tibial artery. There were no significant lesions in the femora-popliteal segments. The left foot X-ray showed osteomyelitis of the fifth metatarsal head (*Figure 2*). The Wound, Ischaemia, Foot Infection (WIfI) score was clinical stage 3, signalling a moderate amputation risk and very low likely benefit from revascularisation.[1]

Intervention

The patient was treated with a combination of systemic antibiotics, Octenisept® antiseptic solution and wound dressings.[5] He was offered outpatient parenteral intravenous antibiotics but initially only accepted ambulatory oral

Box 1. Medical history

- Bilateral ptosis
- Degenerative spine disease
- Diabetes mellitus
- Gastritis
- Hyperlipidaemia
- Hypertension
- Isolated lower-limb weakness from a stroke 8 years earlier
- Normochromic normocytic anaemia
- Peripheral arterial disease (left toe brachial pressure index 0.43)[3]
- Severe autonomic neuropathy
- Severe postural hypotension

Table 1. Clinical grade and investigations

Investigation	Result
Wound, Ischemia, and Foot Infection (WIfI) score	2-0-2
Ankle brachial pressure index	Right 1.07, left 1.09*
Toe brachial pressure index	Right 0.60, left 0.43
Toe pressure on the big toe	Right 105 mmHg, left 75 mmHg
Creatinine	151 umol/l

*Falsely elevated due to medial calcific sclerosis

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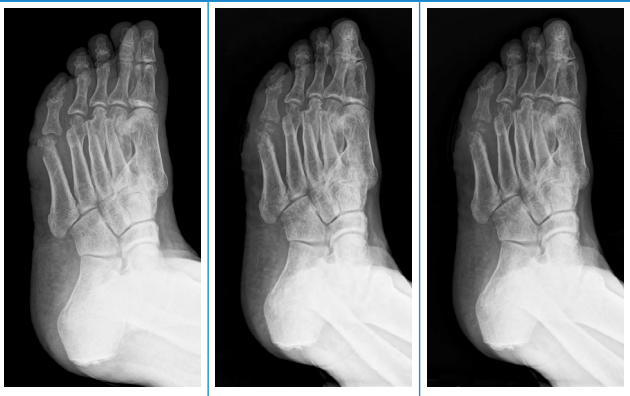
Figure 1. Case study 4.1

Figure 2. Case study 4.1, X-ray



antibiotics. Therefore, based on a previously obtained deep-tissue culture, culture-directed oral ciprofloxacin and amoxiclavulanic were given for 6 weeks. The dressing regimen included a silver Hydrofiber dressing, a polyurethane foam dressing and tape, changed every other day. At each dressing change, Octenisept® antiseptic solution was applied with a saturated swab for 1 minute. The patient also applied his own alternative medication powder containing ginger onto the wound. A low threshold was set for surgical resection if these interventions were unsuccessful.

Outcomes

The wound gradually improved over the following weeks, with a clear trajectory towards healing:

- By day 17, the wound measured 0.6×0.4×1 cm (82% area reduction) and showed 40% granulation and 60% slough on the wound bed.
- By day 45, the wound measured 1.2×0.3×0.8 cm (72% area reduction), and the wound bed showed 50% slough and 50% granulation tissue. The periwound swelling, erythema and warmth had decreased, and there was no maceration. Despite this improvement in the external appearance of the wound, there were evolving signs of osteomyelitis at the fifth metatarsal head. Therefore, the patient was counselled for and agreed to outpatient parenteral antibiotics with piperacillin and tazobactam for a further 6 weeks, initiated in consultation with the infectious diseases team.
- By day 68, the wound measured 0.5×0.4×0.3 cm (85% area reduction), and the wound bed had fully granulated. There was no periwound erythema, oedema or warmth
- By day 73, the wound had almost fully epithelialised
- By day 124, the wound had fully epithelialised, with a small depression remaining over the former ulcer site.

The multimodal treatment, involving antibiotics, dressings and an antiseptic solution, had removed the need for surgical resection.

Conclusions

In this case, an infected longstanding DFU in a complex patient was cleansed with Octenisept®, alongside use of antibiotics and antimicrobial dressings. The resulting accelerated progress towards healing demonstrates the potential of such a multimodal antimicrobial regimen.

Case study 4.2

Background

A 55-year-old man presented with two DFUs on the lateral and plantar right foot. He had a history of hard-to-heal DFUs with recurrent infection and underlying osteomyelitis, which led to extensive forefoot abscess and a complete transmetatarsal amputation 3 years earlier. The patient had a history of poorly controlled type 2 diabetes (glycated haemoglobin 10.9%, 96 mmol/mol), iron deficiency anaemia (haemoglobin 7.2 g/dL), gastritis and bilateral lower-limb chronic venous insufficiency with a history of venous ulcers. The wounds had initially undergone multiple debridements following admission to the orthopaedic department.

Presentation

The lateral wound measured 2.0×0.4×1.0 cm, with periwound skin that was erythematous and 2.5°C warmer than the left foot (*Figure 3*). The plantar wound measured 3.2×2.0×0.1 cm, with periwound skin that was also erythematous and 2.8°C warmer than the left foot (*Figure 4*). There was evidence of underlying osteomyelitis of the fifth metatarsal stump. Peripheral circulation was measured (*Table 2*).

Figure 3. Case study 4.2, plantar foot wound

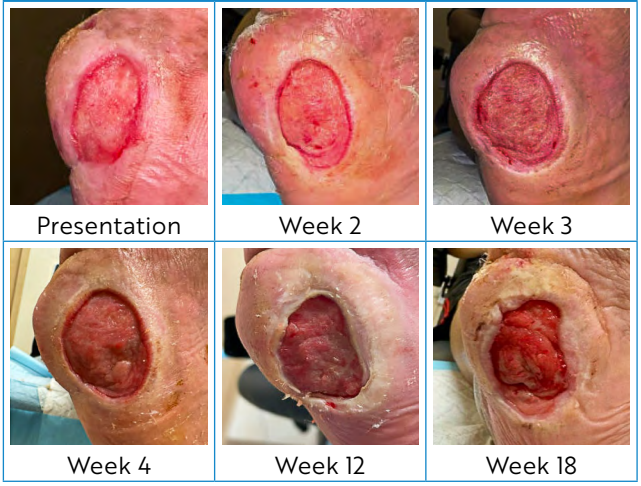


Figure 4. Case study 4.2, lateral foot wound

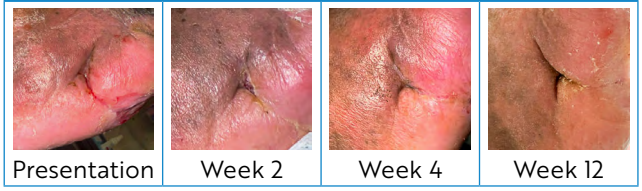


Table 2. Peripheral circulation

Measure	Right foot	Left foot
Ankle brachial pressure index	1.12	1.13
Toe brachial pressure index	N/A	0.79
Posterior tibial artery pressure	173 mmHg	175 mmHg
Dorsalis pedis artery pressure	162 mmHg	164 mmHg

A wound-tissue culture showed infection with multiple bacteria, including *Streptococcus dysgalactiae*, *Bacteroides fragilis* and *Peptoniohilus*. The bone was infected with *S. dysgalactiae* and *B. fragilis*. Following assessment using the WIfI staging system, the patient received scores of 1 (wound), 0 (ischaemia) and 2 (foot infection). The right dorsalis pedis and posterior tibial pulses were both palpable (1+).

Intervention

The wound-management protocol followed relevant guidelines.[1,2] It began with intravenous augmentin for 16 days and oral amoxicillin for 4.5 weeks.

The wounds were treated by cleansing and soaking for 1 minute every other day with Octenisept®, chosen for its broad-spectrum activity and long-term remanence effect. Cleansing and soaking were followed by surgical debridement and dressing with silver antimicrobial barrier dressing, calcium alginate dressing and polyurethane foam dressing.

The wound sites were offloaded with customised orthotics, including a toe filler for the right transmetatarsal amputation. Additionally, rocker soles were added to both shoes, along with a lateral flare and plastic stiffener to the right shoe. However, the patient declined more optimal offloading modalities, such as an aircast or knee-high offloading boot, due to work commitments.

Outcomes

After the first treatment, no growth was noted in bone culture. Following the wound-management protocol, the lateral wound reduced in area by 75% by week 2 and healed by week 3. At week 5, the patient was noted to have good clinical and biochemical response (C-reactive protein levels dropped from 67.3 mg/L to 1.4 mg/L). However, the dimensions of the plantar wound remained static through week 18, when it measured 3.2×2.7 cm with 0.2 cm depth. There were no changes to the dressing regimen.

Conclusions

Despite adequate tissue perfusion and limited risk of ischaemia, the patient's wounds had proven particularly challenging to heal because of unresolved longstanding and recurrent infection. A multimodal intervention involving Octenisept®, debridement and dressings, as well as limited offloading, successfully overcame the infection in the lateral wound. However, healing in the plantar wound remained stalled, likely because of the limitations of offloading with customised orthotics in orthopaedic footwear. The patient was therefore referred for routine review by a prosthetist for insole modification to reduce plantar pressures at the wound site.

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Case series 5

Harikrishna KR Nair and Prishela Banu

These two case studies describe the use of Octenisept® in hard-to-heal wounds in patients with diabetes at a community hospital in Malaysia.

Case study 5.1

Background

A 39-year-old man presented with a surgical wound following extensive debridement and ray amputation of the first and second toes of his left foot due to necrotising fasciitis 6 months prior. The wound had been managed at the district clinic, but no improvement was seen. His comorbidities included type 2 diabetes (diagnosed for 2 years), hypertension and dyslipidaemia. He had poor glycaemic control due to non-adherence to oral hypoglycaemic medications, with typical glycated haemoglobin (HbA1c) of around 11–12.9% (97–118 mmol/mol). He was unable to regularly attend the outpatient department to have his dressings changed. Therefore, he typically changed his dressings himself at home, where hygienic and aseptic conditions were suboptimal.

Presentation

At presentation to the outpatient wound care clinic, the wound measured 14×4 cm and showed 90% granulation tissue and 10% epithelial tissue, yet it had not been healing as expected (*Figure 1*). The patient's self-reported pain score was 1/10 (Ministry of Health Pain Scale). The exudate level was low, and the wound edges were healthy. However, the edges had advanced only very slightly since the amputation (0.1–0.2 cm). Fluorescence imaging revealed a high bioburden and likely biofilm presence (*Figure 2*). Other measurements were taken (*Table 1*).

Intervention

At each dressing change, the wound was first cleansed with Octenisept® antiseptic solution. It was then debrided with a surgical scalpel to remove callus and infected tissue. Finally, the wound was dressed with a silver nanocrystalline dressing and a low-adherent absorbent dressing, as well as moisture barrier ointment. Offloading was not considered

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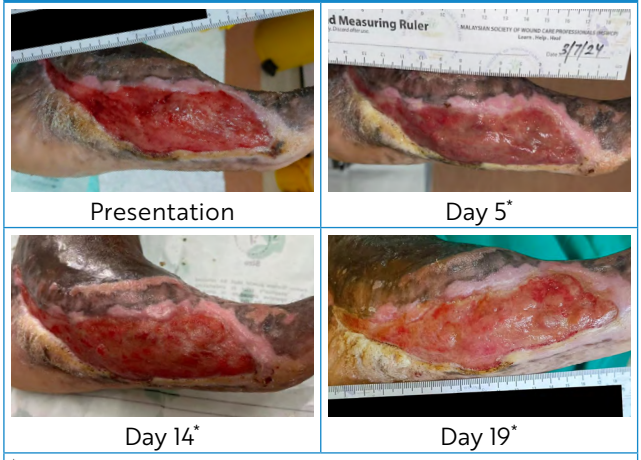
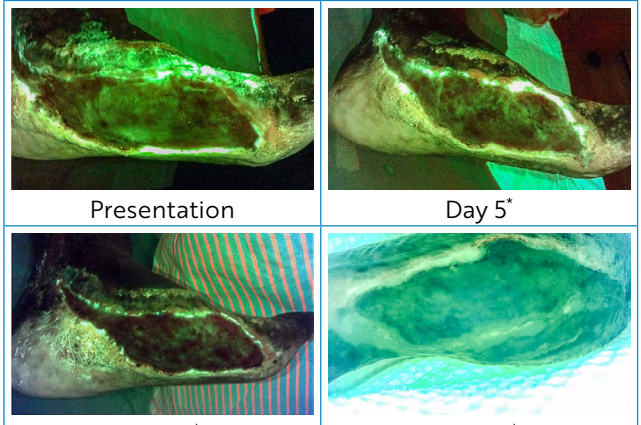
Figure 1. Case study 5.1



Presentation | Day 5* | Day 14* | Day 19*

*Before debridement

Figure 2. Case study 5.1, fluorescence images



Presentation | Day 5* | Day 14* | Day 19*

*Before debridement

Table 1. Measurements

Measurement	Result
Weight	70 kg
Height	168 cm
Body mass index	24.8 kg/m^2
Glycated haemoglobin	6.8%, 51 mmol/mol
Ankle brachial pressure index (left)	1

necessary, as the wound was on the medial aspect of the left foot.

Outcomes

The wound progressed gradually over the following weeks:

- At day 5, the wound bed showed 90% granulation and 10% epithelial tissue, with low-to-moderate exudate
- At day 14, the wound bed showed 85% granulation and 15% epithelial tissue, with no exudate and some fibrin
- At day 19, the wound bed showed 80% granulation and 20% epithelial tissue, with no exudate and more fibrin.

On day 22, the patient was admitted to plastic surgery at a specialist hospital for skin grafting, which was scheduled for 3 days later. Within 2 weeks, the graft had failed, and the patient returned to the outpatient department for dressing. As of month 8, the wound was still present but had reduced in size to 9×2 cm.

Conclusions

This case showed steady improvement in a formerly static longstanding diabetic foot ulcer after introducing a regimen of Octenisept® cleansing, surgical debridement and antimicrobial dressings.

Case study 5.2

Background

A 59-year-old man presented with an infected abrasion wound above the ankle on the right foot, present for 2 months following a reported misstep crossing a drain. The patient had a history of type 2 diabetes (HbA1c 8.4%, 68 mmol/mol) and hypertension, and he was taking oral metformin. The wound was infested with maggots, and the referral mentioned nonadherence to recommended frequency of dressing changes.

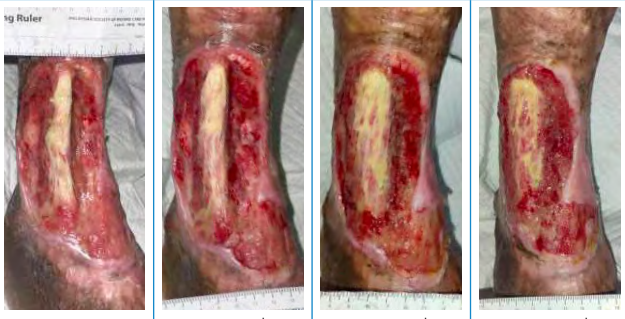
Presentation

The wound measured 16×7 cm and was 1 cm deep. The wound bed showed 10% slough, 80% granulation tissue and 10% epithelial tissue, alongside exposed tendon, which indicated poor blood supply leading to high risk of necrosis, infection and delayed healing (*Figure 3*). There was malodour, and the periwound skin was warm, red, swollen and tender, indicating infection. There was serous discharge and a moderate level of exudate. The wound edges appeared unhealthy and were not advancing, while the periwound tissue was assessed as fibrous and at risk. The patient was neuropathic and reported a Ministry of Health Pain Scale score of 1/10. The wound was assessed with fluorescence imaging, showing likely presence of biofilm (*Figure 4*). The patient also underwent vascular assessment (*Table 2*).

Intervention

The maggots were removed with sterile water and forceps. The patient was prescribed oral ampicillin/sulbactam 750 mg twice daily for 2 weeks. The wound was treated according to the Wound Hygiene protocol of care.[1] At each dressing change, the wound was cleansed with Octenisept® antiseptic solution, before being debrided with a surgical scalpel to remove slough or fibrin. It was then dressed with an antimicrobial silver primary dressing, hydrogel for the exposed tendon and a low-adherent secondary dressing. Compression and offloading were not required.

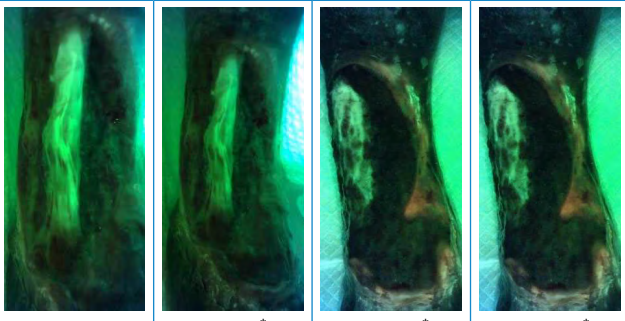
Figure 3. Case study 5.2



Presentation | Day 3* | Day 15* | Day 24*

*Before debridement

Figure 4. Case study 5.2, fluorescence images



Presentation | Day 3* | Day 15* | Day 24*

*Before debridement

Table 2. Measurements

Assessment	Reading
Ankle brachial pressure index	1.07
Dorsalis pedis artery	Feeble
Posterior tibial artery	Present
Anterior tibial artery	Unable to assess due to exposed wound
Capillary refill time	<2 seconds
Oedema	Present
Intermittent claudication	Absent
Varicose veins	Absent

Outcomes

At day 3, the wound measured 15.5×6 cm (17% area reduction) and was 0.8 cm deep. The wound bed showed 90% granulation tissue and 10% epithelial tissue. The wound edges appeared healthy and were advancing. The malodour had ceased, and the pain had reduced to 0/10.

At day 15, the wound measured 14.5×5.5 cm (29% area reduction). The wound bed showed 85% granulation tissue and 15% epithelial tissue. The silver dressing was discontinued.

At day 24, the wound measured 13×5 cm (42% area reduction), while the wound bed showed 85% granulation tissue and 15% epithelial tissue.

Fluorescence imaging showed a gradual decrease in bioburden. The periwound skin condition and moderate exudate remained consistent throughout. The maggot infestation did not recur.

Conclusions

This case shows the wound-healing potential of a multimodal combination of antibiotics, antiseptic cleansing with Octenisept®, debridement and antimicrobial dressings in a hard-to-heal wound in a patient with diabetes. Positive patient outcomes included reductions in wound area and bioburden, resolution of malodour and improved wound edges.

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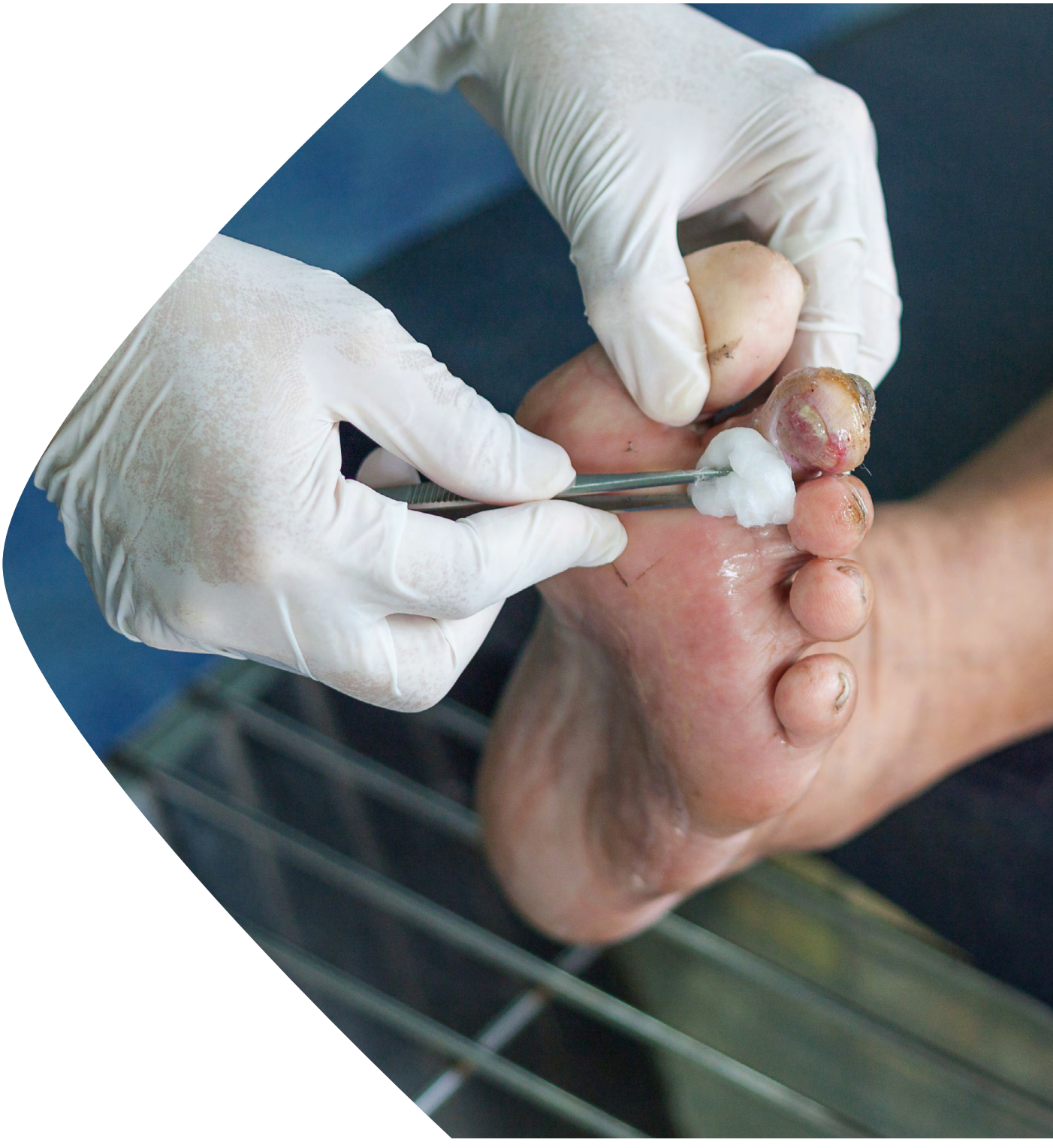

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