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Chronic wound infection: Facts and controversies

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Abstract Chronic wound infections are responsible for considerable morbidity and significantly contribute to the escalation in the cost of health care. Wound infection may initially be manifest as bacterial colonization, and it is only when colonization is combined with other factors, such as decreased vascular supply, intrinsic virulence of specific bacteria (eg, *Staphylococcus aureus*), and host immune factors, that true infection occurs. The microbiology of chronic wounds is complex, and it is difficult to discern which bacteria are culpable. Deep cultures or quantitative biopsies of wound tissue may be necessary. In some instances, such as in the presence of certain mycobacteria, isolation of specific organisms confirms causation. In many instances, it is appropriate to treat these wounds empirically with a combination of topical antiseptics and systemic antibiotics, especially in the presence of invasive infections.
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Introduction

An estimated 1% to 2% of the populace in developing countries will experience a chronic wound during their lifetime.[1] These wounds predominantly affect patients aged older than 60 years.[2] In 1999, the average cost per patient for 2 years of treatment of a diabetic ulcer in the United States of America (USA) was an estimated $27,987.[3] More recently, the cost for the treatment of a single ulcer has increased to $8000, and the cost of an infected ulcer has increased to approximately $17,000 per year.[4] Global wound care expenditures amount to $13 to $15 billion annually.[5]

There is no single agreed upon definition of a chronic wound. Most authors consider a wound to be chronic if it has not healed in 4 to 6 weeks.[6,7] Chronic wounds have also been defined as wounds that have not shown a 20% to 40% reduction in area after 2 to 4 weeks of optimal therapy. Standard surgical textbooks define chronic wounds as those that have not healed in 3 months.[8] Regardless of the duration, wounds that fail to proceed through an orderly process that produces an adequate anatomic and functional result are considered chronic wounds.[8] The most common forms of chronic wounds are related to diabetes mellitus, venous stasis, peripheral vascular diseases, and pressure ulcerations.

Infection is a common problem in chronic wounds, frequently resulting in nonhealing and significant patient morbidity and mortality. Despite extensive basic science and clinical research in chronic wounds during the last decade, several concepts remain controversial. Some of these controversies include:

1. The concept of wound colonization and critical colonization
2. The role of biofilm
3. The clinical diagnosis of infection in a chronic wound
4. When and how to culture a chronic wound
5. The role of topical antimicrobials
6. The role of systemic antibiotics; when to use and for how long

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This contribution will review these controversies and provide clinicians with an evidence-based clinical knowledge base.

The wound infection continuum

The significance of bacteria in wounds presents a continuum from contamination through colonization to critical colonization and, finally, to infection.[9] All wounds become contaminated by bacteria from the surrounding skin, the local environment, and endogenous patient sources. The local environment is particularly relevant for hospitalized patients. Colonization is defined as the presence of proliferating bacteria without a noticeable host response. Colonization of the wound may enhance or impede wound healing, depending upon the bacterial load. Bacterial loads in excess of 10^5 organisms/g of tissue are considered to impede wound healing, although this threshold may be altered by the status of the host immune system and the number and types of bacterial species present.[10] In some instances, colonization may actually hasten wound healing. It has been proposed that local inflammation may increase wound bed perfusion, leading to more rapid healing.[11]

The concept of critical colonization is controversial and not universally accepted. The term needs definitive characterization to validate its consideration in infection management.[12,13] Critical colonization is characterized by increased bacterial burden or covert infection, and the wound at this stage may enter a nonhealing, chronic inflammatory state.[10] Substantial colonization may not cause the obvious signs of inflammation but will likely affect wound healing with failure to heal or slowing of progression. Signs of critical colonization are atrophy or deterioration of granulation tissue, discoloration of granulation tissue to deep red or gray, increased wound friability, and increased drainage.[9]

The transition to infection occurs when bacterial proliferation overcomes the host's immune response and host injury occurs.[14] Several factors determine transition from colonization to infection: the bioburden itself, the virulence of the organisms, the synergistic action of different bacterial species, and the ability of the host to mount an immune response.[15] Diabetes, malnutrition, long-term steroid use, obesity, and advanced age alter the efficacy of the immune system and thus increase the risk of progression to infection. Other factors that may prevent the body from mounting an effective immune response include poor perfusion, necrosis, foreign bodies, undermining, and tunneling.[16]

Microbiology of chronic wounds

Chronic wounds have a complex colonizing flora that changes over time. *Staphylococcus aureus* and coagulase-negative staphylococci are the most commonly isolated organisms. In a study that analyzed the change of ulcer size in relation to the presence of species and quantities of microorganisms in 58 patients with venous leg ulcers, all without clinical signs of infection, *S aureus* was found in 88%, *Enterococcus faecalis* in 74%, *Enterobacter cloacae* and *Peptococcus magnus* in 29% each, and fungi in 11% of the samples.[17] The same species of microorganisms were found in ulcers that decreased (or healed) and in those that increased in size.

More recently, the bacterial profile of chronic venous leg ulcers was studied for 8 weeks.[18] Ulcer samples were collected every second week, and the bacterial species present were identified. More than one bacterial species was detected in all the ulcers. The most common bacteria were *S aureus* (93.5%), *E faecalis* (71.7%), *Pseudomonas aeruginosa* (52.2%), coagulase-negative staphylococci (45.7%), *Proteus* spp (41.3%), and anaerobic bacteria (39.1%). Resident (colonizing) bacterial species were present in all the ulcers, and two or more (up to five) resident bacterial species were found in 76% of the ulcers. This study demonstrated that the chronic wound is colonized by multiple bacterial species and that many of them persist in the wound once they are established.

The longer an ulcer remains unhealed, the more likely it will acquire multiple aerobic organisms (mean, 4.3 species) and a significant anaerobic population (mean, 2.0 species).[19] Chronic wounds tend to have a low tissue oxygen level[20] that facilitates the growth of anaerobes. In fact, chronic wounds have a statistically higher proportion of anaerobes than acute wounds (2.0 species vs 1.1, respectively, $P = .05$).[21] Anaerobes may not be identified on routine microbial culture swabs unless specimens are collected and transported to the laboratory in specific culture medium. Common anaerobic colonizers include *Prevotella*, *Bacteroides, Peptostreptococcus*, and *Porphyromonas.*[22] More than 95% of diabetic foot infections contain anaerobes along with aerobes such as *S aureus*, *Enterococcus* spp., and coliforms.[23] Decubitus ulcers, particularly over the trochanteric and sacral areas, are often exposed to fecal contamination that contains high numbers of anaerobes. Deep decubitus ulcers are at high risk for developing underlying mixed aerobic/anaerobic osteomyelitis and bacteremia and can be an unrecognized source of fever in a debilitated patient.[21]

Hospitalization, surgical procedures, and prolonged or broad-spectrum antibiotic therapy may predispose patients to colonization or infection, or both, with resistant organisms, including *S aureus* (methicillin resistant Staphylococcus aureus - MRSA) or vancomycin-resistant enterococci.[24] The presence of enterococcus, *Candida* spp, or MRSA, especially in low numbers, does not always indicate infection and treatment is not routinely indicated. Some bacteria are always significant, particularly group A β-hemolytic streptococci, *Mycobacteria,* and *Clostridium perfringens*. Rare and unusual secondary infections of chronic wounds may be caused by *Erysipelothrix rhusiopathiae*, seen in raw meat or fish handlers, and *Mycobacterium marinum* and *M ulcerans*,

acquired from aquaria, pools, or water.[21] Special culture media may be necessary for the isolation of mycobacteria, fungi, and other fastidious pathogens.

Whether the microbial profile of chronic wounds changes over time or remains stable is unclear from the limited studies that have examined this issue. One study demonstrated that colonizing ulcer flora was markedly constant over time in the individual ulcers.[17] Another study evaluated the occurrence of new bacterial groups in wounds after initial swabs in chronic leg ulcers and found at least one new bacterial group present in subsequent swabs in 82% of patients.[25] The authors concluded that the microbial populations of chronic wounds alter over time. These studies suggest that although there may be a degree of stability for some microbial populations, the chronic wound appears to be a dynamic environment.

The role of biofilm

A biofilm is a population or community of bacteria living in organized structures at a liquid interface. Bacteria within a biofilm live in microcolonies that are encapsulated in a matrix composed of an extracellular polymeric substance separated by open water channels that act as a pseudocirculatory system for the delivery of nutrients and the removal of metabolic waste products.[26] The extracellular polymeric substance also acts as a physical barrier to the permeation and the action of antimicrobial agents. The biofilm environment provides physical protection to bacteria from a potentially hostile external environment and also a habitat where bacteria can communicate with each other (quorum sensing), which may lead to an increase in virulence and propensity to cause infection.[27] Theoretically, chronic wounds offer ideal conditions for biofilm production because proteins (collagen, fibronectin) and damaged tissues are present, which can allow attachment. The biofilm, in turn, becomes a primary impediment to the healing of chronic wounds.[28]

Most of the chronic wound pathogens, such as methacillin resistant Staphylococcus aureus (MRSA) and *Pseudomonas* spp, are typical biofilm producers. Bacteria that reside within mature biofilms are highly resistant to many traditional therapies. Bacteria within biofilms have been reported to be up to 500 times more resistant to antibiotics than planktonic (unattached, freely living) cells.[29,30] Bacteria in biofilms grow more slowly, and slower growth may lead to decreased uptake of the drug and other physiologic changes that could impair drug effectiveness.[31] Currently, one of the most successful strategies for the management of biofilm-related conditions is physical removal of the biofilm, such as frequent débridement of diabetic foot ulcers.[32]

A recent small case series determined that with a trained eye, biofilm can be visualized in chronic wounds and that its appearance is quite different from that of slough.[33] Because of the differing biochemical compositions of biofilm and slough, different management strategies are required for the removal and control of these substances. Pulsed lavage and enzymatic (proteolytic) débriding agents were efficacious in removing slough, but were ineffective against biofilm. Physical débridement (sharp or use of a sterile gauze pad) was more effective than other modalities in removing biofilm, and the daily application of a nontoxic antiseptic solution prevented biofilm redevelopment.[33]

Clinical diagnosis of infection in a chronic wound

A survey of wound care clinicians in the USA showed that most practitioners rely heavily on clinical characteristics for the diagnosis of wound infection.[34] They used these findings 98% of the time, followed by patient-reported symptoms (88%) and wound culturing (70%). The clinical identification of local infection in a chronic wound can be challenging because of the frequent absence of the typical clinical signs of infection. Redness, heat, pain, swelling, and exudate may be minimal or absent as a result of the presence of factors that commonly contribute to the formation of chronic wounds. Advanced patient age, poor tissue perfusion, poor oxygenation, immunocompromised state, diabetes mellitus, and use of anti-inflammatory drugs can dampen the host response that leads to the typical signs of inflammation and infection.

In 1994, empiric data generated in a large, multidisciplinary clinical practice was used to propose a list of clinical criteria for the identification of chronic wound infection.[35] On the basis of these criteria, red friable tissue, exuberant granulation, increased discharge, and new devitalized tissue, along with other criteria (a total of 15 items) reflected greater probability of chronic wound infection.

The classic signs of inflammation may not be the most sensitive or specific for identifying infection in the chronic wound. One study evaluated quantitative biopsy specimens from patients enrolled in a large multicenter venous leg ulceration clinical trial who had ulcerations that appeared clinically uninfected. Specimens from 26% were positive for infection despite the lack of signs of infection. The investigators' analysis suggested that physical examination was unreliable in the diagnosis of wound infection in venous leg ulcerations.[36]

Clinical criteria from various guidelines have been used to define infection in chronic wounds. The Consensus Development Conference on Diabetic Foot Wound Care[37] agreed that a diabetic foot ulcer should be considered infected, when there are purulent secretions or the presence of two or more signs of inflammation, including erythema, warmth, tenderness, heat, and induration. Guidelines for the management of chronic venous leg ulcers produced by the British Association of Dermatologists and the Royal College of Physicians[38] recommend that infection should be considered if

one of the following is present: pyrexia, increased pain, increasing erythema of surrounding skin, lymphangitis, or rapid increase in ulcer size.

An interesting clinical bedside mnemonic, NERDS, has been devised to differentiate critical colonization and infection: **N**onhealing of the wound, presence of inflammatory **E**xudate, friable or **R**ed granulation tissue, tissue **D**ebris, and **S**mell suggest critical colonization and requires topical treatment; whereas, STONEES—increased wound **S**ize, increased local wound **T**emperature, extension of the wound to b**ON**e (Os), new wound breakdown, **E**xudate/**E**dema/erythema, **S**mell or odor—reflects progression to infection.[39]

A recently reported cross-sectional validation study involving 112 patients estimated the specificity and sensitivity of clinical assessment variables individually, and combined, to determine the presence and quantity of bacteria in the wound.[40] Wounds with debris, increased exudate, and friable tissue were five times more likely to have scant or light bacterial growth (critical colonization); whereas, wounds with elevated temperature were eight times more likely to have moderate or heavy bacterial growth (infection). When any three clinical signs were combined, the sensitivity was 73.3% for scant or light and 90% for moderate and heavy bacterial growth, and the specificity was 80.5% and 69.4%, respectively. The authors pointed out that the two clinicians who conducted the assessments were experts and familiar with the mnemonic. Content validity and reliability studies using other expert or nonexpert clinicians have not been conducted, however.

Clinicians should recognize that these proposed signs of infection can also be observed in several noninfectious conditions such as recurrent trauma, deep structure injury, vasculitis, or pyoderma gangrenosum.[40] Finally, contact dermatitis from the use of certain topical agents can also cause inflammation of surrounding tissues.

How a chronic wound should be cultured?

Because the signs of local and even systemic infection can be subtle or misleading in a chronic wound, bacterial quantitation was proposed 40 years ago as a potential technique to diagnose wound infection.[41] Quantitative culture of wound tissue has now become the gold standard in the diagnosis of wound infection.[42] A strong association does exist between the number of organisms in a wound and the ability of that wound to achieve subsequent healing, with an overall trend toward impaired wound healing once bacterial growth attains a quantity of 10^5 CFU/g of tissue or greater. Quantitative microbiology, however, is expensive, time consuming, and rarely used in the management of most chronic wounds.[43]

Most laboratories process swab specimens using semiquantitative or quantitative techniques, but many do not perform a quantitative swab culture unless it is specifically requested. Quantitative swab cultures are placed in 1 mL of diluent and vortexed to release microorganisms from the swab. The resulting fluid is serially diluted, plated, and incubated, usually under aerobic conditions.[44] The type and number of bacteria is measured and reported as the number of organisms per swab.[44] Semiquantitative swab cultures are inoculated onto a solid medium and streaked on four quadrants, the number of CFU are counted in each quadrant, and results are reported as 1+ to 4 + according to the number of quadrants with bacterial growth.

One study showed the sensitivity and specificity of semiquantitative swab cultures from chronic wounds was 79% and 90%, respectively, to detect wounds with greater than 10^5 CFU/cm^2 compared with quantitative swab cultures.[45] A comparison of semiquantitative and quantitative swab cultures with tissue biopsies found a 52% concordance between semiquantitative swab cultures in recovering all organisms.[46] The mean concordance of quantitative swab cultures to tissue specimens to recover all organisms was 72%, leading the investigators to recommend quantitative laboratory processing of swab cultures for optimal results. Another study demonstrated a 75% concordance between swab and biopsy specimen microbiology in samples obtained from chronic pressure ulcers.[47]

The major concern about swab wound cultures is that they reflect only the surface colonizing bacteria rather than the pathogenic strain invading deeper tissues.[48,49] Swab cultures from wounds and sinus tracts, however, can be of diagnostic benefit for two main reasons. First, the identification of certain resistant microorganisms (eg, MRSA and vancomycin-resistant enterococci) indicates the need for infection control measures. Second, the isolation of *S aureus* from superficial cultures has a high degree of correlation with deep cultures.[50] The recovery of other microorganisms correlates poorly with deep cultures.

How should a wound swab be performed?

The most important aspect of obtaining a swab culture is preparation of the wound bed.[51] Studies that have demonstrated adequate correlation between wound swab and biopsy specimen results have used specific wound-bed preparation. The wound beds in these studies generally were cleansed with saline and superficially débrided so that the culture was more likely to represent the microbiology of the deep wound compartment.[51] Because the fatty acids contained in cotton swabs can inhibit bacterial growth for some fastidious organisms, some clinicians have recommended that an alginate or rayon-tipped swab be used.[52] The organisms that routinely cause wound infection do not require these special measures, however. Premoistening a swab improves yields when sampling a dried surface[52] or swabbing a desiccated area.

Some authors have recommended that the swab be simultaneously rotated and zigzagged across the wound to cover as much surface area as possible for the culture.[53] This technique has never been validated and is as likely to increase the yield of nonsignificant superficial colonizing organisms as it is to yield true pathogens.

During the process of débridement, obtaining deep curettings for culture is appropriate because curettings correlate even more closely to biopsy specimen results than swab samples.[54] A recent literature review showed that although swab cultures are commonly performed, the practice is not standardized and most studies do not provide detailed description of swab culture techniques.[55] The following essential elements for obtaining a valid swab cultures were proposed:

1. swab viable tissue and not necrotic tissue eschar or pus,
2. débride necrosis as indicated to access viable tissue,
3. cleanse the wound before swabbing,
4. apply sufficient pressure on the swab to express fluid from the wound tissue,
5. use sterile technique,
6. obtain appropriate supplies and culture media,
7. ensure prompt transportation of the culture to the laboratory for processing,
8. provide complete labeling and a precise description of the specimens, and
9. forward to a laboratory capable of quantitative processing of the sample.[55]

The role of topical antimicrobials in chronic wounds

The term "antimicrobial agents" comprises disinfectants, antiseptics, and antibiotics. Antiseptics are chemical agents that are broadly toxic to microbes, whereas antibiotics are narrow-spectrum antimicrobial agents with specific intracellular targets. Antiseptics are used primarily to prevent infection in a wound. Among the several different types of antiseptics are alcohols (ethanol, isopropanol), anilides (triclocarban), biguanides (chlorhexidine, polyhexanide), bisphenols (triclosan), halogen compounds (polyvinyl pyrrolidone-iodine), sodium hypochlorite in the form of EUSOL (Edinburgh University Solution of Lime) and Dakin solution, heavy metals (silver compounds, including silver nitrate, silver sulfadiazine, ionic silver), peroxygens (hydrogen peroxide), and quaternary ammonium compounds (benzalkonium chloride, cetrimide).[56] Some antiseptics are cytotoxic *in vitro* to microorganisms and the host's own cells in a concentration-dependent manner.[57]

Two official guidelines have been released concerning antiseptic use on wounds. Povidone-iodine has been approved by the US Food and Drug Administration (FDA) for short-term treatment of superficial and acute wounds,[58] having been found to neither promote nor delay wound healing. Guidelines for the treatment of pressure ulcers by the US Department of Health and Human Services,[43] however, strongly discourage the use of antiseptics as solutions and promote the use of normal saline for cleansing pressure ulcers.

Although appropriate systemic antibiotics are considered essential for the treatment of clinically infected wounds,[16,59,60] the use of topical antibiotics is not justified for the routine treatment of colonized or infected wounds.[61] Topical antibiotics can provoke delayed hypersensitivity reactions[62] and superinfections,[63] and more importantly, select for resistance.[64] Routine use of topical antibiotics in the management of clinically infected leg ulcers was of no benefit,[65] and some evidence shows it may be harmful because it encourages colonization by resistant organisms.[66] Another disadvantage of topical antibiotic use is the frequent occurrence of contact allergy; neomycin is a particular example.[67]

Published guidelines on the treatment of chronic wounds do not recommend the use of topical antimicrobials. Guidelines for diabetic foot ulcers recommend only systemic antibiotics for infections,[68,69] and the Scottish Intercollegiate Guidelines Network (SIGN) guidelines on the care of chronic leg ulcers specifically advise against the use of topical antimicrobials because they are frequent sensitizers and have no effect on healing.[70] The SIGN guidelines do, however, state that a short course of metronidazole gel for odoriferous ulcers might be a possible exception.

Systemic antibiotics in chronic wounds

Despite paucity of evidence supporting their effectiveness, systemic antibiotics are frequently used in the treatment of chronic wounds. A Swedish study[71] investigated patients with chronic wounds, consisting of leg and foot ulcers, pressure ulcers, postoperative, and traumatic wounds, that had not healed in 6 weeks. At the time of the study, 26.6% were receiving systemic antibiotics, and a further 33.5% who were not receiving antibiotics at the time of the study had done so in the previous 6 months. During the 6-month period, 60.1% of chronic wound patients had received at least one antibiotic.

Indications for antibiotic therapy and optimal treatment regimens are ill defined. Most published guidelines are based on expert opinion rather than evidence-based consensus. Without proven benefit and in the absence of evidence, misuse of systemic antibiotics may place patients at unnecessary risk for significant adverse side effects and may contribute to the increasing emergence of antibiotic resistance. The polymicrobial nature of chronic wounds is likely to provide an appropriate environment for genetic exchange between bacteria. Indeed, the first two cases of vancomycin-resistant *S aureus* in the USA were both

isolated from chronic wound patients.[72,73] One study found as many as one-half of all *S aureus* isolates from hospitalized dermatology patients with leg ulcers were MRSA, and more than one-third of *P aeruginosa* isolates were resistant to ciprofloxacin.[74]

Several studies have evaluated systemic antibiotics in chronic wounds.[65,66,75] In a study that included 48 patients with chronic venous ulcers,[65] standard care alone was compared with a 10-day course of systemic antibiotics (cotrimoxazole, gentamicin, or amikacin according to sensitivity) plus standard care. Patients' wounds were not infected at baseline. At 20 days, 7 of 26 ulcers (27%) healed with standard treatment alone compared with 5 of 29 (17%) in those who additionally received antibiotics. This difference was not statistically significant. A greater mean percentage decrease in ulcer surface area was observed in the group receiving antibiotics compared with standard care alone, but the between-group difference was not statistically significant.

A three-arm trial compared ciprofloxacin, trimethoprim, and placebo in chronic venous ulcers.[66] The interventions were delivered for 12 weeks to 36 people in an outpatient setting. It was unclear whether the ulcers were infected at baseline. At 16 weeks, 3 of 10 ulcers (30%) healed with placebo compared with 5 of 12 (42%). This between-group difference was not statistically significant. The ciprofloxacin group contained patients who had, on average, larger and more chronic ulcers (surface area of 53 cm^2 and wound duration of 72 months) compared with those receiving placebo (27 cm^2 and 29 months). This means that the observed treatment effect might not be explained by the interventions alone and that the trial was likely biased in favor of placebo.

A recent Cochrane review of antibiotics for chronic venous ulcer concluded that current evidence does not support the use of systemic antibiotics to treat venous leg ulceration.[76] Although clinicians have a low threshold for prescribing antibiotics for diabetic ulcers, the Infectious Disease Society of America (IDSA) guidelines for diabetic foot infection specifically advise against using antibiotics for uninfected ulcers.[77] A brief, culture-directed course of antibiotic use is endorsed, however, in circumstances where it is difficult to decide whether a chronic wound is infected, such as when the foot is ischemic, has abnormal coloration or a fetid odor, has friable granulation tissue, is associated with unexpected pain or tenderness, or when an otherwise properly treated ulcer fails to show healing progress.[77]

Systemic antibiotics are warranted when the degree of wound infection exceeds what can be controlled by local interventions.[78] Therefore, systemic antibiotics should only be used in the treatment of sepsis, osteomyelitis, cellulitis, lymphangitis, abscess formation, and other signs of invasive tissue infection. A surgical probe should be used to explore the wound base and edges of deep ulcers, particularly those with evidence of infection.[79] Contacting underlying bone with the probe has high sensitivity and specificity for osteomyelitis. Magnetic resonance imaging is the most accurate investigation if osteomyelitis is suspected, but plain radiographs or nuclear scanning can still be helpful if magnetic resonance imaging is unavailable.

Initial therapy is usually empiric and should be determined by the severity of the infection and on any available microbiologic data, such as recent culture results or current Gram-stained smear findings. Therapy for severe infections and for more extensive, chronic moderate infections should be started with broad-spectrum agents. These empiric antibiotics should have activity against gram-positive cocci (including MRSA in sites where this pathogen is common) as well as gram-negative and obligate anaerobic organisms. Most mild and many moderate infections can be treated with agents with a relatively narrow spectrum, such as those covering only aerobic gram-positive cocci.[80] Although anaerobic organisms are isolated from many severe infections, they are infrequent in mild-to-moderate infections, and there is little evidence to support the need for antianaerobic therapy in most infections.[77]

For mild-to-moderate infections in patients without gastrointestinal absorption problems and for whom an oral agent with the appropriate spectrum is available, oral therapy is often appropriate, especially with highly bioavailable agents such as the quinolones. The duration of antibiotic therapy in the absence of osteomyelitis is controversial, with risk of treatment failure balanced against risk of microbial resistance. Most authors recommend 2 to 4 weeks of treatment with oral agents for infected chronic ulcers.[81]

Conclusions

Chronic wounds predominantly affect patients aged older than 60 years, and with the aging of the population, their prevalence will continue to increase. Most chronic wounds are invariably colonized, and therefore, superficial swabs cultures should be avoided. Ideally, quantitative or semiquantitative tissue cultures should be obtained to guide antibiotic therapy. A properly obtained swab culture may be helpful in routine clinical practice. The notion that antibacterials decrease the bioburden (critical colonization) and promote healing of chronic wounds remains controversial and needs further study. Topical antibiotics are not recommended in most guidelines because they can provoke delayed hypersensitivity reaction, superinfection, and more importantly, select for resistance. In chronic wound infection, systemic antibiotics should only be used for the treatment of sepsis, osteomyelitis, cellulitis, lymphangitis, abscess formation, or in the presence of other signs of invasive tissue infection.

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