CLINICAL MANAGEMENT

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Heel Pressure Ulcers: Stand Guard

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PURPOSE

To present a comprehensive overview of current information on heel pressure ulcer (PrU) risk, development, prevention, and treatment.

TARGET AUDIENCE

This continuing education activity is intended for physicians and nurses with an interest in wound care.

OBJECTIVES

After reading this article and taking this test, the reader should be able to:

1. Identify risk factors for heel PrUs.
2. Describe assessment findings and staging of a heel PrU.
3. Discuss current heel PrU prevention and treatment.

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Pressure ulcers (PrUs) develop when unrelieved or inadequately relieved pressure is applied externally to the tissue, most often over a bony prominence. The heel is a very common body site of PrU occurrence, second only to the sacrum[1–3]; however, a more recent national study noted the heel as the most common body site for a PrU.[4] PrUs on the heel are painful and expensive to treat and can severely limit mobility.[5] Heel ulcers are one of the most serious lower-extremity ulcers, and all too often can lead to a below-the-knee amputation in individuals with diabetes mellitus (DM).[6]

The incidence of a heel PrU in patients, with or without DM, is approximately 19% to 32%.[7–9] This figure has increased[10] from the late 1980s to 2001.[11] A 2006 study reported a 43% incidence of heel PrUs in acute care.[12] It is estimated that 60% of heel PrUs develop in the acute care setting; however, the overall prevalence is noted to be higher in long-term care.[5]

Given that individuals are living longer and that more health problems occur with advancing age, the chance of an individual developing a heel PrU only continues to increase over time. A thorough literature search did not identify a comprehensive article on heel PrUs; thus, this article presents a comprehensive review of what is known about heel PrUs.

RISK FACTORS CONTRIBUTING TO HEEL PRESSURE ULCER DEVELOPMENT

Anatomy and Physiology

The calcaneus, the largest bone of the foot, is relatively wide for its skin surface area,[13] yet has a pointed shape to the bony prominence, with little subcutaneous fat surrounding it. This leaves the heel very vulnerable to pressure.[1,14,15] Blood flow to the heel comes primarily via the posterior tibial and peroneal arteries. The heel "padding," albeit minimal compared with other body sites, consists of a soft, subcutaneous tissue pad only 18 mm thick, whereas the dermis and epidermis together are about 0.64 mm thick, leaving the heel vulnerable to ischemia.[13]

Mechanical forces lead to tissue ischemia and vessel occlusion, which, in turn, leads to tissue hypoxia and resultant tissue death.[16] Both high tissue-interface pressure and shear force have been identified as factors causing occlusion of vessels,[17] and, in fact, vessel occlusion can occur in the presence of shear force even with low interface pressure.[18] The heel vasculature has a relatively low resting blood perfusion pressure level[19] "and higher amount of surface pressure when under load."[20–23] Therefore, lower blood pressures need lower pressure-relief levels, as the research of Mayrovitz et al[15,24–26] concurred, and this was further demonstrated in 2003 when they compared blood flow in heels (n = 12) before, during, and after direct surface loading and flow reduction with ankle-cuff compression. Although baseline flows and flow reduction did not differ, hyperemia was significantly greater when flow reduction resulted from direct heel loading. This documented that the process of recovery of flow after unloading contributes to tissue injury/breakdown.[24,25] Both animal and human research has demonstrated that an inverse relationship exists between the duration and intensity of pressure; the higher the pressure, the less time it takes for tissue ischemia and damage to occur.[27,28]

The sole of the foot has no sebaceous glands, resulting in a lack of skin lubrication. This leaves the skin dry and vulnerable to damage from friction.[29]

With age, skin thins, and the shock absorptive ability of the heel pad decreases, leaving the skin less able to resist destructive forces.[7,30] With age and arteriosclerosis, circulation can be impaired, more so peripherally and particularly in the lower extremities. Although this impaired circulation is common in older adults, it also occurs in younger individuals who smoke or have diabetes or hypertension.[31] Pressures in the capillaries are reduced, leaving them vulnerable to external pressure.[32]

RISK FACTORS FOR A HEEL PRESSURE ULCER

Perfusion Problems

Peripheral arterial occlusive disease is a risk factor for heel PrUs. The blood supply to this area is located at the end arterial plexus from the posterior tibial and peroneal arteries. The heel area bears body weight, leaving it vulnerable to decreased arterial blood supply.[33] Heel PrUs occur more often on the medial or lateral surfaces, but can also occur on the plantar and posterior aspects.[6] In individuals with diabetes, this could be related to the more frequent involvement of the tibial and peroneal than the dorsalis pedis arteries.[34]

Mayrovitz[15] studied the effects of cycled pressure relief on heel blood flow. When he cycled different pressures over the heel, it resulted in a significantly greater average blood flow with full pressure release as compared with partial pressure release. In another study of the effect of pressure-relief magnitude on heel blood flow in 12 healthy subjects, researchers used a computer to control support surface cell pressure, varying pressure cyclically at 5-minute intervals between a constant 20 mm Hg during loading and 10, 5, and 0 mm Hg during offloading. Researchers measured heel skin blood flow, average blood perfusion during each 10-minute cycle, and the hyperemic response after pressure relief.[26] They found an inverse relationship between relief pressure and heel skin blood perfusion over the cycles and during the hyperemia phase. The researchers reported that "reduced average skin blood perfusion is attributable to blunting of hyperemia when relief pressure is too high. When it corresponded to an interface pressure near diastolic pressure, little, if any, functional pressure relief or hyperemia is realized. Suitable relief pressures are likely dependent on an individual's diastolic blood pressure and the net tissue forces acting on heel blood vessels."[15] Mayrovitz et al[24,26] believed this suggested that individuals with lower blood pressures need lower pressure-relief levels and thus even greater pressure relief, which could have particular relevance for individuals with depressed vascular responsiveness and/or decreased hyperemic reserve. For patients in the intensive care unit (ICU), lower extremities need to be considered at risk until documented otherwise.[35]

Other primary risk factors for developing a heel PrU include pressure, friction, and shear. A significant and all too frequent combination of risk factors occurs when shearing and pressure

forces are concentrated on a small area directly overlying the calcaneus. This can occur in individuals who make voluntary or have involuntary movements of the lower extremities, which creates friction and shear as well as pressure.[18,36] Such patients may rock in bed or dig their heels into the bed, have leg spasms, or have parkinsonian-type tremors.

Friction

Friction, or frictional force, is ''the resistance to motion in a parallel direction relative to the common boundary of 2 surfaces.''[11] Friction occurs when a portion of the skin remains stationary and the underlying tissue shifts, resulting in diminished blood supply to the skin and consequent tissue damage.[36] When friction is present, the amount of external pressure needed to break down tissue decreases.[37] Friction can occur in patients who are restless and agitated, have dementia, cannot lift up or move easily in bed, and/or use their heels to push themselves up in bed.[38]

Shear

Shear is the ''force per unit exerted parallel to the plane of interest,''[11] whereas shear strain is ''distortion or deformation of tissue as a result of shear stress.''[11] Shear is affected primarily by 3 factors: the amount of pressure exerted, the coefficient of friction between the materials in contact with one another, and the extent to which the body is in contact with the surface (support surface).[39] Shear is seen in individuals with elevation of the head of the bed and those sitting in and sliding down in a chair.[36]

Immobility

Immobility is a primary risk factor for developing a heel PrU[40]; it is a present factor in up to 87% of cases.[41] Some degree of immobility is seen in most hospitalized patients; however, those with a fractured leg,[40,42–44] spinal cord injury (SCI),[3] or stroke are at particularly high risk for skin breakdown on the heel. Following a hip fracture, the circulation and innervation to the extremity is impaired, and the patient does not move the fractured leg, increasing the risk for tissue breakdown. Stotts et al[42] reported that patients with a fractured hip and open reduction with internal fixation have a 45.1% probability of developing a partial-thickness (Stage I-II) heel PrU, whereas Duncan and Mataya[43] reported a heel PrU incidence of 53% in 30 hip fracture patients hospitalized for 5 days or longer. Many individuals who fall and fracture a hip may be immobile for hours to a day or more before being found by another person. With impaired innervation and circulation, and with being immobile, the fractured leg is not moved and becomes ''dead weight'' with very high tissue-interface pressures. It is not uncommon for health care personnel to discover a Stage IV, unstageable, or deep tissue injury (DTI) on these individuals, but often not for a few days or more after the fall. In addition, if Buck traction is initiated before surgery, this continues to compromise the mobility of the leg. Duncan and Mataya[43] prospectively studied the effect of heel pressure relief and demonstrated a reduction in incidence to 0%.

COMORBID RISK FACTORS

It is reported that the risk is great in patients who are older, debilitated, incontinent, paralyzed, or unconscious, as well as those with metastatic cancer.[44,45] Patients in the ICU are at risk,[46] as well as patients on a vasopressor and/or a ventilator, those requiring elevation of the head of the bed, or those unable to reposition independently. Another high-risk group of patients is those with DM. Most have some degree of peripheral vascular disease (PVD), which compromises circulation, have neuropathy, and/or have a foot deformity.[40] Krueger[47] reported that 25% of heel PrUs are related to diabetic neuropathy and peripheral arterial occlusive disease. Heel ulcers in the person with diabetes are often associated with neuropathic and ischemic etiologies[48] related to lower resting perfusion pressures and higher superficial and/or deep pressures when under load.[15] An individual with DM has a heel ulcer occur 4 times more often than an individual without DM.[49]

Neuropathy (pathological changes in the peripheral nervous system) is poorly understood, but is seen frequently with age and DM. It occurs eventually in individuals with DM within 5 to 10 years of diagnosis, impairing sensation and increasing the vulnerability to break down primarily by interfering with one's awareness of the presence of pressure.[50] Neuropathy of the foot consists of 3 simultaneous phases: (1) sensory, with loss of sensation and pressure; (2) motor, with loss of intrinsic muscles and ankle-jerk reflex; and (3) autonomic, resulting in the absence of sweat and oil production, leaving the skin dry and inelastic.[51,52]

Edema compromises capillary blood flow, along with impaired oxygen and nutrient transport and waste removal.[53] The weight of the excess fluid can lead to higher resting tissue pressures, further impairing tissue tolerance to pressure.[29] All these factors contribute to the fact that, on the heel, a PrU is frequently of full thickness when initially discovered.[1]

An individual with a cerebrovascular accident is at risk for a heel PrU related to his or her limited ability to move one or both legs, as well as from friction and shear, and/or impaired cognition.[52] Patients who have had an SCI are at risk for neuropathy associated with impaired autonomic, motor, and sensory systems. Skin that is impaired neurologically undergoes metabolic changes that can take 3 to 5 years to stabilize

after injury. These changes include increased collagen catabolism, defective collagen synthesis resulting in fragile skin, abnormal vascular skin reactions, and lessened elasticity of skin. Paralysis causes reduced muscle bulk, including that over bony prominences, exposing skin to injury.[54] Some of these at-risk individuals propel themselves in their wheelchairs using their heels, increasing their PrU risk.[38,39]

A study of 242 patients in a 333-bed community hospital identified risk factors for a heel PrU to be type 2 diabetes mellitus PVD, low serum albumin, and low Braden Scale score.[55] Patients with severe PVD, patients with a history of PrUs, and those with poor nutrition, leg spasms, contractures, or agitation are also at risk.[5,40] A 2006 study[47] reported that additional individual risk factors for heel PrUs include friction and reduced sensation (individual subscales on the Braden Scale), as well as compromised circulation, the presence of antiembolic stockings, fluid intake of less than 1500 mL/d, inadequate sensation of pain and temperature, smoking, surgical procedures longer than 60 minutes, the presence of a lower-extremity cast, and immobility, particularly with restraints. According to Drennan,[5] any patient assessed on the Braden Scale with a score of 15 or less is at risk.

Patients who undergo epidural analgesia for surgery are at risk to develop a heel PrU. Epidural analgesia produces both a sensory and motor block, restricting a patient's movement and leading to prolonged pressure on the heels and the loss of the protective sensation to move the legs in response to the pressure. According to Koziak,[27] ''a constant pressure of 70 mm Hg for more than 2 hours produces tissue ischemia and irreversible tissue damage.''

Although a PrU on the heel may appear within 24 hours of surgery, it often does not develop for several days.[56] Individuals who undergo surgery for more than 3 hours or who are in the postanesthesia care unit for extended periods are among those at risk.[41] Shah[56] reported on 3 cases of fairly young individuals who developed a heel PrU following epidural anesthesia. Other reports have been made of the development of a heel PrU following epidural anesthesia for labor and delivery, as well as for hip replacement or urological surgery.[57–60] Heel PrUs can also occur in children.[61]

DEEP TISSUE INJURY

Some heel PrUs may become a DTI. A (suspected) DTI was defined by the National Pressure Ulcer Advisory Panel[62] in 2007 as ''a purple or maroon localized area of discolored intact skin or blood-filled blister due to damage of underlying soft tissue from pressure and/or shear. The area may be preceded by tissue that is painful, firm, mushy, boggy, warmer, or cooler as compared to adjacent tissue. DTI may be difficult to detect in individuals with dark skin tones. Evolution may include a thin blister over a dark wound bed. The wound may further evolve and become covered by thin eschar. Evolution may be rapid, exposing additional layers of tissue even with optimal treatment.'' In fact, many DTIs have quickly developed into large Stage IV ulcers.[63] The etiology is felt to be related to high levels of pressure at the bone-tissue interface.[64]

The DTI can present as a deep red or purple area, have a bruised appearance, or appear as a fluid or blood-filled blister. A fluid-filled blister would be more indicative of a Stage II PrU, whereas a blood-filled blister is more indicative of a Stage III-IV PrU.[65] This color is related to tissue necrosis and subsequent coagulation of the blood from stasis.[65]

Animal research has revealed that skin is the last tissue layer to lose viability and die under prolonged pressure and can be nonviable and still remain intact for up to 14 days.[66,67] Farid[65] observed, ''The tensile strength of skin rivals that of connective tissue and ligaments, a phenomenon frequently noted in deep tissue pressure damage.''

It is also known that an area of hyperemia can indicate damage from what is called a reperfusion injury. This is seen when ischemia occurs from arterial insufficiency and pressure, followed by pressure relief and reestablishment of circulation (eg, pressure is relieved), and the sudden reperfusion creates additional insult and injury to the tissue. The ''tissue injury increases with each ischemia-reperfusion cycle, the duration of ischemia, and frequency of ischemia-reperfusion cycles.''[65]

A hyperemic area can quickly become a DTI if offloading is inadequate or if offloading is adequate but the patient has an arterial embolus (eg, lower extremity).[65]

If clinicians wait to observe a nonblanchable lesion to call it a ''true'' Stage I PrU, they can miss the opportunity to prevent irreversible tissue damage.[65] In other words, if the DTI or possible DTI diagnosis is missed on admission, it would appear as a nosocomial DTI normally 7 to 14 days later, when the DTI actually was present on admission. For this reason, capillary refill should be assessed at least twice (preferably every 8 hours) during the first 24 hours after admission,[65] to distinguish between a Stage I PrU and a DTI, particularly for a patient with DM and PVD. The recommendation would be to photograph all reddened areas, as well as other obvious lesions, on admission or soon after admission. When the reddened area maintains capillary perfusion, recovery can occur in the following 72 hours.[68–72] In a DTI that ''presents as a demarcated red/purple area, clinicians can count back 7 days to pinpoint when the actual pressure damage occurred.''[65]

Clinicians are cautioned that capillary refill may seem to be present in the first 24 hours after admission; however, this is a phenomenon known as hypostasis—a condition present in

early tissue decomposition (death) that mimics capillary refill.[73,74] It is actually ''loose blood'' accumulating from capillary collapse.[65] Thus, a DTI can appear as a pale, whitish area with a waxy appearance in lighter-skinned individuals. In patients with darker skin pigment, the DTI can appear as a lighter patch of skin surrounded by an abnormally darker area that shows no color change when testing capillary refill.[65] This is seen more commonly on the sacrum, but may also be seen on the heel.

It is important to pay attention not only to the total risk assessment score, but also to the subscale categories with low scores, such as mobility, sensation, and activity.[40] In patients with DM and impaired mental capacity, it is critical to complete a thorough assessment every shift and, more frequently, even up to 3 times a day.[75]

APPEARANCE OF A HEEL PRESSURE ULCER

A Stage I PrU on the heel appears red, generally nonblanchable, and is often painful.[76] This injury has the potential to recover if offloaded continuously.[76] A Stage II heel PrU often appears as a blister or shallow skin tear, whereas a full-thickness (Stage III-IV) PrU appears open and deeper. A heel PrU caused by friction generally appears red and often is blistered from friction to tissue.[47]

A heel PrU can also be a DTI, which can occur very quickly with the minimal padding over the bony angular heel prominence. A DTI can have a bruised, purplish appearance or can appear as a blood-filled blister of intact skin. If pressure continues without relief, there can be tissue necrosis and eschar development.[76] Figure 1 defines PrU stage characteristics.

Figure 1.
CHARACTERISTICS OF A HEEL PRESSURE ULCER BY NPUAP STAGE

Stage I: Feels boggy, should be staged as Stage I or unstageable. Remove all pressure to heels. Protect heel with foam boot or gauze wrap. Protect heel with protective dressing. If arterial disease is present, keep lower extremity warm, but not hot. Abduction pillow does not take place of turning and heel elevation.
Stage II: Open area or fluid-filled blister. Remove all pressure to heels. Apply protective dressings and monitor for signs of deterioration such as increased drainage, odor, warmth, or redness, increase in ulcer size and/or depth, exposure of tendon or bone, increase in pain.
Stage III/IV: If eschar or slough is present and if it is stable and noninfected appearing, let it dry out, protect it, and do not disturb it. If it appears inflamed or infected, debride slough/necrotic tissue (by someone certified to debride), and continue to carefully monitor. Remove all pressure to heels. Topical antimicrobials are often used, and if tissue culture confirms presence of bacteria, systemic antimicrobials can be used. Monitor for osteomyelitis.
Deep Tissue Injury: Stage as unstageable. Total and persistent pressure relief. Protect intact skin on heel with nonadhesive dressing. Assess for open areas and drainage. If open area, use nonadhesive dressing to absorb drainage and protect area. Assess for change in condition or deterioration of area (increased and/or change in drainage, periwound erythema, pain, odor to drainage, enlarging area).

HEEL PRESSURE ULCER PREVENTION AND TREATMENT RESEARCH

Dressings

A 4-phase study of 242 patients was performed in a 333-bed community hospital. The experimental prospective component, trialing a heel PrU prevention protocol, documented a reduction in the incidence of heel PrU development.[12] Bots and Apotheker[77] studied prevention of heel ulcers using a self-adhesive hydropolymer foam dressing on 140 surgical patients in the ICU over 2 years. Risk was assessed using the Norton Scale, the duration of surgery was documented, and a foam dressing was applied postoperatively. Heels were inspected daily for up to 10 days. A 76.7% reduction in heel PrUs was documented with this method.

A similar study by Nakagami et al[18] compared the shear forces exerted over the heel in a sample of 30 older adult patients in a Japanese hospital. One group used a PrU preventive hydrocolloid dressing, whereas the other used a thin film dressing. Mean interface pressures were not statistically different between the 2 dressings on a paired *t* test (70.7 ± 16.5 and 70.2 ± 15.2 mm Hg; $P = .4198$). However, there was a statistically significant difference in shear force between the 2 groups on a Wilcoxon signed rank test (2.2 ± 1.4 and 11.7 ± 5.8; $P < .001$). The specially designed PrU preventive dressing had a slippery surface that helped reduce friction, and the coefficient of kinetic energy was small enough to reduce the shear force. The authors did not evaluate the ability of the specially designed PrU dressing to prevent a PrU and stressed that a longitudinal study was needed to evaluate the dressing's effectiveness in prevention of a PrU.

Overall, study results documented that an external dressing can protect and significantly reduce shear force, but did not reduce tissue-interface pressures and should not be used as an alternative for heel elevation for the immobile patient. Heels need to be offloaded.

Yet another study compared a standard preventive bandage on the heel with the Allevyn (Smith & Nephew, Largo, FL) heel hydrocellular dressing. Of the 111 patients who

completed the study, significantly (P = .001) fewer heel PrUs developed in the Allevyn group (2 of 61 patients, or 3.3%) as compared with the protective dressing group (22 of 50 patients, or 44%).[78]

Pressure-Redistributing Device Research

Products need to be evaluated on actual patients with actual risk for a particular morbidity. A good device to reduce pressure on the heel will separate and protect the ankles from one another, maintain heel suspension, and prevent foot drop.[40] Pressure-redistributing/relieving devices should consistently reduce interface pressure below 32 mm Hg or pressure-reducing support (pressure less than standard support surfaces, but not below 32 mm Hg).[75]

The heel often requires additional protection beyond the use of topical dressings and specialty beds and overlays. An experimental study evaluated the effectiveness of hospital pillows versus a commercial, triangular-shaped heel elevation device in a sample of 52 patients from 27 to 90 years of age. The heel device was 4 times more likely not to suspend the heel off the bed than the pillow.[79] A more recent randomized clinical trial of 338 adults compared 3 pressure-reduction devices (bunny boot, egg crate, and foot waffle) in the prevention of heel ulcers on moderate to high-risk patients: 12 PrUs developed, with 11 Stage I and 1 Stage II PrUs present; and the incidence was not significant (bunny boot = 3.9%, egg crate = 4.6%, and foot waffle = 6.6%).[80] In a 41-patient prospective study comparing 4 pressure-reducing devices used over 12 days, foam splints and eggshell foam were found to be more effective than Duoderm (ConvaTec, Princeton, NJ) and heel protector boots; however, the researchers stressed that use of one of these only serves to enhance meticulous skin care.[44]

A study of 52 patients compared a control group (n = 30) who received ''only high-level nursing care'' with 22 patients who received the Heelift Suspension Boot (DM Systems, Evanston, IL). No PrUs occurred in the Heelift-treated group, whereas 17% of the control group developed a heel PrU (T.I. Bordner, personal communication, April 2003). Yet another prospective study of 30 hip-fracture patients hospitalized 5 days or longer and treated with heel pressure relief documented a reduction in heel PrU incidence from 53% to 0%.[43]

SPECIFIC PREVENTION AND TREATMENT INTERVENTIONS

As most of the interventions to prevent a heel PrU are the same as those used to treat PrUs, prevention and treatment interventions will be discussed together for the purposes of this article. First, the clinician needs to perform a heel PrU risk assessment.

Risk Assessment for a Heel PrU

Individuals need to be assessed for risk of heel PrU development, most particularly those who are mobility impaired, are unable to move on their own, have impaired sensation and/or circulation of the lower extremities, have DM, or have a foot deformity. A well-researched risk assessment scale should be used. Unfortunately, most commonly used risk assessment scales do not have a subscale or factor for nonmovement of the lower extremity. Consequently, a patient with a leg fracture may not be appropriately assessed and scored on the activity subscale. Should individuals with PVD and peripheral neuropathy be considered cognitively impaired related to the lack of cognitive awareness of their lower extremities? The authors recommend that most older adults with a hip/leg fracture should be placed in an at-risk category, and appropriate preventive interventions should be implemented, from the time of the fracture through the application of Buck traction, surgery, and postoperatively until mobility is regained. Figure 2 illustrates the Heel PrU Risk Assessment Tool.

Patients/residents with cognitive impairment are at increased risk for heel PrU development and need to have their heels assessed. Individuals who are wearing TEDs (Kendall, Mansfield, MA) need to have them removed at least once, and preferably twice, a day to assess the heels. Patients with contractures and foot deformities need careful assessment of the feet and heels and for properly fitting footwear. Individuals who use their heels to propel themselves in a wheelchair are at risk as well.[38,39]

Timing of Risk Assessment

All patients need to be assessed for risk upon admission, with a significant change in condition, and periodically thereafter, appropriate to the setting and in accordance with agency policies. In acute care, this should be done *at least* every 48 hours and whenever there is a significant change in condition,[36,39] whereas the authors recommend at least daily assessment. In long-term care, assessments should be made weekly for the first month, then either monthly or quarterly, depending on agency protocol. In home care, assessments should be made during every visit by a licensed nurse. Each agency needs to define its own protocols and time frame for risk assessment based on scientific evidence and the patient/resident population.[36,39]

Figure 2.
HEEL PRESSURE ULCER RISK ASSESSMENT TOOL

Assess for the following risk factors: (count total)
Age > 70 years
Diabetes mellitus
Mental status changes: agitated, confused, stuporous, and/or unresponsive
Lack of movement in any lower extremity
Total number of risk factors
Assess activity level and treat according to risk factor level:

	Total = 0	Total = 1	Total = 2	Total ≥ 3
Ambulatory	Universal	Universal	Universal	Universal
Walks w/ assistance	Universal	Universal	Preventative	Strict
Confined to WC or bedridden	Universal	Preventative	Strict	Strict

Heel PrU Risk Assessment Tool

Blaszczyk et al[81] developed a heel PrU risk stratification tool. A total score is calculated for each patient, with 1 point given to each risk factor present. Then the nurse would determine activity level and refer to the flow chart to determine the patient's risk level (Figure 2). The authors recommended the following courses of action: daily reassessment of heels; float heels of all intubated or unresponsive patients until assessed for risk and further actions implemented; institute strict precautions if heels are red or skin appears damaged; and use heel protector/floats on all individuals at risk for heel breakdown.[82] Further interventions are outlined in that article.

PROTOCOLS

The pivotal focus for preventing and treating a heel PrU is on the interaction between external pressure and the heel vasculature.[3] Prevention strategies for heel ulcers need to be comprehensive and should include the following: identification of comorbidities, including nutrition; at least daily skin assessment; routine risk assessment using a tested scale; implementation of evidenced-based prospective interventions; early aggressive application of pressure-redistributing devices; immediate initiation of prevention and/or treatment interventions; and ongoing product evaluation with frequent documentation of heel integrity.[12,83] Black et al[63] recommended that an individual with both DM and PVD should have twice-daily heel assessments, and those in acute care with impaired mental capacity should be assessed 2 to 3 times a day. During an assessment, it is important to palpate peripheral pulses (popliteal, posterior tibial, and dorsalis pedis). Also, check for foot sensation in at-risk patients. Consider whether the risk assessment score indicates risk and whether the patient will be immobile for 4 or more hours.

Assessment of the Heels

Heels must be assessed frequently, and each setting or institution needs to define its own protocol. The Agency for Healthcare Research and Quality (formerly known as the Agency for Health Care Policy and Research) and Wound, Ostomy and Continence Nurses Society Guidelines[36,39] recommend at least daily assessments. If a patient has difficulty moving, assessments can be performed using a mirror held under the heel, which provides a good visualization of the area, allowing the assessor to see if there is a purple or bruised look, or if there appears to be a ''blood blister'' present. These factors could be indicative of DTI. Capillary refill/blanching should be checked during assessment, and the heels must be felt for a boggy/mushy feeling and undue temperature changes. Keep in mind the importance of heel anatomy, as ''anatomy can be destiny.''[84] Patients wearing compression stockings need to have them removed at least once and preferably twice a day to allow for both visual and palpatory assessments for changes. Pedal pulses need to be routinely assessed and documented. Figure 1 elaborates on the characteristics of heel PrUs by stage.

Repositioning

Turning or repositioning the patient frequently is a good way to help prevent the development of a heel PrU.[85] Defloor et al[86] compared 4 turning schemes (q2hr or q3hr on standard mattress, and turning q4hr or q6hr on a viscoelastic foam [VEF] mattress) on 838 nursing home patients and found that turning every 4 hours on a VEF mattress significantly reduced PrU development.

Pressure-Redistributing Devices

Complete elimination of heel pressure using a pressure-relief device is critical.[40] At-risk patients should have a heel

pressure-redistributing device in place to prevent breakdown and protect from the effects of pressure, friction, and shear forces to the heel tissue.[39,40] Groups of patients for whom these boot-type devices are in order include those with DM, particularly those with PVD and/or peripheral neuropathy; those with poor or limited mobility; those with absent or poor foot pulses; those undergoing surgeries longer than 2 hours and/or those who will be immobile in the recovery room and bed afterward; those with severe PVD without DM; those with a history of a PrU; and those identified at risk on the risk assessment tool.[5] A variety of boot-type devices are available for redistributing pressure to the heels.

According to Black,[40] ''the best heel-pressure–reducing products also separate and protect the ankles, maintain heel suspension, and prevent foot drop.'' Some mattresses on the market have built-in pressure-reducing areas for the heels. A static device that distributes pressure over a surface larger than the body area is often satisfactory for patients who are able to shift their weight. Examples of these devices are foam mattresses and devices filled with air, gel, or water.[11] A dynamic device is best for patients who cannot shift their weight. This category of devices would include powered devices that alternately inflate and deflate to reduce pressure. Some beds on the market have built-in, heel-area pressure-redistribution properties (eg, Clinitron [Hill-Rom, Batesville, IN], ZoneAire [Hill-Rom], Plexus Auto Aire Select [Gaymar Industries, Orchard Park, NY] [not a complete list]), whereas other beds do not have built-in heel pressure relief. No device replaces repositioning, so frequent repositioning of the patient is essential. We do know that some effectiveness of these devices can be nullified when the head of the bed is elevated.

Although the literature varies, it is reported that these devices do not effectively reduce tissue-interface pressure below minimal capillary closing pressures.[18,85] The devices provide some, albeit limited, protection against friction and shear to bony prominences, particularly when the patient is in a side-lying position.[18] Foam boots work well, as the exterior surface helps reduce friction; however, the more rigid the foam boot, the more likely it is that pressure areas can develop on the lateral ankles or the heel plantar surface. Boot-type devices help lessen external and internal rotation, but unless they are more rigid, they do not prevent it. Air boots are lighter weight and help lessen external and internal rotation, but again, they do not prevent it. Feet can sweat in air boots unless air holes are present to facilitate circulation. Some boots have a surface that allows them to slip around more on the bed surface; thus, it is important to allow for more frequent positioning checks. Straps need to be applied so the boot will stay on, but straps should not be too tight as to cause pressure. Bootstraps can cause undue pressure on the lower leg and dorsum of the foot; thus, they need to be routinely removed, and the entire foot and leg assessed for signs of undue pressure. Clinicians must check to see that the patient is actually wearing the pressure-redistributing devices and that they are worn correctly.

Heels can also be ''floated,'' and pressure can be removed with a folded towel or blanket, or a pillow, particularly when in Buck traction. It is helpful to use a pillow along the entire length of the lower extremity to protect the knee and not to place the elevating device under the Achilles tendon. One can verify if the heels are actually floating by slipping a sheet of paper between the heel and bed surface without it touching the heel or by slipping the clinician's hand between the heel and the bed surface. The bed knee gatch can be raised as long as the heel is floated and the Achilles tendon is protected. When the foot of the bed is elevated (eg, to reduce edema), remember to also elevate the patient's knee(s) to prevent suspending the leg by the heel.[40] Ascertain that foot drop is not present, which could allow for heel cord contractures.[5] When possible, get the patient moving and minimize bed rest time. When feet are elevated to reduce edema, it is very important to have the patient flex his or her knees to prevent hyperextension and reduce pressure on the heels.[40]

Skin Integrity of the Heel and Other Foot Bony Prominences

Preserving the integrity of the skin is critical in preventing a PrU on the heel or other foot bony prominences. This can be accomplished by removing the pressure, by using lubricants and/or moisturizers, and by using protective dressings (film, foam, or hydrocolloid) or protective padding.[36,38,39] These dressings *do not* provide pressure relief. With a Stage I heel PrU, relieving the pressure is often all that is needed for the tissues to recover. If a blister is present on the heel, do not break it. Merely elevate the leg and heel. If the PrU has eroded the skin (Stage II-IV), a moldable dressing is appropriate to protect it, keep it clean, and maintain moist wound healing (eg, hydrocolloid, impregnated gauze, or foam). If infection is suspected, do not use an occlusive dressing that can hold bacteria in the wound and encourage spread.[76] Twice-daily moist gauze dressings would be an alternative for this situation.[76] Monitor the heel area for signs of deterioration, including erythema, odor, increasing drainage,

color and type of drainage, fever, increasing pain, and/or exposed bone.[40]

Specific Heel PrU Treatment

See Figure 1 for further elaboration on this topic. Treatment of a Stage III or IV heel PrU remains controversial, and additional research is in order. The current consensus is to not remove stable eschar, allowing it to separate on its own.[38,39] Heel vascularity underlying the eschar is essentially absent, with only a subcutaneous or fat tissue pad normally; thus, it is susceptible to infection with limited to no ability of the body to fight it. In the presence of eschar, wrap the heel in gauze and relieve the pressure.[76] Also assess it at least twice a day for changes, such as bogginess, edema, redness, drainage, or overt signs of infection. The presence of infection necessitates a change in the treatment plan. As the eschar loosens from the underlying tissue, a qualified debridement clinician can trim the loosened eschar and nonviable tissue.[76] When the eschar is open or cracked or infection is present, debridement of the tissue is in order.[38] Containment of drainage is important.[83]

It is important to perform a nursing nutritional assessment, assessing both the nutritional and hydration status of the patient. If the patient is identified at nutritional risk or if the patient has a heel PrU, obtain a full nutritional evaluation and ensure adequate dietary intake to prevent malnutrition.[38,39] As needed, the registered dietitian will recommend, and the physician will order, a nutritional supplementation if the patient has a PrU, has inadequate dietary intake (particularly protein), or is malnourished. Treatment should include a balanced diet with good sources of vitamins and adequate protein and calories as prescribed by the dietitian.[38,87] Research has not yet documented that high doses of vitamin C will accelerate wound healing.[88]

CONCLUSION

The heel, although currently the second most frequent body site for a PrU, is fast on the path to becoming the most common PrU site. Heel PrU prevalence is increasing and is a real and present danger, particularly in individuals with cognitive or sensory impairment and/or limited mobility. It is critical that all health care team members integrate prevention and early intervention strategies into their practice to prevent heel ulcers. Risk identification, comprehensive histories, daily or more frequent foot inspections, and immediate preventive interventions are all vital. Patients and health care workers need to know the latest strategies on preventing heel ulcers, identifying them at an early stage, and implementing appropriate measures for healing. Each clinical setting needs to develop evidence-based protocols specific to its patient population. Heels of at-risk patients need to be assessed and floated. As the annual cost of treating nosocomial PrUs is estimated to be $2.2 to $3.6 billion per year[89] and the cost to treat a Stage IV PrU can be upward from $70,000,[90] spending $35 or more per heel protection boot would seem to be a wise and cost-effective preventive investment. ●

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