PRESSURE ULCERS
Prevention and Management

ABSTRACT: Pressure ulcers are a significant medical problem that greatly affects the geriatric population. We reviewed pertinent published data in the literature concerning the prevention and treatment of pressure ulcers.

The development of pressure ulcers is associated with well known risk factors including unrelieved pressure, skin maceration, shear forces, malnutrition and immobility. Risk factor modification is an important aspect in prevention and treatment. When a pressure ulcer develops, various specialized support surfaces and wound care products exist to accelerate wound healing. Alternative therapeutic modalities such as ultrasonic mist and wound vacuum therapy are increasingly being used with success for such ulcers.

Pressure ulcers are typically the consequence of underlying medical conditions that should be treated appropriately in order for the wound to heal.

INTRODUCTION

Pressure ulcers are a significant health care problem for institutionalized patients, especially for the geriatric population [1]. Data from the United States indicates that about 11% of all hospital admissions and up to 25% of persons in skilled care and nursing home facilities have pressure ulcers [3-5]. In certain high-risk populations such as elderly patients with femoral fractures or hospitalized patients with quadriplegia, the incidence of pressure ulcers is over 60% [6-9].

The national cost of treating pressure ulcers in the United States has been estimated at over $ 1.3 billion with the median cost of caring for a patient with a pressure ulcer in an acute care setting being $ 27,000 [2]. Pressure ulcers carry a high level of morbidity for the patient, causing pain, suffering, and prolonged hospitalization. Frustration for the patient, family and caregivers always accompany the development of such ulcers.

Pressure ulcers continue to be perceived as a sign of sub-optimal health care, contributing to forge a bad reputation for involved hospitals and healthcare facilities often despite clearly unavoidable risk factors [2].

Studies have shown a clear association between development of pressure ulcers and increased mortality [10-11]. Although the presence of a pressure ulcer is associated with a two- to fourfold risk of dying [10-11], this increased mortality is generally attributable to underlying illness and poor functional status rather than the ulcer itself [10].

DEFINITION

Pressure ulcers are localized areas of tissue necrosis that tend to occur when soft tissue is compressed between a bony prominence and an external surface for a prolonged period [4]. Pressure ulcers are referred to by various names including decubitus ulcers, bedsores, and pressure sores. Because pressure is the main causative factor in the development of these ulcers, the term "pressure ulcer" is recommended [4]. Pressure ulcers may occur over any bony prominence of the body, common places include the sacrum, trochanter, heels, knees, ankles, elbows, shoulder blades, occiput, etc. Sixty percent of all pressure ulcers occur on the lower half of the body; in supine patients pressure ulcers occur most frequently over the sacrum, while the ischial tuberosities are most commonly affected in the seated position [4, 12].

CLASSIFICATION

Over the years, numerous systems have been developed to grade or classify pressure ulcers [12-13]. In 1989, the National Pressure Ulcer Advisory Panel sponsored a national consensus conference, during which several commonly used staging systems were combined in an attempt to implement a universally accepted classification system [4]. The following staging definitions were proposed:

- **Stage I**: nonblanchable erythema of intact skin, considered the heralding lesion of skin ulceration.
- **Stage II**: partial-thickness skin loss that involves the epidermis or dermis (or both). The ulcer is superficial and manifests clinically as an abrasion, blister, or shallow crater.
- **Stage III**: full-thickness skin loss and damage or necrosis of subcutaneous tissue that may extend to, but not through, underlying fascia. The ulcer manifests clinically as a deep crater, with or without undermining of adjacent tissue.
- **Stage IV**: full-thickness skin loss associated with extensive destruction, tissue necrosis, or damage to

*Physical Medicine and Rehabilitation, American University of Beirut - Medical Center, Lebanon.


e-mail: md34@aub.edu.lb Tel.: +961 70 104154
muscle, bone, or supporting structures, such as tendons or joint capsules.

Wounds covered by an eschar such that their real depth cannot be exactly evaluated are temporarily classified as unstageable until debridement is performed and the wound extent properly determined.

PATHOPHYSIOLOGY AND RISK FACTORS

External pressures applied to the skin result in tissue breakdown by causing the collapse of capillaries in the area of affected tissue. It is generally accepted that external pressures above 25-32 mmHg will close down the capillary walls. There is an inverse relationship between time and pressure in the formation of pressure ulcers [14]. The body can withstand high pressures for short time periods and low pressures for longer time periods.

In our daily life we commonly encounter situations where the high pressure applied on our skin would cause an ulcer to develop if that pressure was not relieved. However, a healthy individual is able to perceive the pain caused by the local ischemia and relieve the pressure by shifting his position. A pressure ulcer will develop when an individual either does not perceive this ischemic pain signal (impaired sensation, impaired consciousness) or is physically unable to shift position (immobility). Studies have shown that an external pressure of 70 mmHg for two hours will result in irreversible tissue damage [14]. Tissue interface pressures between a standard hospital mattress and different bony prominences of a supine individual either does not perceive this ischemic pain signal (impaired sensation, impaired consciousness) or is physically unable to shift position (immobility). Studies have shown that an external pressure of 70 mmHg for two hours will result in irreversible tissue damage [14].

Tissue interface pressures between a standard hospital mattress and different bony prominences of a supine patient (occiput, shoulders, sacrum, heels, etc.) have been measured in excess of 70 mmHg. Many hospitals have therefore adopted a policy of two-hourly turning schedules for patients who are unable to shift their position in response to pressure.

Different types of tissues and cells have variable tolerances for ischemia and pressure. For example, muscle tissue is more sensitive to ischemia than the tissues of the skin [15]; therefore by the time the skin shows visible signs of pressure damage, the underlying muscle tissues may already be necrotic. As external pressure from a surface is transmitted from the skin to the underlying bone, the bone exerts a counter pressure and all tissue layers in between are compressed to varying degrees. The highest pressure exists at the bone-soft tissue interface; this pressure diminishes as it reaches the skin [14-15]. Therefore, the earliest and greatest damage occurs in the underlying tissues. It is estimated that 70% of a pressure ulcer lies beneath the skin—this is referred to as the iceberg effect. For this reason, pressure ulcers are often under-staged initially and should always be assessed for undermining.

While pressure is a major causative factor in pressure ulcer development, other forces and factors play a role as well. Friction and shear forces, excessive skin moisture or dryness, malnutrition, immobility, and impaired patient mental status also contribute to tissue breakdown [14].

Friction is caused by the skin rubbing against linens or another surface. Friction is commonly encountered in patients who are unable to lift themselves and are inadvertently pulled or dragged across the bed when the healthcare worker or family member repositions them. By itself, friction can cause damage to the epidermis and upper portion of the dermis and result in superficial abrasions.

Shear forces are caused by the relative displacement of adjacent structures against each other. Typically the skin sticks to the bed sheet while the deeper tissues are pulled by gravity or a moving force. This causes angulation, stretching, twisting, or even tearing of capillaries in the affected area, which leads to disruption of blood flow, ischemia, and cellular death [15]. Shearing forces most commonly occur when the head of the bed is elevated greater than 30 degrees and the patient slides down. Additional shearing and friction damage can be inflicted if caregivers use improper lifting techniques and drag the bedridden patient up to the head of the bed or across the surface of the bed.

Moisture, most commonly in the form of incontinence or diaphoresis, can macerate or waterlog the skin, making it softer and predisposing it to breakdown. Moisture also exacerbates friction and shear because the moist skin sticks to the bed surface. The other extreme, excessive dryness, is also a contributing factor to pressure ulcer development [14, 4].

Certain conditions significantly predispose a patient to tissue breakdown. Patients with diabetes are often at risk due to circulatory and neurological impairment. In addition, once a patient with diabetes develops a pressure ulcer, he or she may experience delayed healing [16]. Any circulatory impairment will increase risk, since pressure ulcer development is an ischemic event. Spinal cord injured patients are prime candidates for tissue breakdown, due to immobilization and diminished sensation [16]. Another often-overlooked category of patients affected by immobilization may be healthy patients undergoing lengthy surgery.

The elderly are a group at high risk, due to the naturally occurring changes in the skin and supporting structures brought on by advancing age [15-16]. Aging is associated with a loss of lean body mass and subcutaneous tissue, decreased protein stores, lessened tissue elasticity, and reduced cohesion between the epidermis and dermis [18]. The elderly are also more likely to have circulatory insufficiency. Therefore, if a wound develops in an elderly person, healing capabilities are diminished. Similarly, malnutrition decreases skin tolerance for pressure and is associated with poor wound healing when the pressure develops [15].

PREVENTION

Prevention is the first step in meeting the challenge of pressure ulcers. Each patient entering the healthcare facility should be assessed for his or her relative risk of developing a pressure ulcer. Assessment should be standardized, consistent and quantitative [6]. Many risk assessment tools have been developed to systematically
rate patients’ potential for pressure ulcer development. The Norton Scale was the first of these tools to become widely used in the United States [6]. Others have been introduced and researched in an attempt to find the perfect method of determining risk to prevent pressure ulcers [19-20]. The Braden Scale is gaining wide acceptance because it has undergone extensive testing in critical care as well as in acute and extended care settings. It is composed of six subscales or risk factors: sensory perception, skin moisture, physical activity, mobility (ability to change and control body position), nutrition, and friction and shear [20].

Designating a person “at risk” is of no value unless intervention is implemented in response to the risk. These strategies have been formalized by the US Department of Health and Human Services that issued clinical guidelines for the prediction and prevention of pressure ulcers. The guidelines may be found on the Agency for Health Research and Quality website: www.ahrq.gov. An essential part of prevention of pressure ulcers is education of healthcare providers, patients and family. Education should include all aspects of pressure ulcer prevention, staging, and treatment.

TREATMENT

The treatment of pressure ulcers has evolved into a comprehensive individualized approach, involving more than simple management of the wound itself. It is obvious that pressure ulcers are a visual manifestation of one or more underlying medical conditions, all of which need to be addressed. In caring for a patient with a pressure ulcer, caregivers should take into account the overall goal of the treatment. If the goal of treating a pressure ulcer is to restore function then aggressive treatment is warranted. However if the goal is to provide comfort and relieve suffering, less aggressive measures may be indicated [24]. Treatment principles should include optimization of wound healing environment along with reversal of risk factors (if possible) by trying to relieve pressure, avoid skin maceration, optimize nutritional status, and improve mobility [25].

1. Wound care

The removal of devitalized tissues (debridement) is often an essential part of pressure ulcer wound care. In fact pressure ulcers often present with a necrotic central area. This necrotic tissue delays wound healing by inhibiting epithelialization and granulation tissue formation. Debridement can be achieved by multiple methods depending on the amount and nature of the necrotic tissue and the patient’s overall status. Surgical debridement is the most expedient method but requires special expertise and sterile procedures. Some patients may not be candidates for surgical debridement. Sharp debridement using a scalpel or scissors may be performed in an outpatient setting, but also requires specific training. Mechanical debridement via the use of wet-to-dry dressings, whirlpool, high-pressure irrigation, or scrubbing with gauze or a sponge is easier to perform but is less selective and may damage viable tissue. Enzymatic debridement uses enzymatic agents to break down necrotic tissue; however they may be harmful to adjacent normal tissues hence requiring skin protectant such as zinc oxide or petroleum jelly. Finally, autolytic debridement through the use of moisture retentive dressings to retain endogenous enzymes at the wound surface to digest devitalized tissues is the most selective, least invasive, albeit slowest method.

A moist wound environment is essential to accelerate healing by promoting granulation and reepithelialization [26]. Appropriate use of dressings to maintain a moist wound environment involves careful assessment of the wound to determine what functions are needed from the dressing. Hundreds of dressings exist; these dressings were developed to modify the wound environment, in an effort to facilitate healing. The choice of dressing depends on the stage of the ulcer, its location, the amount and quality of the exudate, the presence or not of an infection, as well as the characteristics of the wound base and wound margins. The general rule is to keep the wound base moist and avoid dessication, however excessive moisture will also delay healing. Pressure ulcers generally tend to have moderate to high levels of exudate and in order to avoid peri-wound maceration, absorbent dressings such as foams or alginates may be appropriate [27-29]. During the later stages of healing, the exudate may taper off and a hydrocolloid or transparent film dressing may be adequate to provide protection and maintain a moist environment [27-28, 30]. For a dry and desiccated ulcer, a hydrating dressing such as an amorphous or sheet hydrogel may be appropriate. For infected ulcers, dressing products containing antimicrobial agents such as silver or iodine are used. Activated charcoal dressings are available for malodorous ulcers.

The direct topical application of disinfecting agents to the wound surface of a pressure ulcer is controversial. The use of topical antimicrobial agents such as silver sulfadiazine for treating pressure ulcers is borrowed from experience in managing patients with burns, whose wounds frequently become secondarily infected. The mechanism of ulcer formation differs considerably in pressure ulcers as opposed to thermal burns. Because the rate of infection, causative organisms, and mechanism of infection differ, one might not expect the treatment to be the same. Irrigation with saline can retard bacterial growth in open pressure ulcers [24]. The topical use of disinfecting agents may actually be counterproductive. Povidone-iodine, acetic acid, hydrogen peroxide, and sodium hypochlorite are cytotoxic to fibroblasts and may impair wound healing [31]. Topically applied antimicrobial agents such as silver sulfadiazine can decrease bacterial counts; however, use of such agents may result in selection of resistant organisms [32]. In general, topically applied antibiotics do not penetrate deeply into the ulcer and may cause hypersensitivity, contact dermatitis, maceration of the surrounding
skin, and systemic toxicity from drug absorption [24]. Topical application of antibiotics or antiseptic solutions is not routinely recommended. If topically applied antibiotics are used, the duration of therapy should be limited to 7 to 10 days, to prevent the selection of resistant organisms.

2. Support surfaces and repositioning

Since the most important cause of pressure ulcers is unrelieved pressure, support surfaces and repositioning of the patient play a paramount role in the treatment of pressure ulcers. Specialty support surfaces (beds, mattresses, mattress overlays, and wheelchair cushions) that reduce the tissue interface pressure should be utilized when pressure ulcers already exist or when the patient is at high risk for development of pressure ulcers. However, the use of a pressure reducing support surface does not replace the need to turn or reposition the patient or the use of pillows or foam wedges to keep bony prominences apart (knees, ankles, etc.) [33]. Whether using positioning techniques or support surfaces, the goal is to redistribute pressure from a bony prominence to the larger surrounding body surface.

2.1. Patient positioning techniques

Patients who are confined to bed should be repositioned at least every 2 hours while chair-bound patients should be repositioned at least every hour [33]. Simple measures such as pillows placed beneath the calves to suspend the heels and pillows placed between the knees or the legs to separate bony prominences when the patient is lying on his side have been shown to reduce the risk of development of pressure ulcers [6, 33].

2.2. Support surfaces

High risk patients should always be placed on a pressure reducing mattress overlay or a chair cushion. Donut type devices should not be used as they create a very high-pressure area around the open central area. Thick foam mattresses, water mattresses, alternating-pressure air mattresses, and static multilayered air mattresses have been found to be useful in the prevention of pressure ulcers [34-35]. Their usefulness in ulcer healing is not as certain, and no one product has been shown to be clearly superior. Low-air-loss beds and air-fluidized beds, which are elaborate support surfaces, probably are the most effective for functionally dependent patients with large, deep, or multiple ulcers.

Low-air-loss beds consist of multiple inflatable fabric pillows that are attached to a modified hospital bed frame. An electric fan maintains the buoyancy of the pillows [36]. The patient is carried by these air pillows in which the pressure can be adjusted to support the different body areas. The use of low-air-loss beds for nursing home patients with pressure ulcers was associated with a threefold increase in the rate of wound healing in comparison with patients in the same facility treated with foam mattresses [36]. An air-fluidized bed (Clinitron bed) is an oval space with up to 900 kg of tiny silicone beads covered by a polyester sheet. The beads are fluidized by a flow of warm, pressurized air, which floats the polyester cover on which the patient is placed. The patient’s body fluids are able to flow through the polyester sheet; thus, the skin is kept dry [37]. In addition to being heavy, these beds are very expensive. The circulating warm air tends to make the bed hot, which can be uncomfortable to the patient. Most studies in which air-fluidized beds were used have shown faster rates of wound healing than with conventional treatment [37-38]. However because of the need for prolonged treatment of pressure ulcers and the high cost of air-fluidized beds, they have been recommended for use in limited, specific cases such as medically stable patients whose prognosis is otherwise good [38].

2.3. Nutrition

Clinical dietitians play an essential role in the multidisciplinary team caring for the patient with a pressure ulcer. Adequate nutrition is important for normal wound healing. Experts do not agree on how much patients with pressure ulcers should be fed or whether vitamin supplementation is beneficial. The effects of several nutrients on pressure ulcer development and healing have been evaluated. Zinc and ascorbic acid are micronutrients necessary for synthesis and stabilization of proteins; however, the published literature presents no convincing evidence that zinc or ascorbic acid supplementation improves healing [15]. Nonetheless, a standard multivitamin supplement may theoretically be beneficial and is safe for virtually every patient.

“Overfeeding” malnourished patients with pressure ulcers has also been much debated. Although there is some evidence that a high calorie, high protein diet may accelerate wound healing in malnourished patients [39], such diets may be associated with serious metabolic complications such as elevated blood urea nitrogen (BUN), electrolyte disturbances and edema [40-41]. Consequently, a conservative approach seems prudent. Patients should be weighed daily, to monitor for inadequate nutrition or accumulation of fluid. Intake of calories should be recorded for at least one 24-hour period. A diet composed of 25 to 30 kcal/kg of body weight, with 1 to 1.5 g of protein per kilogram of body weight, is adequate for most patients [41].

INCONTINENCE MANAGEMENT

Management of incontinence may be critical to the healing of pressure ulcers. Fecal and urinary contamination of the wound can cause continued maceration and lead to infection. Whenever possible, incontinence should be evaluated, in an effort to enhance management. Incontinence may be compounded by physical restraints, confusion, medications (such as diuretics), and hyperosmolar tube feedings (which can result in urinary or fecal urgency). Urinary incontinence can be managed with a program of
scheduled voiding every two hours and scheduled intake of fluids. Intermittent catheterization or an indwelling urinary catheter may also be necessary in some patients. Condom catheters are another option for incontinent men.

SURGICAL MANAGEMENT OF PRESSURE ULCERS

Surgical repair of pressure ulcers may be considered when the ulcer has not responded to optimal conservative care and debridement. Surgical options include direct closure, skin grafts, skin flaps, muscle flaps, or free flaps. The patient must be medically and nutritionally stable, and able to withstand both the surgical and postoperative immobility.

ADJUNCTIVE THERAPIES

Topically applied platelet-derived growth factors and fibroblast growth factor have been used successfully to promote healing of chronic pressure sores [42-43]. Various other innovative techniques and products, each with strong advocates, are also available or being developed. Hyperbaric oxygen, electrical stimulation, vacuum assisted wound closure, and ultrasonic mist treatment have all shown good results in pressure ulcer management [44-47].

CONCLUSION

Elderly patients are at high risk for developing pressure ulcers, especially during hospitalization. Pressure ulcers are often associated with both high morbidity and significant cost. Their incidence has been linked to the quality of care in healthcare facilities. Prevention is a key factor in the fight against pressure ulcers even though implementing the preventive measures can be personnel demanding and time consuming. Pressure relief is the single most important preventive and therapeutic measure that should be implemented. Wound care, nutritional support, along with newly developed therapeutic options are all part of the treatment arsenal from which the clinician should choose judiciously to provide the best and most suitable treatment plan according to the patient’s overall medical and social condition.

REFERENCES


