Pressure ulcers

Pressure ulcers are localized areas of tissue necrosis that tend to develop when soft tissue is compressed between a bony prominence and an external surface for a prolonged period of time.

The consequences of pressure-induced skin injury range from nonblanchable erythema of intact skin to deep ulcers extending down to the bone.

The common pathway that results in necrosis of the skin is an unrelieved application of pressure to susceptible tissues for a prolonged period of time. The resulting ulcer imposes a significant burden not only on the patient but on the entire healthcare system.

The staging, epidemiology, pathogenesis, and clinical manifestations of pressure ulcers will be reviewed here. Prevention and treatment are discussed separately.

Staging

A number of staging systems have been developed to describe the extent of pressure ulcers. The most commonly used system, proposed by the National Pressure Ulcer Advisory Panel, is described as follows:

Stage 1

Stage 1 is characterized by an observable pressure-related alteration of intact skin which, when compared to an adjacent or opposite site area on the body, may include changes in one or more of the following: skin temperature (warmth or coolness); tissue consistency (firm or boggy feel); and/or sensation (pain, itching).

The ulcer appears as a defined area of persistent redness in lightly pigmented skin; in darker skin tones the ulcer may appear with persistent red, blue, or purple hues.

Stage 2

Stage 2 is characterized by a partial thickness skin loss involving the epidermis and/or dermis. The ulcer is superficial and presents clinically as an abrasion, blister, or shallow crater.

Stage 3

Stage 3 is characterized by full-thickness skin loss involving damage or necrosis of subcutaneous tissue which may extend down to, but not through the underlying fascia. The ulcer presents clinically as a deep crater with or without undermining of the adjacent tissue.

Stage 4

Stage 4 is characterized by full-thickness skin loss with extensive destruction, tissue necrosis, or

damage to the muscle, bone, or supporting structures.

These staging systems primarily describe ulcer depth. Wounds also may be characterized on the basis of features such as size, the presence of necrotic tissue, and the undermining of tissue.

Healing scales

Staging systems are also commonly used to monitor and describe pressure ulcers that are healing. However, this practice, known as reverse staging, is not recommended. Healing ulcers do not progress serially from one stage to the next lowest. Instead, they heal through a process that includes granulation, wound contraction, reepithelialization, and scar formation.

The healing process is better described by scales that capture changes in surface area, the extent of necrotic tissue and exudate, and the presence of granulation tissue.

A number of scales have been advocated for use in monitoring the healing of pressure ulcers, including the Pressure Sore Status Tool (PSST), the Pressure Ulcer Scale for Healing (PUSH), the Sessing Scale, and the Wound Healing Scale.

Given the differences among these staging systems, the scale used should be explicitly described to facilitate effective communication among clinicians responsible for patients with a pressure ulcers. Training in the use of these scales is also required to ensure that ulcers are staged in a consistent manner.

Pathogenesis of pressure ulcers

The development of a pressure ulcer is a complex process that requires the application of external forces to the skin. However, external forces alone are not sufficient to cause an ulcer; their interaction with host-specific factors culminates in tissue damage.

External Factors

External factors that lead to the development of pressure ulcers include pressure, shearing forces, friction, and moisture.

Pressure

Pressure applied to the skin in excess of the arteriolar pressure (32 mm Hg) prevents the delivery of oxygen and nutrients to tissues, resulting in the accumulation of metabolic waste products. Pressures are greatest over bony prominences where weight-bearing points come in contact with external surfaces.

A patient lying on a standard hospital mattress may generate pressures of 150 mmHg; sitting produces pressures as high as 300 mmHg over the ischial tuberosities.

Pressure in excess of 70 mmHg for two hours results in irreversible tissue damage in animal models. Ulcer formation occurs more rapidly with higher pressures, and intermittent relief of pressure prevents tissue damage.

Pressure over a bony prominence tends to result in a cone-shaped distribution with the most affected tissues located deep, adjacent to the bone-muscle interface. Tissues vary in their susceptibility to pressure-induced injury; muscle is the most susceptible, followed by subcutaneous fat and then the dermis.

Thus, extensive deep tissue damage may occur with little or no evidence of superficial tissue injury. A deep necrotic wound may be the first evidence of pressure-induced injury, rather than a gradual progression of an ulcer from stages 1 through 4.

Shearing forces

Shearing forces occur when patients are placed on an incline. Deeper tissues, including muscle and subcutaneous fat, are pulled downwards by gravity, while the superficial epidermis and dermis remain fixed through contact with the external surface.

The result is stretching and angulation of local blood vessels and lymphatics. Shear forces alone may not cause ulceration, but appear to have an additive effect so that in the presence of pressure, more severe tissue damage will occur.

Friction

Friction occurs when patients are dragged across an external surface. This results in abrasion with damage to the most superficial layer of skin. Friction is most likely to result in stage 2 pressure ulcers since it does not cause the necrotic changes associated with deep tissue injury; it has only a limited contribution to the development of stage 3 and 4 ulcers.

Moisture

Exposure to moisture in the form of perspiration, feces, or urine may lead to skin maceration and predispose to superficial ulceration. There are little data regarding the magnitude of the contribution of moisture to pressure ulcer development; some have questioned whether a plausible mechanism exists by which moisture leads to the deep tissue necrosis characteristic of stage 3 or 4 ulcers.

Host factors

A number of host factors may contribute to pressure ulcer development including immobility, incontinence, nutritional status, circulatory factors, and neurologic disease.

Immobility

Immobility is one of the most important host factors that contribute to pressure ulcer development. Immobility may be permanent (eg, due to spinal cord injury) or transient (eg, during an acute medical illness or from the use of sedatives). There is a high correlation between a lack of spontaneous nocturnal movements and pressure ulcer development in studies using devices that measure body movement.

However, methods to measure immobility in clinical settings are generally not available. Thus,

investigators have often relied upon other clinical characteristics as markers for immobility and risk for pressure ulcer development, including functional measures (eg, whether patients are able to ambulate or are bed- or chair-bound) and diagnostic information (eg, a history of a cerebrovascular accident)

Incontinence

Urinary incontinence is frequently cited as a predisposing factor for pressure ulcers. Some studies suggest that incontinent patients have up to a five-fold higher risk for pressure ulcer development

However, these studies have generally not considered the strong correlation between incontinence and immobility. A national survey of nursing home discharges, for example, found that 94 percent of incontinent pressure ulcer patients were bed- or chair-bound.

Urinary incontinence often does not remain as an independent predictor of ulcer development when statistical analyses are performed to account for this correlation. Several studies have suggested that fecal incontinence is a predictor.

Nutritional status

The role of nutritional status in the development of pressure ulcers is uncertain. Animal studies have found that more severe pressure-induced skin destruction occurred in malnourished animals than in well-nourished animals exposed to similar amounts of pressure.

In addition, cross-sectional studies have suggested that patients with pressure ulcers are more likely to have hypoalbuminemia

A wide variety of other nutritional measures have also been examined in prospective studies with inconsistent findings. One study found that lymphopenia was associated with ulcer development in acute hospital patients, but this was not confirmed in long-term care settings.

The strongest nutritional measure predicting pressure ulcer development may simply be whether the patient has adequate dietary intake. It is not clear if interventions to increase dietary intakes, such as initiation of tube feedings or use of dietary supplements, can prevent pressure ulcers; this is awaiting evaluation in well-designed, randomized clinical trials.

Circulatory factors

The role of circulatory factors in the development of pressure ulcers has been increasingly recognized. Some authors have suggested that "pressure sores are not irremediable afflictions of long-stay patients but a sign of acute illness".

Contributing factors to the development of tissue ischemia have been postulated to include hypotension, dehydration, vasomotor failure, and vasoconstriction secondary to shock, heart failure, or medications.

- Several studies have demonstrated an