REVIEW ARTICLE

Bedsore: Epidemiology; Risk Factors; Classification; Assessment Scales and Management

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Bedsore is a very important problem in bedridden patients. It affects patients' lives and imposes substantial costs to society. Its incidence approaches 38% and its annual prevalence is estimated to be 14.8% in patients who are admitted in hospitals.
The National Pressure Ulcer Advisory Panel (NPUAP) and the European Pressure Ulcer Advisory Panel (EPUAP) define bedsore as "a localized injury to skin and/or its underlying tissues as a result of pressure, shear or a combination of those and usually present over a bony prominence.
Proposed mechanisms for the development of pressure ulcers include friction or shear force over the skin. Several scales have been introduced in clinical studies in order to assess the risk for development of bedsores. Four of the most important scales are Braden scale, Norton scale, Waterlow scale and Cubbin & Jackson scale.
Based on literature review it seems that, using appropriate dressings, repositioning the patient, optimizing nutritional status, and moisturizing sacral skin are best ways for management and prevention of pressure ulcers.
Keywords: bed sore; epidemiology; classification; assessment scales

Bedsore is one of the most dominant problems reported by patients with mobility limitations [1]. Sometimes, it can even be life threatening, and its treatment imposes financial burdens on patient's family and society. Bedsore, also known as pressure ulcer, pressure sore or decubitus ulcer has been recognized as a disease entity for many years. They have been found in Egyptian mummies, some of which are more than 5,000 years old [2]. In 460-370 B.C, Hippocrates described bedsore in association with paraplegia in the presence of bladder and bowel dysfunction [2]. In the 16th century, Ambrose Pare, a French surgeon, described pressure ulcer as a disease with difficult treatment which could be treated with resting, exercise and adequate nutrition. In 1777, Wolleben studied the problem and emphasized that pressure ulcer can be treated only with long-term lying. Then in 1944, Groth described that local tissue ischemia is not caused only by external pressure, but also by tissue damage [1]. Pressure ulcers are one of the most radical conditions in critically ill patients [3]. The development of bed sore creates a major problem that accompanies excessive pain and suffering in affected patients [4].

Cost estimates for bedsore treatment ranges from $37 800 to $70 000 per ulcer, with total costs in the United States being as high as $11 billion annually [5-6] and 4% of the total health care costs in Great Britain – between 1.4 and 21 billion Pounds – has been attributed to bedsore care [1].

According to the new definition by the National Pressure Ulcer Advisory Panel (NPUAP) and European Pressure Ulcer Advisory Panel (EPUAP), a pressure ulcer is a localized injury to the skin and/or its underlying tissue as a result of pressure, shear or a combination of those, and usually present over a bony prominence [7].

Epidemiology

The 5th National Pressure Ulcer Prevalence Survey conducted in the USA in 1999 found an annual prevalence rate of 14.8% among patients who were admitted in hospitals, and mostly in the Intensive Care Units and among patients aged 70-79 years. In another study, incidence rates of 0.4% to 38% were reported in inpatient departments while prevalence has been reported as 3.5% to 69% [8-9].Some studies in the USA indicate that the prevalence varies from 10 to 18% in critical environments and from 0 to 29% in home care [8]. Long-term setting prevalence was 11-30%. Annual prevalence in neurologically damaged patients was 78%, but the long-term risk of pressure ulcers was 25-85% [10].

Etiology

Pressure ulcers are caused by unrelieved pressure, friction or shear force over the skin and underlying tissue applied with great force over a short period or with less force over a longer period, that disrupts blood supply to the capillary network, impeding blood flow and depriving tissues of oxygen and nutrients. This external pressure must be greater than the arterial capillary pressure to lead to inflow

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impairment and resultant local ischemia and tissue damage [11-13]. Moreover, Delisa and Mikulic have noted that the visible ulcer represents only the tip of the iceberg or the apex of the lesion and that muscle is more sensitive than skin to ischemia caused by pressure [14].

If the pressure persists for a longer time (approximately 2h) and has sufficient power, it leads to damage and necrosis of cells and tissues by compromising the blood and lymphatic flow. Consequently, insufficient levels of oxygen and nutrients will be available and on the other hand harmful products of metabolism will accumulate.

Expected capillary pressure ranges are between 10 and 30 mmHg. Tissue hypo-perfusion occurs when the interface pressure exceeds capillary pressure.

Risk factors of pressure ulcers are listed in (Table 1).

Table 1- Risk Factors for Pressure Ulcers

Intrinsic factors	Extrinsic factors
Limited mobility	Pressure from Any hard surface (bed, wheelchair, stretcher)
CVA, Spinal cord injury	Friction from patient's inability to move well in bed
Progressive neurologic disorders (Parkinson, Alzheimer, MS)	Shear from involuntary muscle movements
Coma or sedation, Fractures, Postsurgical procedures	Moisture
Pain, Arthropathies, Poor nutrition, Poor dentition	Bowel or bladder incontinence
Dehydration, Weak sense of smell or taste	Excessive perspiration
Comorbidities, vascular disorders, ESRD	Wound drainage
Peripheral vascular disease	
Malignancies, Depression or psychosis	
Decreased pain sensation, Diabetes mellitus, COPD	
Dementia, CHF, Vasculitis	
Immunodeficiency or use of corticosteroid therapy	
Aging skin, Changes in dermal PH, Flattening of rete ridges	
Loss of elasticity, Decreased cutaneous blood flow	
Decreased dermal epidermal blood flow	
Loss of subcutaneous fat	

Reducing risk from pressure

Best strategies for care of patients at risk for pressure ulcers have their emphasis on reduction of the effects of intrinsic factors (such as poor nutrition, concomitant disease, or dry skin) as well as extrinsic factors (such as shear stress and friction, or incontinence) [15]. Removal of pressure or its redistribution by spreading weight over a wider surface area reduces the risk of pressure sores (Table 2).

Table 2- Methods of pressure redistribution [16-17].

Pressure redistribution	
Increased contact area reduces interface pressure	Pressure relief removes pressure from vulnerable area
WE MUST DO…	WE MUST DO…
* Patient repositioning to increase contact area e.g. 30˚ tilt position	* Patient repositioning to remove pressure from a particular anatomical location
* Reactive support surface e.g. foam, gel or air filled, air fluidized	* Active support surface e.g. alternating pressure
	* Lifting body part clear
	e.g. heel boots

Localization

Pressure ulcer can be localized to any part of the body but the most common sites are:sacral area (above sacrum); hips; ischium;spine; neck; back; scapular margins; ribs; legs (malleoli, heel); patella; arms (elbows, posterior side of the arms); wrist; and head (occiput, ears, face, forehead, nose, chin, cheeks) [1]. Common locations of pressure ulcers are listed in (Table 3).

Table 3- Common locations for pressure ulcers

Supine	Back of head
	Shoulder blade
	Elbow
	Lower back
	Heel
Semisitting	Back of head
	Shoulder blade
	Lower back
	Sacrum
	Heel
Lateral decubitus	Ear
	Shoulder
	Elbow
	Hip
	Between knees and ankles
wheelchair	Shoulder blade
	Lower back
	Hip
	Sacrum
	Under side and back of heel

Classification of pressure ulcers

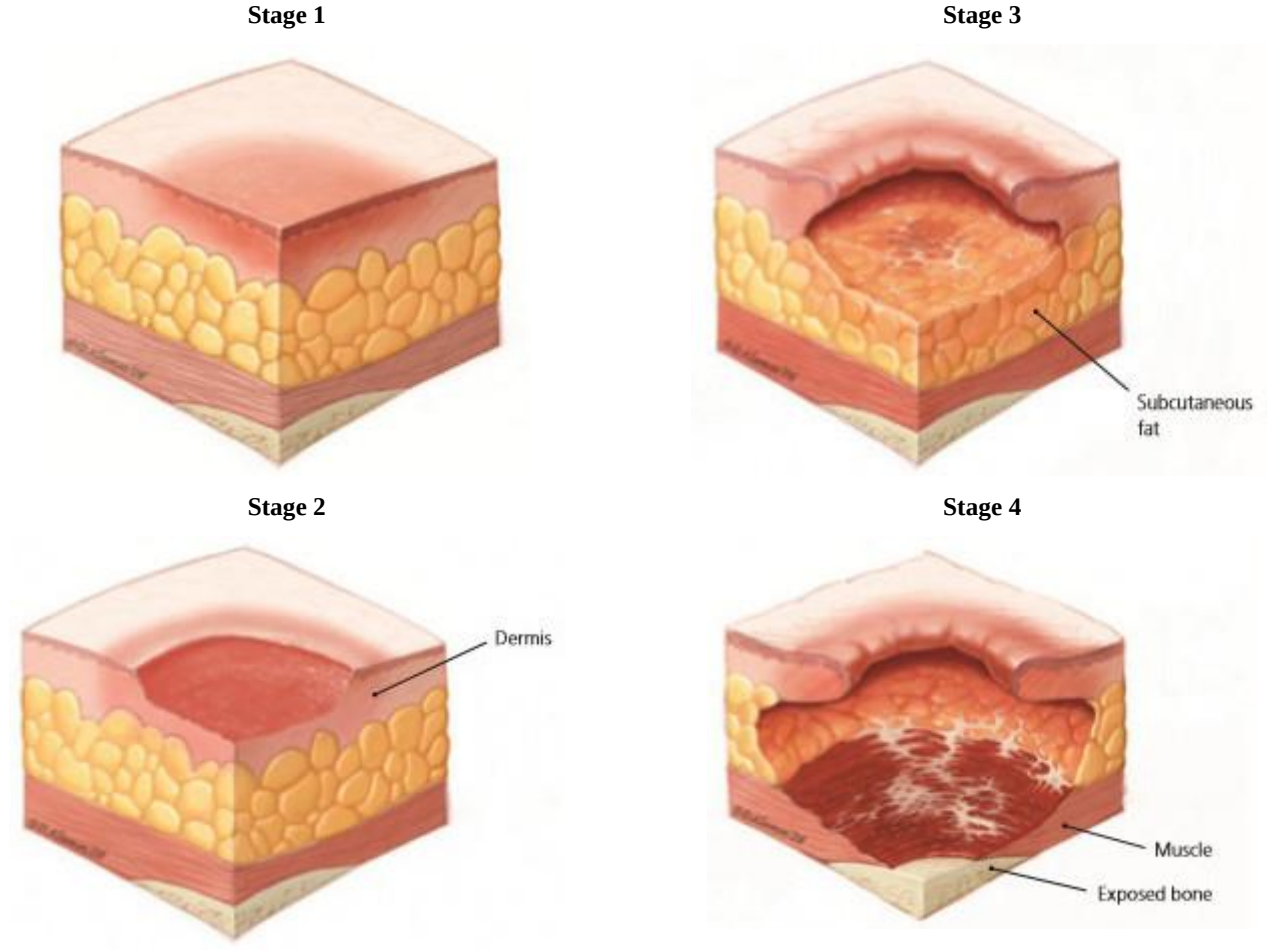
Four stages were described for pressure ulcers that are characterized by the loss of tissue due to molecular destruction. Pressure ulcer stages I to IV and the two additional categories of "suspected deep-tissue injury" and "unstageable" are defined in (Table 4) (Figure 1) [18].

Table 4- NPUAP staging system for Pressure Ulcers

Stage	Description
Suspected deeptissue injury	Purple or maroon localized area of discolored, intact skin or blood-filled blister caused by damage to underlying soft tissue from pressure or shear; the discoloration may be preceded by tissue that is painful, firm, mushy, boggy, or warmer or cooler compared with adjacent tissue
I	Intact skin with non blanchable redness of a localized area, usually over a bony prominence; dark pigmented skin may not have visible blanching, and the affected area may differ from the surrounding area; the affected tissue may be painful, firm, soft, or warmer or cooler compared with adjacent tissue
II	Partial-thickness loss of dermis appearing as a shallow, open ulcer with a red-pink wound bed, without slough; may also appear as an intact or open/ruptured serum-filled blister; this stage should not be used to describe skin tears, tape burns, perineal dermatitis, macerations, or excoriations
III	Full-thickness tissue loss; subcutaneous fat may be visible, but bone, tendon, or muscle is not exposed; slough may be present, but does not obscure the depth of tissue loss; may include undermining and tunneling
IV	Full-thickness tissue loss with exposed bone, tendon, or muscle; slough or eschar may be present on some parts of the wound bed; often includes undermining and tunneling
Unstageable	Full-thickness tissue loss with the base of the ulcer covered by slough (yellow, tan, gray, green, or brown) or eschar (tan, brown, or black) in the wound bed

NPUAP = National Pressure Ulcer Advisory Panel.

Figure 1- Staging of the wound (adopted from References no: 22)



Risk assessment

Several scales have been used to assess the risk for development of pressure ulcers in various clinical settings. A number of studies have examined "pressure ulcer risk assessment scales" in the ICU setting. Several scales have been developed for early screening of patients at higher risk for developing pressure ulcers in the ICU [19]. There is no single scale for risk assessment which can predict all risk factors. Some of the most important scales are Braden scale,

Norton scale, Waterlow scale and Cubbin& Jackson scale.

The Cubbin& Jackson scale was a modification of the Norton scale which was developed and revised specifically for ICU patients, then Cubbin& Jackson scale been have shown to have the best predictive value with 99.3% sensitivity and 55.5% specificity compared to other scores.The most common risk scales used in the United States are the Braden Scale and the Norton Scale. The most common pressure ulcer risk scales used in Britain are the Waterlow and Braden Scales. The Cubbin& Jackson Risk Assessment Score is a pressure ulcer risk tool specific to European critical care units [20-21].

Comparison of the concepts of the risk assessment tools are shown in (Table 5).

Table 5- Comparison of the concepts of the risk assessment tools

Scale	Item	score	Total(min,max)	At risk	Advantages	Disadvantages
Norton	Activity Mobility Incontinence Level of conscousness	Each subscale rated from 1-4	5 to 20	Lower score, higher risk of pressure ulcer formation the score <=14 for patients at risk	highest specificity	
Braden	Activity Mobility Moisture Sensory perception Nutritional status Friction/shear	Each subscale rated from 1-3 or 4	6 to 23	The score for mild risk patients is 15-16, moderate risk 12-14, high risk 11 or below Thus lower score, higher risk of pressure ulcer development	the most widely used ,Higher validity for cardiac surgery ICU, was more reliable in comparison with Waterlow	Not recommended in ICU
Waterlow	Build/weight continence, surgery skin type, appetite, trauma age, mobility, gender, tissue malnutrition neurological deficit specific medications	Each subscale rated from 0-3 or 5-8	<10 to >20	At risk group 10-14 High risk group 15-19 Very high risk group 20 or above Higher score higher risk of pressure ulcer formation, the most sensitive		Not significantly predictive for PrU occurrence, not recommended in ICU
Cubbin and Jackson	Age, mobility, weight, mentalcondition, respiration, generalskin, hemodynamicstatus, nutrition, incontinence, hygiene	Each subscale rated from 1-4	10-40	Lower score higher risk of pressure ulcer development	most effective scale in prediction PrU,higher validity	

Management

The management of pressure ulcers is an interdisciplinary task, which requires a team composed of primary care physicians, dermatologists, infectious disease consultants, social workers, psychologists, dietitians, podiatrists, home and wound-care nurses, rehabilitation professionals and surgeons [22].

In order for the prevention and management of pressure ulcers to be effective, we should maintain tissues capillary pressure at below 30mm Hg, eliminate all of the underlying causes, turn and reposition the patient every two hours, and keep the wound and its surrounding skin clean and free from urine and feces.

Physicians should note the location, size, number, length, width and depth of pressure ulcers and assess for any presence of exudate, sinus tracts, odor, necrosis or eschar formation, tunneling, undermining, infection, healing (granulation and epithelialization) in daily visits, and wound pain assessment should be performed, especially during repositioning, dressing changes and debridement [22]. Necrotic tissue promotes bacterial growth and impairs wound healing, and its scars should be debrided until all necrotic tissue is removed and granulation tissue is present.

Methods of debridement include sharp, mechanical, enzymatic and autolytic debridement.

Physicians and nursing staff should observe the pressure ulcers and measure the size of the ulcers, then they must categorize ulcers with respect to surface area, type of wound tissue and exudate. Then the medical team must compare scores of PUSH tool (pressure ulcer scale for healing) over time look for indications of improvement or deterioration in the process of healing of the pressure ulcer.

Wounds should be cleaned initially and with each dressing change, wound cleaning with antiseptic agents such as acetic acid, povidone-iodine, and hydrogen peroxide should be avoided because they damage the granulation tissue.

Synthetic dressings can reduce healing time and cause less discomfort, and can potentially provide enough moisture for the wound skin. These dressings include transparent films, hydrogels, alginates, foams, and hydrocolloids.

The other components of pressure ulcer treatment are surgical approaches that include direct closure, skin grafts, and skin, musculocutaneous and free flaps, use of growth factors (e.g., platelet-derived growth factor Becaplermin [Regranex]), electromagnetic therapy, ultrasound, and hyperbaric oxygen therapy. However, the role of the last three methods in treatment of pressure ulcer in unclear.

Conclusion

The impact of pressure ulcers on quality of life is significant, considering their effects on physical, psychological, emotional, spiritual, social and financial

dimension of life. Health care professionals involved with pressure ulcer treatment and care at any levels have to recognize the impact of this problem on life quality of patients. Depending on individual characteristics of patients the impact may be significant and long lasting. Therefore, treatment and care should be individualized for each patient.

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