Pressure Necrosis in Geriatric Patients

11

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Contents

11.1 Background

The elderly remain one of the highest at-risk populations for pressure ulcers, with about 70 % occurring in individuals over the age of 70 [1, 2]. The incidence of pressure ulcers in the elderly population varies by care setting, but evidence on risk factors indicates that age will increase the probability of pressure ulcers, particularly in patients with limited mobility [3–5].

11.2 Etiology/Pathophysiology

Pressure ulcers are aptly named because they develop due to pressure. Pressure is a static, direct compressive force on tissue leading to hypoxia of the skin and soft tissue by restricting blood flow. When pressure reaches magnitudes that deform cells, the resulting injury is classified today as deep tissue injury, in that the pressure was applied to the deep tissues (muscle, fascia) and deformed the cells leading to their death [6, 7]. Pressure of less magnitude and of long duration creates tissue ischemia. Ischemia of tissue also leads to necrosis, but the mechanism is due to depletion of oxygen and glucose and accumulation of lactic acid [8].

The time needed to create ischemia in soft tissue and skin which leads to necrosis is elusive. In an ischemic animal models, 70 % of cell viability remained for over 22 h. In contrast, cell deformation which would lead to deep tissue injury was

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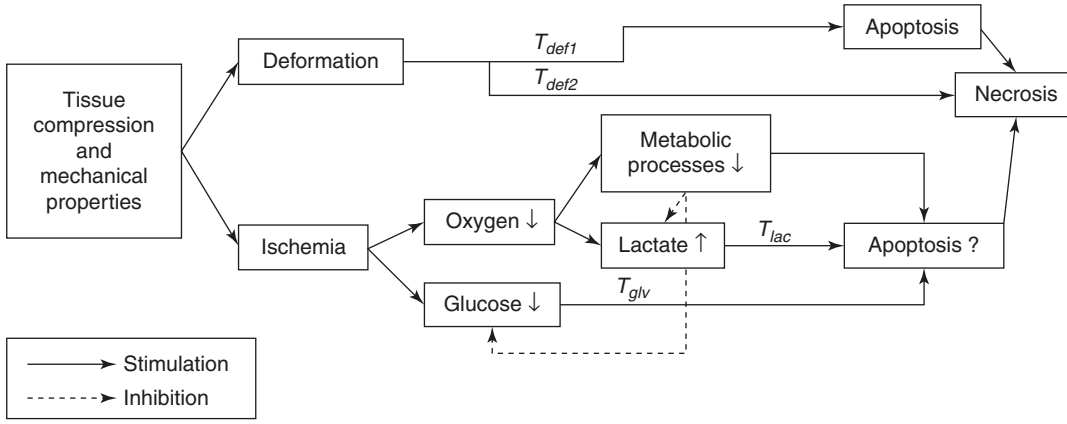


Fig. 11.1 Model of proposed sequence of events leading to necrosis of tissue. Deformation of tissue leads to damage when the threshold is met (T def 1) and cells will start a programmed cell death leading to necrosis. If the deformation of the cell exceeds its tolerance, the cells will die immediately from necrosis. Cells can also be injured from ischemia, reducing the metabolic substrates needed and leading to anaerobic metabolism and accumulation of lactic acid. Both cellular starvation and acidification lead to apoptosis and cellular necrosis (From Stekelenburg [16])

evident within the hour [9] (see Fig. 11.1). Of the various tissues that are at risk of death due to pressure, muscle tissue is damaged first, likely because of its increased need for oxygen and higher metabolic requirements. By the time ulceration is visible in the skin, significant damage of underlying muscle may already have occurred. The tissue fed by the vertical perforators through the muscle remains viable for a while; a series of cases showed the first sign of skin injury from intense pressure was apparent 48 h after the pressure was applied (Black J, The natural history of deep tissue injury pressure ulcers, 2002). An additional finding from this case series is that patients who sustained deep tissue injury were not aware of the ischemia; they were unconscious.

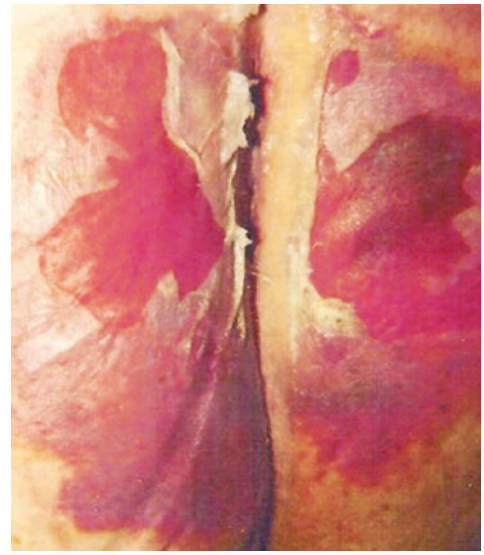
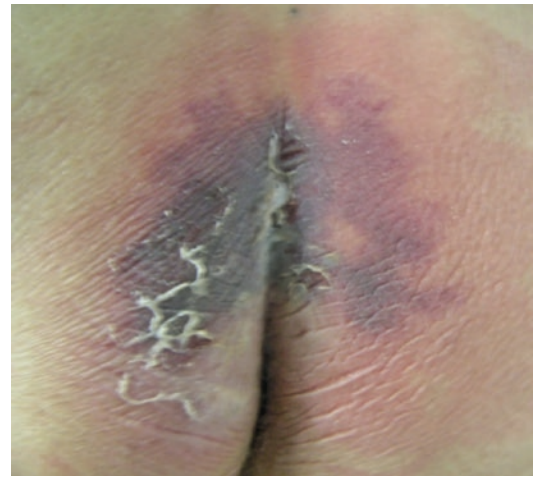
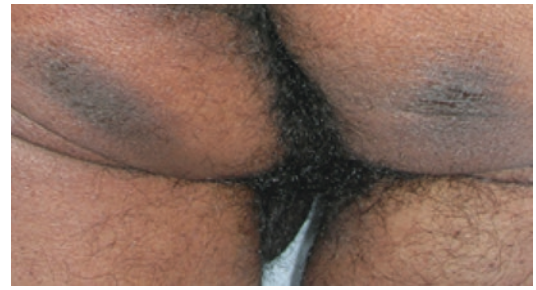
Restoration of blood flow to an ischemic area of tissue, or reperfusion, has recently been suggested as a cause of more damage to the injured area, causing a pressure ulcer to enlarge or fail to heal. One mechanism of harm to tissue may be from reduction in capillary density from repeated loading and unloading along with ischemic insult [10]. The time to develop necrosis is reduced in patients with impaired circulation, such as those with peripheral vascular disease or hypotension.

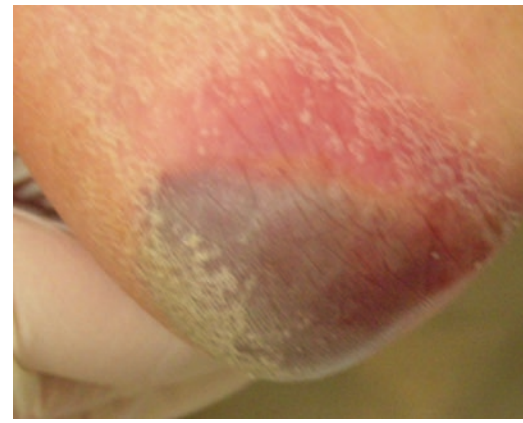
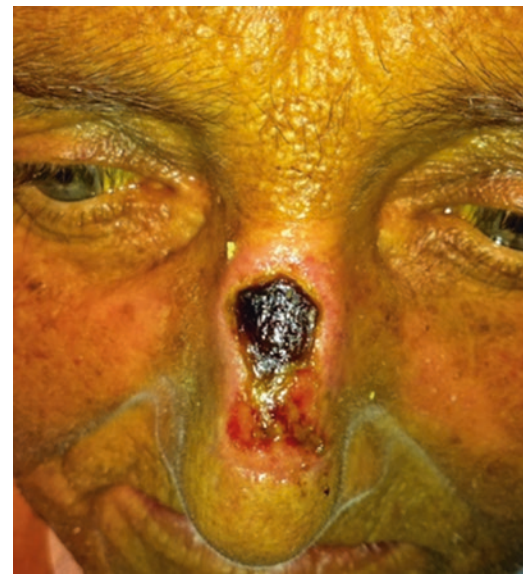
Shear is also a cause of pressure ulcers and undermining in existing ulcers. Shear is a tangential (angular) force associated with movement, for example, sliding down in bed or being pulled over to the side of the bed. Shear forces distort blood vessels in the skin, making the effect of pressure more deleterious because the tissue is already hypoxic. Research has shown that positioning a patient at 45° head of bed elevation leads to the most detrimental combination of pressure and shear on the sacrum, because the shear stresses combined with pressure cause greater obstruction and distortion of capillaries in skeletal muscle around bony prominences than does pressure alone [11].

Microclimate, the moisture and heat of the skin, increases the risk for pressure ulcers because the moisture macerates the skin. The boggy skin does not glide against bed sheets and leads to superficial tissue injury. Skin exposure to urine or stool also injures the skin and increases risk of tissue damage from pressure and shear. Gefen [12] has provided a recent theoretical explanation of the ways in which a changing microclimate may influence the development of superficial pressure ulcers. From a mathematical model, Gefen postulated that four microclimate changes may influence pressure ulcer development – increases in skin temperature, increasing ambient temperature, increasing relative humidity, and decreasing the permeability of sheets or clothing.

11.3 Differential Diagnosis

Pressure necrosis appears on tissue that has been subjected to intense pressure in patients who cannot feel the pressure or respond to it and change positions. High-risk patients and the common locations for pressure necrosis are as follows:

Position	Common location of pressure necrosis	Example pressure necrosis leading to deep tissue injury
Flat in supine position (e.g., during surgery, hypotensive)	Buttocks tissue, unless patient is quite thin, with no buttocks tissue Necrosis appears bilaterally	
Supine with head of bed elevated 30–45° or slouching in a chair	Sacrum and adjacent buttocks tissue	
Sitting erect in chair	Ischial tuberosities	

Position	Common location of pressure necrosis	Example pressure necrosis leading to deep tissue injury
Supine with heels on the bed	Posterior heel in patients with immobile legs, neuropathic legs, or peripheral vascular disease	
Wearing medical devices that are tight	Bridge of the nose from noninvasive positive pressure masks, behind the ears from oxygen tubing, shin, top of foot, and along Achilles tendon from stockings	

Differential Diagnosis

Abscess
Bruising
Cellulitis
Critical limb ischemia
Fournier's gangrene
Hematoma/Morel–Lavellée lesions
Incontinence-associated dermatitis
Ischemia of the skin beneath tightly wrapped dressings
Necrotizing fasciitis
Terminal ulcer
Skin failure

11.4 Prevention

Reducing the duration and magnitude of pressure is paramount. The duration of pressure is reduced by turning the patient off of high-risk areas. Most pressure necrosis develops on the sacrum and therefore immobile patients should be turned to

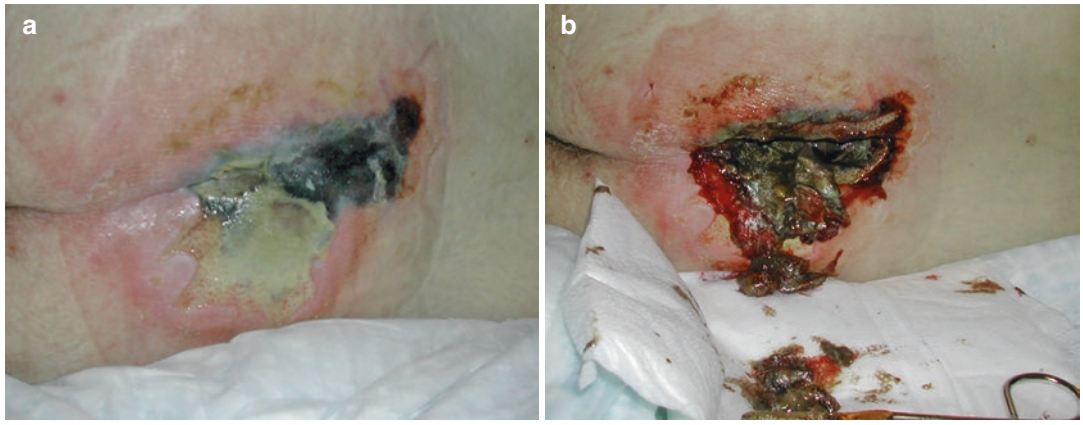


Fig. 11.2 (**a**) This patient is a 65-year-old male who refused to move from his bed at home for several days. When admitted to the hospital, he was septic. Cellulitis and frank necrosis are visible on the sacrum and buttocks. (**b**) His wound was debrided at bedside in the ICU due to the wound being the cause of the sepsis

the side to relieve pressure. The magnitude of pressure can also be reduced by placing the patient on a support surface with adequate envelopment and immersion. High-density foam mattresses have been shown to be effective in reducing pressure injury as long as the patient is moved about in bed. For very-high-risk patients, alternating pressure mattress helps prevent tissue damage; however, the patient still must be moved on these support surfaces. No support surface replaces turning the patient to reduce duration of pressure [13]. Use mattress of 4 in. of viscoelastic foam during times when patients cannot be moved, such as surgical cases over 3 h, cardiopulmonary bypass cases, and in the emergency department.

Heels should be elevated from the bed in high-risk patients. Heel elevation can be done with a pillow placed under the calf of the leg in order to "float" the heel from the bed. Pressure-relieving boots can be used when patients do not stay in place on pillows; however, boots themselves can create pressure points. Therefore, boots need to be removed 2–3 times daily to assess for early signs of pressure injury.

Medical devices should be removed 2–3 times a day, if only long enough to inspect for signs of pressure on the skin [14]. High-risk areas, such as the bridge of the nose and face, should be padded with thin dressings prior to the use of noninvasive positive pressure masks [15]. Oxygen tubing should be padded to reduce the intensity of pressure behind the ear.

11.5 Treatment

Pressure necrosis of the sacrum, buttocks, and ischia will need debridement to viable tissue if healing is the goal for the patient (Fig. 11.2a, b). During the healing process, pressure on the wound must be limited to 1 h 3 times a day (for meals). Nutrition must also be adequate to promote healing and reduce the risk of infection. Biofilm quickly develops in these wounds, so biofilm-resistant antiseptics should be used (silver, cadexomer iodine, honey) [13].

Pressure necrosis of the heel should not be debrided in patients with ischemic limbs. As long as the eschar remains stable, local wound care with topical iodine is recommended. The eschar will lift from the edges and should be trimmed to prevent it from snagging on clothing. If the eschar cap is removed from the wound, or the eschar cap is softened, infection rapidly develops. The poor inherent blood flow in the limb reduces the likelihood of healing and often leads to amputation due to critical limb ischemia [13].

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