NATIONAL PRESSURE
ULCER ADVISORY PANEL

Deep Tissue Injury

Overview

Deep tissue injury is a term proposed by NPAUP to describe a unique form of pressure ulcers. These ulcers have been described by clinicians for many years with terms such as purple pressure ulcers, ulcers that are likely to deteriorate and bruises on bony prominences (Ankrom, 2005). NPAUP's proposed definition, is "A pressure-related injury to subcutaneous tissues under intact skin. Initially, these lesions have the appearance of a deep bruise. These lesions may herald the subsequent development of a Stage III-IV pressure ulcer even with optimal treatment."(NPAUP, 2002).

Why is it important to have another label for pressure ulcers? Aren't four stages enough? Perhaps not, because deep tissue injury represents a dangerous lesion due to its potential for rapid deterioration. Proper labeling will afford clinicians a more accurate diagnosis, and lay a foundation for the development of efficacious interventions.

Background

Shea (1975) is often credited with labeling the stages of pressure ulcers, and his descriptions of four depths of ulcers were adapted by IAET, AHCPR, and NPUAP. Missing from that four stage system label is deep tissue injury. However, Shea did not forget them in his review. He described ulcers that appear innocent yet conceal a deep, potentially fatal lesion. In 1873 Paget may have been the first to describe deep tissue injury describing purple areas with sloughing of tissue and large cavities once they open. In 1942, Groth created deep, "malignant pressure sores" in an animal model in a series of experiments that began in deeper tissues and spread to the surface.

The reasons for the rapid deterioration seen with deep tissue injury may be a combination of direct ischemic injury and reperfusion injury from oxygen free radicals, cytokines and neutrophilic adhesion to microvascular endothelium. When there is a long duration of ischemia, the "direct" damage resulting from hypoxia alone is the predominant mechanism. With shorter periods of ischemia, the indirect or reperfusion mediated damage becomes increasingly more important. For example, it has been shown in an animal model that the intestinal injury induced by 3 hours of ischemia (flow reduced to 20% of normal) and one hour of reperfusion is several times greater than that observed after 4 hours of ischemia alone (Parks & Granger, 1988). These results demonstrate that the injury produced by reperfusion can be more severe than the injury induced by ischemia per se. This same pattern of relative contribution of injury from direct and indirect mechanisms has been shown to occur in the heart, brain, muscle and other organs. Since the body of reperfusion injury literature suggests that significant damage occurs upon reperfusion, the development of methods to block the chemical and biological mediators of reperfusion injury holds promise for treating many conditions – even perhaps pressure ulcers.

Problems with the current labeling system

The current label for stage I pressure ulcers clearly delineates that these ulcers are of intact skin. Hence, the current definition of stage I ulcer could include deep tissue injury in its earliest presentation, when it appears like a bruise. A problem with this approach is that the labeling of deep tissue injury as a stage I can reasonably be interpreted to mean that the lesion is relatively minor and healing is likely with offloading. However, if enough subcutaneous pressure damage has occurred prior to its identification, and if there is reperfusion injury, it is quite possible that no amount of pressure offloading will prevent further deterioration. Such lesions are often identified upon transfer of a patient from one facility to another, and since no treatment or pressure offloading might prevent deterioration, the receiving facility can inappropriately receive quality of care citations or medical malpractice claims for an injury that originated elsewhere.

The current label for stage II pressure ulcers includes blistered lesions. Here again is a situation in which a deep tissue injury may present as a serum blister or blood blister and be appropriately labeled as a stage II. Alternatively, a large area of deep pressure injury can evolve without blister formation but with loss of the outermost cutaneous layers days after the initial injury. Although current staging definitions would also allow such a lesion to be called stage II, the expected deterioration of this injury differentiates it from typical, minor stage II pressure ulcers which will heal in days or a few weeks.

Staging deep tissue injury

The proper labeling of pressure-related damage under intact skin may be most accurately labeled "deep tissue injury". Force fitting these lesions into stage I or stage II ulcer labels is reductionistic and does not represent the true and dangerous potential in them to deteriorate quickly. Several options exist for staging. Because staging was designed to represent the depth of the ulcer, and these ulcers do not have a known depth because the extent of the damage is not known, the term "unstageable" may suffice. However, the term "unstageable is often meant to imply that once debridement occurs, the wound will be accurately staged. Since not all deep tissue injuries should be debrided (e.g., purple areas over bony prominences that look like bruises), "unstageable" does not seem to be an inclusive enough term to unambiguously describe these lesions. Labeling deep tissue injury as stable eschar with the clinical intention of debriding it when there is evidence of infection is another option. However, with such a label, offloading may not be seen as critical to the care plan. Finally, perhaps deep tissue injury needs its own four stages; paralleling the current staging system for pressure ulcers. In this system, the intact skin phase would be labeled a stage I DTI, the blistered phase a stage II DTI and so on.

Differential diagnosis

Several skin conditions need to be included in the differential diagnosis of deep tissue injury:

Bruise: the extravasation of blood in the tissues as a result of blunt force impact to the body. Usually about two weeks is required for healing a bruise under normal conditions. History of trauma is common.

Calciphylaxis: vascular calcification and skin necrosis most common in patients with a long-standing history of chronic renal failure and renal replacement therapy. Lesions may have a violaceous hue and be excruciatingly tender and extremely firm. Lesions are most commonly seen on the lower extremities, not bony prominences. The incidence of these lesions is very low in general patient populations.

Hematoma: deep seated purple nodule from clotted blood, usually associated with trauma.

Fournier's gangrene: intensely painful necrotizing fasciitis of the perineum. May present initially as cellulites .

Perirectal Abscess: commonly presents as a dull, aching, or throbbing pain in the perianal area. The pain worsens when sitting and immediately before defecation; the pain abates after defecation. A tender fluctuant mass may be palpated at the anal verge. These abscesses can open to reveal large cavities which can be confused with deep pressure ulcers.

Implications for research

It is imperative that research be conducted on pressure-related deep tissue injury. While these lesions were likely included in much previous work on pressure ulcer prevalence, incidence, and natural history, since staging system definitions have been ambiguous in how to characterize deep tissue injury under intact skin, the epidemiology and clinical course of these lesions remains unknown. Prospective observational studies can lead to a full understanding of the likelihood of deterioration versus complete resolution, and will allow an unambiguous nomenclature to be developed with respect to reliably diagnosing and predicting the natural history of these lesions.

A second area for research are studies designed to develop diagnostic methods to identify the extent of tissue damage under intact skin (e.g., depth, volume) as well as the viability of injured tissue (e.g., viable, necrotic). These methods may be particularly applicable to patients with darkly-pigmented skin in whom the visual signs of deep tissue injury can be hard to identify.

A third area for research is to identify the most efficacious treatments for each type of deep tissue injury. Interventions should be tested that can prevent further damage, salvage injured yet viable tissue, and maximize overall healing rates.

Implications for education

Education is imperative to assist all health care providers to conduct thorough skin assessments, and understand how to use a precise nomenclature to describe the phenomenon of deep tissue injury.

Regulatory issues

A full understanding of the nature of deep tissue injury and the efficacious methods for treatment should guide the regulation of payment, as well as the implementation of sanctions for suboptimal care.
Questions:

What are the clinical indicators that you would use to define a stage I pressure ulcer?
How can deep tissue injury be best identified in patients with darkly-pigmented skin?
How long does it take to develop a deep tissue injury?
What level of pressure/stress is required to develop a deep tissue injury?
How do co-morbidities (esp. rapid weight loss, loss of sensation) affect the time to develop deep tissue injury?
Does a deep tissue injury that never reaches the skin really a pressure ulcer?

How would a revised staging system affect documentation?
How would a revised staging system impact regulation and reimbursement?
How would the addition of deep tissue injury to the nomenclature of pressure ulcers affect liability?

How would a revised staging system impact health care quality measures?

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