Clinical and Molecular Perspectives of Deep Tissue Injury: Changes in Molecular Markers in a Rat Model

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Abstract	Deep tissue injury (DTI) is a pressure-related injury to subcutaneous tissues under intact skin. DTI has recently been a focus of enthusiastic debates among wound care specialists, particularly regarding how DTI should be categorized into the conventional pressure ulcer (PU) classification. Pathophysiologically, DTI is regarded as an antithesis (bottom-up) of the conventional understanding of PU formation (top-down), and some researchers have suggested that all deep PUs are derived from DTI. On the other hand, the concept DTI can be applied to several specific conditions characterized by subcutaneous tissue damage, some of which were not originally recognized as PUs. In the first part of this chapter, we discuss the novelty and conceptual confusion of DTI from the clinical standpoint, and briefly review several specific types of DTI, as found in immobilized patients, in Asian patients with excess bony prominences, and in patients with spinal cord injury. In the second part of this chapter, we review the molecular aspects of DTI pathophysiology. Damage to subcutaneous tissues such as muscle and adipose tissue is predominantly repaired by the conventional wound healing process with granulation and scar formation. However, there may also be endogenous regenerative reactions in these tissues, such as myogenesis, possibly adipogenesis, and angiogenesis. which will be reviewed at the molecular level later in this chapter. Furthermore. we have established a rat model of DTI. We will present our own experimental results of muscle damage in the DTI model, and the elevation of biochemical and molecular markers indicating muscle damage and regeneration, including creatine phosphokinase (CPK), hypoxia-inducible factor-1 (HIF-1), and Hedgehog (Hh) signaling molecules.
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