Understanding of extracellular matrix of the dermis and granulation tissue is useful for medical practice of cutaneous wound.

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Cutaneous wounds such as pressure ulcer, burn and traumatic ulcer are common in clinical practice. Impaired ADL, protein loss from wound and infection are often complicated with those conditions.

During the healing process of deep wound lacking dermis, transient formation of granulation tissue (GT) is essential. GT contains abundant inflammatory cells and contraction of wound is induced after GT formation. It is important to understand the difference between dermis and granulation tissue from the point of view of ECM.

Structural ECM such as fibrous collagens and hydrated ECM such as proteoglycan, hyaluronan (HA) and plasma proteins orchestrates for desirable wound healing. Once structural ECMs are disrupted by physical stress, hydrated ECM components are recruited to make granulation tissue. Then the hydrated ECM is replaced by the structural ECM to complete wound healing.

A HA-binding proteoglycan, versican functions as a structural component of fibrillin-microfibrils in the normal dermis. In the GT, versican is cleaved by ADAMTS proteases and the fragment containing the G1 domain (VG1F) interacts with serum-derived HA-associated protein (SHAP) to form macrocomplex. Thus, versican forms distinct complex by interacting to other ECM molecules in order to perform tissue/stage specific functions. VG1F-SHAP-HA complex was specifically present in the surface of edematous/inflammatory wound and disappeared along with the stabilization of wound.

Wound in which epithelial tissue is absent can be considered as ‘a visible ECM at bedside’. Therefore, understanding ECM of wound is useful to treat patients with those chronic wounds.

Key words: pressure ulcer, versican, hyaluronan