Fetal wound healing: Clinical implications for keloid treatment

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No one is born with scars. This is because tissue regeneration is possible rather than scar formation as a response to injury during the fetal period. For successful scar treatment, three conditions of so-called "fetal wound healing" should be considered. First, the cytokines that increase the inflammatory response (IL-6,8, TGF-ß1,2) are decreased, and the cytokines that reduce the inflammatory response (IL-10, TGF-ß3) are increased. Second, there is no mechanical force. Third, stem cells and growth factors are abundant. Among these conditions, the first thing to pay attention to in scar formation is mechanical tension. When restoring skin defects after skin cancer surgery, the concave area of the face may have less tension, making the scar less visible. Keloids, which grow like stones in the earlobe, cause tension by themselves, so it is difficult to treat them if they are not addressed through surgical methods early.

Among our patients with earlobe keloids, the most difficult to treat was the case of a postoperative vascular malformation-like response. In 2016, Ogawa et al. published a hypothesis that endothelial dysfunction may play a key role in keloid and hypertrophic scar pathogenesis. This endothelial dysfunction increases the inflammatory cell numbers and the levels of many inflammatory soluble factors that enter the perivascular area from the blood vessels, thereby increasing local inflammation, resulting in collagen over-production of fibroblasts.

In this presentation, I would like to introduce the cases of keloids treated with a combination of the concepts of fetal wound healing and vessel targeting therapy, and consider optimal methods for minimizing scarring.