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LVA (Lymphovenous anastomosis) aids wound healing in lymphedema: Relationship between lymphedema and delayed wound healing from a view of immune mechanisms.

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Abstract

Objective: Delayed wound healing in lymphedema is asuumed to be caused by two reason, pathophysiologic and Immunologic effects of lymphedema. The aim of this review is to establish how impaired lymphatics alter wound healing pathophysiologically and immunologically, and to propose treatment modalities that can promote wound healing in lymphedema.

Approach: Lymphedema is characterized pathophysiologically by localized eripheral edema that compresses the microvasculature and lymphatic vasculature and impairs tissue remodeling. Another suspected mechanism is an imbalance in the ifferentiation of participating immune cells. Profound suppression of Th1 cells is likely to increase the risk of infection, and excessive differentiation of Th2 cells, including M2 macrophage polarization, may promote fibrosis, which disrupts the carefully orchestrated wound healing process.

Results: LVAs were performed to the patients who had recurrent cellulitis several times with lymphorrhea and developed severe ulcers that were refractory to skin grafts, flaps and conservative therapy. The lymphorrhea and the ulcer had healed by 4 weeks. Moreover, the lymphedema improved without compression therapy.

Innovation: Although VAC is useful for treatment of delayed wound healing in lymphedema, LVA may be necessary to treat the fundamental problem of lymphedema. LVA is considered to create a bypass to the lymph nodes via which DCs can transmit antigen information to T-cells. LVA is considered to neutralize chronic inflammation by allowing more DCs to return into the circulation, thereby improving wound healing.

Conclusion: Pathophysiologic effects are temporally improved by VAC threapy. However, permanent improvement for pathophysiologic and Immunologic effects are necessary. LVA may be the candidate.