Further Evidence that Acellular Cadaveric Dermis (AlloDerm®) Decreases Inflammatory Markers of Capsule Formation in Implant-Based Breast Reconstruction

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Abstract

Background: Use of acellular cadaveric dermis (ACD) in implant-based breast reconstruction provides an alternative to total submuscular placement. We previously reported that biointegrated ACD samples from implant-based breast reconstruction patients had statistically diminished levels of granulation tissue formation, vessel proliferation, chronic inflammatory changes, capsule fibrosis, fibroblast cellularity, and foreign body giant cell inflammatory reaction compared with native breast capsule samples. These findings suggested that certain properties intrinsic to ACD may diminish inflammatory changes that may promote peri-prosthetic capsule formation. We sought to further evaluate differences between ACD and native breast capsule specimens through immunohistochemical analysis of key inflammatory markers involved in capsule formation.

Methods: Twenty patients underwent implant-based breast reconstruction using the “dual-plane” ACD (AlloDerm® Regenerative Tissue Matrix, LifeCell Corporation, Branchburg, NJ) technique. During exchange of the tissue expander for the implant, intraoperative biopsies of biointegrated ACD and native subpectoral capsule from the tissue expander envelope were obtained. Immunohistochemical analysis was performed for inflammatory markers: macrophages (CD68), T-cells (CD3), B-cells (CD20), myofibroblasts (αSMA), endothelial cells (CD31), and collagen I and III. Masked biopsy specimens were semi-quantitatively scored by a histopathologist to reflect observed levels of marker staining. Scores were statistically analyzed using the Wilcoxon rank test.

Results: ACD samples had statistically diminished levels of all inflammatory markers assessed compared with corresponding native breast capsule samples ($P<0.005$) (Figure 1). Although all variables were significantly diminished in biointegrated ACD capsules compared with native breast capsules, fibroblast cellularity, granulation tissue, endothelial cells, vessel proliferation, collagen I, and capsule fibrosis had substantially lower $P$ values, suggesting that for those variables, there was an even greater difference between ACD and the native capsule control. Interestingly, although myofibroblasts, collagen III, and B-cell lymphocytes were also decreased in ACD compared with capsular control, these differences were not as significant as the previous variables.

Conclusion: This study further supports the theory that ACD has certain intrinsic properties that may limit capsule formation. The decreased inflammatory changes in the ACD samples as evidenced by decreased fibroblast activity, collagen I deposition, and capsular fibrosis, suggest that use of ACD may result in decreased capsule formation. Further investigation is needed to determine the mechanism by which ACD inhibits these inflammatory cells, whether ACD reduces the incidence of breast capsular contracture, and the longevity of this effect.

Figure 1. Semiquantitative analysis of inflammatory markers with acellular cadaveric dermis (ACD; AlloDerm®) vs. native breast capsule samples (0=None, 3=severe levels of marker staining)

*n=21 samples (a bilateral sample was obtained from 1 patient; all other samples were unilateral). $P<0.005$ for all comparisons.
Reference

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