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ORIGINAL ARTICLE EXPERIMENTAL/SPECIAL TOPICS

Effect of Antiadhesion Barrier Solution and Fibrin on Capsular Formation After Silicone Implant Insertion in a White Rat Model

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Abstract

Introduction One of the most serious complications of breast reconstruction and augmentation using silicone implants is capsular contracture. Several preventive treatments, including vitamin E, steroids, antibiotics, and cysteinyl leukotriene inhibitors, have been studied, and their clinical effects have been reported. However, the problem of capsular contracture has not yet been completely resolved. This study was performed to compare antiadhesion barrier solution (AABS) and fibrin in their ability to prevent fibrotic capsule formation and simultaneously evaluated their effect when used in combination by capsular thickness analysis and quantitative analysis of matrix metalloproteinases (MMPs), tissue inhibitors of metalloproteinases (TIMPs), and type I collagen within the fibrous capsule.

Materials and Methods This study used female six-week-old Sprague-Dawley rats. Eighty rats were equally subdivided into the four following groups: AABS-treated, fibrin-treated, AABS and fibrin combined-treated, and untreated control groups. Each rat received two silicone chips under the panniculus carnosus muscle layer. The test materials were applied around the silicon chips. Four weeks later, the implantation sites including the skin and muscle were excised to avoid the risk of losing the fibrous capsule around the implants. The capsular thickness was analyzed by Masson's trichrome stain. Quantitative analysis of type I collagen, MMPs, and TIMPs was performed by real-time PCR, Western blot, and zymography.

Results The mean capsular thickness was 668.10 ± 275.12 μm in the control group, 356.97 ± 112.11 μm in the AABS-treated group, 525.96 ± 130.97 μm in the fibrin-treated group, and 389.24 ± 130.51 μm in the AABS and fibrin combined-treated group. Capsular thickness was significantly decreased in all experimental groups ($p < 0.05$). Capsular thickness was greater in the fibrin-treated group than in the AABS-treated group ($p < 0.05$). There was no statistically significant difference in capsular thickness between the AABS and fibrin combined-treated group and the AABS- or fibrin-treated group ($p > 0.05$). Compared to the control group, the experimental groups had significantly lower expressions of type I collagen and MMP-1 ($p < 0.05$), but there was no statistically significant difference in expressions of type I collagen and MMP-1 between the AABS-, fibrin-, and AABS and fibrin combined-treated groups ($p > 0.05$). The expressions of MMP-2 and TIMP-2 were not significantly different between the control and the experimental groups ($p > 0.05$).

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