ACNE, ROSACEA, AND RELATED DISORDERS (INCLUDING HIDRADENITIS SUPPURATIVA)

MODIFIED METABOLISM OF EXTRACELLULAR MATRIX BASED ON TGF-β1 SIGNALING DURING ATROPHIC ACNE SCAR FORMATION

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Background: Atrophic acne scar, a persistent sequela from acne, is undesirably troubling patients in cosmetic and psychosocial aspects. Therefore, evolving perspectives on its detailed pathogenic process are promptly needed.

Objective: To reveal the pathogenesis of atrophic acne scar, we investigated the histological, immunological and molecular changes in early acne lesions susceptible to atrophic scarring.

Methods: Thirty subjects who have active acne or acne scar were included. The subjects were divided into two groups; fifteen were prone to acne scar and the others were not prone. Skin samples of early acne lesions were obtained, and the difference of molecular profiles between two groups was examined.

Results: We validated several hallmarks accounting for atrophic scarring. First, devastating degradation of elastic and collagen fibers occurs in the dermis, followed by their incomplete recovery. Second, abnormally excessive inflammation mediated by innate immunity with Th17/Th1 cells is observed. Third, epidermal proliferation is significantly diminished, affecting keratinocytes-fibroblasts cross-talks. Above all, the aberrant TGF-β1 signaling may act as an underlying modulator of all these pathological processes.

Conclusion: These results may ultimately provide a basis for understanding the pathogenesis of atrophic acne scarring as well as a novel idea about preventive intervention to atrophic acne scarring.

Key words: Acne scar, Atrophic scar, Collagen, Elastin, Inflammation, Pathogenesis, TGF-β