Staphylococcus Aureus Infection Induces an Accumulation of Dibromotyrosine in the Thickened Superficial Layer of Chronic Cutaneous Wounds

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Abstract: Chronic wounds are often infected with *Staphylococcus aureus*. Such an infection causes deterioration of tissue repair mechanisms. Wounds are composed of granulation tissue and a superficial cover layer consisting of necrotic tissue and inflammatory cell infiltrates. In the present study, we examined 30 patients with skin wounds, with or without *Staphylococcus aureus* infection. The patients were divided into 3 groups according to the number of *Staphylococcus aureus* at 10^5 CFU/g: lower value group (SA-I), and higher value group (SA-II). The thickness of the superficial layer and *Staphylococcus aureus* invasion were morphometrically analyzed. *Staphylococcus aureus* localized mostly in the superficial layer. The superficial layer becomes thicker in accordance with the increase in the number of *Staphylococcus aureus*. Patients in the SA -II group exhibit a significantly thicker superficial layer than those in SA-I group ($p = 0.0001$); however, no difference was found between the control and *Staphylococcus aureus* -I groups. Furthermore, the distance of *Staphylococcus aureus* invasion, measured histologically, was also significantly greater in SA-II than I ($p = 0.003$). In the superficial layer, myeloperoxidase (MPO)-expressing neutrophils and MPO producing dibromotyrosine (DiBrY), an oxidative product, were increasingly accumulated in accordance with the number of *Staphylococcus aureus* in the wound. These results suggest that DiBrY accumulation, which contributes to superficial layer thickening, is caused by the interaction between the infecting *Staphylococcus aureus* and the MPO-expressing activated neutrophils, which may form a biofilm in vivo. These events create poor conditions for tissue repair in chronic wounds.

Key words: ***Staphylococcus Aureus*, Biofilm, Chronic Wound, Dibromotyrosine, Myeloperoxidase**