Formation of Type III Collagen during Early Wound Healing of Periodontal Defects in Model Rats with Type II Diabetes Mellitus

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Abstract: It has been reported that 95% of diabetes mellitus patients exhibit non-insulin dependent type 2 disease in Japan. Diabetes mellitus is attracting attention as a risk factor of periodontal disease. The formation and accumulation of advanced glycation end products (AGEs) are known to progress faster under diabetes, and there is accumulating evidence that AGEs play a role in the development of diabetes by inducing islet beta cell damage and insulin resistance. In early wound healing, the type III collagen is an important factor during tissue maturity. We created periodontal defects in the maxillary first molars of normal and type 2 diabetes model rats in order to examine the relationship between AGEs and type III collagen. We observed pathologically the early wound healing of periodontal defects.

Palatal dehiscence defects were surgically created on the bilateral maxillary first molars in normal and type 2 diabetes model rats. Rats were euthanized at 3, 5, 7 and 14 days after surgical operation, and perfusion-fixed, followed by removal of the periodontium including maxillary first molar and preparation of paraffin sections. Paraffin sections were stained with hematoxilin-eosin (HE). AGEs and type III collagen were detected immunohistochemically.

At 5 days and 7 days, localization of type III collagen was stronger in the control group than the experimental group. Type III collagen fibers were irregularly arranged in the experimental group at 14 days. There were many amorphous capillary vessels, and AGEs localized the vascular endothelium of the vessels in all experimental groups.

AGEs react with the receptor for AGEs (RAGE), then promote the production of nitric oxide in vascular endothelium. A large amount of nitric oxide might decrease the production of type III collagen and increase amorphous blood capillaries via fibroblasts, vascular endothelium and pericyte.

It is suggested that AGEs inhibit the formation of type III collagen, produce poor amorphous capillary vessels, and interfere in the early period of the wound healing process.

Key words: Diabetes, Early wound healing, Type III collagen, AGEs