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Fundamental Study on Degenerative Joint Disease by D-glucosamine

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INTRODUCTION

In recent years, there are some reports about oral intake of collagen that will be effective for recovery of disordered joint function as same reports as oral administration of D-glucosamine (GlcN)[1, 2]. However, there is no report for pathological experiment of damaged cartilage repair process by collagen administration. In the present study, damaged cartilage repair process was investigated by oral administration of soluble collagen and GlcN using rabbit cartilage damage model.

EXPERIMENTAL

Cartilage Damage Rabbit Model

Under general anesthesia, stifle joint is opened with aseptic surgical technique, and making three holes in distal femur cartilage, one is in medial condyle and two are in trochlea. The hole is 2 mm in diameter, 4 mm in depth, so whole cartilage and surface of undercartilage bone are destroyed. The joint opened is closed aseptically, and antibiotic is administered subcutaneously for 3 days.

Reagents

Three types of collagen solution were supplied by Nippon Meat Packers Inc. (Osaka). Original collagen was extracted from cockscomb, and was depolymelized by proteinase. Molecular weight (mean) is 500, 1, 000, and 10, 000 D, respectively, and major amino acids contained are glycine: 34.06%, prolyne: 11.6%, alanine: 11.48%. pH of the collagen solutions are 5.6. GlcN was supplied by Koyo Chemical Co. (Tokyo).

GlcN was obtained by depolymelization of crab chitin with HCl, and its pH and purification is 3.8 and 100% (no protein contamination), respectively.

RESULTS

After the experiment, there is no significant difference among 5 groups in body weight. Muscle atrophy of the thigh was effectively prevented in the CoG compare to that of the C group. The CoG showed excellent healing, and holes were clearly observed in C. Repair rate (hole depth at 2or 3 weeks/initial depth x 100) significantly increased in all treated groups compare to it of the C. Histologically, after 2 weeks, the holes were filled by fibroblasts and chondroblasts in the treatment group, whereas in the control group, the holes were not filled completely like in the treatment group even in 3 weeks. After 3 weeks, in the CoG, the holes were completely filled by proliferating chondroblasts. In addition to healing of the holes, reconstruction of bony trabecular was observed. The CoG significantly increased both glycans not only in repair site but also in normal cartilage.

CONCLUSION

Enzyme degraded collagen, 10, 000 D, effectively absorbed from digestive tract quickly and induces damaged cartilage repair with an acceleration of cartilage matrix synthesis. Simultaneous administration of GlcN and collagen showed synergistic effect on cartilage repair with regeneration of hyaline cartilage.

REFERENCES

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