The Effect of Sodium Diclofenac on Callus Formation in White Male Rat (Rattus Norvegicus) Cruris Fracture Healing

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INTRODUCTION

Prostaglandins are formed mainly in the fracture site in the inflammation and soft callus formation stage. They are formed in the healing process and stimulate osteoclast accumulation and increase its activity.1 Cyclooxygenase (COX) is a rate-limiting enzyme involved in the conversion of arachidonic acid to prostaglandin H2.2 Sodium diclofenac intake inhibits the cyclooxygenase enzyme. In a previous study,3 NSAID intake interfered with the fracture healing process. NSAID was caused by the disruption of osteoclast and osteoblast activities which decreased callus quality and fracture healing.

MATERIALS AND METHODS

Thirty-six rats were randomly allocated into 2 groups which were group I (treatment group) and group II (control group) with eightteen rats in each group. Anesthesia was performed with a intravenous method. Then the fracturation and immobilization of one side of its lower limbs were performed. After fracturation, group I was given 1.8 mg sodium diclofenac/150 grams a day by “sonde”. The duration of the intake was 28 days. Group 2 was treated with a placebo solution of CMC-Na 0.5% using “sonde” with the same volume as the sodium diclofenac in group 1. On the 28th day, all the rats were sacrificed, the diameter of each callus was measured in millimeters with a dissecting microscope. The strength of each callus was measured with the Shimadzu Autograph in newtons (N).

DISCUSSION

Fracture healing was evaluated through bone density and bone strength parameters. Sodium diclofenac triggers changes in bone metabolism and fracture healing because sodium diclofenac affects the healing inflammation phase. By inhibiting cyclooxygenase, sodium diclofenac decreases the first synthesis of the inflammation mediator, including prostaglandin which is responsible for chemotaxis in the first phase of fracture healing. It also decreases the cell number in the fracture site, absorbs the tissue again, and allows the modification of the number of cells for callus formation. In healing process, there is tendency to differentiate into chondroblast and fibroblast which are responsible for extracellular matrix synthesis. Thus, the immature and less mineralized bone callus would be produced.

RESULTS

Figure 2. The Results of Callus Strength

![Figure 2. The Results of Callus Strength](image)

Figure 3. The Average of Callus Diameter

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CONCLUSION

NSAID could delay bone regeneration by inhibiting the prostaglandin at an early stage of healing through the subtraction of quality (diameter and strength) of fracture healing callus.

REFERENCES