Use of a Subchondral Bone Plate Micropick Technique to Augment Healing of Articular Cartilage Defects

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The technique of subchondral bone micropicking offers advantages over conventional debridement in promoting healing of articular cartilage defects in the horse. The technique is presumed to allow access of mesenchymal stem cells and growth factors from cancellous bone into the defect site without compromising the subchondral bone plate and also possibly provides an improved attachment of the repair tissue. Authors' addresses: Equine Orthopaedic Research Laboratory, College of Veterinary Medicine and Biomedical Sciences, Colorado State University, Fort Collins, CO 80523 (McIlwraith, Frisbie, Trotter, Oxford, and Howard) and Steadman-Hawkins Research Foundation, Vail, CO 81657 (Rodkey and Steadman). © 1998 AAEP.

1. Introduction

Joint injuries are a leading cause of disability among both equine and human athletes, and a common sequela of joint injury is the loss of articular cartilage.1 Articular cartilage is a highly specialized skeletal tissue that is dependent on an intact matrix for its unique biochemical and physiologic properties. Once damage or loss of articular cartilage has occurred, the repair process is typically inadequate.1 Various therapies have been utilized to augment the healing of osteochondral defects in human and equine patients, including full thickness curettage, spongialization, subchondral bone drilling, abrasion arthroplasty and periosteal autografts, osteochondral grafts, sternal cartilage autografts, and most recently, chondrocyte transplantation.1,2 Although penetration of the subchondral bone plate provides access to the cancellous bone and presumably mesenchymal stem cells and growth factors, we have become concerned about disrupting the subchondral bone plate and feel that the biomechanical changes lead to extra stresses on the new repair tissue.2 This paper describes our evaluation of a technique of subchondral bone (microfracture) or micropicking that was developed by one of the authors (JRS).3

2. Materials and Methods

Two research projects, one a long-term study and the second a short-term study (evaluating gene expression of critical matrical components) have been done. In the long-term study, ten skeletally mature horses had a 1-cm full thickness osteochondral defect created in both radial carpal bones and both medial femoral condyles. One carpal defect and one femo-
ral condylar defect had the subchondral bone plate perforated with an orthopedic awl. Arthroscope and instrument portals were sutured and sterile bandages were placed on the carpi. Horses were given phenylbutazone postoperatively and confined to a box stall for the first 2 weeks. They were hand walked from 2 to 6 weeks and then trotted on a treadmill from then until 4 months, at which time galloping was also done in the 12-month group. All horses were exercised; five were evaluated after 4 months, and five horses were evaluated after 12 months. A gross histologic and histomorphometric examination of defect sites and repair tissues was performed, as was collagen typing (using high-performance liquid chromatography) of the repair tissue.4

In the short-term study, the same equine model system was used with defects in both femoral condyles and both distal radial carpal bones. Care was taken to remove all calcified cartilage while leaving the subchondral bone plate intact. One randomly chosen defect in the femorotibial joint and the opposite carpus were treated with the surgical awl to create subchondral bone plate microfractures, while the other joints served as nontreated controls. Perforations were created uniformly within the defect site to a depth of 3 mm and were 2 mm apart. In the short-term study, the same aftercare was instituted following surgery as described in the long-term study. There was no exercise program. Horses were euthanized in accordance with Colorado State University ACUC guidelines. Two were euthanized at 2 weeks, two at 2 weeks, two at 6 weeks, and six at 8 weeks after creation of the defect. A gross examination (with photography) was done. A histologic and histomorphometric evaluation was done. Tissue was also evaluated by using RNA extraction and reverse transcription-coupled polymerase chain reaction (RT-PCR) to evaluate the gene expression for types I, II, and III collagen, as well as aggregan (and GAPDH was used as a control). Primer sequences were chosen and annealing temperatures were determined with the aid of oligo software. The PCR was carried out in a semiquantitative manner. In situ hybridization was also done on the tissues from each joint to evaluate the sites of gene expression of critical matrical components. Equine-specific antisense and sense riboprobes for type I collagen, type II collagen, and aggregan were used.

3. Results

In the long-term study, a greater volume of repair tissue filled the treated defects (74%) versus the control (45%) defects at both time periods on gross examination. Histomorphometry confirmed more repair tissue filling the treated defects, but there was no difference in the relative amounts of tissue types (hyaline cartilage, fibrocartilage, and fibrous tissue). There was also an increased percentage of type II collagen in the treated defects compared with controls, and there was evidence of earlier bone remodeling.

In the short-term study, there was a higher percentage of repair tissue filling the defects compared with the respective control defects at 2 and 4 weeks after microfracture treatment. On histology the percent of granulation tissue in the defects decreased over time, but the process was delayed in the control versus the treated defects. With RT-PCR detection of messenger RNA, there was an enhancement of type II collagen gene expression in treated femorotibial defects compared with controls, but there was no enhancement in the expression of type II collagen in treated carpal defects compared with controls. The aggregan message level increased with time, but there was no enhancement observed in treated defects compared with controls. Specificity for the riboprobes used in the in situ hybridization was confirmed. Type I collagen was found preferentially expressed at the surface of the repair tissue, while the message for type II collagen was found at the deeper levels in the repair tissue. Immunohistochemistry confirmed the distribution of protein in parallel with the messenger RNA expression, demonstrating that type I collagen protein was found primarily at the surface of the repair tissue with lower concentrations throughout the tissue while type II collagen was found at discrete foci located at deeper levels in the repair tissue.

4. Discussion

Both long- and short-term studies show an enhancement of tissue formation with the subchondral microfracture technique. However, in the short-term study, an enhancement of gene expression for type II collagen was not demonstrated in the carpal defects, and it is obvious that there are differences depending on location and joint. The lack of enhancement of aggregan synthesis while augmenting type II collagen synthesis is in agreement with earlier work done by this research group, in which it seems the biggest limiting factor to long-term functional healing is a decreased aggregan content. This laboratory is now investigating the use of growth factors in artificial matrices to augment this repair further. The microfracture technique is being used routinely in clinical cases in the horse when there is retention of subchondral bone plate but loss of articular cartilage. It is suggested that in clinical cases with a sclerotic subchondral plate that the value of micropicking is increased compared with the differences in experimentally created defects.

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References and Footnotes

1. McIlwraith CW, Nixon AJ. Joint resurfacing: attempts at repairing articular cartilage defects. In: McIlwraith CW,


a Linvatec Corp., Largo, FL 33773.
b Lifesciences Software Resources, Long Lake, MN 55356.