AWARD-WINNING ABSTRACTS FROM THE JAPAN SHOULDER SOCIETY ANNUAL MEETING 2018

INTRAARTICULARLY INJECTED MESENCHYMAL STEM CELLS STIMULATE ANTI-INFLAMMATORY MOLECULES AND INHIBIT PAIN AND CHONDROLYTIC ENZYMES IN A RAT MODEL OF ARTHRITIS

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Mesenchymal stem cells (MSCs) possess anti-inflammatory properties and tissue differentiation potential. We prepared a rat shoulder arthritis model by injecting monoiodoacetate (MIA) into the shoulder and investigated the intraarticular administration of MSCs from the aspects of the cartilage protective effect associated with the cell's anti-inflammatory activity and inhibition of the central sensitization of pain. When MIA was administered in this model, anti-calcitonin gene related peptide (CGRP) was expressed in the shoulder joint and C5 spinal dorsal horn. Expression of ADAMTS5, a marker of joint cartilage injury, was similarly elevated following MIA administration. When MSCs were injected intraarticularly after MIA administration, the expression of CGRP in the spinal dorsal horn was significantly deceased, indicating the suppression of the central sensitization of pain. The expression of ADAMTS 5 in joint cartilage was also significantly inhibited by MSC administration. In contrast, a significant increase in the expression of tumor necrosis factor-alpha was evident. Stimulated gene/protein 6 (TSG-6), an anti-inflammatory and cartilage protective factor that is produced and secreted by MSCs intraarticularly extended to the cartilage tissue following the administration of MSCs. In this manner, the intraarticular injection of MSCs inhibited the central sensitization of pain and increased the expression of the anti-inflammatory and cartilage protective factor TSG-6. As the least invasive conservative strategies possible are desirable in actual clinical settings, the intraarticular administration of MSCs, which appears to be effective for the treatment of pain and cartilage protection in early-stage arthritis, may achieve these aims.

COMBINED THERAPY WITH PLATELET RICH PLASMA AND BASIC FIBROBLAST GROWTH FACTOR IN THE EARLY HEALING OF ROTATOR CUFF TEAR IN A RAT MODEL

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Introduction: Retears are a common complication after surgical repair of large and massive rotator cuff tears. Basic fibroblast growth factor (b-FGF) and platelet rich plasma (PRP) reportedly enhance the healing of rotator cuff tears. The purpose of this study was to evaluate the efficacy of PRP and b-FGF using a gelatin hydrogel sheet (GHS), which preserves these healing factors and facilitates their sustainable release until degradation of the sheet during the early healing of rotator cuff tear using a rat model.

Methods: To create a rotator cuff defect, the infraspinatus tendon was resected from the greater tuberosity. Infraspinatus tendons were repaired and covered with GHS impregnated with phosphate buffered saline (PBS) as the control, b-FGF alone, PRP alone, and b-FGF+PRP. Histological examinations and mechanical examinations were performed.

Results: At 2 weeks, the tendon maturing score ascertained from hematoxylin and eosin staining of the b-FGF+PRP group was statistically higher than other groups. Safranin O staining revealed intense staining of proteoglycan at enthesis in the b-FGF+PRP group compared with the other groups. Vascular staining with isolectin B4 in the b-FGF+PRP group was higher than in the other groups at 2 weeks. The number of Col2-positive cells was significantly greater in the b-FGF+PRP group than in the other groups. The ultimate failure load of the b-FGF+PRP group was significantly higher than the control group.

Discussion: In the b-FGF and PRP groups, tendon maturing and fibrocartilage regeneration at enthesis were promoted and mechanical strength was higher than in the other groups. The findings suggest that b-FGF and PRP enhance both tendon and bone-tendon junction healing, with synergistic activity when both b-FGF and PRP are supplied.

WHEN DO BONY DEFECTS OCCUR IN PATIENTS WITH ANTERIOR SHOULDER DISLOCATION?

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Background: Bony defects are common injuries associated with anterior shoulder dislocation. It is generally thought that these defects are created at the time of dislocation. However, there have been no reports demonstrating exactly when they occur. The purpose of this study was to clarify when these bony defects occur using cadaveric shoulders.

Methods: 12 fresh-frozen cadaveric shoulders (mean age: 81 years) were fixed in a custom-made testing machine. First, the glenohumeral joint was inspected by arthroscopy. Then, the arm was forced manually to extend horizontally under fluoroscopy, keeping the arm at 90 degrees of abduction and maximum external rotation followed by arthroscopic inspection. Next, the humeral head was pulled in line with the pectoralis major muscle at 800 N-force with use of the air cylinder. Under this condition, we waited until the arm came to equilibrium. Finally, the glenohumeral joint was arthroscopically examined.

Results: Bankart lesions were observed in 11 out of 12 examined shoulders, and anterior bony fragment of the glenoid was observed in 4 shoulders after dislocation. However, Hill-Sachs lesions were not observed. After the arm came to equilibrium at a lower abduction angle, Hill-Sachs lesions (6 shoulders) and erosion-type glenoid defects (5 shoulders) were observed.

Conclusion: In our cadaveric model, Hill-Sachs lesions were not created at the time of dislocation but after the arm came to equilibrium at a low abduction angle. The fragment-type glenoid defect was observed at the time of dislocation, whereas the erosion-type defect was observed as a compression fracture when the arm came to equilibrium.