Costal Perichondral Grafting for Articular Cartilage Defects in the Rabbit Knee—Process and Comparison with Fascia Grafting—

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Abstract

Autologous costal perichondrium was transplanted to a full-thickness articular cartilage defect in a rabbit knee. The result was compared with a non-treated defect in the contralateral knee and with autologous fascia grafting. In the costal perichondrium grafting group, repair tissue was created by mesenchymal cells from bone marrow, not by the perichondrium. The non-grafted and fascia grafting group showed no improvement.

INTRODUCTION

Over the past three decades, extensive research has been carried out to identify the mechanism regulating anabolic/catabolic homeostasis. This has been done in an attempt either to correct imbalanced cartilage metabolism or to promote the healing of damaged cartilage. So far, studies have mainly focused on biological and physico-chemical aspects.

When a full-thickness articular cartilage defect in the subchondral bone marrow is created, marrow cells capable of participating in an inflammatory response have access to the defect. Even without treatment, some full-thickness defects undergo repair by the marrow cells. Perichondral grafting has been used to restore cartilage defects in animals and human. Few experiments have been carried out on early-stage development of the perichondral graft.

The purpose of this experimental study is to determine, in the costal perichondral grafting for a full-thickness articular cartilage defect into the subchondral bone marrow, whether the perichondrium itself is regenerated or bone marrow cells are differentiated to the hyalin-like cartilage. Results are compared to those from fascia grafting.

key words: costal perichondral graft, articular cartilage defect
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MATERIALS and METHODS

Sixteen mature Japanese white rabbits, weighing 2.7-3.5 kg, were used. The animals were anesthetized by intravenous injection of Nembutal. Arthrotomy of both knees was performed by a medial parapatellar incision, and the patella was dislocated laterally. A cartilage defect measuring 10 × 5 mm was created in the intercondylar groove down to the cancellous bone.

Subsequently, in fourteen rabbits, a strip of perichondrium dissected from the cartilaginous part of one of the lower ribs was applied to the defect of the right knee with Tisseel, a human fibrin glue prepared by mixing fibrinogen (70 to 110 mg/ml) with aprotinin (300 KIE/ml) and thrombin (4 IE) with calcium chloride (40 mmol/l). After heating these mixtures to 37°C, they were applied to the defects. In two rabbits, a fascia was taken from the fascia lata and was applied as above. The perichondrium or fascia was firmly pressed against the cancellous bone for three minutes. The perichondrium was placed with the chobral side facing the knee joint; with the fascia graft, the fascia was placed with the muscle side facing the knee joint. The defect in the left knee of all rabbits was untreated and used as a control. The rabbits were left to move freely about in their cages.

Perichondral grafting group animals were euthanized at 2 and 5 days, and at 1, 2, 4, 6, 10 and 12 weeks. Fascia grafting group animals were euthanized after 2 weeks. The grafts were examined macroscopically and compared with the appearance of the control defects. The specimens were fixed in neutral formalin, embedded in paraffin, sectioned at 5 μm, and stained with hematoxylin-eosin, safranin O-fast green, and azan. Microscopic examination was performed.

RESULTS

Macroscopically, all grafts were found in the place where they had originally been positioned. In the perichondral grafting group of rabbits euthanized at two and five days, repair tissue had not reached the level of the joint surface. In the group examined at two weeks, the tissue had grown to the thickness of the normal adjacent cartilage. At twelve weeks, normal-appearing cartilage had formed (Fig. 1). The control defects remained unchanged and showed progressive degeneration (Fig. 2). In the fascia grafting group, there was no repair tissue.

Microscopically, in the perichondrial grafting group (N=14) two days after the opera-

Fig. 1 Repaired cartilage defect of the intercondylar groove after 12 weeks.
tion, the perichondral graft was thicker, with an increased number of cells. However, the tissue formed had not reached the level of the joint surface. At one week, mesenchymal cells from the bone marrow invaded the defect. At two weeks, mesenchymal cells were oriented towards the joint surface (Fig. 3). At four weeks, the defect was seen with mainly mesenchymal cells, and this newly formed tissue had good safranin-O staining, indicating the presence of large amounts of proteoglycans which is characteristic of normal articular cartilage. After ten weeks the prichondral graft had disappeared and the basal layer contained isogenous groups of chondrocytes in pallisade formation. And after twelve weeks the defect was filled with hyalin-like cartilage (Fig. 4). In the fascia grafting group (N=2), no mesenchymal cells were found in the defects (Fig. 5). The control defect of the left knee (N=16) remained clearly visible owing to its depressed level and irregular surface (Fig. 6).

DISCUSSION

Several attempts have been made to repair cartilage defects with transplantation of perichondral grafting. However, results are difficult to compare because the grafting methods used vary considerably between studies. Engkvist et al.\textsuperscript{3}) created cartilage defects down to the cancellous bone in the glenoid surface of the humero-scapular joint, and perichondral grafts were anchored to the margins with
Homminga et al. confirmed that the perichondrium can be used to repair articular cartilage defects in rabbit knees. He did not expose the cancellous bone by removing the subchondral bone. We created the defect in the patellofemoral joint and removed the subchondral bone.

Only a few experimental studies have been carried out to compare the results of perichondral arthroplasty with spontaneous healing. In our study the defects did not spontaneously heal with fibrocartilaginous tissue. Furthermore, there are few studies which discuss the early changes that occur in perichondral grafting. Ohlsen and Widenfalk reported that chondrogenesis was found to occur in the median and fibrous layer of the perichondrium. Widenfalk et al. demonstrated that fibrin glue gives firm fixation of the graft when combined with an external fixation device for a period of two weeks. We did not use this device because the defect in this study was deeper than that in other studies.

This study considered macroscopic and histologic results during periods of up to three months. Results show that a full-thickness articular cartilage defect in which the subchondral bone plate was opened was repaired by costal perichondral grafting. The repair tissue was a hyalin-like cartilage and...
originated from mesenchymal cells from the bone marrow. The perichondral graft itself had disappeared. It is speculated that differences between the results of Homminga's study, which showed that the perichondrium itself was differentiated to hyalin cartilage, and those of our study, derive from the existence of subchondral bone plate. In comparison to perichondral grafting, fascia grafting did not permit cells from the marrow to invade the defect, and did not repair the defect. Therefore, in the environment of this study, osteochondral defect could not be repaired by simply covering the defect with fascia grafting. Therefore, it is speculated that the perichondrium is a healing stimulator similar to a pretreatment of the defect with chondroitinase ABC or an addition of growth factors. We conclude that the perichondrium makes a good environment for chondrogenic differentiation of mesenchymal cells from the bone marrow, and cannot regenerate the hyalin-like cartilage by itself.

Detailed knowledge of these processes may ultimately provide clues about the mechanism of healing of full-thickness cartilage injuries which do not heal spontaneously with fibrocartilaginous tissue.

REFERENCES

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