Allograft Replacement for Absent Native Tissue

Salma Chaudhury, MD, PhD, MRCS,*† Florian Wanivenhaus, MD,† Alice J. Fox, MSc,† Russell F. Warren, MD,† Maureen Doyle, PA,† and Scott A. Rodeo, MD†

Context: Structural instability due to poor soft tissue quality often requires augmentation. Allografts are important biological substitutes that are used for the symptomatic patient in the reconstruction of deficient ligaments, tendons, menisci, and osteochondral defects. Interest in the clinical application of allografts has arisen from the demand to obtain stable anatomy with restoration of function and protection against additional injury, particularly for high-demand patients who participate in sports. Traditionally, allografts were employed to reinforce weakened tissue. However, they can also be employed to substitute deficient or functionally absent tissue, particularly in the sports medicine setting.

Objective: This article presents a series of 6 cases that utilized allografts to restore functionally deficient anatomic architecture, rather than just simply augmenting the degenerated or damaged native tissue. Detailed discussions are presented of the use of allografts as a successful treatment strategy to replace functionally weakened tissue, often after failed primary repairs.

Keywords: allograft; instability; reconstruction; ligament; tendon

CASE SERIES

Case 1: Allograft Augmented Repair of a Distal Biceps Tendon Tear in a College Football Player

A 23-year-old in-season college defensive end presented 2 weeks after sustaining an injury to his nondominant left elbow during a football game. Despite returning to sports immediately after the injury, the patient experienced discomfort and mild weakness in his elbow. On examination there was minimal wasting of the left biceps compared with the contralateral side, with mild tenderness and decreased supination strength. Magnetic resonance imaging (MRI) confirmed a complete distal rupture of the biceps tendon from the radial tuberosity. Completing the season was a priority, so surgical repair was performed at the end of the season (41 days after injury).

Because of the delay since injury, the biceps tendon had retracted proximally and could not be sufficiently mobilized for direct repair. In view of the shortened biceps tissue and the requirement for high-level performance, a posterior tibial tendon allograft was used for reconstruction. The allograft tendon was proximally fixed to the radius; then, the 2 arms of the tendon allograft were weaved through the native biceps tendon and muscle and sutured in place (Figure 1), with the elbow maintained at 60° of flexion and maximum supination.

From †Hospital for Special Surgery, New York, New York
*Address correspondence to Salma Chaudhury, MD, PhD, MRCS, Hospital for Special Surgery, New York, NY 10021 (e-mail: salmachaudhury@doctors.org.uk).
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significant decrease in strength, flexion, and supination. Allografts have been used for biceps repair. Palmaris longus, flexor carpi radialis, and Achilles tendon power during supination. Supination strength may increase elbow flexion strength but does not restore primary repair is recommended. A biceps-to-brachialis repair tuberosity of the radius can be very difficult. Thus, early anatomic reinsertion of the ruptured tendon to the bicipital proximally. With the concomitant formation of adhesions, ruptures. Steroids or chronic deep radial bursitis may contribute to degenerative changes of the tendon due to the use of anabolic muscle while the elbow is in flexion. In addition to trauma, an acute extension load acting against a contracting biceps muscle-tendon unit by glenohumeral extension and/or external rotation or during contact sports or an activity such as bench press. The recommended treatment for a ruptured pectoralis major tendon is surgical repair. Injury to the pectoralis major muscle is usually caused by a sudden forceful lengthening of the muscle-tendon unit by glenohumeral extension and/or external rotation or during contact sports or an activity such as bench press. There have been approximately 150 cases of pectoralis major ruptures reported, with the majority in professional and recreational athletes; an association with anabolic steroids has also been reported. An Achilles allograft was used in this case to obtain length. To our knowledge, only 1 case report of successfully repairing the pectoralis major with an Achilles tendon allograft has been reported in the literature. This case involved chronic retraction of the pectoralis major, with medial stripping of the tendon into the muscle belly. The sternal and clavicular heads

The patient was placed in a posterior splint at 90° for 2 weeks, before the arm was placed into a hinged brace. The elbow extension increased by 10° per week. For the first 6 weeks, active extension and passive flexion were recommended. Active elbow flexion was begun at 6 weeks and moderate active strengthening exercises were permitted after 3 months. Within 4 months, the patient had no residual pain, had reestablished excellent elbow strength with full range of motion, and was able to return to full training regime that included weightlifting. The patient returned to full contact football at 6 months and was later signed by a professional team.

Distal biceps tendon ruptures usually occur as a result of an acute extension load acting against a contracting biceps muscle while the elbow is in flexion. In addition to trauma, degenerative changes of the tendon due to the use of anabolic steroids or chronic deep radial bursitis may contribute to ruptures. Completely ruptured distal biceps can retract proximally. With the concomitant formation of adhesions, anatomic reinsertion of the ruptured tendon to the bicipital tuberosity of the radius can be very difficult. Thus, early primary repair is recommended. A biceps-to-brachialis repair may increase elbow flexion strength but does not restore supination strength and is not an option for athletes needing power during supination. Fascia lata, semitendinosus, palmaris longus, flexor carpi radialis, and Achilles tendon allografts have been used for biceps repair.

Nonoperative treatment of distal biceps tears results in a significant decrease in strength, flexion, and supination. If the distal biceps tendon is retracted and cannot be adequately mobilized, it can be anatomically reconstructed using an auto- or allograft. Alternatively, a nonanatomic repair of the distal biceps to the brachialis muscle can be performed.

Case 2: Pectoralis Major Repair With an Achilles Tendon Allograft

A 42-year-old right-hand-dominant man sustained a pectoralis major rupture during a motor vehicle accident 7 years earlier. He presented 3 years after a failed right pectoralis major repair. Examination of the right shoulder demonstrated clear failure of the repair with abnormal morphology detected in the anterior chest wall. The patient had a full range of motion of the right shoulder with weakness in internal rotation. Tenderness was detected anteriorly around the retracted pectoralis major tendon. Right shoulder radiographs were grossly normal. MRI of the right chest wall demonstrated chronic retraction of the pectoralis major muscle belly medial to the deltopectoral groove. The medial ridge of the tendon demonstrated an 11-cm gap from the proximal humerus. The clavicular head was also affected and had retracted to the deltopectoral groove, yielding a smaller gap, 75 cm.

A retracted tear involving the sternal head of the pectoralis major was detected during an open surgical exploration. The clavicular head was partially damaged. The tendon could not be fully mobilized, which prevented restoration of the native tendon anatomy. An Achilles tendon allograft was overlaid and woven into the native pectoralis major muscle tendon unit using No. 2 OrthoCord (DePuy, Warsaw, Indiana; Figure 2). The woven tendon and allograft complex was mobilized and inserted into the natural insertion site, the intertubercular groove of the humerus using 3 double-loaded suture anchors.

A progressive strengthening therapy program was advised whereby a gradual increase in motion was permitted 6 weeks postoperatively. After 3 months, the patient had full overhead elevation and had reestablished full external and internal rotation with normal power, although some residual weakness was detected in internal rotation and adduction. By the fourth postoperative month, the patient began chest flies and bench presses.

The recommended treatment for a ruptured pectoralis major tendon is surgical repair. Injury to the pectoralis major muscle is usually caused by a sudden forceful lengthening of the muscle-tendon unit by glenohumeral extension and/or external rotation or during contact sports or an activity such as bench press. There have been approximately 150 cases of pectoralis major ruptures reported, with the majority in professional and recreational athletes; an association with anabolic steroids has also been reported. An Achilles allograft was used in this case to obtain length. To our knowledge, only 1 case report of successfully repairing the pectoralis major with an Achilles tendon allograft has been reported in the literature. This case involved chronic retraction of the pectoralis major, with medial stripping of the tendon into the muscle belly. The sternal and clavicular heads
yielded sizable gaps, and despite efforts, the native tendon could not be restored to the anatomic insertion site. In such cases, because of the chronic nature of the muscle tendon rupture with associated atrophy, improvements in strength and motion are often a gradual process, and there is expected to be some degree of permanent weakness.

Case 3: Reconstruction of the Lateral Compartment of the Knee After Failed Meniscal Transplantation

A 25-year-old female basketball player presented with a 1-year history of right-sided lateral knee pain. The patient had undergone a previous right subtotal lateral meniscectomy 11 years earlier, which was later followed by a lateral meniscal transplant with concomitant placement of 2 osteochondral allografts into the lateral femoral condyle. Examination revealed a valgus deformity and large effusion of the affected knee with lateral joint line tenderness with full range of motion. MRI revealed lateral compartment arthrosis with incongruity of the lateral femoral condyle over the location of the previous osteochondral allografts and localized loss of articular cartilage. Failed incorporation of the lateral meniscal transplant was also evident. The patient was diagnosed with early arthrosis of the lateral compartment of her right knee, secondary to lateral meniscal deficiency and valgus alignment.

A varus-producing distal femoral osteotomy was performed to realign her knee (Figure 3). A fresh-frozen lateral tibial plateau allograft with an attached lateral meniscus allograft was implanted, and a fresh cold-stored osteochondral allograft was used to repair the defect in the lateral femoral condyle. Postoperatively, she was nonweightbearing with the knee in full extension for the first 6 weeks. Early range of motion was begun with partial weightbearing at 6 weeks with progression to full weightbearing by 12 weeks.

The patient reestablished full range of motion, was pain-free following surgery, and was able to participate in light sporting activities such as hiking. After 2 years, mild activity-related discomfort occurred over the lateral side of her knee but was otherwise asymptomatic.

Allograft tissue has been used to successfully restore the cartilage articular surface and meniscus. There is increasing interest in osteochondral allografts for the treatment of localized chondral defects. Cold-stored tissue with viable chondrocytes is now available for transplantation, which may increase longevity of these transplants. Transplanted meniscus allografts may degenerate over time, likely due to the challenging mechanical environment and incomplete biological incorporation. Thus, meniscus transplantation may be considered as an interim step to facilitate joint preservation in young patients and delay more definitive surgery. The ideal patient to receive a meniscal allograft transplant is young, has already had a total meniscectomy and complains of subsequent pain localized to the affected tibiofemoral compartment.

The biomechanical environment for the allograft requires consideration. Reconstruction of the knee requires correction of knee malalignment prior to any type of articular cartilage resurfacing or meniscus replacement; ligamentous instability also needs to be addressed. Even with the use of allograft tissue, it may not be possible to fully restore normal anatomy or joint kinematics, and progressive arthrosis may occur. By restoring joint alignment and architecture, current symptoms may resolve, and progression of joint degeneration may slow.
Case 4: Quadriceps Tendon Repair With Allograft in a Diabetic Patient

A 67-year-old man presented 1 year after a traumatic quadriceps tear that was sustained while playing golf, which was acutely repaired. The primary repair failed following early infection, as did the subsequent revision surgery. Surgical failures were likely to have been contributed by the patient’s poorly controlled non-insulin-dependent diabetes. On examination, a patella baja and palpable quadriceps tendon defect were detected. The patient had full passive extension, had a 45° extensor lag, and was able to flex to 120°.

Revision reconstruction of the ruptured quadriceps was undertaken using a semitendinosus allograft. The quadriceps tendon showed clear signs of atrophy, retraction, and fibrosis. A semitendinosus allograft was woven through the native quadriceps tendon using a Bunnell stitch with a 2.0 Ethibond suture (Ethicon, Johnson & Johnson, Somerville, New Jersey) and fixed to the patella. A dermal extracellular matrix graft (GraftJacket, Wright Medical Technology, Arlington, Tennessee) was overlaid on top of the repair to support tissue formation and further augment the repair.

Three months after this procedure, the augmented repair failed. Because of the multiple failures, it was elected to use a bone-tendon graft to allow bone-to-bone healing at the patellar site. There were no visible signs of an acute infection or inflammatory changes around the failed semitendinosus allograft. An allograft consisting of patellar bone with an attached patellar tendon was used and secured with 3 screws. To promote osteointegration of the bony allograft, an INFUSE collagen sponge embedded with rhBMP-2 (Medtronic, Memphis, Tennessee) was applied around the patella allograft. A Bunnell stitch and No. 2 Ethibond suture were used to attach the tendinous portion of the patellar tendon to the native quadriceps tendon.

Postoperatively, the knee was locked in full extension in a brace for 6 weeks. The patient was permitted to begin flexion to 60° at 6 weeks and then gradually progress to 90° of flexion by 12 weeks. The brace was removed at 12 weeks, and the patient continued with progressive quadriceps strengthening. The patient continued to maintain knee stability and improved knee function after 13 months (Figure 4).

There is limited consensus about the ideal management of chronic, retracted quadriceps ruptures. While the majority of quadriceps tears are repaired directly, chronic retracted tears usually do not have enough length to allow a tension-free repair, and there is thus a relatively high failure rate.

Case 5: Patellar and Trochlear Chondral Defects Treated With DeNovo Natural Tissue Graft Juvenile Allograft Cartilage

A 43-year-old man presented with a 9-year history of increasing pain in his right knee during activities of daily living with no history of preceding trauma. After 7 years, the patient underwent an open debridement of a patellar tendinopathy, microfracture of a trochlear chondral defect, and anterior interval release for apparent arthrofibrosis. Despite initial improvement, the pain recurred, particularly in full extension or with prolonged activities such as walking and stair climbing. There was no history of catching, locking, or instability.
On examination, the patient had a normal gait and neutral knee alignment, with anatomic patellar tracking and mild patellofemoral crepitus. There was a negative patellar apprehension sign, although the patellar tendon was thickened with mild tenderness to palpation. There was a full range of motion with intact ligamentous stability. Plain radiographs yielded normal results, and MRI demonstrated a 1-cm full-thickness lesion on the medial facet of the patella. Changes in the trochlea were consistent with the previous microfracture procedure. The patellofemoral chondral lesions and patellar tendinopathy were diagnosed as the source of the patient’s pain.

Arthroscopic examination of the joint demonstrated delamination of the articular cartilage overlying the medial patellar facet. The central trochlea had an area of fibrocartilaginous tissue (15 × 8 mm) at the site of previous microfracture. Upon probing, the central trochlea was noted to be soft and unstable. A final lesion (15 × 8 mm) was cleared down to the tidemark. The focal patellar chondral defect and trochlear lesions were resurfaced using DeNovo natural tissue allografts (Zimmer, St Louis, Missouri), which consist of minced human articular cartilage recovered from juvenile donor joints containing viable cells. The allograft cartilage pieces were suspended in thrombin and fibrin before molding, and fibrin glue was used to attach the implants to fit the lesion bed. Excellent fill was achieved.

To address the patellar tendinopathy, the nodular area in the midportion of the patellar tendon was sharply excised, and 2 platelet-rich fibrin matrix (PRFM) were implanted. A suture was utilized to reapproximate the edges of the healthy tendon around the PRFM implants. The implanted DeNovo cartilage graft appeared stable during flexion of the knee. The leg was positioned in a hinged knee brace locked in extension.

A period of strict nonweightbearing for 6 weeks and gradual progression to full weightbearing by 8 weeks, followed by physical therapy for 12 to 16 weeks, was recommended to the patient. Six months after surgery, MRI demonstrated excellent fill of cartilage lesions over the patella with overall fairly good integration with the remaining cartilage. The fill over the trochlea was moderate with good fill superiorly and more decreased fill inferiorly. There was no adverse synovial response (Figure 5). There was progressive remodeling of the patellar tendon at the repair site. Physical examination demonstrated good quadriceps tone and bulk, no effusion, and good patellar mobility. The knee motion was 0° to 130°. The patient noted that the pain had improved compared with preoperative status.

Articular cartilage has very limited healing potential because of lack of vascularity, high extracellular matrix–cell ratio, and the lack of local progenitor cells. Simply debriding a chondral defect or bone marrow stimulation techniques often produce suboptimal outcomes, as these approaches result in the production of biomechanically inferior fibrocartilage that consists of predominantly type I rather than type II collagen. Attempts to permit greater function and more durable repair tissue have led to the evolution of cartilage treatment strategies. Cartilage treatment strategies aim to restore the articular surface with a chondral or osteochondral fragment, using autogenous, allogeneous, or synthetic materials.

DeNovo natural tissue graft was used in this case, as it is composed of particulated allogeneic juvenile cartilage that was secured on the subchondral bone with a fibrin adhesive. The advantage of this approach was that no harvesting procedure was necessary. The rationale for the use of this tissue is that juvenile cartilage has higher proliferative capacity compared with adult tissue. In an equine model, local cloning and matrix reformation were observed after implantation.
Case 6: Bilateral Anterior and Posterior Glenohumeral Stabilization Using Achilles Tendon Allograft Augmentation in a Patient With Ehlers-Danlos Syndrome

A 28-year-old woman affected by hypermobility-type Ehlers-Danlos syndrome presented with a 10-year history of recurrent left shoulder subluxation and pain during driving or overhead activities. Shoulder subluxations persisted despite multiple stabilization attempts at an outside institution, which included a Magnuson procedure and a modified Bankart repair. On examination of the left shoulder, there was full range of motion, normal strength, and multidirectional laxity. The patient requested surgical stabilization because of her persistent symptoms.

The patient initially underwent anterior capsulorrhaphy with concomitant augmentation of the capsule using an Achilles tendon allograft. The superior, middle, and inferior glenohumeral ligaments were reconstructed with an Achilles allograft that was fixed to the glenoid rim using 4 suture anchors and to the humeral side with 2 rows of suture anchors laterally. Postoperatively, the patient was advised to maintain her arm in a sling for 8 weeks. External rotation was limited to neutral for the first 6 weeks because of the underlying connective tissue laxity. Progressive motion was gradually introduced after 6 weeks postoperatively. She began strength training at 12 weeks. Gradually the patient regained full functional range of motion and was pain-free at long-term follow-up.

Five years later, the patient complained of recurrence of her left shoulder symptoms, and clinical examination was consistent with a diagnosis of left shoulder posterior instability. She underwent a left posterior capsule reconstruction augmented with an Achilles tendon graft. Allograft augmentation was used because of the multiple repair failures of her native capsule during the previous management of the anterior capsule. Arthroscopic inspection revealed good tension in the anterior Achilles tendon allograft. To reconstruct the posterior capsule, 3 Mitek G2 suture anchors were used to fix the Achilles tendon allograft (Figure 6). A further 6 Mitek G4 suture anchors were used on the humeral side in medial and lateral rows. She was advised to avoid cross-body adduction and internal rotation for 6 weeks, and progressive restoration of motion was permitted between 6 and 12 weeks. At the two year follow-up, the patient noted significantly improved stability with resolution of her anteroposterior shoulder laxity.

Seven years later, the patient presented with multidirectional instability of the right shoulder and was treated with simultaneous anterior and posterior capsular reconstructions with Achilles tendon allografts. Again, an allograft was used to augment native capsule given the underlying connective tissue abnormality (Ehlers-Danlos syndrome). Following surgery, she avoided cross-body adduction to protect the posterior aspect of the repair and to avoid external rotation beyond 30° to protect the anterior repair. Active range of motion was delayed until 6 weeks postoperatively. To date, the patient’s pain and range of movement are better, without recurrent instability of the right shoulder.

A number of published reports describe the successful use of allografts for the revision of failed glenohumeral stabilization procedures and capsular deficiency (Achilles tendon, iliotibial band, tibialis tendon, and fascia lata grafts). The clinical applications for allograft augmentation are rapidly expanding. Most soft tissue defects can be successfully repaired with a direct primary closure, such as quadriceps tendon tears. Additional mechanical augmentation is often warranted when the natural structural integrity of soft tissues is compromised. This can occur in association with a number of conditions, such as diabetes, renal disease, rheumatoid arthritis, steroid use, fluoroquinolone antibiotic use, connective tissue disorders, lupus erythematosus, or secondary hyperparathyroidism; these increase tissue susceptibility to tearing and impair healing following primary repairs. Soft tissue allografts have mechanical and biological functions that are closely related and determine the clinical success. From a mechanical perspective, allografts provide a structural framework with the potential for incorporation into host tissue, and they closely approximate the normal anatomic structure. Allografts provide a biological scaffold to support ingrowth of host tissue during graft incorporation and eventual remodeling.

DISCUSSION

This series of cases demonstrates that allografts can be used to successfully repair a number of anatomical structures with functionally absent tissue. The clinical applications for allograft augmentation are rapidly expanding. Most soft tissue defects can be successfully repaired with a direct primary closure, such as quadriceps tendon tears. Additional mechanical augmentation is often warranted when the natural structural integrity of soft tissues is compromised. This can occur in association with a number of conditions, such as diabetes, renal disease, rheumatoid arthritis, steroid use, fluoroquinolone antibiotic use, connective tissue disorders, lupus erythematosus, or secondary hyperparathyroidism; these increase tissue susceptibility to tearing and impair healing following primary repairs. Soft tissue allografts have mechanical and biological functions that are closely related and determine the clinical success. From a mechanical perspective, allografts provide a structural framework with the potential for incorporation into host tissue, and they closely approximate the normal anatomic structure. Allografts provide a biological scaffold to support ingrowth of host tissue during graft incorporation and eventual remodeling.
Augmentation with allograft tissue may be useful for management of chronic retracted tendon tears. The decision to augment soft tissue repairs with an allograft rather than an autograft can be affected by a number of factors. Advantages of tendon allografts include a shorter operative time, smaller incisions, ease of sizing, and the availability of grafts. A number of disadvantages associated with autografts can also be avoided, such as the need for additional incisions for graft harvesting and donor site morbidity.

In cases where the native tendons may be diseased or atrophied, a primary repair is less likely to be suitable, particularly if the primary repair will be subjected to excessive tension. In such cases, graft augmentation is usually more likely to provide additional structural support that will maintain the integrity of the repair. Successful outcomes following the use of allografts are dependent on several factors, including the underlying status of the tissue being repaired or augmented, the local biological and biomechanical environment, and the type of allograft used. Further information is required to identify the optimal protocols for allograft processing and storage, as this likely has an important effect on graft incorporation and eventual function. Careful attention also needs to be paid to the postoperative management following allograft reconstruction. Gradual introduction of mechanical loading of the tissue through progressive joint motion is advised to allow gradual incorporation of the transplanted allograft tissue.

A number of concerns exist about the use of allografts. After implantation, tendon autografts and allografts undergo avascular necrosis followed by revascularization by host tissue and remodeling. The rate of incorporation is slower in allografts compared with autografts. For example, Jackson et al demonstrated in a goat model that tendon allografts show a greater decrease in structural properties, slower biological incorporation, and prolonged inflammation compared with patellar tendon autografts.

There is wide variation in the current protocols used for allograft processing, cleansing, and storage. There are different requirements for different tissue types (ie, tendon versus articular cartilage). The inherent immunogenicity of the allograft has the potential to elicit an inflammatory reaction that may affect graft revascularization, incorporation, and remodeling. Concerns also exist about the potential for disease transmission with the use of musculoskeletal tissue allografts. Disease transmission has been reported following allograft transplantation and includes both viremia such as HIV and hepatitis C and bacteriemia such as clostridium. The American Association of Tissue Banks has formulated guidelines to improve allograft safety. The risk of viral transmission has significantly decreased, and since 2007, all grafts are mandated to undergo nucleic acid testing for HIV and hepatitis C. Despite the distribution of millions of grafts prior to 2002, there were only 26 reported cases of bacterial contamination, implying a very low rate of bacterial transmission. Retrospective analyses comparing ACL autografts and allografts found that the latter were associated with a lower risk of surgical site infection, likely due to decreased operative time and smaller incisions when using allograft.

With the increasing use of allografts and a rising number of tissue banks, supplying sufficient tissue to meet demands and maintaining regulatory oversight remains a challenge. The requirement of sterilization and preparation can compromise the mechanical properties of the allograft and may contribute to the reported differences in graft incorporation. Exposure to 2 mrad of gamma irradiation at room temperature has been shown to affect tendon allograft material properties, such as strain and the maximum failure force, and result in a reduction in cortical bone strength in bending and torsion. No consensus has been reached to date on a universally accepted sterilization technique.

**CONCLUSION**

We have described a series of cases demonstrating the effective use of allograft tissue to augment functionally absent native tissue. Allografts are advantageous in cases with absent and/or poorly functioning native soft tissue. Ultimately, the quality of native tissue and surgeon preference are likely to determine whether augmentation of surgical repairs is warranted in cases with functionally absent tissue. In the future, the advent of synthetic scaffolds and other novel materials may reduce the need for allografts. However, until the mechanical properties of synthetic scaffolds are closely matched to native tissue and further understanding of biological incorporation of these materials is gained, the advantage offered by allografts will be difficult to surpass.

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