Allograft use in anterior cruciate ligament reconstruction surgery: a review of the current literature

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Abstract

Anterior cruciate ligament reconstruction (ACLR) surgery plays an important role in restoring stability and function to the knee joint following ACL rupture. Owing to an increase in activity levels and sports participation, ACLR has become one of the most commonly performed procedures world-wide. Graft choice may influence clinical outcomes, and therefore the optimal graft remains widely debated. Whilst, historically, autograft tissue has been the preferred choice, the past decade has seen a steady increase in the popularity of allografts. This demand is partly driven by improvements in graft availability, procurement processes and safety; but more importantly a desire to eliminate issues related to donor site morbidity from graft harvest. Despite this, there remains controversy surrounding the use of allograft in ACLR surgery, with much of the literature demonstrating conflicting evidence on functional and survivorship outcomes. In this article we review the current literature surrounding allograft use in ACLR, from the biology of allograft integration, through to outcomes in clinical practice.

Keywords ACL; allograft; anterior cruciate ligament; reconstruction; surgery

Introduction

The anterior cruciate ligament (ACL) is a complex central knee ligament that plays a primary role in providing stability to the knee joint in the sagittal plane by preventing anterior translation of the tibia relative to the femur.[1] In addition, the ACL contributes to rotational stability of the knee through limiting internal tibial rotation,[2] and it also plays an important role in limiting joint hyperextension.[3]

Anatomically, the ligament is made up of two components, the anteromedial bundle, which predominantly contributes to stability with increased flexion of the knee joint, and the posterolateral bundle, which plays a greater role in stability with the knee in extension and also rotation.

Knowledge of the biomechanics of the native ligament is important when selecting an optimal graft substitute for reconstructive surgery. An average load transmission of between 150 and 450 N is thought to transfer through the ligament during normal physiological gait, with a maximum load to failure of the native ligament being in the region of 2200 N.

Optimal ACL graft choice remains an open debate. Ideal graft characteristics (in no specific order) include a graft that is readily available, with minimal donor site morbidity. Furthermore, the graft properties should ideally match those of the native ACL. Relevant to allografts, there must be a low risk of disease transmission and immunogenicity. Rate of graft incorporation also plays an important role, particularly with relation to return to sport.

Historically, bone patellar tendon bone (BPTB) autograft has been considered the gold standard of practice since pioneering work by Kurt Franke in the 1970s.[4] In 1999, the American Orthopaedic Society for Sports Medicine (AOSSM) reported it as their graft of choice. Despite this, BPTB autograft is not without its limitations, notably associated graft harvest site morbidity. As a consequence of these drawbacks, alternative graft choices have been utilised, including hamstring tendon or quadriceps tendon autograft and allograft.

For the purpose of this article, we aim to focus on the use of allografts for ACL reconstruction (ACLR) surgery.

The background of allograft use

Early experimental studies reported positively on the biomechanical, biological and functional properties of freeze-dried allografts when compared to autografts in the animal model. In the 1980s, Webster et al. published their early results comparing freeze-dried flexor tendon allografts to patellar tendon autografts and the normal ACL in a dog model. They concluded that there was no significant difference in graft strength and mode of failure between the autograft and the allograft substitute.[5] Furthermore, in 1985 a study by Curtis et al. described their experience of freeze-dried fascia lata allografts and concluded no overt evidence of biologic incompatibility. Their histological analysis demonstrated the allograft to function as a collagenous scaffold, allowing revascularization and fibrovascular creeping substitution.[6]

It was these early studies that were the predominant driving force behind the popularization of the use of allografts in humans. The demand for the use of allografts in ACLR surgery has been steadily increasing over the past decade, with its popularity driven by completely eliminating the disadvantages associated with donor site morbidity. In 2013 an AOSSM survey of 833 surgeons found that allograft was selected in 27% of cases of ACLR surgery, with a greater propensity towards its use in patients 40 years and older. The justification for allograft use was decreased donor site morbidity, decreased postoperative pain,

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improved cosmesis and decreased surgical time. Data analysis from the Kaiser Permanene ACL Reconstruction Registry reported that of the 21,926 ACLR performed between 2005 and 2013, 41.4% were performed using allograft.

The biology of allograft integration

The vast majority of biological and biomechanical studies on the use of allograft versus autograft for ACLR have been limited to *in vitro* or animal studies. Though early biological and structural properties of the two groups have been reported as being comparable, the big difference appears to be in the rate of graft incorporation, with slower incorporation reported with the use of allograft.[7,8]

When using tendinous grafts, a 'tendon-to-bone' healing process occurs via bone ingrowth into the fibrovascular interface tissue that forms between the tendon and the bone. The development of Sharpey-like collagen is viewed as the earliest sign of osteointegration, and this is usually evident during the first 3–4 weeks. Granulation tissue surrounding the graft expresses high levels of vascular endothelial growth factor (VEGF) and fibroblastic growth factor (FGF), resulting in a high concentration of fibroblasts and blood vessels. There is an initial higher concentration of Type 1/2/3 collagen tissue which, with time, becomes more organized and concentrated Type 1/3 collagen. Progressive re-establishment of collagen fibre continuity between the tendon and bone facilitates the formation of the tendo-osseous junction, with most studies reporting the onset of bony ingrowth at the 6–8-week stage. Weiler et al.[9] reported a mature fibrocartilaginous tendon-to-bone junction at 12 weeks following interference fit fixation, which may be represented as a calcified cartilaginous zone at the intra-articular aperture. The late stages of soft tissue graft incorporation are characterized by a decrease in cartilage metaplasia and a resultant bony ingrowth, with significant ossification demonstrated at 6 months post-reconstruction. This process continues along the length of the tunnel for up to 2 years, as demonstrated by Hunt et al. in animal models.[9]

In contrast to this, BPTB graft healing follows a different process. Initial granulation tissue develops at the bone graft–tunnel interface, with a lesser degree of fibrous tissue formation. There is an initial increase in osteoclast activity, resulting in partial necrosis of the bone block followed by new bone formation via a direct healing process. The process of healing is comparably faster, with partial incorporation into the tunnel demonstrated by 6 weeks and full incorporation reported by 6 months.[10–12]

During the incorporation stages, the intra-articular portion of the graft undergoes a distinct remodeling process. Both animal and human *in vitro* and *in vivo* studies have demonstrated three characteristic stages of graft healing after ACL reconstruction: an early graft healing phase, with central graft necrosis; followed by a phase of proliferation, during which remodelling and revascularization is observed; and finally, a ligamentization phase, with characteristic restructuring of the graft towards the properties of the intact ACL. It is during the early remodelling process that the graft is most susceptible to intrasubstance failure.

Furthermore, histologically and radiologically, allografts demonstrate a slower rate of re-vascularization and a prolonged inflammatory response. The consequent delayed biologic incorporation, of up to 2 years in some animal studies,[13,14] is likely to correlate with the lower strength to failure rate and weaker graft construct reported early on and in some cases for up to 1 year post-surgery.[15]

Allograft procurement and processing

The procurement of allograft tissue varies between countries and healthcare systems. In the UK, supply is predominantly coordinated by tissue banks such as the National Blood Service, which in turn are regulated by the Human Tissue Authority (HTA).

The allograft retrieval process usually begins with procurement of tissue ideally within 24 hours of death of the donor. Donor screening plays a major role in reducing the risk of disease transmission. This is performed through both a detailed medical history and social history. The donor tissue then undergoes nucleic acid testing. All donor tissue is screened for human immunodeficiency virus (HIV), hepatitis B and C (HBV, HCV), *Treponema pallidum*, human transmissible spongiform encephalopathies, and bacterial and viral swabs.

Following procurement, the harvested graft undergoes the process of disinfection or sterilization, depending upon the tissue bank used. Disinfection is the process of removing contamination from allograft tissue. Sterilization is defined as the process of killing all forms of life. It is important to appreciate that not all commercially available allografts undergo the full extent of sterilization. Steps involved often combine techniques of mechanical and chemical washing using biological detergents, alcohols, antibiotics and hydrogen peroxide as part of the initial clean. This is followed by the process of terminal sterilization prior to tissue distribution for implantation.

The process of radiation sterilization has superseded conventional techniques such as ethylene oxide (EO), due to associated host tissue reaction with EO-treated grafts.[9] Irradiation is based on the ability of ionizing radiation to kill microorganisms, and remains a popular method for tissue allograft sterilization. Gamma irradiation has both bactericidal and virucidal properties. The technique involves exposing the soft tissue allograft to gamma rays from radionuclide isotopes ^{60}Co and ^{137}Cs. This in turn induces genetic damage and inhibition of cell division of microorganisms via one of two mechanisms: the direct or indirect effects of radiation. As the name suggests, the direct effect of radiation induces structural damage to DNA polymeric chains, inhibiting DNA synthesis and resulting in subsequent cell apoptosis. The indirect effect occurs due to aqueous free radical formation as a result of radiolysis of water in microorganisms. The generated free radicals and peroxy radicals then damage DNA molecules.

Whilst gamma radiation is considered an effective process, a number of studies have demonstrated a dose-dependent deleterious effect on the biomechanical properties of soft tissue allograft. Fideler et al.[16] have reported that the initial biomechanical strength of fresh-frozen allografts was reduced up to 15% when compared with fresh-frozen controls after 2.0 MRad of irradiation. Furthermore, work carried out by Curran et al.[17] studied the cyclic and failure mechanical properties of paired BPTB allografts, with and without low-dose irradiation of 20 kGy (2.0 MRad). Failure load averaged 1965 ± 512 N for irradiated grafts

and 2457 ± 647 N for non-irradiated grafts. Low-dose radiation, however, between 15 and 20 kGy has shown not to affect these biomechanical properties.[18,19] As a result, despite The International Atomic Energy Agency (IAEA) having defined the optimal radiation dose of 25 kGy (2.5 Mrad) for terminal sterilization of medical products, many tissue banks opt for lower-dose radiation to avoid compromising the biomechanical properties of the grafts.

Alternatively, application of radio-protective agents such as N-acetyl-L-cysteine and 1-ethyl-3-(3-dimethylaminopropyl) carbodiimide has also been described. The technique works by not only scavenging free radicals, but also promoting cross-linking within the collagen molecules, so as to reduce the risk of structural damage, allowing for higher dose radiation (50 kGy) to be delivered without compromise to the graft's biomechanical properties.[20,21]

Following the terminal sterilization process, the allograft is usually stored frozen (fresh frozen allograft), usually at a temperature of −40 °C. This technique of preservation is thought to have the least impact on the internal structure of the soft tissue, hence reducing the effect on the biomechanical properties of the graft. It does however, require facilities for storage at these extreme temperatures and limits the overall shelf-life of the graft. In contrast, there is a lesser role for freeze-dried (lyophilized) allograft use, despite it allowing easier storage at ambient temperature. This is due to the potential degradation of graft mechanical properties during the process, which involves reducing the water content to 6% of the initial weight by primary (sublimation) and secondary (desorption) drying processes.

Allograft safety

The potential for disease transmission with allograft use remains a concern for orthopaedic surgeons, with 72% of members of the AOSSM expressing concerns with regards to disease transmission in non-sterilized allografts.[22] The ability of all connective tissue allografts to transmit disease is documented in a number of experimental studies.[23,24] Disease transmission may occur via one of two principle methods; from an infected donor, due to failure of the initial screening process, or from cross-contamination during tissue processing. Sterility is expressed as a mathematic probability of relative risk. The Food and Drugs Administration (FDA) considers a sterility assurance level (SAL) of $\times 10^{-3}$ as an adequate level for implantation of biological material. This essentially translates as a probability of 1 per 1000 that a viable microbe exists on the implant.

Despite this, however, the overall risk of disease transmission remains very low, with the estimated risk of transmission of a viral infection from a screened donor being reported as ranging from 1 in 500,000 to 1 in 1.6 million.[25,26,27]

Deep infection following ACLR can be devastating. A recent large cohort study of 10,000 patients by Yu et al.[28] looked at the incidence of deep infection at 90-day follow-up following ACLR using allograft. They reported an overall incidence of 0.15%, with the most commonly identified organism being methicillin-sensitive *Staphylococcus aureus*. Interestingly, when the authors compared the incidence of infection in processed versus non-processed allograft, no statistically significant difference was identified.

Outcomes of allograft ACLR

Historically, ACLR has been regarded as a successful operation, with satisfactory outcomes reported in up to 97% of cases.[29,30] Whilst failure of ACLR requiring revision surgery occurs in up to 15% of cases, there is no clear consensus in the literature as to the true definition of a failed graft. Revision surgery itself is technically challenging and often associated with poorer outcomes.[31] A prospective study by the Multicentre ACL Revision Study (MARS) group published in 2010 reviewed the epidemiology of ACLR failure. They noted that the most common mode of failure was recurrent trauma (32%) followed by technical error (24%).[32] The cause of failure is often multifactorial, hence, identifying a single factor such as graft choice can be difficult.

Clinical outcomes between autograft and allograft use for ACLR have been examined in a number of studies, typically by measuring anteroposterior laxity with a KT1000 arthrometer to evaluate side-to-side difference, with greater than 3 mm deemed significant. Earlier animal studies provided sufficient ground to question the structural integrity, biological incorporation and overall strength of allograft when compared to autograft use for ACLR.[7] These findings were later echoed in a meta-analysis by Prodromos et al. in 2007.[33] In their study they reported a significantly higher abnormal stability rate with ACLR using allograft versus autograft (three times higher). Sub-group analysis of the allograft group demonstrated an increased abnormal stability with irradiated grafts versus non-irradiated grafts (31% vs 12%). This conclusion has been supported by a more recent meta-analysis by Kan et al. who reported that autograft was noted to be superior to irradiated allograft, but no difference was noted between autograft and non-irradiated allograft.[34]

Kraeulter et al. in 2013 published a meta-analysis of 5182 patients comparing BPTB autograft vs allograft. They noted that whilst KT1000, Lysholm and Tegner scores were significantly better in the autograft arm, return to pre-injury sport and anterior knee pain scores were significantly in favour of the allograft choice. Importantly, in their study, allograft had a threefold increase in re-rupture rates (12.7% vs 4.3%).[33]

The Multicentre Orthopaedic Outcomes Network (MOON) study initiated in 2002 and published in 2011 looked at failure rates of allograft versus autograft at 2 years. They reported two factors contributing to an increased failure rate of ACLR; namely patient age and graft type. They noted a fourfold increase in rupture rate in allograft when compared to autograft use. Furthermore, they also reported that the odds of graft rupture increased 2.3-fold for every 10-year decrease in age, with the highest risk group being 10–19 year olds.[35] They concluded that whilst allograft use may be suitable for the older cohort of patients, it must be used with caution in the younger population, where autograft may be the most appropriate option.

Despite the presented evidence thus far, there remains little consensus in the literature on outcomes of allograft use for ACLR, with a number of prospective studies from the late 1990s reporting no significant difference in outcomes between the two cohorts.[36,37] More recently, a systematic review by Foster et al. in 2010[38] compared BPTB and hamstring allograft versus autograft. Their results failed to show a statistically significant difference between the two groups, with respect to graft failure. Interestingly, they did report a statistically significantly higher IKDC 'A'

score (normal knee) in the allograft group. They also reported a higher complication rate in the autograft group. Edgar et al. in 2007[39] reported their results of a prospective randomized control trial comparing the use of autograft hamstrings versus allograft hamstrings. Akin to Foster et al., they failed to identify a significant difference in side-to-side laxity between the two cohorts. Both cohorts had similar failure rates (three cases in the autograft group and two cases in the allograft group). Functional outcome scores in the form of Lysohlm, Tegner and IKDC indices again failed to show a statistically significant difference between the two cohorts.

Summary

Allograft tissue plays an important role in ACLR surgery, especially in the revision setting. There is a global increased demand for allograft tissue for musculoskeletal reconstructive surgery. This is partly driven by improvements in graft procurement processes, sterilization and safety. Furthermore, the benefits of allograft, including predictability of graft quality and lack of donor site morbidity, have increased its popularity. Historical, *in vitro* and animal studies have reported potential disadvantages in relation to allograft incorporation and strength. However, the current available clinical outcome data remain inconclusive on this. What is evident from the current literature is that the cause of failure of ACLR is often multifactorial, and hence further research in the form of high quality randomized controlled trials with matched cohorts is needed to draw a more decisive conclusion on the subject of allograft utilisation. ◆

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