This case study describes a new technique in the treatment of severe ankle arthritis. Traditionally, degenerative joint disease (DJD) of the ankle is treated through traumatic surgical modalities such as total ankle replacement (TAR) or fusions. In this case, a less traumatic technique was performed with outstanding results. Ankle anterior spurring was resected, talar dome lesions were filled with calcium phosphate, and living amniotic cells were injected into the ankle joint and extensor retinaculum.

Case Presentation

Prior to seeing us, the patient was treating her severe ankle pain with routine corticosteroid injections via her extensor tendon complex. The ankle capsule was then closed and the extensor retinaculum was repaired and both were infiltrated with 2 ml of frozen, living amniotic cells.

Procedure

A curvilinear incision was made at the anterior aspect of the right ankle. The incision was carried deep utilizing both sharp and blunt dissection taking special care to retract and protect the superficial vessels and nerves. The tibialis anterior tendon was identified and freed from its surrounding scar tissue and the extensor retinaculum was completely released. The ankle capsule was incised and reflected. The spur across the anterior ankle was resected with a sagittal saw and rough surface was rased and smoothed. A severe loss of cartilage and a large osteochondral defect measuring 1.5 cm in diameter was noted on the talar dome. The defect was located on the dorsal medial aspect of the dome. Subchondral drilling was performed on the talar dome. From a lateral, separate port, a probe was drilled into the talar body and calcium phosphate paste mixed with flowable frozen amnion was utilized to fill the area. Some flowable amnion was utilized at the sites of subchondral drilling as well. Prior to closure an amniotic membrane matrix was laid over he anterior lip of the tibia and curved into the ankle joint partially over the distal talar body. Another amniotic membrane matrix was laid over the extensor tendon complex. The ankle capsule was then closed and the extensor retinaculum was repaired and both were infiltrated with 2 ml of frozen, living amniotic cells.

Literature Review

Osteoarthritis (OA) is a condition characterized as a degenerative joint disease. OA is the most prevalent form of arthritis in the U.S. due to aging population and obesity (5). An osteoarthritic joint has a cellular cartilageous scaffold, which undergoes stresses at the cellular level leading to extracellular matrix (ECM) degradation thus ultimately destroys the articular cartilage. Cartilage is OA initially recognized as an inflammation in the early stages of the disease process (3). On a molecular level, osteoarthritic joints have a decrease in proteoglycans (PG) in the cartilage matrix. PGs are in part in effectively providing a scaffold in the ECM to assist in developing cartilage. A lack of PGs ultimately causes lesions to form and causes an inflammatory cascade effect releasing several proinflammatory cytokines which are upregulated and lead to structural changes of the joint and cartilage degradation (3). Amniotic membrane is a multilayer tissue that is connected to the chorion layer, which together form the placenta. Amnion/chorion tissue is widely studied in orthopedic literature for arthritis knees or shoulder but rarely shown to be used in the foot and ankle joint for osteoarthritis. Amniotic tissue is an ECM which acts as a scaffold for building new cells which comprise of collagen types I,II,III,IV, V, VI, hyaluronic acid (which is also important for cartilage for healing) and a multitude of growth factors (2). Amniotic tissue can be used to widen an anti-inflammatory effect, which inhibits the aforementioned inflammatory cascade. Amniotic tissue also helps in homodifferentiation and in several in-vitro studies reported about human osteoarthritic tissue scaffolds, the amniotic cells did not differentiate into other cellular processes, rather, they integrated and repaired damaged articular cartilage (2). In a study by Willett et al, it showed that intrarticular injection of an amniotic membrane allograft can shock cartilage damage in a rat medial meniscal tear model (MMT) model of OA (4). In measuring off the synovium of the injected rats model after 21 days, a “single intra-articular injection of dehydrated human amnion chorion membrane (dHACM) had a significant chondroprotective effect, slowing proteoglycan loss and preventing lesion formation” (4).”

Results

Post-operatively, she was non-weightbearing in a cast for 3 weeks transitioning to a CAM boot and additive 25% weightbearing for the next 5 weeks. ROM physical therapy began at week 6 post-op. At 1 year post-op, she has no evidence of equinus, no ankle pain, no gait abnormalities, and is able to fully squat and be active.

References