ABSTRACT

Humeral shaft non-unions occur in 2-10% of all fracture cases. Increased incidence of these non-unions can be associated with ORIF, comminution, high impact injuries, bone loss or fracture gap. Treatment guidelines for fracture non-union state that fractures with gaps greater than 4 cm should be treated with vascularized fibular autografts or transportation with an external fixator. Unfortunately these modalities carry considerable donor site morbidity and patient will experience considerable discomfort, especially when dealing with an external fixator. This report demonstrates how the use of a nonvascularized fibular strut can be effectively utilized as an alternate treatment modality for large humeral shaft non-union gaps. Further studies should be conducted to support this method as a viable treatment option for non-union gaps greater than 4 cm.

Index words: fibular, strut, allograft, cancellous, chip, non-union

Bone loss can complicate fracture healing and, as previously mentioned, is a risk factor for developing a non-union. Managing patients with extensive bone loss can be a challenge and advances in treatment have been studied. A defect of 3 to 4 cm can undergo acute shortening and gapping without the arm being impaired. For defects greater than 4 cm, the literature supports the use of an autologous fibular strut transfer and bone transport (17-20); however they offer significant donor site morbidity and the utilization of an external device for extended periods of time (respectively).

To our knowledge, the use of fibular allograft (nonvascularized) as a strut graft for large gaps of bone loss (> 4 cm) has not been described. In this report, we present the case of a diabetic woman who presented with history of humeral shaft infected nonunion that failed two prior attempts of surgical treatment at another institution, and presented with loose hardware and a 10 cm gap of bone loss with positive CRP and ESR parameters. We will describe the use of a fibular strut allograft for augmentation of dual compression plating and the use of both BMP and DBM for biologic augmentation of fracture healing, which resulted in excellent bone formation with a rigid fixation and functional arm.

Case History

This is the case of a 47 year-old woman with a past medical history of hypertension and Diabetic Mellitus type II, 3 years ago she was in a car accident while driving and presented to our Orthopedic clinics with instability in her left arm and associated with decreased ROM of elbow, shoulder, and forearm. Patient refers that she was in a car accident three years ago, which resulted in a humeral fracture, and required surgical intervention for open reduction and internal fixation by a community orthopedic surgeon at the time. X-rays from two years prior to visit showed infected fracture non-union with poor bone formation and mild scarring around compression plating (see Figure 1). Subsequent films three years after initial surgery showed progressive osteolysis, bone resorption and hardware loosening with an evolving non-union with an 8 cm gap in the humeral shaft (see Figure 2).

Laboratory data revealed WBC count in 4.9, CRP was found to be positive, glucose level was at 250, and Hba1C was at 8. Due to the high probability of chronic infection, the patient was scheduled for removal of hardware and bone sauerization. A posterior approach to the humeral shaft was taken. Areas of non-vascularized bone and large area of fibrotic nonunion were found with no gross purulent discharge. The loose hardware was removed in its entirety as well as debridement of large areas of bone fragments, which left a 10 cm bone loss gap in the humerus. Cultures were taken and the area was irrigated with 3 liters of 0.9 normal saline solution. The incision was closed using vicryl (for muscle tissue) and prolene (skin). Patient was subsequently placed in prophylactic IV vancomycin therapy for two weeks. During this time, pathological report showed no abnormality in tissue specimen except for necrotic fragments of bone, cartilage and connective tissue. Microbiology report from orthopedic device and tissues revealed no growth after 5 days.
THE USE OF A FIBULAR STRUT ALLOGRAFT WITH DBM, CANCELLOUS CHIPS AND BMP FOR A 10 CM HUMERAL SHAFT INFECTED NON-UNION: A Case Report

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INTRODUCTION

Humeral fractures make up about 5% to 8% of all fractures and 10% of all long bone fractures (1-4). Approximately 30% of all humeral fractures occur at the diaphysis. Most of these fractures can be treated non-surgically with excellent outcomes. Sarmiento et al (5) reported union rates of 94% for open and 98% for closed fractures of the humeral shaft after functional bracing. Nonunion of the humeral shaft occurs in 2% to 10% of non-surgically treated fractures and close to 15% of fractures treated by open reduction and internal fixation (4-7). Increased incidence of nonunion is associated with open fractures, high impact injuries, bone loss or fracture gaping, soft tissue interposition, unstable fracture patterns, segmental fractures, impaired blood supply, infection, and internal treatment with traction (4). A humeral fracture is classified as nonunion if no progression towards healing is witnessed in three months or there is evidence of a lack of union six months (7). Union is expected to occur between 12 to 16 weeks. Risk factors for developing nonunion include both biologic host factors (smoking history, disrupted blood supply, medical comorbidities) and mechanical (fracture displacement and inadequate immobilization) factors. Patients who fail nonsurgical management for humerus fractures and develop a nonunion should preferably undergo surgery with the ultimate goal of creating a biologic environment that favors bone healing and providing a stable mechanical construct that allows for early motion. Currently, the standard of care for humeral nonunion is open reduction and internal fixation with rigid compression plating and autogenous bone grafting (8). Success has been reported with the use of demineralized bone matrix (DBM) with or without bone morphogenic protein (BMP) (9-12) and with plating (9-12). Biologic augmentation with BMP-2 and BMP-7 has been performed with continued success (9-12). A defect of 3 to 4 cm can undergo acute shortening and gapping without bone healing, but function and motion of the arm is not impaired. For defects greater than 4 cm, the literature supports the use of a fibular strut transfer and bone transport (17-20); however they offer significant donor site morbidity and the utilization of an external device for extended periods of time (respectively).

Bone loss can complicate fracture healing and, as previously mentioned, is a risk factor for developing a nonunion. Managing patients with extensive bone loss can be a challenge and advanced treatment options are needed. A defect of 3 to 4 cm can undergo acute shortening and gapping without bone healing, but function and motion of the arm is not impaired. For defects greater than 4 cm, the literature supports the use of a fibular strut transfer and bone transport (17-20); however they offer significant donor site morbidity and the utilization of an external device for extended periods of time (respectively).

ABSTRACT

Humeral shaft non-unions occur in 2-10% of all fracture cases. Increased incidence of these non-unions can be associated with ORIF, comminution, high impact injuries, bone loss or fracture gaping. Treatment guidelines for fracture non-union state that fractures with gaps greater than 4 cm should be treated with vascularized fibular autografts or transportation with an external fixator. Unfortunately these modalities carry considerable donor site morbidity and patient will experience considerable discomfort, especially when dealing with an external fixator. This report demonstrates how the use of a nonvascularized fibular strut can be effectively utilized as an alternate treatment modality for large humeral shaft non-union gaps. Further studies should be conducted to support this method as a viable treatment option for non-union gaps greater than 4 cm.

Index words: fibular, strut, allograft, cancellous, chip, humeral, shaft, infected, non-union

To our knowledge, the use of fibular allograft (nonvascularized) as a strut graft for large gaps of bone loss (> 4 cm) has not been described. In this report, we present the case of a diabetic woman who presented with history of humeral shaft infected nonunion that failed two prior attempts of surgical treatment at another institution, and presented with loose hardware and a 10 cm gap of bone loss with positive CRP and ESR parameters. We will describe the use of a fibular strut allograft for augmentation of dual compression plating and the use of both BMP and DBM for biologic augmentation of fracture healing, which resulted in excellent bone formation with a rigid fixation and functional arm.

Case History

This is the case of a 47 year-old diabetic woman with a past medical history of hypertension and Diabetic Nerve type II, I, and IV. She presented to our Orthopedic Clinics with instability in her left arm associated with decreased ROM of elbow and wrist. Patient refers that she was in a car accident three years ago, which resulted in a humerus fracture, and required surgical intervention for open reduction and internal fixation by a community orthopedic surgeon at the time. X-rays films two years prior to visit showed infected nonunion with poor bone formation and mild scarring around compression plating (see Figure 1). Subsequent films three years after initial surgery showed progressive osteolysis, bone resorption and hardware loosening with continuing non-union with an 8 cm gap in the humeral shaft (see Figure 2).

Laboratory data revealed WBC count in 4.9, ESR level was reported at 96, CRP was found to be positive, glucose level was at 250, and HbA1c was at 8. Due to the high probability of chronic infection, the patient was scheduled for removal of hardware and bone sauerization. A posterior approach to the humeral shaft was taken. Areas of non-viable bone and a large area of fibrotic nonunion were found with no gross purulent discharge. The loose hardware was removed in its entirety as well as debridement of large areas of bone fragments, which left a 10 cm bone loss gap in the humerus. Cultures were taken and the area was irrigated with 3 liters of 0.9 normal saline solution. The incision was closed using vicryl (for muscle tissue) and prolene (skin). Patient was subsequently placed in prophylactic IV vancomycin therapy for two weeks. During this time, pathology report showed no abnormality in tissue specimen except for necrotic fragments of bone, cartilage and connective tissue. Microbiology report from orthopedic device and tissues revealed no growth after 5 days.
On the second week after the first surgery, patient was taken again to the operating room for open reduction and internal fixation. Patient was placed in right lateral decubitus position. Skin incision was done through previous scar. After verifying correct humeral length, non-union was fixed utilizing dual compression plates with 6 distal and 6 proximal screws. A 10 cm non-vascularized fibular allograft was then placed as a strut to act as support for the 10 cm bone gap and was fixed utilizing one screw distally. Cancellous chips were first placed around fibular strut, followed by demineralized bone matrix (DBM) paste around the cancellous chips. Bone Morphogenic Protein (BMP) was then placed between the plates, posterior to the DBM paste, cancellous chips and fibular strut construct. The wound was again closed using vicryl and prolene sutures.

Patient was discharged from orthopedic ward two days postop with only oral antibiotic prophylaxis with instructions for limited motion and no lifting of objects. After two months of follow-up and physical therapy, x-rays revealed callus formation over fibular strut graft in both distal and proximal segments, with both graft and hardware in place (see Figure 3). Elbow range of motion improved significantly from 30 degrees of extension to 20, and from 80 degrees of flexion to 90 degrees. Laboratory results showed CRP levels less than 6 and an ESR level of 20. The patient was judged to have a fixed humeral shaft non-union and was returned to full activity and asked to return every four months for the next year to follow up fracture healing. After two year follow up, no evidence of deterioration or infection was found and patient reported return to full activity with no pain (see Figure 4). Physical examination showed rigid construct with no instability. Patient was discharged from clinics and asked to return as needed.

**DISCUSSION**

Recommended treatment for humeral shaft non-union with a gap greater than 4 cm includes the utilization of vascularized fibular autograft for restoration of length while providing live bone tissue. Another described alternative is the use of external hard-ware for bone transport. While effective, both these techniques have serious downsides: Autologous bone transport has significant donor site morbidity, while bone transport is a lengthy procedure that can take months to complete, all with an uncomfortable external device.

This article describes a possible alternate treatment for large gap non-union humeral shaft fractures, specifically those greater than 4 inches with great instability. The 47 year old woman described most likely attained her non-union due to chronic infection, possibly facilitated by her uncontrolled diabetes mellitus, as evidenced by her elevated blood sugar and Hba1c levels. This patient with a 10 cm humeral shaft non-union gap was adequately treated using non-vascularized fibular allograft as a strut to maintain gap length, while utilizing DBM, BMP and cancellous chips to create an osteogenic and osteoconductive environment that eventually caused callous formation that effectively closed the 10 cm gap and completely healed the non-union in two months as evidenced by serial x-rays films.

The use of a non-vascularized fibula along with BMP, DBM and cancellous chips can be effectively utilized as an alternate treatment modality for large humeral shaft non-union gaps. Unlike the current treatment recommendations, the use of non-vascularized fibula as a strut has no donor site morbidity and contains no external hardware, therefore providing the patient with a much more comfortable recovery. Future studies can be directed towards determining the safety and efficacy of this treatment option compared to fibular autograft and bone transportation.

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![Figure 2: Subsequent x-rays 3 years after initial surgery showed progressive osteolysis, bone resorption and hardware loosening with continuing non-union with an 8 cm gap in the humeral shaft.](image2)

![Figure 3: 2 month follow-up x-rays showed callus formation over fibular strut graft in both distal and proximal segments, with both graft and hardware in place.](image3)

![Figure 4: Two years after most recent surgery, graft remains in place and fracture non-union appears rigid with no instability.](image4)
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ABSTRACT

Kaposi’s sarcoma is a rare malignant requiring infection with human Herpes virus for development. We report a case of a 76-year-old immunocompetent male with recurrent leg cellulitis. The cellulitis eventually developed into a non-healing ulcer and a palpable nodule consistent with nodular Kaposi’s sarcoma.

Index words: rare, neoplasm, immunocompetent, patient, Kaposi, sarcoma

INTRODUCTION

Kaposi’s sarcoma (KS) is a rare malignant, slowly progressing, mesenchymal neoplasm characterized by proliferation of connective tissue and capillaries. Clinical presentation is usually as nodules and red-purple plaques. It occurs most often in older men of Mediterranean or Central/Eastern European ancestry, in whom the lesions usually occur on the distal extremities, particularly the lower legs and feet.

The exact nature of the disease is not clear. It remains controversial whether the endothelial cells are of vascular origin, lymphatic origin or both. Current data support the notion that KS is a vascular hyperplasia with a tight link to human herpes virus 8 infection. The virus was first identified in KS cells of a patient with AIDS but later, it had been linked convincing with all four types of KS, an association that is necessary, but not sufficient to develop KS. Other factors are also important in the etiology of Kaposi’s sarcoma, such as immunosuppression. We report the development of KS in an immunocompetent male patient.

Case History

This is a 76-year-old male patient with past medical history of hypertension, unstable angina pectoris, chronic obstructive pulmonary disease, gastroesophageal reflux, and colon adenocarcinoma status post resection in year 2012 on complete remission. The patient presented to his primary care physician with the chief complaint of a left heel lesion at the medial aspect of three months of evolution (see Figure 1). This lesion was an open ulcer of approximately one cm of diameter, elevated approximately 3mm, with well-defined borders, draining serous but as purulent material, and friable (see Figure 2). There was absence of pain or tenderness upon palpation of the area. The patient received oral and

Case Report/Reporte de Casos

A VERY RARE NEOPLASM IN AN IMMUNOCOMPETENT PATIENT

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