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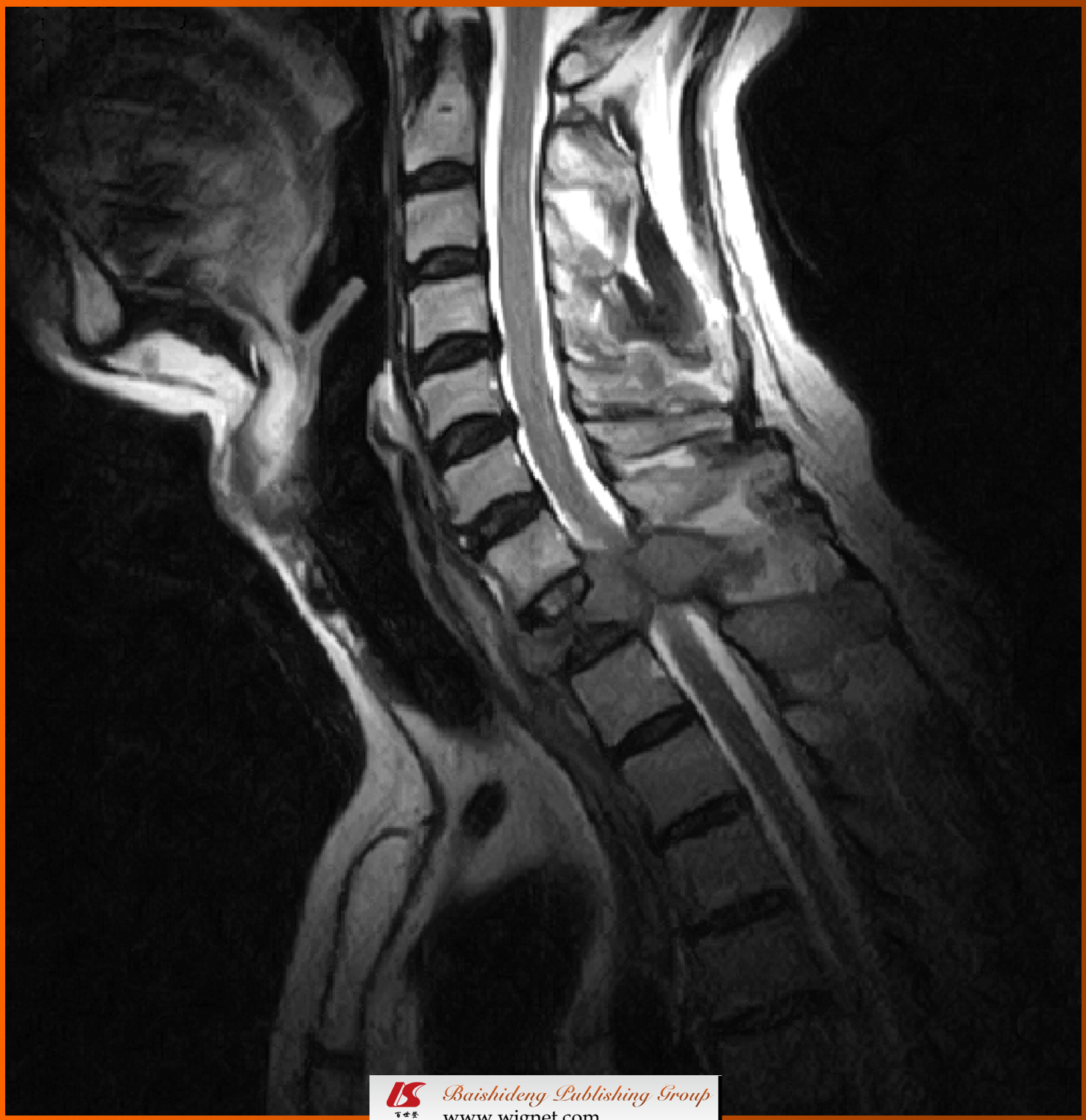

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TOPIC HIGHLIGHT

Quanjun Cui, MD, *Series Editor*

Reconstruction options for acetabular revision

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Abstract

This article summarizes reconstruction options available for acetabular revision following total hip arthroplasty. A thoughtful methodology to the evaluation and treatment of patients with implant failure after joint replacement is essential to guarantee accurate diagnoses, appropriate triage to reconstruction options, and optimal clinical outcomes. In the majority of patients who undergo acetabular revision, factors such as bone loss and pelvic discontinuity provide a challenge in the selection and implementation of the proper reconstruction option. With advanced evaluation algorithms, imaging techniques, and implant designs, techniques have evolved to rebuild the compromised acetabulum at the time of revision surgery. However, clinical outcomes data for these techniques continue to lag behind the exponential increase in revision hip arthroplasty cases predicted to occur over the next several years. We encourage those involved in the treatment of patients undergoing hip replacement surgery to participate in well-designed clinical studies to enhance evidence-based knowledge regarding revision acetabular reconstruction options.

Key words: Hip; Arthoplasty; Revision; Acetabulum; Tantalum

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EPIDEMIOLOGY OF REVISION HIP ARTHROPLASTY

Over the past several decades, total hip arthroplasty has become recognized as an effective treatment option for the reduction of pain and disability associated with advanced degenerative arthritis with successful clinical outcomes. As population factors, such as aging and obesity, drive the demand for primary hip arthroplasty, the demand for revision joint replacement will continue to rise as well. Recent data analyses have shown that in addition to the substantial increase in prevelance of primary total hip arthoplasty procedures, the rates of revision procedures are expected to rise as well[1]. Despite attempts to improve implant design and evade the previous pitfalls of early reconstruction techniques, the prevalence of revision hip arthroplasty cases has not declined[2]. In the United States, 46 000 hip revisions were performed in 2004 and this number is expected to be more than double by 2030[3].

In 2009, Bozic *et al*[4] released data detailing the causes of revision total hip arthroplasty. Their review demonstrated the most common causes of total hip revision, regardless of component, included instability/dislocation, mechanical loosening, and infection. Isolated acetabular component revision comprised 12.7% of all revision hip procedures and instability/dislocation was reported as the most common indication. As such, a surgeon'

s pre-operative planning and understanding of suitable reconstruction options for acetabular revision is essential for the growing population of patients who will undergo total hip replacement.

PATIENT EVALUATION

There is a wide spectrum of signs of symptoms that can occur in the setting of acetabular component failure. Pain is a common presenting complaint and often times groin pain can represent acetabular component failure while thigh pain may be correlated to femoral component failure. Clinical patient presentation ultimately depends on the underlying cause, whether it be infection, polyethylene wear, instability, or aseptic loosening. The steps towards comprehensive evaluation of a painful total hip have been described in the arthroplasty literature, and these guidelines must be implemented to eliminate systemic or infectious etiologies that could preclude a definitive single-stage reconstruction of the acetabulum[5]. Leg-length discrepancy, joint deformity, location of prior incisions, functional status and baseline neurologic deficits should be detected and documented during the pre-operative evaluation as well. It is important to note that the patient population in this setting could be older with osteopenia, compromised soft tissues, and multiple medical comorbidities.

In addition to obtaining a good history and physical examination data, radiographic and advanced imaging modalities are useful in defining the extent and location of bone loss associated with acetabular component failure. Anterior-posterior pelvis radiographs and frog-leg lateral views of the involved hip can be supplemented with Judet views for evaluation of the acetabular columns. They can also give clues to the underlying cause of the component failure. Three-dimensional computed tomography is often helpful in quantifying the presence and severity of osteolytic lesions. This is especially important in the setting of prior revision hip surgery or prior irradiation where radiographs may under-estimate the amount of bone loss that is present. The information obtained from these studies assists in classifying the extent of the acetabular defects, thereby guiding treatment options. Preoperative computed tomography (CT) angiogram of iliac vessels is advised when protrusion of the failed acetabular component past Kohler's line is substantial.

CLASSIFICATION SYSTEMS FOR ACETABULAR DEFECTS

In orthopaedics, classifications are judged on their reliability, reproducibility, and ability to guide treatment plans and predict outcomes. With regard to revision hip arthroplasty, classification systems for acetabular defects have been used to present the severity of bone loss that will likely be found intra-operatively, allowing for appropriate selection of reconstructive options[6]. In 1989, D'Antonio *et al*[7] first described what is now commonly known as the American Academy of Orthopaedic Surgeons (AAOS) classification system of acetabular abnormalities after total hip replacement. The AAOS classification system distinguishes between segmental and cavitary defects, and also subdivides the presence of pelvic discontinuity. Though widely used in the literature, this classification system does not account for the location or size of acetabular defects. A decade after the introduction of the AAOS classification system, Saleh *et al*[8] released results validating the Gross classification system, which quantified the extent of contained versus uncontained bone loss and implications related to use of morselized bone graft during revision reconstruction.

Perhaps the most widely cited and clinically implemented system, the Paprosky Classification was developed to establish acetabular defect type, size, and location for a collective guidance towards the selection of appropriate reconstructive options for revision surgery. Developed from a systematic review of bone loss seen in 147 failed acetabuli, this system was based on four radiographic measures obtained from an anterior-posterior radiograph of the pelvis: superior hip center migration, ischialosteolysis, the position of the implant relative to the Kohler (ilioischial) line, and teardrop osteolysis (Table 1). Unique to this methodology, defects were classified by type to indicate whether the remaining acetabular structures are completely supportive (Type I), incompletely supportive (Type Ⅱ), or unsupportive (Type Ⅲ) of an implanted component. In the original study, reconstructive guidelines for allograft selection were determined by the extent of remaining structural support according to defect type. Today, this classification system continues to provide a useful treatment algorithm, even with the availability of a wider variety of modular metal augmentation and reconstruction options.

SURGICAL RECONSTRUCTIVE OPTIONS

Treatment of acetabular component failure and associated bone defects depends on patient characteristics, the degree and location of bone loss, the ability of the columns to support biologic fixation, and the presence of pelvic discontinuity. The ultimate goal of revision acetabular reconstruction should be to obtain stable fixation and restore the hip center[5]. The various traditional and newer revision options for the acetabular component are discussed below and outlined in Table 2.

Isolated polyethylene liner exchange

The technique of isolated polyethylene liner exchange is useful in the setting of substantial polyethylene wear and osteolysis with evidence of a stable acetabular component. Previous studies have demonstrated the relationship between polyethylene wear and progressive osteolysis with compromised bone stock[9]. Liner exchange with highly cross-linked polyethylene has been shown to decrease average wear rates significantly[10].

Table 1 Paprosky classification system for acetabular defects[26]

Type	Description			
	Superior hip center migration	Is chialosteolysis	Kohler line	Teardrop
I	Minimal	None	Intact	Intact
ⅡA	Mild	Mild	Intact	Intact
ⅡB	Moderate	Mild	Intact	Intact
ⅡC	Mild	Mild	Disrupted	Moderate lysis
ⅢA	Severe	Moderate	Intact	Moderate lysis
ⅢB	Severe	Severe	Disrupted	Severe lysis

To justify isolated liner exchange, the modular metallic shell should be well-fixed and appropriately oriented[11]. This should be evaluated both pre-operatively and intra-operatively. Once the stability of the acetabular prosthesis is confirmed and liner exchange is contemplated, it is then important to consider the adequacy of the locking mechanism between the liner and the metallic shell. If the locking mechanism is compromised, one may consider cementing a new liner into the fixed metallic shell to prevent micromotion between the two surfaces for primary fixation. The clinical track-record and historical performance of the implant should be considered along with the available liner and head size options offered by that particular component.

Hemisphere reconstruction

Historically, treatment options for acetabular component instability or malposition included use of cemented acetabular all-polyethylene prostheses implanted with the same techniques that had been employed for the primary arthroplasty procedure. The results of the cemented revision procedures were poor, resulting from mechanical failure secondary to poor cement interdigitation and fixation leading to excessive micromotion within the acetabular bed[12]. In contrast, hemispheric metal cups with porous coating and associated techniques have been developed that encourage bone in-growth and held the promise of durable biologic fixation.

Cementess hemispherical porous-coated implants are the most commonly used implants for acetabular reconstruction in North America. With supportive and viable host bone and a reliable in growth surface, the cups address most revision problems encountered. Initial stability is provided with a press-fit and screw fixation. Cavitary defects are addressed with morselized bone graft.

These components are acceptable for patients who have not shown evidence of hip center migration or significant pelvic discontinuity (Paprosky types Ⅰ, ⅡA and ⅡB), which can be assessed pre-operatively as well as intra-operatively[5]. It is generally accepted that at least 50% of the bone stock must be present to support the cup. Internal fixation with screws is also advocated to supplement the in-growth of the press-fit component. When there are focal superior segmental or cavitary defects identified at that time of revision, modular metallic augments, structural allograft, or morselized impacted allograft[13] may be added to supplement the acetabular bed. Care must be taken to maintain the appropriate orientation of the revision cup despite the presence of augments in the dome. Park *et al*[14] recently published long-term data from their cohort of patients who underwent revision hip arthroplasty with use of a cememntlessacetabular shell. In this group, survivorship, with revision of the shell for aspetic loosening or evidence of loosening as the endpoint, was 95% at 20-year follow-up.

Table 2 Reconstruction options for acetabular revision

Acetabular revision option	Clinical pearls
Isolated liner exchange	The stability and orientation of the acetabular metal component should be confirmed at the time of revision, liner may be cemented if needed
Hemispheric porous-coated cup	May be used in conjunction with adjunct techniques of bone grafting, screw fixation recommended
Highly porous metal cup	Appears to be effective in achieving biologic fixation in cases of severe bone defects, augments may be used for structural support, cup-cage construct can be used to offload cup
Antiprotrusio cage	Useful in cases of severe bone defects or pelvic discontinuity, spans areas of healthy host bone and accommodates bone grafting deep to the cage, relies on mechanical fixation alone
Customized triflange implant	Requires several weeks or month to obtain implant, serves as a good salvage option in cases of catastrophic bone loss and discontinuity, may achieve biologic fixation

Jumbo cups following allografting for focal defects also have a role in acetabular revision surgery. There is no universally-acceted definition of what diameter defined the jumbo cup. Jumbo cups are loosely defined by the ratio of component size to the pelvis and the hip joint, as compared to the size of the original implant[15]. These jumbo cups offer numerous advantages in regards to maximizing the contact area between the cup and host bone when a larger reamer is necessary to establish rim contact. The larger components can also accommodate larger femoral heads, reducing the rate of dislocation. A large mismatch between a large shell and a small femoral head may increase the rate of impingement and reduce the soft tissue constraints to dislocation and is to be avoided. Another potential disadvantage with the jumbo cup comes with displacement of the femoral head hip center into a lateral inferior position, which has been reported. Nevertheless, satisfactory mid-term results and survivorship are documented with use of acetabular jumbo cups in revision arthroplasty, with survival rates as high 92% at 14 years[16,17].

High hip center placement of an uncementedacetabular hemispheric component is another option when there is a defect in the superolateral dome or posterior column that precludes the standard placement of a hemispheric shell in a more anatomic location. To accommodate the defect, the shell is placed in a more superior position. Accordingly, it is often necessary to do concomitant procedures at the time of revision

to ensure that soft tissue tension and appropriate leg lengths are restored. As superior placement of the hip center can also be associated with lateralization of the component, there have been some reports of increased dislocation or loosening rates with high hip center placement[16]. A high hip center is also disadvantageous from a biomechanical standpoint and will typically result in a limp. Hip stability may be compromised due to the small head size and bony impingement. A long term follow-up study by Hendricks et al, however, reported survivorship of 89% after 15 years in their cohort of patients who underwent high placement of noncementedacetabular components[18].

A traditional contraindication to use of a cementless hemisphere revision component was recent pelvis irradiation, although preliminary reports of successful results using newer porous metal technology suggests that this recommendation may need to be revisited. The presence of pelvic discontinuity and severe bone loss (Paprosky type ⅡC or Ⅲ) may warrant the use of techniques that can provide more stability for the implant in the setting of compromised bone stock.

Highly porous metal components

In recent years, highly porous metal components have become popular options for both primary and revision arthroplasty procedures. Tantalum implants (Trabecular Metal, Zimmer, Inc, Warsaw, Indiana, United States) were developed to provide increased porosity and a trabecular bone-like configuration to allow for rapid and extensive bone in growth along with good initial stability in bone. Some designs incorporate a locking mechnism for the polyethylene insert, whereas others require a cemented polyethylene liner, which allows for placement of the shell in the areas of large defects and compensatory orientation of the cemented liner to re-establish femoral head coverage and hip stability.

Modular revision systems that use porous metal augments have been developed. These augments are assembled intraoperatively based upon the defects encountered and act like structural bone graft substitutes (Figure 1). The cup may also be supplemented with a cage fixed into the ilium (the so-called "cup-cage" construct) to offload the porous metal cup to allow time for bony in growth and cup stabilization.

A large published series by Skytta *et al*[19] reviewed the surgical short-term results of 827 revisions performed with the Trabecular Metal acetabular shell. After 3 year follow-up, the overall survivorship was 92% with the rates of aseptic loosening documented as 2%. In another retrospective series comparing titanium and tantalum cups, similar results in hip revision cases with minor bone deficiencies (Paprosky Ⅰ, ⅡA, and ⅡB) were demonstrated[20]. However, the performance of the two implants differed significantly in the cases associated with severe bone loss (Paprosky ⅡC and Ⅲ), with 12% of tantalum cups and 24% of titanium cups demonstrating evidence of loosening and failure. To investigate this distinction further, Fernandes *et al*[21] performed a retrospective review to evaluate the outcome of TM acetabular components used in revision cases with major bone deficiency. They demonstrated satisfactory midterm results with only 1/46 patients showing evidence of loosening over an average follow-up of 50 mo.

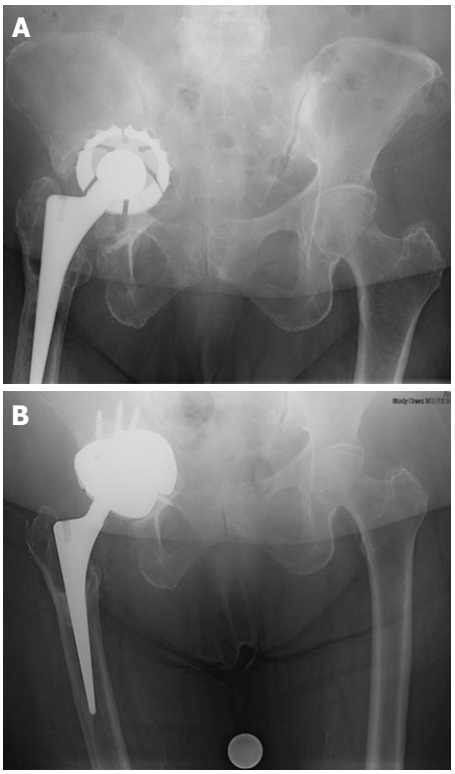

Figure 1 Preoperative radiograph showing failed acetabular component with large medial defect and intact rim (A) and postoperative radiograph demonstrating the revision acetabular construct using tantalum augments as "footings" to support the cup (B).

Antiprotrusio cages

The armamentarium for treatment large bone defects (Paprosky ⅡC or Ⅲ) has traditionally included antriprotrusio cages. These expansile implants are indicated for cases in which stability cannot be obtained with an uncementedhemispheric cup or in situations where the remaining host bone is too compromised to achieve biologic fixation of a porous implant. Antiprotrusio cages provide a larger contact area between the remaining host bone and the implants, which potentially reduces the likelihood of implant migration. Current implant designs also allow for concomitant treatment of bone defects with either morselized or bulk allograft materials protected by the cage construct. Use of antiprotrusio cages requires wide surgical exposure as they span the acetabular defects or area of discontinuity. Solid fixation into the posterior column is essential and, in the most severe cases, additional internal fixation with posterior column plating may be warranted.

In the setting of combined segmental and cavitary defects, impaction bone grafting with compressed par-

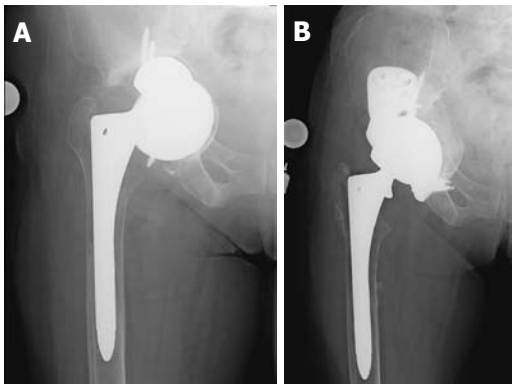

Figure 2 Preoperative radiograph demonstrating failed revision acetabular component with massive bone loss and pelvic discontinuity (A) and postoperative radiographs showing custom triflange cup reconstruction (B).

ticulate graft used in conjunction with an antiprotrusiocage construct has shown successful clinical results. With this construct, the healthy host bone is bridged by the cage implant, which protects the grafted area while consolidation and reconstitution of the acetabular bed occurs[22]. In one original study, a review of patients with combined segmental and cavitary defects (Paprosky Ⅲ) treated with an antiprotrusio cage and allograft demonstrated radiographic remodeling of the graft behind the cages at 5-year follow-up[23]. The rate of aspetic failure was 12% in this group, which exceeded the historial results with these implants. The increased rate of loosening and need for revision in these cases, compared with other acetabular reconstruction options, is likely multifactorial and includes the increased severity of discontinuity and defect found in the patients for whom use of a cage is indicated. This construct does rely on mechanical fixation alone with no potential for long-term biologic incorporation.

Custom triflange implants

Custom triflangeacetabular prostheses are indicated for the treatment of massive acetabuluar bone loss and pelvic discontinuity, situations where the amount of bone loss exceeds the limits of defect-matching technqiues (Figure 2)[16]. They are also considered as reconstructive options when the host bone stock has been compromised by radiation. The implant is customized from data obtained from 3-dimensional CT reconstruction imaging, which details the degree and location of bone loss as well as the orientation of the pelvic dissociation. Accordingly, the time needed to design, manufacture, and sterilize these prostheses can take up to several months and must be taken into consideration during the pre-operative planning process. Modern triflange cups encorporate porous ingrowth surfaces to encourage biologic fixation to host bone. The high cost of these implants and lack of intraoperative modularity is a consideration. Many surgeons consider the use of the custom triflange implant as a final salvage procedure when there is catastrophic bone loss.

In 2007, DeBoer *et al*[24] published the results of their study of 30 hips with failed hip arthroplasty and pelvic discontinuity treated with custom manufactured acetabular prostheses. The authors found definite radiographic healing of the discontinuity without evidence of implant migration or screw breakage at the mean ten-year follow-up. They documented a marked improvement in Harris hip scores and stability of the implant over the years of follow-up. The dislocation rate was 16%, however no revisions were required in their study group. Christie *et al*[25] also published results from a retrospective review of 76 hips reconstructed with custom triflange prostheses. In their group, re-operation occurred in 7.8% of their patients for dislocation, but no triflange components had to be removed. Almost all patients showed radiographic evidence of remodeling and there was marked improvement in the Harris hip scores.

Contraindications to use of custom triflange prostheses include urgent clinical situations that do not allow for the wait period required to manufacture the customized implant. In addition, given the complexity of the prosthesis, the technical challenge and the extensive surgical exposure required for its implantation, custom triflange prostheses should not be used in cases where defect-matching techniques can be employed and less complex and costly acetabular reconstruction options are suitable.

FUTURE OF ACETABULAR RECONSTRUCTION

The goals of acetabular revision are to extract failed implants with minimal host tissue and bone destruction, implant an acetabular prosthesis that will provide durable function and lasting pain relief, and to address osseous defects or dissociation by effectively restoring bone stock. From acetabular bone loss, polyethylene wear and osteolysis, to catastrophic pelvic discontinuity, there is now a spectrum of reconstruction options that allows for consideration of patient factors and the condition of the acetabular bed to guide the treatment algorithm. Requirements for a successful and durable long-term result include supportive host bone and stable implants. With the improvements being made in currently available biomaterials and implant designs, there is still a significant amount of research that needs to be done in the form of well-designed clinical studies to ensure that we are providing the optimal services to the growing number of patients that will stand in need of revision reconstruction in the future.

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TOPIC HIGHLIGHT

Federico Girardi, MD, *Series Editor*

Establishing proof of concept: Platelet-rich plasma and bone marrow aspirate concentrate may improve cartilage repair following surgical treatment for osteochondral lesions of the talus

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Abstract

Osteochondral lesions of the talus are common injuries in the athletic patient. They present a challenging clinical problem as cartilage has a poor potential for healing. Current surgical treatments consist of reparative (microfracture) or replacement (autologous osteochondral graft) strategies and demonstrate good clinical outcomes at the short and medium term follow-up. Radiological findings and second-look arthroscopy however, indicate possible poor cartilage repair with evidence of fibrous infill and fissuring of the regenerative tissue following microfracture. Longer-term follow-up echoes these findings as it demonstrates a decline in clinical outcome. The nature of the cartilage repair that occurs for an osteochondral graft to become integrated with the native surround tissue is also of concern. Studies have shown evidence of poor cartilage integration, with chondrocyte death at the periphery of the graft, possibly causing cyst formation due to synovial fluid ingress. Biological adjuncts, in the form of platelet-rich plasma (PRP) and bone marrow aspirate concentrate (BMAC), have been investigated with regard to their potential in improving cartilage repair in both *in vitro* and *in vitro* settings. The *in vitro* literature indicates that these biological adjuncts may increase chondrocyte proliferation as well as synthetic capability, while limiting the catabolic effects of an inflammatory joint environment. These findings have been extrapolated to *in vitro* animal models, with results showing that both PRP and BMAC improve cartilage repair. The basic science literature therefore establishes the proof of concept that biological adjuncts may improve cartilage repair when used in conjunction with reparative and replacement treatment strategies for osteochondral lesions of the talus.

Key words: Osteochondral lesion; Cartilage repair; Platelet-rich plasma; Bone marrow aspirate concentrate

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INTRODUCTION

The ankle is one of the most common sites of injury in athletes, with a sprain being the most frequent mechanism[1]. The incidence has been described to be as high as nearly 1 per 1000 athlete exposures, leading to 27 000 ankle sprains every day in the United States[2,3]. These injuries are known to lead to cartilage insult in up to 50% of patients[4] and therefore potentially resulting in an osteochondral lesion of the talus (OCL). The increasing recognition of the prevalence of these lesions has brought the etiology and treatment of OCLs to the forefront of sports medicine.

The first description of an ankle OCL was likely given by Monro[5], who removed a loose body from the ankle caused by traumatic injury. Since then, various etiologies have been described which may contribute to the formation of these lesions, including acute trauma, chronic microtrauma, endocrine or metabolic factors, genetic predisposition, joint displacement, osteoarthritis, and avascular necrosis[6-13]. Trauma, however, remains the most common instigating factor, with Flick and Gould[7] concluding that of 500 patients with OCLs, 90% of lateral dome and 70% of medial dome lesions could be attributed to a traumatic event.

Multiple treatment strategies have been defined for managing osteochondral lesions of the talus. These include conservative management, in the form of immobilization or protective weight bearing, and surgical treatment, consisting of either reparative or replacement therapies[14].

Articular hyaline cartilage is avascular and therefore has a poor propensity for healing, additionally when the osteochondral lesion does not extend beyond the subchondral plate, the body does not mount an inflammatory response to promote regeneration. With lesions that involve the subchondral bone, an inflammatory response stimulates marrow cells to produce repair tissue in an attempt to fill the defect[15]. This is the principle behind bone marrow stimulation techniques such as microdrilling and microfracture.

The replacement techniques for treating OCLs consist of substituting the lesion with viable tissue, such as an autologous osteochondral graft or an osteochondral allograft. Studies on both reparative and replacement treatment strategies, however, have shown concern with regard to poor post-operative cartilage repair[16-19]. Both surgical techniques have proven to demonstrate good short to medium term clinical results, however further long-term studies, second look arthroscopy, and radiological investigation have shown reasons for concern[16-20]. This review will describe and address the issue of poor cartilage healing following surgical treatment of osteochondral lesions of the talus, and the use the biological adjuncts to improve cartilage healing. The two most common surgical modalities currently in practice and those used by the senior surgeon are microfracture and autologous osteochondral transplantation, and will be the two strategies addressed in this article.

MICROFRACTURE

The microfracture procedure involves arthroscopically breaching the subchondral plate in order to stimulate an inflammatory response and migration of subchondral derived mesenchymal stem cells (Figure 1). The recruited cells differentiate into fibrocartilage in an attempt to fill the defect and protect the underlying subchondral bone from excessive loading over time[21].

Short to medium term clinical outcomes following microfracture have been good. Saxena and Eakin[4] reported their results in the athletic population (n = 26) at an average follow-up of 32 mo. 96% of patients reported good or excellent post-operative AOFAS scores, with the same percentage of the study group returning to their sporting activity. Chuckpaiwong *et al*[22] published results detailing the outcomes of 105 consecutive patients whom had the talar OCLs microfractured. Of those patients who had lesions smaller than 15 mm in diameter, there were no failures of treatment at a mean follow-up of 31.6 mo.

These results were further pooled and corroborated in a systematic review article by Zengerink *et al*[23]. The authors reviewed 18 studies reporting the outcomes of arthroscopic microfracture surgery and found the reported success rate to be 85%. While the satisfaction of the patient is paramount to determining whether a procedure is successful, the majority of these studies have a short to medium term follow-up.

Despite good clinical follow-up, microfracture relies on a biologic infill that is fundamentally flawed. The underlying issue prevalent with subchondral stimulation is that it stimulates a fibrocartilage repair, which is biomechanically inferior to hyaline cartilage (Figure 2). Additionally, upon further mechanical loading of the joint, fibrocartilage progressively degenerates with an increase in type I collagen[18,19,24-26].

In a study correlating clinical results with second-look arthroscopic findings of 20 ankles, Lee *et al*[16] found at 12 mo post-operatively, 90% of patients reported a good to excellent outcome with regard to their AOFAS score. However, on second-look arthroscopy, 35% of ankles were determined to show incomplete healing, only 30% of lesions were integrated with the native hyaline cartilage, and 80% had cracks and fissures. The authors found no correlation between clinical AOFAS scores and arthroscopic appearance of the lesion site. This may possibly be due to the short follow-up, and the patients may not experience a deterioration of their symptoms at 1 year post-operatively.

Becher *et al*[17] echoed these results when assessing the outcome of microfracture surgery using the Hannover Scoring System for clinical outcome and magnetic resonance imaging (MRI) in 45 cases. While the clinical outcomes were successful, with 4 ankles necessitating further surgery to address the chondral defect, MRI assessment indicated that 100% of the cases had cracks and fissuring of the regenerative tissue at a mean follow-up of 5.8 years.

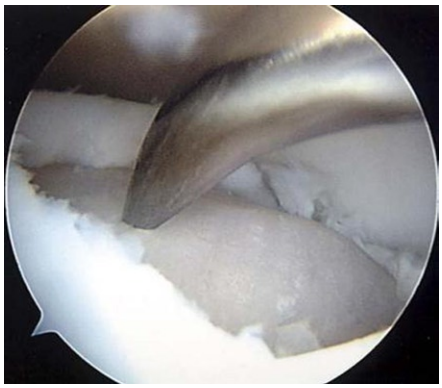

Figure 1 Arthroscopic microfracture of osteochondral lesion.

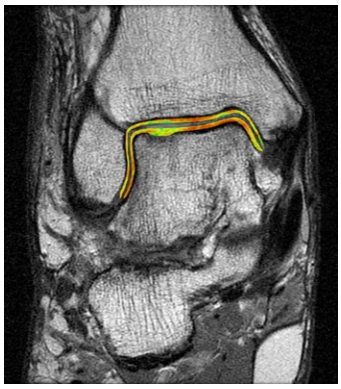

Figure 2 Magnetic resonance imaging with T2-mapping demonstrating significantly shorter relaxation times in both superficial and deep zones of repair tissue-indicating poor cartilage infill 6 mo following microfracture.

There is an indication that the post-operative clinical outcome scores may deteriorate with time as the lesions fails to heal with adequate repair tissue. Ferkel *et al*[27], reporting on 50 patients with a mean follow-up of 71 mo, found that 36% of patients had fair to poor results, as measured by the modified Weber scale. Furthermore, 17 patients had been seen 5 years previously and evaluated using the same criteria. Of these 17 patients with a longer-term outcome, 35% demonstrated deterioration in their outcome scores over time. Hunt and Sherman[19], on reporting clinical outcomes as measured by the Martin Score at 66 mo follow-up (33 ankles), found fair or poor outcomes in 61% of patients. Presently, there is no consensus or evidence base as to the optimal size defect that should be treated with microfracture.

AUTOLOGOUS OSTEOCHONDRAL TRANSPLANT

Autologous osteochondral grafts (OATS) involves replacing the damaged tissue on the talus, with a healthy osteochondral graft harvested from a non-weightbearing portion of the ipsilateral knee (Figure 3). This procedure has predominantly been advocated for treating large cystic lesions, or in patients who have failed previous

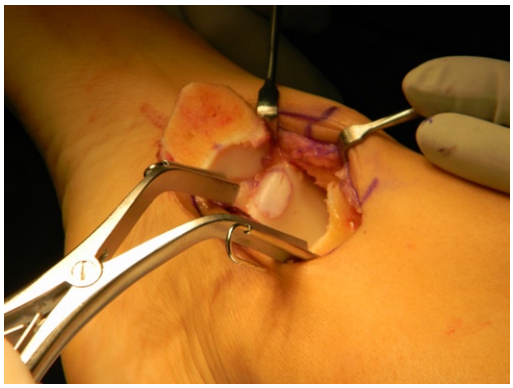

Figure 3 Autologous osteochondral graft.

subchondral stimulation[28-30].

Hangody *et al*[31] were the first to publish results following osteochondral autograft transplantation (mosaicplasty). In a study examining 34 cases with an average follow-up of 48 mo, the authors described good to excellent outcomes in 94% of patients, as measured by the Hannover scoring system.

In a retrospective study examining the outcomes of 50 patients with a cystic talar defect, Scranton *et al*[30] measured patient outcomes at a mean post-operative time point of 36 mo using the Karlsson-Peterson Ankle Score. 45 patients (90%) had a good to excellent outcome score, with a mean of 80.3.

These successful clinical post-operative outcomes were mirrored in one of the largest case series published by Kennedy and Murawski[32]. In a retrospective study reporting the outcomes of 72 patients with a mean follow-up of 28 mo, outcome was assessed using Foot and Ankle Outcome (FAOS) and Short Form-12 scores (SF-12). FAOS scores improved from 52.67 pre-operatively to 86.19 post-operatively. Similarly, the SF-12 scores improved from 59.4 pre-operatively to 88.63 post-operatively. One patient required a revision surgery for a decompression of a cyst that developed below the graft site through a standard retrograde sinus tarsi approach. Despite these encouraging results, there are some concerns that this procedure has inherent problems that may only manifest at a later time point.

An autologous osteochondral transplant allows degenerative tissue to be replaced with a viable hyaline cartilage graft. However, there remain issues with graft healing, particularly at the interface between the graft and native tissue[33]. In animal models it has been shown that there is poor integration at the cartilaginous border of the graft and surrounding talar cartilage[34]. Additionally, in the process of harvesting the graft from the ipsilateral knee and press fitting it into the cored out lesions site, up to 25% of cell death may occur at the periphery[35].

In a case series published by Valderrabano *et al*[20], reporting the outcomes of 21 patients treated with the osteochondral graft procedure with a mean follow-up of 72 mo, the authors described not only the clinical outcomes using the AOFAS ankle score, but also their

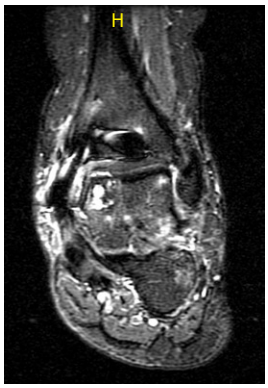

Figure 4 Magnetic resonance imaging showing cyst formation following autologous osteochondral graft surgery at 3 mo post-operative.

MRI and SPECT-CT findings. While the patients reported a satisfaction rate of good to excellent in 92% of cases, and mean AOFAS score improved from 45.9 pre-operatively to 80.2 points post-operatively, radiological findings were less encouraging. On MRI, there was evidence of recurrent cyst formation in 75% of patients. SPECT-CT showed that some level of cyst formation in all cases.

A poorly healed interface may allow synovial fluid ingress around the osteochondral plug and cause cyst formation (Figure 4). When the subchondral bone is under stress, such as from increased hydrostatic pressure from synovial fluid, it leads to upregulation of interleukin-1 and interleukin-6[36]. The upregulation of catabolic factors causes increased osteoclastic activity and ultimately bone resorption. This may cause cyst formation, therefore undermining the graft, and leading to failure of the procedure.

DIAGNOSIS OF OSTEOCHONDRAL LESIONS

In order to detect poor cartilage healing, adequate imaging must be ordered that is able to visualize the problem. Standard weightbearing radiographs on the ankle are still used in the initial post-operative assessment. However, it is known that up to 50% of osteochondral lesions may not be visualized on X-ray[29]. Helical computed tomography is favored by many surgeons as an initial assessment of OCLs. It is useful in assessing bony detail and determining specific size, shape and extent of subchondral cystic formation[37]. The visualization of cartilage though, is not possible with CT imaging.

Soft tissue pathology, which is the object of concern when determining if proper regeneration of cartilage has occurred, is best assessed using magnetic resonance imaging[38]. MRI is useful is detecting the degree of cartilage repair and if any other soft tissue insult has occurred. Additionally, it has been shown that the radiological images correlate well arthroscopic findings[39]. The authors prefer using the recently developed quantitative MRI technique of T2 mapping which provides quantitative and qualitative information about cartilage repair (Figure 2)[40].

Table 1 Summary of the effect of growth factors contained in platelet-rich plasma

Growth factor	Activity
TGF-β1	Stimulates MSCs and chondrocytes inhibit catabolic activity of IL-1
FGF	Stimulate bone growth Decrease aggrecanase activity
EGF	Cellular proliferation epithelial cell differentiation
PDGF	Stimulation of fibroblasts and collagen synthesis stimulation of osteoblasts
VEGF	Promotes angiogenesis and vasculogenesis

TGF-β1: Transforming growth factor-β1; FGF: Fibroblast growth factor; EGF: Epidermal growth factor; PDGF: Platelet-derived growth factor; VEGF: Vascular endothelial growth factor; MSCs: Mesenchymal stem cells; IL-1: Interleukin-1.

IMPROVEMENT OF CARTILAGE REPAIR

Improving the biological environment is crucial in order to stimulate the regeneration of cartilage-like tissue and prevent long-term deterioration of outcome following cartilage repair and replacement surgeries. Growth factors and mesenchymal stem cells have long been of interest to the orthopaedic community as a potential adjunct to both microfracture and osteochondral graft transplantation. Historically, individual growth factors have been study in isolation in their recombinant form[41-45]. However, given the vast assortment of growth factors and their interaction within the joint environment, it is doubtful that any single growth factor will lead to comprehensive cartilage regeneration[38]. Therefore, biological adjuncts in the form of platelet-rich plasma (PRP) and bone marrow aspirate concentrate (BMAC) are currently being investigated for their chondrogenic and anti-inflammatory effects and may improve poor cartilage healing as a post-operatively[46-61].

Platelet-rich plasma

PRP is defined as a sample of plasma with a twofold or more increase in platelet concentration above baseline level or greater than 1.1×10^6 platelets/μL[62]. Platelets' physiological role in healing has led to the concept that PRP may improve cartilage restoration. Additionally, the multitude of growth factors (Table 1) stored within the platelets' alpha granules are believed to improve the biological environment within which cartilage may heal[63]. Multiple *in vitro* and *in vivo* studies are present in the literature delineating the potential of PRP to improve chondrogenesis in ankle cartilage repair[46-61].

In a study culturing porcine chondrocytes in 10% PRP, the authors reported a 115% ($P < 0.001$) increase in proteoglycansynthesis compared to the control (fetal bovine serum). Furthermore, PRP augmented collagen production by 163% ($P < 0.001$)[41]. The proliferative

effect of PRP is not limited to chondrocytes alone. Human mesenchymal stem cells (MSC), which may be recruited through subchondral stimulation (e.g., microfracture) or by the addition of BMAC, have also been shown to be positively affected by PRP. Subchondral progenitor MSCs are stimulated to migrate in the presence of PRP[47]. This is particularly relevant to improving the outcomes of the arthroscopic microfracture technique as this is the reasoning behind the use of this procedure. In addition, human MSCs, when cultured in 10% PRP, demonstrate increased levels of DNA compared to an FBS control[48]. Kruger *et al*[47] demonstrated that the addition of PRP caused MSCs to undergo chondrogenic differentiation and increase type Ⅱ collagen matrix deposition. MSCs are also difficult to recruit in significant amounts as their concentration in peripheral blood and bone marrow is relatively low, representing only 0.001% to 0.01% of mononuclear cells in bone marrow aspirate[49,50]. Their proliferation though has been shown to increase when cultured with PRP[51-53]. Increasing the synthetic capacity and proliferation of chondrocytes and mesenchymal stem cells potentially improves the cartilage infill of both an OCL that has undergone microfracture and the interface between an osteochondral graft and native tissue.

Osteochondral lesions cannot be managed in isolation if the surgeon hopes to avoid a poor outcome. The development of an OCL indicates the presence of an intra-articular inflammatory environment. While surgical intervention in the form of subchondral stimulation or an osteochondral graft treats the focal defect, the presence of catabolic cytokines may cause further cartilage degeneration and inhibit the production of regenerative tissue. Haemarthrosis following trauma or surgery, causes iron-catalyzed oxygen metabolites to induce macrophage activation and matrix metalloproteinase (MMP)-2 and MMP-9 production by synoviocytes[54]. Additionally, neutrophils chemotactically drawn to the intra-articular space produce interleukin 1 beta (IL-1β) and tumor necrosis factor (TNF)-α which further increase matrix metalloproteinase, ADAMTS, and elastases by both synovial cells and chondrocytes[55]. This catabolic cascade serves to alter the composition of synovial fluid and promote degradation of cartilage extracellular matrix, ultimately causing detriment to any surgical procedure.

PRP is known to counter the catabolic mediators in order to reduce the inflammatory damage to the joint. PRP has been shown to increase the production of hyaluronic acid and hepatocyte growth factorby synoviocytes excised from arthritic patients[56]. The increase in hepatocyte growth factor is particularly relevant as there is evidence that it blocks the activity of nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB)[57]. Furthermore, in human chondrocytes cultured in IL-1β to simulate an osteoarthritic environment, PRP decreased IL-1β mediated inhibition of COL2A1 and ACAN gene expression. Additionally, itdecreased the IL-1β induced increase of *ADAMTS4* and *PTGS2* gene expression[58]. These findings have been further confirmed by additional study culturing chondrocytes in IL-1β and TNF-α with collagen matrix enhanced PRP, showing increased chondrogenesis, collagen type Ⅱ deposition and inhibition of IL-1β and TNF-α[59]. The combination of both the anabolic effect and the inhibition of inflammatory catabolism may contribute to improving cartilage repair and decreasing the risks of a poor outcome following surgery for OCLs. These studies have since been translated to an *in vivo* model.

In a rabbit model, PRP treated polylactic-co-glycolic acid (PLGA) scaffolds improved osteochondral lesion healing compared to OCLs treated with a PLGA scaffold alone. The authors noted that while the controls showed fibrous healing with deep fissures, the PRP treated group showed hyaline-like infill which was well integrated with the surrounding tissue[60]. Milano *et al*[61] reported on the results of using PRP and PRP combined with fibrin as an adjunct to microfracture surgery for osteochondral defects in a sheep model, comparing their treatment groups to microfracture surgery alone. The study showed evidence that the PRP treated group showed improved cartilage repair that was both histologically differentiated and mechanically competent.

Bone marrow aspirate concentrate

BMAC is obtained through density gradient centrifugation of bone marrow typically aspirated from the iliac crest. Similar to PRP, BMAC contains platelets, and therefore growth factors, but in lesser concentrations[64]. The principle reason for using BMAC as a biological adjunct to osteochondral lesion surgeries of the talus is to introduce MSCs to the site[64]. Wilke *et al*[65] demonstrated, in an equine model, that the introduction of MSCs to a full thickness cartilage defect improves cartilage repair. Furthermore, the authors noted that the repair tissue contained primarily type Ⅱ collagen and was therefore more hyaline-like. The principle that MSCs improve cartilage healing has been further corroborated in the literature[66,67].

The role of BMAC in improving the outcomes of OCL surgery has also been investigated in an *in vivo* setting. In an equine model, BMAC augmented microfracture was compared to microfracture alone for treatment of a full thickness chondral defect, 15 mm in diameter. At the 8 mo post-operative time point, the authors reported a vast improvement in both ICRS macroscopic and histological scores, 9.4 ± 1.2 compared with 4.4 ± 1.2 and 11.1 ± 1.6 compared with 6.4 ± 1.2 respectively. Moreover, there was improved collagen orientation and collagen type Ⅱ content in the BMAC treated group[68]. Saw *et al*[69] showed similar results in a study extrapolating the use of BMAC to a goat model. In a comparison of treatment between microfracture alone, microfracture plus hyaluronan, and microfracture plus hyaluronan and BMAC, there was a statistically significant difference in repair tissue with the last group showing the most favor-

able results. At the 24 wk following surgery, the BMAC treated group demonstrated almost complete coverage of the defect with evidence of hyaline cartilage repair. In comparison, the group that received microfracture in isolation, showed only partial healing of the lesion with predominantly scar tissue.

CONCLUSION

Osteochondral lesions are currently treated predominantly by either attempting to repair the lesion with arthroscopic subchondral microfracture or replacement of the non-viable tissue with an autologous osteochondral graft. The short to medium-term clinical results of these surgeries are positive, however longer-term clinical outcomes, as well as radiographic and arthroscopic findings, indicate that surgeons must improve the quality of regenerative tissue in order to avoid long-term postoperative deterioration of outcome.

PRP and BMAC, with their array of bioactive factors have been shown to improve cartilage regeneration in both *in vitro* and *in vivo* models. They amalgamate two of the three factors of the tissue engineering trifecta, bringing stem cells and growth factors to the site of injury. These biological adjuncts are simple and easy to generate and are not known to cause any adverse clinical event. Additional research is required to analyze the long-term outcomes of employing biological adjuncts in a clinical setting using carefully designed randomized levelI clinical trials. As we seek to improve the outcomes of surgical treatments for osteochondrallesions, the body of evidence surrounding PRP and BMAC will grow to encompass long-term clinical outcome studies. Researchers are encouraged to continue investigating these biological adjuncts using rigorous scientific methodology.

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CASE REPORT

Giant pseudomeningocele after spinal surgery: A case report

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Abstract

Very few reports have described giant pseudomeningoceles ⩾ 8 cm in diameter. We report this case of the biggest giant pseudomeningocele at the unusual cervicothoracic level. A 59 year old man who underwent cervicothoracic laminectomy had a giant pseudomeningocele detected and the lesion gradually grew to about 15 cm in diameter by 2 years postoperatively. Cerebrospinal fluid leak closure was performed and the postoperative course was favorable. We present this case, review the literature and discuss the size and portion, mechanism of formation, symptoms and treatments of giant pseudomeningocele.

Key words: Pseudomeningocele; Spinal surgery; Dura tear; Cerebrospinal fluid; Complication

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INTRODUCTION

A pseudomeningocele is an abnormal collection of cerebrospinal fluid that communicates with the arachnoid space[1-3]. Pseudomeningocele was first reported by Hyndman and Gerber[1] in 1946. The causes of pseudomeningocele have been classified into three categories: congenital, traumatic and iatrogenic. Pseudomeningocele, rarely reported in the literature, seldom occurs after spine surgeries, particularly after laminectomy or discectomy at the lumbar level in late middle-aged patients[4]. Cervical pseudomeningocele can occur in young patients with traumatic brachial plexus injury as these patients can experience direct trauma to the dural sac, causing cerebrospinal leakage, and muscular weakness from nerve injury can promote growth of the lesion[5]. The true incidence of pseudomeningocele following incidental durotomy is unknown. Swanson *et al*[6] and Teplick *et al*[7] reported incidences of pseudomeningocele after laminectomy of 0.068% and 2%, respectively. Oppel *et al*[8] found the incidence of durotomy during bone removal or retraction to be 5.9%.

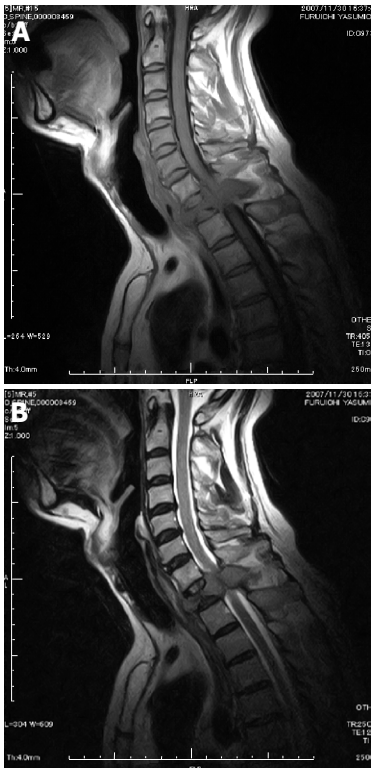

Figure 1 Magnetic resonance imaging revealing compression of the spinal cord at the level of the first thoracic vertebra by a lesion that was considered a metastasis of an unknown primary tumor. A: T1-weighted image; B: T2-weighted image.

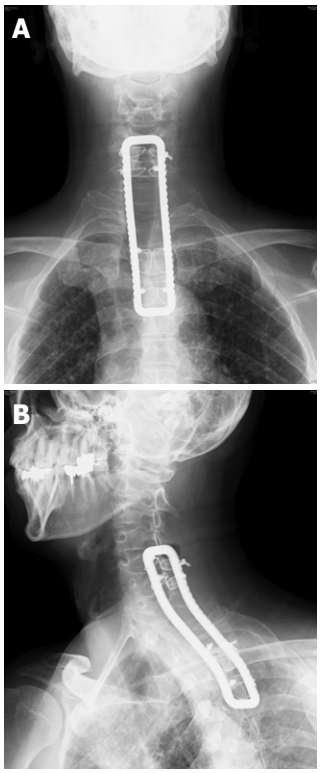

Figure 2 Radiography after first surgery. The laminectomy of C7, Th1 and Th2 and fixation of C5-Th4 were performed by using a sublaminar wire and a rectangle rod. A: Anteroposterior view; B: Oblique view.

The onset of giant pseudomeningocele is a rare complication of spinal surgery. Very few reports have described giant pseudomeningoceles ⩾ 8 cm in diameter[4,9]. We encountered a patient with a giant pseudomeningocele of about 15 cm in diameter that had developed after posterior thoracic decompression surgery. We report and discuss this case with reference to the literature.

CASE REPORT

The patient was a 59 year old man whose major complaint was an uncomfortable feeling in the cervicothoracic back region and who had a history of surgery for the metastasis removal in the first thoracic vertebra. About 3 years earlier, he had experienced the sudden onset of difficulties with walking. His muscle strength of the lower extremities was manual muscle testing (MMT) 3+ and moderate paresthesia of his trunk and lower extremities was noticed. Magnetic resonance imaging (MRI) (Figure 1A and B) indicated compression of the spinal cord at the level of the first thoracic vertebra (Th1) by a lesion that was considered a metastasis of an unknown primary tumor; thus, the patient was subjected to an emergency operation. The surgical procedure included laminectomy of C7, Th1 and Th2 and fixation of C5-Th4 using a sublaminar wire and a rectangle rod (Figure 2A and B). No clear damage to the dura mater was observed intraoperatively.

Based on the pathological findings from tissue obtained intraoperatively, adenocarcinoma of the lung was diagnosed. Walking difficulties improved postoperatively, his muscle strength of the lower extremities was MMT 4 and moderate paresthesia of his trunk and lower extremities was improved. The patient then received chemotherapy in the Department of Respiratory Internal Medicine. The lung cancer was stage Ⅳ, cT4N0M1 according to the TNM classification system. Because this patient's prognosis was very poor, only 5-FU (600 mg/d) for 6 mo was prescribed as his chemotherapy.

The MRI obtained 4 mo after the surgery showed a 7 cm × 3 cm mass that was hypointense on the T1-weighted image and hyperintense on the T2-weighted image, and a pseudomeningocele due to cerebrospinal fluid leak was detected. However, since the patient presented no symptoms attributable to the tumorous lesion, he was placed under observation. The lesion gradually grew to about 15 cm in diameter by 2 years postoperatively, as shown on MRI (Figure 3A, B and C). Since he felt uncomfortable, as if he were carrying a heavy weight on the cervicodorsal region, he was admitted for cerebrospinal fluid leak closure.

Findings on admission indicated a favorable general condition. He had no headache or nausea and could walk unaided. The operation was started following the skin incision from the previous surgery to remove the metastatic tumor. The subsequent subcutaneous deployment led to a 15 cm × 5 cm × 3 cm pseudomeningocele.

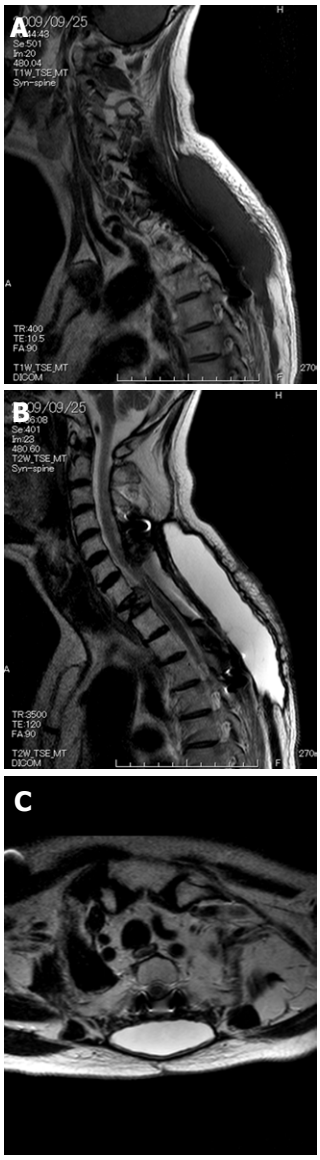

Figure 3 Magnetic resonance imaging 2 years after first surgery. The pseudomeningocele gradually grew to about 15 cm in diameter from C5 to Th6 level. A: T1-weighted sagittal image; B: T2-weighted sagittal image; C: T2-weighted axial image.

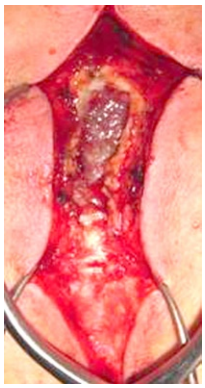

Figure 4 Intraoperative photo before capsule incision of pseudomeningocele. About 50 mL of colorless clear fluid was released after the capsule incision and no damage to the dura mater was noted.

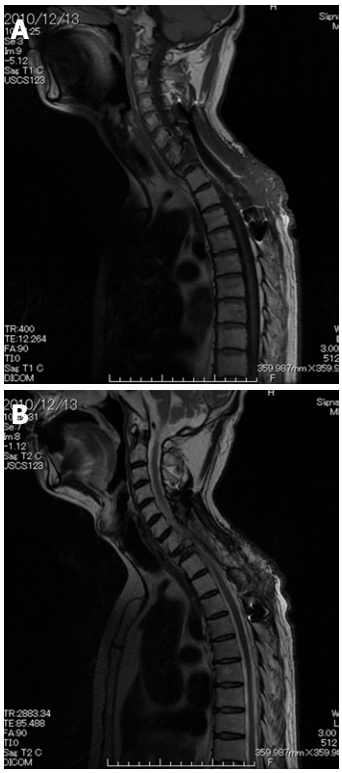

Figure 5 Magnetic resonance imaging showing the pseudomeningocele had disappeared 3 mo after closure of the cerebrospinal fluid leak. A: T1-weighted image; B: T2-weighted image.

The very thin capsule was broken (Figure 4) and about 50 mL of colorless clear fluid was released. No damage to the dura mater was noted. Even although it was not possible to detect small holes from which cerebrospinal fluid was leaking, fatty tissue was collected from under the skin and retained over the dura mater, over which fibrin paste was applied. We closed the paraspinal muscle and soft tissue in a layer-by-layer manner to reduce the third space as much as possible. Spinal drainage to reduce intradural pressure was then performed from the L3/4 level and adjustment was made to drain 200-300 mL/d of cerebrospinal fluid after the operation.

The postoperative course was favorable and his preoperative complaint of discomfort on the cervicodorsal region disappeared with no remarkable complications. The spinal drainage was withdrawn after about 2 wk postoperatively and the condition of the wound was confirmed to be favorable. According to the MRI (Figure 5A and B) obtained 3 mo after closure of the cerebrospinal fluid leak, the pseudomeningocele had disappeared and no recurrence has been observed for 2 years postoperatively. His muscle strength of the lower extremities was MMT 4 and slight paresthesia of his trunk and lower extremities was noticed at the follow-up 2 years after second operation. Chest and abdominal CT obtained at that time showed multiple metastases of ribs, vertebrae, pelvic bone and multiple enlarged lung lesions; however, the patient had almost no complaints.

DISCUSSION

A giant pseudomeningocele is defined as a lesion > 8 cm

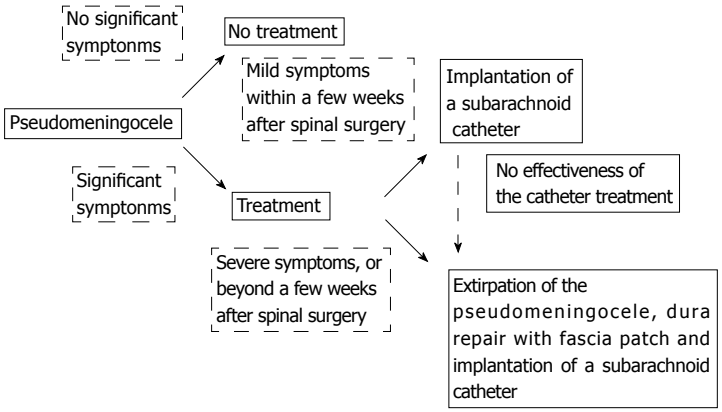

Figure 6 Algorithm for treatment of pseudomeningocele. Conservative treatment is generally recommended in patients without significant symptoms and surgical treatments should be performed in those with significant symptoms.

Table 1 Seventeen cases of giant pseudumeningocele reproted in the literature

Author	No. of cases	Level	Size (cm)	Treatment
Weng *et al*[4]	11	Cervical:2	8-11	Extirpation of the pseudomeningocele, dura repair with fascia patch and implantation of a subarachnoid catheter
		Lumbar:9	8	Extirpation of the pseudomeningocele and paraspinal fascia suture
Liu *et al*[12]	1	Lumbar	8.3	Extirpation of the pseudomeningocele and dura repair with fascia lata graft
Hamilton *et al*[10]	1	Lumbar	10	No surgery
Jame A	1	Lumbosacral	10-12	Extirpation of the pseudomeningocele and paraspinal fascia suture
Miller *et al*[4]	3	Lumbar: 3		

in diameter[4] and only 17 cases of giant pseudomeningoceles (Table 1) have been reported in the literature[4,9,10-12]. Of these 17 cases, 11 patients were men (64.7%) and 6 were women (35.3%). The mean age of the patients was 39.7 years (range 19-68 years). Diagnosis at the time of the initial operation was herniated intervertebral disc in 12 cases (70.6%), spondylolisthesis in 4 (23.5%) and unknown in 1 (5.9%). The mean size of the lesions was 9.6 cm in diameter (range 8-12 cm) and most pseudomeningoceles were lumbar (15 cases, 88.2%) rather than cervical (2 cases, 11.8%). The present case represents the biggest pseudomeningocele occurring at an unusual level.

The formation of a pseudocyst is a mechanical process. Lesion size depends on the size of the defect in the dura-arachnoid, the pressure of spinal fluid and presumably resistance from the surrounding soft tissues. If a small tear in the dura and intradural pressure causes a constant outflow of spinal fluid, the lesion will also gradually enlarge. The intradural pressure is higher in the lumbar spine than in the cervical spine; this potentially explains why pseudomeningoceles occur more often at the lumbar level. A giant pseudomeningocele can develop in patients with a large dural defect or high intradural pressure[7].

Diagnosis of pseudomeningocele typically depends on MRI which shows low signal intensity on T1-weighted image and high signal intensity on T2-weight image. However, MRI of some congenital defects, such as arachnoid cyst, sacral meningocele, arachnoid diverticulum and meningeal cyst, and tumors, such as neurinoma and cavernous angioma, may show the same indication as pseudomeningocele. To differentiate these, the age of the patient, neurological findings and surgical history should be considered to make a diagnosis.

The signs and symptoms associated with pseudomeningocele vary widely, including back pain, sciatic pain, headache, neck pain, nausea, vomiting, tinnitus and a palpable mass, although most pseudomeningoceles remain asymptomatic[7]. Symptoms may appear at any time and the severity of symptoms does not necessarily correlate with the size of the pseudocyst. The treatment modalities for pseudomeningocele include conservative management, placement of a blood patch, lumbar subarachnoid drainage and surgical repair. There are no absolute surgical indications of pseudomeningocele but significant symptoms, including vomiting, tinnitus, intense pain and paralysis, may be relative indications.

Previous reports of giant pseudomeningoceles have not recommended any definitive treatment[7,13]; however, we tried to show the algorithm for treatment of pseudomeningocele in Figure 6. Conservative treatment may be generally recommended in patients without significant symptoms. In some cases with mild symptoms within a few weeks after spinal surgery, implantation of a subarachnoid catheter might be effective. On the other hand, in cases with severe symptoms or beyond a few weeks after spinal surgery, extirpation of pseudomeningocele, dura repair with fascia patch and implantation of a subarachnoid catheter could be appended to deal with the symptoms.

In the present case, a 59 year old patient with lung cancer, the pseudomeningocele measured 7 cm × 3 cm at 4 mo after laminectomy of C7, Th1, Th2 and fixation of C5-Th4. The patient showed no symptoms for up to 2 years postoperatively while the lesion progressed to 15 cm in diameter. At the second operation, no clear damage to the dura mater was observed but we retained the fatty tissue over the dura mater, coated this tissue with fibrin paste and placed a spinal drainage at the L3/4 level. In this case, there appears to be three possible causes for

the development of the giant pseudomeningocele. The first was that the soft tissues and paravertebral muscles at the cervicothoracic level were damaged in the previous spinal surgery. The second was that high intradural pressure caused leakage of cerebrospinal fluid from a very small dural defect and this gradually pooled, causing enlargement of the lesion. The third was that the patient was asymptomatic for 2 years, allowing sufficient time for the formation of the giant pseudomeningocele.

In conclusion, we report this case, showing the biggest giant pseudomeningocele at the unusual cervicothoracic level.

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7th National Conference: Orthopaedics and Sports Medicine 2012 London, United Kingdom

February 16-19, 2012
Orthopaedic MRI and Small Parts
Scottsdale, AZ, United States

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Format

Journals

English journal article (list all authors and include the PMID where applicable)

1 **Jung EM**, Clevert DA, Schreyer AG, Schmitt S, Rennert J, Kubale R, Feuerbach S, Jung F. Evaluation of quantitative contrast harmonic imaging to assess malignancy of liver tumors: A prospective controlled two-center study. *World J Gastroenterol* 2007; **13**: 6356-6364 [PMID: 18081224 DOI: 10.3748/wjg.13.6356]

Chinese journal article (list all authors and include the PMID where applicable)

2 **Lin GZ**, Wang XZ, Wang P, Lin J, Yang FD. Immunologic effect of Jianpi Yishen decoction in treatment of Pixu-diarrhoea. *Shijie Huaren Xiaohua Zazhi* 1999; **7**: 285-287

In press

3 **Tian D**, Araki H, Stahl E, Bergelson J, Kreitman M. Signature of balancing selection in Arabidopsis. *Proc Natl Acad Sci USA* 2006; In press

Organization as author

4 **Diabetes Prevention Program Research Group**. Hypertension, insulin, and proinsulin in participants with impaired glucose tolerance. *Hypertension* 2002; **40**: 679-686 [PMID: 12411462 PMCID:2516377 DOI:10.1161/01.HYP.0000035706.28494.09]

Both personal authors and an organization as author

5 **Vallancien G**, Emberton M, Harving N, van Moorselaar RJ; Alf-One Study Group. Sexual dysfunction in 1, 274 European men suffering from lower urinary tract symptoms. *J Urol* 2003; **169**: 2257-2261 [PMID: 12771764 DOI:10.1097/01.ju.0000067940.76090.73]

No author given

6 21st century heart solution may have a sting in the tail. *BMJ* 2002; **325**: 184 [PMID: 12142303 DOI:10.1136/bmj.325.7357.184]

Volume with supplement

7 **Geraud G**, Spierings EL, Keywood C. Tolerability and safety of frovatriptan with short- and long-term use for treatment of migraine and in comparison with sumatriptan. *Headache* 2002; **42** Suppl 2: S93-99 [PMID: 12028325 DOI:10.1046/j.1526-4610.42.s2.7.x]

Issue with no volume

8 **Banit DM**, Kaufer H, Hartford JM. Intraoperative frozen section analysis in revision total joint arthroplasty. *Clin Orthop Relat Res* 2002; (**401**): 230-238 [PMID: 12151900 DOI:10.1097/00003086-200208000-00026]

No volume or issue

9 Outreach: Bringing HIV-positive individuals into care. *HRSA Careaction* 2002; 1-6 [PMID: 12154804]

Books

Personal author(s)

10 **Sherlock S**, Dooley J. Diseases of the liver and billiary system. 9th ed. Oxford: Blackwell Sci Pub, 1993: 258-296

Chapter in a book (list all authors)

11 **Lam SK**. Academic investigator's perspectives of medical treatment for peptic ulcer. In: Swabb EA, Azabo S. Ulcer disease: investigation and basis for therapy. New York: Marcel Dekker, 1991: 431-450

Author(s) and editor(s)

12 **Breedlove GK**, Schorfheide AM. Adolescent pregnancy. 2nd ed. Wieczorek RR, editor. White Plains (NY): March of Dimes Education Services, 2001: 20-34

Conference proceedings

13 **Harnden P**, Joffe JK, Jones WG, editors. Germ cell tumours V. Proceedings of the 5th Germ cell tumours Conference; 2001 Sep 13-15; Leeds, UK. New York: Springer, 2002: 30-56

Conference paper

14 **Christensen S**, Oppacher F. An analysis of Koza's computational effort statistic for genetic programming. In: Foster JA, Lutton E, Miller J, Ryan C, Tettamanzi AG, editors. Genetic programming. EuroGP 2002: Proceedings of the 5th European Conference on Genetic Programming; 2002 Apr 3-5; Kinsdale, Ireland. Berlin: Springer, 2002: 182-191

Electronic journal (list all authors)

15 Morse SS. Factors in the emergence of infectious diseases. Emerg Infect Dis serial online, 1995-01-03, cited 1996-06-05; 1(1): 24 screens. Available from: URL: http://www.cdc.gov/ncidod/eid/index.htm

Patent (list all authors)

16 **Pagedas AC**, inventor; Ancel Surgical R&D Inc., assignee. Flexible endoscopic grasping and cutting device and positioning tool assembly. United States patent US 20020103498. 2002 Aug 1

Statistical data

Write as mean ± SD or mean ± SE.

Statistical expression

Express *t* test as *t* (in italics), *F* test as *F* (in italics), chi square test as χ^2 (in Greek), related coefficient as *r* (in italics), degree of freedom as υ (in Greek), sample number as *n* (in italics), and probability as *P* (in italics).

Units

Use SI units. For example: body mass, m (B) = 78 kg; blood pres-

sure, p (B) = 16.2/12.3 kPa; incubation time, t (incubation) = 96 h, blood glucose concentration, c (glucose) 6.4 ± 2.1 mmol/L; blood CEA mass concentration, p (CEA) = 8.6 24.5 μg/L; CO_2 volume fraction, 50 mL/L CO_2, not 5% CO_2; likewise for 40 g/L formaldehyde, not 10% formalin; and mass fraction, 8 ng/g, *etc.* Arabic numerals such as 23, 243, 641 should be read 23 243 641.

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Italics

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Genotypes: *gyrA*, *arg* 1, *c myc*, *c fos*, *etc.*
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