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Journal of Arthroscopy and Joint Surgery (JAJS) is the official and peer-reviewed publication of *International Society for Knowledge for Surgeons on Arthroscopy and Arthroplasty* (ISKSAA). The Journal is committed to bring forth scientific manuscripts in the form of original research articles, current concept reviews, meta-analyses, case reports and letters to the editor. The focus of the Journal is to present wide-ranging, multi-disciplinary perspectives on the problems of the joints that are amenable with Arthroscopy and Arthroplasty. Though Arthroscopy and Arthroplasty entail surgical procedures, the Journal shall not restrict itself to these purely surgical procedures and will also encompass pharmacological, rehabilitative and physical measures that can prevent or postpone the execution of a surgical procedure. The Journal will also publish scientific research related to tissues other than joints that would ultimately have an effect on the joint function. The journal is published bi-annually (July and December) by Reed Elsevier India Pvt.Ltd. Contributors are invited to submit their manuscripts in English through the Online Manuscript Management System at **http://ees.elsevier.com/jajs**

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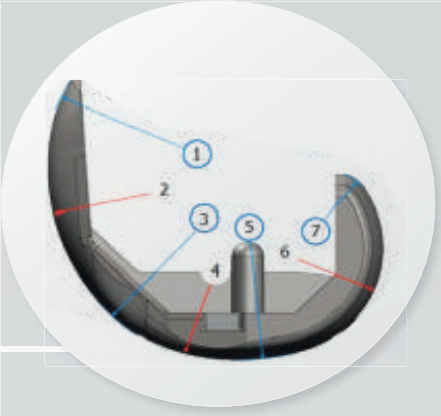

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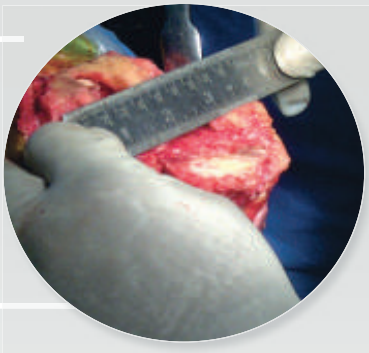

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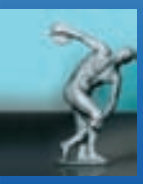

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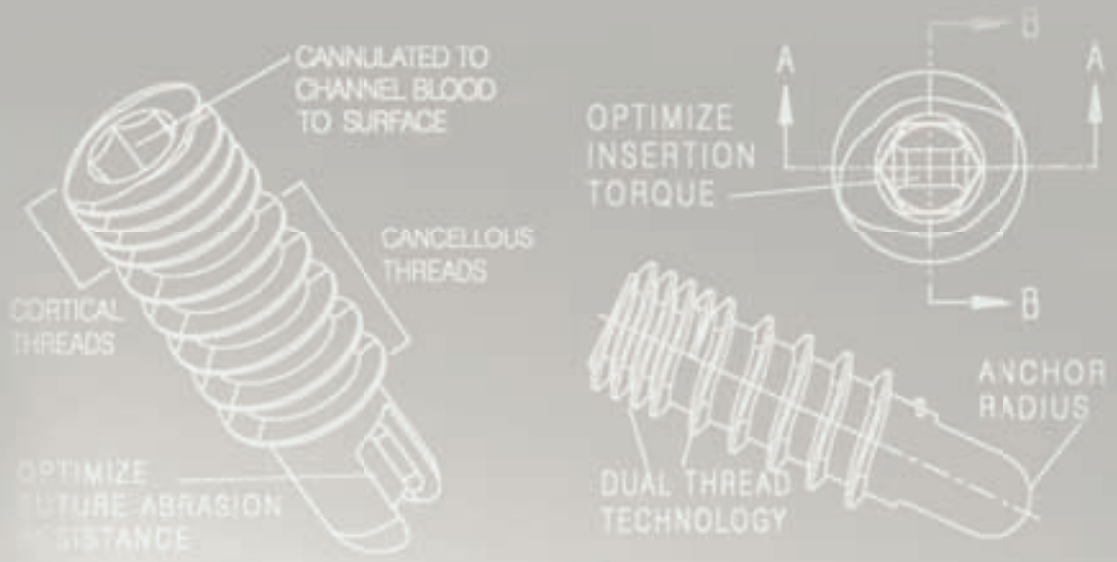

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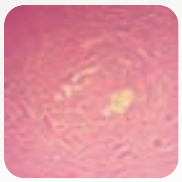

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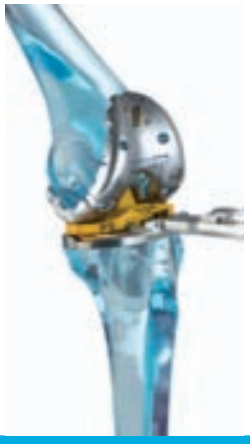

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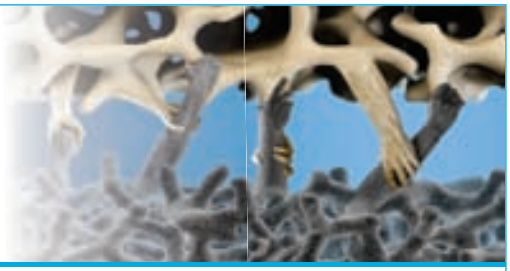

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Editorial

Total elbow arthroplasty today

Elbow is a sensitive and important joint essential for upper limb function. Elbow joint has to be pain-free, stable and mobile for a useful function. Functional impairment occurs in case of disruption in any of these components. Such a joint then requires total elbow arthroplasty.

Total elbow arthroplasty has evolved considerably over the last 20–30 years. The earlier implant designs of hinge elbow arthroplasty were of inferior quality. The fixation of the implant to the bone was of poor quality too, resulting into early loosening and high failure rate.

However, the art and science of total elbow arthroplasty have improved due to the better understanding of biomechanics, implant designs and materials and surgical techniques.

Generally the following 3 types of triceps (surgical) approaches are used in total elbow arthroplasty:

(i) Triceps sparing
(ii) Triceps reflecting, and
(iii) Triceps splitting

The choice of approach depends upon the underlying pathology, implant design and the surgeons preference.[1]

The triceps sparing approach is indicated for total elbow arthroplasty in acute fractures of the distal humerus.[2,3] It maintains the integrity of the triceps better, intraoperatively.

The triceps – reflecting (or Brian–Morrey) approach has been conventionally used for total elbow arthroplasty. The reflected triceps is reattached to the bed in ulna using non – absorbable sutures. However, triceps insufficiency emerges as a complication of this approach.[1]

The triceps – splitting approach involves either longitudinal division of the triceps in continuity with the forearm fascia over the dorsal ulna or splitting of the proximal triceps muscle belly with a V-Shaped turn – down of the triceps tendon and leaving intact its insertion on the olecranon. This approach allows for lengthening of extensor mechanism in cases of extension contracture.[1]

Enough biomechanical data is available to prove that the conventional simple hinge did not replicate the mechanics of elbow. This knowledge resulted into the development of two implant designs: joint resurfacing and linked prosthesis.[1]

In the joint resurfacing total elbow implant, the collateral ligaments of the elbow are preserved to maintain stability. The intact soft tissue envelope and adequate bone stock (because of the resurfacing design of the implant) are responsible for the success of the resurfacing implant.

The stresses across the elbow are absorbed, in part, by the ligamentous constraints, which theoretically results in lower rates of implants loosing, Unlinked designs demand precise replication of the axis of rotation. Poor component alignment and ulno-humeral incongruity result in high failure rates.[4]

With linked prostheses, stability is provided through a coupled articulation between the humeral and ulnar components. Modern linked implants have been modified from fully constrained articulations to semi-constrained designed that allow a few degrees of varus–valgus and rotational laxity. This reduces stress on the bone cement interface and the incidence of loosening.[5]

In theory, unlinked implants should be more prone to instability, whereas linked implants should show greater rates of loosening. However, in practice, mid-term outcomes have been reported with both types of implants.[6,7]

Recent reports have shown equal rates of clinical loosening.[7]

The modern cementing techniques have improved the mechanical fixation of implant to the bone. The techniques include the use of cement restrictors to occlude the canal, delivery of cement in the liquid state and pressurization of the Cement.[1]

The early success with total elbow arthroplasty in rheumatoid arthritis has encouraged the use of total elbow arthroplasty in more demanding pathology.[8] In patients with rheumatoid arthritis, total elbow arthroplasty provides reliable pain relief and functional improvement.[9,10]

Currently, the indications for total elbow arthroplasty are growing most rapidly for the late sequelae of trauma (i.e. post traumatic conditions) and acute traumatic injuries of the elbow.[3,11–16]

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Review Article

Clinical assessment of posterior shoulder joint instability

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ABSTRACT

Posterior shoulder instability is less common than anterior and is not as readily recognised. There are numerous clinical tests for posterior instability. They all have benefits and disadvantages, depending on the type of instability and strength of the patient. In this article we describe the most common clinical tests for posterior instability and review the literature supporting each test. In this manner, we hope that this will provide the clinician with a better understanding of each test and it's value.

1. Introduction

The shoulder is capable of the widest range of movement of all joints: for these to be normal and asymptomatic they depend on the interaction of both static and dynamic stabilisers of the shoulder. Static stabilisers include the bony anatomy, the glenoid labrum, the negative intra-articular pressure, the joint capsule, and the glenohumeral ligaments. The dynamic stabilisers are the muscles of the rotator cuff, and those surrounding the joint.[1] Unlike the hip and knee joints, the shoulder glenoid fossa is shallow. Glenohumeral stability from the glenohumeral ligaments of the capsule is effective primarily when the range of motion is at the extremes.[2] To have extensive movement at the glenohumeral joint the ligaments are required to be relatively lax. This requires combined involvement of dynamic and static stabilisers through range of motion.

The shoulder also benefits from the concavity compression mechanism, where the convex head of the humerus is compressed into the concave glenoid fossa to stabilise it against translating forces. The depth of the concavity and the magnitude of the compressive force influence joint stability with the depth of the bony glenoid being significantly less anteroposteriorly (2.5 mm) than superoinferiorly (9 mm), hence the stability against anterior and posterior forces was less than inferiorly and superiorly directed forces.[3] The labrum is a fibrocartilaginous ring around the glenoid increasing the depth of the glenoid upto 50%, contributing to the concavity compression mechanism.[4] The labrum also works alongside the synovial fluid to form a suction effect by adhesion-cohesion forces, providing stability to the articulation.[5] The negative intra-articular pressure also contributes to this effect and centres the humeral head into the glenoid. The attachment points for the glenohumeral ligaments and the long head of biceps arise from the labrum.

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The glenohumeral ligament structure consist of three parts; the superior glenohumeral ligament (SGHL), which resists translation inferiorly with the arm adducted and in neutral rotation; the middle glenohumeral ligament (MGHL), an anterior stabiliser in adduction and the inferior glenohumeral ligament complex. This comprises the anterior band of the inferior glenohumeral ligament (IGHL), which is the primary static stabiliser in a neutral position; and the posterior band of the IGHL (PIGHL), the primary static posterior stabiliser when the arm is flexed and internally rotated. The coracohumeral ligament (CHL) resists posterior and inferior translation when the shoulder is suspended and inferiorly when the arm is adducted.[1] Tension in the ligaments and capsule provide additional proprioceptive feedback to the rotator cuff muscles helping to prevent abnormal joint translation.[6]

The rotator cuff muscles have independent actions that in combination contribute to stability during mid and end range motions of the glenohumeral joint, working in both a concentric and eccentric manner. The rotator cuff muscles also provide compressive force across the joint, helping to centralise the humeral head in the glenoid fossa.

Injury to either the static or dynamic stabilisers of the shoulder may compromise function resulting in instability. In general terms this can be anterior, posterior, multi-directional, traumatic or atraumatic. We like to use the Stanmore classification system, which is based on three polar groups – traumatic structural, atraumatic structural and habitual non-structural (muscle patterning).[7] Basing these three poles as the points of a triangle it is possible to establish a continuum where a patient may fit into one of the three groups, or as is often the case, overlapping and moving between more than one group.

2. Pathogenesis

Posterior instability is less common than anterior instability, and accounts for between 2 and 12% of cases of instability.[8,9] It was typically described as occurring in patients who have experienced posterior dislocation due to seizures, electrocution. In an anatomically normal shoulder it is now considered in three broad etiological categories: acute trauma, repetitive microtrauma and purely atruamatic.[10–12] The most frequent cause being repetitive microtrauma to the posteroinferior shoulder complex often seen in young, active people performing activities such as bench pressing, rugby, rowing and swimming.[13] These activities result in repetitively loading the glenohumeral joint in a flexed internally rotated position, stretching and injuring the PIGHL and posterior labrum. Anatomical abnormalities in glenoid version, hypoplasia and humeral retroversion can also contribute.[8,14,15] We have also found traumatic posterior instability in a high number of contact athletes [REF].

3. Clinical assessment of the posteriorly unstable shoulder

The basis of diagnosing posterior instability is a careful history and physical examination of both the symptomatic and asymptomatic shoulders. Factors to bear in mind during assessment include:

- How the problem affects their activities of daily living
- How the problem affects their work or sporting lives
- What pathology is present or likely to be present
- An appropriate management plan

Often the diagnosis is not clear and several shoulder complaints can arise from different shoulder relate disorders. The primary complaint is often an aching pain with weakness located around the posterior joint line, biceps tendon or superior aspect of the cuff. The physical examination aims to reproduce the symptoms experienced by the patient. Often in cases of posterior instability symptoms are exacerbated with the arm placed in 90° flexion, adduction and internal rotation.[16]

The patient should be assessed for generalised laxity using the Beighton Score. A score of 6/9 or greater indicates hypermobility but not necessarily benign joint hypermobility syndrome.[17] Throughout the clinical assessment it is necessary to bear in mind the difference between laxity and instability. Lax patients can have the same degree of glenohumeral translation as an unstable patient but report no symptoms or discomfort.[18] In fact ligamentous laxity is often seen in athletes where it may provide an advantage in their sport, but this can be associated with an increased incidence of joint instability, for example in rugby union players, laxity in the shoulder joints may confer increased risk for dislocation.[19]

4. Clinical tests for posterior laxity

4.1. *Posterior drawer test*

In 1984 Christian Gerber and Reinhold Ganz discussed the lack of attention in the literature of clinical diagnosis of shoulder instability; instead most accounts were focussed on the surgical procedures themselves.[20] They attributed some of the failures of the surgeries to not adequately detecting anterior and posterior instabilities and so described the anterior and posterior drawer tests. The posterior drawer test requires the patient to be supine with the examiner level with the shoulder, the proximal forearm is held by the examiner who then flexes to the elbow to approximately 120° and moves the shoulder to be abducted from 80° to 120° and flexed forward of 20°–30°. Holding the scapula with the other hand, with the thumb placed lateral to the coracoid process. The humerus is then slightly medially rotated and flexed further to 60° or 80°, the thumb placed lateral to the coracoid subluxes the head of the humerus posteriorly which can be felt by the fingers behind the shoulder. The patient often responds with apprehension when this is performed. There is a lack of published research showing sensitivity and specificity figures for this test (Fig. 1).

4.2. *The load and shift test*

The load and shift test examines glenohumeral translation and should be performed with the patient sitting in an upright neutral position and also supine.[20] With the examiner behind the shoulder a hand over the scapula helps to stabilise it and then the humerus is held and “loaded” into the glenoid fossa

Fig. 1 – Posterior drawer test.

by applying an axial load, compressing the joint. The humeral head can then be moved anteriorly and posteriorly. The test is repeated in the supine position with the arm positioned in slight abduction and forward flexion.[21] The amount of translation felt varies and as such is graded[22]:

- +0 No translation from being centred in the glenoid fossa
- +1 Translation but not to the rim
- +2 Translation to the humeral head onto the glenoid rim
- +3 Translation over the glenolabral rim
- +4 Translation with complete dislocation and manual reduction required

Other variations of the load and shift test exist with the patient seated with the arm relaxed by their side, and the patient supine with 20° and 90° abduction. These give sensitivity and specificity figures for posterior load and shift as 14% and 100% respectively (Fig. 2).[23]

5. Clinical tests for posterior instability

5.1. *The jerk test*

The jerk test can be performed sitting or supine, the examiner takes the arm and flexes the elbow to 90° and abducts it horizontally.[24] Holding the arm at the elbow and stabilising the scapula with the other hand, the humerus internally rotated and then adducted across the patient's body. A sudden clunk or jerk as the humeral head slides off the back of the glenoid is a positive result.

Kim et al[25] concluded that in a shoulder with symptomatic posteroinferior instability the presence of pain when the jerk test was performed was indicative of a posteroinferior labral lesion. Pain with the jerk test was 89.7% sensitive and 85% specific, with a positive predictive value of 72% and a negative predictive value of 94% (Fig. 3).

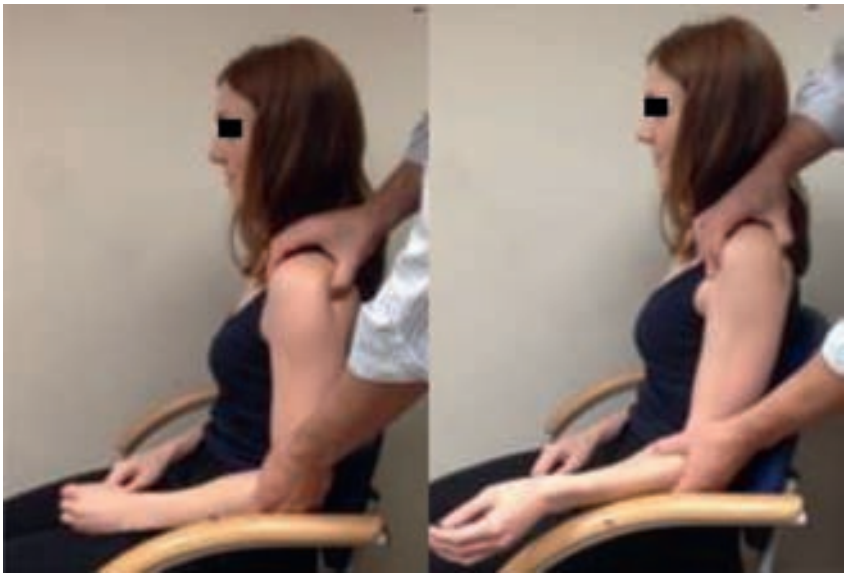

Fig. 2 – Load and shift test, with anterior and posteriorly directed loading.

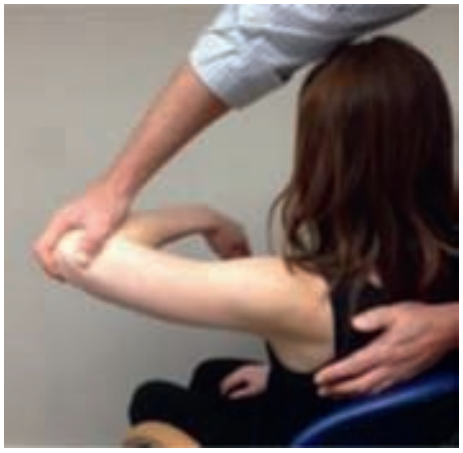

Fig. 3 – The jerk test is shown in a seated patient. The examiner stabilises the scapular, and provides flexion and internal rotation with a posteriorly directed force at approximately the 7 o'clock direction. A positive test reproduces the patient's symptoms when the shoulder is provoked in this manner and is consistent with the diagnosis of posterior instability.

5.2. *The Kim test*

The Kim test is performed with the patient seated and the arm in 90° of abduction (Fig. 4).[26] To perform this test, the clinician grasps the patient's elbow with one hand, while with his or her other hand, the clinician grasps the lateral aspect of the proximal arm, applying an axial loading force. While elevating the patient's arm to 45°, the clinician applies a downward and posterior force to the upper arm. Pain signifies a positive test regardless of an accompanying clunk. They reported a sensitivity of 80%, specificity of 94%, positive predictive value (PPV) was 0.73 and negative predictive value was 0.95. Combined with a jerk test they concluded the sensitivity of detecting a posteroinferior labral lesion was 97%.

5.3. *Posterior stress test and posterior apprehension test*

Again this is performed in a seated position.[27] The scapula is fixed medially whilst applying a posterior force to the arm held in a 90 forward flexed position, adducted and internally rotated position. It is considered positive if it reproduces the patients symptoms along with subluxation or dislocation. For the posterior apprehension test the patient is once again supine, the examiner holds the elbow and stabilises the shoulder with the other hand. The arm is positioned with the shoulder flexed to 90° and internally rotated; the examiner then applies pressure along the axis of the humerus in a posterior direction. A positive test occurs when the patient responds with apprehension and guarding, to prevent the shoulder from subluxating (Fig. 5a).

Jia et al published the results of their study that involved 1913 patients undergoing shoulder surgery at their centre from 1995 to 2008. Posterior instability was one of the diagnoses they examined and collected data on. Their results showed a sensitivity and specificity of the posterior apprehension test were 19.2% and 99.2% respectively with a likelihood ratio of 25.[28] Therefore in a person who gives a clear history of posterior subluxation or dislocation this would be valuable in confirming the suspected diagnoses, however, in a person giving a vague history of an unstable shoulder this test could not be used to rule out posterior instability (Fig. 5b).

5.4. *Wrightington Posterior Instability Test (WPIT)/ Modified O'Brien's Test*

In many cases of posterior instability, patients present with posterior pain and clicking instead of true dislocations. We have found this predominantly in muscular contact athletes. These patients have excess posterior laxity and translation, posterior glenohumeral joint pain in hyperabduction and external rotation. This is a form of subclinical instability. These patients will exhibit marked weakness and pain in resisted flexion in full adduction and internal rotation at 90° – a similar position to the O'Brein's test. This is probably due to

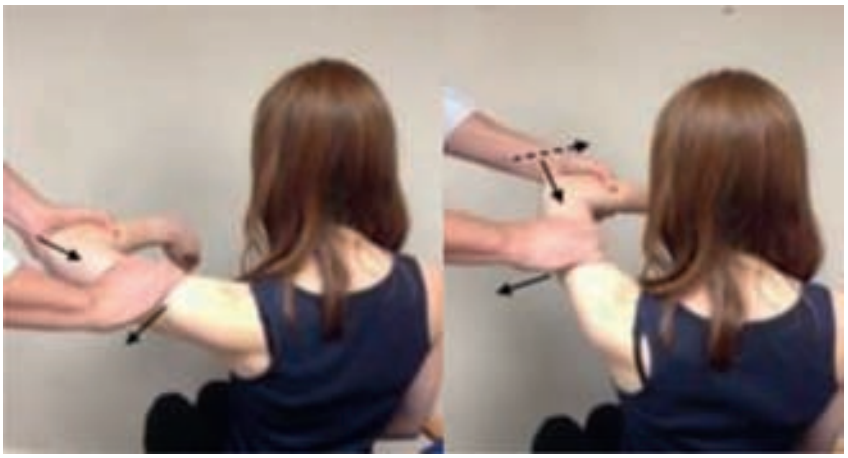

Fig. 4 – The Kim test.

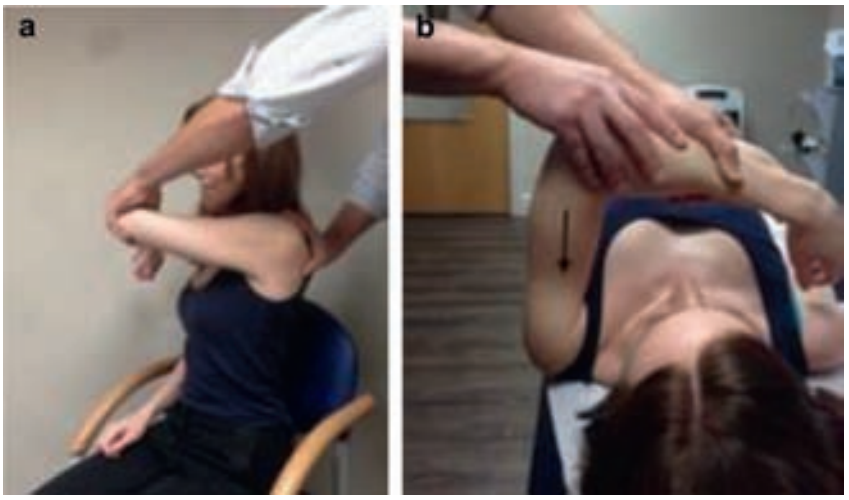

Fig. 5 – a: Posterior stress test. b: Posterior apprehension test.

posterior translation of the humeral head in the position of flexion and internal rotation, with resultant posterior cuff weakness. We are currently validating this test (Fig. 6).

6. Imaging

As an adjunct to history and examination the role of magnetic resonance imaging (MRI) has become a mainstay. MRI is a static study so instability alone cannot be diagnosed, but the presence of labral pathology in conjunction with clinical findings are utilised. Most commonly used is direct MRI arthrogram with gadolinium injected intra-articularly into the glenohumeral joint. Multiple studies have reported sensitivities and specificities of over 90% in detecting labral lesions.[29,30] The use of indirect MRI (I-MRI) has been advocated in the past.[31] The technique involves an intravenously administered contrast agent, which enhances the joint space producing an arthrographic effect. Its perceived weakness is not distending the joint space to show subtle labral detachment. Recent work on I-MRI for labral tears showed a sensitivity and specificity of 95% and 91%.[32]

7. Summary

The diagnosis of posterior instability comprises a good clinical history and detailed examination of laxity and instability. The shoulder may be lax but not symptomatic of any instability, so for appropriate management the pathological must be differentiated from the physiological. The presence of multiple tests to diagnose a condition is usually indicative of no one test being conclusively diagnostic. The validated tests for posterior instability, in particular the load and shift test and the posterior apprehension test, have high specificity but low sensitivity. This suggests the most useful time for these tests

Fig. 6 – Modified O'Briens/WPIT (Wrightington Posterior Instability Test).

is when posterior instability is already the main differential diagnosis based upon the history. In the future, clinical trials around assessment of posterior instability should focus on identifying tests with high sensitivity, which could be used as screening tests during examination of the shoulder, where a classical history of posterior instability is not present. We expect the WPIT test may fulfil this option.

Conflicts of interest

All authors have none to declare.

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Review Article

Current concepts in articular cartilage repair

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ABSTRACT

Articular cartilage is a specialized connective tissue covering vrious joint surfaces. Due to its poor repair potential and no nerve supply early injuries can be easily missed. Articular cartilage injury poses a challenge to treating orthopaedic surgeons and with various treatment options available it becomes difficult to treat due to the limited self-healing capacity, affliction of a young active patient and risk of progression to secondary osteoarthritis. There is no universally accepted successful treatment for these lesions. The ideal treatment should provide good repair fill with hyaline cartilage and maintain quality of subchondral bone. There is an increasing need for high quality studies to evaluate and compare outcomes between different techniques currently available. This article discusses articular cartilage injury and the various treatment options available to the treating surgeon along with the future upcoming treatment options.

1. Introduction

Articular cartilage is a specialized connective tissue covering joint surfaces. It also has no nerve supply and is therefore not sensitive to early injuries. It also has poor repair properties, because there are relatively few cells in the tissue, the metabolic rate is low, and the matrix fibres restrict the capacity of chondrocytes to divide and migrate in the articular cartilage.[1] As a consequence, it is generally agreed that articular cartilage does not repair significantly after injury.[2]

Articular cartilage injury poses a major challenge to the treating orthopaedic surgeons due to the limited self-healing capacity, affliction of a young active patient and risk of progression to secondary osteoarthritis.[3] There is no universally accepted successful treatment for these lesions. The ideal treatment should provide good repair fill with hyaline cartilage and maintain quality of subchondral bone. There is an increasing need for high quality studies to evaluate and compare outcomes between different techniques currently available. This article discusses articular cartilage injury and the various treatment options available to the treating surgeon along with the future upcoming treatment options.

2. Response to injury

Deep lacerations of articular cartilage extending beyond the tidemark heal with fibrocartilage produced by undifferentiated mesenchymal cells. Superficial lacerations do not heal, although some proliferation of chondrocytes may occur.[4] Immobilization of joints leads to atrophy of the articular cartilage and therefore continuous passive motion is believed to be beneficial to healing. In arthritic cartilage, chondrocytes are recovered in clusters of upto thirty cells, which probably represents an attempt at tissue regeneration.[5]

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3. Why do we need hyaline cartilage repair tissue?

In fibrocartilage the matrix component is minimal, and the fibrous one greatly predominates. The chondrocytes are less numerous and much more widely separated than in other types, but most of them are still enclosed in lacunae.[5] Repair tissue that fills osteochondral defects is less stiff and more permeable than normal articular cartilage. The orientation and organization of the collagen fibrils in even the most hyaline-like chondral repair tissue do not follow the pattern seen in normal articular cartilage. The decreased stiffness and increased permeability of repair cartilage matrix may increase loading of the macromolecular framework during joint use and result in progressive structural damage, thereby exposing the repair chondrocytes to excessive loads. The remaining cells often assume the appearance of fibroblasts as the surrounding matrix comes to consist primarily of densely packed collagen fibrils. This fibrous tissue usually fragments and often disintegrates, thus leaving areas of exposed bone. The inferior mechanical properties of chondral repair tissue may be responsible for its frequent deterioration.[6]

4. Natural history of focal chondral defects

The natural progression of untreated chondral defects is still unclear.[7] Linden noticed 55% of patients who were diagnosed with osteochondritis dessicans after the closure of distal femoral physis progressed to osteoarthritis compared to zero percent of patients who were diagnosed as osteochondritis dessicans before the closure of distal epiphyseal line.[8]

Widuchowski retrospectively analysed 25, 124 arthroscopies. Cartilage lesions were classified in accordance with the Outerbridge classification.[9] Focal cartilage lesions were localized in 67%, osteoarthritis in 29%, osteochondritis dessicans in 2% and other types in 1% of the patients in this study. The patellar articular surface (36%) and the medial femoral condyle (34%) were the most frequent sites of the cartilage lesions. Curl noticed that patients under 40 years of age with grade IV lesions accounted for 5% of all arthroscopies.[9]

Lars Engebretsen in a prospective study on 993 knee arthroscopies noticed articular cartilage pathology in 66% and a localized cartilage defect was found in 20%.[10] A localized full-thickness cartilage lesion (ICRS grade 3 and 4) was observed in 11% of the knees. Of the localized full-thickness lesions, 55% of lesions (in 6% of all knees) had a size above 2 cm. Brittberg[11] in another prospective study of 1000 arthroscopies noticed focal chondral defects (ICRS grade 3 and 4) in 19% of patients with average size 2.1 cm^2. The medial femoral condyle was the commonest site for articular cartilage pathology in this study.

5. Clinical diagnosis

The spectrum of chondral pathologies seen in practice are osteochondral traumatic injuries, focal chondral defects and early osteoarthritis.[5] Traumatic osteochondral defects are common with patella dislocations and other significant knee trauma. Patello-femoral joint assessment should include an assessment of hypermobility and maltracking. These should be suspected by the presence of acute onset of significant swelling soon after the injury with lipo-haemarthrosis and with or without osteochondral fragment on radiographs.

Chondral defects have to be differentiated from early OA and the duration of symptoms could help in the decision making.[12] Patients with articular cartilage defects commonly present with knee pain often exacerbated by impact or weight-bearing. These can commonly be misinterpreted clinically with the meniscal injury in the presence of generalized degeneration.

Plain radiographs are essential in the initial assessment especially to rule out early osteoarthritis. Weight bearing long-leg alignment X-rays to assess normal knee alignment is mandatory before consideration of cartilage repair.

MRI scans using cartilage-sensitive sequences like fast spin echo or spoiled gradient-recalled echo are useful to estimate the cartilage loss, fissuring and delamination, underlying subchondral bone and the other structures in the knee.[12] In addition to diagnosing the location and size of defects, detailed cartilage MRI can identify reduction in cartilage volume, changes to GAG (Glycosaminoglycans) and collagen content and can also assess repair tissue. Standard MRI using a cartilage-sensitive sequence (e.g., spoiled gradient-recalled echo or fast spin echo) can show cartilage fissuring, delamination, and focal loss as verified by arthroscopy.[13,14] Quantitative and semi quantitative cartilage imaging techniques are now available and include dGEMRIC (delayed gadolinium-enhanced MRI of cartilage), sodium-23 imaging, T1rho, T2*, and T2 mapping techniques.[13] In comparison with traditional MRI, which emphasizes morphology, these additional techniques help to evaluate cartilage composition. In broad terms, dGEMRIC, sodium, and T1rho are sensitive to proteoglycan content, while measurement of T2 or T2* relaxation times are sensitive to collagen architecture, specifically collagen orientation. To assess the collagen orientation and free water content of repair tissue, T2 mapping techniques can be used.[15]

6. Arthroscopy

Arthroscopy is still the gold standard for assessment of cartilage lesions especially to assess lesion grade and edges and also to identify those suitable for repair.[16]

Numerous cartilage defects classification systems are in place including Insall, Outerbridge, Beuer and International cartilage repair society (ICRS) grading system.[9,11,17] ICRS grading system is more comprehensive and is increasingly used by the surgeons.[18]

The ICRS grading system is graded into 4 grades with each grade further subgraded to accurately evaluate the cartilage injury.

Grade 0 Normal (Fig. 1)
Grade 1 Superficial lesions
A Soft indentations (Fig. 2)
B Superficial fissures/cracks (Fig. 3)
Grade 2 Abnormal lesions extending down to <50% of cartilage depth (Fig. 4)
Grade 3 (Fig. 5)

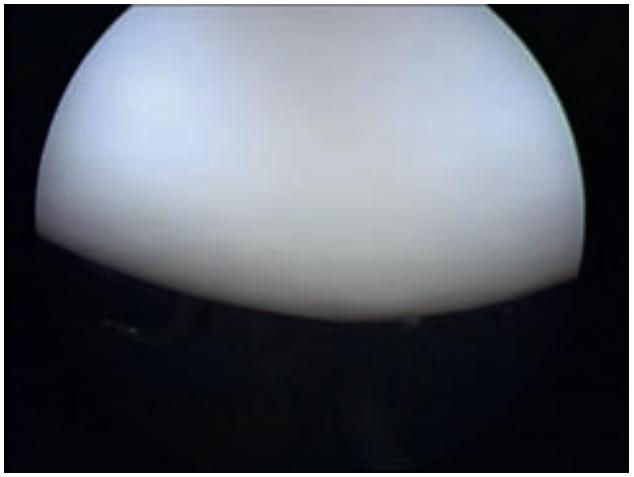

Fig. 1 – ICRS Grade 0: Normal articular cartilage.

A Severely abnormal cartilage defects extending down >50% of cartilage depth
B As well as down to calcified layer
C Down to but not through the subchondral bone D Blisters

Grade 4 Severely abnormal subchondral bone exposure (Fig. 6)

6.1. *ICRS classification of OCD lesions*

ICRS OCD 1: Stable, continuity: softened area covered by intact cartilage
ICRS OCD 2: Partial discontinuity, stable on probing
ICRS OCD 3: Complete discontinuity, "dead in situ", not displaced
ICRS OCD 4: Displaced fragment, loose within the bed or empty defect
If lesion is <10 mm deep (IV A)
If lesion is >10 mm deep (IV B)

Fig. 2 – ICRS Grade 1a: Superficial lesions with soft indentations.

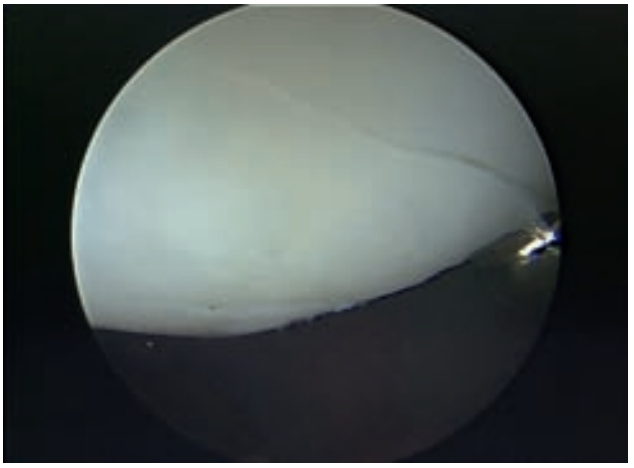

Fig. 3 – ICRS Grade 1b: Superficial lesions with superficial fissures/cracks.

7. Indications for surgery

Each patient treatment should be individualized based on lesion aetiology, size and location of lesions, duration of symptoms, state of subchondral bone, number and type of previous interventions. Patient characteristics that influence outcome are activity level, smoking history, demographics, body mass index and rehabilitation compliance.[13] An understanding of the knee as an organ especially considering the menisci, ligaments and knee alignment is necessary before embarking on any treatment. The duration of symptoms, patient's age, body mass index, previous failed treatment including physiotherapy and patient's compliance with rehabilitation also play a significant role in outcome.[13] Patellofemoral lesions respond less favourably to cartilage repair than femoral condyle lesions.[19] Smokers also have poor outcomes following cartilage repair.[20]

The previously published precise indications for surgery[5] include

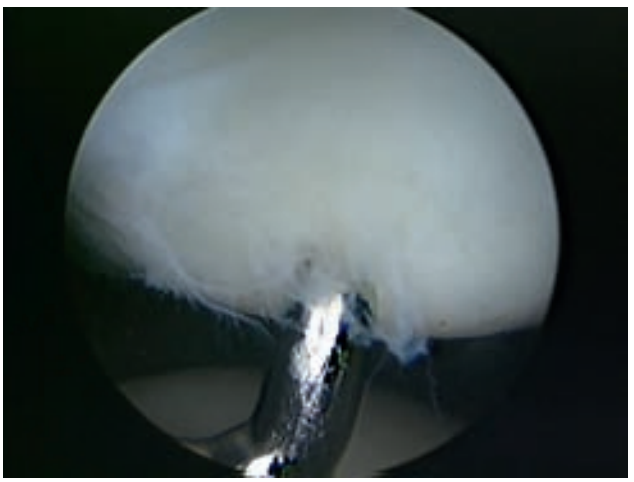

Fig. 4 – ICRS Grade 2: Abnormal lesions extending down to <50% of cartilage depth.

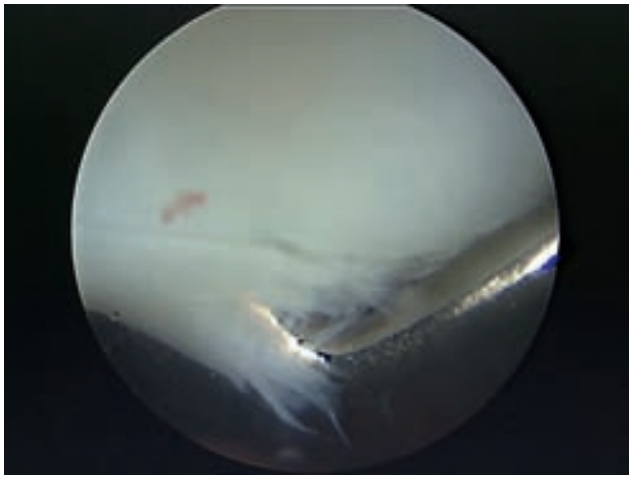

Fig. 5 – ICRS Grade 3: Abnormal cartilage defects extending down >50% of cartilage depth.

- Acute traumatic lesions more than 1 cm^2
- Symptomatic lesions of Grade 4
- Asymptomatic lesions in active individuals
- Distal femoral and trochlear lesions

8. Cartilage repair options

Different cartilage options are:

1 Arthroscopic debridement of localised defects (chondroplasty)
  a Mechanical
  b Thermal
2 Bone marrow stimulation techniques
  a Microfracture
  b Subchondral drilling
3 Chondral and osteochondral autograft/allograft
  a Mosiacplasty/OATS
  b Autologous chondrocyte implantation (ACI)
  c One step stem cell therapy
4 Synthetics and scaffolds

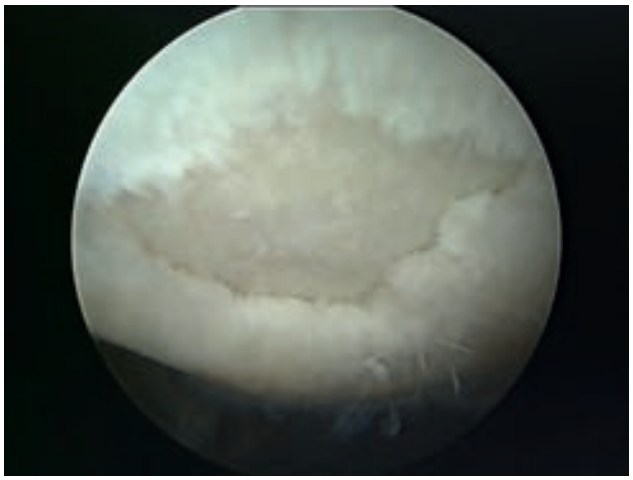

Fig. 6 – ICRS Grade 4: Subchondral bone exposure.

Non-operative treatment such as physical therapy, non-steroidal anti inflammatory drugs (NSAID), hyaluronic acid should always be considered in small lesions with minimal symptoms.

Traditionally, treatment options have been on a stepladder approach with progression from palliative to reparative treatment. Whilst techniques like chondroplasty and microfracture are used widely as first-line treatment, reparative options like osteoarticular autograft/allograft or autologous chondrocyte implantation are often provided as second line treatment.

8.1. *Arthroscopic debridement of localised defects (chondroplasty)*

The term chondroplasty is used for mechanical or thermal reshaping of uneven articular cartilage. The aim is to debride loose chondral flaps and fibrillated articular cartilage to a smoother surface while avoiding any damage to healthy surrounding cartilage.

There are two types of arthroscopic chondroplasty:

- Mechanical – performed using mechanical instruments and arthroscopic shavers
- Thermal – performed using radiofrequency energy

Chondroplasty has good success rate in improving pain and mechanical symptoms.[21] However, the natural history of progression is not clear and the long-term effects of radiofrequency treatment on cartilage remain unknown. Mechanical chondroplasty using a shaver can still leave behind a fine fibrillated surface. Some authors have reported superior results with Radiofrequency (RF) compared to mechanical shaver.[22–24]

Thermal chondroplasty produces chondrocyte death in the surrounding cartilage; potentially even upto subchondral bone.[21,25,26] Lu reported that Bipolar RF could produce a wider and deeper zone of cell death compared to Monopolar.[21] Lavage fluid at 37 °C produces less chondrocyte damage than fluid at 22 °C. Caffey showed that for treatment times of 1 and 3 s, cell death measurements ranged from 404 to 539 μm and 1034 to 1283 μm, respectively.[25] When probes were kept a 1.0-mm distance above the cartilage, no cell death or cartilage smoothing was noted. Both shavers and RF probes should be used like a paintbrush to minimise any damage.

Arthroscopic debridement for focal chondral lesions is commonly performed but there are very few comparative studies with other cartilage repair techniques.

Hubbard prospectively compared debridement ($n = 40$) and washout alone ($n = 36$) for localised medial femoral condyle lesions at 4.5 years. The washout group performed poorly. 19 of a total of 32 survivors in the debridement group were painfree.[27]

Freddie Fu retrospectively compared arthroscopic debridement and autologous chondrocyte implantation (ACI)[26] and showed ACI patients had better outcomes in function and pain relief at 3 years but far higher reoperations in the ACI group.

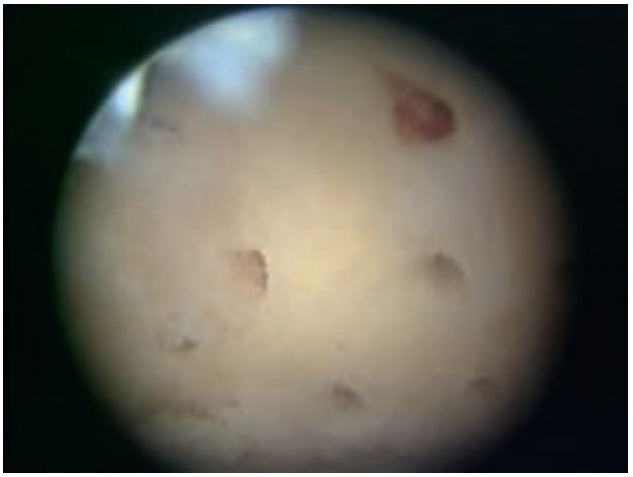

Fig. 7 – Showing arthroscopic view of the microfracture.

The key technical tips in Chondroplasty are -

- Using copious washout
- Use of suction along with application of a non-aggressive shaver blade like a paintbrush
- Swapping portals with arthroscope and shaver to improve surface finish

8.2. *Bone marrow stimulation techniques*

Various marrow simulation techniques have been described in the literature and all of them are based on the principle of exposing the chondral defect to the bone marrow thus trying to create an environment of fibrocartilage healing. These marrow stimulation techniques include microfracture and subchondral drilling.

Pridie developed a drilling technique to bring pluripotent stem cells in to a chondral defect[28] but this was superseded by microfracture, a technique devised by Steadman et al to reduce thermal damage potentially produced by drilling. Steadman and Rodkey[29] described the technique for microfracture along with exacting rehabilitation programme and showed statistically significant improvement in function and pain. Multiple perforations 4 mm deep and 4 mms apart are made using awls in to the subchondral plate (Fig. 7). The calcific layer covering the defect is curetted and the edges are prepared in such a way to create healthy vertical margins. The perforations are commenced from the periphery and uniformly spaced each being perpendicular to the subchondral plate.

Mithoefer in a systematic analysis looked at 28 studies with microfracture of which only 6 were randomized controlled studies.[30] The outcomes of microfracture were improved at 24 months but subsequently deteriorated. The problem with microfracture has been poor cartilage fill and more fibrous or calcific repair tissue. Newer techniques to improve the results of microfracture by addition of a scaffold or by changing the technique of drilling are more promising.[30]

8.3. *Chondral and osteochondral autograft/allograft*

Osteochondral autograft treatment is potentially useful in small lesions with subchondral bone loss. Small lesions of less than 2 square cm can be effectively restored to hyaline-like cartilage using autologous ostechondral plugs harvested from a non-weight bearing of the knee.[31,32] Instrumentation is provided by Arthrex (OATS)® Arthrex Inc. and Smith and Nephew (Mosaicplasty) trademark of ®Smith and Nephew USA. There is donor site morbidity but there are also advantages in this being a one step technique with consistent survival of hyaline cartilage and ability for early aggressive rehabilitation especially in elite sports participants.[33] In cases of large defects with subchondral bone loss there are many published successful reports of the use of fresh osteoarticular allografts.[34] Minced cartilage autograft and particulated juvenile cartilage allograft have now also been reported as grafts for chondral repair.[35]

8.4. *Autologous chondrocyte implantation (ACI)*

This is a two-stage biological treatment procedure aiming to produce hyaline-type cartilage repair. Firstly, a biopsy of healthy cartilage is taken from the affected knee and the chondrocytes are cultured in a suitable environment. The second stage is an open procedure when the cells are reimplanted a few weeks later into the defect beneath a periosteal patch or alternative scaffold.

Until recently ACI has been used for failed primary treatment in a full-thickness chondral lesions and its superiority compared to microfracture was questioned.[36] Newer generations of ACI (Characterised chrondrocyte implantation (CCI) and Matrix-guided autologous chondrocyte implantation (MACI)) that involve cells placed underneath scaffolds have reduced the complications of periosteal hypertrophy seen earlier and have shown improved outcomes on comparative trials.

There are two cell therapy products (®CCI and ®MACI) currently available that have the Advanced Therapeutic Medical Licence in Europe. Economic modelling using some assumptions about long-term outcomes suggests that ACI would be cost-effective because it is more likely to produce durable hyaline cartilage and delaying osteoarthritis.

8.5. *One step cell therapy*

Active research is in progress to achieve stem cell based treatment as a single step technique. Though various sources of progenitor cells have been identified and tried in animal studies to produce cartilage, but there is no safe reliable technique yet identified for cartilage repair. Bone marrow aspirate concentrate has been used successfully as an adjunct to microfracture and platelet rich plasma (PRP) is increasingly thought to have growth factors to initiate cartilage repair. PRP is prepared by differential centrifugation of autologous whole blood and contains a higher concentration of platelets compared with untreated blood, but more specific methods of preparation and attributes have not been uniformly defined. In particular, the presence of leukocytes, monocytes, macrophages, and mast cells in many platelet concentrates is controversial. Randomized controlled clinical studies are required to evaluate the potential of such options in patients.

8.6. *Synthetics and scaffolds*

A lot of acellular commercial products have been available to treat focal defects. These are scaffolds or synthetic plugs. Some scaffolds that have been used are as Trufit plug (Smith & Nephew), Chondromimetic (Tigenix), BST Cargel (Biosyntech Canada). These are plugs or hydrogels that act as a scaffold and some are biphasic and augment a marrow stimulation technique.[37] Though early results with MRI show repair fill, there is concern that the repair is fibrous tissue with foreign body giant cells identified at revision surgery.[16,37]

Synthetics resurface the local defect as a plug and many products are being evaluated such as SaluCartilage-polyviny alcohol-hydrogel (Solumedica) and Chondrocushion-polyurethane plugs (Advanced Bio Surfaces, Inc).

9. The future

Tissue repair and regeneration has an exciting future. The combination of gene therapy, stem cell therapy, and tissue engineering as well as interdisciplinary collaboration between orthopaedic surgeons, material scientists, biomechanical engineers and molecular biologist is crucial for the future success of these technologies. The difficult proposition would be to develop an approved reliable technology to treat the varying complexities of articular injuries and early degenerative lesions.

10. Conclusion

The articular cartilage and its response to injury remain a very exciting area of orthopaedic research. It is important to understand the basic science of repair, this may help alter the course of acute chondral injury and potentially avoiding secondary damage. Despite the development of new cartilage repair procedures, the quality of the existing clinical evidence is limited.[38] Detailed methodological recommendations and a consensus statement were developed the ICRS for the statistical study design, patient recruitment, control group considerations, study end point definition, documentation of results, use of validated patient-reported outcome instruments, and inclusion and exclusion criteria for the design and conduct of scientifically rigorous cartilage repair study protocols. The authors recommend that until such evidence is available, guidelines for treatment of chondral lesions are developed by individual surgical societies and develop registries to gather good quality data.

Conflicts of interest

All authors have none to declare.

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Review Article

The anatomy and relevance of the iliopsoas in the young adult with hip pain: Role of arthroscopic intervention

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ABSTRACT

Hip pain is a significant problem in the young adult (15–40 years) affecting atleast one in 20 patients. Though most sources of pain are from the hip joint, it may also be caused by structures external to it, such as the iliopsoas muscle complex. Recent advances in radiological imaging and hip arthroscopy have increased our understanding of this muscle and its surgical management. We present a comprehensive review of the iliopsoas and its pathologies, with specific emphasis on its arthroscopic treatment.

1. Introduction

Hip pain is reported as affecting 9.2% of the general population,[1] with approximately 2.5% due to sports related injuries.[2] A study found that the prevalence of hip pain in East German adolescents (13–18 years) to be 6.5% while the North West Adelaide Health Study found hip pain to be self-reported in 5.2% of 20–49 year olds.[1,3] Pain in the hip can be caused by labral pathology and femoroacetabular impingement, however the iliopsoas complex can also be involved in a number of conditions affecting the hip.[4–6] With recent advances in magnetic resonance imaging (MRI) and ultrasonography (US), our understanding of the functional anatomy of the iliopsoas tendon and its associated problems has greatly improved.[7] Detailed knowledge of its anatomy and clinical presentation is required to treat them effectively. Hip arthroscopy offers an ideal means of identifying this problem as well as a minimally invasive technique of treatment.[8] In this article, we review the pathologies affecting the iliopsoas, modes of investigation and in particular the results of arthroscopic treatment.

2. Methods

We have provided a comprehensive review on the anatomy of the iliopsoas, conditions affecting it, investigations and its treatment. We also carried out a web-based search (PubMed) of all articles published in English-literature using the terms 'iliopsoas', 'psoas' and 'arthroscopy' (ending date May 2014). We excluded any reports on the open release of psoas tendon.

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Due to the paucity of Level 1 studies, we included all levels of evidence for this review.

3. Results

Our initial search under 'iliopsoas' revealed 1176 studies. However, only 39 studies were retrieved on combining the search with the term 'arthroscopy'. Of these 39 studies, there were 13, which were not relevant to our review. Hence 26 studies were included in the review for analysis. The studies regarding the outcomes of arthroscopic iliopsoas decompression and release are shown in Table 1. They have all shown an improvement of functional scores after arthroscopic release with very few complications. There was only one randomised controlled trial by Ilizaturri et al, which compared short-term results of 2 different techniques of arthroscopic release of iliopsoas for internal snapping syndrome.[9] They found no difference in the functional scores when the psoas tendon was lengthened at the lesser trochanter compared to the transcapsular region.

Treatment of conditions affecting the iliopsoas tendon would require adequate knowledge of its anatomy. We present here its gross and arthroscopic anatomy below before describing the relevant conditions and their management.

3.1. *Gross anatomy*

The iliopsoas is a compound muscle consisting of three muscles: iliacus, psoas major and psoas minor. Iliacus takes its origin from the iliac crest, the superior two-third of the iliac fossa, the ala of the sacrum, the anterior sacroiliac joint and the iliolumbar ligaments. Psoas major arises from the sides of the bodies and the intervening discs of the vertebrae T12 to L5, and the transverse processes of all the lumbar vertebrae.[10,11] It runs inferiorly and laterally along the posterior abdominal wall to pass beneath the inguinal ligament, where it is joined by iliacus to form the common iliopsoas tendon. The psoas minor muscle, absent in 40% of the population, originates from vertebral bodies of T12 and L1. It then inserts on the iliopectineal eminence and iliacus fascia. The musculotendinous junction of the muscle complex is situated anterior to the hip capsule between the iliopectineal eminence and the anterior inferior iliac spine.[12] The common iliopsoas tendon then inserts in to the lesser trochanter of the femur and a short segment of the proximal femoral shaft below. However the tendon could be bifid as reported by Shu et al in 2011, during revision arthroscopy.[13] The psoas tendon lies lateral to the femoral artery, while the iliopsoas bursa lies between the musculotendinous junction and the pelvic brim.[11] Hence the psoas tendon is easily palpable lateral to the femoral artery pulsations. Communication between the bursa and the hip joint occurs in approximately 15% of adults.[14] The cross-section of the iliopsoas at different levels delineates a higher tendon to muscle fibre ratio closer to its insertion.[15] The iliopsoas tendon-muscle complex at the level of the labrum, transcapsular iliopsoas release site in the peripheral compartment and at the level of the lesser trochanter is composed of 40% tendon/60% muscle belly, 53% tendon/47% muscle belly, and 60% tendon/40% muscle belly, respectively.[16] This has an implication on the site of iliopsoas tendon release or lengthening when it is planned arthroscopically. There is also a close anatomic relationship of the psoas tendon to the anterior capsulolabral complex suggesting that it may be a cause of labral injury.[12]

3.2. *Arthroscopic anatomy*

Identification and access to the psoas tendon arthroscopically can be done through the routine portals in either supine or lateral position. The tendon is accessible in the central as well as peripheral compartment. In the peripheral compartment the tendon is closely placed above the medial synovial fold at the junction with the zona orbicularis. Occasionally fraying of the capsule is visualised suggesting potential impingement of the tendons shown in Fig. 1. The capsule overlying the tendon can then be carefully dissected with a radiofrequency probe to reveal the tendon as shown in Fig. 2 and Fig. 3. In the central compartment a modest capsulotomy can identify the tendon at the 3'0 clock position in the paralabral sulcus.

3.3. *Pathologies involving the iliopsoas*

3.3.1. *Iliopsoas tendinopathy*

Iliopsoas tendinitis is described as inflammation and thickening of the iliopsoas tendon.[17] However, pathologically the process is a tendinopathy,[18] which is a disease of overuse.[19] This is commonly seen in young athletes, while secondary iliopsoas tendonitis is seen following hip arthroplasty due to chronic attrition.[20] It is commonly associated with a painful

Table 1 – Clinical studies reporting outcome of arthroscopic intervention for iliopsoas tendinopathy and impingement.

Study	Patients	Follow up	Outcome
Ilizaturi et al[9]	19	12 months	Improvement in WOMAC score in all patients
Contreras et al[52]	7	24 months	All patients had resolution of snapping, no complications or weakness in the musculature around the hip
Domb et al[5]	25	21 months	Improvement in mHHS and HOS postoperatively
Tey et al[48]	1	N/A	Asymptomatic postoperatively
Ilizaturi et al[8]	7	21 months	Good resolution of snapping
Fabricant et al[49]	67	6 months	Improvement in all patients but less in those with increased femoral neck anteversion ($p = 0.031$)
Nelson et al[50]	30	2 years	Improvement in 77% patients
Ilizaturi et al[47]	20	12 months	Improvement in all patients with one recurrence and no complications
Anderson et al[51]	15	12 months	Improvement in all patients with return to sport in 9 months

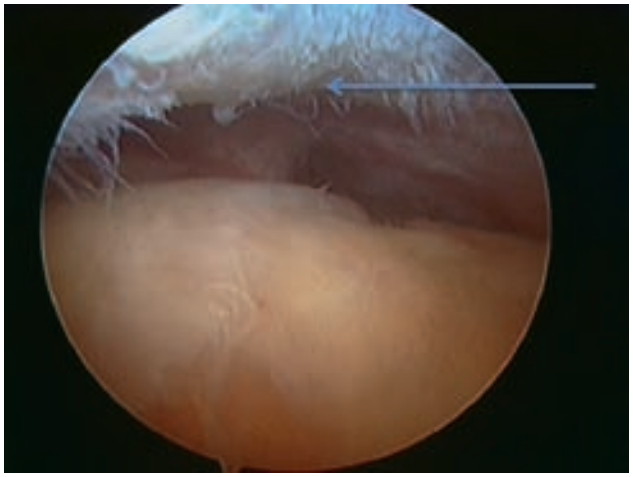

Fig. 1 – Arthroscopic view of the peripheral compartment showing capsular fraying in the region of the psoas (see arrow).

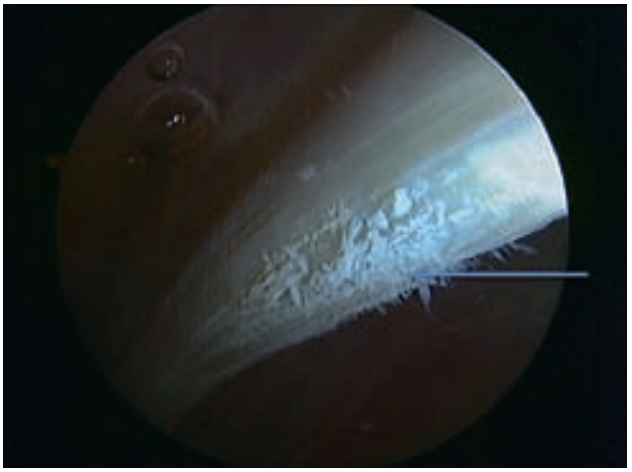

Fig. 3 – Arthroscopic view of the psoas tendon which shows an area of fraying of the tendon due to chronic attrition and previous steroid injection (see arrow).

snapping sensation and is therefore frequently considered within the context of the snapping hip syndrome[21]; more specifically internal snapping.[22]

3.3.2. *The snapping hip (Coxa saltans)*

Coxa saltans or the snapping hip syndrome is characterized by a sudden, painful, and audible snapping of the hip.[23] It is most frequently seen in women between 15 and 40 years of age.[21] It is also common in young athletes, most notably ballet dancers[24]; being self-reported as affecting 90.8% of questioned elite ballet dancers.[25] Snapping can be subdivided into three main categories: external, internal and intra-articular snapping. Intra-articular snapping is attributed to loose bodies within the joint itself due to; labral tears, osteochondral defects or synovial chondromatosis.[26] However, external snapping is caused by the iliotibial band (ITB) or glutaeus maximus snapping over the greater trochanter.[27–29]

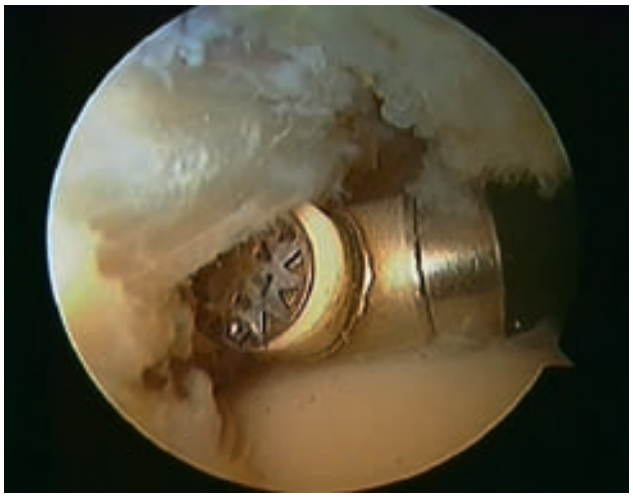

Fig. 2 – View showing decompression of the psoas tendon after capsular dissection with a radiofrequency probe.

Our discussion is only of the internal snapping which occurs due to a taught iliopsoas tendon snapping from lateral to medial over the closely associated iliopectineal eminence or femoral head during hip extension from a flexed position.[21,29,30] Deslandes et al in 2008, using dynamic sonography have now shown it to occur due to the psoas tendon abruptly rolling over iliacus to audibly snap against the superior pubic ramus. This occurs as the hip is brought back to neutral from a flexed, abducted, externally rotated (FABER) position.[31] They distinctly identify the snapping occurring at the inguinal level before iliacus has merged with psoas major to form the conjoint iliopsoas tendon. Other causes identified included; bifid iliopsoas tendons where the medial head abruptly flips over the lateral head and an anterior paralabral cyst which caused iliopsoas tendon impingement. Winston et al in 2007, also describe a mechanism in which the iliopsoas tendon becomes imbedded within the substance of the muscle belly producing a snap both on initial hip movement and the return to neutral.[26] The different mechanisms identified for snapping of the iliopsoas complex are strongly in keeping with this being a spectrum of the above similar clinical situations.[31]

While most cases are asymptomatic, patients may complain of a pain, which localises to the front of the hip or groin.[26,27] Snapping can be reproduced by moving the hip from the FABER position to an extended, adducted and internally rotated position confirming the diagnosis clinically.[26,27,32] In cases where eliciting snapping is difficult, it can be useful to get patients to demonstrate themselves.[21,33] Even if snapping is elicited, clinically distinguishing between internal and intra-articular causes can remain difficult. The tests that may exclude intra-articular causes of snapping are a negative impingement test and a negative McCarthy's sign, however they are not very specific.

3.3.3. *Iliopsoas impingement*

It has been suggested that a number of labral tears found incidentally at the time of arthroscopic iliopsoas release, performed for painful snapping hips could be as a direct consequence of iliopsoas tightness.[34,35] The most common

location for labral tears to be seen arthroscopically is in the anterosuperior region, which is described as the 1 to 2 o'clock position.[36] These are frequently seen in association with femoroacetabular impingement (FAI). Domb et al in 2011 report a distinct pattern of labral pathology seen at the direct anterior location of the labrum or 3 o'clock position, unattributed to any previously described aetiology for labral injury. These injuries are seen directly beneath the iliopsoas tendon, which consistently lies immediately adjacent to the capsule at the 2–3 o'clock position.[8] This close proximity strongly implicates iliopsoas as the causative factor in a process, which Domb et al term iliopsoas impingement (IPI). It is thought to be a repetitive attrition injury, which explains its prevalence in young athletes. It is seen almost exclusively in females with an average age of presentation of 19 years of age (range 12–37).[5,37] On examination, patients could present with a positive impingement sign and focal tenderness over the iliopsoas at the level of the anterior joint line.

3.4. *Investigations*

As with any hip pathology, plain radiographs should always be performed routinely to exclude bony abnormalities but they are often of little help in diagnosing snapping or IPI.[35] While magnetic resonance imaging (MRI) has been used in the past, magnetic resonance arthrography (MRA) has been found to increase the sensitivity and specificity for detecting labral tears from 30% to 36% respectively, to 90% and 91%.[38] The next investigation, which can be diagnostic, is iliopsoas bursography or tenography followed by fluoroscopy. This can be useful in demonstrating the abnormal movement of the iliopsoas tendon during hip motion, thus confirming it to be the cause of the snapping[28,39] (see Fig. 4). However, ultrasonography is the preferred technique for establishing a correlation between snapping and abnormal iliopsoas tendon dynamics allowing both static and dynamic evaluation of the tendon.[33,40]

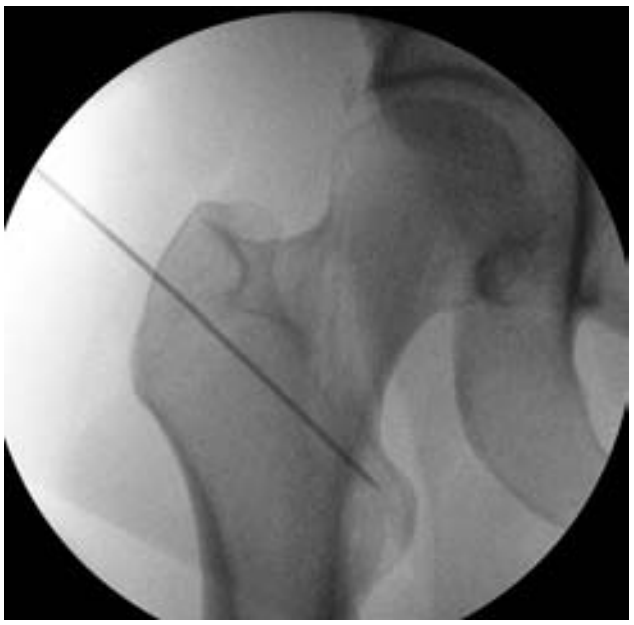

Fig. 4 – Fluoroscopic air bursogram performed before an iliopsoas injection.

It also has the advantage of being able to identify other associated signs of tendinopathy such as a tear or bursitis.[31] It can be used to perform a diagnostic local anaesthetic and steroid injection.[41] It however cannot exclude intra-articular causes such as labral tears. These are better seen on MRA, which have a high sensitivity and specificity for detecting labral tears.[38]

3.5. *Treatment options*

Most cases of iliopsoas tendinopathy and snapping can be treated conservatively with a structured physiotherapy programme, activity modification and anti-inflammatories.[4,17,27,34] For example a review of 30 patients by Gruen et al in 2002, found that 63% improved with 3 months of conservative management alone, and required no further intervention.[29] Where symptoms persist, steroid injection of the iliopsoas bursa may be of some benefit but results are inconsistent, with symptoms often returning after 2–8 months (see Fig. 3). Wahl et al in 2004 reported better results in three professional athletes by using ultrasound-guided steroid injections, which saw a pain free return to sport in four weeks.[19]

For those that fail to resolve with conservative management or steroid therapy then surgery may be indicated. While these were traditionally performed as open procedures, complications have been reported to occur in 43%–50% of patients, often in relation to the surgical incision.[22,27,41,42]

More recently arthroscopic release of the iliopsoas tendon has shown results comparable to or better than those seen with open procedures.[8,35]

The main procedures described are tendon decompression, step lengthening of the iliopsoas tendon[22,27,29,33] and iliopsoas tendon release.[28,43]

Hip arthroscopy also has the added benefit of allowing visualisation and treatment of any associated intra-articular pathology, which has been reported in more than half of patients undergoing hip arthroscopy for internal snapping hip syndrome.[4,35] It is also less invasive which allows earlier rehabilitation and return to function. Complications of this procedure are potential weakness of flexion and those associated with hip arthroscopy in general. While non-traumatic hip dislocation following arthroscopic iliopsoas tenotomy has been reported,[44] it is rare and tendon regeneration has been shown to occur on MRI studies.[45]

3.6. *Arthroscopic technique*

The iliopsoas tendon can be lengthened or released through the central compartment, peripheral compartment or at the lesser trochanter. The technique has been well described by Dienst et al in the peripheral compartment.[46] The medial synovial fold is identified and the capsule is dissected just medial and above it.

There is no consensus on the level of tendon lengthening, whether at the level of the labrum through transcapsular approach or at the mid-femoral neck region via the peripheral compartment or at the lesser trochanter using an extracapsular approach. In cases, where there is a labral tear due to the iliopsoas impingement, the release is done through the central compartment.[5] The labral injury itself is also addressed

most commonly by debridement or repair. It is important to perform an adequate capsulotomy to identify the tendon and to ensure that it is not bifid.[12] Postoperatively this is followed by physiotherapy and rehabilitation focussed on the psoas.

4. Discussion and conclusions

Iliopsoas pathology can be a common cause of hip pain in young patients particularly athletes and ballet dancers.[19,21,25] These comprise of iliopsoas tendinopathy, snapping iliopsoas syndrome and iliopsoas impingement (IPI).

Diagnosis of patients with a snapping iliopsoas is evident on history and clinical examination. Dynamic ultrasound examination is an investigation of choice, as it allows one to examine the tendons causing the snapping and also inject it with local anaesthetic and steroid.[33,40]

However the diagnosis of IPI can be difficult, as it can mimic other intra-articular causes of hip pain. An MR arthrogram can identify labral tears with good sensitivity and specificity,[38] while hip arthroscopy is the best way to confirm and treat a labral tear due to iliopsoas impingement. An injection of the psoas bursa either under fluoroscopic control or ultrasound guidance can help confirm diagnosis and provide symptomatic relief.[28]

In summary, adequate clinical examination along with key radiological investigations can help diagnose the problem effectively.[6,7,31,32] In case where all conservative measures fail, arthroscopic surgery seems to be an ideal choice and has shown good functional results. In patients with an increased femoral neck anteversion, the functional results are poorer.[50] Hence, before considering patients for an iliopsoas lengthening or release, it is imperative to examine their femoral anteversion.

The modes of arthroscopic treatment are decompression, lengthening and complete release. This can be done either paralabrally through the central compartment or transcapsularly in the peripheral compartment or extracapsularly near the insertion into the lesser trochanter. Performing a psoas release in the central compartment can avoid a large capsulotomy in order to access the peripheral compartment, however this will not allow access to deal with a concomitant cam deformity. The advantage of releasing at the lesser trochanter is avoiding entry into the hip joint. The exact location of tendon release is still a matter of debate, with all of the three sites of release showing equivalent results.[9,52] We postulate that this should be according to surgeons' preference and experience.

Conflicts of interest

All authors have none to declare.

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Original Article

The use of antibiotic impregnated absorbable calcium sulphate beads in management of infected joint replacement prostheses

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ABSTRACT

Aims: A multimodality approach is needed for management of infected joint replacement prostheses. We present our results in four patients managed surgically with standard techniques, with the addition of a local antibiotic delivery system using absorbable calcium sulphate beads.

Methods: A retrospective study was undertaken of 4 patients with infected hip or knee joint prosthesis. Two patients had infection in the hip and two had infected prosthetic knee joints.

Results: Patients were followed up in clinic for resolution of inflammatory markers and subsidence of signs of infection. Cure of infection was achieved in three patients at average 19 months follow up.

Conclusion: In this preliminary study, we found local antibiotic delivery using absorbable calcium sulphate beads to be an effective adjuvant to standard debridement, parenteral antibiotics and revision of implants.

1. Introduction

Infections in joint replacement prostheses are a source of significant morbidity. The management of infections involves a multidisciplinary approach. The role of the surgeon, primarily is achieving a reduction of bacterial load – through extensive debridement with or without removal of infected metalwork as appropriate. Additionally, targeted antibiotic therapy is essential to treat residual infection and achieve cure. In most studies, a combination of antibiotic loaded cement and systemic antibiotic therapy was used. The duration and route of administration of antibiotic therapy is a matter of some conjecture.

A prime objective of antibiotic therapy is to achieve a high concentration within the infected joint. Antibiotics in cement are an effective modality, but the exothermic reaction of cement polymerisation limits the choice to only heat-stable antibiotics.

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Table 1 – Summary of patients treated with Stimulan.

Patient	Existing prosthesis	Medical – comorbidities	Infecting organism	Operative procedure	Antibiotics used in Stimulan
AG	Revision knee replacement (three previous revisions for infection)	Diabetes, obesity	Group G streptococcus	Revision total knee replacement – single stage	Vancomycin
DW	Knee revision (for infection in primary knee)	Hemophilia, diabetes mellitus, compromised skin around knee	Coagulase negative staphylococcus	Fusion of knee using antegrade nail	Vancomycin
VW	Hip replacement – dislocated	Haemodialysis, diabetes, obesity	Staphylococcus aureus	Single stage hip revision	Vancomycin
EC	Femoral osteomyelitis with metalwork and displaced intracapsular fracture	Diabetes, long standing femoral osteomyelitis	Corynebacterium, coagulase negative staphylococcus, coliforms, pseudomonas	Cemented hip replacement	Daptomycin

We report our results on the use of Stimulan (Biocomposites Ltd, Keele, United Kingdom) as an absorbable medium for local antibiotic delivery in four patients with infected joint replacement prostheses.

2. Patients and methods

This retrospective analysis was undertaken with the approval of local audit department. Clinical notes, laboratory results and radiographs were studied.

Stimulan was used in two patients with infections in hip prostheses and in two patients with infected knee prostheses. The decision to use additional intraarticular antibiotics was based on –

1. Perceived complexity of the operative procedure.
2. Co-existing medical co-morbidities in the patient, which compromised host immune response.

The patients and their procedures are summarised in Table 1.

All operations were done in a scheduled list. Preoperative identification of the organism was based on joint aspiration in three patients, and on previous culture results in one patient (EC). All patients had raised inflammatory markers (Erythrocyte Sedimentation Rate – ESR, and C reactive protein – CRP) preoperatively consistent with the diagnosis of prosthetic infection. One patient (DW) had a discharging sinus anteriorly. The time lag between presentation and definitive surgery was less than 2 weeks. A close interaction with the microbiology service was maintained for all patients.

Surgery was carried out with removal of all infected metalwork and cement. A through debridement was done, and further samples obtained before administration of perioperative antibiotics. A clean set of instruments was used after debridement, with change of drapes, gowns and gloves.

The antibiotic beads are prepared intraoperatively by mixing the powdered antibiotic with the Stimulan rapid cure powder. The recommended dose of Vancomycin is 1 g in 10 cc of Stimulan powder. The mixing solution is then added and mixed for 30 s. The mixture is applied to the bead mat (Fig. 1) where it sets in 3–5 min. After setting, the beads are removed from the mat. Tobramycin has the same recommended dose but takes 10–20 min to set.

Three patients had Vancomycin in the Stimulan, while the fourth had Daptomycin based on sensitivity. Daptomycin was added in the dose of 1 g in 10 cc Stimulan. The bead mat acts as a template and allows three different sizes of beads (Fig. 1) and these can be chosen on the basis of clinical requirement. After the final wash, the largest beads were placed within the joint space, and the smaller beads inserted within the medullary canal (Fig. 2). Vancomycin 2 g was also added to each 40 g of cement. Cement was not used in knee fusion. Absorption of beads in vivo is complete by 4 weeks (Fig. 3).

Postoperative rehabilitation was with full weight bearing in all patients. All patients received intravenous antibiotics for 2 weeks followed by oral antibiotics for 4 weeks.

3. Results

Control of infection was monitored by resolution of clinical signs of infection and normalisation of inflammatory markers. All patients were followed up at 6 weekly intervals in clinic. Average follow up was 19 months. Three patients achieved resolution of infection, with primary healing of the

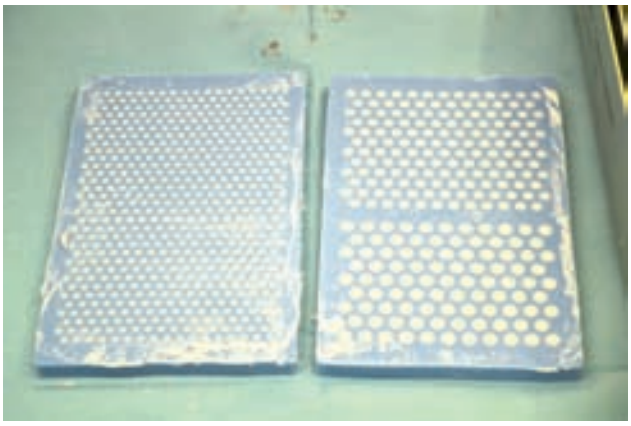

Fig. 1 – Intraoperative preparation of Stimulan beads mixed with antibiotics.

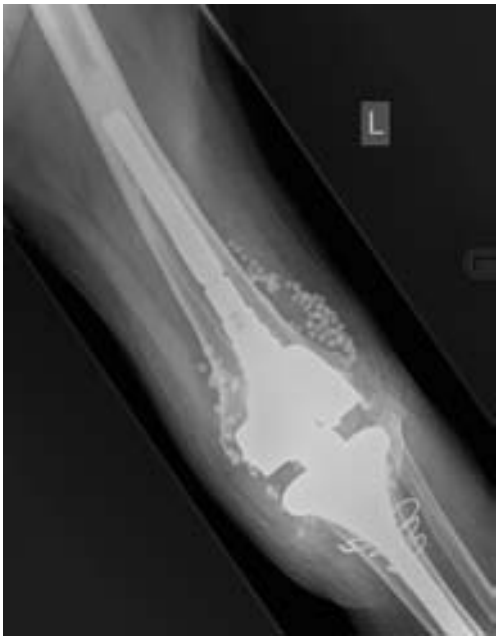

Fig. 2 – Postoperative radiograph showing the beads in the joint space and in the medullary canal.

operative wound. One patient (VW) had a painfree, functioning hip but persisting culture negative discharge, which was managed by regular change of dressings. Further surgery was deemed inappropriate in view of the multiple medical comorbidities, and compromised immune function.

4. Discussion

The prevalence of infection after prosthetic joint replacement varies between 0.86% and 1.5%.[1,2] Treatment of infection involves revision arthroplasty with removal of infected implants, all cement and thorough debridement. The success of revision arthroplasty depends on multiple factors – including immune response of host, appropriate antibiotic therapy, pathogenicity of infecting organism and rigour of debridement.[3]

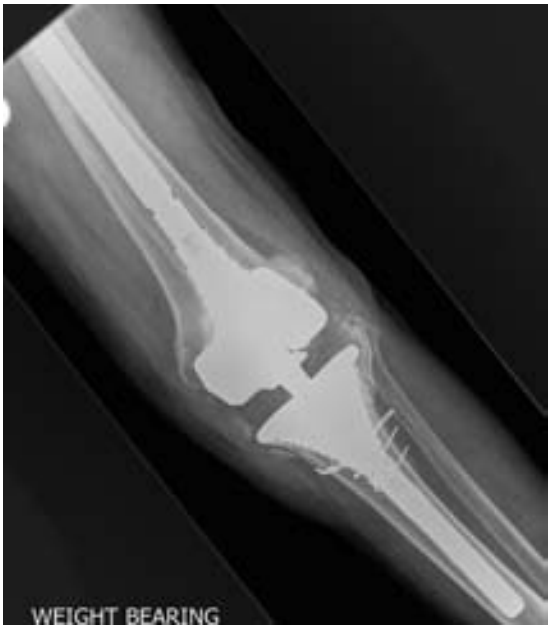

Fig. 3 – Radiograph after 6 weeks showing resorption of the beads.

Systemic administration of antibiotics may cause toxicity at higher doses, and hence local antibiotic delivery is a useful option.[4] An ideal local delivery system would be able to provide a local dose and be biodegradable so as to avoid a second surgical procedure for removal.

Antibiotics in cement are an effective method to enhance local concentration of antibiotics. However, in many cases, the cement is completely covered by the implant, hence restricting access of the cement to the joint space. Elution of antibiotics from the cement is limited[5] and only a small amount is able to permeate into the joint space. Cement beads can provide a high concentration in the joint, but again have a limited choice of antibiotics. As these are not absorbable, they require removal as a second operation and this can often be difficult due to the fibrous reaction around the beads. Additionally, only heat – stable antibiotics can be used with the cement, and this severely restricts the choice of antibiotic.

The present report focuses on an absorbable local antibiotic delivery system. Calcium sulphate has been used as filler in orthopaedic surgery for many years.[6] Mixing the hemihydrate powder form with water leads to formation of dihydrate, which can be moulded into beads. Mixing the antibiotics with the powder results in antibiotics loaded beads. The antibiotic is slowly released as the beads are resorbed. A variety of antibiotics can be added to calcium sulphate[7] including Vancomycin, tobramycin, teicoplanin, cefazolin and fucidin. The setting time is the time taken for conversion from hemihydrate to dihydrate. Vancomycin shortens the setting time, while tobramycin delays setting. Daptomycin can be chosen in situations where Vancomycin resistant Gram positive organisms are grown on cultures. Daptomycin is considered an appropriate choice in this setting[8] as it is effective against bacteria found in the biofilm. The elution of Daptomycin from the pellets starts at a high level and then reduces rapidly over the next 3 days.[9]

Stimulan is synthetic, biodegradable calcium sulphate and is fully absorbed in vivo. As it is prepared synthetically, it does not contain impurities which may be present in naturally occurring forms of calcium Sulphate. It cures at lower temperature and hence enables use of a wider range of antibiotics locally. It is completely resorbed in three to four weeks, and hence entire antibiotic is eluted into the joint space.

This report is a preliminary study involving four patients. All patients had debridement, removal of metalwork and cement and reimplantation/refixation as would be done for infected joint replacement prosthesis. All had antibiotics in the cement and postoperative antibiotics for 6 weeks. The addition of Stimulan with antibiotics was based on clinical complexity of the revision operation and medical comorbidities of the patients.

Three patients had undergone multiple previous operations and had recurrence of infection. The fourth patient (VW) – had gross infection of hip through haematogenous spread from a dialysis canula site. In this study, one patient required Daptomycin locally, and it was possible to deliver this using the calcium sulphate beads as a vehicle for antibiotic delivery.

Clinical studies involving the use of Stimulan are currently limited. One report of 250 cases[10] described its use in aseptic and infected joint replacement revisions. Nearly half (124 patients) in this series had revision for aseptic loosening. Six patients had ongoing infection. 3.2% patients had persistent wound discharge, and this was directly related to the quantity of beads used in the operation. The volume of beads used in their series was between 5 and 70 cc, while we have used a maximum volume of 20 cc. One patient in our series had persistent discharge, although it is difficult to definitely state if that was related to the Stimulan beads, or to multiple previous operative procedures and local scar tissue. A high bead volume was also related to increased risk of Heterotopic ossification.

Local antibiotic delivery using Stimulan has been used in the treatment of chronic osteomyelitis of the lower extremity. In one study of 354 patients, there was an overall resolution of infection in 93% patients.[11] In 86.4% patients, resolution of infection was achieved with surgical debridement and local antibiotics, without intravenous antibiotic usage.

In an experimental study,[12] osteomyelitis was induced in the tibia of 72 rabbits. 36 of these had moxifloxacin impregnated Stimulan beads locally. Of the remaining 36, 18 were used as controls with no antibiotics and the other 18 had Stimulan only. Moxifloxacin was found to be effective in treating Methicillin resistant Staphylococcus aureus osteomyelitis with lower bacterial load locally throughout the study period.

5. Conclusion

Stimulan is a synthetic, biodegradable calcium sulphate that enables delivery of local antibiotics including those that are not suitable for use in cement because of their heat lability. As it is fully absorbable, local antibiotics can be delivered without the need for an operation to remove the beads. It can be used in the management of bone and joint infections and in this series was used in four cases of complicated prosthetic joint infection.

Contribution of authors

1. Sanjeev Agarwal – data collection, preparation of manuscript.
2. Brendan Healey – preparation of manuscript.

Conflicts of interest

All authors have none to declare.

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Original Article

Using a combination of tranexamic acid and rivaroxaban in total knee replacements reduces transfusion requirements: A prospective cohort study

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ABSTRACT

Introduction: The risk of venous thromboembolism (VTE) is high in orthopaedics. Oral direct factor Xa inhibitors have been introduced to help reduce the incidence of VTE. To reduce post-operative bleeding antifibrinolytics are used.

Aim: We aimed to ascertain the effect of two drugs on post-operative bleeding and transfusion requirements.

Methods: We prospectively recorded patient demographics, operative details, complications, transfusion incidence and VTE incidence in TKR patients. We also sent out a questionnaire to patients asking about wound bleeding and VTE. All patients were given 10 mg rivaroxaban 8 h post-operatively and then once a day for 14 days. Patients given tranexamic acid were given 500 mg IV, 5 min prior to wound closure at the discretion of the surgeon. VTE was confirmed by Doppler or CTPA as Deep Vein Thrombus or Pulmonary Embolism. Minor bleed was categorised as dressing soakage or reported wound leakage, major bleed as haematoma requiring revision within 30 days.

Results: 509 patients underwent TKR: 200 (39%) only received rivaroxaban (Group 1), 296 (58%) also received tranexamic acid (Group 2). 13 (3%) of patients had no data available. 5 patients had a VTE: 4 (2%) Group 1, 1 (0.3%) Group 2 ($P < 0.05$). 39 patients had a minor bleed: 17 (8.5%) Group 1, 22 (7.4%) Group 2 ($P = 0.5$). 2 patients had major bleeds: 1 (0.5%) Group 1, 1 (0.33%) Group 2 ($P = 0.69$). Blood transfusions 21 (10.5%) Group 1, 9 (3%) Group 2 ($P < 0.0001$).

Conclusions: We have demonstrated a reduced requirement for blood transfusions in the tranexamic acid group. However our results whilst they show a trend towards decrease bleeding rates in both the minor and major bleeds are not significant, requiring larger studies looking at wound bleeding and leakage.

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1. Introduction

It is estimated that the risk of venous thromboembolism (VTE) following inpatient orthopaedic surgery is around 10–40%.[1] In the mid 1990s and early 21st century, low molecular weight heparins (LMWHs) such as enoxaparin, and later fondaparinux (an indirect Xa Inhibitor) were introduced. These had to be given subcutaneously and were associated with significant bleeding.[2] More recent advances in VTE prophylaxis have seen the introduction of oral agents. Rivaroxaban, a synthetic direct Factor Xa inhibitor in orthopaedic surgery, has been approved by NICE for the prevention of VTE in adult patients undergoing elective hip or knee surgery.[3–5] Rivaroxaban has been shown in four large studies conducted by RECORD (The Regulation of Coagulation in Orthopaedic Surgery)[6–9] to prevent Deep Vein Thrombosis and Pulmonary Embolism when compared to enoxaparin after total knee replacement (TKR). It has a rapid onset of action, a short half life of 7–11 h and very few drug interactions.[10] It has shown to be a cost-effective alternative to LMWHs.[11] The RECORD data and a further meta-analysis by Yong et al[12] presented results that showed no significant difference in bleeding rates between rivaroxaban and enoxaparin. Recently studies have raised concern that the rate of haemotama formation and wound healing. These studies have focused on the rate of wound complications, infections and return to theatre in patients undergoing TKR and Total Hip Replacements.[13]

Total knee arthroplasty has also been associated with major blood loss[14] which produces a longer length of stay, increased post-operative infections and increased mortality.[15] Pharmacological measures intended to minimise blood loss include the use of tranexamic acid. This drug acts as an antifibrinolytic agent that competitively inhibits the activation of plasminogen to plasmin preventing fibrin degradation. It has been shown to be as effective as topical fibrin sprays at reducing intra-operative and post-operative blood loss[16] without increasing the incidence of venous thrombosis.[17,18]

Following recent articles raising concerns[13] about the effects of rivaroxaban on complications, infections and returns to theatre this study aims to report on the effects of using rivaroxaban combined with tranexamic acid on the incidence of wound complications, infections and return to theatre rates in patients undergoing TKR.

2. Method

2.1. Data collection

A retrospective audit was done on prospective data on all patients who had undergone a TKR in the period 6th January 2009–1st October 2010 at the Victoria Hospital, Kirkcaldy in Fife. The following exclusion criteria were used:

- patients who had undergone bilateral operations
- a patients already taking warfarin (these patients were given dalteparin instead of rivaroxaban after TKR)
- a patients who were prescribed an anticoagulation agent other than rivaroxaban
- a patients where there was insufficient data collection

Each patient was seen at a pre-assessment clinic 3 weeks prior to surgery. They were assigned an ID number and data was collected including age; sex; height; weight; BMI; current and previous medical conditions. All patients were risk-assessed for VTE according to local protocol. VTE risk assessments were done based on a series of risk factors for VTE including BMI, family history, medical history and type of operation. The patient was prescribed rivaroxaban for 14 days if they were low risk or 35 days if they were high risk as per the recommendations for consideration of prolonged prophylaxis in orthopaedic patients.[19] All data was recorded on a local database (Fig. 2).

Patients were admitted the day before or on the day of surgery. All TKR procedures were carried out by a standard medial parapatellar approach and used a tourniquet which was deflated at the end of the procedure. Drains were not used in any of our cases. Operative details such as surgeon, type of anaesthetic, ASA grade, lateral release rate and length of surgery were recorded. The first dose of rivaroxaban was to be administered 6–10 h after wound closure. It was then at the discretion of the surgeon whether tranexamic acid was used, however the protocol used was that 500 mg of tranexamic acid was administered intravenously by the anaesthetist 5 min prior to commencement of wound closure.

Post-operative details such as length of stay; post-operative haemoglobin levels; blood transfusion details; tranexamic acid administration; complications (wound infection, DVT/PE, haematoma and any subsequent revisions within 30 days were recorded.

Sources of information included a prospective database backed up by surgical and ward notes. The outpatient VTE clinic was also contacted for a list of patients that were referred for a suspected DVT or PE. See Fig. 1 for a breakdown of patients included in the study.

Finally questionnaires were sent out to all patients following discharge to ask whether they had taken rivaroxaban for the required length of time; whether they had experienced any wound complication (infection, bleeding or bruising), or if they had a swollen leg that was investigated by ultrasound (Table 3).

Any documented "wound soakage" that resulted in the patient having rivaroxaban discontinued was considered to be a *minor bleed*. Furthermore if a patient answered yes and commented on bleeding to the question "did you have any problems with your wound in the hospital or at home?" They were considered to have had a *minor bleed*. Any documented haematoma that required a return to theatre within 30 days was considered a *major bleed*.

Our unit has a blood transfusion policy. Patients with a haemoglobin <8.5 g% were transfused. Patients who were symptomatic with a haemoglobin of between 8.5 g% and 10 g% were also transfused.

3. Results

Six-hundred and two patients underwent a TKR at our centre in the period 6th January 2009–1st October 2010. Ninety-three patients were excluded from the study using the exclusion criteria detailed in the method section; sixty-one were

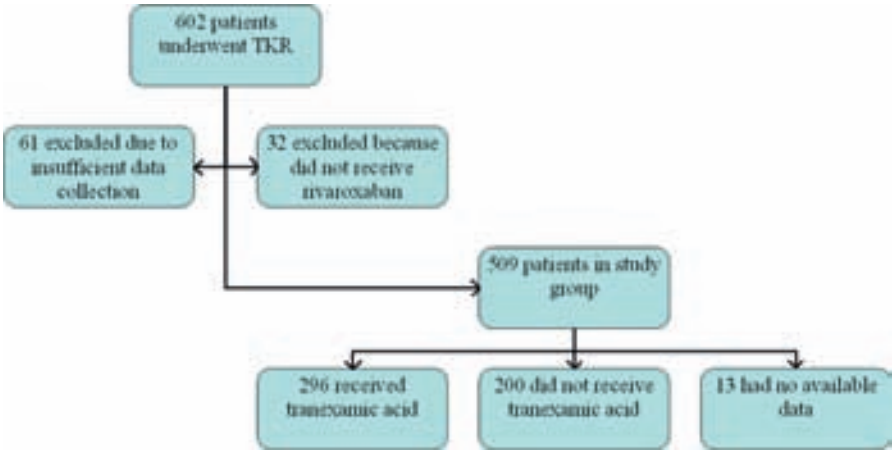

Fig. 1 – Breakdown of patients receiving rivaroxaban and tranexamic acid.

Venous Thrombo-Embolism Risk Assessment Elective patients
To be completed on all adult patients

PATIENT NAME.....

DATE.....UNIT NUMBER.....

Patient details	
Obese BMI>35	[2]
Immobile prior to admission (bed rest >24 hours, long haul flight >8hrs within last 6 weeks	[2]
Current medication	
Oral contraceptive pill	[2]
Hormone Replacement Therapy	[2]
Family History	
Family history of deep vein thrombosis (DVT) or Pulmonary Embolus (PE) 1st degree relative	[3]
Medical History	
Cancer/ chemotherapy	[3]
Previous history of DVT	[3]
Previous history of PE	[3]
Pregnant or within 6 weeks of child birth	[3]
Diagnosis	
knee replacement	[3]
Hip Replacement	[4]
Anticipated Bed Rest >48 hours	[3]
Foot and ankle Surgery	[1]
Anterior Cruciate Injury (ACL)	[1]
Knee arthroscopy	[0]
TOTAL SCORE	

Signature of Patient.....

Signature of clinician.....

Fig. 2 – Local VTE Risk Assessment form.

Table 1 – Overall VTE, minor bleed and major bleed rates and blood transfusion rates for the time period January 2009–October 2010 ($n = 509$).

Clinical VTE incidence (number)	Minor bleed rate incidence (number)	Major bleed incidence (number)	Blood transfusion incidence (number)
5/509 (1.0%)	39/509 (7.7%)	2/509 (0.39%)	30/509 (5.9%)

excluded due to insufficient data collection and 32 excluded because they didn't receive rivaroxaban. Our study group therefore included 509 patients. Two hundred and ninety-six patients received tranexamic acid (58%), 200 did not receive it (39%) and there was no data available for a further 13 patients (3%). See Fig. 1 for breakdown.

The response to the questionnaires was encouraging with 375 completed returns. Thirty-five patients reported experiencing a swollen leg and of these 23 attended for Doppler ultrasonography, two of which were positive for a DVT. Our records indicated that there were three pulmonary emboli. Therefore, in total, there were 5 recorded VTEs.

Clinical notes following surgery showed 40 patients had rivaroxaban withheld or discontinued: 15 of which were due to documented wound soakage. In addition 27 patients reported a bleed on the questionnaire; three of these bleeds reported were already accounted for from the clinical notes taken. Therefore there were 24 new cases of bleeding reported that produced an overall minor bleeding number of 39 out of 509.

There were 2 patients who had a documented haematoma that required a return to theatre within 30 days (one patient required a full revision 7 days later and the other required a washout 16 days later). There were a total of 30 (5.9%) blood transfusions.

The tranexamic group had a significantly lower transfusion rate of 3% compared to 10.5% in the non tranexamic group. Whilst there was a statistically significant difference in the VTE rate, this may not be clinically significant. There was no significant difference in the minor and major bleeding rate between the two groups.

Table 1 shows VTE rates, minor and major bleeding rates and transfusion rates for the overall period January 2009 to October 2010 and Table 2 shows the comparison between tranexamic use and non tranexamic acid use for the same period.

4. Discussion

Routine chemothromboprophylaxis is recommended by NICE[3] and SIGN[20] guidelines for all patients who have lower limb arthroplasties in the UK. Despite this, chemothromboprophylaxis is shrouded in controversies, with reported complications including an increase in post-operative bleeding,[12] upper GI bleeds,[21] thrombocytopaenia[22] and necrotizing skin lesions. In addition there are reports that non pharmaceutical interventions like foot pumps can reduce the VTE rate to similar levels as chemothromboprophylaxis like LMWH.[23] These controversies existed before rivaroxaban was in general use, and recent retrospective research by Jenssen et al[13] has called into question the surgical complication rates experienced when using rivaroxaban.

In the Record trials the minor bleeding rate was 4.3% in RECORD 3. This was a lot lower than our overall minor bleeding rate of 7.7%, which may be explained by a difference in defining "minor bleeding". In RECORD 3 they defined non-major bleeding as "*including hemorrhagic wound complications (excessive wound haematoma or bleeding at surgical site)*". We had a lower threshold for defining minor bleeding, as we believe that our thresholds more accurately reflect the current opinion about what is minor bleeding from an arthroplasty wound, and it is recognised that prolonged wound leakage can lead to a higher infection rate, reoperation rate and prolonged hospital stays.[24] This increase in minor bleeding should be investigated further to ascertain whether there is a higher incidence of late periprosthetic infections in patients with a reported minor bleed. No joints in our study required revising after a minor bleed, and there was no recorded infection requiring a return to theatre in our study period. We believe that there was no significant difference between the minor bleeding rates in each group as a result of this being a binary denominator between no bleeding and the presence of bleeding. It would be expected that a certain proportion of wounds would bleed and it difficult to define how much blood is lost in minor bleeds, other than using transfusion as a marker, which was significantly different between the two groups.

We reported a low major bleeding rate of 0.39%, in comparison to RECORD 3 with a "major bleeding" rate of 0.6% and RECORD 4 a rate of 0.7%. In these studies, Galat et al RECORD 3 and RECORD 4 defined "major bleeding" as "*bleeding that was fatal, that involved a critical organ, or that required reoperation or clinically overt bleeding outside the surgical site what was associated with a decrease in haemoglobin level of 2 g/dL or more or requiring infusion of 2 or more units of blood*", which was different to our definition. Our results were also similar to Galat et al[25] in regards to their return to theatre rate within 30 days following post TKR haemotoma at 0.24%. Our results differ from those reported by Jensen et al[13] who demonstrated in a similar retrospective cohort study that the use of rivaroxaban compared to tinzaparin produced a return to theatre rate of 2.4% following TKR. They concluded that rivaroxaban needed to be studied further to assess its return to theatre rate. Our

Table 2 – Comparison of VTE, minor bleed and major bleed rates and blood transfusion with tranexamic acid and without.

Study Group (number of patients)	Clinical VTE incidence (number)	Minor bleed rate incidence (number)	Major bleed incidence (number)	Blood transfusion incidence (number)
Tranexamic acid (296)	1/296 (0.3%)	22/296 (7.4%)	1/296 (0.33%)	9/296 (3.0%)
Non tranexamic acid (200)	4/200 (2.0%)	17/200 (8.5%)	1/200 (0.50%)	21/200 (10.5%)
P-value (Chi-square test)	$P = 0.0411$	$P = 0.5102$	$P = 0.6924$	$P < 0.0001$

Table 3 – Rivaroxaban compliance questionnaire.

Patient details			
HIP/KNEE			
ORTHOPAEDIC DEPARTMENT – VICTORIA HOSPITAL		Yes	No
Were you given rivaroxaban (blood thinning tablets) after your operation? (pink tablet)			
How many days were you prescribed rivaroxaban?	-14 days -35 days		
Did you take the tablets everyday completing the full course?			
Did you miss any doses either in the hospital or at home?			
Did you have any problems with your wound in the hospital or at home?			
i.e Bruising, bleeding or infection			
Comment:			
Did you have a swollen leg that was investigated with an ultrasound scan?			
Did you experience any other problems with rivaroxaban?			
Comment:			
Did Rivaroxaban have to be stopped because of any of the above problems?			

study included double the number of TKR patients compared to Jensen et al[13] and produced a lower return to theatre rate when using rivaroxaban, this may reflect a higher threshold of return to theatre rates, or it may be due to the timing of the first dose post-operatively as this has a significant impact on the efficacy of rivaroxaban. The current NICE guidelines[3] recommend that rivaroxaban be prescribed 6–10 h post-operatively.

Our results show that the overall clinical VTE rate for rivaroxaban following TKR (1.0%) was low although it was higher than the rate reported in the RECORD 3 and RECORD 4 trials (both 0.7%). Our study included all patients including high risk patients and our study was too small to determine a clinically significant difference in VTE rate compared to previous trials.

Tranexamic acid has been demonstrated to reduce the intra-operative and post-operative blood loss following TKR.[16] We found that the minor bleeding rate was slightly lower in those who received tranexamic acid (7.4%) and higher in those who did not receive tranexamic acid (8.5%) with an overall minor bleeding rate of 7.7%. However there was not enough evidence to support a cause effect. This is likely to be due to our study size. Furthermore, in a standard TKR most of any blood loss is hidden[26] and therefore will not be picked up by examining patient notes. There were 2 major bleeds, one patient received tranexamic acid and one did not.

Tranexamic acid has also been shown not to increase the risk of VTE.[27] In this study there was one VTE (0.3%) reported in the patients who had received tranexamic acid and 4 documented VTEs in the patients who had not received tranexamic acid (2.0%) this difference whilst statistically significant may not be clinically significant and larger studies are required to explore the significance of this finding.

From our study it cannot be determined with any statistical significance whether the use of tranexamic acid had any effect on minor or major bleeding rates. However encouragingly unlike other studies, we can conclude that the rates of major bleeds remained low at our centre.[13]

There was a lower rate of blood transfusion (3.0%) in those who had received tranexamic acid compared to those who had not received tranexamic acid (10.5%). Large multi-centre studies have reported blood transfusion rates following TKR as high as 39%.[14] From our results it can be concluded that the use of tranexamic acid reduces the number of blood transfusions needed at our centre, which is in line with current research.[27]

The VTE rate in our study was comparable with that reported in the literature despite our study including all high risk patients. The post-operative minor bleeding rate was higher than reported in the literature but there were only two patients who experienced a major bleed. The use of tranexamic acid produced a lower minor bleeding rate however the numbers in this study were not large enough to determine whether this change was of statistical significance. The use of tranexamic acid did, however, produce a statistically significant reduction in the number of blood transfusions required following TKR with rivaroxaban. Our results would support the continued use of rivaroxaban in the routine prophylaxis of VTE as we did not experience a high return to theatre rate that has previously been reported, it would also support the addition of tranexamic Acid to reduce the requirement of post-operative blood transfusions. There is however a need for large randomised trials to be conducted to assess the effects of using a combination of tranexamic acid and rivaroxaban on preventing VTE, reducing wound complications and overall long term outcomes following TKR.

Conflicts of interest

All authors have none to declare.

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Original Article

Results of surface replacement proximal interphalangeal joint arthroplasty

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ABSTRACT

Objectives: To evaluate the clinical results and functional outcome measures of surface replacement proximal interphalangeal joint arthroplasty.

Methods: Proximal interphalangeal joint surface arthroplasties (PIP-SRA) performed by a single surgeon were retrospectively reviewed. Arthroplasties were analysed by radiological and clinical review. Clinical review measured: preoperative and postoperative flexion and extension of the PIPJ; arc of motion; distance to the distal palmar crease (DPC); and Disabilities of the Arm, Shoulder and Hand (DASH) score survey.

Results: Forty-eight PIPJ replacements were performed on 24 women and 9 men from 2001 to 2011. Eight patients had more than one joint replacement. The average patient age was 64 years (range 40–84). The average length of follow up was 18 months (range 2–91). The arc of motion improved on average 26° from 55° preoperatively to 81° postoperatively (range 15–150). The average postoperative DPC was 1.8 cm (range 0–8.0) and the average postoperative DASH score 28 (range 1–67). Eleven of the forty-eight joints hyperextended greater than 0° and of these three joints hyperextended greater than 10°. There were four severe flexion contractures.

Conclusions: Most patients achieved a functional range of motion and the improvement in arc of motion was excellent. Several patients hyperextended and four had severe flexion contractures. There was a low operation rate but a short follow up makes this difficult to interpret for significance.

1. Introduction

Small joint replacement for arthritis has traditionally been considered a difficult problem and designs have tended to lag behind larger joint innovations. The traditional implant treatment for the proximal interphalangeal joint (PIPJ) has been Swanson silicone replacements[1] with some large series supporting good results.[2] Since 1979 PIP joint replacements such as surface replacement have become a viable alternative to arthrodesis for treatment of arthritis.[3] These are

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particularly suited for painful osteoarthritic PIPJ with minimal deformity provided there are intact collateral ligaments and good bone stock.[4] The purpose of this study was to determine the functional outcome of patients after surface PIP joint replacement performed by a single surgeon.

2. Materials and methods

A review of PIP-SRA performed by a single hand surgeon with a specialist interest in PIPJ replacement was performed. Clinical examination including ROM was recorded by the same examiner throughout. Proximal interphalangeal joints were examined preoperatively for range of motion. Postoperatively PIPJ were analysed by: measuring flexion and extension; distance to the distal palmar crease; and Disabilities of the Arm, Shoulder and Hand (DASH) score survey. For measuring PIPJ flexion and extension active ROM was recorded. T testing was used to analyse the pre and postoperative range of motion scores. All implants used were metal on polyethylene PIPJ surface replacement (Small Bone Innovations™ formerly AVANTA). Institutional Review Board approval was obtained from our institution.

2.1. *Surgical technique*

Proximal interphalangeal joint radiographs were templated preoperatively. A dorsal approach was used in all surgeries. The extensor tendon was split centrally and the central slip elevated for later reattachment. The collaterals were partly released if needed to allow adequate exposure. Osteophytes were removed. Bony cuts were made perpendicular to the shaft with an oscillating saw of both phalanges and the proximal condyle volar lip also cut. An awl and then sequential broaches were used to prepare both canals. Implants were trialed for stability. Irrigation and canal preparation was performed and then cement and the implant inserted (Fig. 1). The joint was held in extension to facilitate cement pressurisation. The central slip was re-approximated with a previously placed intraosseous 2/0 Ethibond suture.

The patients were placed in a volar plaster splint for 2 weeks with the wrist in neutral and the MCPJ and PIPJ in slight flexion. Patients were encouraged to keep their hands elevated to minimise swelling. Rehabilitation began at 2 weeks under supervision of an experienced hand therapist. Early active mobilisation was commenced with a static dorsal splint in 20° of flexion worn at rest. At 6 weeks protective splinting was discontinued.

3. Results

Forty-eight PIPJ replacements were performed on 24 women and 9 men from 2001 to 2011. No patients were lost to follow up. Eight patients had multiple joint replacements including one patient who had four joint replacements as shown in Fig. 2. The average patient age was 64 years (range 40–84). The average length of follow up was 18 months (range 2–91). Underlying diagnoses include: 45 patients had osteoarthritis, 1 posttraumatic osteoarthritis, 1 psoriatic arthritis and 1 rheumatoid arthritis. Forty-seven implants were cemented; one uncemented press fit implant was inserted. Preoperatively the average extension was 15° (range −30 to 80°) and flexion was 69° (20–110°), for an average total arc of motion of 55°. Postoperatively the average extension was 4° (range −15 to +45°), and the average flexion was 85° (range 2–120°), for an average arc of motion of 81° (range 15–150°). The arc of motion improved significantly with surgery, increasing from 54 to 81° ($P = 0.0001$). The average flexion to the distal palmar crease was 1.8 cm (Range 0–8 cm). The average postoperative DASH score was 28 (range 1–67). Four patients developed a significant flexion contracture.

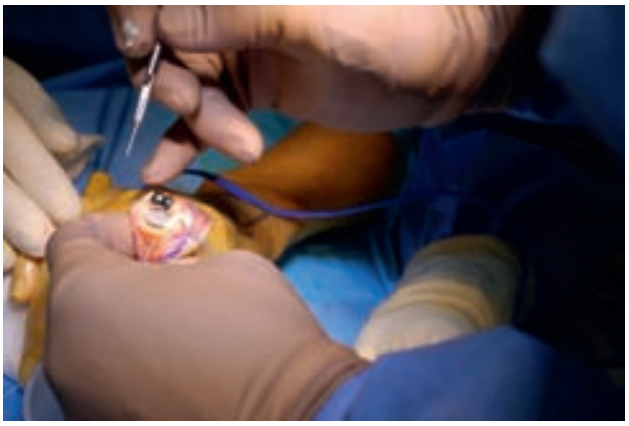

Fig. 1 – Definitive PIP-SRA implant inserted following cement.

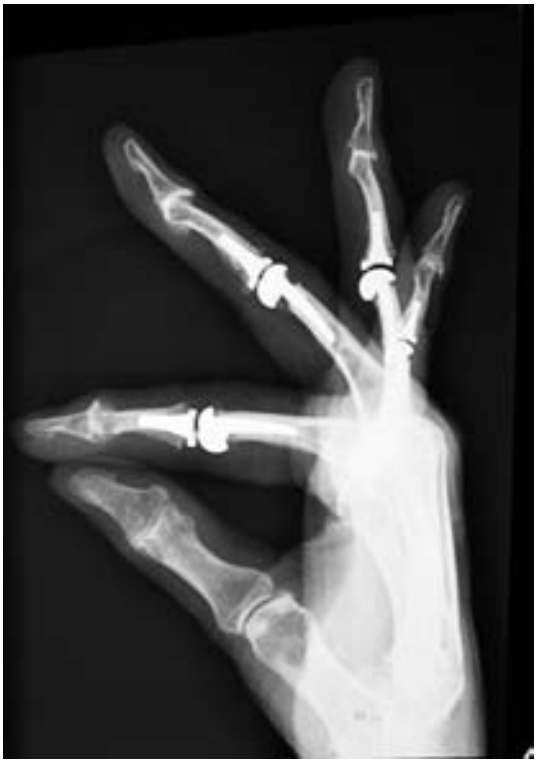

Fig. 2 – Lateral X-ray of patient with two year follow up and four PIP-SRA replacements in situ.

Eleven of the 48 PIP-SRA extended greater than 0°; three of these hyperextending greater than 10°. All three patients were offered a blocking split to prevent hyperextension. Whilst with time the hyperextension did not progress, all three patients continued to report difficulty with initiation of flexion. All felt they could work and function without impairment.

One patient with underlying rheumatoid arthritis developed severe postoperative stiffness in the two PIPJ replaced as well as local irritation from a stitch. Under a general anaesthetic the stitch was removed and the joints manipulated. Whilst 90° of flexion was achieved on the table, at 6 months follow up the patient could only flex the joints 2° and 5°. Another patient with osteoarthritis achieved poor flexion postoperatively despite aggressive hand therapy. She declined a second operation to try and improve range of motion.

No differences in results were found between digits particularly between index and smaller digits. None of the patients complained of significant pain and all thirty three reported that they would have the surgery again and all patients.

There was no evidence of implant subsidence, radiological loosening, or coronal plane angulation on final check radiographs. There were also no incidents of deep infection. No revision procedures were performed.

4. Discussion

Ideal surgical treatment for proximal interphalangeal joint osteoarthritis is an unsolved problem. Surgical options include arthroplasty or fusion. Compared to fusion, replacement of the PIPJ has the advantage of preserving ROM; which is particularly useful to allow ulnar digits to grip. Joint replacement has however the potential to fracture, loosen and dislocate whereas arthrodesis rarely requires further surgery. Proximal interphalangeal (PIP) joint surgery was first described as early as 1914.[5] Initial surgical treatment consisted of arthrodesis or excision arthroplasty.[6] Burman described PIP joint arthroplasty in 1940 as an alternative to arthrodesis.[7] Since then, many modifications and materials have been used with mixed success. Rather than being a simple hinge joint the PIP joint has a variable centre of rotation due to the varying tensile and relaxing mechanism of the collateral ligaments. These mechanics are difficult to reconstruct with arthroplasty and hence PIPJ replacements have not been widely considered to treat PIPJ disease.

The first generation of PIP joint replacements were hinged joints. They failed due to their non-anatomic design, which resulted in higher friction and debris formation, leading to breakage.[8] Implants developed following that copied principles of lower limb implant arthroplasty, but again design flaws and failure prevented widespread acceptance.[8–11] Silicone implants have widely been the most widely used for PIP OA for many years despite variable results[12] and the fact that they act largely as a spacer. Highlighted problems with silicone include implant fracture, longevity, synovitis and poor range of motion.[13] Jennings and Livingston (2008) proposed Swanson silicone implants as the gold standard for PIP OA[1] because an ideal PIPJ replacement did not exist.

Linscheid et al (1979) developed the PIP joint surface replacement as used in this study.[3] These have the advantage of being a "non-hinged" prosthesis with a more anatomical design requiring less bone resection.[9] They reproduce normal joint kinematics and by preserving collaterals allow greater stability and ROM compared to silicon replacements. They are most suited for patients with osteoarthritis or posttraumatic arthritis who tend to have limited deformity, good bone stock and soft preserved tissues. Whilst they are becoming more accepted the largest published studies on the PIP-SRA are still by Linscheid the implant's designer.[14,15]

Post surgery the PIP-SRA analysed achieved an excellent arc of motion of 81°. This arc is very functional and superior to the 30–45° achieved with Swanson PIP replacements in several studies[16,17] however these study groups included a high proportion of patients with inflammatory arthritis rather than patients with OA. Few PIP-SRA studies report the amount of improvement (27°) in ROM as achieved in our study[1,4,15] although this is an average and some patients did better than others.

No implants required revision in our study. This result must be interpreted with the fact that PIP-SRA is a developing rather than long standing treatment for PIP OA and this is a short follow up study. Murray et al (2012)[15] showed the longer the time from surgery the greater likelihood of the need for PIP-SRA revision: there was a failure rate of only 3% at 1 year but 16% at fifteen through 25 years. Hence one would expect that a later review of our group at 5–15 years would show several had required revision. Our results do compare favourably with other studies where the revision rate may be as high as 26%.[1] As well as a short follow up we believe our favourable results were most likely secondary to almost all having OA, the use of cement, and the fact that all operations were performed by an experienced hand surgeon with a specialist interest in PIPJ replacement.

We had 11 PIPJ hyperextend and of these 3 extended greater than 10°. At this range of extension patients can become locked in hyperextension and have trouble initiating flexion. Indeed all three joints required another finger to help push the joint out of hyperextension to initiate flexion. Whilst his hyperextension did not progress at review we have since changed our postoperative regime. At the two weeks postoperative check PIPJ extension is now reviewed closely. Any PIPJ with hyperextension are now fitted with a 30° flexed dorsal blocking splint to be worn constantly, whilst active flexion rehabilitation continues. The ROM is reviewed again at six weeks and if the PIPJ hyperextends the splint worn until 3 months postoperatively.

Arthritides such as rheumatoid or inflammatory arthritis should be a relative contraindication to surgery because of the effect on the surrounding soft tissues.[18] We had 2 patients attain poor postoperative flexion. In the first case the patient was warned of a high risk of postoperative stiffness due to his underlying juvenile rheumatoid arthritis, but requested surgery after successful metacarpophalangeal replacements. Despite the poor range of motion the patient was satisfied with the pain relief and doesn't regret having had the surgery. The second patient developed poor flexion due to excessive soft tissue scarring most likely due to her underlying Dupuytren contracture and a tendency to form keloid. She

declined further surgery and was very satisfied with the pain relief. We believe patient selection is important and patients with rheumatoid arthritis or Dupuytren's can still be offered PIP-SRA for pain relief but should be counselled regarding the likelihood of postoperative stiffness.

Of the four PIPJ which developed significant flexion contractures, two are easily explained. One of these patients was an 82-year-old farmer, the second oldest patient in our study. He was happy with the pain relief provided by surgery and declined hand therapy postoperatively. Another patient had severe erosive bone loss, and surgery was performed early on in our experience with the prosthesis. We would now insert a silicon replacement in the future if confronted with similarly poor bone stock. We think these two flexion contractures were contributed by old age, a lack of hand therapy, and by significant bone loss but it remains unclear why the other two joints developed contractures.

Whilst frequently used to measure outcomes of hand surgery, we believe the DASH score used is problematic. The DASH score average of 27 from our study was similar to the 24 achieved by Luther, but higher and worse, than the average of 14 achieved by Stoklein.[4,19] However most of our patients were very happy with the results of our surgery. Many had a raised DASH score due to problems such as rotator cuff disease and osteoarthritis affecting other joints rather than due to their PIPJ replacement. If preoperative DASH scores had been taken this might help control for the effect of associated limb pathology. Sweets and Stern used the Michigan Hand Outcomes Questionnaire and compared involved to non-involved hands to evaluate a PIPJ replacement.[20] We believe that a more hand-based scoring system such as this would be more accurate assessment tool and would have demonstrated a greater outcome in our patients postoperatively.

The choice of whether or not to cement implants is currently not clear. Johnstone showed cemented implants to have less subsidence (4%) than uncemented implants (68%) in their study.[21] Similarly, Jennings concluded that all implant loosenings in their study group were exclusively associated with a lack of cement.[1] Murray (2007) recommends against using cement and suggests in cases of a capacious canal, packing the canal with bone allograft[18]; the equivalent of the "Ling technique" used in revision hip arthroplasty.[22] Significantly this is mentioned as technical advice rather than results from a study. However having only inserted one press fit implant for our youngest patient (40 years of age) we cannot directly compare uncemented with cemented implants. However the capacious canal, alluded to in the paper by Murray (2007) as a rare indication for cementing in PIP-SRA, we encountered in the majority of cases after broaching the proximal phalanx.[18] We feel the proximal canal unsuitable to a press fit technique even with insertion of bone allograft and hence recommend cementing in most cases. In support of cementing we had no instances of subsidence or failure in our study at follow up.

There are several different approaches to the PIPJ including the volar, lateral, dorsal chamay and dorsal used in this study with there be no clear consensus of which is superior.[17] Many authors favour the dorsal approach over a volar approach.[14,23] although the dorsal chamay technique would have the same advantages of easy access and PIPJ exposure whilst potentially preserving the central slip.[24] Like Linscheid, we believe a volar approach poses a risk to the flexor sheath and volar plate.[14] Moreover we disagree with Stoecklein that a volar approach allows preservation of the central slip of the extensor tendon.[20] In our experience bony cuts for the implant in the middle phalanx often include the bone where the central slip attaches. We therefore do not believe we could preserve the central slip after our bony cuts are made if a volar approach or even a dorsal chamay approach was used. We appreciate that there may be some disadvantages of using a dorsal approach. Some of our patients did hyperextend postoperatively despite due surgical care as discussed. Potential causes for this include: the volar plate was injured by the saw or rendered incompetent post surgery; injury to the collaterals at the time of surgery; and failure of the dorsal lip of the implant to resist PIPJ hyperextension. Deformity may also occur secondary to imbalance between skeletal length and extensor mechanism length especially if the central slip was over tensioned in repair. Our patients do require protection of the extensor mechanism whereas patients operated through a volar approach can rehabilitate more freely straight away. We agree with Stoecklein that a prospective randomised trial comparing volar and dorsal approaches in PIPJ implant arthroplasty would help decide which result is superior.[19]

Alternatives to the cobalt chrome implant used in our study exist but with mixed results. Sweets and Stern reported significant complications with pyrolytic carbon implants including: dislocation, squeaking, loosening and migration.[20] Field also found an unacceptably high revision rate with ceramic coated cobalt chrome.[23] Ceramic implants have been used, though with the potential for a high rate of loosening (10%) requiring reoperation.[25] Whilst concerns have been raised with all materials, clearly further research is required before confirming which is the ideal material for PIPJ surface replacement though the cobalt chrome and polyethylene used in our study appears to be one of the most widely used.

The major limitation in this study is the short 18 month average follow up. In contrast the mean follow up for Linscheid's group was 4.5 years in his first study and 8.8 years in his most recent paper.[14,15] Thus the significance of some of our results may need to be borne out with time. With longer follow up one might expect particularly implant subsidence requiring revision and a higher failure rate in the index finger digit compared to the smaller digits. Given the lack of PIP-SRA studies and certainly of large studies we believe our results are useful. Our study group whilst relatively homogenous includes three patients without OA. Whilst it could be argued that our results might be more meaningful with a group made up only of OA patients only we wanted to report a complete surgeon's series and hence included these patients. Proximal interphalangeal joint surface replacement is still in its infancy[1] however from our very early results it appears an excellent option for hand surgeons to offer patients with PIPJ OA.

Conflicts of interest

All authors have none to declare.

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Original Article

Tightrope-suture button fixation for type III tibial eminence fractures – Case series and review of literature

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ABSTRACT

Purpose: Arthroscopy is the preferred method for anterior cruciate ligament avulsion. Successful fixation methods have been described recently. Here we are introducing a series of 24 patients treated using ACL tightrope and suture disc. This study was performed to evaluate the functional outcome of a consecutive group of patients who underwent reduction and fixation of ACL avulsion fractures fixed with tightrope and suture disc.

Methods: All 24 patients were evaluated using anterior drawer, Lachman test, Tegner activity scale and Lysholm knee scores. The mean age was 29 years (range 17–52). All 24 patients had Meyers and Mckeever type III fracture pattern. The mean follow-up was 41.4 weeks (range 28–57 weeks).

Results: The results of the anterior drawer, Lachman, and pivot-shift tests were negative. The mean Lysholm score improved to 96.

Conclusions: Arthroscopic stabilization by use of tightrope was possible in all cases using this fixation method.

Level of evidence: Level IV, therapeutic case series.

1. Introduction

ACL avulsion injuries from its tibial attachment, also known as tibial spine fractures or intercondylar eminence fractures have been reported in the last 100 years. They represent a variant of anterior cruciate ligament injury. Pringle in[3] 1907 first reported avulsion of the anterior tibial spine in children and it was only in 1959 that Meyers and McKeever[1,2] described an account of surgical management of type II injuries of tibial spine.

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In this injury, the integrity of the anterior cruciate ligament – tibial spine complex which is so essential for proper knee kinematics is compromised, leading to potential complications such as instability, non-union, malunion, arthrofibrosis, growth disturbance in children and residual laxity if left untreated.[4–6] Consequently, accurate diagnosis and prompt treatment are important to restore stability and function to the knee.

These injuries once thought to be common in children aged between 8 and 13 years are also commonly seen in adults due to high energy trauma like road traffic accidents.[1,2,7,8] Reduction and fixation of the tibial spine fractures along with treatment for associated injuries is essential for a good outcome. Treatment of these injuries has evolved from closed treatment to operative treatment and arthroscopy has become the gold standard of treating these injuries as it involves less soft tissue dissection, less pain, quicker recovery and in many cases there is no need for implant removal.[9,10]

Many arthroscopic techniques have been described including screw fixation, pull-out sutures, staples and suture anchor fixations.[4,6,10,12–22]

In this study, we aim to publish results of our series of patients treated with this simple and effective technique to fix Type III non-comminuted fractures using the Tightrope implant (Arthrex).

2. Materials and methods

During August 2012 to December 2013, 24 patients were treated with Type III tibial eminence fractures with arthroscopic Tightrope – Suture button technique and they were included in this case series study. Diagnosis was based on X-rays and MRI. While patients with concomitant meniscal injuries were included, patients with injuries to other ligaments were excluded.

In this study, 19 males and 5 females with a mean age of 29 (range 17–52) were included. Road traffic accidents involving two wheelers were the major cause of injury[22] followed by falls and twisting injuries. All of them presented with pain, swelling and varying amounts of difficulty in bearing weight. A pop or snap was reported only by 4 patients. Most patients had Grade 1 Lachman, varus stress test positive in 8 patients and inconclusive in other patients. All 24 patients (100%) had Meyer and McKeevers classification Type III fracture. Two patients had minor OA changes radiologically. Associated injuries include medial meniscal tears in 4 patients, lateral meniscal tears in 2 patients, Grade 1 medial collateral ligament injury in 8 patients and chondral damage in 2 patients.

The mean time from injury to surgery was 8 days (range 3–26 days) with most of them visiting us within 10 days of injury (17 out of 24). The mean hospitalisation was 3.9 days (range 3–5 days) and the mean follow-up period was 41.4 weeks (range 28–57 weeks). Examination of the knees under anaesthesia before surgery showed grade II anterior instability in 4 and grade III in 20 which was initially inconclusive at the time of admission.

Spinal anaesthesia was used in all the patients. Patients were positioned supine with a thigh support and tourniquet was used in all cases. Anterolateral and anteromedial portals were made and joint lavage was given to evacuate the haematoma. Thorough inspection was carried and meniscal tears if present were addressed. The avulsed bony fragment was circumferentially exposed (Fig. 1A) and for this some portion of the anterior fat pad had to be excised. Entrapment of the anterior horn of medial meniscus and intermeniscal ligament is quite common in these cases and if present it should be carefully pulled out using a probe. Any comminuted pieces of the fracture were removed. The fracture was reduced (Fig. 1B) with the knee in 45–50° flexion, the reduction can be temporarily held with a K-wire. The K-wire was passed percutaneously from an accessory portal. An anterior cruciate ligament aiming device was passed from anteromedial portal and kept over the avulsed fragment, thereby holding the reduction. At an angle of 55°, a tightrope drill guide (4.0 mm) (Fig. 1C & D) was passed across the fracture and into the joint. Ethibond sutures were introduced into the joint (Fig. 1E), from the tibial tunnel using the eyelet of a beath pin and the sutures were retrieved out through anteromedial portal. The leading sutures of the tightrope implant was loaded into the ethibond

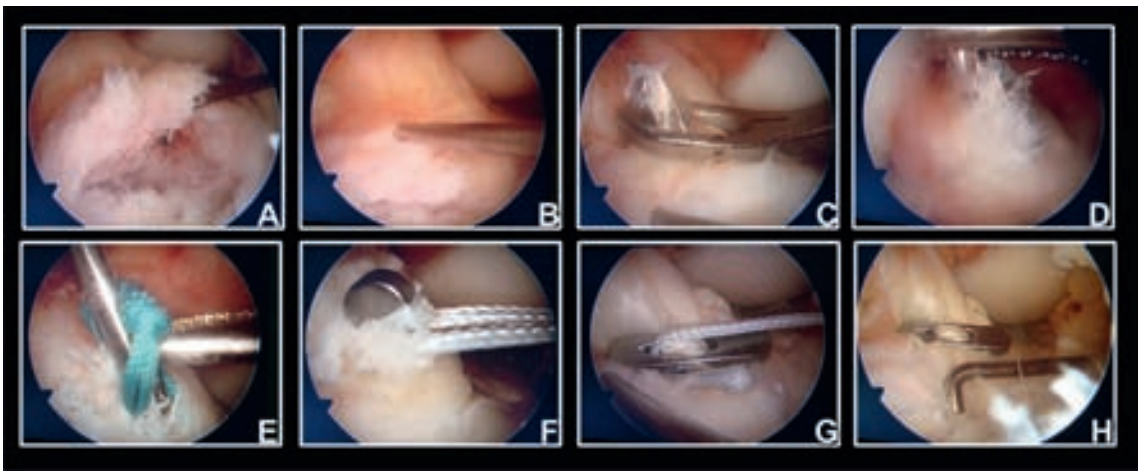

Fig. 1 – Surgical procedure. A – Fracture fragment isolated and prepared. B – Fracture reduced. C & D – Fracture fixed with tightrope drill guide wire. E – Ethibond retrieved into the joint. F – Endobutton retrieved into the joint. G & H – Endobutton flipped over the fracture fragment.

Table 1 – Tegner activity score.

	Pre-op		Post-op		Significance
	Mean	SD	Mean	SD	
Tegner activity score	0.63	0.495	5.04	0.908	0.000 (***) ($P < 0.001$)

Pre-op – Pre-operative; Post-op – Postoperative; SD – Standard deviation.

Table 2 – Lysholm knee scores.

	Pre-op		Post-op		Significance
	Mean	SD	Mean	SD	
Lysholm score	47.63	6.599	96.92	5.672	0.000 (***) ($P < 0.001$)

Pre-op – Pre-operative; Post-op – Postoperative; SD – Standard deviation.

loop from the tibial end and pulled into the joint. Once the titanium button exits the avulsed fragment (Fig. 1F), it is flipped (Fig. 1G & H) and made to sit on the fragment. On the tibial side, the ends of the tightening loop (white sutures) are passed into a suture button and tied over it after a few cycling loadings.

2.1. *Postoperative management*

Knee was initially immobilised with a knee brace and patients were advised non-weight bearing ambulation for a period of 4 weeks followed by partial weight bearing for another 3 weeks. Quadriceps exercises, ankle pumps, 4-way straight leg raises were started from day one. From 3rd week onwards knee brace was removed to perform range of movement exercises. From 2nd month onwards, full weight bearing was allowed and gradual introduced to exercises like bicycling, Stair-Master, leg presses and swimming.

3. Results

All patients underwent periodic clinical and radiological assessments at 4, 8, 12 and 24 weeks postoperatively. Patients were assessed by clinical examination, Tegner activity scale (Table 1) and Lysholm scores (Table 2), by an independent observer. Knee radiographs in standing anteroposterior and lateral views were examined for alignment, joint space narrowing, degenerative knee changes and to assess pre and post-op fracture reduction (Fig. 2a–d). Descriptive and inferential statistical analysis was carried out in this study. Results on continuous measurements are presented on Mean ± SD (Min–Max) and results on categorical measurements are presented in Number (%). For continuous data Paired-T test/Wilcoxon signed rank tests were used. For categorical data Chi-square test was used. The level of significance is considered to be at 5%. SPSS software for windows (version 17) was used.

Of the 24 patients, 23 reported no pain during moderate or strenuous activities; 1 patient reported inconstant and slight pain with moderate or strenuous activities.24 patients followed up regularly, no patients had extensor lag and less than 50 loss of terminal flexion (Fig. 2e & f) when compared to contralateral knee.

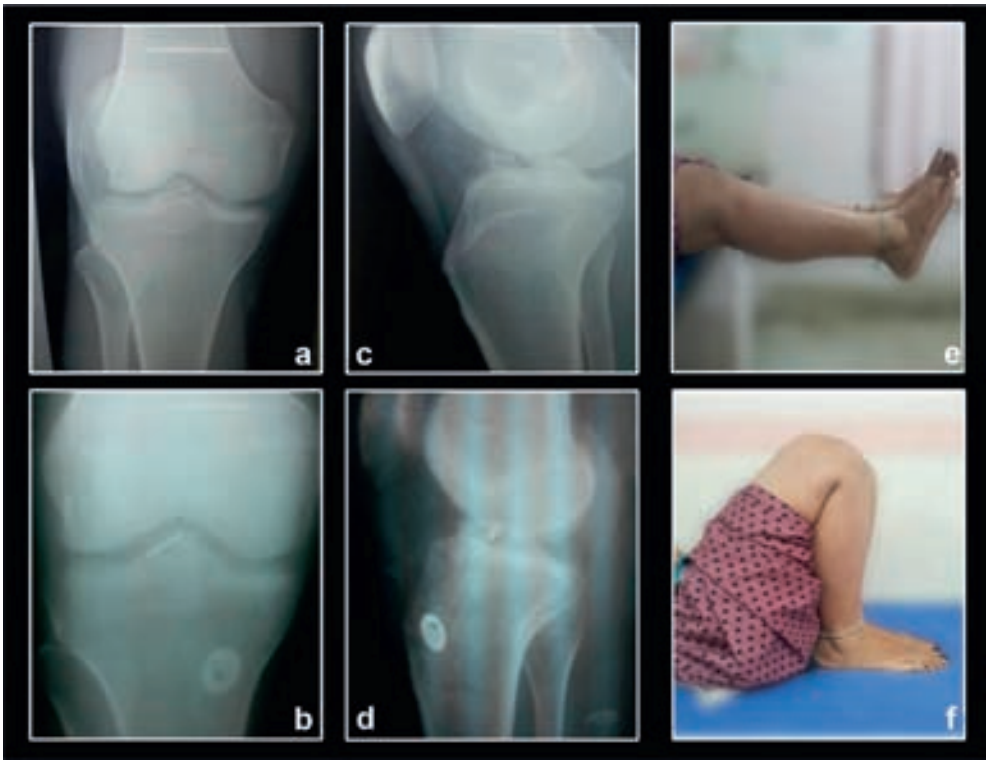

Fig. 2 – a & c – Pre-op AP & Lat view of knee joint with Type III fracture of tibial eminence. b & d – Post-op AP & Lat view showing the reduced fracture fragment with endobutton and suture disc. e & f – Near normal flexion and extension of knee.

Postoperatively, all patients had negative anterior drawer, Lachman tests and pivot-shift phenomena. By 3 months post surgery, radiologic assessments showed solid union in all 24 fractures at final follow-up.

On the functional 1-leg hop test at final follow-up, 22 patients were able to hop 90% of the distance or greater using their healthy limbs, 2 were able to hop 76%–89% of the distance using their healthy limbs.

No complications like deep infection, thrombophlebitis or vascular injury was noted in this series.

4. Discussion

Arthroscopic reduction and internal fixation has become the standard care for ACL avulsion fractures. It also allows complete inspection of the joint in regards to associated injuries and is associated with decreased morbidity, early mobilization, faster rehabilitation, and decreased hospital stay.[9–11]

Buttons and loop based fixation implants are being used in various situations like AC joint separation and syndesmotic injuries.[23,24] In this technique we have used the Tightrope implant and loop with an additional button on the tibial side to fix the avulsion fracture. The availability of the aimer makes it easy to drill the tunnel and flip the button on top of the avulsion fracture. This technique is extremely useful for Type II and Type III fractures. This technique may not be suitable for a comminuted fracture. While many techniques have been described, this one in our opinion is a simple way to fix non-comminuted fractures of tibial eminence. While the patients in this study had good results, surgeons have to be familiarised with the implant construct. Faivre et al has published his report of 8 cases of tibial eminence fractures treated with tightrope device achieved good union.[25] The acknowledged limitations of this study are lack of a control group, small sample size, and a very short observation period.

Conflicts of interest

All authors have none to declare.

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Resident's Corner

Swelling after a knee injury

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ABSTRACT

This article describes the presentation of a patient with knee swelling following injury. It tests and explains the various clinical aspects that are important for a resident to know in assessment, diagnosis and management of this presentation.

1. Case summary

A 25 year old university student presented with a right knee injury following an incident while playing football. He felt severe pain and noticed immediate knee swelling after the injury. He could not return to play for the match on that day. There was no significant past medical history or previous injury to the knee. During the initial assessment in the accident and emergency, there was swelling of the knee and tenderness around the lateral femoral condyle. Passive and active knee movements were restricted by discomfort. Assessment for ligament integrity was difficult due to the existing pain. There was no distal neurovascular deficit. Plain radiograph of the knee was performed (Fig. 1).

2. Questions (answers overleaf)

1. What are the common causes of swelling after a knee injury?
2. What factors in history would suggest that patient had sustained a significant injury?
3. What is your diagnosis from plain radiograph of knee joint?
4. What is the characteristic MRI scan presentation of anterior cruciate ligament (ACL) injury?
5. What are the commonly associated injuries with ACL injury of knee?
6. How would you further manage this patient?

1. What are the common causes of swelling after a knee injury?

Acute knee swelling following injury is due to bleeding in the joint (haemarthrosis) and should be regarded as a serious injury until proven otherwise. The common causes of haemarthrosis of the knee joint includes intra-articular ligament injury (40%) most commonly the anterior cruciate ligament, patella dislocation (25%), meniscus injury (10%) and osteochondral fracture.[1]

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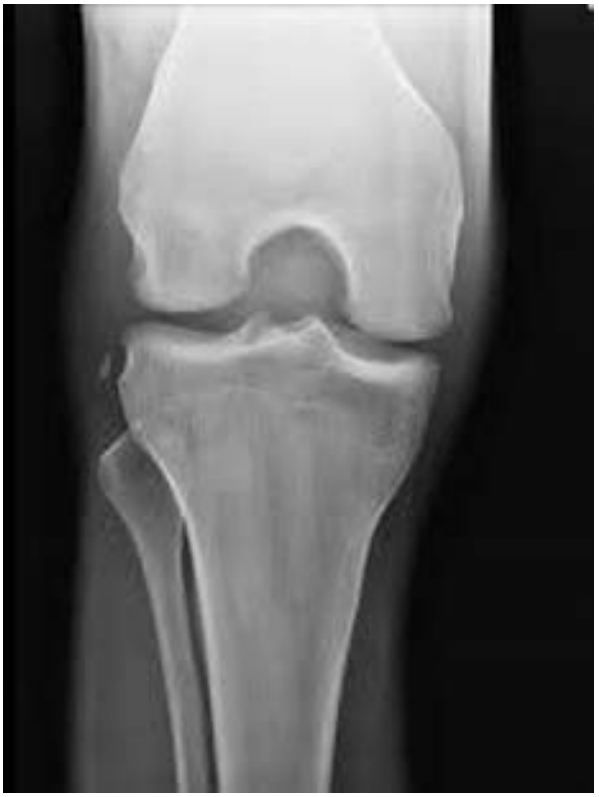

Fig. 1 – AP view of right knee joint.

2. What factors in history would suggest that patient has sustained a significant injury?

Patient with ACL injuries frequently hear a 'pop' or feeling of something tearing in the knee joint. Inability to weight-bear following injury and onset of knee swelling within few minutes of injury, suggest a significant intra-articular injury. Injuries leading to isolated meniscal tears usually cause swelling to develop over few hours.

3. What is your diagnosis from plain radiograph of knee joint?

Antero-posterior radiograph of knee joint for this patient shows a typical fracture, eponymously called Segond fracture. Segond fracture is an avulsion type fracture of lateral tibial condyle at the knee joint. These fractures may occasionally be erroneously ignored as minor avulsion fractures. It is important to identify these fractures as they signify significant capsular or anterolateral ligament injury.[2] Presence of this fracture on plain X-ray is considered pathognomonic of and predicts an associated ACL injury.

4. What is the characteristic MRI scan presentation of ACL injury?

A characteristic bone oedema pattern on MRI scans has been described following acute ACL injuries. Due to rotatory forces on the knee joint at the time of injury, femur subluxes back and impacts on to posterior part of tibia. This movement would only happen if ACL stretches or tears. As the knee relocates, characteristic bruising of lateral femoral condyle and posterolateral tibial plateau is seen on MRI scans (Fig. 2).

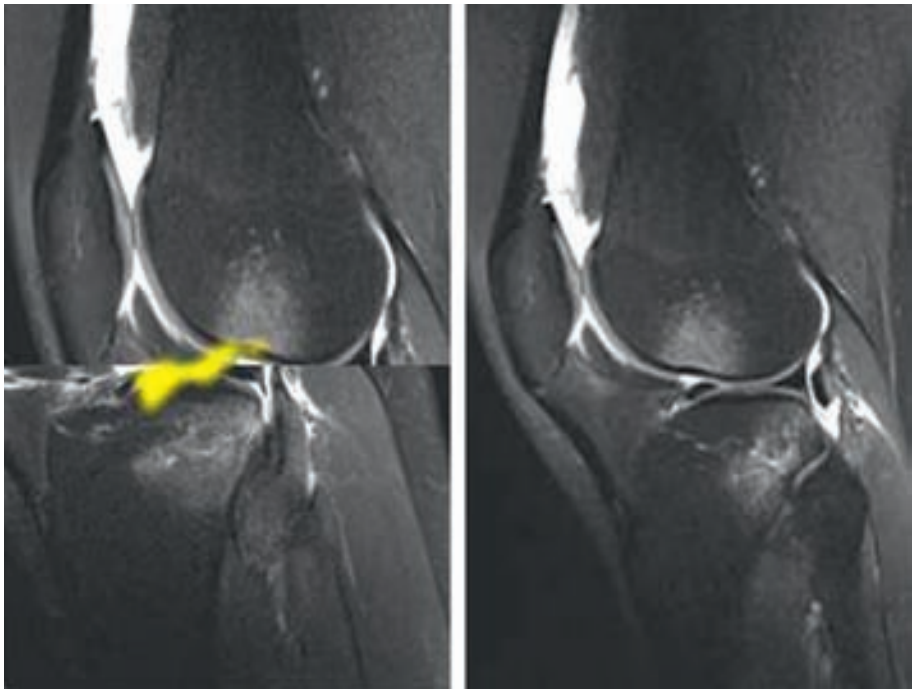

Fig. 2 – MRI scan views of right knee joint showing injury mechanism, with posterior translation of femur leading to ACL tear and characteristic bruising pattern.

In a series of 98 consecutive patients with ACL injuries, Graf et al, suggested 71% of the MRI images showed evidence of bone bruising when obtained within 6 weeks of injury while no bruising was reported on MRI scan taken after 6 weeks of injury.[3]

5. What are the commonly associated injuries with ACL injury of knee?

ACL injuries can be associated with other intra-articular or ligamentous injuries to the knee. Meniscal injuries are commonly associated with ACL injury. Lateral meniscal injuries are more common in acute ACL injuries whilst medial meniscal injuries are more common in chronic ACL deficient knees. O'Donoghue triad is classically described as combined ACL, medial collateral ligament (MCL) and medial meniscus injury. Lateral meniscus injury is more frequently seen than medial meniscus injury in the triad presentation.[4]

ACL injury can be part of a multi-ligament knee injury commonly seen in knee dislocation. Thirty nine percent of ACL tears happen in a multiple ligament injury setting, with commonest association being with MCL injury followed by posterolateral corner injury.[5] It is important to identify and address other ligamentous injuries to improve success rate of ACL reconstructions. Missed posterolateral and posteromedial instability can be a cause of failure of ACL reconstruction.[6]

6. How would you further manage this patient?

Management of ACL injuries is individualised based on the patient and the type of knee injury. Presence of associated injuries may dictate the treatment protocol. It is important to rule out collateral and posterior cruciate ligament injuries in an acutely swollen knee, as their presence in an ACL injured knee may suggest an early intervention. Examination under anaesthesia may help to confirm the injury in an acute setting if MRI scan is inconclusive but there is strong suspicion of associated ligamentous injuries.

For isolated ACL injuries, there is a debate on the role of physiotherapy vis-à-vis surgical treatment.[7,8] Non-operative or surgical treatment pathways should be chosen following consultation with the patient, considering their chosen sports, activity levels and aspirations.

Surgery is usually indicated for symptomatic instability (giving way on pivoting activities), experienced by the patient. Common graft options for ACL reconstruction include hamstring graft, bone-patellar tendon-bone graft and allograft. Graft choice is often based on surgeon's experience though patellar tendon graft may be avoided in patient's involved in kneeling activities. Various meta-analyses have failed to show any significant difference in clinical outcome between patellar tendon and hamstring graft.[9,10] Allografts avoid donor site morbidity but are expensive and can have higher failure rates.[11]

Conflicts of interest

All authors have none to declare.

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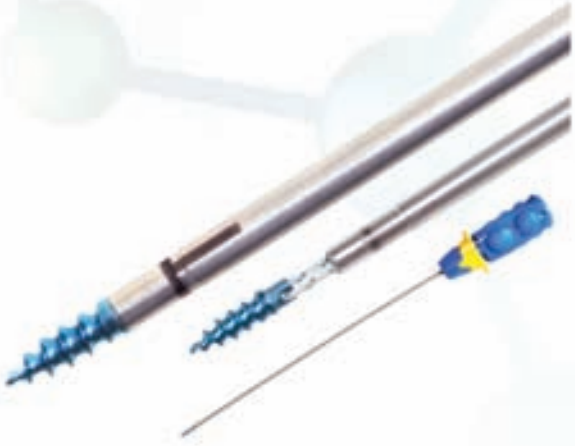

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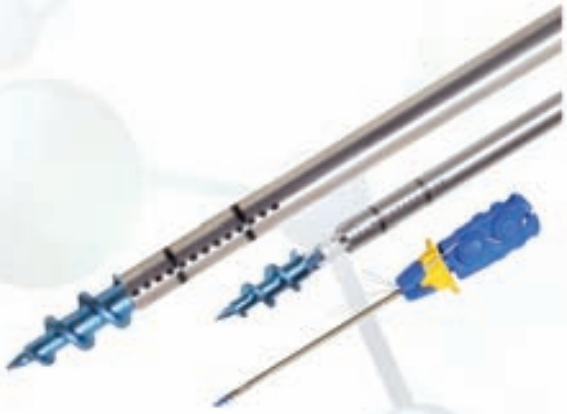

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