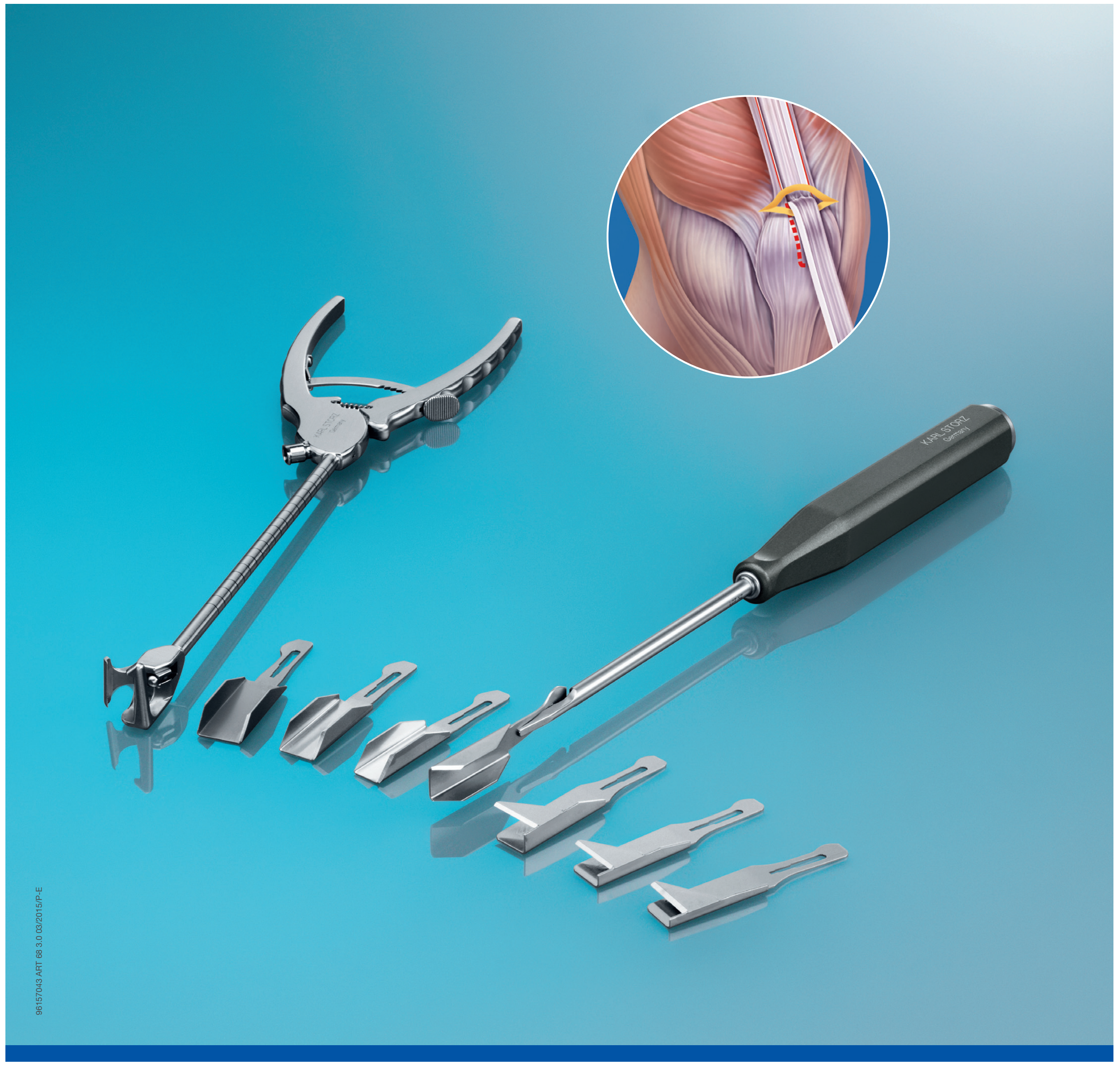

JAJS VOLUME 8 NUMBER 4 OCTOBER–DECEMBER 2021 PAGES 309–386

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Knee & Shoulder Arthroscopy And Arthroplasty • ISKSAA •

JOURNAL OF ARTHROSCOPY AND JOINT SURGERY

JAJS

Official Journal of the International Society for Knowledge for Surgeons on Arthroscopy and Arthroplasty (ISKSAA)

Indexed In Scopus & Embase

Volume 8 Number 4 October-December 2021

E-ISSN: 2214-9635
P-ISSN: 2542-6001

Available online at www.sciencedirect.com

ScienceDirect

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4. The Clinical posts would start in August 2022 although if candidates were to be interested for August 2022 start, they could still apply.
5. The MCh course is at the Edge Hill University and although most of the payment for the course can be made along the way in installments over the 2 years, there would be an initial Commitment of £8,000 to be made to secure the place before the formalities with Royal colleges and GMC are commenced at this End. The salary scales are detailed with the information sheet as well.
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Journal of Arthroscopy and Joint Surgery

An official publication of International Society for Knowledge for Surgeons on Arthroscopy and Arthroplasty

(ISSN: 2214-9635)

Volume 8, Number 4, October–December 2021

Aims and Scope

Journal of Arthroscopy and Joint Surgery (JAJS) 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.

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Journal of Arthroscopy and Joint Surgery

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Published by Elsevier B.V.

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Review article

Arthroscopic management of the stiff knee: A clinical outcome review

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ARTICLE INFO

Article history:
Received 1 October 2021
Accepted 5 October 2021
Available online 9 October 2021

Keywords:
Stiff knee
Arthrofibrosis
Arthroscopic management
Current concepts
Orthopaedics

ABSTRACT

Knee stiffness is not uncommon following knee surgeries. A stiff knee alters the normal gait of the person and is a recognized cause of social humiliation for the patient. Due to its minimal invasiveness, arthroscopic management is gaining popularity among surgeons and patients. Arthroscopy has the potential to treat the majority of the non-bony causes of knee stiffness without much hassle of open surgeries. However, arthroscopic management is not devoid of its limitations and complications. So, awareness of arthroscopic management of knee stiffness and its clinical outcomes is of paramount importance to a practising knee surgeon.

1. Introduction

The knee joint is the largest synovial joint in the body withstanding impressive loads during bipedal locomotion.[1] It is also one of the most stable joints in the human body which can allow a wide range of motion in the sagittal plane from up to -10 to 140° with limited motion in the coronal plane facilitating normal gait. Knee stiffness limits this range of motion impeding the normal function of the knee and impairs the gait and climbing ability of the patient. Since squatting and cross leg sitting are the major parts of the daily chores of the Asian population, a stiff knee causes a significant burden to the patient. Therefore, elaborate knowledge in terms of the treatment of knee stiffness is always indispensable for an Orthopaedic surgeon.

Since the knee is in the load-bearing axis of the lower extremity, its injury is the most common injury accounting for an average of 68% of musculoskeletal injuries happening during sports.[2] It also sustains a significant impact during road traffic accidents. Most of these injuries either with or without surgical intervention are associated with knee stiffness mounting morbidity for the patient.

The global burden of diseases 2010, reported that the age-adjusted standardized prevalence of knee osteoarthritis was 3.8%.[3] An international survey including 18 countries showed that the average rate of primary and revision knee replacement surgeries was 175 and 149 per 1 lac population respectively with a compound annual growth rate ranging 5.3%–17%.[4] Even though the stiff knee is an uncommon complication of total knee arthroplasty (TKA),[5] the number of replacement surgeries being performed every year draws attention to all its complications.

Apart from knee surgeries, various conditions like septic arthritis, inflammatory arthritis, and synovial chondromatosis without early treatment have also landed up with knee stiffness.

For decades, open debridement and removal of adhesions with or without quadricepsplasty have been the standard care of treatment for knee stiffness.[6] But, the discovery of keyhole surgery and its application in treating various joint pathologies has changed the perspective towards the management of knee stiffness.[7] Arthroscopic arthrolysis minimizes surgical morbidity in comparison to open surgery.[8] However, there is a scarcity of literature summarizing the clinical indications and outcomes of arthroscopic stiff knee management. Therefore, the present review aims to highlight and discuss the current application of arthroscopy in the surgical management of knee stiffness.

2. Methodology

Pub Med, Google Scholar, Embase, Web of Science, and Cochrane registry searches were performed using terms “stiff knee” “Knee arthrofibrosis” “Arthroscopic management” “Arthrolysis” “surgical management of stiff knee” and “stiff knee management” from 1960 to January 2020. Only articles relating to stiff knee or arthrofibrosis

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https://doi.org/10.1016/j.jajs.2021.10.002
2214-9635/

management including open or arthroscopic surgeries were selected. The abstracts and the full text of the matched articles were collected and reviewed in detail. References that were cited in the identified articles were also screened for inclusion. A total of 38 articles related to the present study objectives were included and reviewed extensively to consider the therapeutic potential of arthroscopy in the surgical management of stiff knee.

3. Classification of stiff knee

The crux of arthroscopic management of knee stiffness is based on identifying the cause of knee stiffness. Based on aetiology, knee stiffness can be broadly classified as.

1. Knee stiffness due to medical causes
2. Post-operative knee stiffness
3. Post-traumatic knee stiffness

Some of the medical causes of knee stiffness include but are not limited to infective arthritis, synovial chondromatosis, and inflammatory arthritis like rheumatoid arthritis, gouty arthritis, and synovitis due to various reasons. The exaggerated inflammatory cascade is the hallmark of these conditions. Prolonged inflammation along with immobilization finally leads to fibrosis causing knee stiffness.

Post-operative knee stiffness can be either due to the formation of fibrotic adhesions or improper positioning of implants impeding a normal range of motion. Knee stiffness post knee replacement is one of the major causes of postoperative knee stiffness.

Post-traumatic knee stiffness can be broadly classified as intra-articular and extra-articular causes. Intra-articular causes include intraarticular adhesions following knee effusion with or without ligament or meniscal injuries and mal-union or non-union of intraarticular fractures. Extra-articular causes include quadriceps tethering following shaft femur fractures mostly distal 1/3rd fractures and injuries affecting extensor mechanism.[9]

Identifying the cause is critical in selecting the modality of treatment for knee stiffness.

4. Examination

A thorough clinical examination to identify the aetiology of flexion or extension contracture is a must before any treatment. A goniometer can be used to measure the degree of contracture. Preoperative counselling of the patient to discuss the target range of motion required to meet the demands of the patient is mandatory. Identifying any signs of Complex Regional Pain Syndrome (CPRS) in post-traumatic patients is a must since any surgical intervention during the active phase of CRPS is contraindicated. A sequential Bone scan can be used to identify the timing of surgery in knee stiffness patients with CRPS. A successive decrease in uptake in the bone scan can signify the resolving trend of CRPS.[9]

Radiological examination is necessary to identify any bony ankylosis/obstructions due to malunion or implant mal-position. Noting patella height is a must in lateral radiograph since knee stiffness due to Patella Baja requires an open surgical procedure.[9] Radiographs can also identify loosened components in Total Knee Arthroplasty (TKA) causing obstructions in the knee range of motion.

Diagnostic confirmation of arthrofibrosis can also be done by preoperative Magnetic Resonance Imaging (MRI). It also helps in identifying any ligament or meniscal injury that can be addressed during arthroscopy.

5. Indications

Arthroscopic arthrolysis is now a standard technique for post-traumatic knee stiffness.[9] Restriction either in flexion or in extension or both due to fibrotic adhesions can be successfully treated by arthroscopy. Operative intervention done for intraarticular fractures, ligaments or meniscal injuries may lead to intraarticular adhesions, that can be addressed effectively using arthroscopic debridement after a certain duration following healing of ligaments or union of bone.

Amr El Gazzar treated 11 patients with post-traumatic knee stiffness by arthroscopic arthrolysis. Eight among 11 patients were operated on previously for intra-articular and peri-articular fractures and 3 were treated for ligament injuries.[10]

One of the early mentions of arthroscopic arthrolysis for post-operative knee stiffness was by W. Klein et al. and the majority of these patients were previously treated for Anterior Cruciate Ligament (ACL) injuries.[11]

Dhillon et al. have successfully treated quadriceps contracture due to shaft femur fracture using long arthroscopic scissors. Adhesions up to nine inches above the patella were released. They have even claimed to treat bony ankylosis between the patella and femur using arthroscopic assisted debridement with long osteotomes.[12]

Since the arthroscopic procedure is associated with minimal morbidity as compared to open procedure, its role has been extensively studied in treating complications of knee arthroplasty. In 1987, Campbell et al. could treat flexion contracture causing arthrofibrosis post TKA using arthroscopy in eight patients.[13] Klinger et al. used arthroscopy as their diagnostic tool to determine the aetiology for the painful total knee arthroplasty. Arthrofibrosis was the direct cause of painful TKA in 11 out of 27 patients who were successfully treated arthroscopically in the same setting.[14] Most of the authors have considered an aggressive, supervised physiotherapy regimen in the initial three months for the management of post-TKA knee stiffness before attempting arthroscopic arthrolysis.[13]

6. Contraindications

Infection is an absolute contraindication that requires thorough open debridement. Grade III degenerative joint disease, the active phase of CRPS, poor compliance of the patient, axis deviation of $>5^\circ$ are relative contraindications for arthroscopic arthrolysis.[11]

Post-traumatic stiffness due to malunited periarticular or intraarticular fractures require an open procedure to remove the bony blocks impeding joint movements. Attempting arthrolysis and mobilization before a complete union of fractures is not advisable since the uncontrolled mobilization can precipitate re-fractures. Awaiting till consolidation of reconstructed ligaments is reasonable before attempting to treat knee stiffness surgically.

Patella Infera/Baja requires an open surgical procedure to lengthen or reconstruct the patellar tendon or proximalization of the tibial tubercle.[9] Aseptic loosening or dislodgement of TKA components impeding knee motion may require revision surgery.

7. Arthroscopic technique

After a thorough pre-operative evaluation, a passive range of motion (ROM) of the affected joint should be noted under anesthesia and should be compared with the preoperative recorded ROM.

A pneumatic tourniquet when used should be applied over the proximal thigh as high as possible. It should be deflated before attempting mobilization after arthrolysis for unrestricted gliding of

Table 1
Summary and comparison of several studies evaluating the efficacy of arthroscopic arthrolysis in post-TKA knee stiffness.

STUDY	NUMBER OF PATIENTS	INDICATION	TREATMENT	RESULTS
Sprangueet al[7] 1982	1	Arthrofibrosis	Arthroscopic release	No improvement
Del Pizzo et al.[21] 1985	8	Arthrofibrosis	Arthroscopic release	No details
Campbell[13] 1987	8	Arthrofibrosis	Arthroscopic release	7/8 improved
Parisien[22] 1988	1	Arthrofibrosis	Arthroscopic release	Improved
Wasilewski and Frankl[23] 1989	13	Arthrofibrosis and infrapatellar spur	Arthroscopic arthrolysis	7/13 improved
Hirsch and Sallis[24] 1989	1	Impingement	Arthroscopic debridement	improvement
Lawrence and Kann[25] 1992	1	Arthrofibrosis	Arthroscopic arthrolysis	improvement
Jerosch and Schröder[26] 1996	29	Intraarticular fibrous plicae	Arthroscopic debridement	25/29 cases improved
Markel et al.[27] 1996	46	Peripatellar fibrosis	Arthroscopic debridement	27/46 improvement
Williams et al.[28] 1996	10	Limited ROM	Arthroscopic release of PCL	8/10 improved
Court et al.[29] 1999	4	Arthrofibrosis	Arthroscopic arthrolysis	4/4 improved
Henkel et al.[30] 1999	26	Limited ROM	Arthroscopic arthrolysis	23/26 improved
Corces et al.[31] 2000	11	Pain and Limited ROM	Arthroscopic arthrolysis	10/11 improved
Blumberg et al.[32] 2001	33	Pain and Limited ROM	Arthroscopic arthrolysis	31/33 improved
Scranton[33] 2001	10	Pain and Limited ROM	Arthroscopic arthrolysis	9/10 improved
Ternovyi and Zazirnyi[34] 2001	4	Extension Contracture	Arthroscopic arthrolysis	4/4 improved
Djian et al.[35] 2002	6	Limited ROM	Arthroscopic arthrolysis	6/6 improved
Teng et al.[36] 2002	11	Limited ROM	Arthroscopic arthrolysis	11/11 improved
Klinger et al.[14] 2005	12	Intraarticular adhesions	Arthroscopic arthrolysis	9/11 improved
Schwarzkopf et al.[37] 2013	19	Limited ROM	Arthroscopic arthrolysis	19/19 improved
Bodendorfer et al.[38] 2017	18	Limited ROM	Arthroscopic arthrolysis	17/18 improved

quadriceps muscle.

The surgical procedure requires standard arthroscopic instruments, basket clamps, mayo scissors, motorized shaver, and electrocautery probe. After tourniquet inflation, standard arthroscopic portals-anteromedial, anterolateral portals are made. Suprapatellar portals can be made if required. Inspection of the joint is done in a sequential manner starting from the suprapatellar pouch, lateral retinacular gutter, medial retinacular gutter, infrapatellar region to intercondylar notch. Adhesions noted are removed using a motorized shaver. Arthroscope and instruments interchanged between the portals as per convenience.

In cases with previous ACL reconstruction, one should look for hypertrophic tissue at the base of graft known as 'Cyclops lesion' and should be excised.[10] If there is any impingement of the reconstructed graft causing a limited extension, it might require the notch plasty.[10]

Bansal et al.[15] have described a technique where saline-soaked ribbon gauze was packed in layers between patellofemoral articulation using suprapatellar portals. The ribbon gauze piece at the patellofemoral interface lifts the patellar up, stretching the quadriceps and increases the mechanical lever arm of the extensor mechanism. An increase in the range of motion was noted after the removal of the gauze piece from the joint.

Cases with limited knee extension might have posterior adhesions and capsular contractures. To address it, the posteromedial compartment of the knee joint can be accessed by negotiating scope through the intercondylar notch along the axial axis of the medial femoral condyle. Also, it is recommended to make posterior portals at 90° knee flexion to avoid damage to posterior neurovascular structures[16]). Posterior portals are required for arthrolysis if extensive posterior adhesions are noted. The posteromedial portal is made at around 1 cm above the tibiofemoral joint line, 5 mm behind the femoral condyle through the transillumination technique.[17] The posterolateral portal is made similarly by accessing the posterolateral compartment by passing scope through the anteromedial portal along the axial axis of the lateral femoral condyle. The transseptal portal is made by passing a blunt obturator through the posterior septum just behind Posterior Cruciate Ligament (PCL) from the medial to lateral side.[18] The anterior part of the septum can be resected with the help of a shaver to convert the posteromedial and posterolateral compartment into one posterior compartment. It is recommended to pierce the distal part of the septum just behind PCL to avoid damage to vessels passing through the proximal part of the septum.[19] After releasing the posterior capsule, the origin of gastrocnemius muscles can also be released to facilitate further extension if required.

8. Results

Among 11 patients treated by Amr El Gazzar for post-traumatic knee stiffness, 8 patients showed satisfactory results.[10] W. Klein et al. treated 43 of 56 patients with postoperative knee stiffness successfully by arthroscopic arthrolysis. The majority of these patients were previously treated for ACL injuries. The average time between the two surgeries was 22.8 months and arthrolysis was done following the failure of a minimum of six months of physiotherapy.[11]

Several authors have studied the role of arthroscopy in treating post-TKA knee stiffness [Table 1]. For homogenous assessment and analysis, we have enumerated studies with only arthroscopic management of knee stiffness following TKA. Studies that were done before 2000 had shown the efficacy of arthroscopic management in 75% of patients. However, studies after 2000 have shown improvement in around 94% of the patients. This plausible difference in the outcome could be attributed to the advancement in the techniques of arthroscopy in recent years.

Fitzsimmons et al.[20] in their review article have shown the efficacy of arthroscopic arthrolysis up to one year after index surgery. And they had also emphasized that arthroscopic arthrolysis in combination with Mobilization under Anesthesia (MUA) has a superior outcome than the open procedure.

9. Complications

Since the contracted joint has limited space to manoeuvre the instruments, the chance of instruments breakage with inexperienced hands is inevitable. Post-arthrolysis infection is also a common complication. Patellar tendon avulsion, patella fracture can also occur during mobilization after arthrolysis. Due necessary precautions to avoid these complications should be taken during the procedure.

10. Conclusion

Arthroscopic arthrolysis is a better alternative to open arthrolysis for various causes of knee stiffness. It should be attempted after the failure of a minimum of 3 months of extensive physiotherapy post-primary surgery. One can achieve a good functional outcome up to 1-year post-primary surgery if combined with MUA.

Funding

No funding or grants were received or will be received from any commercial party relating to the subject of this article.

Ethical approval

Ethical approval not required as per our Institute Review Board. All procedures performed in this study involving human participants were in accordance with the ethical standard of the institutional and/international research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Author's contribution

Hemant Bansal: Conceptualization, Methodology, Writing-Reviewing and Editing. Vivek Veeresh: Data curation, Writing-Original draft preparation. Hiralal Nag: Supervision, Validation.

Declaration of competing interest

All authors declare that there is no conflict of interest to disclose.

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Journal of Arthroscopy and Joint Surgery 8 (2021) 313–318

Contents lists available at ScienceDirect

Journal of Arthroscopy and Joint Surgery

journal homepage: www.elsevier.com/locate/jajs

The management of osteochondral loss in the skeletally immature knee

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ARTICLE INFO

Article history:
Received 16 February 2021
Received in revised form
13 May 2021
Accepted 25 May 2021
Available online 29 May 2021

Keywords:
Osteochondral fractures
Osteochondritis dissecans
Cartilage loss
Chondral defect
OCD

ABSTRACT

Osteochondral lesions in children and adolescents can be managed by different techniques. There is a paucity of evidence with regards to the optimal management of these particular lesions. Salvage options are mostly inferred from the adult literature, with Autologous Chondrocyte Implantation the most popular technique. The use of fresh allograft has good documented outcomes in this cohort.

1. Introduction

Osteochondral defects of the knee in the skeletally immature encompasses Osteochondritis Dissecans (termed Juvenile OCD) and traumatic osteochondral fractures (OCF). If untreated, both can lead to worsening pain and osteoarthritis. It is not uncommon to encounter neglected cases with loose fragments, or cases where both operative and non-operative treatment has failed. Where defects of the articular surface and subchondral bone are present in the paediatric cohort, techniques utilized in the adult sector often have limited evidence for use. Here we review the evidence for treatment of the paediatric cohort with osteochondral loss within the knee joint.

2. Non-operative management

Conservative management has been successfully applied for small fragment OCFs (<1.5 cm).[1,2] The decision to treat JOCD conservatively is dependent on the patient's skeletal maturity, symptoms, location and stability of the fragment. Small defects (<2 cm^2), and those located at the 'classic' area (posterolateral aspect of medial femoral condyle) are more likely to heal. Cahill proposed that 50% of these lesions heal within 12–18 months as long as the physes remain open and patients are compliant with restricted activity.[3] Other studies have shown healing rate up to 67% by 6–12 months.[4]

There is also no consensus as to the ideal method of non-operative management. Modalities range from limiting weight bearing and activity restriction to cast or brace immobilization.[1] Many children at higher levels of sporting activities struggle with compliance and may require counselling and reinforcement regarding the benefit of conservative management during this stage.

Presentation with mechanical symptoms, joint effusion, large lesions and radiological evidence of subchondral sclerosis at 6 months are all poor prognostic factors.[1,5–7] Patients with these findings and larger, displaced defects will likely suffer deleterious effects if managed conservatively. Studies in animals have shown correlation between the size of the defect and resultant surrounding chondral degeneration. Heuijerjans et al. sought to biomechanically recreate the joint and assess the effect of imparting varying forces and changes with defect sizes. They found that a small increase in defect size lead to an exponential increase in surrounding superficial collagen fibre strain. The authors proposed that filling this defect would increase the surface area able to partake in load bearing, thereby decreasing surrounding cartilage strain.[8] A cadaveric study in 8 knees also showed that in defects ≤

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https://doi.org/10.1016/j.jajs.2021.05.004
2214-9635/

8 mm there was no increased stress concentration around the defect. In this case, the menisci functioned significantly in the redistribution of this load. However, defects ≥10 mm were associated with increased stress concentration along the defect rim and within surrounding cartilage, increasing the risk of progression to osteoarthritis[9]

3. Operative management

For both OCF and JOCD, operative intervention is undertaken in patients who have failed conservative management with continued symptoms or if radiographic evidence of progression to fragment instability exists.[1,5] The aim of surgical treatment is to stabilise the cartilage, maintain joint congruity and repair the osteochondral defects, while causing minimal damage to the physes or iatrogenic injury to the joint, thereby decreasing the risk of osteoarthritis.[3] Interventions are either reparative or restorative.[10] Though multicentre studies are lacking, it is generally agreed that drilling (transarticular or retroarticular) is the best option for symptomatic stable lesions, with good outcomes demonstrated in systematic reviews.[1] Reduction and fixation of unstable but salvageable fragments is also recommended. However, difficulty exists in deciding the best options for unsalvageable lesions. Concomitant conditions such as patellar instability, Anterior cruciate ligament deficiency and limb deformity (leading to malalignment) may also need to be addressed.[11]

3.1. Debridement and fragment excision

Debridement and fragment excision has been suggested for small or irreparable OCDs. Excision of fragments <1–1.5 cm, especially if of poor quality, has been described. This may be performed as a standalone procedure or along with microfracture. Isolated excision, though beneficial in the short term, is not recommended due to long term findings of degenerative changes and poor functional scores, with the worst outcomes if performed in weightbearing areas.[1]

3.2. Osteochondral repositioning and fixation

Fixation of the osteochondral fragment can be performed for OCF or unstable JOCD and involve either in situ fixation or fragment repositioning and fixation if the fragment is viable. It is essential to remove the fibrous tissue from the base of the lesion in JOCD, though bone on the cartilage flap should be preserved.[4] Open and arthroscopic techniques are described and fixation can be performed using bioabsorbable screws and pins or metallic headless or headed compression screws. Fig. 1a and b shows the successful use of bioabsorbable pins for the fixation of an unstable OCD of the lateral femoral condyle. Metallic implants will warrant surgical removal after healing. The benefit of this is the opportunity to review stability and healing of the fragment at the time of surgery.[1] If a viable cartilage flap is present, it can be partially hinged open, its base debrided with microfracture and bone graft inserted if necessary.[10]

A French multicenter study identified 14 skeletally immature patients with OCFs of the lateral condyle and patella that underwent open or arthroscopic surgical repositioning and fixation within 20 days of injury. Over a mean follow up of 30 months, no patient needed revision of fixation.[2] Though patients in this study had surgery acutely and the recommendation is for early treatment of OCFs, case studies have documented successful surgical treatment at 2 months post injury.[12] Increase in the fragment size, cartilage degeneration and subsequent mechanical symptoms can occur if displaced fragments are not treated early.[1]

Repositioning and fixation techniques utilized for JOCDs are similar to that described for OCFs with good success rates of 91–100%. If necessary, autologous bone grafting (from the proximal tibia or iliac crest) can also be utilized.

Fixation of purely chondral lesions in children has been proposed due to greater healing potential.[2] In a retrospective multicenter study of 15 patients with isolated chondral lesions, debridement of the lesion and subchondral drilling was performed, followed by reduction and fixation of the chondral fragment with bioabsorbable screws, tacks and sutures. This was supplemented with fibrin glue in 3 patients. One patient suffered re-injury 8 weeks postoperatively and underwent fragment excision while another had surgery at 1 year to remove unrelated loose bodies. For the latter, arthroscopy showed evidence of healing of the chondral fragment. MRI was performed within a median of 12 months postoperatively and showed cartilage contour restoration in 5 patients and thinning in 2. These patients had evidence of subchondral advancement into the deep cartilage layer. Cartilage thickening occurred in 1 patient while subchondral oedema and cartilage fissuring occurred in another. All patients returned to sports and other activities post-operatively within 6 months. It was suggested that these lesions may in fact have microscopic bone attached which improved healing capability.[13] Of note, in either study, no recommendations were made as to the fragment size that was amenable to fixation. Limited studies of high caliber are available to further assess fixation of isolated chondral lesions.

4. Salvage options

Multiple methods of treatment have been described if repositioning and fixation of the fragment is not possible. Such defects have limited treatment options due to the lack of intact articular cartilage (Fig. 2). Options include bone marrow stimulation techniques (such as microfracture and autologous matrix induced chondrogenesis), autologous osteochondral or chondrocyte implantation and allograft implantation.

4.1. Microfracture

Microfracture is a minimally invasive procedure advocated for small (≤2 cm^2) OCDs not amenable to repair. Subchondral perforations are performed resulting in haematoma and subsequent fibrous clot formation. This contains mesenchymal stem cells (MSC) and growth factors. Fibrocartilage, which is less durable than hyaline cartilage, forms after MSC differentiation. This method cannot be used to address bone defects and is less effective in JOCD due to the presence of abnormal subchondral bone in some instances.[11,14]

Microfracture was used by Lee et al. for 5 patients with irreparable OCFs (average size 1.2 cm^2) secondary to patellar dislocation. Four (average size 3.2 cm^2) had fixation performed. All were postoperatively assessed with the Knee Injury and Osteoarthritis Outcome score and International Knee Documentation Committee outcome measure. Patients receiving microfracture had better outcome stores but results may be attributable to the difference in severity of injury and subsequent lesion size.[15]

Steadman et al. retrospectively reviewed 26 patients (<19 years of age) who had microfracture for full thickness cartilage defects. Twenty-two patients were followed up for a minimum of 2 years. The defect size ranged from 10 to 600 mm^2. One patient had trochlear groove microfracture and required revision 1 year postoperatively. Patient satisfaction was excellent. Age and gender were not prognostic.[14]

Microfracture has however been shown to have poor long term outcomes compared to other restorative techniques. Gudas et al. compared this method with mosaicplasty via a prospective

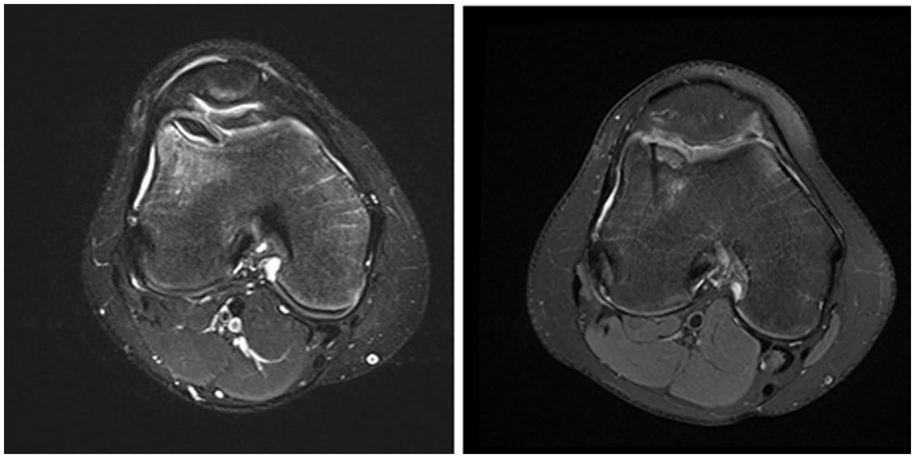

Fig. 1. a: Pre-operative axial MRI image of an unstable OCD, 23 mm diameter, of the lateral femoral condyle. b: Axial MRI image of OCD at 28weeks post fixation with one of four bioabsorbable nails demonstrated.

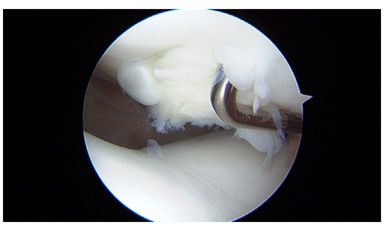

Fig. 2. An arthroscopic image of a patient with long standing unsalvageable patella OCD.

randomized controlled trial in patients less than 18 years and found that at 4.2 years only 63% of patients maintained good to excellent outcomes compared to 83% in the OAT cohort despite comparable results at 1year after surgery. Microfracture was still recommended as there was improvement of the preoperative clinical status.[16]

Due to its shortcomings, 'PLUS microfracture' was developed. This included the use of a membrane (synthetic or periosteum) after microfracture. The principle is to contain and concentrate MSCs at the defect site instead of continued intraarticular expulsion. This procedure has further evolved into autologous matrix induced chondrogenesis (AMIC) which consisted of applying a collagen membrane or matrix after microfracture. Sutures and biological glue are then applied to maintain a stable matrix.[17,18]

4.2. Osteochondral autograft transplantation system (OATS) and mosaicplasty

This is single stage surgery, open or arthroscopic, taking a cylindrical osteochondral graft from a healthy non weight bearing area of the patient's knee and implanting it into the defect. In the mosaic type, multiple small cylinders may be taken from the donor site to fill the defect. Fibrous cartilage is therefore interposed between hyaline cartilage of the osteochondral graft. The success depends on the size and site of defect and matching the radius of curvature on the donor and recipient site. Outcomes are best for defects <2 cm,[2] but it can be used to treat defects up to 4 cm^2 [17]. Valtanen et al. have shown OATS to be useful in lesions up to 3 cm^2 with good to excellent clinical outcomes.[14]

As the graft is from the same patient, there are no risks of disease transmission or graft rejection issues, though donor site morbidity limits the size of the graft harvested. Osteochondral bone plugs have also been used for in situ fixation or splinting of stable fragments.[10] Larger defects can be filled with a 'MEGA-OATS' technique, in which larger grafts can be taken from the dorsal or posterior femoral condyle and inserted via the press-fit method.[17]

Hybrid methods of OATS with stabilisation with bioabsorbable or metal implants have been advocated in adults, but there is no literature evidence available for skeletally immature.[4]

4.3. Osteochondral allograft transplantation

Osteochondral allograft transplantation (OCA) is advantageous in the management of large (>2 cm^2) defects that are unsalvageable. A fresh, fresh-frozen or stored allograft, matched for size and contour is transplanted into the defect after it is prepared. An example of this is shown in Fig. 3a–e.

Ninety-eight percent of chondrocytes remain viable at 7 days in fresh allografts and decreases to 70% after 1 month.[14,19] OCA replaces abnormal subchondral bone with normal bone and allows for early structural stability. With time, creeping substitution occurs and the donor bone is replaced.[20] OCA is expensive and microbiological and immunological investigations must be performed during its preparation to decrease the risk of disease transmission and associated morbidity.[19] Some studies address OCA use specifically for OCD in the paediatric age group. At 10-years post OCA in children with a mean age of 16.4 years treated for cartilage defects secondary to JOCD, avascular necrosis and trauma, graft survivorship was 90% with 89% of patients satisfied with their clinical outcome. OCA is also performed as a salvage procedure after failure of other modalities.[20] Availability and cost, often with logistical difficulties due to the shelf life make use of fresh allograft a challenge.

4.3.1. Autologous chondrocyte implantation

The two-stage procedure, autologous chondrocyte implantation (ACI), was initially described for defects >2 cm^2 and involved an initial biopsy of non-articulating cartilage (such as the superomedial or superolateral trochlear edge and the intercondylar notch) with associated subchondral bone.[21] After in vitro multiplication, the chondrocytes are placed within the defect and covered with periosteum harvested from the tibia. If the depth of the subchondral defect is significant (>6–8 mm), autologous grafting is recommended in conjunction with ACI. The graft may be soaked in autologous bone marrow aspirate obtained from iliac crest prior to insertion. Chondrocyte maturation results in hyaline cartilage formation but with a known risk of graft hypertrophy and

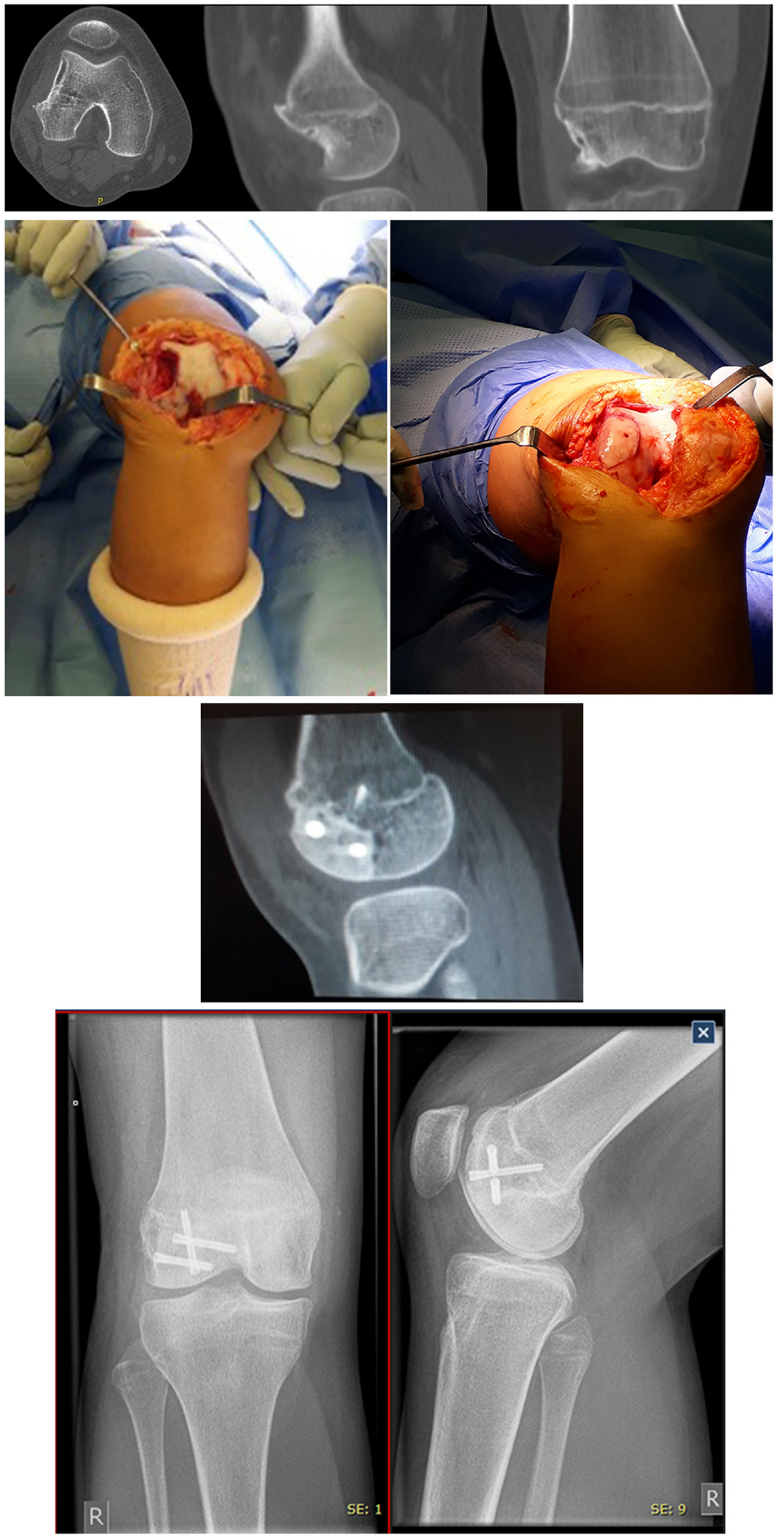

Fig. 3. a. Axial, sagittal and coronal CT images of a large osteochondral defect in the lateral femoral condyle.b. Intra-operative image after the bed of the osteochondral defect has been debrided to bleeding bone.c. The intra-operative appearance after allograft fixation in the same patient. d. A post-operative sagittal CT image of the patient after fixation with metallic variable pitch head-less screws at 6 weeks. e. Plain radiographs 24 months after the initial surgery. There is good bone incorporation of the allograft.

arthrofibrosis.[17,21,22] Long term results were poor when implemented for the treatment of kissing lesions. Hence it is not recommended.[17]

This first-generation procedure has been refined to include artificial membranes instead of periosteum and can be classified as synthetic, proteic and polysaccharid. Hyaluronic acid is commonly used as it aids inhibition of chondrocyte apoptosis and regulates the membrane. Further evolution of the Matrix induced ACI (MACI) involved porcine derived collagen I and III being used as the scaffold for chondrocyte implantation, resulting in less morbidity and even chondrocyte distribution. High-density ACI, in which the chondrocyte density is increased over MACI fivefold, has been described but no comparative studies are available to assess its benefit.[22] The ACI 'sandwich technique' describes the use of 2 collagen membranes. The membranes are placed over the impacted bone graft and secured with fibrin glue and sutures. The chondrocyte matrix is then placed between the 2 membranes. Though recommended in the paediatric age group for large defects, there is a paucity of literature regarding its use.[11,23,24]

MSC can also be implanted into the defect with the aid of scaffolds including platelet-rich fibrin. The cells are usually derived from bone marrow and can be implanted within a single procedure. The added benefit is that these cells are multipotent and therefore regeneration of both subchondral bone and cartilage is possible 25. Further advancement has led to Autograft Cartilage Transfer (ACT) involving the combination of autologous bone marrow aspiration and concentration, autologous cartilage harvested from the non-articular femoral condyle and a scaffold of allograft cartilage extracellular matrix. These procedures eliminate the need for multiple surgeries but do carry an increased risk of donor site morbidity. They are recommended for young patients with unsalvageable lesions. Likely as a result of its novelty, no studies were identified in the English literature to assess their efficacy in the skeletally immature.[26]

Further benefits of tissue engineering are currently being investigated. The expression of chondrogenic genes can be increased by the in vitro addition of specific growth factors and bone morphogenetic protein 2 (BMP2) to MSC. Tensile strength can be improved by increasing collagen concentration and cross linking with glycosaminoglycan-depleting enzymes and cross linking agents respectively. Non-articular chondrocytes are also being engineered into viable cells, thereby expanding the availability of autologous donor sites.[27]

4.4. *Particulate juvenile articular cartilage allograft transplantation (PJAC)*

PJAC is an emerging single stage procedure involving harvesting cartilage from young donors (0–13 years) and mincing them into small 1 mm^3 fragments. These chondrocyte rich particles are then implanted into the defect and covered with fibrin glue. Its benefit is in its ability to be used for chondral defects with multiplanar contours such as within the patella. Studies involving both adults and children have shown good short-term outcomes.[28]

4.5. *Acellular scaffolds*

Recently, the use of acellular scaffolds has been advocated for JOCD, highlighting the benefit of a single stage procedure over ACI with no need for in vitro chondrocyte manipulation and the costs associated with it. A study in 20 children described implantation of a biphasic acellular scaffold consisting of collagen type 1 and hydroxyapatite at varying concentration ratios to mimic the osteochondral unit as best possible. A press-fit technique was ensured and fibrin glue applied. Assessment of regeneration was via MRI performed at multiple intervals post-operatively with 11 patients undergoing all scans. At a mean follow up of 6 years, clinical outcome measures showed improvement from baseline pre-operative levels for all despite non-reassuring findings on final MRI. Though two-thirds had intact articular cartilage, the majority had abnormalities within the subchondral bone.[25,29]

Multiple systematic reviews encompassing the different techniques and their outcomes have been performed. Methods of OCD description varied. Abouassaly et al. in the review of 25 level 4 studies found that both stable and unstable defects were addressed with drilling, bone pegs and bioabsorbable screws while metallic screws and fixation were employed for only unstable lesions. A systematic review identified drilling for stable OCDs and bioabsorbable pin fixation for unstable OCDs as the most common techniques employed. No specific documentation was made of OATS or OCA usage.[30] Two other systematic reviews, each analyzing 11 studies and including only one level 1 study, noted that ACI was the most common treatment method in the paediatric population. OCA had the highest revision rate.[14,31] Across all studies, some general observations were made. Regardless of the modality of treatment, majority of defects healed and resulted in clinical improvement. Limitations were noted in the strength of studies, sample size, documentation of additional surgical procedures performed, assessment of outcomes and duration of follow-up.[14,30,31]

5. Conclusion

Osteochondral loss in the young patient is associated with the development of pain and osteoarthritis. Fixation of loose defects should be the focus of management. For cases where this is not possible, microfracture of the defect can be carried out, although this facilitates symptomatic benefit only in the short term. Other restorative procedures can be considered but evidence in this age group is lacking at present. There are emerging techniques, however these are mostly documented in adult case studies with small sample populations. Furthermore, the majority of larger powered studies are performed in a heterogenous population of adults and children with varying aetiologies for osteochondral loss. Though it is well documented that most defects will heal, further investigation is warranted as to the best methods of treatment in the young, especially for unsalvageable lesions.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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