Heterotopic Ossification After Arthroscopic Procedures

A Scoping Review of the Literature

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Background: Heterotopic ossification (HO) is the formation of bone in soft tissue resultant from inflammatory processes. Lesion formation after arthroscopic procedures is an uncommon but challenging complication. Optimal prophylaxis and management strategies have not been clearly defined.

Purpose: To present a scoping review of the pathophysiology, risk factors, diagnostic modalities, prophylaxis recommendations, and current treatment practices concerning HO after arthroscopic management of orthopaedic injuries.

Study Design: Scoping review; Level of evidence, 4.

Methods: A scoping review via a PubMed search was performed according to the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-analyses) guidelines. The search strategy was based on the terms "heterotopic ossification" AND "arthroscopy." The clinical outcomes review included studies on the arthroscopic management of orthopaedic injuries in which the primary subject matter or a secondary outcome was the development of HO. An analysis of the pathophysiology, diagnostic modalities, and management options was reported.

Results: A total of 43 studies (33,065 patients) reported on HO after hip arthroscopy, while 21 (83 patients) collectively reported on HO after arthroscopic procedures to the shoulder, elbow, knee, or ankle; however, management techniques were not standardized. Identified risk factors for HO included male sex and mixed impingement pathology, while intraoperative capsular management was not suggested as a contributing factor. Diagnosis of ossification foci was performed using radiography and computed tomography. The rate of HO after hip arthroscopy procedures approached 46% without prophylaxis, and administration of nonsteroidal anti-inflammatory drugs (NSAIDs) decreased occurrence rates to 4% but carries associated risks. External beam radiation has not been exclusively studied for use after arthroscopic procedures.

Conclusion: HO is a known complication after arthroscopic management of orthopaedic injuries. NSAID prophylaxis has been demonstrated to be effective after hip arthroscopy procedures. Patients with persistent symptoms and mature lesions may be indicated for surgical excision, although variability is present in patient-reported outcome scores postoperatively.

Keywords: heterotopic ossification; myositis ossificans; arthroscopic surgery; ectopic bone formation

Arthroscopic surgery is generally considered a safe and effective modality in the treatment of musculoskeletal injuries, with an estimated 1.77 million procedures performed annually in the United States.[73] Technical advances and burgeoning knowledge in orthopaedics have expanded the breadth of pathologies addressed with arthroscopic techniques. A known postoperative complication after surgical intervention of orthopaedic injuries is heterotopic ossification (HO).

HO refers to the ossification of soft tissues that results in the formation of histologically normal lamellar bone in an abnormal location.[3,6,42,81,87] Known predisposing factors include direct muscular trauma, arthroplasty, spinal cord and head injury, prolonged sedation, and mechanical ventilation.[11] While HO has been noted to occur after arthroscopic procedures of the shoulder, elbow, and knee, the largest body of orthopaedic literature involves that of the hip joint.[4,7,48,52,74] In fact, the incidence of HO after arthroscopy of the hip has been reported to be as high as 46% in the absence of prophylactic treatment.[6]

We present an updated literature review of current prophylactic measures, diagnostic modalities, and management

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options pertaining to HO after the arthroscopic management of orthopaedic injuries.

METHODS

A systematic literature review following the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-analyses) guidelines was conducted using the PubMed database. A search was performed using the keywords "heterotopic ossification" AND "arthroscopy." A total of 187 studies were identified. Only articles available with full text in English were reviewed. Editorial articles were excluded. In our clinical outcomes review, studies were excluded if HO was not the primary subject matter, a secondary outcome measure, or the sequela of an arthroscopic procedure related to an orthopaedic injury. A total of 64 eligible studies were reviewed, including 43 studies[¶] (33,065 patients) involving the hip and 21 studies[#] (83 patients) examining other anatomic locations (shoulder, elbow, knee, or ankle) (Figure 1).

RESULTS

Pathophysiology

Development of HO is dependent on the presence of several environmental parameters, such as an inflammatory process, the availability of local osteogenic cells, and a rich vascular environment.[42,81] Although widely studied, the exact mechanism by which HO develops has not yet been well established.[3,27,81,88] A suggested model is that the interaction between the injured periosteum and necrotic or damaged muscle may induce periosteal bone production.[2] Additionally, direct trauma to the soft tissue causes the release of bone morphogenic protein 2, which upregulates the release of inflammatory markers; this results in the recruitment of immune cells and the initiation of processes essential for nerve remodeling, fibroproliferation, and angiogenesis, which are necessary for HO formation.[3,86]

Transplant of primitive mesenchymal stem cells capable of osteogenesis into soft tissue adjacent to bone induces the release of factors that create a suitable environment for ectopic bone formation.[6,63,64] An osteogenic cascade ensues, resulting in lesions ranging from clinically insignificant bone foci to large symptomatic formations with associated pain, impingement, or joint stiffness.[3,47] HO has been reported to form at triple the rate of normal bone and contain twice as many osteoclasts.[13] These formations of bone are radiographically evident by 6 weeks and typically cease to progress in development after 12 to 24 weeks.[3,4,47,88]

Risk Factors

Several predisposing risk factors have been reported for the development of HO after arthroscopic procedures. Intrinsic factors are male sex, a history of HO, and hypertrophic osteoarthritis.[3,6-8,27,42,64,88] Other postulated factors include hips with markedly diminished range of motion, age >60 years, and coexisting bony pathologies, such as ankylosing spondylitis, diffuse idiopathic skeletal hyperostosis, and Paget disease.[22,32,52,63] Relative tissue hypoxia has been associated with periosteal bone formation, placing smokers and patients with chronic obstructive pulmonary disease at an inherently higher risk profile.[6]

Extrinsic risk factors typically pertain to the procedure performed and operative technique utilized. Arthroscopic treatment of femoroacetabular impingement of the hip is the most widely reported cause of HO after orthopaedic procedures. Relevant predisposing factors were mixed-type impingement necessitating acetabuloplasty and femoroplasty, size of cam lesion, and anchor placement.[42] Unrepaired capsulotomy and capsulectomy have been postulated to lead to higher rates of HO formation when compared with capsular closure.[61] However, in a case-control study of 100 consecutive hip arthroscopies, Amar et al[4] found no statistically significant differences in postoperative HO rate between 50 patients with capsular closure and 50 patients without. In a systematic review examining supine versus lateral decubitus positions for hip arthroscopy, the supine position was associated with an increased risk of HO (0.21% vs 0%), although statistical significance was unreported.[23]

Failure to administer HO prophylaxis after hip arthroscopy increases a patient's relative risk. In a randomized controlled trial comparing HO prophylaxis with nonsteroidal anti-inflammatory drugs (NSAIDs) versus placebo, the prevalence of HO was >10 times higher in the placebo group (46% vs 4%).[8] Although radiographic HO is not

[¶]References 3-6, 8, 9, 14-16, 18, 19, 21, 23, 27, 30, 31, 33, 42, 43, 45, 47, 50, 53-55, 57, 59, 61, 63-68, 70, 75, 77, 78, 81, 83, 86-88

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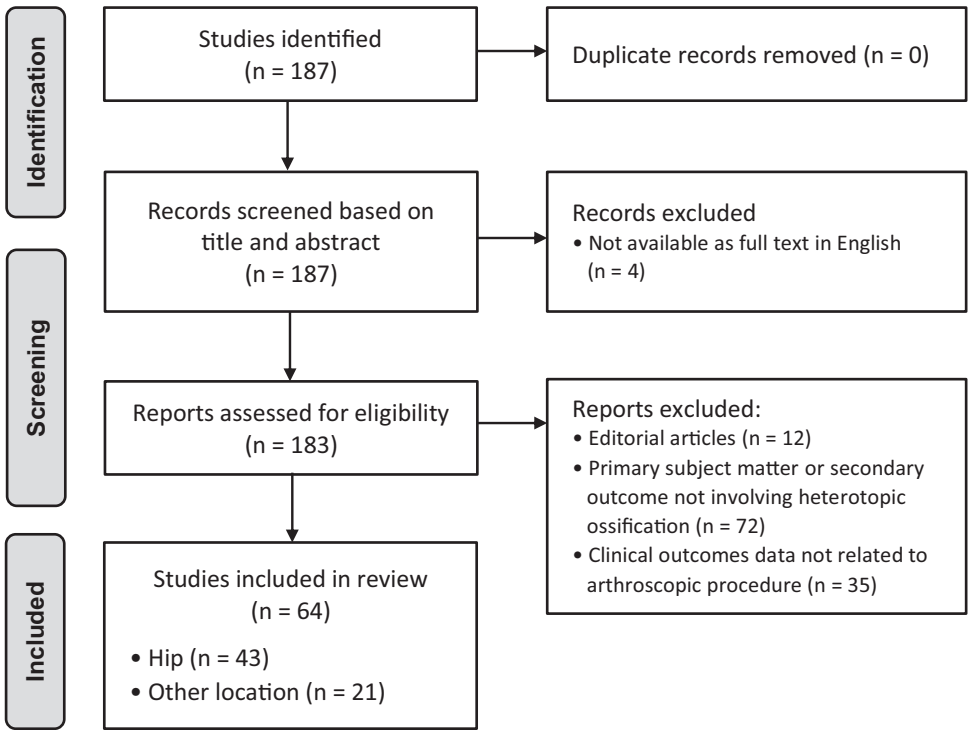

Figure 1. PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-analyses) study selection via PubMed database for clinical outcomes review.

TABLE 1
Brooker Classification of Heterotopic Ossification

Grade	Description
1	Islands of bone within the soft tissues around the hip
2	Bone spurs from the pelvis or proximal end of the femur, leaving at least 1 cm between opposing bone surfaces
3	Bone spurs from the pelvis or proximal end of the femur, reducing the space between opposing bone surfaces to <1 cm
4	Apparent bone ankylosis of the hip

uncommon after hip arthroscopy, symptomatic HO requiring revision surgery is present in <1% of cases.[42]

Classification

The severity of HO is commonly described using the Brooker classification. This radiological classification system, designed in 1973 for use in hip arthroplasty, has recently been interpreted to characterize HO of the hip after arthroscopic procedures.[42] Findings are based on anteroposterior radiographs and divided into 4 grades (Table 1). Clinically relevant HO, in which functional disability has been indicated, typically presents as Brooker grade 3 or 4.[47] Despite its widespread use, the Brooker classification system has received criticism in that an apparent bridging ankylosis may be bone located anterior or posterior to the hip on radiographs, presenting as a false source of restricted hip motion (Figure 2).[3]

Clinical Findings

Clinical findings after the development of HO vary by the severity and stage of disease. Differential diagnoses include infection (cellulitis, septic arthritis), thrombophlebitis, tumor, and soft tissue nonosseous calcification.[37] In cases of clinically significant HO, pain and swelling gradually progress and can lead to ankylosis. Early findings (at 0-4 weeks) are pain, swelling, and subjective stiffness of the site. Intermediate findings (at 5-8 weeks) comprise increasing subjective and objective stiffness. Late findings (at 8-12 weeks) include decreased range of motion, with possible ankylosis.

A recent study analyzed the severity of HO after total hip arthroplasty. While the incidence of HO was 29.9%, those with Brooker grades 1 to 3 were largely asymptomatic. Only Brooker grade 4, present in 0.57% of patients, resulted in significantly worse patient-reported outcome measures.[69] In a series of 327 patients, a recent study found 14 cases (4.28%) of radiographically confirmed HO after hip arthroscopy, with 12 patients asymptomatic at the last follow-up.[88] In most cases, HO was in the central area of the arthroscopic portals or capsulotomy. Two patients with Brooker grade 2 disease required revision hip arthroscopy to excise symptomatic HO. Both returned to their previous levels of recreational activities and had no recurrence at 2-year follow-up.

Imaging and Laboratory Findings

Imaging modalities that are helpful in assessing HO include radiography, ultrasonography, computed

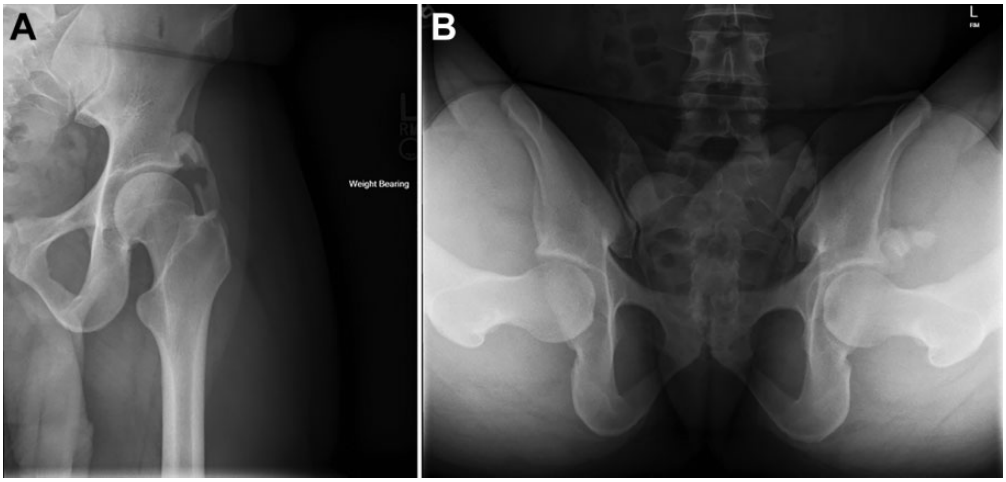

Figure 2. (A) Anteroposterior radiograph of a patient with heterotopic ossification of the left hip. Apparent bridging ankylosis is visualized. (B) A 90° Dunn view of the same patient demonstrating that the lesion is anterior to the femoral neck, without evidence of ankylosis.

tomography (CT) scan, technetium-99 bone scan, Raman spectroscopy, and near-infrared imaging. Radiographs remain the imaging modality of choice for initial evaluation. Ultrasonography is quick and convenient but is largely user dependent. While bone scans detect HO earlier than radiographs, they are expensive, and it can be difficult to distinguish postoperative inflammation versus HO. CT scans may facilitate preoperative planning when excision is indicated. Raman spectroscopy and near-infrared imaging detect disease earlier than radiography by spotting mineralized collagen within tissues, but availability remains limited.[62]

Commonly performed laboratory tests include serum calcium, phosphorus, and alkaline phosphatase. These test values are high in early-stage disease but tend to return to normal by 9 to 12 weeks.[34] Other laboratory values that may be elevated in acute-stage disease are C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and serum creatine kinase.[17,29,72] CRP is more specific than ESR, and both tend to normalize over time.[29] While CRP and ESR are elevated in cases of infection, the addition of calcium, phosphorus, and alkaline phosphatase is collectively more specific for HO. Finally, biopsy may be considered if tumor is on the differential, but it tends to exacerbate the symptoms of HO.[37]

Prophylaxis

HO prophylaxis in the surgical setting may be broadly categorized into 2 modalities: NSAIDs and radiotherapy. Three factors govern the prevention of HO: disruption of signaling pathways, reduction of the osteoprogenitor cells in the area, and modification of the cellular environment. NSAIDs lower prostaglandin levels and disrupt signaling pathways, thereby inhibiting osteogenic differentiation of progenitor cells. Radiotherapy acts more broadly and is thought to reduce the number of osteoprogenitor cells in the irradiated area.[37] These treatments can be used exclusively or in combination.

NSAIDs remain the most common prophylaxis for arthroscopic procedures, owing to their ease of use and availability. A recent systematic review consisting of 1662 patients undergoing hip arthroscopy cited a 13.4% incidence (range, 1.3%-46.0%) without NSAID prophylaxis as opposed to 3.3% (range, 1.8%-8.3%) with it.[86] The authors found all NSAIDs to be effective, without superiority of nonselective NSAIDs. Regimens included the following nonselective NSAIDs: naproxen (500 mg, twice daily for 3 weeks), aceclofenac, ketoprofen, indomethacin alone, or indomethacin (75 mg, daily for 4 days) followed by naproxen (500 mg, twice daily for 3 weeks). Selective NSAIDs were celecoxib (200 mg, daily for 3 weeks) and etoricoxib (90 mg, daily for 3 weeks). This corresponds to a separate systematic review of 808 patients undergoing total hip arthroplasty, which had a similar incidence of HO with nonselective versus selective cyclooxygenase (COX) inhibitors.[84] A recent randomized controlled trial using naproxen (500 mg, twice daily for 3 weeks) demonstrated a 46% prevalence of HO in the placebo group versus 4% in the naproxen group.[8]

Radiotherapy has not been studied exclusively in a cohort of patients undergoing primary arthroscopic procedures and is less commonly used than NSAIDs for HO prophylaxis. However, radiotherapy regimens utilizing external beam radiation have been applied in arthroplasty, trauma, and tumor surgery. As compared with NSAIDs, radiotherapy has marginal superiority at doses >600 cGy.[40,56] Radiation can be administered 24 hours preoperatively to 72 hours postoperatively at a dose of 700 to 800 cGy.[62] In a recent systematic review of procedures predominantly involving the hip, there was no difference in postoperative versus preoperative radiotherapy and no biologically effective radiation dose in the development of

HO.[60] The most common regimen was a dose of 700 cGy administered postoperatively.

HO After Hip Arthroscopy: Management Outcomes

Although uncommon, revision surgery to excise HO is patient specific and may be indicated in circumstances of refractory pain and/or restricted motion. Resection of immature HO may lead to increased complications and recurrence; therefore, characterization of HO maturity before surgery is paramount.[3] Criteria include radiographic assessment of the ossification reflecting dense cortical bone, normalized levels of serum alkaline phosphatase, calcium, phosphorus, and normal bone scan findings.

Open and arthroscopic techniques have been described for HO excision. Theoretical advantages of arthroscopy in comparison with open HO excision include decreased potential for wound complications, less surgical morbidity, and accelerated rehabilitation. In a series of 616 hip arthroscopy procedures, 29 hips (4.7%) developed HO postoperatively, with 7 (1.1%) undergoing revision procedures to excise HO at a mean 11.6 months after the index case.[9] Of these 7 patients, 6 were male; 6 did not receive prophylactic NSAIDs because of an existing allergy; and all 7 had undergone arthroscopic osteoplasty with a capsular cut. No patients had recurrence of HO after excision, although the number treated with open versus arthroscopic techniques was not specified. Ong et al[54] described an arthroscopic technique using spinal needle localization of lesion sites under fluoroscopy to establish working portals for HO excision. Contraindications to their technique were Booker grade 4 HO, posteriorly located lesions, immature HO, arthritis, and inaccessibility attributed to patient obesity. In the 3 patients who underwent the procedure, the mean modified Harris Hip Score improved from 73 to 92 at 26-month follow-up.

In hips with Brooker grade 3 or 4 HO, the extensive nature of the ectopic bone may necessitate open excision. Bedi et al[9] reported on 4 patients who underwent this procedure, followed by immediate dosing of 700 cGy of radiation therapy for prophylaxis, with no cases of recurrent HO after the treatment protocol. Proximity to major vessels has also been cited as an indication for open HO excision. Kurz et al[42] recommended CT angiogram as part of the routine preoperative imaging workup, particularly as a means to rule out vascular abutment or encasement within the HO mass. Future studies directly comparing arthroscopic and open techniques are needed to clarify outcomes.

HO After Arthroscopy Outside the Hip: Management Outcomes

Shoulder. While uncommon, studies of HO after arthroscopic interventions have been conducted outside the hip (Table 2). There are several case reports involving HO of the shoulder. Sanders et al[71] presented a case of severely symptomatic HO within the deltoid after arthroscopic rotator cuff repair that was discovered on 3-month follow-up radiographs. The patient underwent preoperative radiation with 750 cGy, followed by open excision of HO and arthroscopic lysis of adhesions. He had full functional recovery with no recurrence at 2-year follow-up. Similarly, Dhollander et al[25] noted symptomatic HO within the deltoid and coracoacromial ligament after rotator cuff repair, which was managed successfully with excision and NSAIDs.

Reossification and fusion across the acromioclavicular joint after distal clavicle resection were reported in a 44-year-old woman.[79] While the patient had an uneventful recovery in the 3 months after the index procedure, she did not return for follow-up until she was referred back to the operating surgeon 12 months later for recurrent symptoms. The acromioclavicular joint ankylosis was excised with 1.5 cm of the distal clavicle through an open approach, with no recurrence at 9-month follow-up. Three studies involving 13 patients reported on HO within the subacromial space, subdeltoid fascia, and supraspinatus tendon after subacromial decompression.[10,39,49] All patients presented with impingement symptoms, with 11 patients experiencing pain resolution after revision arthroscopic decompression. Severe HO after arthroscopic acromioplasty in a 48-year-old man at 22-month follow-up has been reported, with bony bridging of the acromion to the humerus.[13] Disuse osteopenia of the humerus had developed in the absence of glenohumeral motion. The patient underwent glenohumeral fusion, with 85% pain reduction at 1-year follow-up and forward flexion to 90°.

Elbow. To date, there have been 8 case reports on HO after arthroscopic procedures of the elbow.[2,24,32,35,36,51,74,85] Gofton and King[32] reported on a 47-year-old farmer who developed HO anteriorly and posteriorly to the distal humerus after arthroscopic loose body removal. At 4 years postoperatively, the patient underwent an open debridement and capsulectomy for residual stiffness and recurrent locking sensations. Adjunctive irradiation with 600 cGy and perioperative indomethacin was initiated for prophylaxis, with no recurrence at 6-month follow-up. Sodha et al[74] described a case of HO after arthroscopic debridement for posteromedial olecranon impingement in a 17-year-old baseball pitcher, with range of motion limited to 30° to 120° at 22 months after the index procedure. After excision of the mature lesion and anterior capsular release, the patient regained full range of motion and returned to competitive pitching. Desai et al[24] presented a case of HO after arthroscopic debridement of lateral epicondylitis in a former collegiate basketball player. Early HO anterolateral to the lateral epicondyle was found on radiographs 7 weeks postoperatively, and he underwent open HO excision with repair of the common extensor origin 11 weeks later, with a single 700-cGy dose of adjunctive radiation therapy on the day of surgery. No recurrence was present at 14-month follow-up. Hughes and Hildebrand[35] described ankylosis of the anterior elbow after arthroscopic debridement and ulnar nerve decompression. Although HO removal was performed 6 months after the index case, the patient had recurrence of the lesion 2 weeks later. Four additional studies involving 46 patients have reported on excision of HO

TABLE 2
Studies of HO After Arthroscopic Procedures Outside the Hip[a]

Lead Author	Sample Size	HO Location	Index Procedure	Clinical Findings	Management; Outcome
Sanders[71]	1	Shoulder (deltoid)	RCR	Pain	Preoperative radiation, open HO excision, LOA; full recovery
Tytherleigh-Strong[79]	1	Shoulder (AC joint)	DCR	Impingement pain, ankylosis	Open excision; no recurrence
Boynton[13]	1	Shoulder (acromion/humerus)	Shoulder arthroscopy, SAD	Pain, restricted ROM, disuse osteopenia, ankylosis	Glenohumeral fusion; 85% improvement in pain, 90° FF
Dhollander[25]	1	Shoulder (deltoid/coracoacromial ligament)	RCR, biceps tenotomy, acromioplasty	Impingement	Arthroscopic excision, NSAIDs; no recurrence
Merolla[49]	2	Shoulder (supraspinatus tendon)	Shoulder arthroscopy, SAD	Pain, impingement	Arthroscopic excision; no recurrence, slight pain in 1 patient
Kircher[39]	1	Shoulder (subdeltoid fascia)	Shoulder arthroscopy, SAD, ACJ resection, mini-open RCR	Pain, restricted ROM	Open excision, radiation, diclofenac prophylaxis; no recurrence
Berg[10]	10	Shoulder (subacromial space/AC joint)	SAD	Impingement (8 patients), asymptomatic (2 patients)	Arthroscopic SAD (and DCR when indicated), radiation (1 patient), indomethacin (2 patient); recurrence (1 patient, asymptomatic)
Gofton[32]	1	Elbow (anterior/posterior compartments)	LBR	Stiffness, locking	Open debridement, capsulectomy, postoperative radiation/NSAIDs; no recurrence
Sodha[74]	1	Elbow (olecranon fossa)	Debridement for posteromedial impingement	Extension bock	Open excision, anterior capsular release; full ROM
Desai[24]	1	Elbow (common extensor origin)	Debridement of lateral epicondylitis	Stiffness	Open excision, postoperative radiation; improved ROM, no recurrence
Hughes[35]	1	Elbow (anterior)	Debridement/ulnar nerve decompression	Stiffness, ankylosis	Open excision, preoperative radiation; recurrence
Intravia[36]	14	Elbow	Elbow arthroscopy	Unreported	Nine cases requiring resection; unreported
Yang[85]	25	Elbow (intra-/periarticular)	Capsular release	Categorized as minor/moderate/severe, otherwise unspecified	Repeat arthroscopy in 4 patients; unreported
Nelson[51]	6	Elbow (intra-/periarticular)	Osteocapsular arthroplasty	Uncategorized	Excision; unreported
Adams[1]	1	Elbow	Arthroscopic osteophyte resection and capsulectomy	Uncategorized	Open excision; unreported
Ogilvie-Harris[52]	4	Knee (femoral tunnel)	ACLR	Lateral knee pain, clicking	Open excision, indomethacin; no recurrences
Kumar[41]	1	Knee (PCL femoral tunnel)	ACLR/PCLR, hamstring autograft	Pain	Patient declined intervention
Bhandary[12]	6	Knee (femoral tunnel)	ACLR	Unreported	Unreported
Berndt[11]	1	Knee (medial arthroscopic portal)	Partial medial meniscectomy	Pain, mass	Arthroscopic excision; no recurrence
Patton[58]	3	Knee (posterior capsule)	Reconstruction of knee dislocation (ACL/PCL / ± PLC and MCL)	Pain, restricted ROM (2 patients), asymptomatic (1 patient)	Anterior soft tissue releases performed; HO not pain generator
Willis[82]	1	Ankle (tarsal tunnel)	Ossicle excision	Pain	Open excision, indomethacin; no recurrence

[a]AC, acromioclavicular; ACJ, acromioclavicular joint; ACL, anterior cruciate ligament; ACLR, anterior cruciate ligament reconstruction; DCR, distal clavicle resection; FF, forward flexion; HO, heterotopic ossification; LBR, loose body removal; LOA, lysis of adhesions; MCL, medial collateral ligament; NSAIDs, nonsteroidal anti-inflammatory drugs; RCR, rotator cuff repair; ROM, range of motion; PCL, posterior cruciate ligament; PCLR, posterior cruciate ligament reconstruction; PLC, posterolateral corner; SAD, subacromial decompression.

after elbow arthroscopy, although no outcomes data are available.[2,36,51,85]

Knee/Ankle. Ogilvie-Harris and Sekyi-Otu[52] reported on HO development in a series of 4 patients who underwent anterior cruciate ligament reconstruction with bone–patellar tendon–bone autograft, utilizing an outside-in drilling technique for the femoral tunnel. In each case, the ectopic bone developed at the tunnel entry site at the lateral cortex, causing lateral knee pain and clicking sensations. After HO excision and a 3-month course of postoperative indomethacin, no recurrence was found at minimum 6-month follow-up. A similar series was published by Bhandary et al[12] in 6 patients, although management was not described. Two studies described HO after multiligamentous knee reconstruction. Kumar et al[41] reported on the development of HO at the posterior cruciate ligament (PCL) femoral tunnel aperture after combined anterior cruciate ligament and PCL reconstruction in a 29-year-old man 8 months after the index procedure. The patient's symptoms were mild, and he declined further intervention. Patton and Tew[58] presented a series of 3 patients with knee dislocations who underwent multiligamentous reconstruction. HO was identified within the posterior knee on radiographs obtained at 4- to 6-week follow-up. Interestingly, 2 patients underwent manipulation under anesthesia and lysis of adhesions attributed to knee flexion deficit and anterior knee pain. The authors attributed the development of posterior HO to a combination of PCL tibial reaming and a favorable environment for mesenchymal cell differentiation given the high-energy mechanism of injury, but they concluded that the HO was not responsible for the patients' symptoms. Berndt et al[11] presented a case of HO within the medial arthroscopic portal after partial medial meniscectomy, which presented as a painful lump. After excision, complete pain relief and normal knee function were achieved. A single case of HO after ankle arthroscopy was presented by Willis et al,[82] in which a 19-year-old woman athlete developed tibial nerve entrapment within the tarsal tunnel after ossicle excision. The patient underwent surgical resection and postoperative indomethacin prophylaxis, without further recurrence at 1-year follow-up.

Discussion

After arthroscopic procedures, we typically reserve the use of HO prophylaxis for arthroscopic procedures of the hip and in patients with a known history of HO. Our standard protocol is to start prophylaxis on postoperative day 1 with celecoxib (200 mg, daily for 6 weeks). Our preference for using a selective COX-2 inhibitor is predicated on the reduced risk of gastrointestinal adverse effects in comparison with nonselective inhibitors. The lack of evidence for usage of radiation therapy exclusively in primary arthroscopic procedures, coupled with its potential for malignant transformation at higher doses, obviates its usage in our practice. In addition to the proven efficacy, NSAIDs are less costly, more readily accessible, and easier to administer.

After the presentation of symptomatic HO, it is our preference to identify bony maturation before surgical excision (typically occurring 6-12 months after surgery) to minimize recurrence. Radiographs should demonstrate a lesion density similar to cortical bone, and serum alkaline phosphatase levels and bone scan findings should be normal. Low-grade lesions may be excised arthroscopically, while more advanced HO often requires open excision. Postoperatively, pharmacologic prophylaxis is identical to our protocol used for primary cases.

CONCLUSION

HO is a known complication of arthroscopic procedures, particularly after arthroscopy of the hip, although other sites include the shoulder, elbow, and knee. Abnormal proliferation of bone within nonskeletal tissue is dependent on local inflammation and environmental factors. Risk factors include male sex, history of HO, and excessive bony resection during surgery. Prophylaxis after hip arthroscopy has been shown to reduce occurrences. Once HO is present, most cases are asymptomatic, although larger lesions may result in pain and restricted range of motion, which may necessitate surgical resection upon lesion maturation.

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