The Non-operative Management of Hip Disease in Young Adults

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Introduction

Hip pain is a common complaint in the young adult population. Up to 10 % of patients presenting to sports medicine clinics have a primary complaint of chronic hip or groin pain [1–3]. Groin injuries account for up to 16 % of all athletic injuries in elite football players [4]. A high incidence of groin injury is also noted in ice hockey [5], American Football [6] and sports involving running, twisting or kicking [7–9]. Chronic groin injury often presents insidiously and may not always result in an abrupt cessation of sporting activity. The true mechanism of these injuries may therefore be unclear and the incidence under reported. Holmich proposed a 'clinical entities' approach to categorize groin pain as primarily adductor, psoas or rectus abdominis related [10]. However, within that report, the proportion of patients with hip pathology presenting primarily as sports-related groin pain was remarkably small. Only 3 of 207 athletes were noted to have hip joint related pain.

In a 7 year prospective study of 23 professional European football clubs, 12–16 % of injuries requiring time off from training were related to the hip and groin [11]. Adductor injuries were the most common (64 %) and 6 % of cases were diagnosed as hip joint pathology. Of the latter, the most common cause was hip joint synovitis but labral tears and chondral injuries were also noted. Only two patients were diagnosed with femoroacetabular impingement (FAI). This may be explained by the fact that only 16 plain radiographs were performed, perhaps suggesting a lack of awareness of this condition. A prospective cohort study of patients with chronic groin pain in private practice demonstrated hip pathology as the most prevalent group of conditions [12]. A 10 year retrospective study of professional American Football players reported that 3 % of all injuries were localized to the groin [13]. Of these, 5 % were intra-articular hip injuries with the majority being fractures. Only five labral tears were reported in 23,806 injuries recorded in the National Football League (NFL) between 1997 and 2006. The recent increase in utilization of MRI as an imaging modality has identified labral injuries to be a common and significant source of morbidity in the young athlete's hip [14–16].

Subtle morphological abnormalities around the hip joint are being increasingly identified in symptomatic and asymptomatic young adults [17]. Collectively termed FAI, this condition is now a recognized cause of hip pain secondary to chondrolabral dysfunction and a precursor to secondary osteoarthritis (OA) of the hip. It is therefore important for medical practitioners to have a high index of suspicion for FAI in young adults presenting with hip or groin pain. Clear management protocols are also essential to direct appropriate and timely investigations and guide treatment strategies.

Patients presenting with activity related hip pain, biomechanical dysfunction or anatomical abnormalities around the hip require a medical management plan in addition to consideration for surgical intervention. Medical management in these patients may encompass physical therapy, pharmacological interventions and intra-articular injections. Even in patients with a surgically correctable pathology of the hip, a rehabilitation plan focused on improving function and activity is critical for long term success.

The differential diagnosis of hip pain is extensive and accurately identifying the cause of hip pain on history and physical exam alone can be a challenge even for the seasoned physician. Furthermore, multiple etiologies may be present in up to 34 % of patients with chronic groin pain [10]. Normal biomechanics of the hip joint depend on well-coordinated muscle activity around a stable and congruent pelvis and proximal femur. Damage to a single structure may result in an imbalance that requires alterations in activity. These alterations can subsequently place abnormal stresses on other

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structures within the pelvis leading to secondary injury which may be detected clinically [18–20]. Iliopsoas muscle related pain was the most common secondary origin of pain in the Holmich study, consistent with its role as the major hip flexor and its importance in lumbo-pelvic function and stability.

The clinical entity of OA involves a number of different pathophysiological processes in its progression and development. Articular cartilage degradation, tissue synovitis and subchondral bone remodeling are just three examples of pathological processes which may be active in isolation or co-exist. Appropriate identification of active pathology should enable effective and targeted management strategies.

This chapter provides an overview of medical interventions aimed to assist the clinician in developing an overall management strategy for dealing with hip and groin pathology. It discusses a range of non-operative treatment options available including the role of physical therapy, oral medication, intra-articular injections and radiofrequency ablation. The potential role of these modalities in specific pathologies around the hip is discussed.

Physical Therapy

Appropriate physical therapy is a cornerstone for effective management of hip injury in the young athlete. The aim of exercise is to improve function, pain or pathology through the selection or avoidance of particular activities.

Femoroacetabular Impingement (FAI)

FAI is diagnosed when a bony abnormality exists at the proximal femur (cam type) or the acetabulum (pincer type) resulting in abnormal contact between the acetabular rim and the femoral head neck junction during hip flexion results. This results in reduction in range of movement and fissuring at the chondrolabral junction [21–23]. FAI can be painless or painful and limit athletic activity.

The prevalence of cam-type impingement in young asymptomatic individuals is around 15 % [24–26], but it is notably more common in males [25, 27, 28]. Pincer-type impingement is more common in females [29]. It is important for clinicians treating young athletes to be aware of the at risk positions of the hip joint which can increase the likelihood of impingement. Sprinters are at risk during the first few steps after the block start when the hip is in a flexed position [30] and the drive phase causes an internal rotation shear on the hip joint. In ice hockey, the initial push off requires abduction and external rotation of the hip [31, 32], a vulnerable position for the anterolateral acetabular labrum [33]. This is followed by hip flexion and internal rotation, a second at-risk position for the anterolateral labrum. As speed increases, the rate and degree of rotation of the hip joint also increases. The risk of symptomatic impingement and damage to the labrum is likely to be greater at higher velocities [34, 35]. FAI is a likely risk factor for and often misdiagnosed as groin strain [35, 36].

In athletes with recognized FAI, it may be prudent to limit the volume and intensity of the type of training which puts the hip into a vulnerable position. For a sprinting athlete this may mean less time spent doing block starts or hill sessions. The range of motion and joint position that athletes adopt during stretching and drills should also be considered. A muscle strengthening program can be devised to improve deceleration during rotation movements at the hip. Targeted strengthening to ensure optimal force attenuation through the kinetic chain will also reduce impact load on the labrum, chondral and bony surfaces during sporting activity.

The normal range of hip motion is 30–40° of internal rotation in 90° of hip flexion [37]. It has been reported that reduction in the normal range of motion is a risk factor for the subsequent development of groin pain [36]. In patients with FAI, hip internal rotation at 90° of hip flexion is limited to less than 15° [38–40]

When the usual joint range of motion for an athlete with underlying FAI is reduced, the clinician should be prompted to identify triggers and modify activity as required. It is also important to note that aggressive physiotherapy aimed at increasing range of motion is only likely to result in further micro-trauma at the labrum and is not recommended.

In addition to range of motion restriction, there have been some recent studies on FAI related kinematics which can help inform clinical decision making. Painful hip adduction and internal rotation during high intensity dynamic activities has been noted in a case report [41]. There is some good quality research on hip kinematics during walking demonstrating a reduction in hip flexion angle and reduced peak hip abduction angle [42, 43]. It is interesting to note that similar changes are seen in patients with OA and may allude to the role of FAI in the continuum of OA. This notion is supported by a recently published kinematic study which has described a reversal of these changes following FAI surgery [43, 44]. However, a significant portion of the altered biomechanics in FAI may result from hip muscle weakness. A recent study has compared hip muscle strength and EMG activity in patients with symptomatic FAI [45]. Patients with FAI were noted to have significantly reduced maximal voluntary contraction strength, in the order of around 16 %, for hip adduction, flexion, external rotation and abduction. Weakness in these muscle groups, particularly the external rotators and abductors could increase antero-medial bony contact stresses in the hip joint during dynamic activity [46]. There have been some preliminary studies which have demonstrated symptomatic and functional improvement in patients with FAI with a targeted strength and co-ordination program [46].

However, long term benefits of conservative treatment, when reported in the orthopedic literature, are usually limited [17, 47]. Unfortunately, while kinematic studies have been performed both pre and post operatively, these have not usually followed a conservative strengthening program. While the presence of a painful impingement will limit activation and rehabilitation of related muscle groups, the effects of a targeted exercise program on outcomes after FAI surgery warrant further investigation, particularly if combined with other medical strategies to reduce pain.

Labral Pathology

Labral tears of the hip joint can be a significant source of pain and dysfunction [14]. The labrum has a role in shock absorption, lubrication, stability and distribution of forces within the hip joint [33]. There is a clear association between labral tears and early onset OA [48, 49]. The occurrence of labral tears may be associated with trauma, FAI, dysplasia or capsular laxity [50]. In addition to athletes with predisposing anatomy, labral tears often occur in those who undertake repetitive rotational movements on a loaded femur [51, 52]. These movements increase stress on the capsular tissue and iliofemoral ligament. The resultant rotational instability can increase pressure on the anterior superior labrum. Activities requiring frequent external rotation of the hip such as ballet, golf and football have all been associated with labral pathology [15, 16, 53].

Exercise regimens should be based on the predisposing etiology and extremes of movement which place additional stresses on the labrum should be avoided. There is limited literature in this area and one orthopaedic review concluded that physical therapy is not recommended [54]. A therapy protocol has been described in the literature but there has been no critical assessment of its efficacy [55]. The principles of the program were strengthening of iliopsoas, hip abductors and external rotators and addressing gait dysfunction, with the aim of limiting hip hyperextension which would subsequently reduce anterior joint reaction forces [56]. However, there is no strong evidence or rationale to support conservative management and surgical intervention may well be required in athletes with symptomatic labral tears.

Early Osteoarthritis

In early OA, articular cartilage degeneration, subchondral bone remodeling and tissue synovitis can all contribute to progression of clinical symptoms. Pain is the predominant symptom and is often associated with joint stiffness, reduced range of joint motion, instability and muscle weakness. This may result in impaired global physical function and the development of compensatory movement patterns with load transfer to other musculoskeletal structures. With worsening OA symptoms, patients may experience physical and psychological disability which limits activities of daily living and impair their quality of life.

Exercise traditionally plays a role in the management of early hip OA and is specifically targeted towards improving muscle strength, range of motion, joint control and stability. The goals of exercise are to reduce pain, improve physical function and optimise participation in social and recreational pursuits. Whilst these generic goals are applicable to older patients with hip OA, they are equally relevant to the young athlete whose early functional restriction may cause significant psychosocial problems. Although exercise can provide symptomatic relief in hip OA, there is currently no evidence to suggest that it can influence underlying structural disease or modify it [57].

Findings from studies involving patients with knee OA cannot be directly extrapolated to the hip, due to differences in joint biomechanics, type of functional impairment, rapidity of progression and risk factors [57]. Recent systematic reviews have concluded that there is insufficient evidence to support the use exercise as a sole management approach in the short term, for reducing pain, or improving function and quality of life [58, 59]. However, a meta-analysis by Hernandez-Molina et al. [60] which included hydrotherapy, concluded that physical therapy was effective treatment for hip OA when supervised specialist exercises and muscle strengthening were incorporated into a program. In clinical practice exercise normally forms part of a package of care in OA. This includes analgesics, NSAIDs, structure-modifying slow-acting drugs. One feasibility study has found preliminary evidence that hip and knee OA patients can obtain health-related benefits from the combination of glucosamine sulphate and a progressive home-based walking program [61]. Furthermore, in overweight adults with knee OA, the combination of modest weight loss and exercise provided better overall improvements in self-reported outcomes and performance measures when compared to either intervention alone [62]. Clinically, the optimal mode and intensity of exercise for hip OA is unknown and few studies have compared different exercise programs [57].

Exercise regimens for hip OA should be individualized and patient-centered. They require assessment of specific impairments relative to the underlying etiology and degenerative change. In FAI with early OA, addressing strength and co-ordination of specific muscle groups, aimed at reducing anteromedial stress during activity, may improve symptoms and joint function [63]. Aerobic fitness and patient preferences will also influence the regimens used. Individualization of the exercise program to the unique requirements of the patient as well as ensuring availability of resources can be effective in maximizing compliance [57, 64]. There is also evidence that supervision may improve outcomes during an exercise program. Marked improvements in locomotor function and pain have been shown by supplementing a home-based exercise program with

physiotherapist-led group sessions [65], and there is evidence from meta-analyses that increasing the number of directly supervised exercise sessions improves the treatment effect [58].

Obesity

When treating the patient with hip OA, weight-reduction strategies form an important component of the overall management strategy. Being overweight (BMI 25–30 kg m^{-1}) or obese (BMI >30 kg m^{-1}) are well-known risk factors for OA. Leptin is an adipose-derived hormone which circulates at levels proportional to body fat and is therefore overexpressed in the obese [66, 67]. It is present in the synovial fluid and, under physiological conditions, stimulates synthesis of IGF-1 and TGFβ-1 by binding to leptin receptors on articular chondrocytes [68]. These mediators are important for chondrocyte proliferation and extracellular matrix (ECM) synthesis and thus have a positive anabolic effect on the joint by increasing cartilage matrix production [69]. However, in pathological concentrations leptin mediates catabolic effects on articular cartilage [70]. Leptin enhances the synthesis of several pro-inflammatory mediators, including PGE_2, IL-6, IL-8 and nitric oxide (NO) [71]. High NO levels result in reduced production and increased degradation of ECM and chondrocyte apoptosis [72]. Leptin also induces synthesis of matrix metalloproteinases (MMP), a large family of enzymes that degrade proteoglycans and other cartilage components, leading to structural damage of cartilage.

These factors suggest that obesity, mediated by leptin, can lead to chondrocyte apoptosis and degradation of the ECM [69]. Obesity can therefore be regarded as a significant modifiable risk factor for OA both as a result of biomechanical joint overload and its adverse metabolic effects. There is therefore a rationale for exercise in OA specifically as part of a weight-reduction strategy.

Oral Medication

Paracetamol

Paracetamol is a widely used simple analgesic with antipyretic properties [73]. It does not have a particular anti-inflammatory effect but is recommended by numerous guidelines in the treatment of early OA [74–76]. It is considered safe at a maximum dose of 4 g per day. Paracetamol is hepatotoxic at higher doses and should be avoided in patients with liver disease and chronic alcohol abuse. The use of an effective analgesic in hip pathology can be of particular importance in conjunction with the overall management plan. If pain is controlled early and appropriate management instituted to address the injury, secondary consequences may be avoided.

A number of reviews and meta-analyses on the role of paracetamol in mild to moderate OA have shown that it is effective in providing early pain relief but that NSAIDs are marginally superior in improving hip and knee pain, particularly in advanced OA [77–79]. It is widely accepted that OA is an inflammatory arthropathy and it is to be expected that reducing inflammation will result in greater improvements in pain. The majority of studies have included hip and knee OA within the same group. Recent studies have noted moderate clinical heterogeneity between patients with knee or hip OA and therefore recommended that future research considers these as separate clinical conditions [80].

NSAIDs

Non-steroidal anti-inflammatory drugs (NSAIDs) are recommended for use in the management of hip OA [74, 75, 81]. NSAIDs function both centrally and peripherally, and are primarily effective in reducing inflammation and nocioceptor-mediated pain through Cyclo-Oxygenase (COX) inhibition [82]. Inhibition of COX results in a decrease in prostaglandin synthesis.

Oral NSAIDs are essentially divided into those that are selective for COX-2 inhibition and those that are nonselective for COX-1 and COX-2 [83]. COX-2-selective NSAIDs were developed to reduce the risk of gastric bleeding and ulceration since nonselective COX inhibition reduces synthesis of certain prostaglandins which protect gastric mucosa against acid attack. Significant gastro-intestinal complications such as bleeding or perforation occur in 0.2 % of patients taking COX-2-specific agents, compared with 2 % taking non-selective NSAIDs [84]. However, COX-2 inhibitors have potentially substantial cardiovascular risk [85], and as a direct result, two widely distributed COX-2 inhibitors (rofecoxib and valdecoxib) were recently withdrawn from the market. NSAIDs can also adversely affect renal function and both NSAIDs and COX-2 inhibitors can adversely effects bone and tendon healing [86–88].

NSAIDs are routinely recommended in OA if paracetamol alone cannot control symptoms or if there are signs of clinical inflammation [74, 75, 81]. They should be used at the lowest effective dose and consideration should be given to the concomitant use of a gastro-protective agent such as a proton pump inhibitor or misoprostol in patients with increased gastrointestinal risk. One systematic review found NSAIDs to be slightly more effective than paracetamol in patients with hip OA [80].

In non-arthritic hip conditions, the rationale for using NSAIDs should be based on the presence of concomitant inflammation. In labral injury or FAI, the clinical presentation can include episodes of joint synovitis which may respond to short-term use of NSAIDs. OA has not previously

been synonymous with inflammatory arthropathy, though we now know that inflammatory mediators are expressed in the cartilage and synovial tissues in the early stages of OA and that they are involved in cartilage degeneration [69]. NSAIDs in early OA may have a disease-modifying role.

Codeine Based Medication

Opioids have been shown to be of some benefit for the treatment of pain associated with arthropathy [89, 90]. However, their use may be associated with adverse events, particularly nausea, dizziness and constipation. This may limit their role in the treatment of the young adult hip. They may be helpful for short term pain relief but should not be used regularly as a long term treatment option.

Glucosamine and Omega-3 Fatty Acids

Articular cartilage has limited ability to regenerate or adapt to altered mechanics. It is avascular and receives nutrients by diffusion from surrounding tissues and joint fluid. Chondrocytes maintain composition and organization of the ECM which consists of a network of collagen and elastin within a proteoglycan gel [69]. Proteoglycans have a net negative charge and hold a large amount of water within the cartilage. They confer resilience and elasticity to cartilage and aid in lubrication of the joint system. Proteoglycans are large molecular complexes, composed of a central hyaluronic acid (HA) filament, to which aggrecan molecules composed of chondroitin sulfate and keratan sulfate are attached. In OA, the balance between catabolic and anabolic processes within articular cartilage is disturbed and chondrocytes are unable to compensate for the loss of collagen type II fibers and proteoglycans despite increased synthesis [91].

The amino-monosaccharide glucosamine is an essential component of proteoglycan synthesis. The availability of glucosamine, synthesized from glucose in human tissues, is one of the rate-limiting steps in proteoglycan production [69]. As a dietary supplement, glucosamine may overcome this rate limitation and support joint health as suggested by numerous in vitro studies [92–94]. Glucosamine enhances production of aggrecan, collagen type II, and HA [93]. It may prevent collagen degeneration in chondrocytes by inhibiting lipoxidation reactions and protein oxidation. It may also inhibit the predominant cleavage enzymes in cartilage (MMP and aggrecanases) and hence prevent proteoglycan degradation [94, 95].

Inflammation in OA is not simply a secondary event [96, 97]. Inflammatory mediators are expressed in cartilage and synovium in early OA in response to mechanical overload, trauma, and obesity. Benito et al. [98]. have found that expression of both inflammatory mediators and transcription factors from the inflammatory cascade is significantly higher in the earlier stages of OA. A combination of inflammation and oxidative stresses leads to cartilage degeneration and chondrocyte apoptosis. Glucosamine has been shown to act in a number of ways to modulate the inflammatory cascade and exert an anti-oxidant effect. In particular, glucosamine may suppress the IL-1 induced expression of COX-2 and NO [99], two mediators which trigger inflammation and are implicated in chondrocyte apoptosis.

In clinical trials, glucosamine has been shown to delay progression of knee OA. Similar effects have not been demonstrated in hip OA, for reasons that are unclear. There are a number of contentions why this may be so. The anatomy, vascular supply and cartilage loading within the hip are very different to that in the knee. Nevertheless, in evaluating the evidence from available clinical trials, meta-analyses and reviews in knee OA, authors have concluded that long term treatment with glucosamine reduces pain, improves function and mobility of the joint, reduces disease progression and reduces risk of total joint replacement [100, 101]. These conclusions have also been applied to recommendations for hip OA despite the limited clinical evidence. Glucosamine sulphate is taken as a daily dose of 1,500 mg and most trials have demonstrated tolerance of this dose at least the same as placebo and better than for NSAIDs. There has been conflicting evidence on the effect of glucosamine from both clinical trials and meta-analyses, with high placebo effect, subject heterogeneity and bias due to industry funding all cited as potential confounding factors. A network meta-analysis by Wandel et al. [102]. in the British Medical Journal concluded that "compared with placebo, glucosamine, chondroitin, and their combination do not reduce joint pain or have an impact on narrowing of joint space". Furthermore, they recommended that patients on these supplements may continue their use based on good safety and perceived benefit, but that new prescriptions should be discouraged given the lack of putative clinical relevance. However, Bruyere [103] has challenged their trial selection, high study heterogeneity and the use of a complex Bayesian analysis. Glucosamine supplementation is recommended by European and international guidelines on the treatment of OA and there is a wealth of data from in vitro studies and clinical trials and reviews which provides a sound rationale for its use in chondropathic conditions [101, 104–106].

Chondroitin sulphate is a natural glycosaminoglycan and an important component of the extracellular matrix. The European League Against Rheumatism recommendations regarding knee OA gave chondroitin sulphate the highest evidence grade and recommend that effects may be noticeable within 3 weeks [107]. In addition to its role as constituent of the ECM it can increase hyaluronan production and stimulate further anabolic effects [108, 109]. There are some

clinical and in vitro studies which suggest that chondroitin and glucosamine may have synergistic effects [110, 111].

The role of glucosamine and chondroitin in the synthesis and composition of large proteoglycans, such as aggrecan, has led some researchers to question their use in patients with tendinopathy [87]. In reactive tendinopathy which is characterized by tendon swelling and aggrecan production [112, 113], an increase in proteoglycan synthesis may be detrimental. Although tendon pathologies around the hip are usually inflammatory in nature, it may be prudent to avoid the use of glucosamine in iliopsoas or gluteal tendinopathy especially in patients with concomitant reactive patellar or Achilles tendinopathy.

Omega-3 polyunsaturated fatty acids are known to have anti-inflammatory and antioxidant effects and have been used as dietary supplements in rheumatologic conditions. Polyunsaturated Fatty Acids (PUFAs) are also important components of dietary therapy in OA. Reactive oxygen species are generated in OA and have been shown to be involved in cartilage degradation [114, 115]. A recent study has demonstrated a synergistic effect between glucosamine and omega-3 fatty acids s in markedly reducing morning stiffness and pain in hip and knee pain OA [116]. The anti-inflammatory effects of omega-3 PUFAs have been shown in several studies [117–119] and they may be useful in inflammatory hip disease.

Vitamins and Minerals

There is limited clinical evidence demonstrating increased oxidative stress and reduced total antioxidant capacity in patients with OA [120]. Vitamin C and E are antioxidants which may stimulate collagen and proteoglycan synthesis [121, 122]. The role of Vitamin D in muscle strength is well established and a few small studies have noted that low levels of Vitamin D can increase progression of OA [123]. Selenium, Zinc, Manganese and Copper all have theoretical beneficial effects on proteoglycan synthesis and chondropathy but clinical evidence is currently limited and they cannot be strongly recommended.

Calcitonin

Calcitonin is produced by parafollicular C cells in the thyroid. It has a key role in calcium and phosphate regulation through increasing the effect of Parathyroid Hormone (PTH) and limiting calcium mobilization from bone. It is a weak inhibitor of osteoclasts and has also shown to inhibit MMP and block collagen degradation in chondrocytes [124]. In animal studies, calcitonin has been shown to be a disease modifying agent [125, 126]. A small clinical study has also noted improved functional scores in patients with knee OA using calcitonin [127]. It is recognized that subchondral bone changes and remodeling are involved in the initiation and progression of early OA. They are also usually a concomitant feature of acute intra-articular pathology. The precise nature of the interaction between articular cartilage and subchondral bone is not completely clear. It has been proposed that subchondral bone changes precede the development of cartilage degradation [128] and that bone produces a number of cytokines and eicosanoids that can induce these cartilage changes [129, 130]. Other studies suggest that subchondral bone changes occur secondary to cartilage degradation and subsequent microfissuring [131–133]. Regardless of the timing of these events, it would appear that the relationship between subchondral bone and cartilage is a key factor in both joint health and pathology [134]. With improvements in MRI scanning it is possible to observe bone marrow activity at subchondral sites [135–137]. While clinical studies are still awaited, treatments targeted at subchondral bone such as calcitonin and strontium may prove to be effective in improving subchondral bone homeostasis and subsequent intra-articular health.

Intra-articular Injections

Corticosteroids

Corticosteroids are strong anti-inflammatory agents that limit the inflammatory cascade through a reduction in vascular permeability and inhibition of leucocyte activation. They also inhibit inflammatory mediators such as prostaglandins, MMPs and interleukins [138–140]. MMPs are catabolic enzymes that are implicated in cartilage matrix degradation. Interleukins 1 and 6, amongst others, are associated with the synovitis that is present in inflammatory and degenerative joint disease and implicated in cartilage breakdown early in the pathological progression of OA [141, 142].

There are notable consequences of repeated intra-articular corticosteroid injections (IACSI). Corticosteroids inhibit fibroblasts and collagen production. Inhibition of osteoblastic and osteoclastic function limits bone remodeling. Cartilage breakdown has been reported following IACSI [143, 144] Cystic lesions and thinning of articular cartilage have been noted in weight bearing joints injected with corticosteroids. There is also a marked reduction in the elasticity of articular cartilage following IACS due to a degradation of the cartilage matrix [145–147]. Corticosteroid, particularly if combined with local anaesthetic is chondrocyte toxic [148–150]. With repeated injections and subsequent chondrocyte death, cartilage may be unable to regain its natural physical properties [151]. The injection of corticosteroids into the joints of young patients should therefore be considered carefully. An early return to running following steroid injection is more detrimental to cartilage. It may be preferable, to inject into the inflamed synovium rather than the joint fluid.

The most common and significant local adverse effect of IACSI is pericapsular or intracapsular calcification [139, 152]. These calcifications, composed of hydroxyapatite, may become inflamed and interfere with normal joint mechanics. Atrophy of the adjacent soft tissues is also a possibility. The psoas muscle lies directly anterior to the hip joint. Degeneration and atrophy of psoas fibres is certainly a possibility following injections into the hip joint. This can be minimized by guiding needle placement into the joint before attaching the syringe containing steroid and by avoiding injecting steroid during needle withdrawal. Avascular necrosis is a recognized complication, and usually follows several injections within a short time frame. Rapid destruction of the femoral head has been described in women with unilateral hip OA [153]. On microscopic assessment, total necrosis of the underlying trabecular bone is noted and it is recommended to consider avoidance of IACSI in severe chondral disease with underlying bone marrow edema and microfissuring into the subchondral bone. Joint infection is another serious complication and it is essential that an appropriate antiseptic and no-touch technique is performed. It is recommended that all injections of the hip are performed under radiographic guidance and after joint aspiration if an effusion is present.

A number of expert opinion studies have suggested a role for corticosteroids in therapeutic pain relief and in patients who are not candidates for total hip arthroplasty due to co-morbidity or young age [154, 155]. Clinical guidelines for the use of corticosteroids in OA are generally based on studies performed on knee OA patients [74, 75]. The evidence suggests some short term benefit in pain over the course of 4–6 weeks but this is not maintained and improvements in function and stiffness are minimal [156]. Predictors of improvement in some studies were the presence of synovitis and successful joint aspiration prior to injection [139, 157]. A prospective cohort study on hip OA has shown improvements in pain and stiffness at 6 and 12 weeks [158]. In young athletic patients with active synovitis, bursal inflammation, intact cartilage surfaces and normal subchondral bone requiring short term pain relief or reduction in inflammation after an acute incident (e.g. a labral injury), IACSI may be an appropriate option. It can provide short term relief in patients with FAI and associated peri-articular inflammation. This may be particularly useful during certain stages of an athletic season. However, if limited mobility rather than pain is the most significant presenting feature, short term improvement with intra-articular HA may be more appropriate, prior to surgical consideration.

Viscosupplementation

Viscosupplementation is the intra-articular injection of HA and was first presented as a therapeutic option over 20 years ago [159, 160]. The rationale for its use is based on the importance of HA in synovial joints. HA is a polysaccharide produced by chondrocytes and synovial cells [161] with a molecular weight of around 1×10^7 Da. It is the major constituent of synovial fluid and a component of the ECM of cartilage and the superficial synovial membrane. It has an important role in directly maintaining the structural and functional integrity of cartilage and indirectly in enabling normal joint mobility and effective shock absorption. The viscoelastic properties of HA can increase viscosity to provide lubrication during low shear movements and, alternatively, it may provide shock absorption by reducing viscosity and increasing elastic properties during high shear and faster movements [162, 163]. In OA the composition of synovial fluid changes with reductions in viscosity and elasticity [164] thereby increasing susceptibility to injury. The average molecular weight of HA in OA is also reduced to around 2×10^5 Da.

In addition, to its role in joint mobility and cartilage health, HA has an important function in maintaining joint homeostasis through modulation of the inflammatory response. HA can inhibit the release of arachadonic acid and Interleukin-1 (IL-1) [161, 165]. IL-1 is an pro-inflammatory cytokine which may induce cartilage degradation in culture models [166] and can be detected in inflamed synovial tissue [142]. IL-1 also stimulates the production of prostaglandin E2 (PGE2), a pro-inflammatory factor present in early OA [98].

HA preparations differ in their origin, molecular weight, biological characteristics and pharmacodynamics [167]. A number of proposed mechanisms exist for improved outcomes following intra-articular HA injections. HA injection may immediately reduce the activity of nociceptive afferents [168] and provide short term pain relief. Additionally, HA can modulate an anti-inflammatory effect through the reduction of PGE2, IL-1 and other inflammatory cytokines [165, 169]. This provides the rationale and supportive evidence [170, 171] for effective initial reduction in pain following intra-articular HA injection to a painful, inflamed joint with potential advantages for future cartilage preservation. However, a number of large meta-analysis and systemic reviews on knee OA have generally found delays in efficacy of around 4 weeks [172, 173].

HA injection is effective in stimulating synovial cells to synthesize endogenous HA [174–176]. This may be one potential mechanism for long term effects following injection since retention within the joint is only short-term [177]. Intra-articular retention may be increased to several weeks by the use of high molecular weight preparations. There is, however, conflicting evidence regarding clinical efficacy of high molecular weight HA (HMWHA) relative to low molecular weight HA (LMW HA). Some studies have identified that HMWHA is more effective in pain relief for knee OA [178], proposing that higher viscoelastic properties improve efficacy [160]. Other studies have found no difference in clinical efficacy between different molecular weight

HA injections in hip and knee OA [179, 180]. While HMWHA is more biologically active and similar to endogenous HA, there is some evidence that it may be less effective in penetrating the synovial ECM and reducing synovial inflammation [181]. A rational interpretation of the currently conflicting literature on the differences between various preparations may be that HMWHA is more appropriate for the functional restoration of joint mobility and that LMWHA more appropriate to target active synovitis.

The ability of intra-articular HA to directly preserve or improve cartilage structural integrity is currently unclear [182]. It has been reported that HA may improve chondrocyte density and articular cartilage reconstitution in vitro [183]. Cartilage preservation has been also identified in experimentally induced models of knee OA [184]. However, in clinical studies HA has not been shown to be a long term disease modifying agent [185].

The most comprehensive systematic review assessing intra-articular HA is a 2006 Cochrane review on knee OA [167]. There was support for a small reduction in pain over 3 months with maximal efficacy at 5–8 weeks following injection. In comparison, a recent meta-analysis identified greater pain relief following corticosteroid injection at 2 weeks but not at 4 weeks and greater benefit of HA at 8–26 weeks [186]. It is difficult to extrapolate the evidence for HA in the knee joint to the hip. The hip is clearly a very different joint biomechanically and anatomically. It should be recognized that in a significant number of patients there is a communication between the hip joint and iliopsoas bursa [187, 188] with the potential consequences of dispersal of injection from the joint.

In hip OA, there have been a number of recent studies of generally poor methodological quality. Migliore [189] performed a prospective non controlled study on the symptomatic effects of HA, using Hylan G-F20. They noted improvement in pain, functional scores and NSAID consumption. A number of other studies have shown similar improvements with a variety of HA preparations and no differences between preparations [179, 190]. A more recent study in 120 patients noted significant improvements in hip pain, mobility and function with 6 monthly HA injections [191]. The same study group also reports a 6-month RCT comparison of HA to Mepivicaine and noted a reduction in pain and improved function following HA injection [192]. While there have been no high quality long term studies of the efficacy of HA in hip OA the available evidence, albeit with a possible positive publication bias, does point to a role for HA in hip joint OA. From the previous discussion regarding the mechanism of action of HA it is possible to rationalize that this would be most effective in hip joint synovitis, early chondropathy and synovial restrictions in hip joint range. In early chondropathic states, the cartilage is likely to be more responsive to a normalized synovial fluid environment. It is likely to be less helpful in restriction due to bony impingement or in advanced chondropathic or subchondral bone disease.

Most studies investigating HA injection into the hip have commented on the importance of ultrasound or fluoroscopic guidance [193, 194]. The hip is a difficult joint to inject without guidance [195] and there is a high risk of adverse events. It is our recommendation not to inject intra-articular local anaesthetic during the intervention, due to the chondrotoxicity of local anaesthetic [150]. The anterior approach is recommended due to the large target area between the femoral head and neck within the anterior recess of the anterior capsule. This approach also prevents damage to the labrum or articular cartilage from the needle tip. If an effusion is present, this should be aspirated prior to HA injection to prevent dilution. Injection of HA into the hip joint appears to be safe and well tolerated and reported complications in the literature are rare [196, 197]. The most commonly reported side effect is a transient increase in minor localized pain, within the first week following injection [191, 198].

Platelet Rich Therapies

Growth factors (GF) are essential for the repair of injured tissue through the stimulation of various aspects of tissue healing. Platelets contain growth factors, such as insulin-like growth factor, transforming growth (TGF) and vascular endothelial growth factor (VEGF) in their α-granules. These are released at the site of injury and aid repair. This theory has led to the development of a variety of therapies based on delivering more platelets (and therefore GFs) to the site of injury. Platelet therapies, including platelet rich plasma (PRP), have been used for more than 20 years in the fields of dentistry and maxillofacial surgery and more recently in the treatment of musculoskeletal injury [199, 200]. In the context of intra-articular hip pathology, TGF β and platelet derived GF are known to have important roles in cartilage regeneration [201–203]. Laboratory studies have also shown the efficacy of platelet rich therapies in reduction of the inflammatory effects of IL-1 on human chondrocytes [204]. While these basic science studies are encouraging, there have been limited clinical studies in hip pathology to date. A number of pilot studies on patients with knee and hip OA [205–207] have shown encouraging results particularly in young patients with early chondropathic changes. Further research in this area is needed.

Radiofrequency Ablation

The hip joint capsule is innervated by sensory branches of the obturator, femoral and superior gluteal nerves [208]. The groin and medial thigh pain which is often present with hip

pathology is usually mediated by the articular branches of the obturator nerve. It is recognized that minor pathology in the groin can have numerous secondary effects on the function of other structures, particularly the iliopsoas and adductor musculature. In the young athlete with hip pain arising from an acute synovitis or FAI, the secondary effects on pelvic function may be more debilitating on athletic performance than the pathology itself. Assuming that the overall management plan can address the underlying biomechanical or structural problems, a short term pain relieving procedure may be particularly effective for athletic performance and minimizing secondary dysfunction.

Radiofrequency ablation can effectively block nociceptive conduction. Continuous radiofrequency (CRF) of sensory articular branches of the hip can provide long term relief of joint pain [209–211]. However, as CRF works through thermal coagulation of nerves, it may be complicated by neuroma formation [212]. Pulsed radiofrequency (PRF) has been described as an alternative technique which effectively blocks nociceptive signals through the application of an electric field but does not induce structural nerve injury [213–215]. It is also associated with less post-procedure neuro-inflammation and is not complicated by loss of sensation. There are a number of case studies which have produced promising effects in patients with hip and groin pain [215, 216]. There are insufficient high quality studies to draw conclusions about the efficacy of this intervention at present but if appropriately targeted, it appears promising for the future.

Conclusion

Hip and groin injuries in young adults are a common presentation in sports medicine and orthopaedic outpatient clinics. A small but significant proportion of these patients will have an intra-articular pathology which must be thoroughly investigated. Physicians should have a low threshold for early MRI in patients where the diagnosis is uncertain and when symptoms are refractory. An accurate diagnosis based on functional and anatomical hip abnormality is critical to directing appropriate treatment. FAI is being increasingly recognized as a cause of hip pain and restriction of movement in young adults and can potentially lead to chondrolabral damage and early hip OA. Although surgical intervention may well be needed in a proportion of patients with structural abnormalities around the hip, the role of medical treatment is well recognized, both as an adjuvant to surgery as well as to delay progression of irreversible joint damage and the subsequent need for early arthroplasty in relatively young patients.

In athletes with symptomatic labral tears, ‘early’ surgical intervention may be required.

Physical therapy may provide symptom relief in hip OA and is especially effective when supervised by trained specialists and incorporated into a formal training program. Obesity is a significant modifiable risk factor for hip OA and the role of leptin in obesity-related chondrocyte damage is well established. Supervised exercise appears to have a number of benefits in hip OA; it improves muscle strength, locomotor function and aids weight loss.

NSAIDs in addition to paracetamol are routinely recommended in OA especially if concomitant signs of inflammation are present. Glucosamine taken orally has been shown to reduce pain and improve knee joint function and may therefore also have a role in hip OA. Further clinical studies are needed to assess the effects of treatments targeted at subchondral bone such as calcitonin and strontium.

Intra-articular joint injections of corticosteroids, HA and platelet rich therapies have all been described in hip OA. Radiographic guidance during injection is recommended as routine. The effects of intra-articular corticosteroids and HA are short lived and their long term use is generally not recommended. The use of intra-articular platelet rich therapies and pulsed radiofrequency has shown promising results in reducing inflammation around the hip joint and this is a potential area for future research.

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