Can too much physical activity at a young age be counter-productive?

The negative impact of sports on the developing hip joint

A vast body of evidence exists advocating the many benefits of daily physical activity from an early age, from reducing cardiovascular disease and the risks associated with obesity, to enhancing metabolic fitness and promoting a healthy state of wellbeing. While there is a growing interest in enhancing the health and fitness of children and adolescents, the associated increased incidence of musculoskeletal injuries as a by-product is often overlooked; the distinction between competitive and recreational involvement in physical activities such as sports best highlights this.

The desire to achieve and be 'the best' is an innate quality and the potential rewards can often be a catalyst for the increased intensity of sporting involvement for the passionate athlete. With the increased profile of professional sports, involvement typically begins at an early age and young athletes are socialised to focus exclusively on their sporting success. With this sport-centred identity manifesting, it leads to an increase in competition, whether this is competing for a league title, scholarship for university or to emulate a sporting hero.

This high expectation for success can only be accomplished with an increase in the intensity of training, which can often result in misapplication of valid training sessions. The goal of many coaches and sports trainers to maximise the development of young athletes, with the aim of achieving athletic success at an adult age, is also a contributing factor. While it has been shown to take 10 years, or 10,000 hours of deliberate practice, to develop one's talent and become an 'expert', it is hard to imagine this comes without a physiological cost to the athlete.

In the past number of years, the incidence of sports-related hip injury and subsequent surgery have risen, owing in part to the increased intensity with which our young athletes are training and competing and from an improved awareness of the symptoms and signs of progressive hip injury.

The young musculoskeletal system is a dynamic structure, highly influenced by and responsive to environmental and external cues. Repetitive and high-impact forces associated with running, cutting, rotational and kicking movements makes the hip joint and open growth plates vulnerable to overuse injury at the critical developmental stages.

A mechanical obstruction

Femoro-acetabular impingement (FAI) is the most commonly recognised hip pathology in athletes, leading to chronic hip and groin pain. FAI develops when regular abnormal contact between the acetabular rim and the femoral head or neck results in progressive damage to the soft tissues of the hip joint, including the acetabular labrum (seal) and the articular cartilage. When these structures are compromised, patients will predominantly experience pain and stiffness, usually in the groin but which can also be localised to the front, side or the back of the hip.

Two morphological abnormalities of the hip joint have been identified as causal factors of FAI: a 'cam' deformity results from the progressive loss of femoral head sphericity; and a 'pincer' deformity exists with global or focal over-coverage of the femoral head by the acetabulum. In the majority of cases, both cam and pincer abnormalities co-exist as a 'mixed' impingement.

Signs and symptoms

The development of symptoms associated with hip impingement (FAI) is insidious, often progressing for many years undiagnosed. This is representative among the excess of 1,460 athletes who have undergone surgery for hip impingement at The Hip and Groin Clinic, Whitfield Clinic, Waterford. Over three-quarters (78 per cent) of athletes described the onset of their symptoms as gradual and present in excess of six months to more than five years before an accurate diagnosis was confirmed. Athletes' complaints of activity-related hip and groin stiffness, loss of speed and agility, persistent hamstring tightness and lower back discomfort are often put down to a heavy training session and are initially managed with regular physiotherapy, including stretching programmes, gluteal activation and core strengthening exercises.

When rudimentary movements such as starting/stopping, squatting, twisting, sprinting, or kicking a ball become limited and/or associated with pain, for some athletes this may begin to signal the presence of underlying hip impingement (FAI). For others, it is not until the severity of the symptoms force a withdrawal from sports altogether that the diagnosis is eventually considered.

While there is the obvious drawback of FAI impacting on performance, there is also the additional psychological impact that this reduced performance, limited involvement or subsequent withdrawal from their chosen sport has on the passionate athlete. High-performing GAA players, from senior club through to county level and athletes regularly competing at national level, appear particularly prone to symptoms of FAI; frustration and low mood is a common finding among such athletes referred for intervention.

Typical presentation

The average age of athletes undergoing surgery for FAI at our clinic is 27.5 years; seven-in-10 are competitively involved in one or more sporting types, with an average commencement of sport at eight years of age. At time of presentation, 80 per cent of athletes have previously attended two or more healthcare practitioners (including physiotherapists, GPs, chiropractors and sports doctors) for assessment and/or treatment.

Unfortunately, for the majority of these athletes the presence of the underlying hip impingement has either been misdiagnosed or greatly underestimated. By the time these athletes attend our clinic, 95 per cent have significant labral tearing, with the overwhelming majority having irreversible cartilage damage to the joint surface as a direct consequence of continued engagement in their chosen sports in the presence of symptomatic FAI. (The relative risk of significant articular cartilage damage approximately doubles in the presence of a labral lesion.) Early detection of symptomatic FAI and subsequent referral for definitive surgical treatment for deformity correction and labral repair is important for a successful return to sports and to delay, or possibly prevent, early hip joint degeneration.

Clinical examination classically reveals a reduction in the range of hip movement, with flexion, adduction and internal rotation most significantly reduced. A positive impingement test, as assessed by the reproduction of pain as the hip joint is moved through flexion, adduction and internal rotation (FADIR), is a highly sensitive physical examination test for FAI and is positive in more than 70 per cent of cases presenting at the clinic. Pain may also be experienced in 52 per cent of cases utilising the FABER test, where the hip is moved through flexion, abduction and external rotation.

Plain radiography and MR arthrogram are utilised to establish the nature and extent of bony impingement and confirm the presence of labral and articular cartilage pathology. Pincer deformity (over-coverage of the femoral head) is almost universally present and is the primary cause for hip impingement in all athletes treated, with a CAM deformity present in 62 per cent of cases (Figure 1). Labral tearing and articular cartilage damage is almost invariably evident at time of surgery (Figure 2).

Treatment options

As an initial intervention, conservative measures can be applied to help address the associated pain. Activity modification to reduce weight-bearing forces and reduction or cessation in intensity or frequency of high-impact sporting activities, as well as core strengthening and use of non-steroidal anti-inflammatory drugs (NSAIDs), are the mainstays of non-surgical treatment. While this measure may serve as a temporary relief, the success of conservative care for FAI is largely dependent on the patient's willingness to modify their sporting activity, ultimately becoming less active in impact sports. Where a morphological abnormality is present and is contributing to adverse symptoms, conservative measures do not alter the bony changes or rectify the pre-existing impingement. Structural bony deformities, as is the case with FAI, require a structural solution. Any significant delay in the surgical treatment of symptomatic FAI, either from misdiagnosis or as a result of lengthy conservative therapy, results in irreversible damage to the labrum and articular cartilage of the hip joint. This is associated with a poorer outcome from surgery, inability to return to competitive sports and the development of early osteoarthritis of the hip, eventually requiring hip replacement.

The success of surgery from an athlete's perspective is the ability to return to competitive sports without stiffness or pain; significant improvements in range of hip movements and enhanced athletic performance can be expected with meticulous surgical technique, removing all bony deformities with a repair of the labrum and the hip capsule. Over the past 10 years, our surgery has continued to evolve, culminating in the development of the 'Sports Hip Repair' technique, utilised specifically for the treatment of athletes with symptoms of FAI. Post-operative results from this unique procedure demonstrate clinically and statistically significant improvement in associated pain, function, physical activity level and quality of life, permitting early resumption of competitive sports.

With a 93 per cent success rate, this minimally-invasive technique permits accurate restoration of the hip anatomy and anatomical repair of injured intra-articular soft tissues. A structured and proactive postoperative rehabilitation programme is a requisite to achieve optimal outcome and a rapid return to sports.

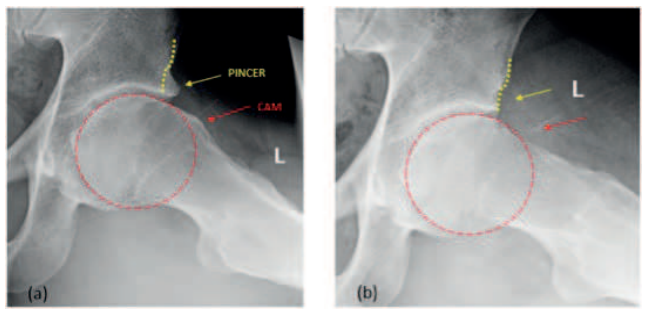

Figure 1: (a) Pre-operative x-ray of a 30-year-old professional athlete with significant pincer and cam deformities consistent with FAI; (b) post-operative x-ray following resection of bony abnormalities

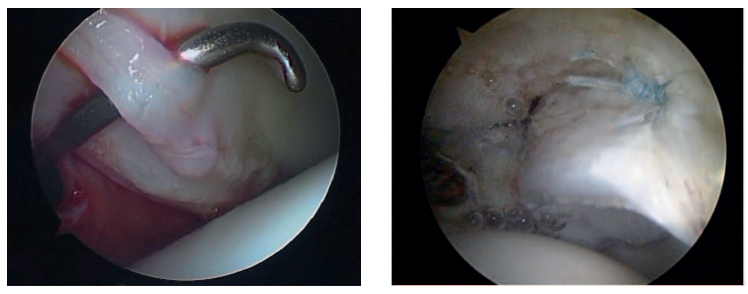

Figure 2: (a) labral tear; (b) labral repair

Early and accurate recognition

Athletes with early stages of FAI are more likely to have a better outcome than those with more extensive articular cartilage delamination or exposed bone. Our results clearly indicate that athletes who present with symptoms of FAI for longer than two years (although still expecting significant improvement in pain and function) will have a statistically significant decrease in overall success compared to those treated with symptoms for less than two years; a worrying statistic which is supported by other international researchers. This reinforces the importance of early diagnosis and appropriate surgical correction to avoid poorer outcome and irreversible damage, leading to osteoarthritis and total hip replacement.

Healthcare professionals, including GPs and physiotherapists, are becoming more familiar with the classical signs and symptoms of underlying hip pathology, leading to earlier diagnosis and intervention, with better outcomes for the athlete. With an increased awareness of typical warning signs and the acceptance that ongoing hip pain and stiffness are not normal by-products of a good training session, further injury, misdiagnosis and the potential for further degeneration can be avoided with the underlying problem surgically corrected.

The age-old, misleading 'no pain, no gain' ethos held by many budding young athletes needs to be re-evaluated. Training and coaching should be orientated to maintain player welfare, both psychosocial and physical, with holistic training regimes in place to prevent injury risk factors. The expectations of and training regimes executed at a senior level should not be transferred among younger emerging athletes. While there is an increased emphasis on competitive success in our young athletes, the associated high-intensity training to achieve and maintain this 'winning status' often compromises the very same.

The participation in high-level competitive impact sports is commonly associated with an increased risk of future osteoarthritis; the classic symptoms and clinical signs of FAI not only act as an important 'early warning' sign, but present an opportunity to surgically correct the deformities and repair damage structures to delay or prevent further deterioration and the requirement of premature hip replacement.

References available on request

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ADENURIC 80 mg and 120 mg film-coated tablets: Abbreviated Prescribing Information Please consult the Summary of Product Characteristics (SmPC) for full prescribing information. **Presentation:** Film-coated tablets containing 80 mg or 120 mg febuxostat. Also contains lactose monohydrate. **Use:** Treatment of chronic hyperuricaemia in conditions where urate deposition has already occurred (including a history, or presence of, tophus and/or gouty arthritis) in adults. **Dosage and administration:** Oral use with or without food. Recommended dose is 80 mg once daily. If serum uric acid is > 6 mg/dL (357 µmol/L) after 2-4 weeks, 120 mg once daily may be considered. *Older people:* No dose adjustment required. *Renal impairment:* No dosage adjustment necessary in patients with mild or moderate renal impairment. Efficacy and safety not fully evaluated in patients with severe renal impairment. *Hepatic impairment:* Recommended dosage in patients with mild hepatic impairment is 80 mg. Limited information available in patients with moderate hepatic impairment. Efficacy and safety has not been studied in patients with severe hepatic impairment. *Children and adolescents:* Safety and efficacy in children under 18 has not been established. *Organ transplant recipients:* No experience therefore not recommended. **Contra-indications:** Hypersensitivity to the active ingredient or to any of the excipients. **Warnings and precautions**: *Cardio-vascular disorders:* **Not recommended in patients with ischaemic heart disease or congestive heart failure.** *Product allergy/hypersensitivity:* Advise patients of signs/symptoms of allergic/hypersensitivity reactions and monitor closely for symptoms. Stop treatment immediately if serious reactions occur, including Stevens-Johnson syndrome, Toxic epidermal necrolysis and acute anaphylactic reaction/shock; do not re-start febuxostat at any time. Severe hypersensitivity reactions, including Drug Reaction with Eosinophilia and Systemic Symptoms (DRESS) associated with fever, haematological, renal or hepatic involvement in some cases. *Acute gouty attacks (gout flare):* Do not start treatment until an acute attack of gout has completely subsided. As with other urate lowering medicinal products, gout flares may occur during initiation of treatment. At treatment initiation flare prophylaxis for at least 6 months with an NSAID or colchicine is recommended. If a gout flare occurs during treatment, do not discontinue. Manage the gout flare concurrently as appropriate. Continuous treatment decreases frequency and intensity of gout flares. *Xanthine deposition:* As with other urate lowering medicinal products, in patients in whom the rate of urate formation is greatly increased (e.g. malignant disease and its treatment, Lesch-Nyhan syndrome), the absolute concentration of xanthine in urine could, in rare cases, rise sufficiently to allow deposition in the urinary tract. As there has been no experience of treating gout in these patients with febuxostat such use is not recommended. *Mercaptopurine/azathioprine:* Not recommended in patients concomitantly treated with mercaptopurine/azathioprine. Where combination cannot be avoided, monitor patients closely. Dose reduction for mercaptopurine/azathioprine is recommended. *Theophylline:* No pharmacokinetic interaction shown with febuxostat 80 mg, no data for 120 mg. *Liver disorders:* Liver function test is recommended prior to the initiation of therapy and periodically thereafter based on clinical judgement. *Thyroid disorders:* Caution in patients with alteration of thyroid function. *Lactose:* Contains lactose. Patients with rare hereditary problems of galactose intolerance, the Lapp lactase deficiency or glucose-galactose malabsorption should not take this medicine. **Interactions:** *Mercaptopurine/azathioprine:* On the basis of the mechanism of action of febuxostat on xanthine oxidase inhibition concomitant use is not recommended. No data is available regarding the safety of febuxostat during cytotoxic chemotherapy. *Rosiglitazone/CYP2C8 inhibitors:* No dosage adjustment required. *Theophylline:* No special caution advised for 80 mg febuxostat, no data available for 120 mg. *Naproxen and other inhibitors of glucuronidation:* Can be co-administered with naproxen with no dose adjustments necessary. *Inducers of glucuronidation:* Monitoring of serum uric acid is recommended 1-2 weeks after start of treatment with a potent inducer of glucuronidation. Cessation of treatment of an inducer might lead to increased plasma levels of febuxostat. *Colchicine/indometacin/hydrochlorothiazide/warfarin:* Can be co-administered with colchicine or indomethacin with no dose adjustments necessary. No dose adjustment necessary when administered with hydrochlorothiazide. No dose adjustment necessary for warfarin when administered with febuxostat. *Desipramine/CYP2D6 substrates:* Co administration with other CYP2D6 substrates is not expected to require any dose adjustment for those compounds. *Antacids:* May be taken without regard to antacid use. **Pregnancy and lactation:** Do not use during pregnancy or breast-feeding. Effect on fertility unknown. **Side-Effects:** *Clinical Studies and post-marketing experience:* Common (1-10%): Gout flares, headache, diarrhoea*, nausea, liver function test abnormalities*, rash, oedema. Uncommon (0.1–1%): Blood thyroid stimulating hormone increased, diabetes mellitus, hyperlipidemia, decrease appetite, weight increase, decreased libido, insomnia, dizziness, paraesthesia, hemiparesis, somnolence, altered taste, hypoaesthesia, hyposmia, atrial fibrillation, palpitations, ECG abnormal, hypertension, flushing, hot flush, dyspnoea, bronchitis, upper respiratory tract infection, cough, abdominal pain, abdominal distension, gastro-oesophageal reflux disease, vomiting, dry mouth, dyspepsia, constipation, frequent stools, flatulence, gastrointestinal discomfort, cholelithiasis, dermatitis, urticaria, pruritus, skin discolouration, skin lesion, petechiae, rash macular, rash maculopapular, rash papular, arthralgia, arthritis, myalgia, musculoskeletal pain, muscle weakness, muscle spasm, muscle tightness, bursitis, renal failure, nephrolithiasis, haematuria, pollakiuria, proteinuria, erectile dysfunction, fatigue, chest pain, chest discomfort, blood amylase increase, platelet count decrease, WBC decrease, lymphocyte count decrease, blood creatine increase, blood creatinine increase, haemoglobin decrease, blood urea increase, blood triglycerides increase, blood cholesterol increase, haematocritic decrease, blood lactate dehydrogenase increased, blood potassium increase. Rare (0.1-0.01%): Pancytopenia, thrombocytopenia, anaphylactic reaction**, drug hypersensitivity**, blurred vision, weight decrease, increase appetite, anorexia, nervousness, tinnitus, pancreatitis, mouth ulceration, hepatitis, jaundice**, liver injury**, Toxic epidermal necrolysis**, Stevens-Johnson Syndrome**, DRESS**, angioedema**, generalized rash (serious)**, erythema, exfoliative rash, rash follicular, rash vesicular, rash pustular, rash pruritic**, rash erythematous, rash morbillifom, alopecia, hyperhidrosis, rhabdomyolysis**, joint stiffness, musculoskeletal stiffness, tubulointerstitial nephritis**, micturition urgency, thirst, blood glucose increase, activated partial thromboplastin time prolonged, red blood cell count decrease, blood alkaline phosphatase increase. *Treatment-emergent non-infective diarrhoea and abnormal liver function tests in combined Phase III studies more frequent in patients concomitantly treated with colchicine. **Adverse reactions coming from post-marketing experience. Rare serious hypersensitivity reactions including Stevens-Johnson Syndrome and anaphylactic reaction/shock have occurred in post-marketing experience. Hypersensitivity reactions to febuxostat can be associated with the following symptoms: skin reactions characterised by infiltrated maculopapular eruption, generalised or exfoliative rashes, also skin lesions, facial oedema, fever, haematologic abnormalities such as thrombocytopenia, and single or multiple organ involvement (liver and kidney including tubulointerstitial nephritis). Gout flares commonly observed soon after treatment start and in first months. Frequency decreases after time. Gout flare prophylaxis is recommended. Please consult the SmPC for further information. **Pack sizes:** 80 mg and 120 mg tablets: 28 film-coated tablets. **Legal category:** POM **Marketing authorization number:** EU/1/08/447/001 & 003 **Marketing authorization holder:** Menarini International Operations Luxembourg S.A., Avenue de la Gare, L-1611 Luxembourg, Luxembourg **Marketed by:** A. Menarini Pharmaceuticals Ireland Ltd. Further information is available on request to A. Menarini Pharmaceuticals Ireland Ltd, 2nd Floor, Castlecourt, Monkstown Farm, Monkstown, Glenageary, Co. Dublin or may be found in the SmPC. **Last updated:** May 2015.

References: 1. Adenuric 80 mg SmPC. February 2014. **2.** Adenuric 120 mg SmPC. April 2015.

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