[Athletic Training]

The Running Athlete: Stress Fractures, Osteitis Pubis, and Snapping Hips

CEU

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Context: Pelvic stress fractures, osteitis pubis, and snapping hip syndrome account for a portion of the overuse injuries that can occur in the running athlete.

Evidence Acquisition: PubMed searches were performed for each entity using the following keywords: *snapping hip syndrome, coxa sultans, pelvic stress fracture*, and *osteitis pubis* from 2008 to 2013. Topic reviews, case reports, case series, and randomized trials were included for review.

Study Design: Clinical review.

Level of Evidence: Level 4.

Results: Collectively, 188 articles were identified. Of these, 58 were included in this review.

Conclusion: Based on the available evidence, the majority of these overuse injuries can be managed non-operatively. Primary treatment should include removal from offending activity, normalizing regional muscle strength/length imbalances and nutritional deficiencies, and mitigating training errors through proper education of the athlete and training staff.

Strength of Recommendation Taxonomy: C

Keywords: stress fracture; osteitis pubis; snapping hip; coxa sultans

PELVIC STRESS INJURIES

Stress fractures account for 15% of injuries suffered by running athletes, 4% of which occur about the pelvis.[21] They represent a spectrum of bone disorders from early injury (stress reaction) up to catastrophic failure of the bone (fracture).

Normal bone begins to fail when exposed to repeated bouts of excessive forces. These forces may result from a failure of the nearby muscle to absorb shear forces acting on the bone or occur as a result of direct muscle traction on the bone itself.[34] These overloading forces alter the normal bone homeostatic mechanism allowing osteoclastic function to predominate and incite the injury cascade.[21,32]

Several risk factors for developing a stress injury have been identified. Intrinsic risk factors include female sex, amenorrhea, low bone mineral density, low lean body mass, poor aerobic fitness at onset of an exercise regimen, gene valgum, and leg length discrepancy.[21,32,34] Extrinsic factors include rapid progression of a training program, running on an irregular or angulated surface, running and jumping sports, deteriorated footwear, smoking, and poor nutrition.[2,16,21,27,28,32-34]

Pelvic stress injuries can occur at the sacrum, inferior pubic rami, and femoral neck.[2,16,21,27,32-34,48] Stress fractures about the sacrum and inferior pubic rami are considered low risk. Femoral neck fractures account for less than 10% of all stress fractures and are considered high-risk injuries as they can displace with a high rate of morbidity (avascular necrosis and nonunion).[21,32,34,48]

Imaging

Kuhn et al[28] retrospectively reviewed plain film imaging of 54 patients treated for femoral neck stress fracture compared with patients with normal imaging findings. They found that 57% of those with femoral neck stress fracture had acetabular retroversion, compared with 31% of those without a fracture.[28,32]

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Table 1. Arendt classification and grading scale for stress fractures[4]

Low grade	Grade 1	+ STIR (short tau inversion recovery)
	Grade 2	+ STIR and T2-weighted image
High grade	Grade 3	+T1 and T2 images without fracture line
	Grade 4	+T1 and T2 images with a fracture line

Triple phase bone scans are highly sensitive to stress-related injuries; however, they lack specificity.[48] Shin et al[49] compared bone scan to MRI in the evaluation of hip pain in naval personnel in heavy endurance training programs. Bone scans had a sensitivity of 100% and positive predictive value and accuracy of 68% in the diagnosis of femoral neck stress injuries.[49] However, bone scans cannot distinguish between stress reaction/fracture and competing diagnoses such as avascular necrosis or bone cysts.

Magnetic resonance imaging is the preferred modality in the evaluation of stress injuries about the pelvis,[35] being highly sensitive and specific in the localization of stress injuries.[38,48] MRI findings in subjects with sacral stress injuries revealed intermediate intensity T1-weighted images, high signal changes on T2, and short tau inversion recovery.[2] The fracture line tends to start within the upper portions of the sacral alae and radiate toward the first or second sacral neural foramen in a vertical fashion parallel with the sacroiliac joint.[2] The signal changes normalize at 5 to 6 months after the onset of symptoms.[2]

A retrospective study assessed the return to sport time in 52 high-level athletes with a variety of lower extremity stress fractures (only 1 located in the femur).[13] Those with a low-risk fracture returned to sport at a mean 95 days versus 143 days for the high-risk fracture group ($P = 0.01$). Using MRI grading criteria (Table 1), the low-grade stress fracture group returned to sport at a mean of 61 days compared with 153 days for high-grade injuries ($P = 0.005$).[13] In a 5-year prospective study of collegiate track and field athletes, severe marrow and periosteal edema with or without a fracture line and a low bone mineral density correlated with a longer time to return to sport (23.6 ± 2.4 weeks).[35]

Management

Generally, management involves removal from the offending activity until symptoms have resolved.[17,32] During this time, it is important to maintain cardiovascular fitness, muscular strength, and neuromuscular control while not aggravating/stressing the injured region. Non/reduced weightbearing activity such as deep water jogging or cycling can often be performed to maintain physical fitness. A 4-week deep water running program maintained leg strength, Vo_2max, and running performance.[17]

Muscular strength and length imbalances along with impaired neuromuscular control identified during the initial physical examination should be corrected. Leg length discrepancies can be treated with heel lifts or custom shoe inserts.

Prevention of future stress injuries should be attempted through education of the patient and training/coaching staff. Proper progression of a training program is necessary to mitigate overloading of the musculoskeletal system. Rapid progression of training or sudden introduction of heavy interval training should be avoided. Cross training and regular exposure to different training surfaces may also reduce cyclic overloading of bones. Running shoes should be replaced after 300 to 500 miles of use.[32]

Other health maintenance measures such as smoking cessation and maintenance of ideal body weight should be addressed.[21,30,38] Inadequate caloric consumption for energy expenditure can adversely affect bone health, especially in female runners.[34,47,59] Low caloric intake whether related to disordered eating or inadvertent low caloric intake negatively affects estrogen balance and can induce amenorrhea or oligomenorrhea.[9,14,32] This in turn can lead to higher osteoclastic activity and increased risk of bone stress injury.[36] Cobb et al[8] assessed the effect of oral contraceptives on stress fracture development in recreational runners and found a nonsignificant trend toward reduced risk in the treatment group.[21,32] This study was underpowered because of noncompliance within the treatment group and restoration of normal menses in some of the control group subjects.[8,32]

The effect of calcium and vitamin D intake on bone health in female athletes has also been assessed.[2,16,27,32-34,37] Each additional cup of skim milk consumed daily in a female athlete reduced the incidence of stress fracture by 62%.[37,48] In female naval basic training recruits, a 20% reduction in the incidence of stress fractures was found with the consumption of 2000 mg calcium and 800 IU of vitamin D daily for 8 weeks.[28,29] Single doses of no more than 500 mg of calcium led to improved alimentary absorption, so multiday dosing may be more effective.[49,53]

Femoral neck stress fractures are managed differently, given the risk of developing a displaced fracture. Currently, tension side injuries are managed with hardware fixation of the fracture (Figure 1).[32,35] Management of compression-sided stress fractures that involve more than 50% of the femoral neck width are treated with surgical fixation.[38,48] Those with a fracture line less than 50% of the femoral neck width can be managed nonoperatively.[2,48]

OSTEITIS PUBIS

Athletes that develop osteitis pubis typically participate in sports producing twisting/shearing forces about the pelvis such as soccer, rugby, ice hockey, running, American football,

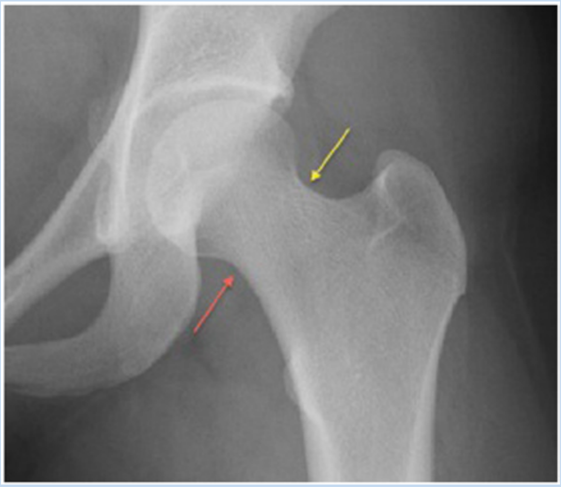

Figure 1. Femoral neck stress fracture zones. Anteroposterior radiograph of normal hip. Yellow arrow, tension side; red arrow, compression side.

basketball, and tennis.[2,5,6] Early diagnosis and management is important as this injury can lead to a high degree of disability and time away from the sport.

Evaluation

Plain film imaging may reveal degenerative changes such as subchondral cysts and osteophytes suggesting prior injury. Bone scan images are sensitive to stress injuries, but their lack of specificity limits the assessment of competing diagnoses. MRI correlates well with stress injury about the pubic symphysis, demonstrated by edema within the adjacent pubic bone.[2,57]

Management

Rehabilitation

A systematic review found patients treated with a progressive rehabilitation program targeting pelvic girdle strengthening and stretching exercises along with a graduated running program were able to return to activity within 4 to 13 weeks.[6,13]

A retrospective review of professional soccer players diagnosed with osteitis pubis over an 8-year period found 35 of 44 players had stage I to III severity (Table 2).[44] Treatment consisted of 14 days of nonsteroidal anti-inflammatory drugs and modalities (electrical stimulation, ultrasound, laser, and cryomassage) along with a progressive strength/conditioning program. Initial exercises included strengthening/stretching and non/reduced weightbearing aerobic exercises. Once tolerated, jogging was initiated and followed by kicking drills in a progressive fashion. Using this strategy, the mean time to return to play was 3 weeks for stage I, 6.8 weeks for stage II, and 10 weeks for stage III.[44]

Holmich et al[20] compared active physical therapy to passive physical therapy (modalities plus stretching) in patients with chronic groin pain attributed to either osteitis pubis or insertional adductor tendinopathy.[20] Subjects were randomized to either an active or passive physical therapy group. The active physical therapy group performed progressive strengthening exercises targeting hip adductors and abdominal muscles along with neuromuscular control and endurance exercises for the core and pelvic girdle muscles. They also stretched the lumbar spine and pelvic girdle muscles with exception of the hip adductors. The passive physical therapy group received modalities (ultrasound, laser, electrical stimulation, and transverse friction massage) in addition to stretching of lumbar spine and pelvic girdle muscles. Both groups performed aerobic conditioning on an exercise cycle initially with gradual introduction of jogging activity after 6 weeks of treatment. Treatment lasted for 8 to 12 weeks depending on symptom resolution with treatment or jogging. Twenty-three actively trained subjects versus 4 subjects with passive treatment were able to return to sport without pain. This trend of improved outcomes for the active treatment group continued when subjects were recontacted 8 to 12 years later in a follow-up study.[19]

In addition to resolving symptoms, 2 functional tasks may help determine when patients can return to activity/sport.[61] In a small case series, performing 5 minutes of simulated skating on a 3-m slide board and 3 sets of 12 repetitions of adductor strengthening using 6 kg of resistance determined when the athlete could return to competitive soccer without recurrence of symptoms.[61]

Injections

Corticosteroid and prolotherapy injections can be used in cases refractory to relative rest and rehabilitation.[6] A systematic review reported that 58.6% of patients with an average of 12.9 weeks of pain were able to fully return to their sport 8 weeks after corticosteroid injection.[6] In a case series, a prolotherapy solution consisting of 12.5% dextrose and 0.5% lidocaine was used in 2 sets of injections given 1 month apart targeting tender areas about the thigh adductors, abdominal tendon insertion, and pubic symphysis.[54] Twenty-two of the 24 (91.7%) athletes had significant reduction in pain and were able to return to full unrestricted activity in an average of 9 weeks.[54]

Surgical Intervention

Operative management of recalcitrant cases may include pubic symphysis debridement curettage, pubic symphysis fusion, or preperitoneal retropubic polypropylene mesh placement.[6,18,41] There are no trials comparing one technique over another. While all of the approaches led to reduced pain in most patients, the mesh placement has had fewer complications and faster return to play (mean of 7.2 weeks vs 6 months for the curettage or fusion techniques).[6]

SNAPPING HIP SYNDROME

Snapping hip syndrome, coxa sultans, refers to a painful popping sensation that occurs with movement about the pelvic girdle. It occurs in 5% to 10% of the general population, but the

Table 2. Osteitis pubis severity, re-created from description by Rodriguez et al[44]

Stage	Location of Pain	Pain Level Worsens
I	Unilateral kicking leg	After training
II	Bilateral inguinal pain	After training
III	Bilateral inguinal and lower abdominal muscle pain	Kicking, sprinting, change in direction, longer walks, and transition from sit to stand
		Unable to play sport
IV	Bilateral inguinal, lower abdominal, and lower back pain	Defecation, sneezing, walking
		Unable to perform activities of daily living (ADLs)

true incidence in running athletes is not known.[31] The location of the snapping has been broadly divided between intra- and extra-articular domains, with the extra-articular version occurring more frequently.[31]

Intra-articular

Intra-articular causes include loose fragments, fractures, synovial chondromatosis, and femoral acetabular impingement, which may lead to labral tears and articular cartilage injury.[60] A recent meta-analysis assessing conventional MRI versus MR arthrography in the diagnosis of labral tears found no significant differences in sensitivity or specificity when comparing MR field strengths.[51] The pooled analysis of MRI studies using a 1.5-T scanner revealed a sensitivity of 70% and specificity of 82%. Pooled data on MR arthrograms on a 1.5-T scanner revealed a sensitivity of 83% and specificity of 57%.[51] The prevalence of labral tears in asymptomatic volunteers using a 1.5-T MR scanner without arthrography and 2 blinded radiologists assessing 42 hips (age range, 27-43 years) found tears in 34 of 42 (80.9%) and 36 of 42 (85.7%), respectively.[45] Similarly, a prospective assessment of 45 asymptomatic volunteers (age range, 15-66 years) with a 3.0-T scanner found abnormalities in 73%; 69% had a labral tear.[43]

Extra-articular

Internal/Medial

Extra-articular internal or medial snapping involves the iliopsoas tendon abruptly striking the superior pubic ramus/iliopectineal eminence, anterior hip joint, or lesser trochanter.[31,60] Dynamic ultrasonography of 18 snapping hips demonstrated snapping of the iliopsoas tendon while the patients reproduce their symptoms by moving the hip from a flexed, abducted, externally rotated position back down to a neutral hip position through extension, adduction, and internal rotation.[11] Fourteen of 18 patients had snapping as the iliopsoas tendon moved from anterolateral to posteromedial positions caused by sudden contact of the tendon against the superior pubic ramus. Three patients had snapping of a bifid iliopsoas tendon, where the medial tendon would migrate back and forth over the lateral tendon. One tendon was seen snapping over a paralabral cyst.[11]

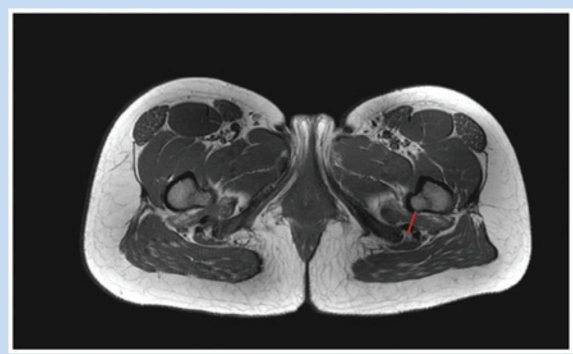

Figure 2. Ischiofemoral space. Hip magnetic resonance image, axial T1-weighted image. Red line, measurement of the ischiofemoral space; top, anterior; bottom, posterior; right, left; left, right.

Lateral

Lateral snapping occurs when the iliotibial band and/or the anterior leading edge of the gluteus maximus muscle snaps over the greater trochanter of the femur.[7] In a case series, either the iliotibial band (5 of 7 cases) or the leading edge of the gluteus maximus (2 of 7 cases) was sonographically visualized abruptly moving anteriorly over the lateral facet of the greater trochanter when the patient flexed or extended the adducted thigh.[7]

Posterior

Posterior snapping can occur with ischiofemoral impingement (IFI).[3,52,55,56] The true etiology of this rare entity is unknown. The snapping may arise as a result of a narrowed ischiofemoral space, the distance measured between the lateral border of the ischial tuberosity and medial border of the lesser trochanter on axial T1-weighted MRIs (Figure 2).[55] Narrowing of the ischiofemoral space potentially allows the lesser trochanter to strike against the ischial tuberosity with activity, especially during maneuvers that involve hip extension, adduction, and external rotation.[52] This may lead to injury of the quadratus femoris muscle that spans this region. A retrospective review of patients with ischiofemoral impingement reported a sensitivity/specificity of 83% and 82%, respectively, when comparing an ischiofemoral space measuring 13 ± 5 mm in symptomatic patients versus 23 ± 8 mm normally.[55]

Two separate case reports describe snapping of the proximal hamstring tendons (biceps femoris and semimembranosus) over the ischial tuberosity.[42,46] Both involve partial tears of the respective tendon snapping across the ischial tuberosity during extremes of hip flexion.[42,46]

Management

Rehabilitation Treatment Strategies

Nonoperative management for all forms of snapping hip syndrome is initially employed. Activities that initiate or aggravate the painful snapping should be modified or avoided altogether. Exercise or sport training errors should be investigated and corrected to prevent further injury.[40] Cross training exercises can be used to maintain aerobic condition and strength during the recovery period. Corticosteroid injections can be used to identify the source of the pain as well as reduce pain if the patient is unable to tolerate the exercise prescription.[1,25] Intra-articular injections are performed with either fluoroscopic or ultrasound guidance. Extra-articular injections for a snapping iliopsoas tendon or quadratus femoris muscle injections for ischiofemoral impingement are generally performed with fluoroscopy, ultrasound, or computed tomography guidance.[25,55] Trochanteric bursa injections for lateral snapping can be performed with or without guidance. Fluoroscopically guided compared with non-guided injections are more accurate; however, the clinical effectiveness with regard to pain reduction did not differ between methods, even at 3 months from the initial injection.[9,10]

Intra-articular Snapping

A systematic review of nonoperative treatment of femoroacetabular impingement (potential cause of internal snapping in a runner) found that the majority recommend activity modification (84%), conservative treatment (65%), nonsteroidal anti-inflammatory drugs (75%), and physical therapy (48%) without supportive evidence.[58]

Extra-articular Snapping

Internal (iliopsoas tendon). Stretching of the iliopsoas muscle should be performed along with modification of activity until symptoms begin to resolve.[26] Strength and neuromuscular control exercises should focus on the pelvic girdle and lower extremity muscles while limiting activation of the iliopsoas.[39]

External/lateral. Sixteen percent of those with external/lateral snapping have weakness during eccentric contraction of the gluteus medius when compared with control subjects.[24] Using surface electromyography, the gluteus medius was most consistently activated with side lying hip abduction.[12] Eccentric training of the gluteus medius from the side lying position may be helpful. Stretching of the iliotibial band, gluteus maximus, and tensor fascia muscle should also be performed.

External/posterior. There are no studies of a nonoperative treatment measure for ischiofemoral impingement.[52] However, knowledge of the mechanism of the injury/snapping can guide treatment. Widening the gait pattern may reduce the impingement between the lesser trochanter and ischial tuberosity. Prevention of hip adduction during the stance phase of walking/running by progressive strengthening of the hip abductors and stretching of the adductors and hip external rotators may relieve symptoms.

Operative Management

As with most musculoskeletal injuries, surgery is generally reserved for cases that have not responded to 3 to 6 months of standard nonoperative treatment. Open and endoscopic procedures are available for intra-articular and extra-articular internal and external snapping hip syndromes.[3,16,22,23,50,52] A review of open and endoscopic release of the iliopsoas tendon and iliotibial band found evidence that the endoscopic approach may more completely resolve symptoms.[22] Resolution of ischiofemoral impingement may require surgical debulking of the ischiofemoral space and removal of the lesser trochanter.[3,52]

SUMMARY

Stress fractures, osteitis pubis, and snapping hip syndrome are overuse injuries that can occur in the running athlete. Stress fractures represent a continuum of stress injury to bone from simple stress reaction through fracture. Osteitis pubis occurs as a result of repetitive shear stress across the pubic symphysis and adjacent abdominal and adductor tendons. Snapping hip syndrome can occur as a result of hip joint pathology or involve the iliopsoas and proximal hamstring tendons, tensor fascia or gluteus maximus muscles, or iliotibial band snapping against adjacent bone or soft tissue structures. Identifying potential etiologies such as muscle length/strength imbalances or training errors are key to developing an effective nonoperative treatment plan and prevention of future injury. On a rare occasion, surgery may be needed to help manage these disorders.

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