Diagnosing Running Injuries: A Primer for Physiatrists

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Clinical Vignette

SD is a 31-year-old female runner without significant past medical history who presented with four weeks of bilateral “shin pain,” left more severe than right. She had been a recreational runner (15 miles per week) for about five years, but in the three months prior to presentation, she increased her running frequency and intensity to 40+ miles per week in preparation for her first marathon. She described the pain as “achy” and noted it began to emerge after running for about 15 minutes. She denied any trauma or specific inciting event. She initially treated herself with ice and ibuprofen after each run, as well as orthotics prescribed by her chiropractor. She saw her primary care physician (PCP) within a week of the pain onset, and he diagnosed “shin splints” after x-rays were negative. She completed 10 days of relative rest and began a stretching program. However, her symptoms returned once she resumed her previous level of activity. Physical exam was unremarkable, without visible deformity, redness, or swelling. She was not tender to palpation along the anterior lower leg or in any compartments. Bilateral knee and ankle exams were unremarkable. She demonstrated intact strength, sensation, and reflexes. She became frustrated because of the pain and sought further evaluation from a physical medicine and rehabilitation specialist so that she could resume training for her marathon.
Defining the Problem

Recreational running is a very popular sport in the United States. Current estimates reveal that 20 to 30 percent of the American population participates in running. In general, the average runner is getting older and heavier. The average age of participants in road races is 40. Many runners are inexperienced and new to the sport, and approximately half of all runners will sustain an injury during a given year. Consequently, there is a high likelihood of seeing a running-related injury in an outpatient musculoskeletal medicine clinic. Recent review articles have described the incidence and prevalence of several running-related injuries, most of which occur in the lower extremities.1,2

A comprehensive evaluation of the injured runner includes assessment of the runner’s weekly mileage, recent changes in mileage, footwear, training regimen (hills, sprints, cross-training), running injury history, and running goals. Training errors include increasing weekly mileage too rapidly, lack of variation in terrain, running on the same side of banked roads, or wearing improper footwear. Any of these factors may predispose runners to an injury. The entire lower extremity kinetic chain should be evaluated, as deviations in lower extremity alignment have been associated with different injuries.3

Kinesiology of Running

Gait mechanics may be impacted by intrinsic factors, such as leg length discrepancy, lower extremity ligamentous pathology, or alignment issues.4 During running, the limb spends >60% in swing phase and <40% in stance phase, and there is no double stance phase but rather a float phase.4,5

Joint position and individual muscle group activation in the legs while running have been studied in detail by Mann6 and more recently by Dicharry,5 and this biomechanical data can aid physicians in assessing the gait of runners. It is important for physicians to remember that major joints are part of a coupled kinetic chain, and that poor mechanics at one location can lead to injury or pain at another.

HIP

Tightness of the hamstrings may prevent appropriate hip flexion. This forces the pelvis to tilt further anteriorly and, subsequently, places greater strain on the lumbar spine. Conversely, altered pelvic alignment may place increased strain on the hip flexors and hamstrings. The running hip also differs from the walking hip in the initiation of hip extension prior to initial contact in running. This serves to bring the center of mass under the body to maintain speed.1

KNEE

In running, the knee typically does not achieve full extension at any point in the gait cycle. A running knee reaches flexion greater than 90 degrees in swing phase (up to 130 degrees in sprinting) but extends only to 25 degrees of flexion at contact and 45 degrees at midstance.5 With increased speed, the quadriceps must exert more force to maintain stability in flexed single leg stance. The concentric and eccentric demands on the quadriceps make strength and endurance critical to maintaining appropriate biomechanics of the hip and knee joint in running.3

ANKLE

Compared to walking, a running ankle moves through an increased range of motion. Whereas a walking ankle will plantarflex to achieve foot flat, the running ankle remains dorsiflexed to allow forward tibial lean to load the limb in stance phase and propel the runner’s center of gravity anteriorly. It is important to maintain flexibility of the gastroc-soleus complex to facilitate this movement. It is notable, however, that as speed increases to a sprint, dorsiflexion decreases due to the propensity of sprinters to run on their toes rather than achieve full foot flat.5,6

Differential Diagnosis of Running Injuries

Achilles Tendon Injuries

The Achilles tendon originates as the distal terminus of the gastrocnemius and soleus muscles. The tendon inserts onto the calcaneus. The tendon proper is surrounded by the paratenon, a membrane that originates from the fascia of the deep posterior compartment of the leg and supplies most of the blood to the tendon.7

Achilles tendinopathy most commonly occurs at either the relatively poorly vascularized area 2 to 6 cm proximal to the insertion or at the insertion proper. Maximal forces on the tendon during running approach 6 to 8 times the athlete’s body weight, and peak at late and terminal stance. Risk factors for tendinopathy include age, male gender, foot pronation, and higher mileage.8 Injury is thought to develop from shear stress and microtrauma to the tendon during the running gait. The repetitive pronation-supination motion of the hindfoot creates shear force across the tendon that tends to be greatest on the medial side. Histology studies have shown that most patients exhibit degenerative changes rather than acute inflammation, meaning that the problem is truly more tendinosis than tendonitis.9
On physical exam, the affected area may be tender to palpation. There may be associated thickening and tendon nodularity, particularly on the medial side. In cases of acute inflammation of the paratenon, there is crepitus with ankle movement and diffuse swelling along the length of the tendon. The differential diagnosis for Achilles tendinopathy includes retrocalcaneal bursitis (distinguished by pain on palpation of the bursa between the Achilles tendon and the calcaneus), Haglund’s deformity (prominent superior lateral aspect of the calcaneus frequently associated with retrocalcaneal bursitis), and posterior ankle impingement, which can coexist with Achilles tendon problems. Full rupture of the tendon is usually a straightforward clinical diagnosis, but partial tears cannot be ruled out by history and physical examination alone. Imaging modalities used to evaluate the tendon are ultrasound and MRI.

Conservative management of Achilles tendinopathy includes correction of training errors, relative rest, and addition of low-impact cross training. In cases of acute inflammation, non-steroidal anti-inflammatories (NSAIDs) or oral steroids play a role. Corticosteroid injection is not recommended due to increased risk of progression to tendon rupture. A heel pad or heel cup is generally of limited benefit but can be considered. The rehabilitation program should emphasize gastrocnemius and soleus flexibility, followed by progression to eccentric exercises. There are some data to support the use of nitroglycerin patches and platelet-rich plasma (PRP) injections. Surgery is recommended if conservative measures fail to control pain or restore function. When standard treatment fails, tenotomy and debridement are the most common surgical procedures.

**Patellofemoral Pain Syndrome (PFPS)**

PFPS is the most common cause of knee pain in runners. There is a female predominance in PFPS, and the mechanism of injury is thought to be increased stress at the patellofemoral compartment of the knee joint, leading to articular cartilage damage. Non-modifiable biomechanical risk factors associated with PFPS include increased Q angle (formed by a line drawn from the ASIS to the central patella and a second line drawn from the central patella to the tibial tubercle), valgus knee alignment or bony dysplasia of the patella, or the trochlea. More dynamic and modifiable factors include foot overpronation, tight lateral retinaculum, poor flexibility, imbalanced lateral and medial quadriceps strength, or weak hip external rotators and abductors.

Patients with PFPS complain of insidious onset anterior knee pain that is usually present under or around the patella. Special tests for PFPS include the patellar grind test performed by applying axial compression of the patella leading to pain +/- crepitus during quadriceps activation. In the seated position, lateral maltracking may be seen with active knee extension (positive “J” sign). Patellar motion and position are noted with patellar glides, tilt, rotation, and anterior-posterior position. This is assessed in the supine position with the knee in extension and then passively flexed to approximately 20-30 degrees. Patient apprehension or pain during mobility assessment may suggest an unstable patella. Imaging is not usually indicated in PFPS unless the patient has a history of trauma or is not improved with conservative treatment. Although much literature is dedicated to treatment of this condition, at present there is no high-quality evidence to predict which patients will benefit from a particular intervention. Acute management includes activity modification and control of pain and inflammation. Correction of training errors is essential. For those with foot alignment issues, such as pes planus, orthotics are indicated. A brace should be considered for patients with patellar maltracking, although McConnell taping may be effective in cases when the brace is not. This involves placing a strong, rigid tape on and around the patella to keep it aligned in the trochlear groove. It has been shown to improve tracking, reduce pain, and even promote proper quadriceps firing patterns. Exercise programs should focus on strengthening the quadriceps, hip external rotators, and hip abductors, and progress from open to closed chain and eccentric strengthening. In addition, stretching of the quadriceps, hamstrings, and gastrocnemius should be considered based on the examination findings. Iliotibial band stretching also should be considered when Ober’s test is positive.

**Plantar Fasciitis**

The plantar fascia originates at the medial tuberosity of the calcaneus and consists of lateral, medial, and central bands. At the level of the metatarsal heads, the central band divides into slips that insert on the proximal phalanx of each toe. During static standing, the plantar fascia supports the arch of the foot. During the latter part of the stance phase of gait, dorsiflexion of the toes places tension on the plantar fascia at the metatarsal heads, shortening it and aiding in foot supination. This process is known as the windlass mechanism.

Plantar fasciitis accounts for approximately 10% of all running injuries. The most common site of abnormality is around the origin of the central band at the calcaneal medial tuberosity. The most common cause is repetitive microtrauma leading to mechanical overload at the enthesis. This leads to inflammation that can progress to eventual scarring and shortening of the fascia. Risk factors have been identified and include obesity, pes planus, pes cavus, tight Achilles tendon, and training errors.
Patients present to the physician with inferior heel pain. A common complaint is pain that is worst with the first few steps in the morning and gradually improves with activity. Pain is usually worse with barefoot walking. In severe cases, rest pain can be seen. Passive dorsiflexion of the toes may reproduce pain. While the diagnosis of plantar fasciitis is generally straightforward, other pathology should be considered in the differential diagnosis. This includes rupture of the plantar fascia, calcaneal stress fracture, bony pain due to heel spur or fat pad atrophy, seronegative spondyloarthropathy, radiculopathy, or medial tarsal tunnel syndrome. Diagnosis is typically made on the basis of history and physical examination alone. Rarely, imaging or other testing may be indicated but is usually reserved for patients who present atypically or do not respond to initial treatments.

First line treatment should include stretching of the plantar fascia and gastroc-soleus complex, as well as night splinting. Over-the-counter arch supports and heel cups have been shown to be effective. Patients should be instructed to avoid excessive barefoot walking and flat shoes. Home therapy programs may include ice massage, strengthening foot intrinsic muscles with towel exercises, and ankle strengthening. Initially, the training regimen should be adjusted and high impact activities scaled back until pain improves. Corticosteroid injection is controversial — in some studies, only those patients who undergo injection go on to rupture the plantar fascia. For patients who do not respond to first line treatment, other options include iontophoresis or Graston Technique, which uses specialized instruments to mobilize soft tissue. Steroid injection or botulinum toxin injection is also considered for non-responders. With some combination of these treatments, 95% of patients’ symptoms will resolve within one year. For those who do not respond, third-line treatments include extracorporeal shock wave therapy or surgical plantar fasciotomy. There is some emerging evidence for PRP in the treatment of chronic plantar fasciopathy.15

Hamstring Strain

Hamstring injuries are more common in sprinters than longer distance runners. In sprinters, this is the most common muscular injury. The long head of the biceps femoris is the most commonly injured hamstring muscle. The entire muscle group is at highest risk for injury during terminal swing phase and terminal stance phase, with a high eccentric load placed on the muscles.16,17

Hamstring injuries are classified traditionally as grades 1-3, with grade 1 representing overstretching but mostly maintained structural integrity. Grade 2 has fiber disruption less than half the tendon or muscle width, and grade 3 indicates complete muscle tear. A more recent MRI-based scoring system graded injuries by assigning points in several categories: number of muscles involved, location, insertion involvement, percentage of cross-sectional injury, retraction, and length in long axis.16 The grading system has been shown to be an accurate predictor of return to play for National Football League (NFL) players.18

Treatment includes activity modification and an exercise program to address strength and flexibility deficits. Sherry et al describe a hamstring program with progressive agility and trunk stabilization exercises that has been shown to reduce re-injury rates.19 A common error in early rehabilitation is a focus on hamstring stretching when, in fact, the muscle should be protected and worked at shorter lengths. Goals of rehabilitation should include strengthening the injured hamstrings to within 5-10% of the contralateral side and increasing the hamstring-to-quadriceps strength ratio. Core strengthening also should be addressed. Intramuscular dexamethasone injection was studied retrospectively in the NFL and can be safe and effective in controlling pain and inflammation if performed in the first 48 hours.20 More recently, PRP has been investigated in the treatment of hamstring injuries.16 Surgery is usually reserved for cases of proximal avulsion or distal tendon rupture.

ITB Syndrome (ITBS)

The iliobial band (ITB) resists hip adduction and internal rotation, as well as anterior tibial translation and internal rotation during gait.21 The ITB has been shown to translate posteriorly over the lateral femoral epicondyle between 0 and 45 degrees of knee flexion under ultrasound examination.22 Runners will complain of lateral knee pain during a run. In severe cases, there can be pain with walking or even rest pain. Physical exam typically reveals tenderness to palpation of the distal ITB at the lateral femoral epicondyle. The Noble compression test assesses for a painful arc of motion as the knee moves into extension. Ober’s test can reveal contracture/tightness of the ITB. The differential diagnosis includes a tight lateral retinaculum, lateral meniscal tear, lateral collateral ligament injury, popliteal tendonopathy, and biceps femoris tendonopathy. Imaging is rarely required in the workup for ITBS.

Many predisposing risk factors have been investigated.21 Training errors remain the most common risk factor for injury. Downhill running has been associated with ITBS.21 Biomechanically, increased femoral external rotation and greater hip adduction angles in stance phase have been shown in female runners who go on to develop ITBS compared to controls.24 Muscle imbalances also may play a role, as hip
abduction and internal rotation weakness are associated with poor treatment outcomes. Rarely, leg length discrepancy can predispose to ITBS on the shorter limb.

Fredericson and Wolf have described a comprehensive rehabilitation program for ITBS in runners. Stretching of the ITB and release of myofascial restrictions is addressed early. Foam rolling involves using a foam cylinder to apply pressure to various tender points and is a popular method of soft tissue mobilization but has little literature to support its use. Hip stabilization is targeted through strengthening of hip abductors and internal rotators. Most athletes are able to return to running without pain after about six weeks. Faster-paced running may be initially recommended, as it is actually less stressful on the ITB with more acute angles of knee flexion on initial contact. Distance should gradually be increased based on symptoms.

### Stress Fractures

The incidence of stress fracture in the general athletic population is less than 1%, but it can be as high as 15% in runners, with a higher incidence in women and Caucasians. The tibia (49.1%), tarsals (25.3%), and metatarsals (8.8%) are most commonly affected, although runners may also suffer pelvic, femoral shaft, and femoral neck stress fractures. Stress fractures are due to an imbalance of absorption and resorption activity and frequently occur in the relative watershed regions of the affected bones. This imbalance is theorized to result from increased strain on bone due to either an abnormal force, such as occurs with muscle fatigue or poor alignment, or a normal force on an abnormal bone. Stress fractures may be categorized as low or high risk for complications (see Table 1). High-risk fractures are at greater risk of delayed union, nonunion, or even AVN, and are generally surgically managed.

It is critical to evaluate patients for additional health factors that may contribute to poor bone integrity — especially those individuals with multiple or recurrent stress fractures. Patients should be screened for a history of metabolic disorders, autoimmune disorders, alcohol use, smoking, steroid use, low calcium and vitamin D intake, eating disorders or other causes for poor nutrition, as all of these may negatively impact bone health. A major risk factor in female runners is amenorrhea or oligomenorrhea, conditions that may be associated with disordered eating, which can further impact the formation and maintenance of healthy bone.

**TABLE 1: Classification of Lower Extremity Stress Fractures (Adapted)**

<table>
<thead>
<tr>
<th>Low Risk</th>
<th>High Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pelvis</td>
<td>Femoral Neck</td>
</tr>
<tr>
<td>Sacrum</td>
<td>Patella</td>
</tr>
<tr>
<td>Pubic rami</td>
<td>Anterior cortex tibia</td>
</tr>
<tr>
<td>Femoral shaft</td>
<td>Medial malleolus</td>
</tr>
<tr>
<td>Tibial shaft</td>
<td>Talus</td>
</tr>
<tr>
<td>Fibula</td>
<td>Tarsal navicular</td>
</tr>
<tr>
<td>Calcaneus</td>
<td>Fifth metatarsal</td>
</tr>
<tr>
<td>Metatarsal shaft</td>
<td>Second metatarsal base</td>
</tr>
<tr>
<td>Great toe sesamoids</td>
<td></td>
</tr>
</tbody>
</table>

On exam, athletes generally demonstrate significant point tenderness at the site of fracture with a raised periosteum. Individuals with femoral neck stress fracture have pain with all range of motion. Evaluation for femoral shaft fracture should include a fulcrum test, in which the patient is seated and the examiner places one forearm under the thigh while applying a downward force at the knee or distal thigh. The position of the forearm is gradually moved proximally, with reproduction of pain considered an indicator of stress fracture at that point on the femur.

The first diagnostic test performed for a suspected stress fracture is commonly an x-ray, given its ease and low cost. However, 85% of stress fractures may not be visible on x-ray if performed in the early days of fracture development, and even repeat imaging may not show the fracture in 50% of patients. The most sensitive and specific diagnostic imaging test is MRI (68-99% and 4-97%, respectively), and it should be performed preferentially to bone scan unless contraindications exist.

**INTERVENTION**

Athletes are maintained in a CAM boot until healing takes place. Femur stress fractures are generally managed with limited weight-bearing until imaging shows evidence of healing and the patient is pain free with activity — generally a minimum of six weeks. Sacral stress fractures may require 4-12 weeks of rest to heal. Regardless of the location of the fracture, should pain recur during return to activity, it is critical to return to protected weight bearing until activity is again pain free. In women with disordered menses, oral contraceptives may be recommended to improve bone health by normalizing menstrual cycles. Pulsed ultrasound has been suggested as an adjunctive therapy, but current data is inconclusive. However, extracorporeal shock wave therapy has been shown to improve healing in animals and may be useful in the treatment of refractory stress fractures. Surgical intervention is needed.
in the case of high-risk stress fractures. Tension side femoral neck stress fractures should be surgically pinned, as they frequently become displaced.13 While navicular stress fractures show good healing without surgical management, there must be strict non-weight-bearing for at least six weeks. For athletes who hope to return to sports more quickly, percutaneous screw placement is recommended. Similarly, type 2 Jones fractures may heal with strict non-weight-bearing, but the risk of nonunion or recurrence of fracture can be lessened with surgical pinning.27

The differential diagnosis for bone pain with activity includes medial tibia stress syndrome or “shin splints,” which are a stress reaction of bone with an incidence of 13.6%–20% in runners.2 Patients present with pain along the posterior medial tibia, and x-rays must exclude fracture but may show sclerosis. Pes anserine bursitis may also cause pain along the medial tibia but generally more proximally than in shin splints.

Compartment Syndrome

Chronic Exertional Compartment Syndrome (CECS) presents as exercise-induced pain due to an increase in intracompart-mental pressure that compresses the vasculature or nerves, leading to ischemic pain or paresthesias, respectively.14,35 It occurs most often in the anterior compartment with the posterior, lateral, and superficial posterior compartments less frequently affected. It may be bilateral in 37–82% of affected athletes.13 CECS is fairly rare — Styf confirmed the diagnosis in only 25.6% of suspected cases. Thus an attempt should be made to exclude more common diagnoses, such as tibial stress fractures, fascial defects with muscle herniation, medial tibia stress syndrome (shin splints), fibular nerve injuries, and arterial or venous disorders.35,16,34,17

EVALUATION

Possibly the most challenging factor in diagnosing CECS is the transient nature of the symptoms. In general, patients will have a normal exam and no complaints when seen in clinic. As symptoms are brought on by activity, it is critical to evaluate the patient while symptomatic, as this should reproduce both subjective complaints and exam findings. Patients may be asked to run on a treadmill or run laps around the parking lot until symptomatic. Immediately following provocative exercise, exam findings may include a sense of fullness in the compartment, pain with palpation, weakness in the limb, paresthesias, and diminished reflexes. After a period of rest, some or all of these symptoms and signs may disappear.16

Clinical evaluation is critical but alone is not adequate evidence for a diagnosis, and most individuals will undergo various imaging and diagnostic testing to exclude other conditions with similar presentation. Formal testing for CECS involves measuring intracompartmental pressures (ICP) at rest and following exercise that reproduces the patient’s symptoms, usually at 1 minute and 5 minutes after exercise. Measurement of ICP is performed by inserting a needle attached to a negative pressure syringe into the affected compartment.36 During exercise, average pressures may rise as high as 150mmHg in both unaffected and CECS legs but should return to normal within 1 minute of cessation of exercise. A generally accepted normal ICP is less than 15mmHg at rest, and CECS may be diagnosed with resting pressures greater than 15mmHg, 1-minute pressures greater than 30mmHg, or 5-minute pressures greater than 20mmHg.16 Frequently, athletes with suspected CECS have normal or borderline resting or post-exertion pressures.

MANAGEMENT OF CECS

Historically, compartment fasciotomy was performed for patients with clinical signs of compartment syndrome, with a success rate of 60–100%.37 Pressure measurement theoretically provides a more accurate diagnosis of CECS and reduces the chances of unnecessary surgery. However, Verleisdonk et al37 found that while 83% of individuals with elevated pressures (>50mmHg after exercise) benefitted from fasciotomy, an additional 67% with borderline or normal pressures received relief from surgery. Surgery may be considered if symptoms persist longer than three months.36 Non-surgical management of CECS involves relative rest, avoidance of symptom-provoking activities, physical therapy with or without modalities, and orthotics.1,34 Compared to running (19.1mmHg), cycling (12.2mmHg) produces significantly lower anterior compartment pressures, presumably secondary to less eccentric contraction of the tibialis anterior. This may be a viable alternative for those who cannot tolerate running but want to stay active without undergoing fasciotomy.18

Clinical Vignette Outcome

SD showed no symptoms during examination but continued to complain of anterior lower leg pain with running. She was instructed to run laps around the clinic parking lot until symptomatic. Anterior compartment pressure (performed only on the more severe left leg) was 10mmHg prior to run and 13mmHg immediately following run (within 1 minute). However, following her run, she had weakness of bilateral dorsiflexion and ankle eversion with reflexes intact. A Tinel’s sign was found at the fibular nerve at the fibula head. After 15 minutes of rest, her strength returned and her pain resolved.
Due to concern for exertional compression of the common fibular nerve, she was recommended to initiate physical therapy with a focus on hamstring and anterior/lateral compartment stretching and strengthening.

After about four weeks of therapy, she still complained of pain with running but the discomfort was milder and only provoked by runs longer than 1 hour in duration. An electrodiagnostic study was normal. She continues to train through the pain as she prepares for her marathon. Exploration of the fibular nerve was recommended if she did not have spontaneous resolution of her symptoms.

References

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