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A step-wise approach to exertional leg pain

This review, differential table, and case to test your skills can help you avoid overuse of costly tests and delayed treatment.

PRACTICE RECOMMENDATIONS

› *Consider the possibility of vascular and neurologic problems as the source of exertional leg pain (ELP).* C

› *Order magnetic resonance imaging to evaluate patients with ELP and negative x-rays for stress fractures.* C

› *Measure lower extremity intracompartmental pressures both before and after exercise when you suspect chronic exertional compartmental syndrome. Doing so is the gold standard for the diagnosis of this condition.* B

Strength of recommendation (SOR)

A Good-quality patient-oriented evidence

B Inconsistent or limited-quality patient-oriented evidence

C Consensus, usual practice, opinion, disease-oriented evidence, case series

Most family physicians are accustomed to treating active patients with shin splints and stress fractures. But many are less familiar with, and slower to recognize, other sources of exertional leg pain (ELP), defined as exercise-related pain that localizes in the lower extremity distal to the knee and proximal to the talocrural joint.[1]

ELP has a broad differential diagnosis that includes other musculoskeletal conditions—most notably chronic exertional compartment syndrome (CECS), which has been found to affect 33% of athletes with chronic ELP[1]—as well as a number of vascular and neurologic causes.[2-4] In addition, etiologies may overlap. Greater awareness of the many causes of ELP can help you to avoid the unnecessary use of expensive diagnostic tests as well as delayed diagnosis and treatment.

A thorough medical and activity history, symptom review, and physical examination are your most important tools when patients present with ELP. When the cause is not obvious or the patient fails to respond to conservative measures, x-rays, magnetic resonance imaging (MRI), vascular studies, electromyography and nerve conduction studies, and/or intracompartmental pressure testing may be needed to find the source of the symptoms. In the text that follows, we review both common and relatively uncommon sources of ELP, using a step-wise diagnostic approach. You'll find a diagnostic challenge, in which you can test your skills, on page 676, and a more comprehensive differential diagnosis in the **TABLE**.[1-3,5-9]

Musculoskeletal injuries: Shin splints and beyond

Medial tibial stress syndrome (MTSS), commonly known as shin splints, is characterized by pain and tenderness over the posteromedial aspect of the distal tibia.[3] It typically results in diffuse pain that occurs with exercise, but may persist at rest in severe cases.[3-6] Less often, localized swelling may also be present.[2]

TABLE

Differential diagnosis for exertional leg pain[1-3,5-9]

Diagnosis	History and physical clues	Evaluation/diagnostic tests
Musculoskeletal: Common		
CECS	Consistent bilateral symptoms; numbness and weakness may occur as activity progresses	Lower extremity compartment pressure testing before and after activity
MTSS	Common in runners, often bilateral, diffuse tenderness	Hx and physical often adequate; MRI may confirm diagnosis
Muscle strain	Immediate onset of symptoms, unilateral, no pain at rest	Hx and physical generally adequate
Stress fracture	Common in athletes, especially female athlete triad; unilateral symptoms, focal pain, tenderness	Hx and physical often adequate; x-rays lack sensitivity; MRI may confirm diagnosis
Tendinopathy	Gradual onset of symptoms related to overuse; symptoms localized to tendon	Hx and physical often adequate; MRI may help in advanced cases
Vascular: Common		
Deep vein thrombosis	Unilateral pain, swelling, discoloration; risk factors include smoking, recent travel, cancer, certain drugs	Duplex ultrasound
Peripheral vascular disease	Bilateral claudication; history of smoking, atherosclerosis	Ankle-brachial index and/or angiography
Vascular: Uncommon		
Arterial endofibrosis	Associated with repetitive hip flexion, unilateral, most common in cyclists; ischemic pain and loss of power	Arterial ultrasound useful; MRA is test of choice
Cystic adventitial disease	Men in mid-40s, intermittent claudication with activity	Duplex ultrasound may show stenosis; MRA confirms diagnosis
Popliteal artery aneurysm	Most common peripheral aneurysm, more common in males, associated with smoking and high BP; acute or chronic ischemic pain or arterial insufficiency, often found without symptoms	Pulsatile mass often palpable; duplex ultrasound or arteriography confirms diagnosis
Popliteal artery entrapment syndrome	Lower limb pain and ischemia with high-intensity exercise associated with excessive dorsiflexion and plantar flexion of the ankle	Ankle-brachial index done with the ankle in various positions as screening tool; arteriography for definitive diagnosis
Neurologic: Common		
Lumbar radiculopathy	Possible association with low back pain, symptoms in distribution of dermatome, more likely unilateral	Lumbar x-rays and MRI
Peripheral neuropathy	History of diabetes, vitamin deficiency, or other systemic disease; pain and sensory loss; may be acute or chronic	EMG/NCS, nerve biopsy, evaluation for vitamin deficiency and/or chronic disease
Spinal stenosis	Age ≥50 y, lower back pain, symptoms worse with activity, relieved with sitting or flexing the spine; numbness and tingling from buttocks into legs	Lumbar x-rays and MRI
Neurologic: Uncommon		
Nerve entrapment	Trauma more likely than overuse; pain and tingling worse with activity in distribution of affected nerve	EMG/NCS are tests of choice, but lack sensitivity

BP, blood pressure; CECS, chronic exertional compartment syndrome; EMG/NCS, electromyography/nerve conduction studies; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; MTSS, medial tibial stress syndrome.

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While ice, NSAIDs, proper conditioning, physical therapy, and activity modification are all appropriate treatment for shin splints, none of these interventions are more effective than rest alone.

MTSS accounts for between 6% and 16% of all running injuries.[2,10] It is associated with a spectrum of tibial stress injuries, including periostitis, tendinopathy, and stress reaction, with dysfunction of the tibialis posterior, tibialis anterior, and soleus muscles thought to be contributing factors.[2,11] Intrinsic factors include high body mass index (BMI), female sex, excessive internal and external hip rotation, hyperpronation, and hyper plantar flexion.[2,10,12]

X-rays of the leg are typically normal in patients with MTSS and should be considered only if the clinical presentation suggests the possibility of an alternative diagnosis, such as a stress fracture or tumor.[2-4,13] Advanced imaging such as MRI or triple phase bone scans (TPBS) are useful when the diagnosis is in question and will reveal an abnormally high signal along the posterior medial tibial surface or the classic train-track appearance of nucleotide uptake in patients with MTSS.[2] MRI readily shows periosteal reaction and bony edema and has a sensitivity of 78% to 89% and a specificity of 33% to 100% for the diagnosis of MTSS.[14,15]

Initial management of MTSS is conservative, with the mainstay of treatment consisting of rest, ice, and nonsteroidal anti-inflammatory drugs (NSAIDs).[3,13,16] While ice, NSAIDs, proper conditioning, physical therapy to stretch and strengthen the calf musculature, rigid orthotics to correct foot hyperpronation, and activity modification are all appropriate treatments, randomized controlled trials have shown none of these interventions to be more effective than rest alone.[2] Non-operative treatment is usually successful, but surgery may be required for severe or refractory cases. Procedures include posteromedial fasciotomy, release of the medial soleus fascial bridge, deep compartment fasciotomy, or removal of a section of the distal tibia periosteum.[3,4]

Lower extremity stress fracture. Stress fractures are caused by repetitive loading that results in microtrauma, including bony microfractures. The vast majority of cases—80% to 95% of stress fractures—affect the lower extremities, and most involve the tibia.[2-4,6,13,17] The most common presentation is an insidious onset of pain over a specific bony area with a normal appearance, although localized swelling or erythema may occasionally be present.[3,14,17,18] The pain may be reproduced or worsened by weight-bearing activities and relieved by rest.[14,18]

Consider the female athlete triad. In evaluating a patient with a stress fracture, pay close attention to dietary history, BMI, and, in female athletes, take a detailed menstrual history. Such patients are at risk for amenorrhea, low bone mineral density, and nutritional deficits—the "female athlete triad," which carries an increased risk of stress fractures.[3,14,17-19]

Stress fractures can often be diagnosed with a thorough medical history and physical, with imaging used for confirmation.[6,14,17,18] Historical features of a stress fracture that may differentiate it from MTSS include pain that is unilateral and absent at rest and occurs with more prolonged activity, as well as post-exercise and/or nocturnal pain. Notable physical exam features include pain that is reproduced in a focal area with a single leg hop or percussion with a tuning fork or ultrasound.[5,11,17]

Initially, sensitivity for a plain radiograph is as low as 10%.[2,11] Abnormalities on x-ray are usually seen after 2 to 8 weeks of symptoms[2,7,11] and may include a faint periosteal reaction, a fluffy area of callus, or a cortical lucency sometimes referred to as the "dreaded black line."[3,6,17] If a radiographic exam shows evidence of a stress fracture, further imaging is typically unnecessary. MRI or TPBS is suggested, however, when x-rays appear normal but suspicion of a stress fracture remains.[3,17,18] MRI may show edema within 3 days of symptom onset and is more sensitive and specific than computed tomography (CT) or TPBS for diagnosing stress fractures of the tibia.[2,16]

Treatment of tibial stress fractures is typically non-operative and consists of alterations in activity (eg, non weight-bearing), correction of nutritional deficits, such as inadequate caloric intake or too little calcium or iron, and addressing problems with footwear, training regimen, and/or running surface.[3,14,18] Fibular and posteromedial tibial stress fractures are considered low risk and heal with weight-bearing restrictions and rest, initially for a minimum of 2 to 4 weeks.[3]

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Posteromedial tibia injuries tend to heal well because they are on the compression side of the bone. Anterior tibia stress fractures, which are located on the tension side of the bone[2,7] and account for approximately 5% of all tibia stress fractures, are more prone to non-union or progression to a complete fracture.[7,20] Thus, anterior tibia stress injuries warrant a more aggressive approach, with treatment options including non-weight bearing status that may last longer than 8 weeks, pneumatic brace casting, and/or orthopedic referral to evaluate for surgical intervention.[7,20-22] Time for radiographic evidence of healing may exceed 8 months, so early surgical intervention should be considered, especially for high-level athletes.[7,20,21]

Pain associated with a stress fracture can typically be reproduced in a focal area with a single leg hop or percussion with a tuning fork.

When to suspect chronic exertional compartment syndrome

Leg pain in CECS results from increased pressure within the lower extremity fascial compartments temporally related to exercise.[2,23,24] Its incidence in the general population is unknown, but CECS has been found to range from 14% to 27% in patients with previously undiagnosed leg pain[1,14,25] and to affect about a third of athletes with chronic ELP. In addition, CECS has been found in 90% of patients who have both diabetes and ELP with normal findings on vascular studies.[1,3,4,26]

The anterior compartment is most commonly affected, followed by the lateral, deep posterior, and superficial posterior compartments.[3,13,23,27] Symptoms are bilateral 60% to 95% of the time.[2,13,14,25] Factors contributing to CECS include fixed muscular compartment constraints, muscle swelling, thickened fascia, muscle hypertrophy related to resistance training, dynamic muscular contraction patterns, and low muscle capillary supply. Stretching of fascial pain receptors and pressure fibers and inadequate myocyte response to increased metabolism may play a role, as well.[14,28]

The initial clinical presentation is usually predictable leg pain—ie, pain that begins at about the same time, distance, or intensity of a workout and resolves with rest; numbness and weakness may occur as the workout progresses. In time, leg pain associated with CECS may be present with everyday activity or at rest. The physical exam may be normal or

Test your skills: A diagnostic challenge

Janine T, a 24-year-old long-distance runner, presents with left lower leg pain that occurs with activity. There was no injury, Ms. T reports; the pain began about 6 weeks ago, shortly after she began training for a marathon and running more than 30 miles per week. The pain is not relieved with intermittent rest or over-the-counter analgesics, she says. But it usually abates within 15 to 30 minutes after she completes her run.

Ms. T is underweight, with a body mass index <17 kg/m^2. She denies any dietary restrictions and has normal menstrual cycles. The patient reports taking oral contraceptives, but no other medications. An initial x-ray is normal, as is magnetic resonance imaging to evaluate for a stress fracture.

You suspect Ms. T has shin splints, advise her to rest for a few weeks and to consider getting orthotics for her running shoes, and schedule a follow-up visit.

When she comes in 6 weeks later, the patient reports that she resumed running after a 3-week rest; shortly after, she noticed pain in both legs. What's more, she now experiences tingling in her feet after running a few miles.

What's wrong with this patient?

Ms. T's symptoms—bilateral persistent leg pain, with tingling in both feet, and little improvement with rest—strongly suggest that she has chronic exertional compartment syndrome. Intracompartmental pressure testing, which reveals pre-exercise values ≥15 mm Hg and post-exercise values of ≥30 mm Hg at one minute, confirms the diagnosis.

Activity avoidance or modification will allow Ms. T's symptoms to subside, but they're highly likely to recur when she resumes running. The definitive treatment is intracompartmental fasciotomy, which has a success rate of approximately 80%.[1,28]

reveal swelling, tenderness over the involved compartments, pain with passive digit or ankle motion, and palpable muscle herniation.[14]

Measurement of intracompartmental pressure before and after exercise is the gold standard for diagnosis of CECS.[2,14,27] Pre-exercise values ≥15 mm Hg and post-exercise values ≥30 mm Hg at one minute or ≥20 mm Hg at 5 minutes are all considered diagnostic of CECS,[11] although these widely accepted criteria for bilateral testing of all compartments yields a false-positive rate of 5%.[27] CECS is almost always bilateral,[29] and some clinicians advocate limiting the number of needle insertions by taking only post-exercise measurements and testing only symptomatic compartments in one limb.

Imaging has limited value, as both x-rays and MRIs are usually normal.[14] However, post-exertional T2-weighted MRI findings of muscular edema correspond to increased intracompartmental pressures, with a sensitivity of 87% and a specificity of 62%.[14,24,30,31] Infrared spectroscopy, which measures levels of oxygenated and deoxygenated blood, is sensitive for CECS when the post-exercise ratio of deoxygenated to oxygenated blood remains elevated.[14,24] Neither of these screening modalities is routinely obtained or considered diagnostic, however. Their chief role is to exclude an alternative diagnosis.[14]

Treatment and symptom relief. Discontinuing or modifying the aggravating activity typically brings relief of CECS. But this is not a long-term solution, as symptoms are likely to recur when the patient returns to the activity in question.[1] The definitive treatment is compartment release via fasciotomy. Success rates for anterior and lateral compartment releases are >80%.[1,28] The success of fasciotomy of posterior compartments, however, is <50%—a finding attributed to more complex anatomy, difficult visualization, and the presence of additional compartments.[1,32]

When the cause is vascular

Arterial endofibrosis—the fibrotic thickening of the intima of an artery—is thought to be caused by repetitive hip flexion.[8] This results in hyperplasia, wall thickening, and eventual stenosis of the vessel, with 90% of cases affecting the external iliac artery.[8,33] The condition is most common in activities such as cycling, but is also seen in such activities as running, skiing, soccer, and rugby. Symptoms are typically unilateral, but an estimated 15% of patients experience bilateral symptoms.[8,33]

Loss of power in the affected leg, with intermittent claudication and pain due to presumed ischemia from the vascular defect, is the usual presentation, although some patients develop cramping of the buttocks and/or paresthesia of the affected leg and foot during uphill running or cycling.[8,33] The physical exam is often normal, but there may be a post-exercise arterial bruit over the femoral artery when the hip is flexed.[8,34]

Pre-exercise ankle-brachial index (ABI) <0.5 and post-exercise ABI <0.66 at one minute is suggestive of moderate arterial endofibrosis, with 90% sensitivity and 87% specificity.[8,33,34] Arterial ultrasound and color Doppler may also be used for diagnosis, but are often operator dependent. Magnetic resonance angiography (MRA), while more expensive, can detect excessive kinking or compression of the vessel and is not operator dependent.[8,33] Angioplastic balloon catheter dilation and stenting, bypass surgery, vascular reconstruction and endarterectomy with vein patch are the options for treatment. The success rates of the various interventions are unknown due to a lack of head-to-head studies and long-term follow-up.[8,33]

Popliteal artery entrapment syndrome (PAES) is a constellation of symptoms caused by vascular impingement in the popliteal fossa of the knee.[8,34] The typical presenting symptoms are lower limb ischemia and pain caused by intense exercise that resolves quickly afterwards. Symptoms correlate more with the intensity than the duration of exercise.[3,8]

PAES is usually caused by a variant of the gastrocnemius muscle in which a medial head passes behind the popliteal artery in males younger than 30 years.[8,33-35] Less commonly, it is the result of an overuse or acute orthopedic injury that irritates structures surrounding the popliteal fossa.[8,34] PAES affects football, basketball, and soccer players, as well as runners because of excessive

Consider spinal stenosis in patients who report bilateral lower extremity numbness and tingling that radiates down the legs.

dorsiflexion and plantar flexion of the ankle.[3,4]

The physical exam for a patient with PAES is typically normal, but a post-exercise popliteal bruit with weak peripheral pulses may be elicited.[8,33] An ABI in the neutral, forced dorsiflexion and forced plantar flexion positions can serve as a useful screening tool. An ABI <0.9 is abnormal, with a sensitivity and specificity of 90% and 98%, respectively, for stenosis >50%.[2,36]

Arteriography is the gold standard for diagnosis of PAES. Contrast arteriography is most commonly used because of its availability and cost. But MRA better differentiates functional from anatomic entrapment—a differentiation that less invasive tests, such as duplex ultrasound studies, lack the specificity to reveal.[8,34] Treatment requires either surgical removal of the offending musculotendinous structures or arterial bypass and grafting of the chronically impinged area, as conservative therapies lack efficacy.[2,8,34]

An ankle-brachial index in the neutral, forced dorsiflexion and forced plantar flexion positions is a useful screening tool for popliteal artery entrapment syndrome.

Cystic adventitial disease (CAD) is the narrowing of an artery by mucoid cysts in the arterial wall or adventitia.[8,9] It is a rare condition, accounting for just 0.1% of all vascular diseases, most commonly occurring in men in their mid-40s.[8,33] CAD is thought to be the result of mucin-producing cells being haphazardly incorporated into the adventitia during arterial development. About 85% of patients whose popliteal artery is affected in this way will experience intermittent claudication with activity.[8,9]

On exam, such patients often have diminished ankle-brachial pressure indices, and duplex ultrasound often reveals stenosis in the affected artery, as well as a collection of mucoid cysts in the adventitia.[8,9]

Diagnosis can be confirmed by MRA.[8,37] Evidence for the treatment of CAD is largely anecdotal.[9] Cysts may be aspirated but tend to recur, and stenting does not correct the cystic-induced narrowing of the vessel. Surgical removal of the cysts is the only successful treatment.[8,9]

Neurologic causes to consider

Spinal stenosis is caused by central canal narrowing secondary to congenital abnormalities, trauma, or, most commonly, degenerative changes in the lumbar spine. Spinal stenosis is generally seen in men or women ages 50 to 70 years.[38] Patients experience unilateral or bilateral claudication that improves with sitting or flexion of the spine[5] and may develop bilateral lower extremity numbness and tingling from the buttocks that radiates down the legs. Diagnosis is typically made with a combination of a lumbar x-ray and an MRI, which will show nerve compression and bony overgrowth.[38] CT myelogram, another imaging option, is less sensitive in the acute phase, but can be used to monitor the disease course.

Initial treatment includes physical therapy and NSAIDs.[5] If conservative therapy fails, epidural or nerve root corticosteroid injections and surgical decompression or laminectomy are options.[38]

Nerve entrapment is a less common source of lower extremity pain in which the superficial peroneal nerve is most often affected.[4,12,17,39] Trauma is the usual cause of nerve entrapment, but it may also be associated with overuse, most notably related to dance, soccer, or tennis.[2,14,40,41] The most likely anatomic site is where the nerve exits the deep fascia within the lateral compartment in the lower third of the leg.[39,40] Less frequently, the common peroneal nerve at the fibular neck, the saphenous nerve as it passes through Hunter's canal, the posterior tibial nerve at the tarsal tunnel, and the sural nerve in the posterior calf may be affected.[3,4,12,17,20,40,41] Entrapment of the peroneal nerve may be associated with activities involving repetitive inversion and eversion, such as running and cycling. Injury of the saphenous nerve is seen in sports involving repetitive knee flexion like rowing and cycling. Sural nerve entrapment is a result of crural fascia compression of the nerve during activities like running and track.[3,14,40,42,43]

Patients typically experience burning, tingling, and radiation of pain with activity. Symptoms worsen with continued exercise. The physical exam is often normal, especially early in the disease process, but may reveal sensory loss, motor weakness, and a loss of reflexes.[2,40] Patients with superficial peroneal nerve involvement may have distal lateral leg pain that radiates into the dorsum of the foot, often exacerbated by lower leg percussion and resulting in diminished sensation.[1]

Common peroneal nerve involvement may alter sensation of the lateral leg, as well, but may also cause foot drop.[2] The saphenous nerve can cause medial knee or leg symptoms, while the sural nerve can yield pain in the lateral ankle and foot.[2]

To diagnose nerve entrapment, electromyography and nerve conduction velocities at the level of the lesion may yield positive results 3 to 4 weeks after symptom onset.[2,13,40] There are wide ranges of sensitivity and specificity for these studies, but they are nonetheless considered the tests of choice for nerve entrapment.[1,44] Conservative treatment with activity modification, physical therapy, massage, and NSAIDs is often sufficient,[2] with surgical management warranted only for refractory cases.[2,14,40,41] JFP

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