Tibial Stress Injuries

Tibial stress injuries are by far the most common cause of lower leg pain in athletes, accounting for up to 75% of exertional leg pain [7]. Tibial stress injuries include various types of bone lesions that represent a continuum of abnormalities from asymptomatic osteopenia to fracture, all occurring in response to abnormal repetitive stress applied to normal bone [5–7]. This spectrum of lesions includes periostitis, cortical osteopenia, cancellous bone, and cortical fractures, often associated with various degrees of reactive soft-tissue and bone marrow edema [5–7]. Although both proximal and distal metaphyses and the whole diaphysis can be involved by stress injuries, such injuries more frequently occur in the cortex of the distal two thirds of the tibia and cause a clinical syndrome known as medial tibial stress syndrome or shin splints.

Periostitis can occur both as an isolated abnormality or in association with bone stress injuries. Although on MRI, periostitis appears as a thin hypointensity surrounded by hyperintense edema (Fig. 1). Cortical lesions include osteopenia, cavitations, striations, and fractures. Radiography

OBJECTIVE. Our purpose is to describe the imaging features in athletes with chronic lower leg pain, emphasizing the role of MRI and CT, which are the diagnostic tools with the highest sensitivity and specificity in the differential diagnosis of lower leg pain. Moreover, a diagnostic algorithm in patients with chronic lower leg pain is proposed.

CONCLUSION. Plain radiography has a low sensitivity but may reveal tibial stress fractures, bone tumors, and soft-tissue calcification. CT and MRI may be useful to better evaluate the abnormalities shown by plain radiography.

Exercise-induced chronic leg pain is a common condition in competitive and recreational athletes. By definition, lower leg pain is pain between the knee and ankle [1, 2]. The causes of chronic lower leg pain in the athlete are numerous, and therefore the differential diagnosis is quite broad (Appendix 1).

According to the literature, despite the wide range of potential diagnoses, medial tibial stress syndrome and stress fractures are the most common sources of exercise-induced chronic lower leg pain, followed by chronic exertional compartment syndrome and popliteal nerve entrapment [1–4]. Although a high index of suspicion, careful physical examination, and detailed history are essential in athletes with chronic lower leg pain [1–3], “even for an astute clinician, distinction between the different medical causes may be difficult given that many of their presenting features overlap” [5]. Consequently, the role of diagnostic imaging remains fundamental in detecting the cause of chronic lower leg pain.

The objectives of this article are to describe the imaging features in athletes with chronic lower leg pain, emphasizing the roles of MRI and CT, which are the diagnostic tools with the highest sensitivity and specificity in the differential diagnosis of lower leg pain [5, 6], and to propose a diagnostic algorithm in patients with chronic lower leg pain.

**Exercise-induced chronic leg pain**

**Tibial Stress Injuries**

Tibial stress injuries are by far the most common cause of lower leg pain in athletes, accounting for up to 75% of exertional leg pain [7]. Tibial stress injuries include various types of bone lesions that represent a continuum of abnormalities from asymptomatic osteopenia to fracture, all occurring in response to abnormal repetitive stress applied to normal bone [5–7]. This spectrum of lesions includes periostitis, cortical osteopenia, cancellous bone, and cortical fractures, often associated with various degrees of reactive soft-tissue and bone marrow edema [5–7]. Although both proximal and distal metaphyses and the whole diaphysis can be involved by stress injuries, such injuries more frequently occur in the cortex of the distal two thirds of the tibia and cause a clinical syndrome known as medial tibial stress syndrome or shin splints.

Periostitis can occur both as an isolated abnormality or in association with bone stress injuries. Although a bone scan can be positive in this condition, MRI is the most sensitive examination for diagnosing periostitis [5]. Periostitis appears as a soft-tissue edema near the cortex. Often, on STIR or fat-saturated T2-weighted images, detached periostium can be seen as a thin hypointensity surrounded by hyperintense edema (Fig. 1).

Cortical lesions include osteopenia, cavitations, striations, and fractures.
detects only a small number of cortical fractures. The other cortical abnormalities and the majority of cortical fractures (up to 94%) remain undetected [8] (Fig. 2). Both MRI and CT have high accuracy in detecting the spectrum of cortical abnormalities. MRI is the most sensitive single diagnostic tool for patients with medial tibial stress syndrome given its excellent soft-tissue contrast to show soft-tissue abnormalities [5] (Figs. 3 and 4). However, sometimes CT permits the early diagnosis of cortical abnormalities not visible on MRI [6] (Fig. 5).

**Chronic Exertional Compartment Syndrome**

Chronic exertional compartment syndrome is a cause for claudication in athletes. It is caused by abnormally increased pressure within muscular compartments that are enclosed by relatively noncompliant fasciae. Of the four lower leg muscular compartments, anterior and lateral compartments are affected more frequently than others (76%), followed by deep posterior (16%) and posterior superficial (12%) [9].

The diagnosis of chronic exertional compartment syndrome requires measuring compartment pressure with a slit or weak catheter. However, some problems exist: Pressure measurement is an invasive technique. Potential risks include muscular hernia or neurovascular damage [9]. Multiple compartments are affected in about half of chronic compartment syndrome cases and symptoms in the bilateral legs occur in approximately 75% of cases [10]. Chronic exertional compartment syndrome may be complicated by the coexistence of periostitis and tibial stress fracture, which cannot be diagnosed by compartment pressure measurement.

MRI is a promising technique for non-invasive diagnosis of chronic exertional compartment syndrome [11, 12]. A recent work has shown that the sensitivity of MRI in diagnosing chronic exertional compartment syndrome was comparable to that of intra-compartmental pressure measurement and near-infrared spectroscopy [11].

MRI must be performed immediately after exercise-inducing pain. MRI findings in chronic exertional compartment syndrome include muscular hyperintensity on T2-weighted or fast STIR images with or without muscular swelling (Figs. 6 and 7). Inhomogeneous hyperintensity within affected compartments can be seen (Fig. 6). MRI may detect involvement of more than one compartment (Fig. 6) and concurrence of medial tibial stress syndrome.

Further studies are necessary to define the appropriate role of MRI in the diagnostic algorithm of chronic exertional compartment syndrome. In the meantime, MRI should be used as a problem-solving examination in confusing circumstances, in patients refusing compartment pressure measurement, or in patients with contraindication for compartment pressure measurement (coagulation disorders).

**Peripheral Neuropathy**

Compression or nerve entrapment can lead to a functional disturbance or pathologic change in the peripheral nerve of the lower leg causing lower leg pain and muscular dysfunction. The superficial peroneal nerve and sural nerve can be involved, but the most common cause of extravascular neuropathic leg pain is common peroneal nerve disease, which can be caused by trauma, compression, and intrinsic abnormalities.

Although the diagnosis of common peroneal nerve injury is based initially on electromyography, MRI plays an important complementary role in diagnosing the cause of the nerve sufferance [13]. Moreover, MRI can also show muscular neurogenic edema in the subacute phase and atrophy with fatty degeneration in the chronic phase of denervation [14] (Fig. 8).

Muscular neurogenic edema appears on fast STIR and fat-saturated T2-weighted turbo spin-echo images as a diffuse homogeneous hyperintensity involving the muscles innervated by diseased nerve. Hyperintensity of denervated muscles is usually seen after 2–3 weeks but can appear as early as 4 days after acute traumatic denervation [14]. Muscular neurogenic atrophy with fatty infiltration due to longstanding denervation is well shown by T1-weighted MR images. This is important clinical information because neurogenic atrophy is irreversible damage.

**Peripheral Vascular Disease**

Popliteal artery disease is an uncommon cause of intermittent claudication of the lower leg in young athletes. The most common cause (up to 60%) is popliteal artery entrapment syndrome, which typically occurs in young men [15–17]. This syndrome is caused by anomalous musculature in the popliteal fossa, in which the popliteal artery is entrapped [15, 16] (Fig. 9). Popliteal artery entrapment syndrome may be complicated by thrombosis [15] (Fig. 9). Other rarer causes of intermittent claudication in young athletes are adventitial cystic disease, muscular fibro dysplasia, arteritis, and compression of the artery by exostosis of the distal femur [17].

The diagnosis of popliteal artery disease requires not only depiction of arterial stenosis but also identification of the responsible disease. Although arterial stenosis can be shown on conventional angiography and sonography, CT and MRI are equally better in showing the cause of popliteal abnormalities [15–17] (Fig. 9). However, MRI has some advantages over CT, including lack of ionizing radiation and the need for contrast material as well as higher soft-tissue contrast [16].

**Other Causes**

Among the uncommon causes of chronic lower leg pain in athletes, a number of soft-tissue and bone diseases should be considered, including tumors and infection [1–3], tendinopathy of the proximal Achilles tendon (Fig. 10), other more unusual tendinopathies (Fig. 11), interosseous membrane injuries, and chronic bursitis (Fig. 12).

**Diagnostic Algorithm**

We propose a diagnostic algorithm for differential diagnosis in athletes with chronic lower leg pain (Fig. 13). It is useful to remember that, according to Edwards et al. [2], the first diagnostic step is always represented by radiography. The advantages of radiography are low cost, short imaging time, and easy execution. Radiography has a low sensitivity but may reveal tibial stress fractures, bone tumors, and soft-tissue calcification (calcific tendinitis, calcific soft-tissue tumors, and so on). CT and MRI may be useful to better evaluate the abnormalities shown by radiography.

**References**


APPENDIX 1: Causes of Chronic Lower Leg Pain in Athletes

<table>
<thead>
<tr>
<th>Very common</th>
<th>Tibial stress injuries (medial tibial stress syndrome, stress tibial fractures, periostitis)</th>
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<tbody>
<tr>
<td>Common</td>
<td>Chronic exertional compartment syndrome</td>
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<tr>
<td></td>
<td>Peroneal neuropathy</td>
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<td></td>
<td>Tendinopathy (including Osgood-Schlatter disease)</td>
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<tr>
<td>Less common</td>
<td>Popliteal artery entrapment</td>
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<td></td>
<td>Bursitis</td>
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<tr>
<td>Uncommon</td>
<td>Other popliteal artery diseases</td>
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<td>Bone tumors and infections</td>
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<td>Fibular stress fracture</td>
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<td>Myopathy</td>
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<td>Deep venous thrombosis</td>
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<td></td>
<td>Muscle hernia</td>
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<td>Interosseous membrane injury</td>
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**Fig. 1**—Tibial periostitis in 32-year-old man who was professional basketball player. **A and B,** Axial (**A**) and coronal (**B**) fast STIR images show periosteal edema. Detached and thickened periosteum can be seen as signal-void line (**arrow**).
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Fig. 2—Longitudinal tibial stress fracture in 34-year-old man who was runner and had chronic medial tibial stress syndrome lasting 6 months. 
A, Orthogonal radiograph obtained 10 days before MR and CT examinations does not show fracture. 
B, Axial T2-weighted MR image shows longitudinal tibial stress fracture as cortical hyperintense line. Hypointense calcified periosteal callus (arrow) as well as bone marrow edema can also be seen. 
C, High-resolution CT image shows, with better advantage, longitudinal tibial stress fracture with calcified periosteal callus (arrow).

Fig. 3—Medial tibial stress syndrome in 21-year-old man who was runner. 
A, T2-weighted axial MR image shows multiple osteopenic lacunae (arrows) in anterior and posterior cortices of tibia. 
B, Three-dimensional CT reconstruction image of same patient confirms evident osteopenia (arrows) of anterior and posterior tibial cortices. Note normal density of fibula and lateral tibial cortices.
**Fig. 4**—Tibial stress injury in 27-year-old woman who was handball player and had chronic leg pain.  
**A,** Fast STIR image shows both periosteal (arrowheads) and bone marrow edema (asterisk) but not fracture.  
**B,** Axial turbo spin-echo T1-weighted image confirms absence of cortical fracture. Bone marrow edema (asterisk) and periostitis (arrowhead) are less conspicuously appreciable in comparison with fast STIR image in **A.**

**Fig. 5**—Medial tibial stress syndrome in 20-year-old man who was runner.  
**A,** High-resolution CT image reveals multiple areas of osteopenia and cavities (arrows) of anterolateral tibial cortex representing stress related lesions.  
**B,** Sagittal fast STIR image corresponding to **A** is negative.
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Fig. 6—Chronic compartment syndrome in 30-year-old man who was runner. Fat-saturated T2-weighted axial MR image, obtained immediately after exercise, shows evident edema of tibial anterior and deep posterior compartment muscles (arrows). Slight, questionable hyperintensity can be seen in other muscles of anterior compartment (arrowheads).

Fig. 7—33-year-old female long-distance runner with right lower leg chronic exertional compartment syndrome lasting 3 months. Patient refused catheter pressure measurement. Fat-suppressed T2-weighted axial MR image obtained immediately after pain-inducing exercise shows swelling and hyperintensity of anterior compartment muscles.

Fig. 8—Chronic hypertrophic demyelinating neuropathy in 24-year-old male basketball player. A, Axial T1-weighted turbo spin-echo image shows enlarged common peroneal nerve (arrow) with loss of normal fascicular pattern. Note slight fatty replacement because of early muscular atrophy of denervated muscles. B, Fast STIR image shows enlarged and hyperintense common peroneal nerve (arrow). Muscles of anterolateral and peroneal compartments are diffusely hyperintense because of denervation.
Fig. 9—19-year-old man who complained of right leg pain that appeared with hard exercise and abated with rest. (Reprinted with permission from [15], Utsunomiya D, Sawamura T. Popliteal artery entrapment syndrome: noninvasive diagnosis by MDCT and MRI. *Australas Radiol* 2007; 51(spec no):B101–B103)

**A**, Occlusion of right popliteal artery is seen on 64-MDCT angiography image.

**B**, Delayed phase axial CT image of right popliteal fossa shows abnormal anatomy in which medial head of gastrocnemius muscle (MHG) courses between thrombosed popliteal artery (PA) and popliteal vein (PV).

**C**, MR angiography is comparable to CT angiography (**A**) in showing occlusion of right popliteal artery.

**D**, Axial T2-weighted image also shows abnormal anatomy responsible for entrapment. Black arrow indicates popliteal artery, white arrow indicates popliteal vein, and arrowhead indicates medial head of gastrocnemius muscle.

Fig. 10—Tendinopathy in 38-year-old male long-distance runner.

**A**, Sagittal fast STIR image shows tendinopathy and partial tear (black arrow) of Achilles tendon (white arrows). Edema of peritenonem (arrowheads) also can be seen.

**B**, T1-weighted axial turbo spin-echo image confirms enlargement of Achilles tendon with marked medial hyperintensity (arrow) and posterior peritenonitis (arrowheads).
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Fig. 11—Calcification of interosseous membrane at insertion of tendon of posterior tibial muscle in 29-year-old male professional soccer player complaining of chronic pain of 1 year with recurrent episodes of acute pain. A, Proton density--weighted fat-saturated axial image shows calcification (arrow) of interosseous membrane at insertion of posterior (P) tibial muscle. A = anterior tibial muscle. B, Coronal T2-weighted fat-saturated turbo spin-echo image shows tendon calcification (asterisk) and edema (arrow) of posterior tibial muscle (P) at tendon–muscle junction. Muscle injury was probably due to reduced elasticity of tendon–muscle junction in patient with chronic overuse of muscle. Inflammation or recurrent strain can explain muscular abnormalities.

Fig. 12—Chronic bursitis in 32-year-old male soccer player with slight chronic pain and swelling on medial side of upper part of lower leg. A and B, Axial T1-weighted turbo spin-echo image (A) and coronal fat-suppressed T2-weighted turbo spin-echo image (B) show enlarged bursa (arrowheads) containing multiple ossified loose bodies (arrows). Bursitis is not anserine bursitis because it was located superficial to pes anserinus tendons. This is adventitious bursa caused by chronic friction from upper edge of stiff shin-guard.

Fig. 13—Chart shows suggested diagnostic algorithm in patients with chronic lower leg pain. PES = popliteal artery entrapment syndrome.