Shin pain diagnosis requires multipronged clinical approach

Athletes who participate in sports that require both prolonged and quick bursts of running and/or jumping are familiar with shin splints, one of the most common exercise-induced lower extremity injuries.1 Symptoms may include pain over the anterolateral or the distal two-thirds of the posteromedial aspect of the shin.2-6 These symptoms are usually present with activity and are alleviated with rest.7

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Risk factors that may predispose an individual to develop shin splints include training errors, foot shape and biomechanics, poor preseason conditioning, and high body mass index.

If the athlete continues to train through the pain and a proper treatment program is not initiated, the symptoms and severity of the injury may progress. Activities that involve repetitive ground reaction forces associated with running and jumping may include long-distance running, track and field, soccer, basketball, and volleyball. Military recruits in basic training may also develop this problem. 

During running each foot strikes the ground 50 to 70 times per minute or a total of 800 times per mile, with the force of two to four times body weight. The muscles, tendons, and bones that support the lower extremity can usually adapt to increased workloads associated with running if the training program progresses properly. However, various risk factors may predispose an individual to the development of shin splints by increasing ground reaction forces that act on the lower extremity or on a compromised musculoskeletal structure. These factors include training errors, foot shape and biomechanics, poor preseason conditioning, high body mass index, poor nutritional habits, and age-related degenerative changes.

The initial definition of shin splints was any type of pain from the hip to the ankle. Though broad, it seemed appropriate in the mid-1900s because treatment algorithms for lower extremity injuries were in their rudimentary form and were relatively ineffective. Over the last four decades, medical research has led to advancements in understanding the causes, treatment, and prevention of sports injuries. Although the exact pathophysiology of shin splints remains unknown, several possible etiologies have been considered.

An understanding of the kinesiology, physiology, and anatomy of the lower leg, and of risk factors associated with shin splints, along with a detailed history and physical examination may aid the practitioner in developing a proper treatment and prevention protocol. Most cases of shin splints can be effectively treated with conservative care.

Anatomy of the lower leg

Long bones such as the tibia have a rigid outer layer of compact bone. The external surface of the compact bone consists of several concentric rings of cortical bone, which is surrounded by periostem, a thin layer of dense fibrous connective tissue that is richly innervated by nerves and blood vessels. Anchored to the cortical bone by bundles of connective tissue, or "Sharpey fibers," the periosteum has several functions. It protects the underlying bone and participates in its repair and remodeling process and is the site of attachment of adjacent muscles. Muscles consist of bundles of individual muscle fibers (fasciculi); each fiber is surrounded by endomysium, a thin, delicate layer of connective tissue. The fibrocartilage strands of the periosteum intertwine with the endomysium of abutting musculature to create an extensive origin of attachment. At this attachment, the underlying cortical bone is rough or thickened due to bone remodeling because of muscle traction.

The tibia has three potential sites for muscle attachment: a posterior surface, and an anterolateral and a medial surface that are separated by the anterior tibial crest. The soleus, flexor digitorum longus, and tibialis posterior musculature connect to the posterior surface of the tibia. The tibialis anterior musculature attaches to the anterolateral surface of the tibia (Figures 1A-D). These muscles are contained within the superficial posterior, the deep posterior, or the anterior compartment of the leg (Figure 2).

The superficial posterior compartment houses the soleus musculature and is bounded posteriorly by the crural fascia and anteriorly by the transverse fascia. The deep posterior compartment contains the flexor digitorum longus and the tibialis posterior musculature. The tibialis anterior muscle is contained within the anterior compartment of the leg. These muscles surrounding the shin aid in coordinating movements of the foot and ankle and in reducing ground reaction forces associated with running and jumping.

Lower leg biomechanics during ambulation

Gait consists of the stance phase and the swing phase for each leg. During the stance
Throughout loading response and into early midstance, the foot goes through pronation, a series of transformations that allow the lower extremity to be more efficient in absorbing ground reaction forces. Pronation of the foot and ankle consists of the following movements: the STJ pronates, the heel bone turns outward (everts), and the talus drops downward distally and adducts toward the midline as the medial longitudinal arch (in-step arch) lowers towards the ground; ground reactive dorsiflexion occurs at the talocrural joint, the tibia approximates to the toes, and the forefoot turns outward, or abducts (Figure 3). Normal range of pronation is 4° of heel eversion and 20° of ground reactive dorsiflexion. Pronation of the foot and ankle should terminate before late midstance as the foot prepares for toe-off.24,27,28

Shortly after the foot strikes the ground, the muscles attached to the anterior and posterior aspects of the tibia contract eccentrically; the muscle fibers elongate as that tension attempts to decelerate. The soleus, tibialis posterior, and flexor digitorum muscles have points of origin on the posterior aspect of the tibia and contract eccentrically during loading response and into midstance to decelerate pronation. Shortly after initial ground contact, the tibialis anterior contracts eccentrically to decelerate foot slap, when the forefoot descends towards the ground.24,21

Under normal circumstances, the musculoskeletal support structures of the lower extremity can adapt to these repetitive eccentric loads associated with running. However, training errors, structural abnormalities, and other risk factors may predispose a runner to excessive eccentric loads placed on the lower leg's musculature. This phase, the foot contacts and adapts to the ground surface. The swing phase begins when the stance leg lifts off of the ground. Most sport-related injuries can be attributed to the repetitive ground reaction forces occurring during the stance phase.26

The stance phase of gait comprises five subphases: initial contact, loading response, early midstance, late midstance, and terminal stance. During initial contact, the foot of the swing leg meets the ground. Loading response begins shortly after initial contact as the foot begins to adapt to the terrain. Early midstance commences as the contralateral swing leg is midline with the body and distributes the body weight over the stance leg. Late midstance starts when the foot of the stance leg changes from a mobile adaptor to a more rigid lever as the foot prepares for toe-off. Terminal stance begins shortly after heel lift and ends with toe-off.27,28

Sequenced movements of the articulations of the rearfoot during the stance phases are pivotal in determining the function of the foot and ankle. The rearfoot is composed of the talocrural and the subtalar joints. The talocrural joint (ankle mortise) consists of the articulation of the distal aspect of the tibia and the fibula with the trochlea of the talus.

The subtalar joint (STJ) consists of the articulation of the undersurface of the talus with the calcaneus (heel bone).24,27,28

Figure 2. Cross section of the lower leg shows the position of the tibia and surrounding musculature.

Figure 3. Pronation of the foot and ankle occurs after the foot contacts the ground. The ankle pronates, the talocrural joint dorsiflexes, the subtalar joint everts, and the forefoot abducts.
can result in anterolateral or posteromedial shin splints. Although the exact pathophysiology of shin splints has not been determined, research has come up with several hypothetical causes.

**Possible etiologies**

Various causes have been ascribed to shin pain.

*Traction periostitis/periostalgia.* In 1964 the American Medical Association defined shin splints as “pain and discomfort in the leg from repetitive activity on hard surfaces, or due to forcible excessive use of the foot flexors.” Current research supports the hypothesis that posteromedial shin pain in running athletes may be caused by excessive eccentric contractions of the superficial and deep posterior compartment muscles that originate on the tibia. Training errors that do not allow for proper adaptation of the musculoskeletal support structures of the lower leg to increased workloads may lead to inflammation or degenerative changes to the surrounding fascia (fasciitis) or to the periosteum (periostitis/periostalgia).1,2

Posteromedial shin pain due to traction periostitis has been given several different names. Posterior tibial syndrome (PTS) was first used in 1978 to describe posteromedial shin pain due to a traction periostitis involving the origin of the tibialis posterior musculature.2 Traction tibial stress syndrome (MTSS) was introduced in 1982 to encompass any of the origins of the deep flexor muscles of the leg that attach to the tibia as possible causes of traction periostitis.3

In 1985, Michael and Holder coined the term soleus syndrome. Using bone scintigraphy and biopsies, they diagnosed complaints of posteromedial leg pain, and found increased bone remodeling and periostitis at the origin of the soleus muscle on the medial aspect of the tibia. However, other studies involving biopsies of patients diagnosed with posteromedial shin splints did not confirm inflammation of the periosteum.2 Based on those studies, it has been hypothesized that shin pain may instead be caused by a degenerative periostalgia. MTSS is currently the most common term used to describe posteromedial shin pain thought to be caused by periostitis or periostalgia.1,2

Anterolateral shin pain has been linked to repetitive eccentric contractions of the tibialis anterior muscle. Downhill running accentuates a more pronounced foot slap, which can predispose the tibialis anterior to fatigue. This may result in anterolateral shin pain or anterior tibial stress syndrome (ATSS) due to a similar mechanism of injury as MTSS.4

**Bone stress reaction.** Bone remodeling of the tibia in response to increased workloads commences approximately five days after stimulation.2 Two types of cells are active in bone remodeling: osteoclasts and osteoblasts. Osteoclast cells react to this increased stress by breaking down bone. Bone resorption is followed by osteoblast secretion of osteoid, or uncalcified bone. Osteoid calcifies into new bone after interacting with calcium and phosphate ions. These adaptations should result in a stronger, more rigid skeletal support structure, although the process may take 90 days.2

Overtraining may lead to a higher ratio of bone resorption by osteoclast cells to the production of new bone by osteoblast cells. A compromised skeletal support structure, which is susceptible to a bone stress reaction, may result.4,5 Bone stress reactions may include microfractures, or in more advanced stages, a stress fracture. Stress fractures in athletes occur most commonly in the tibia at about four to five weeks after beginning a new exercise regimen.6,7,8,9,10

**Exercise-induced compartment syndrome.** Increased intracompartmental pressures due to exercise are most commonly found in the anterior and deep posterior compartments of the leg.2 Repetitive eccentric contractions of the calf muscles may lead to the release of protein ions due to microtrauma. This results in increased intracompartmental swelling, decreased blood flow, and ischemia; this eventually can lead to muscle dysfunction and shin pain.5,11 The treatment of posteromedial or anterolateral shin splints in minor injuries is similar whether the cause is a traction periostitis/periostalgia, bone stress reaction, or ECS.1,2 The initial history and physical examination should focus on discovering possible risk factors. Treatment and prevention should then include steps to minimize these risk factors.

**Risk factors**

Risk factors associated with shin splints can be separated into intrinsic and extrinsic etiologies (Table 1).6,12 Intrinsic risk factors may include structural abnormalities such as pes planus (flat foot); forefoot varus (inversion in relation to the rearfoot, Figure 4A); rearfoot varus (inverted position of the back of the heel, Figure 4B); and ankle equinus (limited dorsiflexion of the ankle joint most likely caused by diminished flexibility of the posterior calf musculature). These abnormalities may lead to prolonged or excessive pronation of the STJ and predispose an individual to shin splints.1,2,3,4

Conversely, a pes cavus structure (high arch that is usually restricted in pronation) is limited in its ability to absorb ground reaction forces. This leads to an increased workload on the tibia, which can predispose a person to a bone stress reaction.5,12

Most risk factors are correctable or can be minimized. Severity of symptoms and level of injury are generally scored on a four-grade system:

- **Grade 1.** Pain is present at the end of the workout but is minimal;
- **Grade 2.** Pain is present during the workout but does not affect performance;
- **Grade 3.** Pain during the workout affects performance but dissipates when activity ends;
- **Grade 4.** Pain does not allow participation in sport and is now present during activities of daily living.1 Usually the athlete will seek treatment when pain hinders performance.

**Diagnosing shin splints**

The history should include the following: initial onset of injury; current symptoms; progression of the frequency, intensity, and duration of the weekly training runs; training surface; whether training runs incorporate hills; age and type of running shoes; recent weight fluctuations; past history of stress fractures; lower extremity strength training and flexibility program; and training goals.6,13

Palpation may reveal diffuse tenderness over the distal two-thirds of the posteromedial aspect (MTSS) or the anterolateral aspect of the shin (ATSS).6,13,14 However, palpation that reveals focal tenderness localized to bone and edema, erythema, and an inability to run due to severe pain at that localized sight, may
be a red flag for a bone stress reaction. Vibratory irritation with a tuning fork or ultrasound may exacerbate symptoms related to more severe cases of bone stress reactions; these findings, however, are not typically reliable.

Provocative testing may be needed. These tests are meant to increase eccentric strain on the posterior calf muscles that attach to the tibia; thereby leading to or exacerbating symptoms related to MTSS. These tests may include one-legged hops on an unstable surface, such as a trampoline or a foam cushion; passive dorsiflexion of the foot by the practitioner with the patient prone and the knee flexed; or the patient applying a ground reaction force to the talocrural joint by assuming a lunge position with the affected limb forward, and then pushing upwards on an undersurface of a railing or counter surface with the ipsilateral hand. Visual observation of the medial longitudinal arch and the instep may reveal a pes planus or pes cavus foot structure. Using a goniometer, a more detailed analysis of foot structure and compensatory movements can be obtained in the nonweight-bearing and weight-bearing positions.

If the patient is not improving with conservative care or the clinical diagnosis is unclear, radiographs or advanced imaging techniques can prove helpful in differentiating between shin pain due to MTSS/ATSS, a bone stress reaction, or other pathology.

Advanced imaging techniques
Radiographs are routinely taken of the leg in patients experiencing shin pain. This baseline study may reveal a periosteal reaction, callus formation, or a radiolucent line, which are common findings of a bone stress reaction or frank stress fracture. Other pathological conditions, such as an osteoid osteoma, osteosarcoma, or Ewing's sarcoma, may also be ruled out with an x-ray.

However, bone stress reactions are usually not visualized on x-ray until the second to sixth week postinjury or initial complaints of symptoms. Sensitivity of early fracture detection by radiography can be as low as 15%, and follow-up x-rays may demonstrate diagnostic findings in only 50% of cases.

Figure 4. Intrinsic risk factors associated with shin splints include forefoot varus (A) and rearfoot varus (B), both of which can be measured with a goniometer.

More advanced studies, such as an MRI or a bone scan, may be needed for further evaluation. These studies are more sensitive in detecting bone pathology in the earlier stage of injury than radiographs. They can aid in developing a treatment plan for a gradual return to sport-specific training or one that may require a longer period of modified rest, immobilization, or corrective surgery for more advanced cases of bone stress reactions.

MRI is highly sensitive to edema in musculoskeletal structures. Two different types of sequences may be used: a T1-weighted sequence that depicts anatomy and more advanced signal abnormalities in the cortical bone; and either a short tau inversion recovery (STIR) or a T2-weighted frequency-selective fat suppression sequence, which is more sensitive to edema in the muscle, periosteum, or bone marrow.

Frederickson et al developed a graded classification of MRI findings and the severity of bone stress reactions. Grade 1 injuries demonstrate mild periosteal edema on the T2-weighted images only. Grade 2 injuries show more severe periosteal edema with bone marrow edema than that detected on T2-weighted images only. Grade 3 injuries depict moderate to severe edema of both the periosteum and bone marrow on T2-weighted and also on the T1-weighted images. Grade 4 injuries show a low signal fracture line with changes of severe marrow edema on both T1-weighted and T2-weighted sequences. When pain was present during training and normal ambulation, there was an 81% incidence of a grade 3 or 4 injury as depicted by MRI.

A bone scan is highly sensitive in detecting osteoblast activity that occurs in bone remodeling. Usually a triple-phase bone scan, which involves a preinjection of the technetium-99m methylene diphosphonate isotope, is conducted. In the first phase—blood flow—images are taken over the symptomatic area for 60 seconds. In the second phase—blood pool—images are taken for five minutes immediately after the blood flow phase. In the third phase—delayed skeletal—images are taken two to six hours after the initial injection. The delayed images allow the isotope to clear from the adjacent soft tissue; under normal circumstances, approximately 50% of the isotope is absorbed into the skeletal system. The amount of radiotracer absorption depends on the rate of bone remodeling and osteoblastic activity.

Less severe bone stress injuries are depicted as ill-defined foci of increased isotope absorption and are located predominantly in the cortical region of bone. More severe injuries involve a higher rate of bone remodeling, which results in increased isotope resorption and wide fusiform lesions that extend from the cortical bone into the medullary bone. Acute stress fractures would be positive for increased isotope activity on all three phases. Soft tissue injuries would indicate increased uptake in the initial two phases but not in the delayed skeletal phase. MTSS would show linear foci of increased isotope uptake along the posterior border of the tibia in the delayed skeletal phase only.
An MRI or bone scan can be a valuable tool for early detection and differentiation between shin pain due to MTSS/ATSS, a bone stress reaction, or other pathology. These advanced imaging studies may prove useful in avoiding complications due to undetected bone stress reactions or other pathologies. However, advanced imaging findings can be vague. Previous imaging studies have indicated false-positive findings present in asymptomatic patients; false-negative findings also have been noted. Therefore, the practitioner should correlate MRI and bone scan findings with clinical examination findings.

The hallmark test for ECS is intracompartamental pressure measurement with a slit catheter. These measurements are taken before exercise (see “Shin pain treatments get active patients back on track,” April, page 31), and then at one and five minutes postexercise. Measurements consistent with an ECS diagnosis are a preexercise pressure of ≥ 15 mm Hg; one-minute postexercise pressure of ≥ 30 mm Hg; or five-minute postexercise pressure ≥ 20 mm Hg.

In anterior compartment syndrome, muscle testing may reveal weak dorsiflexion of the foot, and paresthesia may be present on the dorsum of the foot. In deep posterior compartment syndrome, muscle testing may reveal weakness in plantar flexion and inversion, and paresthesia may be observed on the plantar aspect of the foot.2

References

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**TABLE:** RISK FACTORS ASSOCIATED WITH SHIN SPLINTS

| Source Ref. | DUBIN Chiropractic in Quincy, MA, and a member of the Team USA Triathlon/Duathlon International Triathlon Union medical staff since 1999. Rachel Appel Dubin, PT, DPT, is a staff physical therapist at Milford Hospital in MA. Gregory H. Deon D.C., CCSP, is the owner of Bergen Chiropractic and Sports Rehabilitation Center in Cliffside Park, NJ. This is part 1 of a two-part article. Next month, part II will look at prevention and treatment options for shin pain. |

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**Intrinsic**
- Lower limb structural abnormalities:
  - Pes planus, rearfoot varus, forefoot varus, ankle equinus, and pes cavus
- Poor conditioning, overweight (body mass index > 30 kg/m²);
  - Insufficient muscle endurance and strength; limited flexibility

**Extrinsic**
- Training errors: inappropriate progression of intensity, frequency, or duration of training program
- Poor training surface that is either hard and unyielding or uneven
- Unsuitable footwear
- Hill training before a proper base is established

**Female triad—osteoporosis, amenorrhea, and an eating disorder**
- Age-related changes that are especially noticeable after 40

**History of previous stress fractures**
- Type of sport

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