Medial Tibial Stress Syndrome: Current Etiological Theories Part I—Background

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Clinicians who work with athletes who have medial tibial stress syndrome (MTSS) are often asked the question, “What is making my leg hurt?” This question has many possible answers. Until the etiology of MTSS is clearly established, preventing and treating this common condition will involve some degree of uncertainty. The purpose of this report is to review the relevant theoretical history (Part 1) and in ATT’s next issue, recent research findings (Part 2) pertaining to the etiology of MTSS.

History

Through the 1950s and 1960s, many researchers studied overuse injuries of the lower leg, including what was termed “shin splints.”¹⁻³ Many of those studies used terms such as shin soreness, fatigue infraction, tibial stress syndrome, shin splint syndrome, compartment syndrome, and tibial stress fractures to identify the condition that is now designated as MTSS. It wasn’t until the 1970s that clinical researchers began to differentiate various conditions responsible for lower leg discomfort.

In 1974, Clement⁴ was among the first to postulate that “tibial stress syndrome” was caused by a periostitis that could progress to tibial stress fracture. The theory that pain was caused by inflammation of the periosteum was widely accepted through the next decade. An important study that built upon this theory was reported in 1978. A research team in Oregon suggested that the tibialis posterior muscle was involved in the etiology of MTSS.⁵ Tibialis posterior tendonitis was described as the first stage in a progression of MTSS, which was believed to be followed by periostitis and stress fracture. This theorized progression of pathology complimented Clement’s theory. Between 1977 and 1984, several studies were published regarding the use of bone scans to diagnose tibial stress injuries.⁶⁻¹¹ These studies demonstrated that there was not one specific presentation of MTSS, but rather “diverse abnormalities” that were associated with lower leg pain.⁸

The use of tissue biopsy to study MTSS etiology was introduced in 1982 by Johnell.¹² The study included 62 patients presenting medial tibial pain after exercise. The authors reported that 22 of 35 bone biopsies from the medial edge of the tibia demonstrated increased tissue metabolic activity, i.e., osteoblast proliferation, osteoid seams, and vascular ingrowth, and 13 of 33 soft tissue biopsies demonstrated inflammatory changes in the crural fascia. Importantly, only one case involved periosteal inflammation. The author’s conclusions introduced the concept of stress microfracture as a common cause of MTSS. This study found that 2/3 of MTSS patients had bone injury and 1/3 had soft tissue injury, which supported the existence of “diverse abnormalities” and a continuum of pathology.

In 1985, Michael and Holder¹³ suggested that the soleus muscle was a source of the pain associated with MTSS. They noted,

. . . a distinctive pattern of increased uptake on triple phase radionuclide bone scan in patients with MTSS. The location of uptake coincided with the origin of the medial soleus muscle. . . . Because a true inflammatory process would demonstrate increased uptake on the first two phases (angiogram and blood pool) of a triple phase bone scan, the most frequent finding in these patients is diffuse uptake on only the delayed (third) phase of the scan. This finding is more indicative of bone remodeling. (p. 88)
The researchers obtained biopsy samples from 75 patients presenting chronic MTSS. Only four demonstrated evidence of periostitis. Twenty-two biopsy samples demonstrated evidence of soleus fascia inflammation, most commonly affecting the crural fascia. Twenty-four biopsy samples demonstrated vascular ingrowth, osteoblasts, and osteioids, all of which are associated with a bone remodeling response to repetitive stress and/or trauma. They also found that 1/3 of the MTSS patients had soleus fascia inflammation and 1/3 had bony inflammation just underneath the fascial attachment into the bone.

In 1988, Messier and Pittala reported a correlation between MTSS symptoms and high plantar flexion strength, along with limited dorsiflexion range of motion. In 1990, Saxena et al. dissected 10 lower leg cadaver specimens and reported that the origin of the tibialis posterior muscle extended to the lower one-third of the tibia. Because discomfort at the distal one-third to middle one-third of the medial tibia is recognized as a consistent clinical finding associated with MTSS, they concluded that the tibialis posterior muscle pathology plays a role (Figure 1).

Beck and Osternig subsequently dissected 50 cadaver specimens and contradicted the findings of the Saxena et al. study. They identified the attachments of the soleus, flexor digitorum longus, tibialis posterior, and deep crural fascia as possible structures affected by the condition. Their results indicated that the soleus, flexor digitorum longus, and deep crural fascia attach most frequently in the area corresponding to the location of MTSS pain. Further, they reported that the tibialis posterior muscle did not attach in the symptomatic area in any of the specimens. The authors' conclusions supported the view that the soleus was “probably the major contributor to traction-induced MTSS” (p. 1060).

In the mid to late 1990s, magnetic resonance imaging (MRI) became prevalent as a diagnostic tool for identification of tissue lesions, and several studies were published that involved the use of MRI to diagnose tibial stress injuries. In 1997, Anderson et al. used MRI to evaluate the bone and soft tissues of 19 patients with MTSS. They concluded that “Patients with acute shin splints have a spectrum of MR findings, which suggests this clinical entity is part of a continuum of stress response in bone” (p. 827). This confirmed the notion that Clement proposed in 1974, that MTSS was a continuum of injury, rather than one specific injury.

In 1998, Beck presented the theory of tibial bending as a possible MTSS etiologic factor. He suggested that tight plantar flexors could cause the tibia to bend like a bow, which creates a compressive load on the posterior-medial surface of the tibia. The hypothesized relationship between tibial bending and stress injury was explained as follows:

Persistent and increasing strain on the porous bone during remodeling incites a positive feedback loop that re-stimulates remodeling. This results in a protracted hypermetabolic state within the bone. This chronic remodeling in the cortical bone, mediated via the periosteum (with or without periosteal injury), probably represents the pathologic lesion of MTSS. (p. 270)

Two years later, Kortebein et al. presented a report on MTSS that supported Michael and Holder’s soleus theory.

Since the soleus is the primary plantar flexor and inverter of the foot, it is theorized that the medial portion of this muscle must contract eccentrically as the foot moves from relative supination to pronation. Accordingly, this results in increased stress of the fascial origin of the medial soleus, possibly disrupting the Sharpey’s fibers that
traverse through the periosteum to insert in the fibrocartilaginous bone of the tibia. (p. S30)

Individuals who excessively pronate during the midstance phase of the gait cycle may place a greater eccentric load on the medial portion of the soleus, which may explain the widely-held notion that excessive pronation is a risk factor for MTSS.

The correlation between excessive pronation and MTSS symptoms has been studied extensively. Early studies reported that pronation, as measured by navicular drop and/or standing foot angle (SFA), was significantly correlated with MTSS. More recent studies (reviewed in Part 2) present contradictory findings with regard to the relationship between pronation and MTSS. In 1988, Messier and Pittala used high speed cinematography and anthropometric measurements to compare a group of subjects with MTSS to a non-MTSS control group. They found that maximum pronation was significantly greater in the subjects with MTSS, and the difference in maximum velocity of pronation was found to be even more significant.

Sommer and Vallentyne retrospectively evaluated athletes and dancers with MTSS to determine whether or not foot alignment was associated with the condition. They concluded that a standing foot angle (SFA) of <140 degrees and a varus alignment of the hindfoot and/or forefoot were associated with a history of MTSS. Bennett et al. retrospectively evaluated 125 high school cross-country runners for navicular drop. Comparison of runners who developed MTSS with a control group of uninjured runners demonstrated a significant difference in navicular drop (injured = 6.8 mm +/- 3.7; uninjured = 3.6 mm +/- 3.3). The researchers concluded that a pronated foot alignment was associated with MTSS. In 2004, Yates and White prospectively monitored 124 naval recruits, 40 of whom (35%) developed MTSS. Their results indicated a more pronated foot alignment in the MTSS group compared to the control group (P = .002). These four studies found meaningful correlations between pronation and the development of MTSS. Although this does not indicate that all individuals who exhibit excessive pronation will acquire MTSS, it does indicate that the risk is greater. These studies should not be interpreted to mean that pronation is the cause of MTSS, but rather is a possible contributing factor to its development.

In 2002, Couture and Karlson proposed one of the most comprehensive theories concerning the etiology of MTSS, which included tibial bending, bone remodeling, soleus strength and endurance, and pronation. The first part of their theory concerned bone remodeling, which agreed with Johnell’s 1982 conclusions.

Bone remodeling, or adaptation to mechanical properties in response to changes in loading patterns, increases metabolic activity of the bone. When a bone encounters a new, sustained mechanical stress, osteoclasts begin to remove the old bone matrix, thus creating tunnels in the framework of the bone. Osteoblasts then fill the tunnels with new bone matrix. In pathologic conditions, during the time between removing the old bone matrix and filling the tunnels with new bone, the bone cannot accommodate the continued loading adequately and microfissures result, which may progress to stress fracture.

The second part of their theory involved tibial bending and the plantar flexor muscles and agreed with the conclusions of Michael and Holder, Messier and Pittala, and Beck.

Contraction of the gastrocnemius and soleus muscle group can bend the tibia in much the same way that a taut bowstring bends a bow. Thus, high plantar flexion strength and decreased range of motion in dorsiflexion have been demonstrated in patients with tibial stress injuries. Conversely, a weak or fatigued muscle cannot dissipate mechanical stress effectively, so the stress is transmitted to the bone, thereby increasing the risk of injury.

The third part of Couture and Karlson’s theory addressed foot alignment in terms of pronation, standing foot angle, and forefoot/hindfoot measures. “Hindfoot and/or forefoot varus create an unstable point of contact between the foot and ground that is corrected by pronation of the subtalar joint, which in turn, increases the eccentric stress placed on the soleus”.

Couture and Karlson proposed that stress placed on the tibia leads to a chronic bone remodeling cycle, which results in microfissures that cause pain. The “increased stress” is likely attributable to tight and/or fatigued soleus and/or gastrocnemius muscles. If tight, these muscles may cause some degree of tibial bending. If they are fatigued, the tibia may be subjected
to greater loading. If excessive pronation occurs, an increased eccentric load is placed on the medial soleus, which could lead to fatigue and/or increased tension transmitted to the tibia, creating microfractures.

Bone mineral density at the site of MTSS symptoms was studied in 2003 by Magnusson et al. They compared 14 male athletes with MTSS, when symptomatic and after recovery, to 13 non-athletes. Bone mineral density was found to be 11% lower in the MTSS group in the tibial region corresponding to the location of pain. After recovery from symptoms, bone density was not lower than that of the control group. The researchers concluded that low bone mineral density may develop in conjunction with the symptoms, rather than being a causative factor for MTSS.

Up to 2004, synthesis of the available body of knowledge suggested that the pain associated with MTSS was likely due to stress microfractures in the medial tibia associated with a chronic bone remodeling response. The cause(s) of the overload remain unclear and may involve multiple factors. The soleus muscle likely plays a central role in creation of the continuum of injury reported in several of the cited studies. Tightness of the soleus (crural) fascia and the underlying bone suggest soleus involvement in the etiology of MTSS. Excessive pronation appears to exacerbate and/or accelerate development of the condition. Part 2 (March issue of ATT) will review the most current literature (from 2004-2007) concerning the etiology of MTSS and provide recommendations for its management.

References


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