Medial Tibial Stress Syndrome

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KEYWORDS
• Medial tibial stress syndrome • Stress • Reaction • Tibia
• Shin

INTRODUCTION

The first description of medial tibial stress syndrome (MTSS) was in 1958. Devas[1] published the first study and described signs and symptoms of what he termed *stress fracture at the tibia* or *shin soreness*. Other terms like *medial tibial syndrome*,[2] *tibial stress syndrome*,[3] *shin splint syndrome*,[4] and *medial tibial stress syndrome*[5] have followed. Numerous definitions for this condition have been described by experienced sports clinicians, highlighting the vague understanding of this condition. Yates and White[6] most accurately described MTSS as "pain along the posteromedial border of the tibia that occurs during exercise, excluding pain from ischemic origin or signs of stress fracture." The purpose of this review is to summarize the known data about MTSS and to give a formal definition and treatment algorithm, allowing for timely diagnosis and treatment.

RELATED PATHOANATOMY AND PATHOPHYSIOLOGY

MTSS is broadly defined as painful symptoms on the medial aspect of the tibia, often located at the middle or distal portion. There is disagreement about the etiology of MTSS. Numerous theories relating functional anatomy and pathologic biomechanics are the foundation for the development of MTSS. Several cadaveric studies evaluate the relationship between the exact location of the pain and the associated anatomical structures. The traction theory was first published by Devas in 1958,[1] stating that traction to the periosteum can be caused by any strong calf muscle. Namely, the muscle causes tension of the periosteum causing inflammation and eventual bone production. Michael and Holder[7] performed dissection on 14 cadaveric specimens. They concluded the fibers of the soleus muscle insert 4 inches proximal to the medial malleolus. The pain is formed from a fascial covering over the deep compartment of

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Clin Sports Med 31 (2012) 273–290
doi:10.1016/j.csm.2011.09.008 **sportsmed.theclinics.com**
0278-5919/12/$ – see front matter

the leg. Saxena and colleagues[8] believed the tibialis posterior (TP) muscle was the cause for MTSS. By dissecting 10 cadavers, they identified the muscle origin 7.7 cm proximal to medial malleolus and the intersection of the TP and the flexor hallucis longus (FHL) 8.2 cm proximal to the medial malleolus. This intersection point is the source of pain in MTSS and is associated with FHL flexion. Both these studies suggest a traction theory as the pathologic condition.

Beck and Osternig[9] concluded that if a traction etiology was implicated in MTSS, the soleus and the FHL (not the TP) are involved. They dissected 50 legs and found no fibers of the TP muscle on the distal half of the posteromedial border of the tibia. In the upper half of the distal tibia, fibers of the soleus muscle and FHL muscle were abundant on the medial border. Few muscle fibers of the soleus muscle or any other muscle were found at the distal part of the tibia, where MTSS complaints are commonly felt. Garth and Miller[10] suggested that the flexor digitorum longus (FDL) is the cause of MTSS. They evaluated 17 runners with suspected MTSS and noted pain associated with flexion of the metatarsophalangeal joints and a relative weakness of the FHL muscle. They also suggested that MTSS may be associated with mild claw toe deformity. Recently, Bouche and Johnson[11] added support to this theory. Using 3 cadaveric specimens, they applied tension to the tibial periosteum through the soleus, TP, and the FDL muscles. As tension was increased, strain in the tibial fascia and periosteum increased in a linear manner.

Another explanation for the etiology of MTSS is the ability of the calf muscles to cause repeated bending or bowing of the tibia, thereby causing a stress reaction and periosteal reaction.[12] This theory was originally described by Devas[1] and Beck[13] as a possible explanation of posterior calf pain. Several studies found that repeated bending of the tibia causes adaptive reaction of bone, predominantly where bending forces are greatest.[14,15] This is located at the narrowest part of the diaphysis of the tibia, between the middle and the distal third.[16] As described by Wolff's law, repeated loads applied to bone initiate a cascade of signal transduction, detected by the cellular components of bone.[17,18] This response will repair microdamage to a certain threshold. Repetitive load or stress may cause the microdamage to rise above a threshold that escapes repair.[18]

To study the biomechanical properties of the tibia in stress fractures and MTSS, Franklyn and colleagues[19] looked at a cohort of military recruits (men and women) suffering from stress fractures and MTSS. The authors compared them with 2 gender-mixed control groups: aerobically active and sedentary. Using tibial scout radiographs and cross-sectional computed tomography, the aerobic control group had larger cortical cross-sectional area than the inactive subjects in men. Similarly, MTSS and tibial stress fractures had a smaller cortical area than aerobic controls. They calculated that aerobic controls were better adapted to axial loading, torsion, and bending rigidity than subjects with MTSS and tibial stress fractures. They also showed that the MTSS group had lower cross-sectional cortical area compared with aerobic controls in both men and women. This suggests that MTSS is not only a soft tissue problem but may involve the adaption of the distal tibial cortex to repetitive stress.

Studies have also suggested that lower muscle strength has a negative influence on the bone adaptation process. This pathologic condition develops when weak leg muscles cannot oppose the bending forces on the tibia, resulting in greater strain on the tibial cortex.[20,21] Other investigators have suggested a theory in which the adaptation of traction and bony overload is challenged by the traction of the soleus and flexor hallucis longus muscles on the periosteum.[13]

Histologic analysis is also inconclusive. It is thought that tension applied on the periosteum will result in an inflammatory reaction and could explain the pain. Two