
Paul Monaro, Sports & Musculoskeletal Physiotherapist, MAPA, MSPA, MMPA. 2013
Contact: info@cssphysio.com.au ; Further information: www.cssphysio.com.au

This paper is a clinical review of the available literature on medial tibial stress syndrome. It is not a critical analysis - no comment is made on the quality of the included papers. Some areas of the condition, such as pathoanatomy, have been extensively studied. Other areas, including treatment, have had limited investigation, and the research quality is generally poor (32). Current management practices are based on anecdotal evidence, experience, opinion, and findings from the limited data available.

Abstract

Medial tibial stress syndrome is one of the most common acute and overuse leg injuries in runners and ballistic sports. It accounts for up to 50% of all lower leg injuries in certain sports. It is characterised by dull pain over the lower half to one third of the posteromedial tibial border. The pathology is still not clearly understood, but it is thought to represent a bone stress reaction. Over twenty possible contributing factors are described in the literature, with the most attention given to foot biomechanical faults, training errors, anatomical and biomechanical factors, past injury history, and training surface. Females are consistently shown to be at greater risk than males. Numerous treatments are recommended, however there has been limited quality research in this area. There is some evidence for the benefits of shoe inserts and orthotics, however the results are conflicting. Training factors, including adjustments to loading, graded exercise, cross-training, muscle strengthening, and biomechanical correction, have anecdotal evidence or clinical endorsement, but limited supporting evidence.

Introduction

The term medial tibial stress syndrome (MTSS) was originally suggested by D. Drez, and quoted by Mubarak et al in 1982 (24). The authors felt the pain was due to periostitis – an inflammation between soft tissue attachments and the bony periosteum. In 1986 Detmer (4) classified posteromedial shin pain as being due to one of 3 possible causes:

1. A primary bone problem (including stress fracture).
2. Symptoms adjacent to bone at the periosteal / fascial junction*
3. Symptoms due to deep posterior compartment syndrome – a build-up of pressure within one or more muscle compartments.

*Detmer felt the pain was probably due to traumatic avulsion of the periosteum off the bone, & most likely due to the action of soleus, as it partially attached medially into the crural fascia. In his operative series, adipose tissue was consistently found between the periosteum and the underlying bone. At the time he advocated periosteal cauterisation (scarring the periosteum back to the bone) and fasciotomy, as the treatment of choice for chronic cases (4). This is rarely practiced today.

There have been numerous studies into MTSS. A recent Pubmed search identified 55 articles dating back to 1982, which used the term medial tibial stress syndrome, Many studies have been in military recruits, where the findings may not be entirely applicable to the athletic population. Military personnel undergo different training techniques, have different body composition, use different footwear, and generally have lower levels of fitness compared to athletes (8). This makes comparison between groups difficult.

Incidence:

In military studies, rates vary between 3 to 35% (11,20,27,36). In the Australian Defence Force, the rate was found to be 15.4% (36). In sport, MTSS is said to be most common in runners and in ballistic sports,
particularly sprinting, hurdling, gymnastics, the football codes, hockey, basketball, and dancing (4,7,17). Varied incidence rates are reported within and between different sports. MTSS is said to represent up to 50% of all lower limb injuries in selected populations, and 6-17% of all running injuries (3,13,14,20). It is the first or second most common running injury (13). Similar statistics are quoted by Hubbard et al who say 10% to 20% of all runners will experience a bout of MTSS, & that it accounts for up to 60% of all overuse injuries seen in the leg (8). 94.4% of cross country athletes reported a history of exercise related leg pain (14). It is said to account for up to 22% of all injuries in aerobic dance (20). However, overall rates can be difficult to quantify. In one study of navy recruits, 70% of people who developed MTSS did not seek medical intervention (20).

Field Hockey: This sport is known to have a high incidence of MTSS, particularly since the widespread introduction of artificial turf. The incidence of ‘exercise related leg pain’ in field hockey was reported to be 38.9% (14) The following data comes from a systematic review of field hockey injuries known as the ‘Monash Report’ (17). MTSS accounted for 14% of all acute & 16% of all chronic injuries. Of ‘severe’ injuries in field hockey, 7% were MTSS. It represented 10% of chronic complaints. It represented 4.1% of all male & 22% of all female injuries across the sport. It accounted for 5.5% of all injuries on grass & 13% on turf.

Males versus Females: Females are consistently found to be at greater risk. In their study of naval recruits, Yates and White reported an incidence of 58% in females compared to 28% in males (20). Newman et al found among Australian Defence Force recruits, females were 3 times more likely to develop MTSS (36). In a study of high-school cross country runners, 12% of all runners, and 19.1% of female runners had MTSS (1). The incidence in non-professional track & field was 19.7%, with the rate for females being slightly higher at 22.2% (13). In recreational runners, the incidence was 10.7% for males & 16.8% for females (1).

Symptomatology and History:
The typical description of MTSS is dull pain over the posteromedial lower half to one third of the tibia (27,32,36). Occasionally the pain will be anteromedial (19). In the early stages of the disease, symptoms will be present at the beginning of activity, and subside as the athlete warms up. (7,11,15). As the condition becomes more severe, symptoms may persist for longer and even throughout the activity (7,15). In the most severe cases symptoms persist on cool-down and may even be present at rest. Pain may last for several hours or days after exercise & be present during normal activities including walking (20,25).

There is often an increase or change in training load which precedes the onset of symptoms. A comprehensive history should be obtained to evaluate the athlete’s weekly exercise routine, running mileage, intensity, pace, terrain, & footwear (7). Careful attention should be paid to recent changes in training (7). A common scenario will be an increase in intensity or duration, or running on hard or uneven surfaces (7). Past injury history is important, as previous lower limb injury is a known risk factor for developing MTSS (3,7,8,11,12,14,16). Reported recovery time for the condition ranges from 4 weeks to 18 months, with recurrences being common (36).

Clinical Features:
Tenderness will be palpable over the distal one third to half of the posteromedial tibia, with or without mild swelling (15,16). There will also be tenderness in the surrounding soft tissues (36). Swelling, if present will usually be pitting in nature, and will be localised over the area of tenderness. This differentiates swelling due to MTSS from other typical causes of lower leg oedema (36). In one study
palpatory tenderness was found to be more sensitive to diagnosis than pain on hopping or on percussion of the area (11). A recent study found that tenderness and swelling may be present up to several months before the onset of symptoms (36). In this study of 1st year military recruits, the presence of tenderness alone was found to indicate a 4.63 times greater likelihood of developing MTSS, while the presence of oedema indicated a 76.1 times greater likelihood (36). Around 75% of subjects with tenderness went on to develop MTSS. Of the 25% who did not, it could be speculated that healthy adaptation led to recovery over time, or that they had residual symptoms from previous MTSS.

Other important examination findings may include anatomical & biomechanical factors including genu valgum or varum, tibial torsion, femoral anteversion, foot arch abnormalities, and leg length discrepancy (7). However there is no clear data on how important these factors are. It is also recommended to test for inadequate calf flexibility or weakness, and quadriceps and hamstrings inflexibility or imbalance (7). In consideration of kinetic chain factors, it would seem rational to assess lumbo-pelvic-hip control. Tests include the stork, and double and single leg bridge tests (7). Where the diagnosis is not clear, it may be necessary to examine the spine & pelvis to exclude these areas as a source of symptoms (7). For over-pronation of the foot and ankle, the tests most often quoted are the navicular drop and foot posture index (12,19,20) Calf flexibility should be tested in knee extension and flexion, as should 1st MTP joint extension (19). Restriction of the MTP joint may be structural or functional (due to muscle tension or imbalance), so testing in weight-bearing and non-weight-bearing is advisable.

**Differential Diagnosis:**
The most likely alternative diagnoses will be:

1. **Stress fracture:** Tibial stress fracture will usually present differently to MTSS, in that there will be increased localisation of tenderness (11,15,16,27), particularly on weightbearing (40), and usually over the mid or anterior third of the tibia (15,16). There is also greater likelihood of the presence of night pain (11,27), pain on percussion (11), pain at rest, and on walking (27).

2. **Compartment syndrome:** This will be characterised by exercise related pain, with or without neurological symptoms (5,15). It is more commonly associated with the anterior compartment, and the patient will point to the anterolateral lower leg. A posterior compartment syndrome will generally be described as being further posterior, and more over the soft tissues, compared to MTSS (4). The symptoms should resolve within minutes on cessation of activity, with no post-exercise tenderness (5,15). However pain lasting for up to two days has been observed clinically. The possibility that MTSS and compartment syndrome may co-exist cannot be discounted (11).

There is also mention in the literature of the possibility of either nerve entrapment or popliteal artery entrapment. These conditions are thought to be rare causes of distal shin pain and the history should exclude these diagnoses (15).

**Imaging:**
The diagnosis of MTSS is a clinical one and usually straight-forward (11,15) If there is uncertainty, and particularly concern about the possibility of a stress fracture, bone scintigraphy & MRI are the investigations of choice (11,15,27). However research has shown that there will be many false positives with these investigations (11,15) Up to 80% of asymptomatic athletes undergoing bone scintigraphy had
pathological findings (15). MRI, which shows the presence of periosteal & bony oedema, also has many false positives (15). CT scanning has also been used and demonstrated the presence of cortical osteopenia associated with MTSS (15,27). It has higher specificity but lower sensitivity than MRI (11).

**Anatomical Findings related to MTSS:**

**Soft tissue attachments in the region of symptoms:**

There has been speculation in the past that the symptoms of MTSS arose due to trauma to muscle attachments, particularly to tibialis posterior or soleus. However more recent studies have reported the absence of any direct muscle attachments to the area of bone where the symptoms of MTSS are felt.

**Tibialis Posterior (TP):** Support for the role of TP in MTSS includes the large role it plays in pronation control (19). However in anatomical studies there were no fibres of TP found to be attached to the distal half of the tibia (15).

**Soleus:** This muscle was found to attach to the proximal half of the tibia only (18). However it was still felt that soleus may be the major contributor to MTSS, possibly through its attachment to the deep crural fascia (13,16,27). The medial part of soleus is a strong plantar flexor & invertor, and eccentrically contracts to prevent pronation (27). TP, flexor digitorum longus, and possibly gastrocnemius and flexor hallicus longus (FHL) are other muscles that may contribute through this connection (2,16,19,27).

**Deep crural fascia (DCF):** In a cadaveric study (where the average age of the subjects was 83.7 years) it was found that the tibial fascia was the only structure to insert onto the medial tibial crest from the ankle to the knee (2). The most distal part of the fascia was found to be thicker and stronger as it passed distally to form the flexor retinaculum (25). This part of the fascia, proximal to the retinaculum, is termed the soleus bridge (25).

**Histological and Pathological Findings in MTSS:**

**Inflammation:** Inflammation was generally not found in biopsy studies (10,15,27). However an inflammatory response of the periosteum was proposed as being a possible cause of symptoms in the acute stage (15). Degeneration, demonstrated in biopsies, involved the chronic stage (15). When oedema is present, this may indicate “periosteal or bone stress inflammatory reaction” (36).

Current opinion is that the disease is caused either by a traction injury of the crural fascia where it attaches along the posteromedial lower half of the tibia, or to a bone stress reaction. The more recent consensus is bony stress (25,32,36). Both theories are described below:

1. **Fascial Traction Theory** (2,13,15,16,18,19) This theory maintains that tension through the deep, and possibly the superficial plantar flexors, result in traction injury at the DCF attachment to the periosteum (2). The DCF traction injury model “seems viable based on anatomical findings” (18). Soleus, FDL, TP (& possibly gastrocnemius & FHL) are the muscles most likely to contribute (2). Some authors feel the soleus attachment to the DCF is the most likely mechanism (3,13,16,25). During the stance phase of running, eccentric overload of the soleus and other muscles may lead to excessive fascial tension. This may occur in an effort to counter mid-tarsal & subtalar pronation motions (2), or due to muscle fatigue. As tension increases, a tenting effect (2) that exerts tension on tibial fascia & periosteum increases linearly (2,15).

   DCF inflammation has reportedly been found in biopsies obtained from individuals with recalcitrant MTSS (18). This is a possible stress reaction of the tibial fascia, or ‘fasciitis’ (2). Some authors suggest that fascial traction results in a traumatic stripping of the periosteum off
the bone, resulting in subperiosteal bone stress or inflammation (4,19,25). This may imply both soft tissue and bony trauma.

2. Bone Stress Theory: According to Wolff’s law & the Utah paradigm (11) stress and strain will result in bony adaptation. However if strain is greater than the threshold for repair, microdamage can accumulate & outstrip repair (11). There is a possibly that bone resorption outpaces replacement, and this may be a factor in the early stages of the condition (11). MTSS may then represent a bone stress reaction which has become painful (6). Several studies support the notion that bony overload is the primary factor (2,10,13,15,20,25,27,32,36). Bony overload could result from direct mechanical forces through the bone. The posterior tibia is a concave surface, and bending forces on foot contact could result in compressive overload. It is also possible that bony bending forces are exacerbated by muscular contractions. In theory, the calf muscles could cause repeated bowing of the tibia, resulting in bone stress and a periosteal reaction (15). Their attachment at the superior half of the tibial shaft could result in greater bending forces distally. It has also been speculated that eccentric fatigue of soleus could contribute to bony overload through increased tibial bending (3), implying a protective function to muscular forces. Repeated bending could cause an adaptive reaction, especially at the narrowest part of the diaphysis, where the bending forces are the greatest (15). There are several factors supporting the bone stress theory:

(a) Smaller tibial cross-sectional area: Increased bending occurs at the area with the least cross-sectional area, and this is the region of MTSS development (15,16,20). The theory is that chronic repetitive loads induce tibial bending that causes bony stress close to the site of maximal bending.

(b) Tibial bending is accepted as a cause of stress fracture (2), which could be continuum of MTSS (see below**).

(c) Bone density is adversely affected in MTSS subjects (6,9,11,26), indicating that MTSS is not purely a soft tissue injury (6). The bone changes due to exercise are metabolic, characterised by initial bone porosity due to osteoclastic channelling on the compressed concave posteromedial border, followed by laying down of new bone (20). With long-standing MTSS the affected part of the tibia is 15% more porous than in controls, and 23% more so than athlete controls (20,26). On high resolution CT the tibial cortex is found to be osteopenic (11). This was present in both patients and asymptomatic athletes. Patients with MTSS had decreased bone mineral density compared to controls (26) & this returned close to normal with resolution of symptoms (9).

(d) The bone remodelling sequence at the start of an exercise programme commences at approximately five days, and the bone is relatively weakened for the first eight weeks (20). The reasonably early development of symptoms in unconditioned subjects found in Yates & White’s study (at an average of 3 weeks) would tend to support the bone stress reaction theory (20).

(e) On triple phase bone scans in patients with MTSS, the last scan was abnormal, showing that the bone and periosteum were involved (11)

(f) MRI has been shown to demonstrate bone marrow oedema & increased signal along the periosteum in MTSS sufferers (11).

Following on from the bone stress reaction theory, it has been suggested that MTSS may represent an early stage in the development of a tibial stress fracture. This is discussed below.

**Theory of possible continuum toward stress fracture:
Some authors suggest a continuum – from asymptomatic stress reaction, to symptomatic stress reaction, to stress fracture (2,3,36). The evidence quoted to support this argument includes:
1. The symptomatology of the two conditions are often similar. With progression of the injury, pain lasts for longer and may be present at rest (11). Pain at rest is a particular feature of stress fracture.

2. For both men and women, bone cross sectional geometry was similar for both MTSS & stress fracture groups, suggesting MTSS is not just a soft-tissue injury, but involves an effect on cortical bone in addition to the soft tissue and periosteum (6). Both MTSS & tibial stress fracture subjects had reduced cortical cross sectional area compared to controls (6,15). There is evidence of low bone mineral density (BMD) at the junction of the middle and distal third of the tibia in people with MTSS (9,27). This may develop in conjunction with the symptoms and be a cause rather than effect. In their study of athletes, Magnusson et al (2001 & 2003) looked at 14 males who developed symptoms at a mean of 24 years of age (26), & who were followed up for a mean of 6 years (9). They were examined with bone scintigraphy, & dual X-Ray absorptimetry. BMD in other areas of the body in MTSS subjects was often higher than non-athlete controls, but lower than athlete control (26). This could indicate a tendency for MTSS subjects to reduce their activity levels due to their symptoms. It could also indicate that these subjects had a naturally lower BMD and this contributed to the development of MTSS. Follow-up BMD after resolution of symptoms showed that in the region of bone related to the MTSS, density was similar to non-athlete control, but lower than athlete controls (9). Again, this could be due to residual changes, or indicate overall lower density. According to the authors, this suggests that the changes are transient and not inherited (9)

3. In MTSS the bone is more porous than in controls (6), making it more vulnerable.

4. Both stress fractures & MTSS are more common in women (6). Females were said to have 1.5 to 3.5 times the risk of progressing from MTSS to a stress fracture (7).

5. In one study, 46% of the MTSS group had experienced a past stress fracture versus 17% of the asymptomatic group (8).

6. In a case series of three army recruits who developed stress fractures, initial bone scans were negative (39). In these cases, bone tenderness was an early sign of stress fracture, as may be the case with MTSS.

Arguments against the continuum theory:
Some authors disagree with the continuum theory (2,15). Their arguments include:

1. Stress fractures in the posterior distal tibia was said to be unusual (15), and the site of tibial stress fracture typically more proximal than that seen with MTSS (16). However Yates & White stated that tibial stress fractures occurred more commonly in the middle or distal third on the posteromedial border (20), in disagreement with the above authors. Radiographic studies have documented lesions from the distal third to the proximal third of the tibia (39,40), while MTSS is never experienced proximally.

2. In one study (Aoki et al 2004, cited by 11) athletes kept running after the diagnosis of MTSS, and on follow-up MRI there was no evidence of stress fracture. This could mean that only a small percentage of MTSS sufferer’s progress to stress fracture. It could also support the argument that the two processes are different.

3. In one military study the peak of stress fracture onset was 5-8 weeks after the onset of training (Burr 1997 cited by 6). This was in subjects who were generally untrained to begin with. In trained athletes, as opposed to untrained military recruits, stress fractures are often seen years into their training, due to a gradual increase in training load (6). With MTSS, the opposite tends to be the case - there is evidence of a protective effect with a greater number of years of training (8,15). Interestingly, in a separate military study, the onset of MTSS symptoms in a group of naval recruits was an average of three weeks (20), and symptoms lasted throughout the training period of 10 weeks. In military recruits undergoing basic training it can be theorised that those who will develop MTSS will do so within the first 3 to 4 weeks, whereas those developing stress fractures will be diagnosed later, at 5 to 8 weeks.
4. Clinical experience tells us that MTSS tends to be a bilateral condition, whereas stress fractures are often unilateral. In a study of naval recruits, close to 80% of subjects who developed MTSS had bilateral symptoms (20).

**Contributing factors to MTSS:**

Over the years many different causes and contributing factors to MTSS have been proposed:

1. **Over-pronated foot type:** (3,7,8,10,11,15,16,19,20,27). This has received the most attention of all the possible contributing factors to MTSS. In the literature, there is a strong argument to suggest this may be the most significant factor. Moen et al (2009) say there is Level 1 evidence (11). Excessive or poorly timed pronation has been theorised to cause the foot intrinsic & larger extrinsic anti-pronation muscles to fire for longer while contracting eccentrically (19). This leads to earlier muscle fatigue, and increased force being absorbed through the tenoperiosteum and bone (19). TP & tibialis anterior support the medial arch, and both work to control pronation. The entire posterior compartment is stressed with excessive pronation. TP is the most biomechanically efficient to reduce pronation, followed by FDL (19).

   Signs of excessive pronation include excessive or prolonged internal rotation of the leg, eversion of the calcaneus, abduction of the mid-tarsal joints, apropulsive gait, abnormal phasic activity of the muscles (19), and increased varus of forefoot or hindfoot (16). Bennett et al (2001) found a trend toward increased calcaneal eversion (1).

   Various definitions are given for apropulsive gait, with the basic description being that it arises due to inadequate supination at toe-off. This will result in compensatory movement abnormalities further up the kinetic chain, such as excessive knee flexion, forward body lurching, forward head bobbing and excessive arm swing (41).

   To assess pronation, most studies cited the navicular drop test (1,3,11,12,13,15). The average was 6.8mm in MTSS subjects, compared to 3.7mm in asymptomatic subjects (1,15). A study over five university sports found significant difference in navicular drop, with a mean difference of 2mm in those who developed exercise related leg pain (14). In a study of 66 non-professional track & field athletes, there was a significant association between navicular drop & MTSS: (3.0 - 9.5mm in asymptomatic; 6.8 – 8.9mm in those with MTSS) (13). They found no significant difference in rearfoot or calcaneal angles.

   In a study of Australian naval recruits (20), the foot posture index (a grading system from -2 (supinator) to +2 (pronator) was recorded. Normal was considered to range from +1 to +5. The MTSS group scored higher on this index: +7.45 on average, compared to +5.47 for asymptomatic individuals. For a pronated foot type the relative risk of developing MTSS was 1.7 (20). As well as static measures of pronation, certain authors felt the timing of pronation was the critical issue. In some individuals, overall pronation may appear normal, but occurs late in the stance phase, and this results in the foot pronating when it should be supinating (19). Maximal velocity of pronation may be important (3). Pronation at propulsion and in all phases of gait was a negative predictor of MTSS by Tweed et al (19), who felt timing of pronation was the more important factor. They also found that with static measurement there was a significant difference between neutral & relaxed calcaneal stance positions. At propulsion, the foot should be re-supinating. TP assists with this. If this is delayed, there could be overloading of TP (and other muscles including the medial soleus) as it attempts to decelerate rapid internal rotation of the tibia (19). The authors found pronation at propulsion was not a predictor of MTSS, but suggested apropulsive gait was the problem, & this would likely overload TP. Another possible cause of altered timing of pronation is a plantarflexed 1st ray (19). In a patient with a statically supinated foot, in early stance the 1st metatarsal head contacts the ground before the lateral border of the foot. This causes the subtalar joint to supinate when it should be pronating, to bring the lateral border to the ground. This will result in weight transference from lateral to medial on propulsion, & the foot pronating when it should be supinating (19).

2. **Orthotics:** In the Hubbard et al study, subjects who developed MTSS were 1.5 times more likely to be wearing orthotics (8). 53% of those who wore orthotics developed MTSS, compared to 21% of asymptomatic subjects who did not (8). In another study, runners who reported orthotic
use were four times more likely to report a history of lower limb injury (12). These findings could be interpreted in two ways:
(a) People wearing orthotics are more likely to have biomechanical abnormalities, or may have suffered a prior injury, and so are more at risk already.
(b) Pronation is a natural method for absorbing shock. Anti-pronation orthotics may lead to increased shock and contribute to MTSS in certain individuals. Tweed et al found that pronators were less likely to develop MTSS (19). Hyper-pronation is the most likely reason for people to be prescribed orthotics, so the findings above (8,12) suggest more evidence is required to show that orthotics are in fact of benefit in preventing MTSS.

3. **Muscle strength factors.** Both muscle weakness and muscle imbalance have been suggested as possible causes:
(a) Increased plantarflexor strength has been proposed to lead directly to excessive tibial stress (3,15,16).
(b) Reduced lower limb strength could have a negative influence on the bony adaptation process (15), as bone and muscle strength are known to develop together. Also, weak muscles may inadequately oppose tibial bending forces, resulting in increased strain to the cortex (15,29). Early muscle fatigue due to endurance deficits may be another factor leading to increased bony stress (7,15). Decreased lean calf girth has been suggested to be a factor in reduced shock absorption, and reduced ability to bear loads (10,11,15,30,36).

4. **Biomechanical / anatomical factors:** Numerous factors have been proposed, including genu valgum or varum, tibial torsion, femoral anteversion, foot arch abnormalities, and leg length discrepancies (7). Tibial varum could lead to increased compressive loading through tibial bending in a medial as well as posterior direction (37). However no clear evidence is available for any of these factors.

5. **Running biomechanics:** (5,8,19). Various factors relating to running technique have been proposed as possible contributing factors:
(a) Excessive heel striking. A significant increase in anterior compartment pressure was found in positions of full dorsiflexion and knee extension – the position of a hindfoot striker (5). In this study pressures and symptoms were significantly reduced, and running distances increased in changing from a hindfoot to a forefoot strike in 10 military recruits. The authors hypothesised that forefoot running could possibly improve other lower limb stress injuries such as stress fractures & MTSS (5). Excessive heel striking could also have been a factor in that increased stride length was identified in female naval recruits who developed MTSS (20).
(b) Excessive forefoot landing: There is evidence that forefoot landing increases strain on the posteromedial tibia (10). However forefoot striking has also been shown to reduce ground reaction forces, stride, ground reaction time, and anterior compartment pressures (5).
(c) Apropulsive gait: In one study, this was proposed as a very strong predictor of MTSS, with up to 800 times the risk (19). In treadmill analysis the authors found subjects with MTSS displayed early heel lift, abductory twist, and apropulsive gait (19).

6. **Muscle balance factors:** Proposed causes include quadriceps and hamstrings inflexibility or imbalance (7,19), and core strength inadequacies, particularly at the hip & pelvis (7). Pronation at the foot has long been considered an important contributing factor to many lower limb disorders. In the last ten years, a lot of interest has been devoted to control factors at the hip. Poor hip control in walking and running can result in torsion through the entire lower limb, and could even lead to increased or delayed subtalar pronation. A finding that subjects who developed MTSS were more likely to have increased hip rotation range (30), and ankle plantar flexion range (8), may have had a relationship with muscle control factors, as hypermobile joints require additional muscular support.

7. **Load:** A sudden increase in training intensity or duration is a well recognised contributing factor (3,7,8,16,19). Training on hard or uneven surfaces has been suggested (19) but there is no firm evidence. Training errors may be a major factor in up to 60% of cases. This includes an abrupt
increase in intensity, duration or frequency (Fredericson, et al cited in 3). The author quotes an increase of greater than 30% of initial training mileage within a year as being significant (Kortebein et al cited in 3). Another factor may be hill running (3).

8. Activity history: Runners who had been running for less than 8.5 years were more likely to develop symptoms (31). In an athlete study, MTSS subjects had an average 5.3 year running history versus 8.8 years for asymptomatic subjects (8). Adaptation and strengthening of the tibia and / or soft tissues may develop over several years to protect against MTSS.

9. Younger Age: This was quoted as a factor (6), and may relate to activity history.

10. Reduced calf flexibility: This is often suggested as a contributing factor (7), but has not been proven by research. In one study, it was found that there was a difference in talocrural range of motion with the knee extended in those with MTSS (19). The authors suggested gastrocnemius tightness “amplifies the possible role of over-pronation by increasing traction on the soleus origin” (19). The midfoot & forefoot compensate for the decreased dorsiflexion during midstance when the leg moves over the stationary foot to achieve maximum dorsiflexion just before heel lift (19). However in other studies, reduced dorsiflexion was not found to be a cause of increased pronation (20), and reduced flexibility was not found to be a factor in MTSS development (14).

11. Hallux limitus: One study identified restricted 1st MTP joint extension as a factor in the development of MTSS (19). Restriction can be due to soft tissue or bony causes. This may affect toe off and result in an apropulsive gait (19).

12. Increased plantar flexion range of motion: (8,10,15): In a study of soccer, tennis & volleyball athletes, MTSS subjects had an average plantar flexion range of 46°, compared to 40° in asymptomatic subjects (8).

13. Increased hip rotation range: There is said to be level 2 evidence for increased internal and external rotation as a finding in those with MTSS (11). The average internal rotation range was greater by 8 degrees and external rotation by 12 degrees in a group of Australian army recruits who developed MTSS (30). Moen et al felt that biomechanically it is possible that both increased and decreased hip internal rotation can influence running in such a way as to excessively load the posteromedial tibia (10).

14. Altered bone density: Low regional bone density was found in MTSS athletes, & this returned to normal with recovery of symptoms at an average of 5.7 years (26). However it is conceivable that this could be an effect of the injury rather than a cause. Reduced medio-lateral width & small cross-sectional area of bone have been found to be risk factors for stress fracture (6,38). In a military study, female MTSS subjects had smaller bi-malleolar width than active controls (6). In males, the best predictors of injury were ‘low section modulus’ & low cross-sectional area (6,38). In females, section modulus & distal tibial width were the significant parameters (6). Inadequate calcium intake has been suggested as a possible factor (3), but this has not been proven by research.

15. Increased BMI: It would seem logical that increased loading due to weight factors would lead to greater tibial stress. Several authors quote increased BMI as a contributor (6,10,11,12,15). Interestingly, a BMI greater than 21 (with 21-25 considered within the healthy range) was found to be significant (15). One study found a relationship between increased BMI & longer recovery times (Judex et al cited in 10). One study found no significant relationship between BMI and the development of MTSS (13).

16. Body type: One military study found the shortest 25% of females had increased risk compared to the tallest 75% (20). However this could have been related to stride length, with shorter recruits being expected to keep up with taller ones during training (20). In athletes, tibial length was not found to be a factor (6).

17. Female sex (6,7,8,10,11,20): There is said to be level 1 evidence between female sex and risk of MTSS development (11). In a study of Australian naval recruits, 52.9% of female recruits
compared to 28.2% of male recruits developed MTSS symptoms (20). In study of cross country runners females were 5.46 times more likely to develop symptoms (1). 4.1% of male, and 22% of female field hockey injuries were due to MTSS (17). There was a suggested relationship between higher risk for females and bone density factors. Females had a higher incidence of reduced bone density and osteoporosis, particularly related to hormonal imbalances & the female athletic triad (osteoporosis, amenorrhea, and disordered eating) (7,8,11).

18. Previous lower limb injury (3,7,8,11,12,14,16): Hubbard et al found a past history of MTSS or stress fracture was most predictive (8). In their study 87% with MTSS versus 16% without, had a past history. 46% of the MTSS group had experienced a past stress fracture versus 17% of the asymptomatic group (8). They speculate these findings could relate to bone mineral density (8). In another study previous lower limb injury created a two times greater risk of developing MTSS (12). A two times greater risk was also found in a study of non-professional track & field athletes, but not this was not statistically significant (13).

19. Footwear: Several authors suggest the type or quality of footwear may play a role in symptom development. A recent change in footwear was found to be a risk factor (15,19). The quality or condition of the shoes may play a role (3,8,15,16,17). One study suggested that running in wet shoes may also heighten the risk, due to a reduction in shock absorption (7). This has not been explored in field hockey, a sport with a known high incidence of MTSS, and where most games are now played on water-based artificial turf.

20. Surface: The type of playing surface is a possible factor. Running on hard, irregular or inclined surfaces is thought to increase the risk (3,15,16,17). This may be because such surfaces increase eccentric loading (20). A recent change in surface or terrain may be another factor (15). The composition & grade of the surface are other potential factors (8). Higher loading was reported on the front of the foot on Greenset versus clay in tennis players and it was thought this could be a potential risk factor due to hyperpronation (4). In a study of five sports (cross country, running, field hockey, volleyball & soccer), soccer had the lowest incidence of ‘exercise related leg pain’ (14). The authors speculated this may have been because the university soccer teams trained and played all games on natural grass, compared to the other sports where artificial turf was used (14). It was suggested that the ‘unyielding nature of synthetic surfaces’ may be a factor in MTSS in hockey players, where shin pain was more common from turf than natural grass (17). However a comparison between new generation artificial turf & natural grass in male and female soccer players found no significant differences in the rate of MTSS (21). Limited studies have been conducted on synthetic surfaces used for hockey. Physiological response testing (looking at heart rate, lactate threshold, etc.) suggested that more effort was required for slow jogging on turf compared to grass (De Michele et al cited in 22). Players also reported increased perceived physiological effort with running on turf (Andersson et al cited in 22), and fatigue was associated with increased risk of injury (22). Potentially the ‘inferior impact attenuation properties’ of artificial turf could contribute to the formation of micro-damage and the development of chronic injuries (22).

21. Multiple risk factors:
Combined factors which lead to a greater likelihood of developing MTSS included female sex, increased pronation, increased hip internal & external rotation, a BMI greater than 21, decreased lean calf girth, pervious injury history & inexperience (15). Combined navicular drop & female sex had 76% accuracy in predicting the risk of developing MTSS (1). Hard surface & pronation combined resulted in increased eccentric loading of the deep plantarflexors (2).

Management:
Depending on the individual, there are several factors which may be of benefit in the treatment of MTSS:

1. Load management: Suggestions from the literature include:
- Decrease running frequency & intensity by 50% (7). The American College of Sports Medicine recommends at least 7-10 days of rest from painful activities to treat MTSS (16). A graded increase back to full training would then be rational.
- Avoid hills, uneven surfaces, or very firm surfaces (7).
- Add cross training including pool running, swimming, elliptical trainer, bike (7).
2. **Strength & conditioning:**
   - Slowly increase intensity over weeks, and gradually introduce jumps, etc (7).
   - Calf muscle training (11). Bone strength and muscular strength go hand-in-hand, so inclusion of general calf strengthening exercises would seem logical. Include eccentric calf exercises to build endurance and help prevent fatigue, (7) and exercises to focus on muscles controlling pronation (7). Increased strength and endurance of soleus is particularly recommended (3).
   - Strengthen the core and hip, and include proprioceptive & balance training – stance, wobble-board, etc (7).
3. **Running Technique.** As heel striking has been found to increase ground reaction force and anterior compartment pressures (5), change to a midfoot or forefoot running style may assist in reducing tibial stress. Professional assessment of running technique may be beneficial.
4. **Ice:** This has benefit for symptomatic relief (27).
5. **Shin splint strap / brace:** Straps were tested by one study, which found they had no benefit (2). However in severe cases, the short-term use of a leg brace (such as a high pneumatic splint) has been advocated (27). The evidence is anecdotal.
6. **Strapping tape:** the theory behind tape is that it unloads the sensitive tissue. Anecdotally there is some short-term benefit, but no research evidence.
7. **Deep tissue massage:** This is often recommended, but there is no evidence for its benefit (11,27). Assessment and treatment of focal areas of muscle tightness using massage or dry needling is a common clinical intervention. Massage may include digital ischaemic pressure (‘trigger point’), transverse friction, or sustained myofascial tension techniques (27).
8. **Vacuum cupping:** this has been advocated to treat muscle tension (27).
9. **Acupuncture:** This is often recommended but without evidence (11).
10. **Extra-corporeal Shock Wave Therapy:** moderate to good results were reported for this therapy in recalcitrant cases of MTSS (16). However no quality studies have been conducted to properly test this regime (32).
11. **Analgesia:** if required for symptomatic relief (27).
12. **Several other modalities** have been studied, including laser, therapeutic ultrasound, iontophoresis, phonophoresis, and pulsed electromagnetic field therapy, however no good evidence is available for the benefit of any of these therapies for the treatment of MTSS (32).

**Prevention:** Factors which may be effective in treatment and prevention of MTSS include:

1. **Shock absorbing / neoprene insoles:** (3,7,11,15,33). In an army study (33) less recruits developed MTSS when they wore shock absorbing insoles - 12.8%, compared to those who did not - 20.4%. There is no published data on the percentage of shock absorption recommended (3).
2. **Shoes:** Running shoes should be a good fit with a stable heel counter, & should be changed every 400 to 800 km, depending on such factors as body weight, training surface, and running style (3,7). In the sport of field hockey, where the incidence of MTSS is known to be high, elite
players run up to 8.5km per game (34,35). If they run similar distances in training, shoes should be replaced at least every season.

3. **Shoe orthoses:** After 3 months training in military recruits, 8% who had orthotics developed MTSS, compared to 38% who did not (28). However a systematic review in 2008 showed inconsistent results for the benefits of orthotics (3).

4. **Training factors:** Care should be taken with adjustments to training loads and methods (3,7). A common cause of MTSS is a sudden increase in intensity, or an increase in running on hills, hard or uneven surfaces (7). Cross training may be beneficial, including changing running surfaces. Field hockey players might benefit from conducting some of their fitness drills on natural grass. During heavy training, strength and conditioning staff should work with coaches to commit at least one day per week to a pool workout or some other cross training, to allow the bone remodelling response to keep pace with resorption (3).

5. **Preseason strengthening:** This should include graduated running programmes, eccentric and endurance calf exercises (3,7) and core strengthening where appropriate.

6. **Screening:** In asymptomatic first year military recruits, the presence of pain and/or swelling over the area typical for developing MTSS was found to be predictive of development of the condition within the subsequent 5 to 6 months. These tests may be useful screening tests, and could influence ongoing training loads and the use of other possible preventative strategies for at-risk individuals. Screening for other biomechanical or functional risk factors (described above) may also be beneficial in planning training loads and preventative exercises.

**Surgery:**
According to some authors, (4,25,27), in chronic resistant cases surgical release of the involved fascia, with or without compartment fascial release may have some benefit. A review of the available literature found a reported success rate varying between 29% and 86%. However most studies had poor methodology or follow-up (25). In a recent study of 43 patients undergoing a deep posterior compartment fasciotomy (to the ‘soleus bridge’), cauterisation of a 2cm area of periosteum, and scoring of the exposed bone, the authors reported 69% good or excellent results (25). However only 41% of patients returned to their previous level of sporting activity. There was also a high drop-out rate, with 41% of the original 78 subjects failing to return for follow-up. The reported complication rate was high, with 31 patients experiencing ongoing paraesthesia due to saphenous nerve injury (25).

**References**


