MEDIAL TIBIAL STRESS SYNDROME

SHIN SPLINTS: AETIOLOGY, TREATMENT AND PREVENTION

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ABSTRACT

One of the most common causes of exercise-induced leg pain is medial tibial stress syndrome. This review focuses on its aetiology, patient evaluation, treatment and prevention.

INTRODUCTION

Exercise-induced lower leg pain is a frequent complaint in athletes and medial tibial stress syndrome (MTSS) or shin splints is one of the most common of its causes. Incidences vary from 4 to 35% in different sports[1]. This condition is most frequently seen in runners and athletes involved in jumping, for example basketball players and rhythmic gymnasts.

In the past, many different authors used different names to describe MTSS: shin soreness, tibial stress syndrome, medial tibial syndrome, medial tibial stress syndrome and shin splints syndrome. Since MTSS has been the most commonly used term in the scientific literature during the last 10 years, it will also be used in this article.

AETIOLOGY

One of the most intriguing things about MTSS is its aetiology. Over the years many aetiological mechanisms have been proposed. Many doctors/physiotherapists know the subdivision postulated by Detmer in 1986. He stated that MTSS can be divided in three subtypes: a first subtype in which periostalgia is the cause of pain, a second in which stress reaction or a stress fracture of the tibial cortex is involved and a third in which elevated compartmental pressure is postulated to be the reason for the pain[2]. Currently, stress fractures and elevated compartmental pressures are considered separate entities from MTSS. However, even today the subdivision by Detmer is still used.

There have been many hypotheses on the aetiology of MTSS, but none have been confirmed. This presents a huge challenge for researchers on MTSS as it is difficult to target treatment if the aetiology is unclear. The two main hypotheses are described below and a possible explanation is provided for the development of pain due to each of these hypotheses.

> ***pain may even be provoked by activities of daily living***

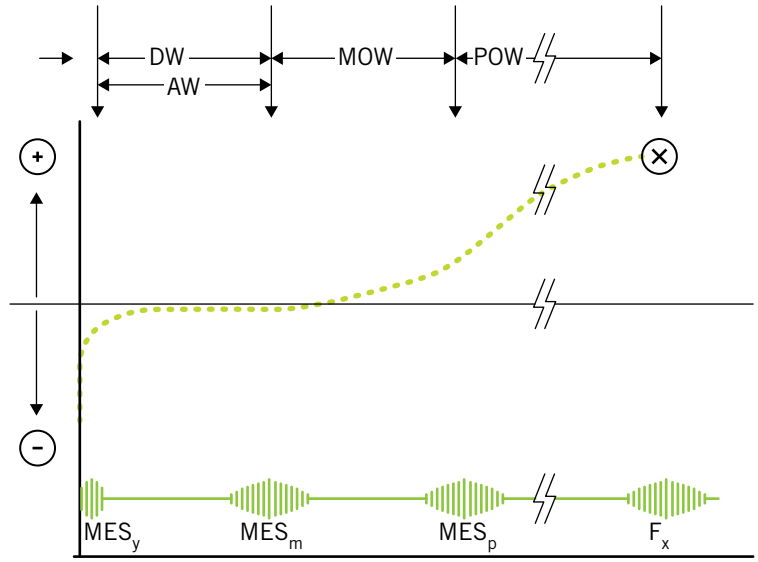

Figure 1: Remodelling graph of load-bearing bones adapted from Frost, 2004. DW=disuse window, AW=adapted window (as in normally adapted young adults), MOW=mild overload window, POW=pathologic overload window. MESy=disuse threshold, MESm=maintenance threshold, MESp=damage threshold, Fx stress fracture or MTSS (Reprint permission granted by Allen Press and Angle Orthod).

Hypothesis I: bone overload

With the bone overload hypothesis it is assumed that MTSS is caused by overload of the tibial cortex. Several studies have shown that MRI can depict bone marrow and periosteal oedema as a sign of bone remodelling. CT scan studies showed osteopenia on the posteromedial side of the tibia in symptomatic legs. Magnusson et al performed dual X-ray absorptiomerty (DEXA) scans in athletes, which showed decreased bone density in symptomatic legs compared to their controls[3].

In addition to the observations described above, it has been shown that even in asymptomatic legs of athletes, bone marrow oedema and periosteal oedema can be present. Bergman et al performed a study in which an MRI scan was performed in 21 asymptomatic runners who ran 50 to 70 miles each week[4]. The MRI scans showed findings in 43% (five unilateral findings and four bilateral findings). The findings varied from mild to moderate periosteal oedema to periosteal oedema and bone marrow oedema on T1 and T2 images. None of the runners developed complaints in the 48-month follow-up period. Moen et al and the study by Batt et al also showed bone marrow and periosteal oedema in asymptomatic legs of athletes with MTSS[5,6]. These MRI findings raise the question of whether bone marrow oedema represents normal remodelling of the bone after loading. It is possible that bone marrow oedema in athletes represents a normal sign of bone remodelling, which may become symptomatic once the loading exceeds a certain threshold.

Loading can affect remodelling of the bone in a variety of ways. Loading applied by muscle forces and axial loading transferred through the joint can deform the bone tissue and create strains which influence cellular processes in the cortex. This concept was placed in a theoretical framework by Frost who called it the 'Mechanostat Theory' (Figure 1)[7]. When the load is minimal and below a genetically derived threshold, the bone becomes weaker (disuse threshold: MESy) by resorbing trabecular or endocortical bone. When the load is higher than the disuse threshold and below a second threshold (maintenance threshold: MESm) the bone maintains its strength. When the load is between the maintenance threshold and a threshold above which cortical micro-damage occurs (damage threshold: MESp), the bone strengthens. Loads above this last threshold lead to microscopic fatigue damage (micro-damage), which accumulates when the loads and subsequent micro-damage are too great to recover from.

Bone pain mechanism

When the bone is overloaded and micro-damage accumulates, pain may occur. The pain mechanism of overloaded bone has only recently been established. Previously, the prevailing opinion was that bone pain was derived from the densely innervated periosteum. However, more recent studies showed that sensory nerve fibres also innervate the mineralised bone and bone marrow. Several studies suggested that these nerve fibres are stimulated by mechanical distortion. Other studies suggested that sensory nerve fibres could be activated by protons, which are released by osteoclasts when resorbing bone. That is because osteoclasts form a highly acidic compartment between themselves and mineralised bone. The acidic environment is sensed by afferent nerves that innervate the mineralised bone and induces a pain signal. Furthermore, microscopic cracks could also provide direct sensory input to intra-cortical nerves. When the crack disrupts the nerve, signalling is interrupted which can potentially lead to (referred) pain (personal communication:Weinans 2012[8]).

Hypothesis II: traction

The alternative theory on the aetiology of MTSS is that the syndrome is caused by traction. In the past, it was generally accepted that the posteriortibial muscles caused traction on the periosteum, leading to periostitis. However, significant evidence for periostitis was lacking, as shown in studies which performed histological research[1]. Furthermore, the location of the posteriortibial muscles (proximal and lateral) does not resemble the location of complaints in patients with MTSS[1].

> ***One of the most commonly described risk factors is pronation of the midfoot at rest and during exercise***

Two articles were published which provided some evidence for the possibility of the traction theory. Stickley et al showed, in an anatomical study with 16 cadavers, that the crural fascia was present along the entire posteromedial border of the tibia in all but three specimens[9]. They propose that traction on the crural fascia could produce complaints along the posteromedial tibial border. Fibres of the flexor digitorum, posterior tibial and soleus muscles were not found along the distal tibia. In 2006, an interesting study was published by Bouche and Johnson[10]. In this study three fresh frozen cadaver limb specimens were disarticulated at the knee and vertically aligned in a frame. The posterior tibial, flexor digitorumlongus and soleus muscles were connected to cables which could pull in an upward direction, simulating muscle pull. Four strain gauges were inserted in the crural fascia at its insertion on the posterior side of the tibia at 3, 6, 9 and 12 cm from the medial malleolus. Load was applied to the different muscles via the cables and strain was measured in the gauges. The result was that increased load to the muscles led to increased strain, recorded in the 3 and 6 cm strain gauges, in a linear manner. The data from the 9 and 12 cm strain gauges was variable and inconsistent[10]. Bouche and Johnson suggested that with dynamic loading, the crural fascia is under tension and may lead to complaints of MTSS through traction.

With the Bouche and Johnson study, the traction theory cannot be ruled out, but more comparable studies are needed to obtain firm evidence for the traction theory. Currently, the evidence is based on just three cadavers. Furthermore, it is not clarified how traction could lead to bone marrow oedema as seen on MRI, osteopenia as seen on CT scans and decreased bone density as seen with DEXA scans.

Pain mechanism

The mechanism of pain for the traction theory is not fully understood. Only one study investigated the crural fascia histologically. According to the Bouche and Johnson theory, Johnell et al found some evidence for inflammation of the crural fascia in the minority of patients but it remains unclear what exactly could cause pain symptoms[11]. They suggested that the aetiology of MTSS is the same as for plantar fasciitis. In this field, some more and larger histological studies have been performed. These studies showed that inflammatory cells are seldom found and that collagen and mucoid degeneration are more likely to lead to complaints. The crural fascia also consists of collagen fibres, which could theoretically become degenerative. If this is the case then free nerve endings at ligamentous attachment sites can be activated mechanically by strain or chemically. The transmission of electrical signals and neurotransmitter release could then cause pain. However, this hypothesis has not been investigated in patients with MTSS.

PATIENT EVALUATION

History

Most patients with MTSS present with exercise-induced lower leg pain. The pain is located along the posteromedial border of the tibia, usually in the middle or distal thirds. Initially symptoms are present on starting activity and subside with continued exercise. In more chronic cases, the pain continues to be present during activity, can persist after exercise and in severe cases may be provoked by activities of daily living.

Physical examination

No articles have been published on physical examination and MTSS. However, most authors agree that the diagnosis of MTSS is established by physical examination[12]. During physical examination pain is present on palpation of the posteromedial border of distal two-thirds of the tibia. Sometimes there is mild swelling of the tibia. Occasionally, resistance tests of the posterior tibial and soleus muscles are painful[6]. It can be difficult to differentiate between MTSS, tibial stress fracture and exertional compartment syndrome. Imaging may be helpful in such cases.

Imaging findings

Despite multiple imaging options, the diagnosis of MTSS is usually clinical. In the literature, radiographs, CT-scans, bone scans and MRI scans have all been used to aid in the diagnosisof MTSS. Radiographs performed in MTSS patients are almost always normal, so these are not useful to confirm MTSS. Two studies from the same study group investigated the role of CT-scans. These studies showed osteopenia in the tibial cortex in most of the patients. However, in 45% of the tibiae in asymptomatic runners, osteopenia was also found. This makes establishing the diagnosis using CT-scans at least difficult, if not impossible.

For bone scans and MRI scans a vast number of false positive and negative cases have been described. Bergman et al[2] showed that 43% of runners who ran between 64 to 112 km per week, showed findings on MRI

which can be compared to the findings in athletes with MTSS. On follow-up these runners did not develop leg complaints. In the study by Drubach et al, 100 young athletes referred for a bone scan because of low back complaints were analysed for asymptomatic bone scan abnormalities in the legs[13]. They found abnormalities in 34% of the legs. In 57% of athletes with longstanding MTSS complaints, no abnormalities were found on MRI scans[6]. It is therefore recommended to establish the diagnosis of MTSS clinically.

TREATMENT

Overall, the methodological quality of studies aimed at the treatment of MTSS is poor. Only a few randomised controlled trials (RCT) have been published. The following interventions were studied in the literature.

Iontophoresis, ice massage, phonophoresis and ultrasound vs control

Patients with MTSS were treated with these modalities in two separate RCTs. Iontophoresis and phonophoresis with corticosteroids and lidocaine were superior to doing nothing in terms of perceived pain. No difference in outcome was found between treatment with phonophoresis and iontophoresis[14,15].

Low-energy laser vs sham laser treatment

An RCT studied the effect of low-energy laser on pain and return to activity. No difference was found between the treatment group and the control group, which received sham laser treatment[16].

Graded running, stretching/strengthening and sport compression stockings

A Dutch RCT studied graded running, stretching/strengthening exercises and sports compression stockings in (mainly) young athletes. No differences were found in days to complete a running programme[17].

Lower leg brace vs no leg brace

Two RCTs were conducted which studied the effect of a lower leg brace on complaints of MTSS. No differences were found between the groups in terms of pain or ability to return to activity[18,19].

Shockwave vs other modalities

Two studies performed a controlled trial (not randomised) on the effect of shockwave (radial and focused) on complaints of MTSS. One study compared shockwave with home exercises[20], while the other study compared shockwave combined with a graded running programme and a running programme only[21]. In both studies, the group in which shockwave treatment was involved, the outcomes for pain and function were better.

Foot orthoses

Two retrospective studies investigated whether foot orthoses had an effect on pain caused by MTSS[22,23]. Both studies reported that the majority of patients noticed a reduction of pain in daily life and during sports.

Of the interventions above, only iontophoresis, phonophoresis, foot orthoses and shockwave therapy showed an effect on complaints of MTSS. It is therefore advised to incorporate (any of) these interventions while treating a patient with MTSS. One has to bear in mind that the quality of the studies that showed the treatment effect is poor.

PREVENTION

Several preventive strategies for MTSS have been studied in the past decades. Of these, level I evidence exists only for a shock absorbing insole[1]. Shock absorption may lead to less strain in the tibia while the leg is axially loaded. Less strain and bending of the tibia result in less microdamage of the cortex[4]. No further preventive measures have been proven to be beneficial[1].

Future attempts to prevent MTSS could be aimed at risk factors known to be associated with MTSS, as described by Professor van Mechelen and the Aspetar Illness and Injury Prevention Programme (ASPREV). One of the most commonly described risk factors is pronation of the midfoot at rest and during exercise. No studies have investigated whether an anti-pronation insole could prevent the development of MTSS. Other risk factors which are described in the literature are more difficult to focus on such as female gender, decreased internal range of motion of the hip joint and increased plantarflexion of the ankle[1].

It could be useful to measure bone mineral density (BMD) in athletes in an effort to prevent MTSS. If the BMD is reduced, the training load could be adapted. Usually, BMD is measured using DEXA scans. This

method is not practical for athletes, since measurements have to be performed in the hospital. Qualitative ultrasound has been used to measure BMD in athletes. Although this is a more practical method of measuring BMD and may be promising, more studies are needed to recommend the technique in practice yet.

CONCLUSION

Medial tibial stress syndrome remains an elusive syndrome affecting many athletes. It is possible to establish the diagnosis clinically and the standard use of imaging is not advised. A few treatment options exist such as iontophoresis, phonophoresis, foot orthoses and shockwave, which can aid the injured athlete. However, there is only level 3 or 4 evidence for these interventions. As long as treatment of MTSS remains difficult, maximal efforts should be put into prevention methods, such as maximising shock absorption.

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