



Update article

Stress fractures: definition, diagnosis and treatment[☆]



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ABSTRACT

Stress fractures were first described in Prussian soldiers by Breithaupt in 1855. They occur as the result of repeatedly making the same movement in a specific region, which can lead to fatigue and imbalance between osteoblast and osteoclast activity, thus favoring bone breakage. In addition, when a particular region of the body is used in the wrong way, a stress fracture can occur even without the occurrence of an excessive number of functional cycles. The objective of this study was to review the most relevant literature of recent years in order to add key information regarding this pathological condition, as an updating article on this topic.

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Fraturas por estresse: definição, diagnóstico e tratamento

RESUMO

A fratura por estresse foi descrita inicialmente em soldados prussianos por Breithaupt em 1855 e ocorre como o resultado de um número repetitivo de movimentos em determinada região que pode levar a fadiga e desbalanço da atuação dos osteoblastos e osteoclastos e favorecer a ruptura óssea. Além disso, quando usamos uma determinada região do corpo de maneira errônea, a fratura por estresse pode ocorrer mesmo sem que ocorra um número excessivo de ciclos funcionais. O objetivo deste estudo é revisar a literatura mais relevante dos últimos anos para agregar as principais informações a respeito dessa patologia em um artigo de atualização do tema.

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Introduction

Stress fractures were first described in Prussian soldiers by Breithaupt in 1855.¹⁻³ They were named “march fractures” and their characteristics were confirmed 40 years later with the advent of radiography.^{1,2} In 1958, Devas made the first report on stress fractures in athletes.¹⁻³

This injury occurs as a result of high numbers of occurrences of cyclical overloading of intensity lower than the maximum bone strength, on non-pathological bone tissue.⁴⁻⁶

These fractures may be the final stage of fatigue or insufficiency of the bone affected.⁶ Fatigue fractures occur after formation and accumulation of microfractures in normal bone trabeculae. On the other hand, fractures resulting from bone insufficiency occur in bone that is mechanically compromised and generally presents low bone mineral density.⁶ In both situations, imbalance between the bone that is formed and remodeled and the bone that it reabsorbed will result in discontinuity of the bone at the site affected.^{7,8} The aim here was to present an updating article on this topic and condense the main information obtained through the most important studies published over the last few years.

Epidemiology

Population

Runners, soldiers and dancers are the main victims of stress fractures.^{6,9,10}

Anatomical region

All the bones of the human body are subject to fracturing caused by stress. This stress is closely related to the daily practice that athletes undertake. The predominance of stress fractures in the lower limbs, over fractures in the upper limbs, reflects the cyclical overloading that is typically exerted on bones that bear the body weight, in comparison with bones that do not have this function.³ Stress fractures are mostly commonly diagnosed in the tibia, followed by the metatarsals (especially the second and third metatarsals) and by the fibula.^{3,11} Stress fractures in the axial skeleton are infrequent and are mainly located in the ribs, pars interarticularis, lumbar vertebrae and pelvis.¹¹⁻¹³

Types of sport

Runners present greatest incidence of stress fractures in long bones such as the tibia, femur and fibula, and also present fractures in the bones of the feet and sacrum.^{11,12} Types of sport in which the upper limbs are used, such as Olympic gymnastics,¹⁴ tennis, baseball and basketball may result in fractures due to stress. The bone most affected is the ulna, especially in its proximal portion, while the distal extremity of the humerus is also affected.^{6,11,13} Stress fractures occur mainly in the ribs in golfers and rowers^{11,13} Jumpers, bowlers and dancers present greatest risk of injury to the lumbar spine and pelvis.¹¹

Sex

Among athletes, the difference in the incidence of stress fractures between men and women is minimal. It is believed that the intensity and type of controlled training for each athlete and the physical preparation that already exists diminish the impact of the training program.^{9,15} In the military population, the incidence of stress fractures among females is greater than among men.^{16,17}

Physiopathology

Six to eight weeks after a sudden and non-gradual increase in the intensity of an athlete's or new patient's physical activity, this cyclical and repetitive physiological overloading may lead to the appearance of microfractures and may not allow the bone tissue to have sufficient time to undergo remodeling and adapt to the new condition, and thus to repair the microlesion.^{4-6,10,18,19} The load applied is considered to be insufficient to cause an acute fracture, but the combination of overloading, repetitive movements and inadequate recovery time make this a chronic injury.¹¹ Elastic deformation occurs initially, and this progresses to plastic deformity until it finally results in microfracturing. If this is not treated, it will evolve to complete fracturing of the bone affected.¹⁰ The bone repair process in stress fractures differs from the process in cases of common acute fractures and only takes place through bone remodeling, i.e. reabsorption of the injured cells and replacement with new bone tissue take place.¹⁹

Markey also proposed that the muscle mass acts toward dispersing and sharing impact loads on the bone tissue.²⁰ Therefore, when fatigue, weakness or muscle unpreparedness occur, this protective action is lost and the risk of bone tissue lesions increases.^{16,20}

Risk factors

The factors associated with increased risk of development of stress fractures can be divided into extrinsic and intrinsic factors. This makes stress fractures multifactorial and difficult to control.^{8,9,20-23} Extrinsic factors relate to sports movements, nutritional habits, equipment used and the type of ground.^{8,9,14,20-23}

Abrupt increases in the intensity and volume of training are often enough for lesions to develop.^{6,9-11} Equipment such as footwear that has low impact absorption, is worn out (more than six months of use) or is a bad fit for the athlete's foot may cause injuries.^{8,23} The quality of the training track may also be a risk factor, when it is uneven, irregular or very rigid.^{17,24} Lastly, if the athlete's fitness level is insufficient for the sports movement or functional technique, this may lead to injury, sometimes without the number of repetitions having been very high.^{8,25}

The intrinsic factors relate to possible anatomical variations, muscle conditions, hormonal states, gender, ethnicity or age.^{8,9,20-22}

Many studies have mistakenly considered that only female gender is a risk factor for stress factors to appear.^{11,16,26} In

reality, hormonal, nutritional, biomechanical and anatomical alterations are the true factors that favor appearance of stress fractures in women.^{11,24}

Age also cannot be considered to be a risk factor in isolation for stress fractures.^{11,23,27} Studies conducted in the United States have attempted to evaluate the incidence of these injuries among white and black athletes, without observing any significant differences.^{11,13} In a military population, the incidence among whites was twice as high as among blacks, without any difference between the sexes. This was attributed to bone density and its biomechanics.²⁴

There is an inverse relationship between bone mineral density and the risk of stress fractures.^{8,10,28} Inadequate nutritional intake may alter bone metabolism and predispose toward appearance of stress fractures.^{8,10,29}

Low levels of physical and muscle conditioning are also an important risk factor for the genesis of this problem.^{6,8,10,30,31} Furthermore, rigid pes cavus, discrepancy of the lower limbs, short tibia, genu valgum, increased Q angle, body mass index lower than 21 kg/m² and short stature should also be taken into consideration in analyzing the risk factors for stress fractures.^{6,8,9,21,32}

Some studies have also suggested that stiffness of the feet, alterations to the plantar arch and limitations of dorsiflexion due to shortening of the sural triceps may be risk factors.^{8,10,33} Runners whose hindfoot presents eversion, particularly with excessive pronation, and athletes with a pronounced high arch have a risk of developing stress fractures that is up to 40% higher.^{10,21,33,34} Moreover, hyperpronation of the forefoot predisposes toward increased risk of stress fractures in the fibula.³⁵ Stress fractures in the second metatarsal have been correlated with presence of Morton's neuroma, hypermobility of the first metatarsal and a relative increase in the length of the second metatarsal.^{20,33} Although use of orthoses and footwear that is more appropriate theoretically decreases the incidence of stress fractures, the number of studies in the literature remains insufficient to sustain this theory.^{10,34}

Other authors have also considered that the following are risk factors: smoking, physical activity of frequency less than three times a week and consumption of more than 10 doses of alcoholic drink per week.⁶

Physical examination

Physical examination of stress fractures is very sensitive but unspecific.^{20,36} Patients present increased sensitivity, pain and edema at the lesion location after an abrupt and/or repetitive increase in physical activity with insufficient rest intervals for physiological tissue recovery.⁶ Initially, the pain is reduced and alleviated through rest and this allows unimpaired physical activity. However, if the aggressive movement is maintained, the injury will progress, thus resulting in increased pain and limitation of practicing this movement.^{9,20} Information regarding any previous fractures, weight, height, body mass index and its changes over the last 12 months, menstrual and puberty history and nutritional evaluations is important for identifying possible intrinsic risk factors for injury during physical examinations.¹⁰

Clinical tests such as use of therapeutic ultrasound and tuning forks are also useful in diagnostic investigations on stress fractures.³ When used directly on the site of the suspected lesion, they may trigger or worsen the pain because of the great local osteoclastic reabsorption, which results in injury to the periosteum.^{3,37} In addition, the skipping rope test (hop test) can be used: this consists of asking the patient to hop on the spot while putting weight on the limb that is under investigation. The test is positive when it triggers strong or incapacitating pain in the region injured.^{6,38}

Some laboratory tests may be useful in investigating stress fractures: serum levels of calcium, phosphorus, creatinine and 25(OH)D3. Nutritional markers should be requested in the presence of clinical conditions of weight loss and anorexia. Hormonal levels (FSH and estradiol) should be investigated when there is a history of dysmenorrhea.¹⁰

Imaging examinations

Imaging examinations are fundamental for diagnosing, prognosing and following up stress fractures.⁶

Simple radiography (X-ray) is the initial imaging examination because of its ease of access and low cost.^{4,13,36,38-42} However, it has a high false-negative rate, given that the alterations caused by stress fractures only appear on such examinations at a late stage (two to four weeks after the start of the pain), which may delay the diagnosis.^{6,14,18,43} Initially, a subtle weak radiolucent area can be observed directly on the bone tissue affected and/or sclerosis, periosteal thickening, cortical changes comprising diminished cortical bone density (gray cortex) and/or appearance of a delicate fracture line. Finally, an attempt by the organism to form a bone callus is observed, with endosteal thickening and sclerosis, which are the commonest findings.^{6,10,14,38,44} The sign known as the dreaded black line occurs in the anterior cortical bone of the tibia and suggests the presence of a fracture with a poor prognosis and a high probability of evolution to a complete fracture because of its location in a region of bone tension and poor vascularization.⁴⁴

Computed tomography (CT) is used mainly when there is a contraindication against using magnetic resonance imaging.⁴³⁻⁴⁶ Chronic and quiescent lesions may be more evident in this examination than on magnetic resonance imaging or bone scintigraphy because they present low bone turnover.⁴⁶ Single photon emission CT (SPECT) has been particularly more useful in investigating stress fractures involving the dorsal spine, and specifically in pars interarticularis (spondylolysis).^{6,45,46}

Nuclear medicine using triple-phase scintigraphy (technetium-99m) presents significant sensitivity (74-100%) to bone remodeling and shows imaging alterations three to five days after the start of symptoms.^{3,6,41,42,47} The radiopharmaceutical becomes concentrated in the regions affected and detects areas of bone remodeling, microfractures of the trabecular bone, periosteal reaction and formation of bone callus.⁴⁶

Magnetic resonance imaging (MRI) is the most sensitive and specific imaging examination for diagnosing stress

Table 1 – Classification of Arendt and Griffiths.

Stages/grades of Arendt and Griffiths for bone injuries due to stress, based on magnetic resonance imaging (MRI) findings		
Grade of injury due to stress	MRI findings	Duration of resting period needed for cure (weeks)
1	STIR-positive	3
2	STIR and T2-weighted positive images	3-6
3	T1 and T2-positive without definition of cortical rupture	12-16
4	T1 and T2-positive with definition of cortical rupture and visible fracture line	16

fractures. It is recommended by the American College of Radiology as the preferred examination in the absence of radiographic alterations.⁶ The abnormalities caused by the fracture can be identified one to two days after the start of the symptoms,^{6,10,12,38,41,43,46,48} with early detection of edema in the bone tissue and adjacent areas.^{10,41,46} This examination makes it possible to differentiate medullary damage from cortical, endosteal and periosteal damage allows gradation of the lesions regarding their severity and prognosis.⁶ Intramedullary endosteal edema is one of the first signs of bone remodeling and may continue to be present for up to six months after the fracture has been diagnosed and treated, while the cortical maturation and remodeling take place.^{16,48} Medullary edema or signs of bone stress may also be present in asymptomatic physically active patients, without any correlation with increased incidence of stress fractures, especially in the tibia in marathon runners.⁴⁶ The fracture line is less commonly visible.¹⁰ It presents sensitivity slightly greater than or equal to that of scintigraphy, but it is considered to be a more specific examination.^{6,38,41}

Classification

Fractures can and should be classified so that the prognosis and treatment can be measured and thus give rise to a better result for the patient.

Arendt and Griffiths apud Royer et al.¹¹ used imaging parameters obtained through MRI to divide stress fractures into four stages. The aim of this classification is to define the length of resting time that is needed for a return to sport, according to the patient's current stage. These stages can also be used for reevaluation during follow-up of the lesion.⁷ Lesions treated at stage 1 require an average of 3.3 weeks of resting, while those at stage 4 require 14.3 weeks⁷ (Table 1).

Table 2 – Classification of low-risk stress fractures.

Low-risk stress fractures	
Upper limbs	Clavicle, scapula, humerus, olecranon, ulna, radius, scaphoid, metacarpals
Lower limbs	Femoral diaphysis, tibial diaphysis, fibula, calcaneus, metatarsal diaphyses
Thorax	Ribs
Dorsal spine	Pars interarticularis, sacrum
Pelvis	Ischiopubic rami

Table 3 – Classification of high-risk stress fractures.

High-risk stress fractures
Femoral neck (superolateral)
Anterior cortical bone of the tibia
Medial malleolus
Navicular bone
Base of the second metatarsal
Talus
Patella
Sesamoids (hallux)
Fifth metatarsal

Stress fractures can also be classified as high and low-risk fractures. The bone location, the prognosis for consolidation and traits ascertained through imaging examinations are some of the characteristics that define whether there is higher risk that a stress fracture might not evolve satisfactorily during the treatment^{6,11} (Tables 2 and 3).

Fredericson proposed a stress fracture classification through using the alterations seen on MRI. The progressive stages of lesion severity are assessed according to periosteal involvement, followed by medullary involvement and going as far as the point at which the cortical bone also becomes compromised^{10,41} (Table 4).

Treatment

In order to adequately treat stress fractures, it is essential to identify risk factors that lead to disease. Treatments for stress fractures are based on prevention of new episodes and on recovery of the injured area.^{6,10,20}

Table 4 – Fredericson classification.

MRI findings according to Fredericson	
Lesion stage	
0	Normal
1	Periosteal edema
2	Periosteal and medullary edema on T2-weighted images
3	Periosteal and medullary edema on T1 and T2-weighted images
4	Periosteal and medullary edema with visible fracture line

Prevention of new episodes is achieved through modifying activities, correcting sports movements, changing sports equipment, changing training locations that might be favoring bone overloading, changing nutritional habits, recognizing hormonal, anatomical and muscle strength alterations and recognizing low cardiomuscular conditioning.²⁰ The ideal type of footwear for each type of sports practice is the external factor that has been studied most with regard to the genesis of stress fractures.²⁰ Some studies have shown that there is lower incidence of injuries when running on asphalt is replaced by running on softer surfaces, such as athletics tracks. Nonetheless, other authors have reported in their studies that there was no relationship between these factors.²⁰ Voloshin⁴⁹ believed that there was interference between the different shock-absorbing surfaces: the stress on the bone tissue is not due solely to the reaction forces from the ground. The combined forces generated by muscle action through the athlete's movement and his adaptation to the training surface may also be considered to be risk factors for a given type of injury.^{20,49}

The treatments for these injuries comprise diminution of the overloading on the site affected, medication for pain control and physiotherapeutic rehabilitation.^{6,10,20}

Analgesics are used for pain relief.⁶ Anti-inflammatory drugs, if used, should be prescribed cautiously and only for short periods. Studies on animals have demonstrated that there may be negative interference in the bone healing process.⁶ However, reviews of the literature conducted more recently have reported that there is no conclusive evidence regarding this negative action.⁵⁰⁻⁵²

The time taken for fracture consolidation is generally between four and 12 weeks when the fractures are low-risk.⁶ For the metatarsals, a time of three to six weeks is expected, while for the posteromedial region of the tibial diaphysis, the femur and the pelvis, six to 12 weeks is expected.^{10,11} The patient should be reexamined every two to three weeks, to monitor the changes to the symptoms and pain during resting and rehabilitation periods.^{6,53-56} IN order to maintain flexibility, strength and cardiovascular physical conditioning during the resting period, the patient needs to be engaged in a physiotherapy program^{6,53,54} and a controlled exercise program.⁵⁷

Immobilization is only rarely used for treating stress fractures because of its deleterious effects on muscles, tendons, ligaments and joints.⁵ However, there are some specific types of fracture for which immobilization is fundamental for obtaining appropriate conditions for a cure: this is the case for the navicular bone, sesamoids, patella and posteromedial region of the tibia.⁵

High-risk fractures commonly evolve to non-consolidation of the bone and surgical intervention by an orthopedist becomes necessary.⁶ Stress fractures of the lateral cortical bone (due to tension) at the femoral neck is associated with catastrophic results, such as complete displacement of the femoral head and osteonecrosis, when this is not treated surgically.^{20,53} Fractures of the anterior cortical bone of the middle third of the tibial diaphysis are another type that, if not treated surgically, mostly presents an extremely poor prognosis.²⁰ Fractures of the base of the fifth metatarsal and of the navicular bone can also be cited as types that commonly

require surgical intervention in order to achieve a satisfactory result from their treatment.²⁰

New types of therapy

Some new types of therapy for stress fractures are being studied with the aim of achieving faster consolidation and an earlier return to physical activities. These can be divided into biological and physical methods.⁵⁰

Oxygen supplementation therapy (hyperbaric oxygen therapy)

In vitro studies have demonstrated that administration of 100% oxygen is capable of stimulating osteoblasts and consequently bone formation.⁵⁰ However, there is still no consensus in the literature regarding its benefits for treating stress fractures.^{50,54}

Bisphosphonates

Bisphosphonates suppress bone reabsorption and inactivate osteoclasts through their bonding with calcium phosphate crystals.^{20,50} Their high cost and various side effects may be the deciding factor with regard to choosing and attempting to use this therapeutic method.^{50,55} There is not yet any scientific basis for their prophylactic use.^{50,56}

Growth factors and growth factor-rich preparations

Growth factors are applied directly to diseased tissues with the aim of accelerating and promoting their repair. The preliminary results from muscles, tendons and ligaments have been encouraging.^{50,57} There are only a few studies on treating stress fractures. Some of them have reported that when these factors are used during surgical treatment of high-risk fractures, they may accelerate and improve the recovery.⁵⁰

Bone morphogenic proteins

Bone morphogenic proteins contain bioactive factors that are responsible for inducing bone matrix activity with an osteoinductive function.⁵⁰ Their primary activity is in relation to differentiation of mesenchymal cells into bone and cartilage tissue-forming cells, through direct and osteochondral ossification. They have an important function in repairing bone lesions. Studies on animals have demonstrated acceleration of the injury cure process in cases of traumatic fractures, but little can be concluded regarding their use in stress fractures.⁵⁰

Recombinant parathyroid hormone

Parathormone acts toward regulating serum calcium levels through gastrointestinal absorption, calcium and phosphorus reabsorption in the kidney, and calcium release from the skeletal tissue.⁵⁰ Although this initially promotes stimulation of osteoclasts through regular administration, when it

is done intermittently in a controlled manner, it gives rise to anabolic stimulation of osteoblasts and results in formation of bone with increased strength and density, followed by remodeling.⁵⁰ Studies have demonstrated that this hormone stimulates bone repair through both endochondral and membranous mechanisms.⁵⁰

Low-intensity pulsatile ultrasonography

High-frequency sound waves that are above the audible limit of human beings interact with bone tissue and the adjacent soft tissues and generate microstress and tension that are capable of stimulating consolidation.^{6,13,50} However, their exact mechanism of action remains unknown.¹⁹ Some studies have demonstrated its efficacy in treating stress fractures.^{50,58} Other studies have completely supported its use for treating these fractures.⁶

Application of magnetic fields

Magnetic fields can be applied by means of a direct current at the focus of the fracture through surgical placement of electrodes, use of an electrical capacitance field device or use of electromagnetic field pulses.⁵⁰ There is still no concrete evidence regarding its use in stress fractures.^{20,50}

Criteria for return to sport

The time taken from diagnosis to cure and until the return to sport depends on multiple factors such as the injury site, sports activity, severity of the injury and possibility of correcting risk factors that are intrinsic to the patient.²⁰ Low-risk stress fractures and non-surgical treatment usually make it possible for the patient to return to his activities four to 17 weeks after the injury.⁶

The criteria that can be used for allowing an athlete to return to his practice may include: total absence of pain at the site affected, especially during sports movements; absence of symptoms during pain provocation tests at the injury site; absence of abnormalities in imaging examinations; and, above all, comprehension by the patient, trainers and technical team of the sport regarding the risk factors and conditions that led to the injury, so that corrections can be made so as to prevent recurrence and reappearance of injuries.¹⁰

The gradual definitive return to sports activity should be started after the patient has been free from pain for 10–14 days, with 10% increases in training intensity per week.^{20,50} Formation of a bone callus and obliteration of the fracture line on simple radiographs and, especially, on computed tomography scans are the factors that determine whether the cure process for the stress fracture has been adequate.⁵⁰

Prevention

Although several methods for preventing stress fractures have been proposed, only some of them present proven validity that can justify their recommendations.⁶ The possible risk factors that contribute to appearance of these fractures need to

be carefully studied, modified and followed up.^{6,10} Constant control and modification of physical activity, with adequate recovery time, are extremely important.^{6,10} It is considered that daily intake of 2000 mg of calcium and 800 IU of vitamin D may be protection factors.^{6,9} The kinematics and biomechanical factors predisposing toward such fractures need to be monitored and corrected, through correct understanding of the sports movements, equipment, orthoses, training surface and all the other factors that may be involved in sports practice.^{6,10,50} Some studies have investigated prophylactic use of bisphosphonates for preventing stress fractures, but there is still no evidence regarding its benefits in prevention of this type of injury.⁶

Complications

The main complications occur in cases of high-risk stress fractures. Inappropriate management may cause progression of the fracture to a complete and displaced fracture line and thus give rise to delayed consolidation, avascular necrosis and pseudarthrosis.^{6,20} Furthermore, bisphosphonates used in treating stress fractures may weaken some bone regions when used over the long term and may predispose toward appearance of fractures due to insufficiency and a potential teratogenic effect among pregnant patients.⁶

Conflicts of interest

The authors declare no conflicts of interest.

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