International Journal of Health Science

IMAGING ASSESSMENT OF STRESS FRACTURES (FATIGUE/FAILURE)

Marina Lucena Aguiar Roriz

Resident of Radiology – São Carlos Imagem / ISCEP, Fortaleza, Ceará, Brazil.

Lorena Chérida Alves Vidal

Radiologists – São Carlos Imagem / ISCEP, Fortaleza, Ceará, Brazil.

Germana Bastos Pontes

Radiologists – São Carlos Imagem / ISCEP, Fortaleza, Ceará, Brazil.

Manuel Joaquim Diógenes Teixeira

Orthopedist and Traumatologist – Hospital Geral de Fortaleza (HGF), Fortaleza, Ceará, Brazil.

Gisele Façanha Diógenes Teixeira

Resident of Pediatric Orthopedics – Hospital Pequeno Príncipe, Curitiba, Paraná, Brazil.

Cláudio Régis Sampaio Silveira

Radiologists – São Carlos Imagem / ISCEP, Fortaleza, Ceará, Brazil.



All content in this magazine is licensed under a Creative Commons Attribution License. Attribution-Non-Commercial-Non-Derivatives 4.0 International (CC BY-NC-ND 4.0).

Summary: Stress fractures are increasingly common in our country. History taking and physical examination are essential tools for diagnosis. However, imaging methods very important in confirming these injuries. Although plain radiography is the initial choice, magnetic resonance imaging (MRI) is the modality with the greatest sensitivity and specificity for the early detection of this type of fracture. The present study aims to review the literature, identifying the most consistent imaging findings and offering a practical approach to the diagnosis of stress fractures through a systematic assessment.

Keywords: Stress Fracture; Fatigue Fracture; Insufficiency Fracture; Bone Stress Reaction; Pathological fracture.

INTRODUCTION

Stress fractures had their first description in the literature in 1855, when Breithraupt, a military doctor, reported overload injuries in the fifth metatarsal bone of Prussian conscript soldiers. About 40 years later, with the invention of radiography, the characteristics of the so-called "gait fractures" were confirmed through imaging examination. However, only in 1958, Devas made the first report of stress fractures in athletes.

Stress injuries occur as a result of a high number of cyclical and repetitive overloads on the bone structure, with forces lower than the load sufficient to fracture the bone in a single acute situation, but with the power to shake the bone microstructure.⁴ In this way, they differ from other fractures because they do not result from acute traumatic events.⁵

There are two categories of stress fractures: fatigue fracture, which results from repetitive abnormal stress applied to a bone with normal elastic strength; and insufficiency fracture, which occurs when there is normal stress on a bone with altered elastic resistance.⁶

Fatigue fractures predominantly affect

active and healthy young people who increase the frequency, duration or intensity of physical exercise.⁷ On the other hand, insufficiency fractures prevail in elderly people with comorbidities, such as osteoporosis, which weaken the bone and predispose to this type of injury even in the face of a habitual routine activity.

For the diagnosis of stress injuries, a compatible clinical history in addition to imaging exams are essential to enable faster treatment initiation, thus improving the prognosis of these fractures.

PATHOPHYSIOLOGY

Stress consists of the force applied to a certain body segment resulting from muscular tension or weight support, being necessary, to a certain extent, to maintain the normal development of bone tissue.⁸

Bone is a dynamic structure that is in constant metabolism and that, in physiological situations, suffers the action of the Law of bone remodeling proposed by Wolff, in which intermittent loads related to daily life activities stimulate changes in bone architecture, in order to to adapt it to the new mechanical environment.8

]When physiological bone remodeling suffers an imbalance between osteoclastic and osteoblastic activity, the cycle of adaptations in response to overloads applied to the bone is compromised, which can culminate in Stress Fracture, which initially occurs through elastic (reversible) deformation. followed by a plastic deformity (irreversible) that then progresses to the appearance of microscopic lines of bone discontinuity (microfractures). If the inciting activity is not interrupted, it may lead to a complete fracture of the affected bone.⁹

Stress fractures usually occur between 6 and 8 weeks after the beginning or increase in the habitual load imposed on the bone structure, the increasing number of repetition cycles

of overload, associated with an inadequate recovery time or even a decrease in surface area over which the force is applied.^{4,10,11,12}

In cortical bone, the periosteal and endosteal reaction, sometimes seen on radiographs, represents the production of new bone at sites of stress as an attempt to reinforce the cortex temporarily weakened by microfractures; while in cancellous or medullary bone, stress culminates in microlesions of bone trabeculae and, as a repair mechanism, there is the formation of microcalluses and trabecular thickening, which are responsible for bone sclerosis.8

Therefore, stress fractures are consequences of the sum of recurrent loads and occur when the rate of accumulated microdamage exceeds the bone's ability to regenerate through the normal process of bone remodeling, while the repair process of these injuries occurs through the reabsorption of damaged cells and replacement with new bone tissue.^{7,13}

EPIDEMIOLOGY

In general, stress fractures represent 1 to 20% of all injuries in sports medicine, and the most frequently affected population is military personnel, runners and dancers. 9,14

Involvement of the lower limbs predominates over the upper limbs due to the overload exerted by supporting body weight on those bones, with the most affected being the tibia, femur and fibula, in addition to the 2nd and 3rd metatarsals, notably in runners. ^{15,16,17} In relation to the upper limbs, the proximal portion of the ulna is the most affected area, followed by the distal end of the humerus.11

While the occurrence of fatigue fractures is uncommon in the axial skeleton, insufficiency fractures commonly affect this site, with the spine being the most affected, resulting in loss of height of the vertebral bodies, instability and kyphotic deformity.¹⁸ The upper extremities

generally are not affected by insufficiency fractures.¹⁹

In non-athlete individuals, it is known that poor physical and muscular conditioning are triggering factors for stress injuries, while there is not a direct relation to age, gender and ethnicity. 11,20,21,22

RISK FACTORS

Some risk factors are associated with an increased chance of developing a stress fracture, for example to sport activities, specially running, level of nutrition, hormonal profile and biomechanics. ²³

Women may have a higher incidence of stress fractures than men. The so-called "female athlete triad" represents a risk factor for females and is characterized by the presence of eating disorders (anorexia), menstrual irregularity or amenorrhea and osteopenia - presumably due to the absence of the protective effect of estrogen against bone loss. Changes in bone tissue related to the combination of hormonal and nutritional disorders contribute to the development of stress fractures in this group. 9, 24,25,26,27

Regarding biomechanical and anatomical aspects, some of the risk factors are: discrepancy in length between the lower limbs, decreased width of the tibia, excessive genu valgus, rigid cavus foot and exaggerated pronation of the feet.^{28,29} The presence of bone deformity, such as lateral curvature of the femur, especially in the elderly, can also be considered a risk factor.^{30,31}

In regard to insufficiency fractures, several conditions reduce the elastic strength of the bone and predispose to this type of injury. The most common cause is osteoporosis, but rheumatoid arthritis, Paget's disease, osteomalacia, hyperparathyroidism, osteogenesis imperfecta, previous irradiation and prolonged therapy with bisphosphonates are some other factors.³²

CLINICAL EVALUATION

The diagnosis of stress fractures is made based on anamnesis, physical examination and complementary imaging methods.³³

Clinical history is characterized by pain related to physical exercise or habitual walking, edema and limitations in range of motion.³⁴ The pain is usually insidious and, a few weeks after the onset of the injury, it can progress and become more intense., causing functional incapacity.^{35,36} initially, it usually occurs at the end of the activity, improves after the guarantee and allows the return to its performance without prejudice. However, as the overload continues, the severity of the symptoms tends to worsen, causing persistent pain even after the stress has stopped, ultimately leading to the suspension of physical activity.^{14,37}

On physical examination, mild soft tissue edema can be observed, although infrequent. Bone palpation is commonly painful, especially in the most superficial regions.² The physical examination is quite sensitive, but somewhat specific.^{37,38} Therefore, imaging studies must be obtained routinely, being fundamental for diagnosis and adequate treatment.¹¹

RISK CLASSIFICATION

Stress fractures can be classified into low and high risk according to their natural history, bone location and the appearance of complications (recurrence, delayed union and progression to complete fracture). These conditionss define the chance of a non satisfatory evolution of the injury during treatment.^{11,16}

Low-risk fractures are considered to be those that have a favorable evolution. They are located in areas of bone compression, and have low rates of complications.³⁹ These preferentially affect: clavicle, scapula, ribs, humerus, radius, diaphysis of the ulna, inferior and medial cortex of the femoral neck (Figure 1 a, b, c), femoral shaft, fibula, tibial shaft and

first to fourth metatarsals.²

High-risk stress fractures have unfavorable natural history, affect locations where traction forces prevail (which act to separate the edges of the fracture and predispose them to displacement) or in areas of poor vascularization, have high rates of complications and can require the need for surgical treatment with internal fixation.³⁹ Those that are serious occur, mainly, in the cortex of the proximal diaphysis of the femur known as "atypical femoral fractures" (Figure 1 d, e, f), in the anterior cortex of the tibia, in the patella, in the medial malleolus, in the navicular bone, in the hallux sesamoids and in the fifth metatarsal.2

IMAGING ASPECTS

CONVENTIONAL RADIOGRAPHY

Plain radiography is still the most routinely used imaging method due to its easy access and low cost and is the initial examination of choice when stress fractures are suspected.⁴⁰

Radiographic sensitivity in the early stages of lesions is low, estimated at around 15 to 35%, as radiological changes become visible two to four weeks after the onset of symptoms, which can delay diagnosis. ¹¹Follow-up images evolutionary are positive in only 50% of cases. ^{41,42}

Insufficiency fractures can be difficult to identify on radiographic studies, due to the frequently associated osteopenia.²²

The initial radiological change of stress fracture includes the "gray cortical" sign consisting of an ill-defined cortical border. As the pathological process progresses, a cortical fracture line becomes visible. Periosteal bone neoformation can be visualized 10 days after the onset of the injury. In cancellous bone, stress fractures have as a pattern the formation of a linear sclerotic band perpendicular to the trabeculae (Figure 2 a, b, c).⁴³

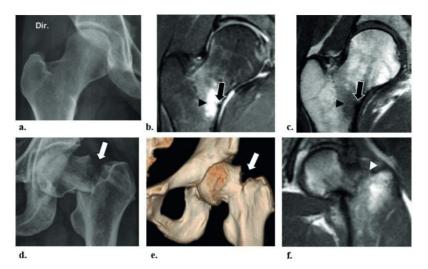


Figure 1 – Low and high-risk femoral fractures. 34-year-old woman complaining of pain in her right hip, beginning three weeks before, while exercising. AP radiograph of the right hip (a) does not show direct signs of fracture. However, proton density-weighted coronal MRI (PD), with fat saturation (b) and T1 coronal MRI (c) demonstrate a linear fracture line (black arrows) with low signal, in the inferomedial femoral neck cortical, surrounded by edema of the adjacent bone marrow (black arrowheads) that exhibits high signal in PD and low signal in T1. - Another female patient, 49 years old, with pain in the anterior compartment left thigh root, of insidious character and worsening for one week after running. Radiograph of the left hip in AP (d) and CT with 3D reconstruction (e) reveal evolution to complete fracture of the superolateral femoral neck cortex (white arrows), with signs of bone misalignment. T1-weighted coronal MRI (f) shows the extensive fracture line (white arrowhead) with low signal, associated with changes in bone angulation / axis in this region.

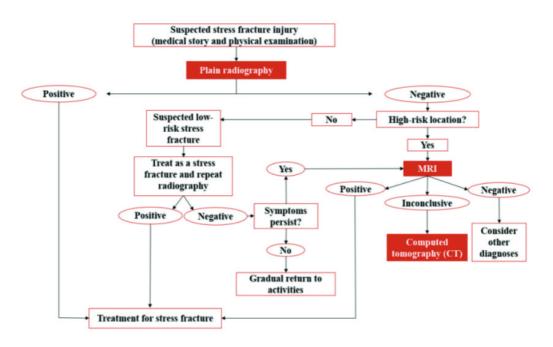


Figure 5 - Flowchart for a principal approach to stress fractures.

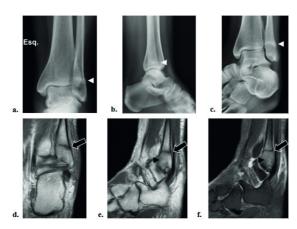


Figure 2 - Low-risk fibular fracture. 41-yearold woman, presenting pain and edema in her left ankle for 15 days, related to functional training at the gym. AP (a), lateral (b) and oblique (c) radiographs of the left ankle demonstrate a range of bone sclerosis (white arrowheads) in the distal metaphyseal region of the fibula, denoting trabecular thickening. MRI in the coronal T1 (d), sagittal T1 (e) and sagittal T2 views with fat saturation (f) better define the lesion, showing a transverse fracture line (black arrows) that extends from the posterior edge to the anterior edge of that region, characterized by low signal in T1 and T2, surrounded by bone edema (black arrowheads), marked by poorly defined medullary zone of low signal in T1 and high signal in T2, as well as edema of the muscleadipose and subcutaneous planes surrounding the distal fíbula.

BONE SCINTIGRAPHY

For a long time, bone scintigraphy was considered the gold standard in the early diagnosis of stress fractures. The degree of lesion uptake depends on the rate of bone metabolism and local vascularization.

It is considered that from 6 to 72 hours after the insult it is already possible to observe the concentration of the radiopharmaceutical in the affected regions, detecting areas of bone remodeling, microfractures of the trabecular bone and periosteal reaction.⁴⁴ This method has high sensitivity, of approximately 100%, but its specificity is lower than radiography, as

other disorders such as tumors, infections or periostitis can generate false-positive results.⁴³

COMPUTED TOMOGRAPHY

Computed tomography (CT) is not the first choice in suspected stress fractures, with lower sensitivity when compared to scintigraphy and MRI. 45,46 However, it can help locate the injury and demonstrate the fracture line that was not well seen on conventional radiography, especially in stress fractures that affect the bones of the pelvis and sacrum. This exam is also used when there is are contraindications for MRI. 47,48

MAGNETIC RESONANCE IMAGING

MRI is the first choice for patients with clinical findings suggestive of stress fractures and without radiographic changes. The findings become evident 1 to 2 days after the onset of symptoms.¹¹ It is the most sensitive and specific modality for diagnosing these lesions, presenting sensitivity similar to that of scintigraphy (100%), but with higher specificity (100%), accuracy (90%), positive predictive value (100%) and negative predictive value (62%).^{34,44,49}

On MRI, typical findings of initial reactions to stress include areas of low signal intensity on T1- weighted images and high signal intensity on T2 and STIR, corresponding to bone marrow edema (Figure 3).²² This method is also used. allows simultaneous assessment of soft tissue structures.

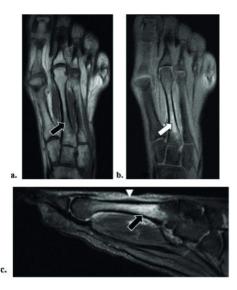


Figure 3 - Bone stress reaction. A 25-yearold female, with a clinical picture of localized pain in her right foot, after running a marathon one month before. Magnetic resonance imaging in the coronal axes of the right forefoot weighted in T1 pre-contrast (a) and T1 post-contrast (b) and in the T2 sagittal view with fat saturation (c) demonstrate accentuating bone edema (black arrows) in the medullar of the second metatarsal, with low intensity signal at T1 and high signal at T2, extending from the base to the distal metaphyseal transition, including significant contrast enhancement (white arrow), compatible with stress reaction. There is also a slight thickening of the cortex (white arrowhead) in the diaphysis region, associated with severe edema of the adjacent soft tissues.

The most advanced stress fractures, classically, are characterized by a linear band, perpendicular to the bone force lines, with low signal at T1 and low signal at T2 / STIR, representing the fracture line, which is surrounded by poorly defined zones of hypo-signal in T1 and hypersignal in T2 / STIR indicating bone edema (Figure 2 d, e, f). In some cases, a high intensity signal in the STIR can also be observed in juxtacortical and subperiosteal locations, translating periosteal reaction. Signal abnormality tends to be

resolved in approximately 6 months. After this period, the altered signal persistence probably represents recurring damage.⁵⁰

In insufficiency injuries, the orientation of the fracture line also tends to be perpendicular to the main trabeculae of the affected bone. This type of fracture is most commonly found in the spine, in which the affected vertebral body collapses / compresses and presents hypo-signal or intermediate signal in T1 and hypersignal in T2 / STIR, reflecting the association with edema or hemorrhage (Figure 4). The fracture line can also be identified. When old and healed, the compression fracture shows a spongy bone marrow signal in all image sequences. ²²

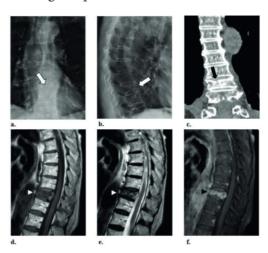


Figure 4 – Compression insufficiency fracture. 82-year-old woman reporting spontaneous pain in her spine for 20 days after sitting up suddenly. AP radiographs (a) and profile (b) of the thoracic spine and CT in coronal reconstruction (c) show flattening / compression fracture of the body of the tenth dorsal vertebra (white arrows), at the expense of depression of its upper plateau (black arrow). T1-weighted sagittal MRI in relation to the intervertebral disc and T2 hypersignal compared to the disc, translating edema or hemorrhage (white arrowheads), with intense uptake by the contrast medium (black arrowhead). It is also associated with the retreat of the superior-posterior wall, reducing the caliber of the medullary canal.

DIFFERENTIAL DIAGNOSIS

PATHOLOGICAL FRACTURE

The patient's medical history and the location of the lesion may be important differentiating between stress pathological fractures. The diagnosis of stress fractures should be favored in young and healthy patients with a history of repetitive activity. Pathological fractures, on the other hand, should be considered particularly in elderly patients with metastatic bone disease. As for its location, stress injuries usually occur in characteristic locations associated with specific activities, while pathological fractures most commonly affect three sites: the subtrochanteric region of the femur, the metaphyseal-diaphyseal junction of the humerus and the vertebral bodies. Although such information is useful, it is not sufficient to reliably discriminate between benign and malignant causes of fractures.7

It is often difficult to differentiate between a vertebral compression caused by an insufficiency fracture and a pathological fracture related to an underlying tumor, specially in multiple myeloma. However, some imaging characteristics may suggest the benign origin of insufficiency fractures, such as the abnormal signal intensity involving only a part of the spinal cord of the vertebral body, the precise linear margin between the normal and abnormal spinal cord, the absence of involvement of the pedicle, the presence of a liquid-filled cleft inside the vertebra in T2weighted images, the absence of a paraspinal mass and the return to the normal spinal cord signal after the injection of the contrast medium (gadolinium).21,33

DIAGNOSTIC SYSTEMATIZATION

In short, due to a nonspecific clinical stress fractures history, complementary imaging studies are essential to offer additional subsidies for the correct diagnosis and management of these injuries. In the scenario of a stress fracture suspicion, conventional radiography in two views of the area of interest is the first test to be performed, as it is accessible and inexpensive, despite its relatively low sensitivity (Figure 5).^{45,46}

If initial radiographs are negative and there is the possibility of a stress fracture at highrisk location, magnetic resonance imaging should be performed due to the high chances of complications.⁴⁶

In the situation where the initial x-ray examination is negative, but that the location of the stress fracture is low-risk for complications, it is recommended that radiography be repeated within 10 to 14 days after the onset of the clinical pain, in an attempt to catch changes that become visible only later in this method.⁴⁶

When there is a strong suspicion of a lowrisk stress fracture and initial radiography is normal, appropriate treatment should begin immediately to prevent progression of the injury, rather than starting the treatment only after confirmation on serial x-ray tests.⁴⁵

Magnetic resonance imaging will also be indicated in cases that persist without radiographic manifestations of the fracture and are refractory to clinical treatment.⁴⁶

In exceptional contexts where there is an immediate need for the diagnosis of a stress fracture, for example, in a professional athlete during a sports season, it is recommended that an MRI be performed directly to speed up the detection and correct recovery of the injury.⁴⁶

Faced with a suspicion of a stress fracture (fatigue or insufficiency) in pregnant women, magnetic resonance imaging is the initial

modality of choice, as it does not expose pregnant patients to ionizing radiation.

Computed tomography is reserved for occasions when magnetic resonance imaging is inconclusive, remaining as a reasonable and less sensitive alternative for the diagnosis of stress fractures, except contraindications.⁴⁶

CONCLUSION

Stress fractures are routinely encountered in medical practice. It is important to understand the terminology and pathophysiology, know the risk factors, symptoms and locations subject to complications of these injuries, as well as understand the complementary radiological resources available, in order to guarantee the identification and characterization of fractures, providing the ideal management to the patient.

The systematic approach to stress fractures, proposed in this article, aims at early diagnosis and, thus, optimizing treatment and adequate follow-up of the injury, as well as preventing complications.

REFERENCES

- 1. Breithraupt MD. Zur pathologie des menschlichen fusses. To the patology of the human foot. Med Zeitung 1855; 24:169.
- 2. Astur DC, et al. Fraturas por estresse: definição, diagnóstico e tratamento. Rev Bras Ortop 2016; 51:3-10.
- 3. Devas MB. Stress fractures of the tíbia in athletes of "shin soreness". J Bone Joint Surg Br 1958; 40(2):227-39.
- 4. Fayad LM, et al. Distinguishing stress fractures from pathologic fractures: a multimodality approach. Skeletal radiology 2005; 34(5):245-259.
- 5. Micheli LJ. Back injuries in dancers. Clin Sports Med 1983; 2(3):473-484.
- 6. Aihara AY, Fernandes AR, Natour J. Valor dos métodos de diagnóstico por imagem na avaliação das reações/fraturas de estresse. Rev. Bras. Reumatol. 2003; 43(3):175-184.
- 7. Marshall RA, Mandell JC, Weaver MJ, Ferrone M, Sodickson A, Khurana B. Imaging Features and Management of Stress, Atypical, and Pathologic Fractures. Radiographics: a review publication of the Radiological Society of North America, Inc. 2018; 38(7): 2173–2192.
- 8. Anderson MW, Greenspan A. Stress fractures. Radiology 1996; 199(1):1-12.
- 9. Bennell KL, Malcolm AS, Thomas AS, Wark JD, Brukner PD. The incidence and distribution of stress fractures in competitive track and field athletes. A twelve-month prospective study. Am J Sports Med 1996; 24(2): 211–7.
- 10. Nivia MH, Mattila VM, Kiuru MJ, Pihlajamaki HK. Bone stress injuries are common in female military trainees: a preliminar study. Clin Orthop Relat Res 2009; 467(11):2962-9.
- 11. Carmont RC, Mei-Dan O, Bennell LK. Stress fracture management: current classification and new healing modalities. Oper Tech Sports Med 2009; 17:81-9.
- 12. Bolin D, Kemper A, Brolinson G. Current concepts in the evaluation and management of stress fractures. Curr Rep Sport Med 2005; 4(6):295-300.
- 13. Mori S, Burr DB. Increased intracortical remodeling following fatigue damage. Bone 1993; 14(2):103-109.
- 14. Cosman F, Ruffing J, Zion M, Uhorchack J, Ralston S, Tendy S, *et al.* Determinants os stress fractures risk in United States Military Academy cadets. Bone 2013; 55(2):359-66.
- 15. Schneiders AG, Sullivan SJ, Hendrick PA, Hones BD, Mcmaster AR, Sugden BA, *et al.* The ability of clinical tests to diagnose stress fractures: a systematic review and meta-analysis. J Orthop Sports Phys Ther 2012; 42(9):760-71.

- 16. Royer M, et al. Stress fractures in 2011: practical approach. Joint Bone Spine 2012; 79:86-90.
- 17. Snyder RA, Koester MC, Dunn WR. Epidemiology of stress fractures. Clin Sports Med 2006; 25(1):37-52.
- 18. Silverman SL. The clinical consequences of vertebral compression fracture. Bone 1992; 13(2):27-31.
- 19. Helms CA, Major NM, Anderson MW, Kaplan PA, Dussault R. Ressonância magnética musculoesquelética. 2ª ed. Rio de Janeiro: Elsevier; 2010.
- 20. Nattiv A, *et al.* Stress fracture risk factors, incidence and distribution: a 3-year prospective study in collegiate runners. Med Sci Sports Exerc 2000; 32(5):347.
- 21. Iwamoto J, Takeda T. Stress fractures in athletes: review of 196 cases. J Orthop Sci 2003; 8(3):273-278.
- 22. Matheson GO, MacIntyre JG, Taunton JE, *et al.* Musculoskeletal injuries associated with physical activity in older adults. Med Sci Sports Exerc 1989; 21(4):379-85.
- 23. Jones BH, Harris JM, Vinh TN, Rubin C. Exercise-induced stress fractures and stress reactions of bone: epidemiology, etiology, and classification. Exerc Sport Sci Rev 1989; 17:379-422.
- 24. Barrow GW, Saha S. Menstrual irregularity and stress fractures in collegiate female distance runners. Am J Sports Med 1988; 16:209-216.
- 25. Friedl KE, Nuovo JA, Patience TH, Dettori JR. Factors associated with stress fracture in young army women: indications for further research. Mil Med 1992; 157: 334-338.
- 26. Carbon R, Sambrook PN, Deakin V, et al. Bone density of elite female athletes with stress fractures. Med J Aust 1990; 153:373-376.
- 27. Warden SJ, Creaby MW, Bryant AL, *et al.* Stress fracture risk factors in female football players and their clinical implications. Br J Sports Med 2007; 41(1):38-43.
- 28. Dixon SJ, Creaby MW, Allsopp AJ. Comparison of static and dynamic biomechanical measures in military recruits with and without a history of third metatarsal stress fracture. Clin Biomech 2006; 21(4):412-9.
- 29. Pohl MB, Mullineaux DR, Milner CE, et al. Biomechanical predictors of retrospective tibial stress fractures in runners. J Biomech 2008; 41(6):1160-5.
- 30. Fujimaki Y, *et al.* The radiological examination for the lateral bowing deformity of femur (in Japanese). Kansetsugeka. 2002; 21: 1144-51.
- 31. Zenke Y, et al. Study of atypical femoral fractures cases coupled in a multicenter study. 2016; 38: 207-2014.
- 32. Park-Wyllie LY, et al. Bisphosphonate use and the risk of subtrochanteric or femoral shaft fractures in older women. JAMA 2011; (305):783-9.
- 33. Sallis RE, Jones K. Stress fractures in athletes: how to spot this underdiagnosed injury. Postgrad Med 1991; 89:185-188, 191-192.
- 34. Edwards PH, Wright ML, Hartman JF. A practical approach for the differential diagnosis of chronic leg pain in the athlete. Am J Sports Med 2005; 33(8):1241-1249.
- 35. Drabicki RR, Greer WJ, Demeo PJ: Stress fractures around the knee. Clin Sports Med 2006; (25):105-15.
- 36. Miller TL, Best TM. Taking a holistic approach to managing difficult stress fractures. J Orthop Sur and Res. 2016; (11):1-8.
- 37. Raasch WG, Hergan DJ. Treatment of stress fractures: the fundamentals. Clin Sports Med 2006; 25(1):29-36.
- 38. Fredericson M, Wun C. Differential diagnosis of leg pain in the athlete. J Am Podiatr Med Assoc. 2003; 93(4):321-4.

- 39. Laurino, CF. Fraturas de estresse e sobrecargas ósseas no esporte. Atualização em ortopedia e traumatologia do esporte. 2009; (1):1-36.
- 40. Montandon C, et al. Sacroiliíte: avaliação por imagem. Radiol Bras 2007; 40(1):53-60.
- 41. Greaney RB, Gerber FH, Laughlin RL, et al. Distribution and natural history of stress fractures in U.S. Marine recruits. Radiology 1983; 146:339-346.
- 42. Nielsen MB, Hansen K, Holmes P, Dyrbye M. Tibial periosteal reactions in soldiers: a scintigraphic study of 29 cases of lower leg pain. Acta Orthop Scand 1991; 62:531-534.
- 43. Greenspan A. Radiologia ortopédica uma abordagem prática. 4ª ed. Rio de Janeiro: Guanabara Koogan; 2006.
- 44. Sofka CM. Imaging of stress fractures. Clin Sports Med 2006; (25):53-62.
- 45. Wright AA, Hegedus EJ, Lenchik L, Kuhn KJ, Santiago L, Smoliga JM. Diagnostic accuracy of various imaging modalities for suspected lower extremity stress fractures: a systematic review with evidence-based recommendations for clinical practice. Am J Sports Med 2016; 44(1):255–263.
- 46. Bencardino JT, et al. ACR appropriateness criteria: stress (fatigue/insufficiency) fracture, including sacrum, excluding other vertebrae. J Am Coll Radiol 2017; 14(55):293-306.
- 47. Shin AY, Morin WD, Gorman JD, Jones SB, Lapinski AS. The superiority of magnetic resonance imaging in differentiating the cause of hip pain in endurance athletes. Am J Sports Med 1996; 24(2):168-76.
- 48. Dixon S, Newton J, Teh J. Stress fractures in the young athlete: a pictorial review. Curr Probl Diagn Radiol 2011; 40(1):29-44.
- 49. Kahanov L, et al. Diagnosis, treatment and rehabilitation of stress fractures in the lower extremity in runners. J Sports Med 2015; (6): 87-95.
- 50. Slocum KA, Gorman JD, Puckett ML, Jones SB. Resolution of abnormal MR signal intensity in patients with stress fractures of the femoral neck. Am J Roentgenol 1997; 168:1295-1299.