Lower Extremity Stress Fractures: General Concepts and Treatment with Focal Shock Waves and Radial Pressure Waves

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Introduction

Stress fractures are secondary injuries, as a result of repetitive loading on the same bone segment, as opposed to fractures due to a traumatic event or insufficiency fractures. This is because stress fractures occur in a normal bone subjected to an abnormal mechanical load, while insufficiency fractures occur in an abnormal bone (with decreased bone mineral density [BMD]) subjected to a normal load [1].

They were first described by Breithaupt in Prussian soldiers suffering from leg pain in the War of 1850, after long and continuous treks carrying heavy equipment on their shoulders [2, 3].

Epidemiology

This type of fracture accounts for between 0.7 and 20% of all injuries treated in the clinical practice of sports medicine [4]. They are generally considered to account for 10% of all sports injuries. About 0.8% of high-performance scholastic athletes will sustain a stress fracture at some point in their sporting lives [5].

The incidence of stress fractures ranges from 1% to 20% of patients consulting for the lower extremity pain who are subjected to intensive physical activity (repetitive mechanical impact) at work or in sports [3, 5].

The most affected population groups are young people, specifically athletes, military personnel, and individuals engaged in the practice of dance [96].

They most frequently affect the lower extremities [5], which are the most exposed to the reaction forces generated by the ground after a given impact. In a U.S. Army series, lower extremity location accounted for 40.3% of all stress fractures [7].

Women are at higher risk than men in terms of developing these injuries [5, 6]. For example, in the U.S. Armed Forces, the 4-year cumulative incidence of stress fracture was 5.7% for male cadets and 19.1% for female cadets [6, 8]. The difference in the incidence of stress fractures found in the literature, with respect to sex, may be explained by certain findings in studies that show that women have smaller bone structures and less muscular strength [5, 6]. In accordance with this, they also have a lower BMD compared to men [6, 9, 10] as well as hormonal irregularities typical of the menstrual cycle [6, 11].

Recent evidence has described a greater incidence of stress injuries in younger and female dancers [12, 13]. In a systematic review by Smith et al., [12] amateur male and female dancers sustained 0.99 and 1.09 injuries for every 1000 dance hours, respectively, whereas professional male and female dancers sustained a total of 1.06 and 1.46 injuries, respectively, during the same period. In professional female dancers, 64% of injuries were related to overuse compared with 50% in professional males; in amateurs, the overuse rate of injury was even higher at 75% [14].

Etiology and Pathogenesis

Stress fractures are similar to the so-called material fatigue failures, which are studied in materials engineering, in the sense that they are due to a large number of repetitions of a mechanical load stimulus, which only repeated once or a few times, does not cause any structural failure [3].

Insufficiency fracture, osteoporotic or pathological, on the other hand, occurs under physiological or normal loads applied on a bone with decreased bone matrix density.

There are intrinsic and extrinsic risk factors that influence the genesis of these injuries. Intrinsic factors include: Mechanical factors, such as alterations in the anatomical or mechanical alignment axis of the lower extremities, with the eventual presence of varus or valgus deformities, type of podal loading (supination loading, which has been associated with a higher incidence of stress fractures of the fifth metatarsal or in association with pes cavus, with a higher rate of stress fractures of the femur or in pronation, either as flat feet or mixed feet, which, in turn, has been related to a higher risk of stress fractures of the tibia, fibula, or tarsal bones), dorsal or plantar flexion of the metatarsals, gastrocnemius with contracture or shortening of the triceps suralis as a global structure, excessively long second metatarsal, etc. [15, 16], and the magnitude or rapidity with which a significant level of muscle fatigue, muscle imbalances, or states of baseline muscle hypotrophy or atrophy are reached. General postural alterations also constitute an influential element; non-mechanical factors, such as metabolic status (alterations in the cyclicity of sex hormones or other hormones that affect the balance between bone resorption and formation by altering calcium/phosphorus metabolism)

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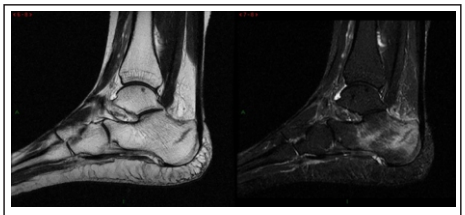

Figure 1: (a and b) Calcaneal stress fracture on T1- and T2-weighted magnetic resonance imaging. Gentleness Dr. Julio Rosales Lecaros.

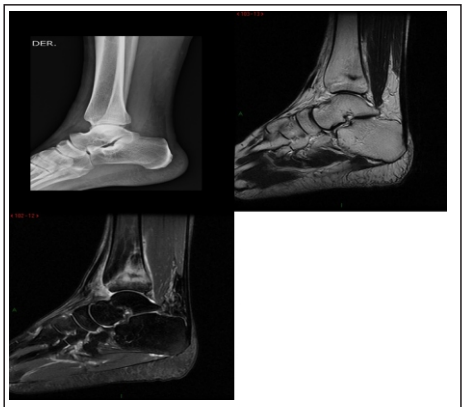

Figure 2: (a-c) Stress fracture of the distal epiphysis of the tibia, where the image of the fracture on the lateral ankle radiograph is not distinguishable, as it is on the magnetic resonance imaging. Gentleness Dr. Julio Rosales Lecaros.

[5].

It is very important to highlight that the hormonal alterations mentioned are present in both sexes; in women, the presence of amenorrhea and oligomenorrhea has been well described in elite athletes [17] and has been related to changes in body composition (loss of fat mass). Menstrual alterations cause a deficit in estrogenic status resulting in a decrease in bone mineral mass and thus increasing the risk of fractures. Ovulatory disturbances associated with alterations in progesterone production and the duration of the luteal phase have also been associated with bone loss [5, 18]. Female athletes with menstrual cycle disturbances are 2–4 times more likely to suffer stress fractures than their counterparts without disorders [5, 18, 19]. In men, it has been also demonstrated abnormally low hormonal states, such as a 25% decrease in testosterone levels within 2 days of vigorous activity; it is known that testosterone inhibits interleukin-6, a cytokine responsible for moderating the development of osteoclasts [20, 21].

Another non-mechanical intrinsic factor is systemic diseases that alter bone vascularization or conditions of deficient bone vascularization as an anatomical characteristic of the bone segment as occurs in the tibia, talus, or navicular or tarsal scaphoid [3, 5, 22].

Among the most common extrinsic factors are: The training regime and the level of basic physical activity (abrupt increase in the duration, intensity or frequency of the activity, inadequate rest period between stimuli, and not respecting a stage of gradual adaptation to the loads in the sporting return after a stage without activity), and dietary habits (especially, deficient intake of calcium and Vitamin D3, low caloric and/or protein intake, and the high and sustained consumption of alcohol). Other extrinsic factors of great importance are inadequate personal equipment and sports infrastructure, such as a type of footwear that does not match the podal load and its null or wrong complementarity with insoles or orthotics and the type and misuse of the surfaces of physical-sports practice: Training or competition areas without proper shock absorption or abrupt changes in the surface of the activity (the transition to harder surfaces). Changes in sporting gestures also play a significant role [1, 6, 21].

Data from a study examining summer adolescent dancers also revealed a high prevalence for stress-related bone injuries with 36 of 239 dancers (15.1%) having a previous history of stress fracture(s) [23].

Ethnicity is also considered influential in the causation of stress fracture, with the lower rates seen in African-American populations compared to Caucasian and Asian groups [24, 25, 26].

Extending the study of favoring factors and based on an analysis of genetic material extracted from leukocytes of two cohorts of military and elite athletes (Israel and United Kingdom), who had suffered stress injuries and with control groups including individuals who had never presented a fracture of this type, it has been possible to identify a pair of functional polymorphisms within the P2X7R gene, which are independently associated with the development of stress fractures. This gene is characterized by its involvement in osteoclast apoptosis and osteoblast activation [27].

A number of genetic factors have been studied in dancers [14]. Genetic variants are common in ballet dancers, with are reported 88% prevalence of variants associated with connective tissue disorders in a professional company, many being collagen genes associated with osseous metabolism [28]. Research suggests that a family history of osteopenia or osteoporosis, frequent fractures, or a combination thereof was strong independent predictors for incidental stress fractures in young women [29]. A study examining elite Korean ballet dancers found that a mutation in the gene a-actinin-3 (ACTN3), which prevents expression of the actinin protein, predisposed dancers to over 4.5•higher risk of sustaining ankle injuries [30]. Dancers with the ACTN3 XX genotype reported markedly less flexibility compared with those with the RX genotype. The ACTN3 XX polymorphism was associated with low fat-free mass in dancers as well. Several genotypes including the estrogen receptor, ESR1 rs9340799 A allele, LRP5 rs2508836 C allele, and LRP5 GCGT/GCAG haplotypes have also been found to be associated with lower BMD at both nonimpact and impact sites in elite and pre-elite dancers [31]. Dancers with small nucleotide polymorphisms of the Wnt/b-catenin signaling pathway and those with higher levels of circulating sclerostin (SOST gene) were also found to have lower BMD, irrespective of other risk factors for low BMD [14].

The most adequate anatomopathological description is to consider these lesions as an entity that encompasses a continuous flow,

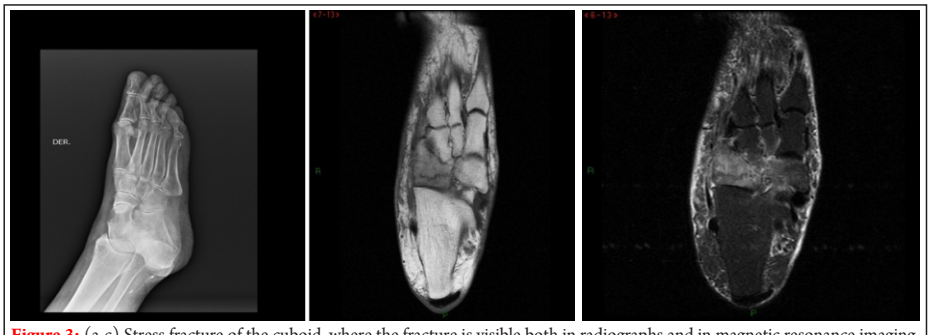

Figure 3: (a-c) Stress fracture of the cuboid, where the fracture is visible both in radiographs and in magnetic resonance imaging. Gentleness Dr. Julio Rosales Lecaros.

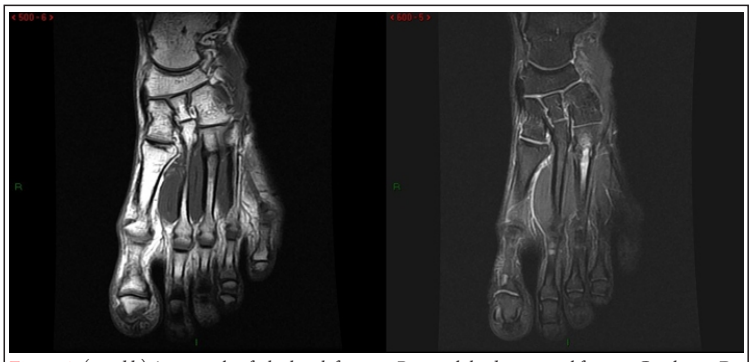

Figure 4: (a and b) An example of a high-risk fracture: Proximal third metatarsal fracture Gentleness Dr. Julio Rosales Lecaros.

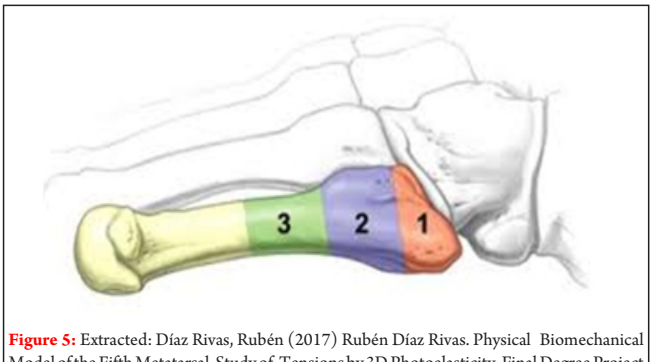

Figure 5: Extracted: Díaz Rivas, Rubén (2017) Rubén Díaz Rivas. Physical Biomechanical Model of the Fifth Metatarsal. Study of Tensions by 3D Photoelasticity. Final Degree Project / Final Degree Project, E.T.S.I. Industrial (UPM).

starting with bone marrow edema, followed by trabecular fracture and cortical rupture or interruption, and ending with complete fracture [5]. Repetitive loading will stimulate osteoclasts to resorption at a faster rate than osteoblasts can form new bone. The normal cycle of remodeling takes 3–4 months [32].

Stress fracture, as we have already said, does not occur as a consequence of a single trauma, but is caused by repetitive or cyclic loads, often occurring in runners who suddenly increase their training load, with lesional compromise of the cortical bone [5, 33]. As a mechanical load is applied, the bone deforms through its elastic range. If the load exceeds the elastic properties of the bone and the bone is unable to absorb it, a fracture of the bone trabecula ("microcrack") occurs. The accumulation of microcracks results in more diffuse damage leading to a fracture itself [5, 34, 35]. Bone tissue remodels permanently, both stochastically and specifically. Bone formation and bone resorption increase with load (directed remodeling). Microfractures appear to increase as the rate of bone repair increases [34]. This increase in bone microdamage leads to apoptosis of osteocytes, with production of pro-osteoclastic osteocytokines, such as RANKL (receptor activator of nuclear factor kappa beta), with differentiation of proosteoclasts [5, 34].

The porosity caused by increased bone resorption weakens and decreases the elasticity of the bone until new bone is completely formed and mineralized. The osteoid matrix has weaker mechanical properties than bone itself. Therefore, directed bone remodeling may predispose to stress fractures [5].

Location

Stress fractures can potentially occur in any bone segment [5]. The location depends on the type of physical activity. In a U.S. Army series, the most frequently injured site was the lower extremities, which accounted for 40.3% of all stress fractures [7]. The location also depends on the physical-sports activity, in joggers, the tibia (33%), tarsal bones (20%), metatarsals (20%), femur (11%), fibula (7%), and pelvis (7%) were the most common locations of stress fractures [5].

Stress fracture of the pars interarticularis is a common injury in young gymnasts and may eventually lead to spondylolysis [5, 36]. Unusual locations are olecranon [37] in gymnasts, hook of the hook [38], thoracic spine [39], radial styloid [40], patella [41], first rib (more common in baseball pitchers, weightlifters, basketball players and dancers) [42, 43], and carpal scaphoid [5, 44].

The location is also influenced by the mechanics of the sport practiced, thus, sprinters, hurdlers, and jumpers have a higher incidence of stress fractures in the feet [5, 45], while long distance runners have more common stress injuries in the pelvis and long bones [5, 45]. In addition, the location also varies with gender, the tibia is the most frequent location of stress fractures in both men and women who participate in jumping and running sports, but women who practice these disciplines present more frequently locations in pelvis and metatarsals, while the fibula is affected less frequently in these activities [5, 45].

Clinical and Imaging

Localized pain is the main symptom of stress fractures. Pain of this type, which is associated with a sudden increase in physical activity or physical activity without adequate rest periods, should raise suspicion of a stress injury [46]. Symptomatology increases with activity and decreases with rest. Local edema may follow painful symptomatology [47]. Local tenderness was present in 66–100% of cases and localized edema in 18–44% of cases [48]. The "hop test" is positive when it causes intense focalized pain. This test is positive in 70% of patients with medial tibial stress fracture, but also in half of the patients with bilateral medial tibial stress syndrome [5, 49, 50].

Magnetic resonance imaging is the imaging test of choice for the diagnosis of stress fractures (Figs. 1a and b), according to the American College of Radiology [5, 48]. The sensitivity of MRI is equal to that of the bone scintigram (100%), but with a higher specificity (85%) [5, 48].

Radiographs of the affected segment are usually normal in the initial stages of the lesion (Figs. 2a, b, c), and some minor findings can be found from the 3rd week of evolution. The initial sensitivity is only 10%, increasing only over 30% after the 3rd week of evolution (Figs. 3a, b, c) [3, 48].

In the author's opinion, the bone scintigram plays some role in the early diagnosis of an asymptomatic stress lesion ("hidden fracture") coexisting with the one that generated the clinical manifestations.

Ultrasound can show findings suggestive of stress fracture, such as periosteal elevation, hypervascularization, hematoma, and even cortical defect, although its sensitivity and specificity are not yet known. Its advantage is its wide accessibility and low cost and it could be considered in the absence of resonance or bone scintigraphy [51].

Laboratory Evaluation

In general, laboratory studies do not aid in the diagnosis of stress fractures unless they become recurrent and more frequent. Vitamin D levels were evaluated prospectively in a large number of military

recruits, and a serum concentration of <20 ng/mL (50 nmol/L) is associated with a higher number of stress fractures than levels >20 ng/mL [52]. For recurrent stress fractures, additional testing would include thyroid-stimulating hormone, parathyroid hormone, and a BMD study [53].

Classification of Stress Fractures

Stress fractures can be classified from a clinical operative point of view, according to their risk of evolution with non-union in the face of conservative management in low-, medium-, or high-risk fractures [54]. Stress fractures of the fibula, lateral malleolus, cuneiforms, distal second to fourth metatarsals, calcaneus, and medial femoral neck (inferior and medial border with the presence of compression trabeculae) are considered low risk. Moderate-risk lesions include: The posteromedial tibia, the femoral diaphysis, the base of the fifth metatarsal, the medial malleolus (except for the configuration as a vertical feature at the junction with the tibial pylon and with complete cortical disruption, a finding that practically requires surgical resolution) [43], and the pelvis. The high-risk category includes fractures of the femoral head and femoral neck at the edge of the tension trabeculae (superior and lateral edge), pars interarticularis (lumbar spine), anterior border of the tibia (anterior tibial cortex), patella (the transverse fracture feature has a greater chance of displacement than the vertical feature) [43], hallux, tarsal bones excluding the cuneiforms or wedges (navicular in particular), the proximal second and third (Figs. 4a and b) metatarsals, and the sesamoids (the medial being the most frequently affected) [43, 51, 55].

In general terms, high-risk stress fractures are loaded in tension and have a poorer natural history, whereas fractures that are loaded in compression have lower risk [14].

However, one-third of low-risk fractures will remain symptomatic after conservative treatment, becoming high-risk fractures [54]. It may be that, in this group, the early indication of shock wave application is the treatment of first choice [54].

A special case of high-risk stress fracture, due to its treatment implications, is the fracture of navicular or tarsal scaphoid fracture.

The treatment of navicular fractures is controversial. Navicular stress fractures can be treated effectively both surgically and conservatively with good clinical outcomes if one has a high index of suspicion. Unfortunately, delays in diagnosis may stall proper treatment and resolution of symptoms. Although the literature does not favor conservative or surgical treatment, the patient is best served when the athletic level and fracture pattern is taken into account [56].

Based on the coronal computed tomography cuts, navicular fractures are classified as follows (Table 1) [57].

For fractures with a dorsal cortical break fractures, the patient is placed in a non-weight bearing cast for 6 weeks. In non-elite athletes with a non-displaced Type II fracture, we will still offer a non-weight bearing cast. For non-displaced Type II fractures that have failed non-weight bearing or in elite athletes who wish to return to sport more quickly, it is recommended screw fixation of the navicular with two screws, typically placed from lateral to medial. For no displaced Type II and III fractures or in fractures with cystic or sclerotic borders, it is recommend open reduction and internal fixation with autogenous bone graft [56].

Fractures of the fifth proximal metatarsal are also a topic that deserves to be analyzed in a special way. (Fig. 5).

The classification of proximal fractures of the fifth metatarsal is as follows (Table 2).

Zone 3 extends distal to the joint of the fifth metatarsal with the fourth and is where stress fractures occur.

Proximal fifth metatarsal stress fractures are potentiated by a cavus alignment, metatarsus adductus, and participation in cutting sports. Proposed bioetiologies include a vascular watershed area between the peroneus brevis insertion and the diaphyseal blood supply [58, 59]. Torg et al. [60] classified these fractures by acuity and radiograph characteristics (Table 3).

Pain and swelling are usually localizable, while radiographs are generally diagnostic. Acute Type I fractures may be treated with a 6-week period of casting and offloading, but some have argued for immediate intramedullary screw fixation to facilitate earlier return to sports and decreased refracture rates, although definitive evidence for these opinions remains lacking [61, 62]. Predisposing cavus or adductus malalignment may influence any decision for early surgery and generates its own controversy in both athletes and non-athletes as to any role for concurrent realignment surgery. Type II and III fractures require operative repair, although canal obliteration may necessitate open plate fixation with bone grafting. Cavus realignment should be heavily considered in such settings, although recovery from its treatment remains difficult to justify in any elite athlete [59]

Treatment

In most cases, conservative treatment consisting of analgesia, immobilization, and/or cessation of the causative activity or external triggers, with gradual return to activity as symptoms permit, will be sufficient. Some authors have suggested the possibility that treatment with non-steroidal anti-inflammatory drugs (NSAIDs) may slow the healing of fatigue fractures and favor the development of pseudarthrosis, so that as far as possible and until adequate studies are available, it is reasonable to limit their use. Local cold and analgesics without anti-inflammatory activity can be used. The use of rigid insoles made, for example, of carbon fiber, can also be a useful aid in treatment [51, 63].

Extracorporeal shockwave therapy (ESWT) involves the application of focused high-energy ultrasound shockwaves, which promote biological healing processes through mechanotransduction. Shockwaves are characterized by high peak pressure, low-tensile amplitude, short rise time, and short duration. The positive phase of the

Table 1 : Saxena classification of navicular fractures.

I.	Dorsal cortex
II.	Extension into the body
III.	Extension from dorsal to plantar cortex with potential avascular , cyst and sclerotic changes.

Table 2: Anatomical classification of proximal fractures of the fifth metatarsal.

- Zone I (pseudo-Jones fracture): Avulsive fracture of the proximal epiphyseal region
- Zone II (Jones fracture): Fracture of the metaphysodiaphyseal junction
- Zone III: Stress fracture of the proximal diaphysis

Table 3: Torg classification of proximal fifth metatarsal stress fractures.

Type description
Type I: Acute
Type II: Delayed union with periosteal and intramedullary bone formation
Type III: Non-union

shockwave produces a direct, mechanical effect, whereas the negative phase produces a cavitation effect in biological tissue [3, 64]. In bone, the application of ESWT is thought to cause neovascularization, periosteal stimulation, and osteoinduction. Periosteal stimulation contributes to cell migration and the development of callus at a bone injury site. Osteoinduction involves osteoblast differentiation from mesenchymal stem cells and inactive cells [64].

The first work with control group, randomized, and double blind, which reported the usefulness of focal shock waves in the treatment of stress fractures, was published in 2001 [3] and considered as study population 26 naval cadets: 13 under treatment and 13 as control group; with 18 years of average age and presenting stress fractures of both tibias, with a similar anatomical and imaging pattern among them; with no response to conservative treatment after 3months.

The analgesic response was evaluated by means of a visual analog scale applied at rest, before and after sports and local pressure. Follow-up was performed for 1year. An evaluation at the beginning and at the end of the follow-up was verified with radiographs, bone scintigram, and nutritional conditions.

Two sessions were applied with 2000 impacts each, with an energy density of 0.1–0.27 MJ/mm2, with an interval of 1week between them.

The results showed that all patients (including those in the control group) were free of symptoms at 1 year of evolution.

All tibiae treated with focal shock waves presented a decrease in their pain level earlier than the control tibiae, including a first analgesic response at 3weeks and this in all four instances of pain assessment (rest, before, and after physical-sports activity and with the local pressure test) [3].

It was concluded that focal shock wave therapy significantly reduces pain and functional recovery time in athletes with high physical requirements and who have been complicated with tibial stress fractures [3].

A retrospective study in 2009 [65], conducted in 10 male patients who practiced soccer at a professional or semi-professional level of competition, with delayed consolidation or frank non-union of stress fractures: 6 patients with involvement of the proximal fifth metatarsal (presence of fine radiolucent line, within 1.5 centimeters distal to the metatarsal tuberosity) and 4 patients with involvement of the tibia.

Three sessions of 4000 impacts each were applied every 48 h for fractures of the fifth metatarsal and four sessions every 48 h for tibial fractures, with an applied energy density between 0.09 and 0.17 MJ/mm2 (patients in the first group were kept in unloading for 6 weeks, using a short open plaster boot, patients with tibial fractures were excluded from any physical sports activity involving jumping or running. Follow-up was clinical and with radiographs.

The results showed that 100% of the patients consolidated under clinical and radiological parameters between 2 and 3 months of follow-up. The return to full sports activity occurred between 3 and 6 months of follow-up.

In another publication [66], experience was presented in five athletes who suffered a fracture of the base of the fifth metatarsal that could be classified as Jones fracture (location of the fracture feature up to 15–20 mm distal to the tuberosity); two patients were soccer players, two patients were basketball players and one was a long jumper. The gender distribution was three males and two females, with an average age of 19.2 years. Two patients had been operated with an intramedullary screw and three patients had undergone conservative management (for the author, it is at least striking, the decision of conservative management in these patients, considering that currently, the management decision in this type of fractures is surgical, given the high risk of inherent nonunion that they imply as an injury).

All patients presented radiographic, ultrasound, and clinical signs of non-union after an average follow-up time of 7.4 months. Three thousand impacts were applied at 0.36 MJ/mm2 of energy density, focused on the site of greatest pain with 1–3 sessions per patient.

In 100% of the patients, there was complete consolidation, in an average time of 11.2 weeks (7–16 weeks) and with a return time to competitive sports activity of 15.8 weeks (11–24 weeks).

The recommendation of the ISMST (International Society for Medical Schockwaves Treatment) as a treatment protocol for stress fractures is as follows:

A single session of 2000 impacts with an energy density of 0.2–0.5 MJ/mm2, under sedation.

Followed by a rehabilitation program including: Immediate active range of joint mobility without mechanical stress, progressive loading, and gradual sport return. Consolidation should be expected 4–6 weeks after the procedure.

In patients with moderate and high-risk stress fractures, unloading and immobilization with orthopedic boot or plastic cast should be performed [67].

Regarding the application of radial pressure waves in stress fractures, there is insufficient evidence available at present to recommend their use in an exhaustive manner [3]. However, there is experience in the treatment of superficial bone non-unions (carpal and tarsal scaphoid, tibia, fibula, medial and lateral malleoli, iliac crest, second, and fifth metatarsals) with radial pressure waves and successful results, which could eventually be ex-trapolated to the treatment of stress fractures [3, 68].

The author, considering the above, has achieved good results (not yet published) in the treatment of stress fractures in tarsal and metatarsal bones with radial pressure waves, mainly in the high-performance sports population.

Conclusions

Stress fractures are a widely represented pathology in traumatology, sports medicine, and rheumatology. Women are the most frequently affected gender, most likely due to ovulatory alterations associated with ovulatory deficiency associated with steroid hormone deficiency and anatomical predisposition. Stress fractures mainly affect the lower extremities. Magnetic resonance imaging is the "gold standard" in imaging standard in imaging diagnosis. Physical sports rest and analgesics are the primary therapy. Shock waves are a very useful tool for the management of refractory conditions or for early and preventive treatment in sports patients [5]. However, to date, there are no randomized control trials that provide Level I evidence for extracorporeal shock wave therapy (ESWT) in stress bone pathology [64, 69].

A multidisciplinary team approach is particularly helpful when managing stress injuries, given their multifactorial nature [14].

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the Journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

Conflicts of Interest: Nil. **Source of Support:** None.

References

1. Carlín AG, Garza JI, Parada CA. Fracturas porestrés enelatleta: Epidemiología y manejo. Orthotips 2016;12:12-27.

2. Devas MB: Stress fractures of the tibia in athletes or shin soreness. J Bone Joint Surg Br 1958;40-B:227-39.

3. Carlos L, D'Agostino C, Garcia SG, Fernandez A. Current concepts of shockwave therapy in stress fractures. Int J Surg 2015;24:195-200.

4. Fredericson M, Jennings F, Beaulieu C, Matheson GO. Stress fractures in athletes. Top Magn Reson Imaging 2006;17:309-25.

5. Saunier J, Chapurlat R. Stress fracture in athletes. Joint Bone Spine 2018;85:307-10.

6. Sandoval JC, Huamán LV, Cruz-Vargas JA. Factors associated with stress fracture: A case-control study in a peruvian navy medical center. Medwave 2020;20:e7936.

7. Changstrom BG, Brou L, Khodaee M, Braund C, Comstock RD. Epidemiology of stress fracture injuries among US high school athletes, 2005-2006 through 2012-2013. Am J Sports Med 2015;43:26-33.

8. Waterman BR, Gun B, Bader JO, Orr JD, Belmont PJ Jr. Epidemiology of lower Extremity stress fractures in the united states military. Mil Med 2016;181:1308-13.

9. Beck TJ, Ruff CB, Shaffer RA, Betsinger K, Trone DW, Brodine SK. Stress fracture in military recruits: Gender differences in muscle and bone susceptibility factors. Bone 2000;27:437-44.

10. Wentz L, Liu PY, Haymes E, Ilich JZ. Females have a greater incidence of stress fractures than males in both military and athletic populations: A systemic review. Mil Med 2011;176:420-30.

11. Kelsey JL, Bachrach LK, Procter-Gray E, Nieves J, Greendale GA, Sowers M, et al. Risk factors for stress fracture among young female cross-country runners. Med Sci Sports Exerc 2007;39:1457-63.

12. Smith PJ, Gerrie BJ, Varner KE, McCulloch PC, Lintner DM, Harris JD. Incidence and prevalence of musculoskeletal injury in ballet: A systematic review. Orthop J Sport Med 2015;3:2325967115592621.

13. Sobrino FJ, Guillén P. Overuse injuries in professional ballet: Influence of age and years of professional practice. Orthop J Sport Med 2017:32 .

14. Jotwani VM, Aflatooni JO, Barter LE, Harris JD. Management of stress fractures in ballet. J Am Acad Orthop Surg 2022;30:543-53.

15. Del Río Martínez PS, García MS, Berges MP, Rubio PB. Fractura de estrés en metatarsos: A propósito de dos casos. Rev Osteoporos Metab Miner 2015;7:67-70.

16. Pecina MM, Bojanic I. Overuse Injuries of the Musculoskeletal. 2nd ed. Boca Raton: CRC Press; 2004. p. 421.

17. Barrow G, Saha S. Menstrual irregularity and stress fractures in collegiate female distance runners. Am J Sports Med 1988;16:209-16.

18. Boyden TW, Pamenter RW, Stanforth P, Rotkis T, Wilmore JH. Sex steroids and endurance running in women. Fertil Steril 1983;39:629-32.

19. Warden SJ, Creaby MW, Bryant AL, Crossley KM. Stress fracture risk factors in female football players and their clinical implications. Br J Sports Med 2007;41:i38-43.

20. Girasole G, Jilka R, Passer G, Boswell S, Boder G, Williams DC. 17beta-estradiol inhibits interleukin-6 production by bone marrow-derived stromal cells and osteoblasts in vitro: A potential mechanism for the antiosteoporotic effect of estrogens. J Clin Invest 1992;89:883-91.

21. Torrengo F, Paús V, Cédola J. Fracturas por estrés en deportistas algoritmo de estudios complementarios actualizado y estadificación. Rev Asoc Argentina De Traumatol Deporte 2010;17:18-23.

22. Taylor D, Kuiper JH. The prediction of stress fractures using a "stressed volumen" concept. J Orthop Res 2001;19:919-26.

23. Thomas JJ, Keel PK, Heatherton TF. Disordered eating and injuries among adolescent ballet dancers. Eat Weight Disord 2011;16:e216-22.

24. Lakmini Bulathsinhala,Julie M Hughes,Craig J McKinnon,Joseph R Kardouni,Katelyn I Guerriere,Kristin L Popp,Ronald W Matheny Jr,Mary L Bouxsein.. Risk of Stress Fracture Varies by Race/Ethnic Origin in a Cohort Study of 1.3 Million US Army Soldiers.First .JBMR.3131 2017:32:1546-53.

25. Bennell K, Matheson G, Meeuwisse W, Brukner P. Risk factors for stress fractures. Sports Med 1999;28:91-122.

26. Vaitkevicius H, Witt R, Maasdam M, Walters K, Gould M, MackenzieS, et al. Ethnic differences in titratable acid excretion and bone mineralization. Med Sci Sports Exerc 2002;34:295-302.

27. Varley I, Greeves JP, Sale C, Friedman E, Moran DS, Yanovich R, et al. Functional polymorphisms in the P2X7 receptor gene are associated with stress fracture injury. Purinergic Signal 2016;12:103-13.

28. Vera AM, Peterson LE, Dong D, Haghshenas V, Yetter TR, Delgado DA, et al. High prevalence of connective tissue gene variants in professional ballet. Am J Sports Med 2020;48:222-8.

29. Loud KJ, Micheli LJ, Bristol S, Austin SB, Gordon CM. Family history predicts stress fracture in active female adolescents. Pediatrics 2007;120:e364-72.

30. Kim JH, Jung ES, Kim CH, Youn H, Kim HR. Genetic associations of body composition, flexibility and injury risk with ACE, ACTN3 and COL5A1 polymorphisms in Korean ballerinas. J Exerc Nutrition Biochem 2014;18:205-14.

31. Amorim T, Duraes C, Machado JC, Metsios GS, Wyon M, Maia J, et al. Genetic variation in Wnt/ b-catenin and ER signalling pathways in female and male elite dancers and its associations with low bone mineral density: A crosssection and longitudinal study. Osteoporos Int 2018;29:2261-74.

32. Denay KL. Stress Fractures. Curr Sports Med Rep 2017;16:7-8.

33. Bennel KL, Brukner PD. Epidemiology and site specificity of stress fractures. Clin Sports Med 1997;16:179-96.

34. Hughes JM, Popp KL, Yanovich R, Bouxsein ML, Matheny RW Jr. The role of adaptive bone formation in the etiology of stress fracture. Exp Biol Med (Maywood) 2017;242:897-906.

35. Chapurlat RD, Delmas PD. Bone microdamage: A clinical perspective. Osteoporos Int 2009;20:1299-308.

36. D'Hemecourt PA, Zurakowski D, Kriemler S, Micheli LJ. Spondylolysis: Returning the athlete to sports participation with brace treatment. Orthopedics 2002;25:653-7.

37. Hetling T, Bourban P, Gojanovic B. Stress fracture and nonunion of coronoid process in a gymnast. Case Rep Orthop 2016;2016:9172483.

38. Van Demark RE Jr., Van Demark RE, Helsper E. Stress fracture of the hook of the hamate: A case report. S D Med 2015;68:157-9, 161.

39. Jha SC, Sakai T, Hangai M, Toyota A, Fukuta S, Nagamachi A, et al. Stress fracture of the thoracic spine in an elite rhythmic gymnast: A case report. J Med Invest 2016;63:119-21.

40. Hashiguchi H, Iwashita S, Ohkubo A, Sawaizumi T, Takai S. Stress fracture of the radial styloid process in a judo player: A case report. J Nippon Med Sch 2015;82:109-12.

41. Park CJ, Suh KT, Lee SM, Cho HJ. Longitudinal stress fracture of the patella in a female weightlifter. J Orthop Sci 2016;21:241-4.

42. Low S, Kern M, Atanda A. First-rib stress fracture in two adolescent swimmers: A case report. J Sports Sci 2016;34:1266-70.

43. Kiel J. Kimberly Kaiser Stress Reaction and Fractures In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2022 [Last accessed on 2022 Jan May 13].

44. Kohring JM, Curtiss HM, Tyser AR. A scaphoid stress fracture in a female collegiate-level shot-putter and review of the literature. Case Rep Orthop 2016;2016:8098657.

45. Bennell KL, Malcolm SA, Thomas SA, 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;24:211-7.

46. Ohta-Fukushima M, Mutoh Y, Takasugi S, Iwata H, Ishii S. Characteristics of stress fractures in young athletes under 20 years. J Sports Med Phys Fitness 2002;42:198-206.

47. Fredericson M, Bergman AG, Hoffman KL, Dillingham MS. Tibial stress reaction in runners. Correlation of clinical symptoms and scintigraphy with a new magnetic resonance imaging grading system. Am J Sports Med 1995;23:472-81.

48. Patel DS, Roth M, Kapil N. Stress fractures: Diagnosis, treatment, and prevention. Am Fam Physician 2011;83:39-46.

49. Clement D, Ammann W, Taunton JE, Lloyd-Smith R, Jesperson D, McKay H, et al. Exercise-induced stress injuries to the femur. Int J Sports Med 1993;14:347-52.

50. Batt ME, Ugalde V, Anderson MW, Shelton DK. A prospective controlled study of diagnostic imaging for acute shin splints. Med Sci Sports Exerc 1998;30:1564-71.

51. Escobar-de-las-Heras MN, Sevilla-Lerena MP, Ochoa-Prieto J. Fracturas por fatiga: Sospecha clínica y perseverancia diagnóstica. Med Familia Sermegen Dic 2010;10:590-2.

52. Moreira CA, Bilezikian JP. Stress fractures: Concepts and therapeutics. J Clin Endocrinol Metab 2017;102:525-34.

53. May T, Marappa-Ganeshan R. Stress fractures. Treasure Island FL: StatPearls. NIH National Library of Medicine; 2021.

54. Haffner N, Smolen D, Dahm F, Schaden W, Mittermayr R, Wang CJ, et al. Significance of extracorporeal shockwave therapy in fracture treatment. Transl Res Biomed 2018;6:42-63.

55. Wang CJ, Schaden W, Ko JY. Shockwave Medicine. Transl Res Biomed 2018;6:42-63.

56. Gross CE, Nunley NA 2nd. Navicular stress fractures. Foot Ankle Int 2015;36:1117-22.

57. Saxena A, Fullem B, Hannaford D. Results of treatment of 22 navicular stress fractures and a new proposed radiographic classification system. J Foot Ankle Surg 2000;39:96-103.

58. Notarnincola A, Moretti B. The biological effects of extracorporeal shock wave therapy (eswt) on tendon tissue. Muscles Ligaments Tendons J 2012;2:33-7.

59. Kaiser PB, Guss D, DiGiovanni CW. Fractures of the foot and ankle in athletes. Foot Ankle Orthop. 2018;(3):1.

60. Torg JS, Balduini FC, Zelko RR, Pavlov H, Peff TC. Fractures of the base of the fifth metatarsal distal to the tuberosity: Classification and guidelines for non-surgical and surgical management. J Bone Joint Surg Am 1984;66:209-14.

61. Coughlin MJ, Saltzman CL, Mann RA. Mann's Surgery of the Foot and Ankle E-Book: Expert Consult-Online. Philadelphia, PA: Saunders, Elsevier; 2013.

62. Quill GE Jr. Fractures of the proximal fifth metatarsal. Orthop Clin North Am. 1995;26:353-61.

63. Wheeler P, Batt ME. Do non-steroidal anti-inflammatory drugs adversely af-fect stress fracture ealing? A short review. Br J Sports Med 2005;39:65-9.

64. Roche M, Abrams G, Fredericson M. Systemic Treatment Modalities for Stress Fractures. United States: Stanford University Stanford University; 2020. p. 141-9.

65. Biagio M, Notarnicola A, Garofalo R, Moretti L, Patella S, Marlinghaus E, et al. Shock Waves in the treat-ment of stress fractures. Ultrasound Med Biol 2009;35:1042-9.

66. Tanaka K, Kanamori A, Kajiwara M, Nishino T, Nishida Y, Yamazak M. Extracorporeal shock wave therapy (ESWT) for refractory fractures at the fifth metatarsal base. Int J Foot Ankle 2019;3:27.

67. Schaden W. DIGEST Guidelines to the Extracorporeal Shock Wave Therapy ISMST (The International Society for Medical Schockwaves Treat-ment) Updated 05/2019.

68. Kertzman P, Császár NB, Furia JP, Schmitz C. Radial extracorporeal shock wave therapy is efficient and safe in the treatment of fracture nonunions of superficial bones: A retrospective case series. J Orthop Surg Res 2017;12:164.

69. Leal C, Berumen E, Bucci S, Castillo A. Extracorporeal shockwave therapy and sports-related injuries. Transl Res Biomed 2018;6:70-86.

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