Return to Play in Stress Fractures of the Hip, Thigh, Knee, and Leg

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31.1 Introduction

31.1.1 Epidemiology and Clinical Presentation

In brief, "stress fractures" occur when a normal (or pathologic) bone is incapable of recovery from repeated strains that occur during physical activity [1]. Typically, such fractures have been linked to specific groups like military personnel or athletes from running and jumping sports, but in fact they can occur in anybody from general population [2].

The relationship between these fractures and military has historical origins. The first description of such injuries comes from the nineteenth century and was described as the "swollen feet syndrome" found after long marches in Prussian soldiers. Such swelling was demonstrated to be due to metatarsal stress fractures [3].

The incidence among military recruits varies from 5% to 30% per year, and it has been shown to be significantly higher among women [2, 4, 5].

Among athletes (nonmilitary), the incidence ranges from 1.1% to 31% per year and is highest among long-distance runners [2, 6–8]. Such fractures represent 10–20% of sports injuries due to overtraining [9–11]. They can affect any bone; however, it has been recognized that 90% of these are lower extremity stress fractures (Fact Box 1) [12]. The most affected bone in footballers is the fifth metatarsal, and the second most affected is the tibia [10, 13–15].

Regardless of competitive level, football is one of the most frequent sports worldwide [16]. Thus, football-related injuries have a high socioeconomic impact [17, 18]. Moreover, concerning high-level athletes, injuries lead to absence from competition (with all related consequences), costs related to treatment, and potential endangerment of an athlete's career [17].

Despite the former, there remains limited data and research in this topic related to footballers [1, 19–24]. In a recent study on elite male football players, stress fractures accounted for 0.5% of all injuries, and the incidence was 0.04/1000 h [19]. Thus, one might conclude that a team with 25–28 players could expect a stress fracture every third season [19]. Another study reinforces the influ-

ence of gender as risk factor, finding a 13.6% incidence of stress fractures among female football players [22].

Fact Box 1

Distribution of lower extremity stress fractures in male footballers [19]	
Stress fracture site	Frequency (%)
Fifth metatarsal	78.4
Tibia	11.7
Pelvis	5.8
Tarsal bones	1.9
Fibula	1.9

31.1.1.1 Clinical Presentation

The early diagnosis of a stress fracture relies on a careful clinical history and physical examination. The team physician must be aware of prodromal symptoms before the onset of more serious injuries. Typically, patients with stress fractures present with insidious onset of localized pain. The athlete's complaints often correlate with, or are aggravated by, physical activity and are relieved by rest, reappearing after repeated effort. The absence of rest pain helps on differential diagnosis of stress fractures with a variety of conditions, including inflammatory processes, acute fractures, and tumors.

Many times, there is a history of change/increase in vigor of training/physical activity, change in pattern or effort, change in footwear, or a different training field (e.g., harder surfaces) [1, 25]. Athletes with stress fractures might experience pain on palpation or strain on the involved bone—the athlete typically recognizes the pain he/she feels after the activity when the examiner presses over [26]. Swelling, redness, or warmth may or may not be present.

31.1.2 Mechanism of Injury, Risk Factors, High- Versus Low-Risk Injuries

The skeleton is subjected to several types of forces during motion and activity. The human bone follows *Wolff's law*, which describes the way this tissue will adapt to mechanical stress [27]. As the force acting on bone rises, it will deform accordingly, given the inherent elasticity of the tissue. If loading on a particular bone increases, the bone will remodel itself over time in order to become stronger and to be capable to resist that kind of stress. The internal architecture of the tissue suffers adaptive modifications (e.g., temporary elastic deformation), followed by secondary changes to the external cortical portion of the bone, which can become thicker as a result. The inverse is also true: if the loading on a bone decreases, the bone will become less dense and weaker due to the lack of the stimulus required for continued remodeling. Thus, balance is required to promote a healthy bone [28].

When the bone is exposed to forces within its capacity to support them, it undergoes elastic deformation, recovering its histological configuration as soon as the load discontinues. However, when these strains exceed bone's resistance, the elastic deformation is superseded by plastic deformation: there is no return to the previous situation, and, in case the repetitive forces remain, microfractures can occur. In such cases, bone reabsorption will occur.

Once this balance is disrupted (e.g., consecutive microtrauma without permitting the bone to recover completely from the initial stress) and microtrauma accumulates, a "stress fracture" can occur [28].

According to the described mechanism, stress fractures occur in three phases: microfracture, propagation of the microfracture, and complete fracture [28, 29]. Such fractures typically occur in places submitted to high tension [25, 27, 29, 30]. This is different from the mechanism involved on a normal acute fracture. In this case, a single load exceeds the bony resistance and crates a failure of the tissue. Opposing, stress fractures typically derive from repeated microtauma. In these cases, not only the strain intensity but also its frequency and duration, among other physical conditions, are determinant [31–33].

There are still a number of questions to be answered concerning the precise conditions, which might lead to these injuries, thus enabling improved prevention strategies and early

diagnosis. However, several risk factors (intrinsic and extrinsic) have been identified, for example, gender and a predilection for young people and athletes [34]. Fact Box 2 summarizes the risk factors which gather higher level of scientific support [1].

Fact Box 2 (Based on [1])

Important risk factors for "stress fractures"	
Intrinsic factors	Extrinsic factors
Diminished physical activity	Changes in training program
Low "body mass index" and low bone mineral density	Running on hard surfaces
Age: In men, the incidence lowers above the age of 17 In women, the incidence increases after the menarche	Long-distance runners
Diminished muscle strength	Alcohol
Anatomical factors: e.g., leg-length discrepancy, low cross-sectional area	Smoking
Eating disorders	Low vitamin D levels
Menstrual dysfunction	

31.1.2.1 Risk Factors

Several intrinsic and extrinsic risk factors have been proposed [1]. Extrinsic factors include change in training program, sports on hard surfaces, and type of sports (e.g., long-distance runners) [35–37]. The influence of shoewear has been proposed but remains somewhat controversial with some discrepancies in literature [1]. In a recent study, it was proposed that a sports shoe with more than 6 months of regular use and training in hard surfaces increases the risk of stress injuries by diminishing the capacity to dissipate energy when the foot strikes the pitch [38]. Caffeine, alcohol, and tobacco can also increase the risk for bone stress injury [39].

Intrinsic factors that have been proposed include gender (female with higher risk), age (higher in young ages), physical condition, anatomic features, and history of previous stress fractures [34, 40–42]. Most studies suggest that females have higher risk of stress fractures, in part related to the "female athlete triad" [43]. In addition, differences among genders also include lower cross-sectional bone area and neuromuscular response [44, 45].

Other intrinsic factors include leg-length discrepancy, history of previous surgeries or trauma, hyperlaxity, instability, and muscle weakness [10, 46, 47]. Muscle weakness has been considered an important risk factor once muscles help to dissipate the energy thus lowering strain transmitted to bone during running or jumping [48]. This theory has been described as the "neuromuscular hypothesis" [28, 37]. Training overload with short recovery times might lead to overtraining [49]. This condition increases risk of bone stress injury and leads to paradoxical lowering of performance [50].

Finally, some stress fractures have been linked to specific anatomic features. For example, tibial stress fractures have been linked to anatomic conditions such as *cavus* foot, smaller tibial bone, and foot overpronation [51, 52].

31.1.2.2 High-Risk and Low-Risk Stress Fractures

High-risk injuries occur on the side of maximal tension (e.g., tension-sided femoral neck fractures, anterior tibial diaphyseal fractures, tension-sided patellar fractures) and hypovascular zones [53, 54]. High-risk lesions have higher probability for longer recovery; are more prone to progression to complete fracture, delayed union, and chronic pain [53, 54]; and more often require surgical treatment [1].

31.2 Imaging of Lower Extremity Stress Fractures

31.2.1 Radiographies

Given the low cost and availability, plain radiographs are usually the first imaging exam to be performed. Plain radiographs provide important anatomic information and can identify cortical thickening, impingement lesions, and displaced fractures. However, they have very low sensitivity

for stress fractures, particularly in the first 3–4 weeks, particularly in the presence of osteopenia [25, 55, 56]. Stress fractures can sometimes be seen as a subtle linear sclerosis, focal endosteal or periosteal reaction, or a fracture overlapped and hidden by a periosteal reaction [25]. So, a negative X-ray cannot exclude a stress fracture. Sometimes the X-ray will be positive later in the course (typically weeks to months) showing bone callus and remodeling in the later phase of this condition (possibly much latter than the full clinical recovery) [57]. If there is the clinical suspicion and a negative X-ray, another imaging exam should be obtained (e.g., MRI, bone scan, or CT) [58].

31.2.2 Bone Scan

Three-phase bone scintigraphy used to be the gold standard given its high sensitivity in detecting the early phases of stress fractures (detects the increased metabolic activity around the injury site) [59]. However, bone scintigraphy is accompanied by a high level of inherent radiation and very low specificity with up to 40% of false positives [60]. It cannot distinguish from inflammatory processes, infection, or tumor diseases [58]. Moreover, it can show increased uptake in the injury site up to 2 years after an injury is clinically resolved [60].

31.2.3 CT Scan

Computed tomography (CT) scans can be highly valuable for assessment of bone stress injuries, despite exposing patients to radiation. In the scope of stress fractures, CT scans remain useful in sacral fractures and spondylolysis and in differentiating tumors, infection, or bone stress reaction from stress fractures [58].

31.2.4 MRI

MRI is currently the most used imaging exam given its high sensitivity and specificity (ranging from 85% to 100%) [9, 61, 62]. Under the clinical suspicion of bone stress injury, negative plain radiography, and pain without defined etiology, MRI is the first-line exam [63]. It is possible to identify fracture lines hypointense in T1 and T2 sequences, usually combined with bone marrow edema, and hyperintensity in surrounding soft tissue on SatFat or STIR sequences [33]. The presence of bone edema is not specific but is highly sensitive for stress reaction [33]. MRI assessment besides confirming the diagnosis is usually helpful in determining the severity of the condition. In this way, it also helps in the decision for choice of treatment and can be useful in deciding the timing to return to play (by assessing the severity of the lesion). One of the most used classifications for tibial stress fractures characteristics in MRI has been described by Fredericson and others [33, 64–68]. Table 31.1 represents the Fredericson classification as one example of how MRI can be used to assess the most common type of stress fracture herein described (tibial). Table 31.2 summarizes another classification method based on MRI and used as guideline for treatment [64].

Table 31.1 MRI Fredericson classification for tibial stress fractures

Grade	Periosteal edema	Marrow STIR SI	Marrow T1 SI	Intracortical sign
0	No	Normal	Normal	Normal
1	Yes	Normal	Normal	Normal
2	Yes	High	Normal	Normal
3	Yes	High	Low	Normal
4°	Yes	High	Low	Focal abnormality
4b	Yes	High	Low	Linear fracture

STIR short-TI inversion recovery; *SI* signal intensity [65]

Table 31.2 Stages/grades of Arendt and Griffiths [64] for stress fracture, based on magnetic resonance imaging (MRI) findings and correlation with period of rest required

Classification of Arendt and Griffiths		
Grade of injury	MRI findings	Required period of rest (weeks)
I	STIR-positive	3
II	STIR and T2-weighted positive images	3–6
III	T1- and T2-positive without definition of cortical rupture	12–16
IV	T1- and T2-positive with definition of cortical rupture and visible fracture line	>16

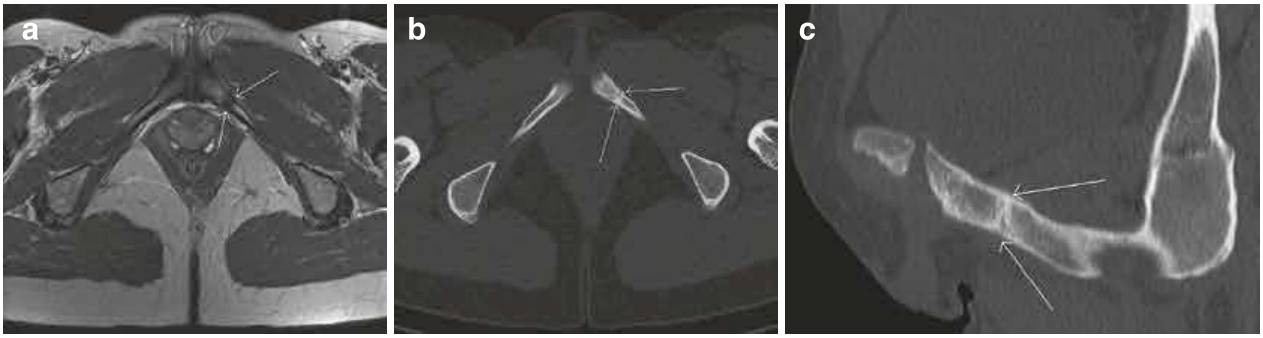

Fig. 31.1 Inferior pubic ramus stress fracture (*white arrows*) on MRI (**a**) and CT views (**b**, **c**)

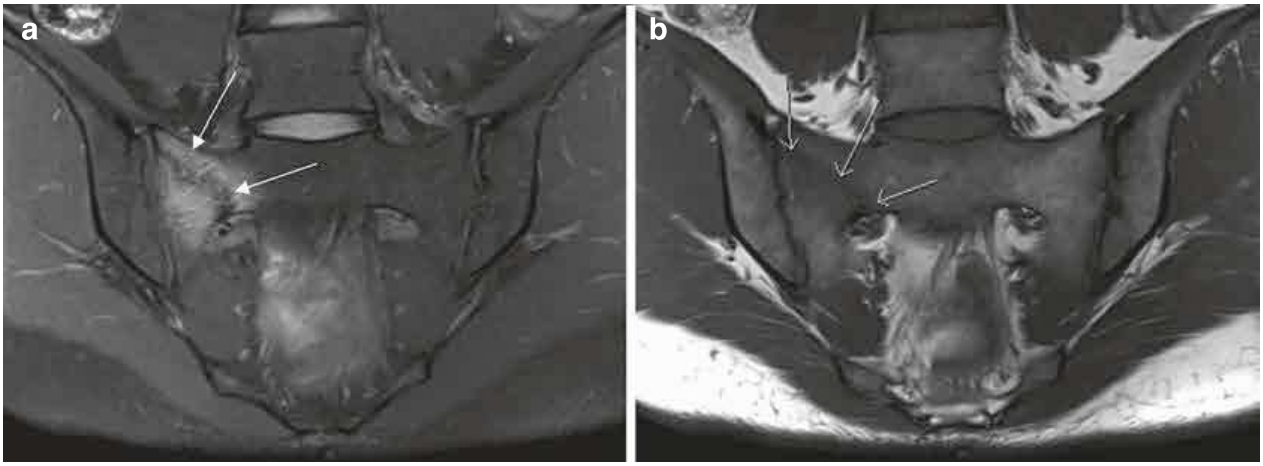

Fig. 31.2 MRI views of sacral stress fracture (*white arrows*) on STIR (**a**) and T1 (**b**)

31.3 Pelvic Stress Fractures

Pelvic stress fractures can occur in a wide spectrum of structures, such as the pubic rami (Fig. 31.1), the sacrum (Fig. 31.2), and the apophyses (e.g., anterior superior iliac spine or the ischial tuberosity). Stress fractures around the pelvis represent the third most common type of stress fractures encountered in football [19]. Stress fractures of the pubic rami are rare and usually occur in the medial portion or at the junction between the inferior pubic ramus and the symphysis pubis [69].

The occurrence of sacral stress fractures (Fig. 31.2) is probably underreported, given the general lack of awareness of this condition and the nonspecificity of symptoms. Stress fractures of the sacrum have been described primarily in long-distance runners, especially females and military [70–72]. They are described as fatigue

and insufficiency fractures. The diagnosis is often delayed or inaccurate due to limited overall awareness of this condition and the lack of specific symptoms [71].

The clinical presentation of athletes with pelvic stress fractures consists of insidious onset of pain in the hip or lower back. Despite its low sensitivity, radiographs can identify pubic ramus stress fractures, which might be visible as non-displaced fracture lines. However, it can be difficult to identify sacral stress fractures on plain radiography, which is particularly difficult given the overlying bowel gas. CT scan or MRI is necessary for accurate diagnosis. Treatment consists of a period of rest and medication. It has been stated that most fractures heal well within 6–10 weeks [51, 73].

31.4 Femoral Stress Fractures

Femoral stress fractures (Fig. 31.3) represent 4.2–48% of all stress fractures in athletes [1, 51, 73]. Femoral stress fractures are less common than pelvic stress fractures and are more common among female runners [74]. Femoral stress fractures (Fig. 31.3) can occur in the femoral neck or the femoral shaft [74].

Plain radiographies often fail to detect femur stress fractures. A high index of suspicion is required in order to achieve early diagnosis in an initial phase of this condition and avoid more severe consequences, such as displacement of a fracture. MRI is an important imaging tool since it enables detection of bone edema and hypointense lines representing fractures on early stages (Fig. 31.3) [74]. Kiuru et al. have described a helpful system for MRI grading of pelvic bones and proximal femur [75].

31.4.1 Femur Neck Stress Fractures

Considering the same biomechanical principles, the superolateral section of the femoral neck corresponds to its tensile site opposing to the inferomedial part (compression). The morphology of the proximal femur probably plays a role in the susceptibility of the patient to develop a stress fracture. *Coxa vara* predisposes to femoral neck stress fracture [76]. *Coxa vara* substantially modifies the biomechanical conditions of the femoral neck, increasing the effect of direct muscle pull and leading to fatigue of opposing muscle groups favoring stress fractures [76]. Femoral neck stress fractures have been linked to female

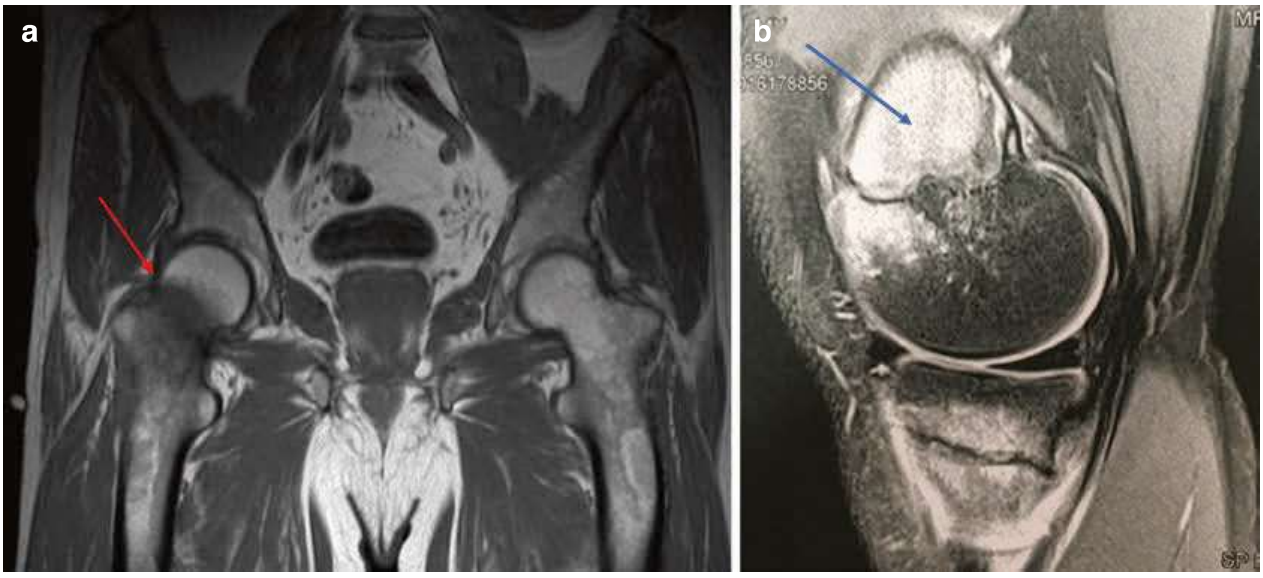

Fig. 31.3 MRI views of femoral neck stress fracture (*red arrow*) (**a**) and stress reaction (*blue arrow*) of the distal femur of an immature footballer (**b**)

athlete triad and might occur in either tension or compression sites [7, 77].

The tension-sided femoral neck fractures are higher-risk injuries with a worse prognosis (i.e., higher risk for displacement) [74]. The presence of displacement increases the risk of a worse clinical outcome (higher morbidity and lower functional results) and increases the risk of complications (osteonecrosis, refracture, or pseudarthrosis) [77]. Considering the potential consequences of complete fracture, tension-sided femoral neck fractures are surgically treated even in early stages (most often by osteosynthesis with three screws) [1]. Considering the biomechanical risk factors in the femoral neck, fractures in the compression side might be managed conservatively, but refractory cases typically require surgical repair.

31.4.2 Femoral Shaft Stress Fractures

Considering human anatomy, the femoral diaphysis has slight anterolateral bowing. Bearing this in mind, the anterolateral surface corresponds to the tension site, while the posteromedial surface corresponds to the compression site. In this case, most stress fractures occur as a consequence of repetitive microtrauma on the compression side, at the junction of its proximal and middle thirds. The pathophysiological explanation is that this is the area of origin of the *vastus medialis* muscle and the insertion of the *adductor brevis muscle*, both of which may be implicated in repeated stresses [78, 79].

Most athletes with femoral shaft stress fractures are managed nonoperatively with good results [80, 81]. This option requires a variable period of rest with return to full sports activity between 12 and 18 weeks [74, 82]. One of the most frequently cited protocols for nonoperative management has been proposed by Arendt and Griffiths (Table 31.2), in which treatment is adapted according to the MRI grading of the injury [64, 81, 83]. Nonoperative treatment must consider the possible complication of displacement dictating the need for surgery.

Currently, when surgical treatment is required, the first option is intramedullary nail fixation [83]. There is no study assessing return to sports after surgical treatment of femoral stress fracture. On a cohort of military recruits, a mean of 3.5 months was required for bone union [84].

31.5 Tibia, Fibula, and Patella Stress Fractures

31.5.1 Shin Splint and Tibia Stress Fracture

The tibia is the second most frequent site for stress fractures among football players (Fig. 31.4) [19]. Stress fractures may occur at any location of the tibia. However, the tibial diaphysis is most commonly affected [1]. Tibial diaphyseal stress fractures may be divided in anterolateral (tension sided) and posteromedial (compression sided). Stress-sided anterior or anterolateral diaphyseal tibial stress fractures are considered high-risk fractures [85]. From an anatomical and biomechanical perspective, the tibia is a component of both the knee and the ankle joints. Therefore, changes in the knee or ankle joint biomechanics play a key role in loading of the tibia during activity. The tibia is bowed anterolaterally due to the powerful tensile stress of the gastrocnemius-soleus complex. In addition, the anterior surface of the tibia is poorly vascularized, increasing the risk for stress fracture [85].

Stress fractures of the tibia must be distinguished from medial tibial stress syndrome (MTSS), also known as "shin splints." "Shin splints" refers to periostitis of the posteromedial tibia that occurs due to the pulling stress of the gastrocnemius-soleus complex [86–88]. It has been proposed that "shin splints" might be an early stage of tibia stress fracture and that both entities are the same in different stages. However, there is no consensus on this statement, and both continue to be considered with their clinical and imaging differences [1].

The most important symptom of tibial stress fracture is pain. For both entities, pain develops insidiously and is aggravated by activity and

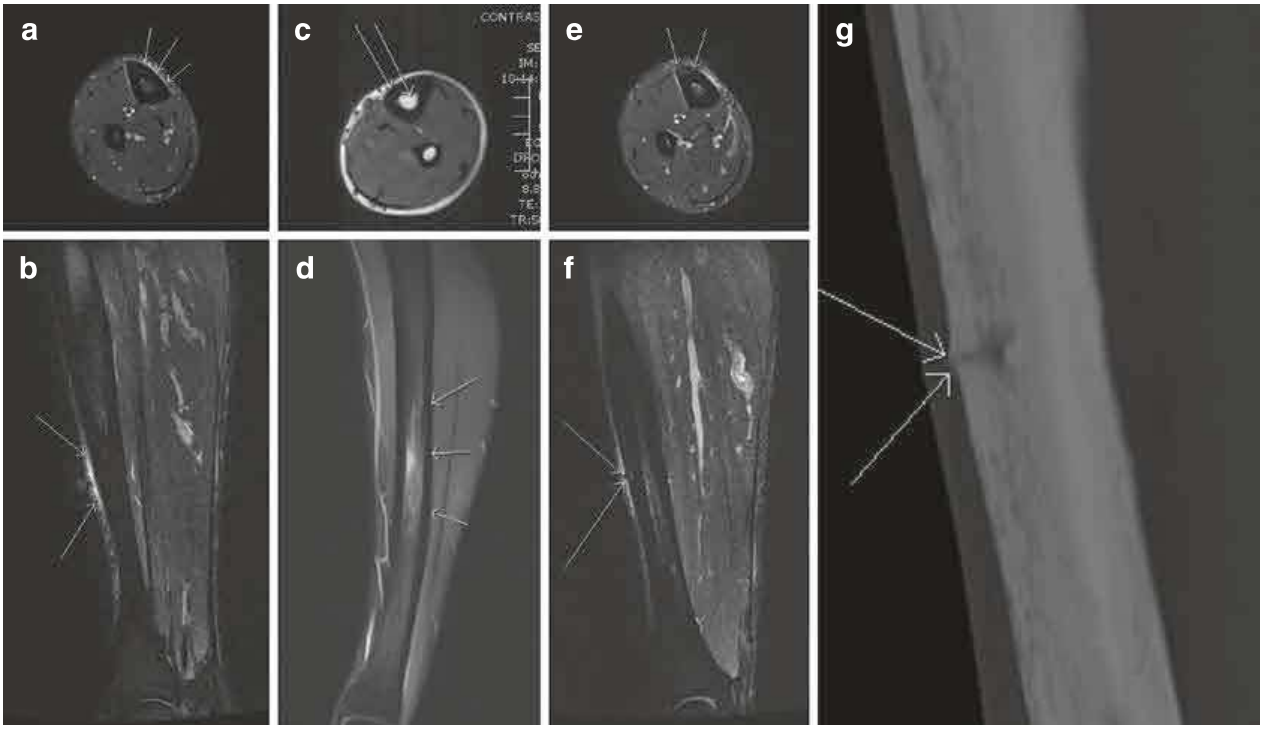

Fig. 31.4 **Fredericson grade I lesion**—Axial (**a**) and lateral (**b**) MRI views of the tibia with visible periosteal edema (*white arrows*), without bone marrow or cortical changes. **Fredericson grade 2 lesion**—without signal changes in T1 (two *white arrows*) (**c**) but bone marrow edema in STIR (three *white arrows*) without cortical changes (**d**). **Fredericson grade 4b lesion**—Axial (**e**) and sagittal views (**f**) with periosteal, bone marrow, and cortical changes (*white arrows*) including linear fracture which is also visible in CT image (*white arrows*) (**g**)

relieved by rest [25]. For tibial stress fracture, pain is located at the site of the stress fracture, and clinical examination reveals focal tenderness localized to the site of injury. Conversely, "shin splint" patients present with diffuse pain and tenderness along the posteromedial surface of the tibia [1]. One must emphasize that changes in training schedule or program as well as return to sports by people with physical deconditioning predispose to the onset of complaints.

A thorough search is required to identify any risk factors. *Pes planus* and *cavus*, tarsal coalition, muscle imbalance, or joint stiffness may alter the biomechanics of the ankle and predispose to "shin splint" and stress fractures [86–88]. Edema and palpable periosteal thickening are often observed in patients with tibial stress fractures while being usually absent from patients with "shin splint" [87]. The vibrating tuning fork test has been nearly abandoned since it is not reliable [89]. The single-leg hop test has also been commonly used in the evaluation of all lower extremity stress fractures; however, its sensitivity and specificity are very low [26]. It is critical to distinguish low-risk from high-risk fractures given their implication on the choice of treatment (surgical versus conservative) but also for prognosis and possible complications.

Stress-sided anterior or anterolateral diaphyseal tibial stress fractures are considered as high-risk fractures [85, 90]. These have higher risk for prolonged recovery, complete fracture, delayed union, nonunion, or chronic pain [53, 54].

However, most tibial stress fractures are posteromedial (compression sided), thus representing low-risk lesions that can be successfully treated nonoperatively [8]. Nonoperative management requires discontinuation of sports activities and avoidance of any activity that may load the tibia significantly until the patient can walk without pain. Non-weight bearing and rarely immobilization in a cast or brace may be needed if the athlete shows no improvement after 3–4 weeks [81]. Most rehabilitation protocols are divided into two stages [91–93]. The first stage is focused on rest and pain management. During this stage, athletes might use

deep water running and/or antigravity treadmill in order to keep cardiovascular conditioning while treating the bone in a protected environment. Some data suggest that this approach might lead to an earlier return to play [91–93].

The second stage is focused on the return to previous activity and competition [9]. The second stage includes correction of risk factors, muscle conditioning, and balance and proprioception training [46].

Resistance training incorporating repetitions with no loading or lower magnitude loading is used to improve muscle performance and bone recovery [46, 94]. The American College of Sports Medicine usually recommends resistance training 2–3 times per week [95]. There is general consensus that athletes should only return to sports after a minimum of 2 weeks free of symptoms [31]. On the other hand, the need for imaging control prior to return to play does not have consensus [96, 97]. For anterior tibial stress fractures, non-weight bearing for a period of 4–8 weeks is recommended, while for posteromedial fractures, a period up to 3 weeks is required [46, 96].

Using Fredericson's scale, it has been proposed that Grade 1 lesions might require 2–3 weeks before return to play, Grade 2 to Grade 4a injuries will take 6–7 weeks, and Grade 4b injuries will require a minimum of 9–10 weeks prior to return to sports [62, 98]. Low-risk fractures not responding to nonoperative treatment might require surgical treatment.

High-risk anterior fractures often require surgical treatment [99]. Intramedullary nailing is the preferred surgical method and is associated with high union rates, low rates of complications, and high return to sport [51, 96, 100, 101]. Approximately 10–12 weeks are required to return to sports activity after surgical treatment of tibial stress fractures [51, 96, 100, 101].

31.5.2 Fibula Stress Fractures

Fibula stress fractures are quite rare and can therefore be overlooked by the team physician (Fig. 31.5). From a biomechanical perspective, the load transmission to the fibula, with the ankle in neutral rotation, is only 6–7% of body weight [102]. The proportion of fibular stress fractures among runners on a recent systematic review was reported as 7–12% of all stress fractures [38]. However, in female and male long-distance runners, this proportion may be as high as 33% and 20% of stress fractures, respectively [103].

Most fibular stress fractures represent a simple injury that can be successfully treated with rest and activity modification in 6–12 weeks [104]. Stress fractures of the fibula are most common in its distal third and cause pain in the lateral distal third of the lower leg [38, 105]. Differential diagnosis of conditions that may cause pain in this location include *fibularis* muscle strain or tendinopathy particularly *fibularis brevis* and lateral ankle ligament sprain [105]. MRI or bone scan is usually required for diagnosis on an early stage. On rare occasions, dysfunction of the ankle syndesmosis may contribute to the development of a distal fibular stress fracture. This fact should be considered in the global assessment of pain along the fibula [104].

Proximal fibula stress fractures are very rare. The mechanism might be repetitive pressure of the proximal fibula, a consequence of repeated jumping with both knees completely flexed in a squatting position, causing a repeated strong pull of the muscles attached to the fibula (e.g., *soleus*, *peroneus longus*, *tibialis posterior*, and flexor *hallucis longus*) [104]. In long-distance runners, *biceps femoris* contraction forces have been implicated [38]. Among football players, its description has been associated with running and jumping [106]. Cessation of activity results in complete healing and strengthening, and flexibility exercises of ankle dorsiflexors, ankle plantar flexors, peroneals, and hamstrings are helpful in recovery and avoid recurrence [104].

31.5.3 Patella Stress Fractures

Although patella stress fractures have been increasingly recognized, the patella remains a

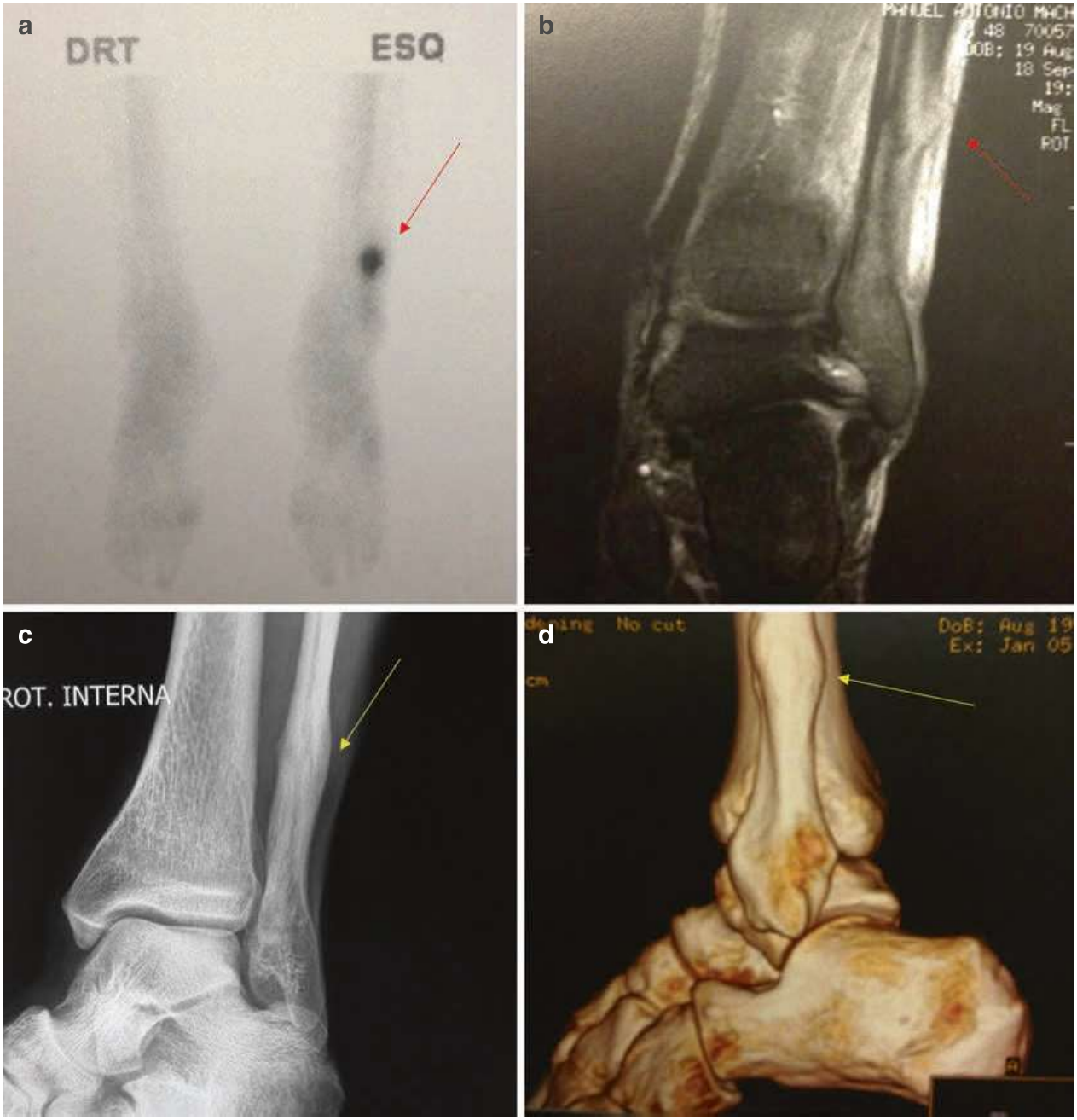

Fig. 31.5 Bone scan showing increased activity in the painful fibula site (*red arrow*) on the early stage of the condition (**a**); MRI view confirming periosteal edema, cortical and bone marrow changes (*red arrow*) (**b**); X-ray after 3 months confirming fracture healing and callus formation (*yellow arrow*) (**c**) which is also visible on CT (**d**) with exuberant bone callus (*yellow arrow*)

rare site for stress fractures [107]. The published data are comprised of individual case reports and small case series. The patella is a sesamoid bone lying within the extensor mechanism of the patellar tendon, linking the quadriceps to the tibia and functions under a quite demanding biomechanical environment of the patellofemoral joint. The force knee vector acting in it reaches more than three times the body weight going up and down the stairs and up to eight times the body weight during deep knee flexion [108].

In order to identify and differentiate a patella stress fracture from the numerous causes of

anterior knee pain, a high index of suspicion is necessary. The anterior (tension side) stress fracture of the patella is considered a high-risk lesion and might require surgical treatment, while compression side fractures will respond better to rest and nonoperative treatment [1].

Patients will present with an atypical history of anterior knee pain. Often, there will have been a recent change inactivity levels or a change in training program or training load, applying higher stresses or shorter periods of rest between exercises. Most cases describe a sudden onset of pain during activity, often associated with a pop or crack, but this is typically preceded by weeks or months of anterior knee pain. Transverse fractures are more frequent considering the axial loading applied, although longitudinal fractures may also occur. The most common injury site is at the junction of the middle and distal thirds of the patella where distal quadriceps and proximal patellar tendon fibers merge and insert.

Plain radiographs may demonstrate an obvious fracture, either non-displaced or displaced, although the features of chronic stress, such as sclerotic fracture margins (Fig. 31.6) or cystic changes, may be noticed. Bone scan and MRI will help to detect the early stages of this condition, and CT scan can help better define it once identified.

Concerning treatment, most often when there is a positive bone scan but X-rays are normal, patients can be managed conservatively by a period of rest. If X-rays show a non-displaced fracture, it is possible to choose conservative treatment by immobilization in extension (cast or brace) 4–6 weeks with partial weight bearing followed by passive range of motion exercises, quadriceps strengthening, and progressive return to activity within a period of 3 months. If a displaced fracture is present, surgical treatment is required by tension band wiring or compression screws. It is important, if sclerotic fracture margins are present, to debride and curettage these margins to create healthy, vascularized surfaces for bone healing. The postoperative protocol will depend on the achieved fixation and bone quality.

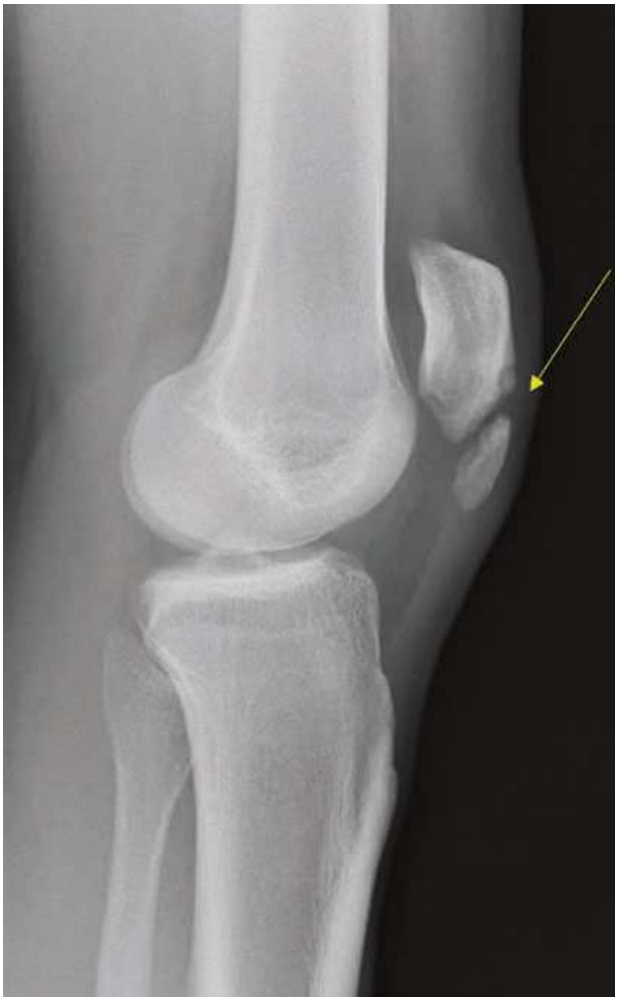

Fig. 31.6 Radiography of lateral view of late stage patella stress fracture (*yellow arrow*). Notice the sclerotic border of fracture line confirming the slowly developing process

31.6 Recent Therapeutic Options

With recent therapeutic biotechnical advances, several new modalities (both biological and physical agents based) are being developed in order to accelerate the healing process and return to play. These are used in combination with general fracture management principles but aim to achieve the maximal benefits of biological and physical stimulation methods [109]. However, many of these options remain experimental and lack evidence-based support.

31.6.1 Hyperbaric Oxygen Therapy (HBOT)

This method consists of intermittently administering 100% oxygen at pressures greater than one atmosphere absolute (ATA) in a pressure vessel. It has been attempted as therapy for several conditions. However, despite some basic-science support as an effective way to stimulate the osteoblasts [110], there is still no clinical evidence on its effectiveness in promoting bone healing [111]. Its use remains controversial and somewhat experimental.

31.6.2 Bisphosphonates

Bisphosphonates suppress bone reabsorption by osteoclasts. By this mechanism, bisphosphonates might prevent bone loss during the initial remodeling phase following high bone stresses and facilitate bone recovery. A small series in collegiate athletes suggested a positive effect of intravenous pamidronate [112]. There is no further evidence of its benefit. Considering the costs and potential adverse effects of bisphosphonates, its use cannot be widely indicated for the treatment of stress fractures in athletes, and prudency is advised [113, 114]. Moreover, their prophylactic effect has also not been demonstrated for bone stress injuries [113, 114].

31.6.3 Growth Factors

The use of growth factors and preparations rich in growth factors (PRGF) has become increasingly popular, particularly in the sports population [115, 116]. These include the growth factors that are produced by platelets in a number of forms of application. Besides remaining controversial in different tissues, with contradictory results found in literature, there is even less evidence concerning its use in stress fractures given the paucity of studies. It has been stated that autologous preparations rich in growth factors might enhance the healing of hypertrophic nonunions when applied during internal fixation surgery and also enhance healing by injection application on stable nonunions [117]. However, more definitive knowledge is required before supporting its widespread use to treat these conditions.

31.6.4 Bone Morphogenetic Proteins (BMPs)

These proteins belong to a family of growth factors (TGF-beta superfamily) that are known to have osteoinductive properties and have been used to promote bone healing [118]. These have been demonstrated to be useful during surgical approaches of fractures and cases of nonunion [119]. Ongoing work is aiming for its clinical percutaneous application, which might be helpful in some stress fracture conditions [118, 119]. Cost-effectiveness must also be taken into account, but this is a promising approach.

31.6.5 Recombinant Parathyroid Hormone

Parathyroid hormone (PTH) increases serum calcium levels by enhancing gastrointestinal calcium absorption, increases renal calcium and phosphate absorption, and releases calcium from the skeleton when required. Although with regular administration of PTH promotes osteoclast activity, intermittent exposure to PTH can also stimulate osteoblasts and results in increased bone formation.

Some studies have shown positive effect in bone healing [120]. Systemic intermittent PTH treatment can enhance either endochondral or intramembranous bone repair [121]. Once more, there is limited knowledge specifically in stress fractures, but this is also a promising and interesting area for future developments.

31.6.6 Low-Intensity Pulsatile Ultrasound Therapy

High-frequency sound waves that are above the audible capacity of humans can influence the bone and the surrounding soft tissues by creating microstress and tension that are capable of stimulate healing. Despite the method being effective in accelerating acute fracture consolidation [122], the exact mechanism remains unclear but is ostensibly related to increased synthesis of extracellular matrix proteins [123]. There remains a paucity of available data concerning ultrasound and stress fractures. A meta-analysis of the effect of low-intensity pulsed ultrasound on the healing of all types of fractures found conflicting results and concluded that most studies had relevant methodological limitations [124].

31.6.7 Magnetic Field Application

Electric fields are recognized to promote bone healing in vitro given the fact they induce cellular stimulation and protein synthesis [123]. There are two main methods to consider: capacity-coupled electrical field (CCEF) devices or pulsed electromagnetic field (PEMF) stimulation [123]. CCEF requires operative placement of an electrode in the fracture site. PEMF promotes release of calcium stored inside the cells, while CCEF uses the calcium ions present in the extracellular fluid. CCEF has been shown to result in higher DNA increase in bone tissue [123]. From a clinical perspective, there are few studies evaluating these methods [125–127]. Further research is needed.

31.7 Return to Sports After Stress Fractures

Fact Box 3

1. A stress fracture should always be considered in an athlete who presents insidious pain, referred to a bony structure, which typically increases after effort and diminishes on rest.
2. Radiographies alone are not feasible (very low sensitivity) for the diagnosis of stress fractures; diagnosis often requires MRI and/or a bone scan.
3. It is mandatory to investigate changes in the training schedule and/or playing surface in footballers.
4. Concerning women athletes, always search history of menstrual disorders, eating disturbances, and weight loss.
5. General rule: return to play (training) is allowed after 2–3 weeks free of symptoms.
6. There is currently no consensus on imaging criteria prior to return to play.

Stress fractures can result in prolonged absence from football, particularly the high-risk variety. The time taken from diagnosis to full recovery and return to play depends on multiple factors: the injury site, sports activity, injury type, and severity and possibility of correcting intrinsic and extrinsic risk factors [1, 81, 83].

Low-risk stress fractures and those manageable by conservative treatment usually make possible for the patient to return to their previous activities 4–17 weeks after the injury [128]. Ekstrand and Torstveit reported that the mean absence from football was 3 months for stress fractures of the tibia and 4–5 months for pelvic stress fractures [19]. However, tibial stress fractures, according to its classification, might range from 2 to 12 weeks prior to return to play [62, 98]. If surgery is required to treat stress fractures, it will typically take at least 3 months after surgery to resume sports activity [51, 96, 100, 101]. Femoral stress fractures usually require 12–18 weeks before full return to sports [74, 82].

The criteria that might be used to allow an athlete to return to play include absence of pain at the affected site during sports activity, absence of symptoms during provocative tests, and absence of abnormalities in imaging examinations. It is of paramount relevance that the athlete, the coach, the manager, and the technical team understand the risk factors and conditions that led to the injury. This way, necessary steps can take place in order to mitigate these risk factors and prevent

recurrence and reappearance of injuries [1, 83]. The gradual 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 [81]. Imaging information confirming complete healing might also be considered as previously discussed.

Fact Box 4

General criteria for athlete's return to play after "stress fractures":
No pain at the injured site during sports activity
No symptoms during provocative tests
Progressive return to sports activity after the athlete is without complaints for 10–14 days, with 10% increases in training intensity per week
No evidence of imaging abnormalities

Take-Home Message

Despite being infrequent conditions, a stress fracture should always be considered in an athlete who presents with insidious pain, referred to a bony structure, which typically increases after effort and diminishes on rest. The tibia is the second most frequent bone affected by stress fracture in football, followed by the pelvis. Understanding the biomechanical feature of tension and compression sites helps establishing higher-risk lesions.

Radiographies have very low sensitivity for the diagnosis of stress fractures; thus, MRI and/or a bone scan is often required for early detection.

In the athletic population, it is mandatory to investigate changes in the training schedule and/or playing surface. Female athletes have specific risk factors that need to be considered. Identifying biologic contributing factors, such as nutritional or hormonal deficiencies, is an important part of management.

During the healing phase, the athlete should focus on conventional methods of relative rest, analgesia, and rehabilitation. Although surgical stabilization involves iatrogenic trauma to the area, the pain related to surgery may well force the athlete to rest, thus promoting healing and recovery.

As a general rule, return to play (training) should be allowed only after the athlete remains 2–3 weeks free of symptoms. There is currently no consensus on imaging criteria prior to return to play; however, some classifications have proven useful and the team physician must be aware of this to assure safe return to play for footballers.

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