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Bone Stress Injuries

Knöcherne Stressverletzungen

CLINICAL REVIEW

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Summary

› **Bone stress injuries,** commonly referred to as stress reactions or stress fractures, are caused by overuse and are prevalent conditions in athletes. These injuries are often seen in sports with high cumulative skeletal loading such as running, athletics, gymnastics, basketball, cricket, or soccer. The main injury pathophysiology is an imbalance between bone microdamage formation and its removal and replacement. A detailed medical history and clinical examination are important for diagnosis, with magnetic resonance imaging representing the gold standard for radiographic confirmation. Treatment and return-to-sport decisions should be individualized and based on injury location, severity, aspects of sports participation, and patient preferences. Initial management includes activity modification, protected weight-bearing, immobilization, optimizing nutrition (caloric intake, dietary calcium, and vitamin D supplementation), and in a few cases surgery. Following initial treatment, progressive loading and return-to-activity are initiated. Overall, a high rate of return-to-sports can be expected. Numerous risk factors that alter bone loading or bone health have been described and their identification is a crucial step towards prevention of future injury.

KEY WORDS:
Stress Reaction, Stress Fracture, Bone Overuse

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Introduction

Bone stress injuries are overuse injuries that develop in the context of repetitive loading due to cumulative bone damage (4). These injuries are commonly seen in athletes and often result in absence from sports participation (7). A delay in diagnosis or inadequate treatment may lead to severe consequences including complete bone fracture, displacement, non-union, immobility, or avascular necrosis (4, 5).

Bone stress injuries were first described in the 19th century in military personnel after prolonged activity causing foot pain (1). However, little attention has been paid to these types of injury, which may be explained by a low incidence in the general population (4). Nonetheless, bone stress injuries may account for up to 20% of injuries in specific populations (e.g., runners), thus leading to substantial morbidity in athletes (4, 7, 15).

Recently, the terminology of bone stress injuries has been further specified (4, 16). Accordingly, a bone stress injury comprises a spectrum of stress-related injuries including early stress reactions to advanced injuries with the presence of a fracture line, referred to as stress fractures. Although impaired load resistance and altered bone biology have been identified as important risk factors, bone stress injuries are caused by excessive loads applied to a generally normal bone. In contrast, bone insufficiency injuries are caused by physiological loads in the context of abnormal bone biology (16).

In this clinical review, we provide an overview of the epidemiology, pathophysiology, risk factors, prevention, diagnosis, and management of bone stress injuries. By using a narrative synthesis method with emphasis to latest references, this review aims to provide an up-to-date overview. Particular attention ›

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Table 1

Low-risk and high-risk classification of bone stress injuries. Injuries to high-risk bone sites are associated with an increased risk of treatment complications such as delayed union or non-union.

LOW-RISK	HIGH-RISK
Humerus	Lumbar vertebral arch
Hand	Femoral neck
Rib	Patella
Sacrum	Anterior tibia
Pubic bone	Medial malleolus
Femoral shaft	2nd metatarsal base
Posteromedial tibia	5th metatarsal base
Fibula	Great toe sesamoids
1st – 4th metatarsal shaft	
Calcaneus	

is given to clinical aspects with the aim to provide a framework for a better understanding of bone stress injuries and to enhance patient care in the field of sports medicine.

Epidemiology

Bone stress injuries are common injuries in the sports context. The injury rate varies between the sports populations, sex, age, and ethnicity. In selected athletic populations, up to one out of five injuries can be expected to be a bone stress injury (4). Athletes participating in sports with high repetitive loading are especially prone to these injuries (4). In high school and collegiate athletes, the injury rate of bone stress injuries was reported to be 1.5 and 5.7 per 100,000 athlete-exposures, respectively, but varied widely depending on the primary sport practiced (2, 11). Sport activities with the highest rate of bone stress injuries were cross-country running, female gymnastics, and athletics (2, 11). Furthermore, women are reported to have greater rates of bone stress injuries than men across different sports and populations (9). Bone stress injuries most commonly occur in the lower back (spondylolysis) and the lower extremities (e.g., tibia and metatarsals), but can also be located at other injury sites (e.g., ribs, olecranon) dependent on the sport-specific loading (4).

Pathophysiology

The development of a bone stress injury is generally attributed to an imbalance between the formation of load-induced localized skeletal microdamage and its removal and replacement (4). However, the underlying mechanism of injury remains incompletely understood at present. Bone matrix predominantly consists of collagen and hydroxyapatite, and bone is generally structured as compact (cortical) and cancellous (trabecular) bone. In theory, the development of bone stress injuries follows a continuum. With repetitive bone loading, microdamage occurs in form of linear microcracks, diffuse damage, or trabecular microfractures. Various skeletal adaptation mechanisms have been described to avoid bone failure. For instance, specific arrangement on collagen fibers serves as a natural resistance to the formation and propagation of microdamage. When microdamage accumulates with elevated stress and strain, repair mechanisms are initiated through targeted bone remodeling. Specifically, apoptosis of matrix-embedded osteocytes leads to activation of bone-resorbing osteoclasts via various signaling pathways (e.g., production of Receptor Activator of NF-κB Ligand) to eliminate microdamage. If bone remodeling cannot exert repair mechanisms in a timely manner, a bone stress injury occurs. The initial failure of toughening mechanisms allows the initiation of microcracks, which may be followed by crack growth or coalescence. The consequence is the clinical manifestation of a bone stress injury or a complete fracture. New insights into bone-specific risk factors that reduce the athlete-individual bone loading capacity are needed to broaden the understanding of bone stress injuries.

Risk Factors and Prevention

Factors contributing to the development of bone stress injuries include both increased bone loading and impaired skeletal health. Overuse is considered the most important factor in athletes as bone stress injuries, by definition (4), are typically seen in the context of high physical demands. The total amount of training volume or intensity is less important but rather a sudden change in training (3). In particular, the presence of a bone stress injury must be considered when athletes increase their sports activities or return from recent breaks due to other injuries, illnesses, or off-season (4).

From a biomechanical standpoint, movement patterns including both kinetic and kinematic variables are thought to play a major role in the pathophysiology of overuse injuries (8). Specific parameters have been related to bone stress such as foot strike pattern, foot morphology, and limb-length discrepancy (4, 6). However, evidence is far from conclusive, and it should be noted that biomechanical characteristics have been suggested to contribute to injury at some bone sites but may not be responsible for an increased risk in general (6). Further risk factors that may alter bone stress and strain include running surfaces, shoes, and inserts (4, 14).

Growing evidence suggests that bone stress injuries are more likely to occur in energy-depleted athletes as caused by low dietary intake and high energy expenditure (4). The result of a low energy availability state may include menstrual dysfunction, male hypogonadism, and reduced bone mass from the syndrome Relative Energy Deficiency in Sports (RED-S) defined by the International Olympic Committee (10, 13). In addition to ensuring adequate energy availability from increased caloric intake and reduced exercise energy expenditure, dietary recommendations also include sufficient dietary calcium intake (1,000-1,500 mg per day) and vitamin D supplementation (if serum 25-OH-D >30 µg/L) as low levels are potentially associated with an increased risk of bone stress injuries (4).

Other risk factors have been identified, including impaired bone microarchitecture, psychological factors, sleep disorders, early sport specialization, genetics, and medications (4, 12). By identifying these factors, clinicians may better understand the multifactorial origins of bone stress injuries. Screening for bone stress injuries in clinical practice is difficult but identification of (modifiable) risk factors is a crucial step towards rehabilitation and injury prevention for the injured athlete (4).

Diagnosis

Bone stress injuries are pathologies that should always be considered as a differential diagnosis in athletes. However, clinicians should be particularly sensitive to athletes participating in "at risk" activities with pain at typical injury sites (e.g., tibia in runners, fifth metatarsal in basketball players, vertebral arch in adolescent throwing athletes). In general, diagnosis of bone

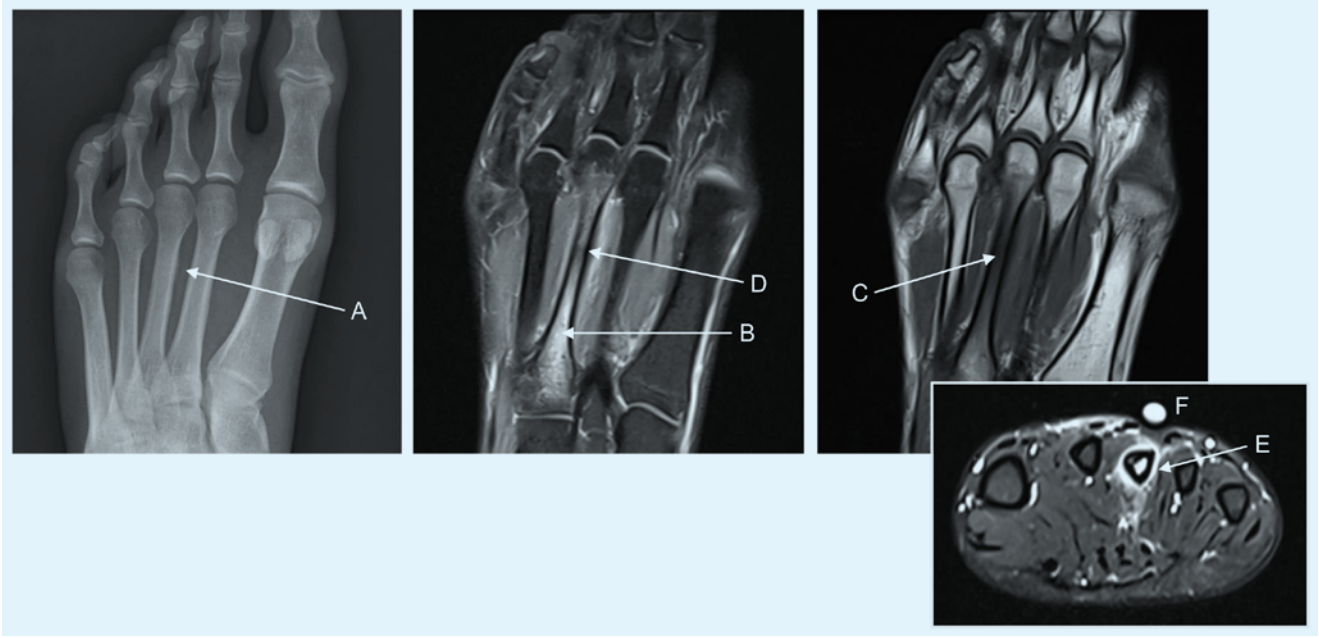

Figure 2

Imaging findings of a third metatarsal bone stress injury. Bone stress injuries are often occult in plain radiography but indirect signs such as callus formation (A) can be indicative of a bone stress injury. Long axis magnetic resonance imaging reveals a diffuse edema (B) in fat-suppressed proton-density imaging with corresponding hypointense signal (C) on T1 imaging. A cortical signal or hypointense fracture line may be present (D). Axial imaging typically shows adjacent soft tissue inflammation (E). Skin marker placement (F) can aid in the diagnosis if placed in the area of maximum pain.

stress injuries is based on taking a detailed history and performing a clinical examination. Attention needs to be given to athletes with a recent change in training volume or intensity, a history of prior bone stress injuries, and indicators of RED-S (4, 13). There is often local bone tenderness on palpation and possibly pain on percussion. Swelling may be present. The hop test may provoke pain. Further tests have been described for certain locations of injury (e.g., fulcrum test for femoral shaft, squeeze test for calcaneus). Typically, pain does not "warm-up" and is described as persistent or worse with continued activity. Imaging is used for differential diagnosis, confirmation or grading of injury, respectively (4). Although plain radiographs (X-rays) are among the first examinations, they have a low sensitivity as they are often inconspicuous. Pathological findings on plain radiographs may only be seen in more severe injuries with a present fracture line or after substantial bone stress injury healing (e.g., callus formation). However, early identification of a bone stress injury is important as a delay in diagnosis can result in catastrophic consequences including fracture displacement or non-union with continued activity (4). Therefore, magnetic resonance imaging (MRI) is the recommended imaging modality in many cases due to its high sensitivity and specificity (figure 1). In particular, fat-suppressed (T2) sequences can reveal bone edema. Adjacent periosteal or soft tissue reactions may be present, and the occurrence of a (hypointense) fracture defines a high-grade injury (that is, stress fracture). In addition, MRI may be used to grade injuries and guide return-to-sports (7).

Management

Individualized treatment strategies should guide management of bone stress injuries. Bone site and injury severity are two important factors when guiding treatment; with pain, level of performance, patient preference, patient compliance and adherence, prior injuries, age, hormonal status, and bone properties being other important factors to consider (4, 5, 7). Practically, initial treatment of bone stress injuries is guided by injury site. Thereto, bone stress injuries are commonly classified as either low- or high-risk (Table 1). Low-risk injuries typically heal without complications and examples are the posteromedial tibial shaft, fibula, pelvic ring, and the first to fourth metatarsal shaft (5). For many low-risk bone stress injuries, activity modification leads to predictable healing and high return-to-activity rates (5). Protected weight-bearing is recommended if pain is provoked by ambulation. In contrast, high-risk bone stress injuries require a much more cautious treatment strategy to reduce risk for non-union, among others (5). High-risk injuries include the femoral neck, anterior tibial shaft, medial malleolus, navicular bone, talus, second and fifth metatarsal base. Activity reduction, immobilization, and protected weight-bearing are typically necessary. Surgical management may be indicated to reduce the risk of complete bone fracture, and non-union, and to allow for early weight-bearing (5). Clinicians with less experience in treating high-risk bone stress injuries are advised to consult physicians with expertise in treating bone stress injuries (4). For both low- and high-risk injuries, optimizing nutrition (increased caloric intake and dietary calcium along with vitamin D supplementation), shockwave therapy, and bone-specific medications are sometimes used by clinicians, but their in-depth discussion is beyond the scope of this review (4). In any case, identification and addressing of risk factors is a key measure to accelerate healing and prevent re-injury.

Bone stress injuries typically result in considerable absence from sports with full return-to-sports usually seen between five and 20 weeks depending on image grade and anatomical location (5, 7). Accordingly, despite its high rate of return-to-prein-jury-level of activity, bone stress injuries are dreaded by both athletes and coaches. Most return-to-sports protocols are based on the gradual introduction of bone loading. An example protocol adapted from Warden et al. (15) on return-to-sports for runners is presented in figure 2. At all rehabilitation stages, patients should be pain-free; and any recurrent pain should ›

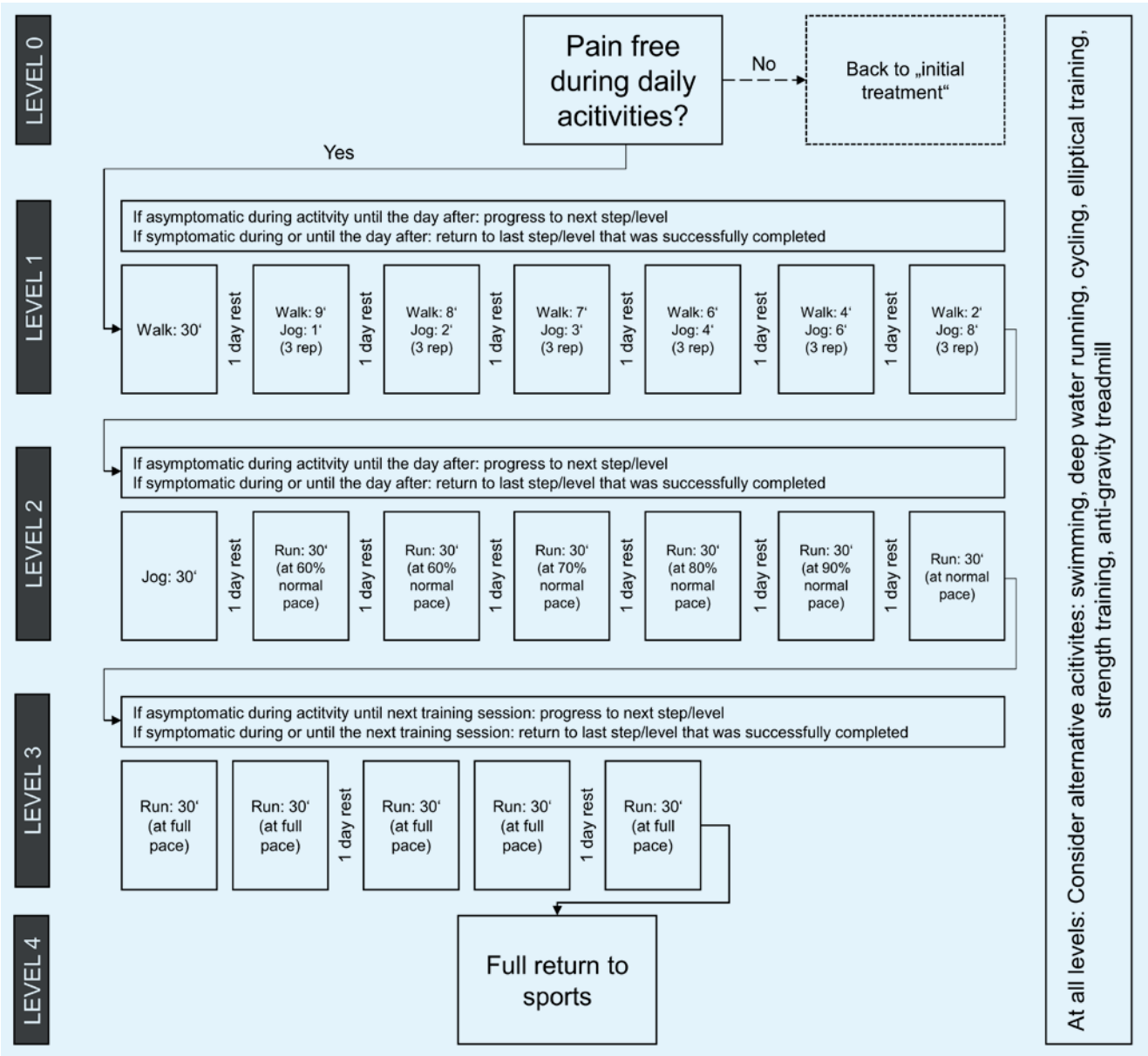

Figure 2

Exemplary return to running protocol after bone stress injuries. Adapted from Warden et al. (15). Minutes are indicated with an apostrophe (‘). Rep=Repetition.

result in a reduction of loading with at least one additional day of rest (5). Low-impact activities (e.g., anti-gravity treadmill, deep water running, cycling, strength training) offer good opportunities to maintain fitness (4).

Conclusion

Bone stress injuries comprise distinct clinical entities in the context of repetitive skeletal loading. A combination of thorough medical history, physical examination and magnetic resonance imaging is the key to diagnosis in the majority of cases. Management of bone stress injuries is guided by injury site and grade of injury, among others. Modification of activity, protected weight-bearing, temporary immobilization, and physical therapy are standard first-line treatment options. Underlying causes should be investigated in order to accelerate healing and to prevent re-injury. Following initial treatment, a rehabilitation program should be designed by a multidisciplinary team to safely guide return-to-sports. While there is a risk of delayed or non-union, the rate of athletes returning to their previous activity level is usually high.

Conflict of Interest

The authors have no conflict of interest.

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