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Review

Current concepts of shockwave therapy in stress fractures

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HIGHLIGHTS

- Extracorporeal shockwave treatments (ESWT) stimulate bone turnover and neovascularization in delayed unions and avascular necrosis.
- ESWT is a safe and effective non-invasive outpatient procedure.
- Medium and high energy focused ESWT has shown excellent results in treating stress fractures, with faster return to competition and athletic activity.

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ABSTRACT

Stress fractures are common painful conditions in athletes, usually associated to biomechanical overloads. Low risk stress fractures usually respond well to conservative treatments, but up to one third of the athletes may not respond, and evolve into high-risk stress fractures. Surgical stabilization may be the final treatment, but it is a highly invasive procedure with known complications. Shockwave treatments (ESWT), based upon the stimulation of bone turnover, osteoblast stimulation and neovascularization by mechanotransduction, have been successfully used to treat delayed unions and avascular necrosis. Since 1999 it has also been proposed in the treatment of stress fractures with excellent results and no complications. We have used focused shockwave treatments in professional athletes and military personnel with a high rate of recovery, return to competition and pain control. We present the current concepts of shockwave treatments for stress fractures, and recommend it as the primary standard of care in low risk patients with poor response to conventional treatments.

1. Introduction

Bone is a very specialized dynamic organ that forms the primary structural element of the human body. It is the solid base of the muscle-joint-bone complex and has the unique characteristic, as an engineering material, of changing form, geometry and physical properties according to mechanical demands. This process has been referred to as mechanical homeostasis, a complex biological response to physical loads that rule not only fracture healing but also bone geometry and even the evolution of species.

Bone remodeling is an essential biological process as old as the bone itself [47]. The fossil records show that the skeletons of the earliest weight bearing vertebrates contained osteonal structures and other evidence of coordinated bone resorption and formation. This process has been essential for a healthy functional skeleton for millions of years. Probably the genes that enable bone remodeling have been continued to be selected because they confer important survival advantages.

The function of bone remodeling has been debated for centuries. The vision of the anatomists and histologist of the 19th century was that the Osteon structure is a nearly perfect mechanical and biological complex and it definitely serves a mechanical function. When the discovery that calcium serum levels must be regulated to prevent muscle tetany the story changed, and the metabolic significance of calcium brought the concept of bone remodeling as a metabolic process. Both visions are correct, meaning the importance of the process of bone remodeling nowadays. But the system is not perfect, because it fails in certain conditions. Microdamage is a biological form of fatigue, creep or other accumulative mechanical processes by which the microstructure of a loaded material is

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Stress fractures are similar to fatigue failures in engineering materials in a sense that are due to a relatively large number of repetitions, which if applied once, do not cause failure. Microdamage ranges from microcracks to diffuse damage, cross-hatching and finally microfractures, depending on the amount of energy, the accumulative fatigue stress, and the mechanical resistance of bone as a material.

The importance of bone remodeling must be emphasized, because microdamage is increased by fatigue loading at physiological strains. Such damage is a normal consequence of skeletal function, thus, microdamage can only be removed from bone by means of resorption and replacement of bone tissue through remodeling [68]. This process is done by the activation of Bone Multicellular units, both randomly to control calcium homeostasis, and targeted to control specific microdamage areas [14,65,66].

This means that if we inhibit the remodeling process we could increase the risk for microdamage. On the other side, if repetitive cyclic loading of bone is excessive, then Fatigue overuse on bone that accumulates more stress than BMU activation will cause microdamage.

The way we study bone as a material is applying known loads to specific areas and recording the mechanical behavior of the material. The elasticity modulus is determined, as well as the yield point and the ultimate failure point. To study fracture risk we simply go beyond the failure point and that way we can determine variations between specimens. For Stress fractures we need to repetitively load the bone with energies lower than the yield point. This energy applied once has no significance, but the accumulative loads will fracture the bone in certain areas.

If we compare the amount of stress needed to fracture a bone, it is clear that a great amount of stress is required to start a bone fracture and very little to finish the job. However, deformation is almost the same, which means that the material becomes more plastic after the initial failure point. The energy required to initiate the fracture is represented in the stress–strain graph by the areas under the curve.

These has been deeply studied by Keaveny and Hayes [45,46], and stress fractures are in the range of the pre yield or linear region, with small amounts of energy loaded in cycles that cause microfractures as a response of misbalanced bone remodeling.

Bone is especially weak under transverse and tensional loads. This means that insertional, or traction forces applied in cycles to bone could result in higher fracture risk. Also tensional areas of normal bone loaded repetitively could represent a higher fracture risk.

Taylor [82] proved how the medial tibia is one of the most vulnerable areas in the skeleton, as a result of the tibial shaft curves, the asymmetric mechanical loads in normal gait and exercise, and it's poor vascularity. He proposed a mathematical model that gives a guide for prediction of stress fractures under certain exercise conditions, time and age.

Repetitive cyclic loading of bones is the most relevant etiologic factor in the genesis of stress fractures. The fine balance between Bone Microdamage & Remodeling marks the outcome of bone failure under repetitive loading conditions [18]. The three possible scenarios for bone failure under fatigue loading are: normal bone & abnormal loading – normal loading & abnormal bone and abnormal loading on abnormal bone. The most common bones affected are tibia, metatarsals, fibula, navicular, pelvis and femur [10,59].

2. Diagnosis and classification

The first cases of stress fractures were described by Breithaupt in Prussian Soldiers with leg pain in the war of 1850 [28]. The global incidence ranges from 1% to 20% depending on the physical activities of the patients [28]. They usually appear as a progressive localized bone pain after physical activity or sports [43]. Symptoms usually disappear with rest and have short recovery periods. The ethiology of stress fractures is a biomechanical misbalance of loads that result in a progressive breakage of the gait kinetic chain [63]. This is very relevant in athletes and military personnel that repetitively overload under-trained skeletons and cause unbalanced bone remodeling resulting in bone failure [43]. Clinical diagnosis is relatively easy with physical examination that shows pain at a pin pressure point that may or not be associated to swelling. There is pain when eccentric loads are applied to the muscles inserted on the affected bone, and specific tests have been described for stress fractures such as the hyperextension, the fulcrum or the hop tests [95].

Stress fractures are classified upon the risk of a complete bone failure, as low, medium or high risk [13,16,64]. Frederickson [29,30] described an image-based classification using both X rays and MRI, associating recovery time with four stages of bone damage. It is especially valuable to determine prognosis. Low risk stress fractures usually respond to conservative treatments, while high risk fractures usually require surgical procedures in order to prevent a complete fracture. Up to one third of low risk stress fractures may not respond to conventional treatments and continue with pain during exercise [30,50,77]. They may evolve into high-risk stress fractures if load conditions and bone turnover is not balanced. It is a primary goal of the sports medicine and orthopedic specialists to prevent the progression of a low risk stress fracture.

Diagnostic images are mandatory in order to determine staging [64]. The first reports of a radiological classification of stress fractures was done by Savoca [76] in 1971, and he correlated clinical symptoms with early metaphyseal sclerosis, periosteal reaction or partial fractures. Magnetic resonance images are the best tool to determine bone marrow edema, periosteal reaction and soft tissue damages in all stages of stress fractures [22]. Bone scans are very sensitive to determine increased bone turnover areas in early stages, but it is not very specific as many other situations may mark as false positive, and is an invasive procedure with potential risks [52,71]. (Fig 1). However, in early stages it is the most specific and sensitive test available, as radiographic findings only appear after three weeks of the initial microfracture [27,79].

3. Treatment

Treatment of stress fractures is based on a mechanical and a

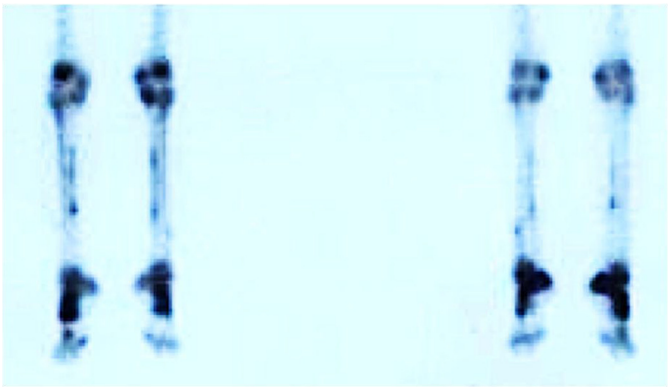

Fig. 1. Bone scans of tibial stress fractures in a high performance athlete. This is the most specific and sensitive test for stress fracture diagnosis. X Rays do not show early changes. Bone scans may remain positive after the patient has recovered from treatments and is painful.