Early identification of stress fractures

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Introduction
Stress fractures occur when normal bone is exposed to abnormal stress. They are seen in professional athletes and in military personnel but, with increasing health awareness, are becoming more common in the general population who are increasing their physical activity. Stress fractures can occur in all age groups and are thought to account for about 10% of all sports injuries. Early diagnosis is vital to the signs of a stress reaction, allow healing and to avoid progression to a frank fracture.
Insufficiency fractures are fractures which occur in abnormal bone when exposed to a normal stress. They most commonly occur secondary to untreated osteoporosis.

Clinical features
The clinical history is important in the diagnosis of a stress fracture. The patients should be asked about the sequence of events leading to the onset of pain and patients with a non-athletic habitus should be questioned about any new physical activity.

The classic clinical feature of a stress fracture is pain associated with a particular activity. The pain is relieved by rest and exacerbated when activity is continued. Often, the activity will be new to the person. Examination findings include localised tenderness, warmth and swelling.

Treatment is predominantly medical by optimising nutrition, excluding endocrine abnormalities and, most crucially, modifying activities to allow the stressed bone time to rest. Most fractures will heal in 6-8 weeks if the repair process has dominated over resorption, although certain sites may take longer.

Pathophysiology
Fatigue fractures
Fatigue fractures result from the application of abnormal muscular stress or torque to a bone that has normal elastic resistance. The following triad is associated with most fatigue fractures:
1. The activity is new or different for the person;
2. The activity is strenuous;
3. The activity is repeated with a frequency that ultimately produces signs and symptoms.
Stress-related bone injuries were first described in 1855 by Briethaupt who recorded the painful swollen feet of marching soldiers. In 1987, this condition was shown to be due to a fractured metatarsal shaft and subsequently termed a ‘march fracture’. Risk factors implicated in these injuries can be intrinsic or extrinsic and are listed in Table 1.
Bone is a dynamic architectural tissue that responds to changes in the muscular activity imposed upon it. Bone requires stress for normal development and constantly remodels itself in response to the changing distribution and quantity of stresses applied through it. As a result, bone optimally adapts to its new mechanical environment (Wolf's law of transformation). When bone is exposed to persistent overuse to which it is unaccustomed, such forces result in microscopic trabecular fracturing. This in turn stimulates local osteoclast-mediated periosteal resorption, which is characterised by local oedema and hyperaemia and peaks at about three weeks. Simultaneously, but at a slower pace, stronger lamella bone replaces the resorbed bone and is laid down along the lines of stress, a process which takes at least 12 weeks. The imbalance results in a transient weakening of the bone cortex which, if rested, will heal, but if further stresses are applied, this weakening will progress to a frank fracture.
A stress response refers to the pre-failure events occurring at a cellular level which results in structural bone weakening and maladaptive bone remodelling. A stress fracture is the final consequence of the preceding events.
Insufficiency fractures
An insufficiency fracture results when normal or physiological muscular activity stresses a bone that is deficient in mineral or elastic resistance.
Bone mineral density, composition and structure are related to bone elasticity and stiffness which together determine bone strength. Any process that affects these parameters could change bone resistance which could result in fracture formation. Conditions predisposing to the development of insufficiency fractures are listed in Table 2.

The lower limb is the most common site for stress fractures, particularly the tibia, metatarsals and fibula. However, they may also occur in the pelvis and non-weight bearing bones such as the ribs. The distribution of stress fractures is dependent on the patient population and the type and level of activity.

Imaging of stress fractures
Imaging plays an important role in the diagnosis of a stress injury. Stress fractures have a variable appearance depending on the amount of time between the onset of injury and the imaging investigation, as the development of these injuries is a continuous process. The main imaging modalities are plain film radiography, computed tomography (CT), bone scintigraphy and magnetic resonance imaging (MRI).

The first imaging technique usually used in the investigation of stress fractures is the plain radiograph (Figure 1). Early radiographs are often normal, with detection rates as low as 15%, and serial radiographs are diagnostic in only 50% of cases. Plain films generally reveal a range of relatively late skeletal responses, from endosteal or periosteal reactions to frank fractures.

Bone scintigraphy (Figure 2) is highly sensitive in identifying stress injury as it detects the osteoblastic activity associated with remodelling; however, it is not specific. CT is less sensitive than scintigraphy or MRI in the early detection of stress injury, but it is more sensitive for the detection of cortical fracture lines. It is therefore useful in demonstrating stress fractures of the sacrum, pars interarticularis, navicular and tibia. Multi-detector CT allows multiplanar and 3D reconstruction of anatomical structures enabling visualisation of subtle fracture lines, but does involve significant ionising radiation.
MRI is able to depict abnormalities weeks before a radiographic lesion. It has comparable sensitivity and superior specificity with bone scintigraphy. It is extremely sensitive in the detection of pathophysiological soft-tissue, bone and marrow changes associated with stress fractures and also demonstrates surrounding muscular or ligamentous injury.

The MR technique should include an oedema sensitive sequence, such as a fat-suppressed T2W or STIR (short tau inversion recovery) images. A T1W image is better to define the anatomy and more advanced fractures. Contrast imaging is not considered essential.

The sensitivity of MR relies on its ability to detect early bone marrow oedema, the hallmark of the stress response. In 2003, Kiuru et al developed a grading system which illustrates how the sequential detection of oedema increases as the severity of the stress response increases. This grading system is summarised in table 3.

MR has also been shown to be useful in the follow-up of healing stress fractures. Resolution of the abnormally bright STIR signal is seen in about 90% of cases within the first six months of the first imaging study.

**Typical locations**

**Tibia**
Due to the increased prevalence of running, the tibia is the most commonly affected bone. Tibial stress fractures usually occur in the proximal or mid shaft and often presents with diffuse tenderness along the tibia.

**Metatarsals**
Stress fractures involving the middle and distal portions of the second and third metatarsal shafts are the most common (figures 1 & 2).

**Talus**
The classic pattern of a talus stress fracture is linear bone marrow oedema perpendicular to the trabecular flow, paralleling the talonavicular articulation at the talar neck (figure 3).

**Calcaneum**
Stress injury of the calcaneum is due to axial compression forces and is often seen in jumpers. It most commonly involves the dorsal posterior aspect (figure 4).

**Navicular**
Navicular fractures are commonly seen in physically active individuals. They are usually linearly orientated in the central third of the navicular. They are often complicated by slow healing, delayed/non-union, osteonecrosis and re-fracture.

**Pelvis and proximal femur**
In this region, approximately 60% of stress fractures are located in the proximal femur (figure 5).

Sacral stress fractures are caused by vertical body forces from the spine to the sacrum and then dissipated onto the sacral ala. MRI is highly sensitive in the detection of early sacral insufficiency fractures, but as diagnosis may be difficult, CT and scintigraphy may also be required.

**Conclusion**
Stress fractures commonly occur in athletic individuals due to repetitive actions. They usually affect the lower limb and, with an appropriate clinical history, imaging plays a pivotal role in diagnosis. The most sensitive and specific modality is MRI, which allows grading of the injury dictating the appropriate rest period to allow healing and preventing overt fracture.

**Extrinsic**
- Excessive load on the body
- Training errors
- Unsuitable training environment
- Poor training equipment
- Ineffective training rules

**Intrinsic**
- Malalignments (eg tibia vara)
- Leg length discrepancy
- Tarsal coalition
- Previous surgery
- Overweight
- Muscle weakness or imbalance

**TABLE 1**
Commonly implicated extrinsic and intrinsic factors in fatigue fracture.

**TABLE 2**
Conditions predisposing to insufficiency fractures.

**Grade**
- Magnetic resonance imaging findings
- I Endosteal oedema
- II Periosteal and endosteal oedema
- III Muscle, periosteal and endosteal oedema
- IV Fracture line
- V Callus at the endosteal +/- periosteal surface of cortical bone

**TABLE 3**
Magnetic resonance imaging grading of stress injuries.

**References**
FIGURE 1
(a) The initial dorsoplantar radiograph of the left foot in a patient with a stress fracture of the second metatarsal, which appears normal. (b) A follow-up dorsoplantar radiograph of the left foot in a patient with a stress fracture of the second metatarsal, which shows a periosteal reaction (arrow).

FIGURE 4
Sagittal fat-saturated T2-weighted image of the left ankle demonstrating a calcaneal stress fracture. The hypointense fracture line is seen surrounded by bone marrow oedema (arrow).
FIGURE 2
Two images from a Tc99m-MDP bone scan of the right foot demonstrating a stress fracture within the shaft of the third metatarsal with increased uptake in the blood pool phase (upper image) and delayed phase (lower image). The plain film was normal (not shown).

FIGURE 3
(a) Sagittal T1-weighted image of the left ankle demonstrating a talar stress fracture with a hypointense fracture line perpendicular to the trabeculae and parallel to the talonavicular joint (arrow). (b) Sagittal fat saturated T2-weighted image of the left ankle demonstrating a talar stress fracture with a hyperintensity in keeping with bone oedema (arrow) with effusions in the ankle joint (curved arrow).
FIGURE 5
(a) Anteroposterior plain film of the right hip demonstrating a proximal femoral stress fracture with a cortical thickening on the medial aspect of the femoral neck but no definite fracture line (arrow). (b) Coronal T1-weighted image of the right hip demonstrating a proximal femoral stress fracture with a hypointense fracture line with surrounding bone oedema (arrow). (c) Coronal STIR of the right hip demonstrating a proximal femoral stress fracture with a hypointense fracture line, surrounding bone oedema and mild oedema in the adjacent soft tissue (arrow).