## **Insufficiency Fractures**

# 16

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Stress fractures occur when bone is repetitively loaded over time without the presence of violent trauma. One type of stress fracture, the insufficiency fracture, is common in older individuals but much less common among younger athletes. In order to develop an understanding of insufficiency fractures it is important to distinguish insufficiency fractures from the pathological fracture and the more common fatigue fracture. Insufficiency fractures occur when normal or physiologic forces are applied on bone with abnormal elastic resistance. In contrast, fatigue fractures occur when repetitive forces are applied to bone of normal elastic resistance. Pathological fractures occur when bone is weakened by infection or tumor [1]. While insufficiency fractures are more commonly experienced in the elderly population, it is important to distinguish between causes of stress fractures in athletes because each type requires its own unique steps in diagnosis and management.

## **Causes of Insufficiency Fractures**

Understanding the causes of insufficiency fracture is of great importance as the prevention and treatment of these fractures must involve correction or

M.J. Tranovich, DO • V.J. Wright, MD, MS (⊠) Department of Orthopaedic Surgery, University of Pittsburgh, 3200 South Water Street, Pittsburgh, PA 15203, USA e-mail: tranovichmj@upmc.edu; wrigvj@upmc.edu prevention of the underlying pathophysiology. Weakened bone leading to insufficiency fracture is commonly associated with osteopenia, osteoporosis, osteomalacia, Paget's disease of bone, or a history of treatment with radiation therapy.

## **Osteopenia and Osteoporosis**

Insufficiency fractures are most commonly associated with bone of decreased density, also known as osteopenic bone. While aging athletes are at risk for osteopenia and consequently insufficiency fracture, it is possible for younger athletes to experience an insufficiency fracture as well.

Causes of osteopenia and osteoporosis can be primary or secondary. Primary causes of decreased bone density include age-related, juvenile, postmenopausal, and osteogenesis imperfecta while secondary causes include several endocrine, hematologic, hereditary, and nutritional disorders. Rheumatoid arthritis, sex hormone deficiency, steroid therapy, hyperparathyroidism, and renal osteodystrophy are a few of the secondary causes of osteopenia associated with insufficiency fracture [2]. Any individual that participates in athletics and possesses one or more of these risk factors for osteopenia also presents with an increased risk of developing an insufficiency fracture.

With the continuous advancement of joint preservation techniques, significantly more individuals are participating in sports well beyond 40 years of age. With increasing age comes an unavoidable increasing amount of oxidative stress and accumulation of free radicals in bone. In addition to physiologic changes, genetics and dietary factors play a role in the development of aging bone. While sports participation may help in maintaining a healthy bone mass, exercise alone will not prevent the development of osteopenia and frank osteoporosis. Therefore, care must be taken to recognize all aging athletes at risk for developing osteopenia and take appropriate dietary and pharmacological steps to prevent osteopenia and decrease the risk of insufficiency fracture.

Diet also plays a role in bone health as protein, calcium, and vitamin D intake are associated with higher bone mineral densities and therefore protection from osteopenia [3]. In a study of female navy recruits it was shown that consumption of 2,000 mg of calcium and 800 IU of vitamin D per day was associated with a 20 % lower incidence of stress fracture [4].

Screening for osteoporosis plays a major role in preventing insufficiency fractures as early diagnosis and treatment can help those at risk to maintain a more normal bone density. In 2009 the American College of Preventative Medicine recommended that all adult patients  $\geq$ 50 years of age be screened for risk factors for osteoporosis. It was recommended that all women  $\geq$ 65 years of age and all men  $\geq$ 70 years of age obtain dual energy X-ray absorptiometry (DEXA) testing to screen for osteoporosis [5]. Younger postmenopausal women and men ages 50–65 with one major or two minor risk factors for osteoporosis (Table 16.1) should also undergo DXA testing [5, 7].

In adults, DEXA is interpreted in terms of *T*-score. By the World Health Organization's reference values, a *T*-score refers to how many standard deviations a patients' bone density lies from the average bone density of a 20- to 29-year-old female. A *T*-score of  $\leq$ -2.5 at the femoral neck, total hip, or lumbar spine generally allows for the diagnosis of osteoporosis [8]. In contrast to the *T*-score, the *Z*-score is based on standard deviations from an age, gender, and race matched reference value. *Z*-scores are used for children and adolescents undergoing DXA testing [9].

Table 16.1	Risk factors	for osteoporosis	5 [ <mark>6</mark> ]ª
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Major risk factors	Minor risk factors
Vertebral compression fracture	Rheumatoid arthritis
Fragility fracture after age 40	Past history of hyperthyroidism
Family history of osteoporotic fracture	Chronic anticonvulsant therapy
Systemic glucocorticoid therapy >3 months	Low dietary calcium intake
Malabsorption syndrome	Smoking
Primary hyperparathyroidism	Excessive alcohol intake
Propensity to fall	Excessive caffeine intake
Osteopenia apparent on X-ray film	Weight <57 kg
Hypogonadism	Weight loss >10 % of weight at age 25
Early menopause (before age 45)	Chronic heparin therapy

<sup>a</sup>Reprinted from American Journal of Preventative Medicine, 36/4, Lim LS, Hoeksema LJ, Sherin K, ACPM prevention practice committee, Screening for osteoporosis in the adult US population: ACPM position statement on preventative practice, 366–75, Copyright (2009), with permission from Elsevier

In premenopausal women, treatment for early osteopenia/osteoporosis includes investigation for underlying causes as well as ensuring adequate intake of calories, calcium (1,000 mg daily from diet), and vitamin D (600 IU D3 supplement) [10]. For premenopausal women, evidence is lacking for common pharmacologic treatments used in postmenopausal women such as bisphosphonates, selective estrogen receptor modulators, teriparatide, and denosumab. Smoking cessation [11], normalization of body weight, avoiding excessive dieting and weight swings [12], and limiting alcohol consumption [13] have been shown to be of benefit in preventing or treating decreased bone mineral density.

In postmenopausal women initial osteoporosis treatment begins with ensuring adequate vitamin D and calcium intake. Along with dietary calcium, supplemental calcium should be taken in doses of 500–1,000 mg/day so that total calcium intake equals around 1,200 mg/day. Intake of vitamin D should equal around 800 IU daily. Exercise and cessation of smoking are also important in this population. The National Osteoporosis Foundation recommends pharmacologic intervention in postmenopausal women with a history of hip fracture or vertebral compression fracture or in those individuals with a *T*-score  $\leq$ -2.5 [14]. Bisphosphonates are considered the mainstay of pharmacologic intervention in postmenopausal osteoporosis [15]. Other effective agents include selective estrogen receptor modulators [16], parathyroid hormone [17], and denosumab [18, 19]. Evidence is lacking or contraindications exist for estrogen replacement, calcitonin, and combination therapy.

Treatment guidelines also exist for the treatment of osteoporosis in men. As with women, diet and lifestyle changes are important in the treatment of osteoporosis. Calcium intake should reach 1,000 mg/day in younger men and up to 1,200 mg/ day in older men. Vitamin D supplementation should equal 600-800 IU per day. Continued exercise, smoking cessation, and limiting alcohol consumption are also important lifestyle changes in men. Testosterone therapy is recommended in younger hypogonadal men with no contraindications to testosterone therapy [20]. In addition to the above measures, pharmacologic intervention is recommended in men with a history of fracture or T-score of  $\leq$ -2.5. Pharmacologic intervention may also be necessary in men with a T-score of -1.0 to -2.5. As with women, bisphosphonate therapy is oftentimes the drug of choice. Studies have recommended weekly treatment with alendronate or risedronate [21]. Other alternatives include the IV bisphosphonate zoledronic acid, and secondline agents such as teriparatide [22] or denosumab [23].

## The Female Athlete Triad and Decreased Bone Mineral Density

Young athletes particularly vulnerable to insufficiency facture are those with the female athlete triad of amenorrhea, eating disorder, and osteoporosis. This triad is specifically observed in physically active females and is now defined as involvement of any one or more of the following components: (1) low energy availability with or without disordered eating; (2) menstrual dysfunction; and (3) low bone

mineral density [24]. Risk factors for the triad that should be assessed in female athletes are menstrual irregularities, criticism of eating habits by coach, family, or peers, depression, dieting, obsessive personality, pressure to lose weight, early sport-specific training, overtraining, recurrent injuries, history of fracture, low BMI, and physical examination signs of an eating disorder [24]. In relation to triad risk factors, a 2014 study found that more triad risk factors are associated with a greater odds of bone stress injury than one factor alone [25]. Specifically, the authors found an increase in bone stress injury from 15 to 21 % for one risk factor to 30 % for two risk factors to 50 % for three triad risk factors [25]. Another 2014 study also found that multiple risk factors exhibit a cumulative risk of lower bone mineral density in young women [26].

Low energy availability in at-risk athletes often leads to menstrual dysfunction and can lead to deleterious effects on the musculoskeletal system. In cases of hypoestrogenism, increased reabsorption of calcium and decreased bone storage of calcium leads to decreased bone mineral density [27]. In terms of menstrual irregularities and bone mineral density, a 2003 study found that female runners experiencing less than 10 menstrual cycles per year had bone mineral densities 3-6 % lower than those female runners having greater than 10 menstrual cycles per year [28]. In addition to the musculoskeletal system, the reproductive, cardiovascular, endocrine, gastrointestinal, renal and neurological systems can be affected by the female athlete triad [24].

Diagnosis of the female athlete triad is multia multidisciplinary faceted and involves approach. Low energy availability can be indicated by a BMI <17.5 kg/m<sup>2</sup> or in adolescents <85 % of expected body weight. While a low BMI can be an indicator of low energy availability assessing energy availability is most often a much more complex measurement. In response to this issue the Female Athlete Triad Coalition provides an energy availability calculator on their website (http://www.femaleathletetriad.org/calculators/). They report that physically active women should aim for at least 45 kcal/kg fat-free mass/day of energy intake [24].

High risk	Moderate risk
<ul> <li>History of a DSM-V diagnosed eating disorder</li> </ul>	• Current or history of disordered eating for ≥6 months
<ul> <li>BMI ≤17.5 kg/m<sup>2</sup>,</li> <li>&lt;85 % estimated weight, or recent weight loss of ≥10 % in 1 month</li> </ul>	<ul> <li>BMI between 17.5 and 18.5, &lt;90 % estimated weight, or recent weight loss of 5–10 % in 1 month</li> </ul>
• Menarche at ≥16 years of age	• Menarche between 15 and 16 years of age
• Current or history of <6 menses over 12 months	• Current or history of 6–8 menses over 12 months
• Two prior stress fractures, 1 high-risk stress fracture, or a low energy nontraumatic fracture	• One prior stress reaction or stress fracture
• Prior Z-score of <-2.0 (after >1 year from baseline DXA)	<ul> <li>Prior Z-score between -1.0 and -2.0 (after &gt;1 year from baseline DXA)</li> </ul>

**Table 16.2** Female athlete triad risk factors [24]

Assessing amenorrhea is a complex process that should be initialized by the primary care physician with appropriate consults to both gynecology and endocrinology specialists. Pregnancy and endocrine disorders such as thyroid dysfunction, hyperprolactinemia, primary ovarian insufficiency, hypothalamic and pituitary disorders, and hyperandrogenic conditions must be ruled out as the causes of the amenorrhea.

Because low bone mineral density is the direct contributing factor to an insufficiency fracture, criteria have been established for obtaining DEXA testing in young woman and girls. The Female Athlete Triad Coalition recommends DEXA testing in athletes with one or more highrisk factors or two or more moderate risk factors (Table 16.2). The Coalition also recommends DEXA testing in athletes with a history of two or more peripheral long bone traumatic fractures when 1 or more high or moderate triad risk factors are identified [24]. Results from DEXA scanning should be interpreted carefully and may need to be repeated every 1-2 years in individuals with ongoing indications for testing. The International Society for Clinical Densitometry (ISCD) provides guidelines for interpreting DEXA testing in children and adolescents. In their 2013 position statement the ISCD maintained that a vertebral compression fracture is indicative of osteoporosis in children and adolescents while densitometry alone is not adequate to diagnose osteoporosis. Total body less head and the posterior-anterior spine are the preferred skeletal areas when performing DEXA testing. In children and adolescents without vertebral compression fracture, osteoporosis is diagnosed with a significant fracture history and a Z-score of  $\leq$ -2.0 on densitometry. The ISCD also reports that a Z-score of >-2.0 does not necessarily preclude skeletal fragility [29, 30]. Young individuals with osteoporosis most often present with fracture before the diagnosis is confirmed; therefore early recognition and prevention of the female athlete triad is instrumental in avoiding insufficiency fractures.

#### **Osteomalacia/Rickets**

Osteomalacia, termed rickets in children, is defective bone mineralization most often caused by a chronic deficiency in vitamin D or phosphate. Consequently, individuals with osteomalacia have a softening of bones which predisposes to fracture. While it is extremely rare in athletes, it should be considered as an underlying cause of insufficiency fracture in athletes with generalized osteopenia. A 2013 study found that DEXA scanning may detect osteoporosis in up to 70 % of individuals with osteomalacia [31]. Patients with osteomalacia often present with generalized bone pain and osteopenia. Generally the best way to prevent osteomalacia induced insufficiency fracture is ensuring adequate vitamin D intake throughout life. In a 2013 position statement, The Society for Adolescent Health and Medicine recommended vitamin D supplementation of 600 IU daily in healthy adolescents and at least 1,000 IU for adolescents at risk for vitamin D insufficiency, in addition to dietary intake. Other guidelines for vitamin D supplementation and management for adolescents are presented in Table 16.3. Some conditions that are potential factors associated with vitamin D deficiency are

**Table 16.3** Vitamin D guidelines as recommended by the Society of Adolescent Health and Medicine [32]

- Vitamin D supplementation of 600 IU daily in healthy adolescents
- Vitamin D supplementation of at least 1,000 IU daily for adolescents at risk for vitamin D insufficiency
- Serum 25(OH)D concentration in at-risk adolescents
- Serum 25(OH)D concentration of 30–50 ng/mL is optimal in adolescents
- In adolescents with <20 ng/mL 25(OH)D, supplement 50,000 IU of vitamin D once per week for 8 weeks
- In adolescents with 20–29 ng/mL 25(OH)D, supplement 1,000 IU per day for at least 3 months
- Use a vitamin D3 preparation if available
- Vitamin D supplementation should be taken with dinner if possible

25(OH)D 25-hydroxyvitamin D

increased skin pigmentation, frequent use of sunscreen, obesity, specific diets such as vegan, cultural body coverage requirements, chronic GI diseases, amenorrhea, pregnancy or lactation, immobilization, bariatric surgery, chronic kidney or liver disease, certain medications such as steroids, anticonvulsants, and HIV medications, and known low bone density status [32]. In older adults the International Osteoporosis Foundation (IOF) states that, on average, 800-1,000 IU of vitamin D are required per day to maintain a serum level of 25-hydroxyvitamin D (25(OH)D) of 30 ng/mL. The required intake also varies per individual as 800 IU per day may be sufficient in healthy individuals with regular sun exposure. On the other hand, obese individuals, those with low sun exposure, with osteoporosis, malabsorption, and in populations such as those of Middle Eastern or Southern Asian decent may need upward of 2,000 IU of vitamin D intake per day [33]. The IOF advises that 100 IU of vitamin D will increase the serum 25(OH)D by about 1.0 ng/mL.

#### Paget's Disease of Bone

Paget's disease of bone is generally a disease of older individuals and is characterized by abnormal bony remodeling and resultant disorganized bony architecture. The disorganized bone growth associated with Paget's disease of bone can initially lie clinically silent and may lead to bone pain, bone deformity, fracture, osteoarthritis, spinal stenosis, cranial nerve compression, tinnitus, deafness, and in a small number, osteosarcoma. Paget's disease of bone is thought to have genetic influences and potentially environmental triggers such as viral infection and low calcium and vitamin D intake. The most common bones affected by Paget's disease of bone are the pelvis, femur, lumbar spine, skull, and tibia [34, 35]. Patients with Paget's disease of bone often have normal calcium, phosphate, and PTH levels on laboratory testing. Variable but often elevated levels of alkaline phosphatase may be observed and depend on the stage of the disease [35]. Another potential presentation finding of Paget's disease of bone is an abnormal radiograph while investigating for other pathologies. Pseudofractures on the convex aspects of affected bones also should raise suspicion of Paget's disease of bone [35]. Patients may also report pain with use of the affected area, with rest, and at night [35]. Other factors useful in diagnosing Paget's disease of bone are localized pain in areas with continued uptake on bone scan and pain improvement with a bisphosphonate trial. Pain that originates in joints is less likely to be due to Paget's disease of bone. While Paget's disease of bone is an extremely rare cause of insufficiency fracture in younger individuals, it should be ruled out as a potential underlying cause of insufficiency fracture in aging athletes as treatment of Paget's disease involves medical and surgical management.

#### **Radiation-Induced Fractures**

While fracture associated with tumor is termed pathological fracture, insufficiency fracture can occur in previously irradiated bone in which a tumor has since resolved. Ionizing radiation is effective as a treatment means for cancer because it causes cell death through DNA strand breaks. This radiation disrupts the bone's blood supply and decreases the number of osteoblasts while increasing the activity and number of osteoclasts. This leads to bone marrow suppression and abnormal bony remodeling, which in effect lowers bone mass and density predisposing affected individuals to insufficiency fracture. These effects are dose dependent and can remain permanent with higher radiation dosages. Therefore, athletes with a history of radiation therapy must be monitored for the development of insufficiency fractures [36].

## Presentation of Insufficiency Fractures

Patients with insufficiency fractures often present with acute pain in a commonly affected area such as the back, groin, or foot. A history of trauma is usually lacking. Depending on the severity of the fracture, the patient may present in a nonambulatory state. Physical examination of a suspected insufficiency fracture involves localization of pain and inspection of the area for warmth and swelling and palpation for tenderness. Range of motion, the fulcrum test, flexion–abduction– external rotation (FABER test), and Flamingo test may assist in evaluation of areas not readily accessible to direct palpation [3].

## **Imaging of Insufficiency Fractures**

Obtaining proper imaging is instrumental in early recognition and treatment of suspected insufficiency fractures. As with any suspected skeletal injury, plain radiographs should be obtained. Plain radiographs may assist in the diagnosis of various insufficiency fractures though magnetic resonance imaging (MRI), computed tomography (CT), or bone scintigraphy may be necessary in cases where plain radiographs are inconclusive and a patient's pain persists.

## Radiography

The most common radiographic imaging finding in patients with insufficiency fracture is a sclerotic band in the affected bone. Other imaging findings on radiography of insufficiency fractures include bone resorption along the fracture line, bony expansion, callus, and osteolysis. Radiography is often more successful in detecting insufficiency fractures in long bones, pubic rami, and peripheral bones while sacral and pelvic insufficiency fractures are more often elusive on standard radiographs [37].

#### Multidetector CT Scanning

Multidetector CT (MDCT) scanning may be the imaging modality of choice in the detection of insufficiency fractures though the high dose of ionizing radiation limits its usage to some extent. MDCT allows for thin slices and detection of otherwise non-visible fracture lines. Callus development can also be readily observed. While bony edema on MRI can be suggestive of fracture as well as other processes such as tumor, high resolution CT scanning has the ability to rule out lytic lesions and those extending into the adjacent soft tissue [37].

#### Magnetic Resonance Imaging

Due to the absence of ionizing radiation, MRI is commonly used as the imaging modality of choice in the diagnosis of insufficiency fracture when plain radiographs prove inconclusive. Hypointense signal to adjacent bone is seen on T1 weighted images while hyperintense signal is observed on T2 weighted images with the possibility of observing the fracture line within bony edema. A commonly cited radiographic and MR grading system of stress fractures can be found in Table 16.4 [38]. Care must be taken as to not confuse an insufficiency fracture with underlying avascular necrosis or tumor.

## **Bone Scintigraphy**

Bone scintigraphy, also termed radionucleotide scanning or bone scanning is very sensitive for insufficiency fractures though generally it is nonspecific. Various uptake patterns can be difficult to interpret and can remain positive well after a fracture occurs. Bone scintigraphy has clinical utility in the diagnosis of insufficiency fractures of the sacrum and pelvis as two or more areas of

Grade	Radiograph findings	MRI findings
Normal	Normal	Normal
1	Normal	Positive STIR image
2	Normal	Positive STIR, plus positive T2-weighted
3	Periosteal reaction	Positive T1 and T2 weighted, STIR without definite cortical break visualized
4	Injury or periosteal reaction	Positive injury line on T1 or T2 weighted scans

 Table 16.4
 Radiographic grading of stress fractures<sup>a</sup>

<sup>a</sup>Adapted from Clin Sports Med, 16/2, Arendt EA, Griffiths HJ, The use of MR imaging in the assessment and clinical management of stress reactions of bone in high-performance athletes, 291–306, 1997, with permission from Elsevier

increased uptake in the sacrum and another in a pelvic site are diagnostic of insufficiency fractures [37]. Grading of stress fractures on bone scans has been proposed as grade 1, a small, ill-defined cortical area of mildly increased activity; grade 2, larger well-defined cortical area of moderately increased activity; grade 3, wide to fusiform involvement, cortical-medullary area of highly increased activity; and grade 4, transcortical area of intensely increased activity [39]. This grading system has also been correlated to MRI findings as Grade 1, mild to moderate periosteal edema on T2 with normal marrow; grade 2, moderate to severe periosteal edema on T2 with marrow edema on T2; grade 3, moderate to severe periosteal edema on T2 with marrow edema on T1 and T2; and grade 4, moderate to severe periosteal edema on T2 with a clearly visible fracture line and marrow edema on T1 and T2 images [40]. It should be noted that a more recent study made note that periosteal edema is often observed on MR but not always present in a bone stress injury [41].

## Specific Sites of Insufficiency Fractures and Management

#### Pelvis

Pelvic insufficiency fractures are more often seen in elderly individuals with osteoporosis though any condition that leads to premature osteoporosis can predispose to the development of an insufficiency fracture. Pelvic insufficiency fractures are also associated with a history of radiation therapy in postmenopausal women. In younger women, a pelvic insufficiency fracture is most often observed in the inferior pubic rami [42]. Insufficiency fractures in the pelvis can often present as low back pain making the proper diagnosis more difficult to determine. Because plain radiographs oftentimes do not visualize these fractures with certainty, MRI of the pelvis is often necessary to confirm the diagnosis. Patients may have previously had an extensive lower back workup with the causative fracture escaping previous imaging attempts. In a 2008 study of MRI and CT imaging of 145 patients with pelvic and proximal femur stress fractures, it was found that 70.3 % of patients had a stress fracture at more than 1 site. In patients with pubic stress fractures 89.2 % had concomitant stress fractures, most commonly in the sacrum and acetabulum. In patients diagnosed with acetabular stress fractures, 76 % had concomitant fractures [43].

Pelvic insufficiency fractures are generally considered low-risk for malunion or nonunion in active individuals, therefore treatment generally consists of conservative management [44]. As mentioned previously, any underlying medical cause must be investigated and treated appropriately. A period of rest with gradual return to activity is usually utilized for pelvic insufficiency fractures. Limited or nonweightbearing is prescribed for 2–6 weeks with gradual progression to full weightbearing. Prolonged low-impact activities should be achieved without pain before resumption of high-impact exercises [44].

#### Sacrum

Like the pelvis, sacral insufficiency fractures can be difficult to diagnose. Bone scanning is generally very sensitive for detecting sacral insufficiency fractures as a characteristic "H" pattern or the combination of concomitant sacral and parasymphyseal uptake being typical of fractures in this region [45]. Sacral insufficiency fractures are also more common in elderly individuals and can be a cause of significant back pain. As with other insufficiency fractures, osteoporosis and conditions that are associated with osteoporosis such as hyperparathyroidism and renal osteodystrophy are the most common underlying causes though a history of radiation therapy and Paget's disease of bone are other possibilities [46, 47]. It is also important to rule out tumor as a cause of the fracture. A 2013 study found that menstrual irregularities were noted in 75 % of female athletes with trabecular bone injuries, which includes the sacrum [41]. This presents further evidence that female athlete triad risk factors are associated with insufficiency fractures normally found in elderly women.

Similar to pelvic insufficiency fractures, sacral insufficiency fractures are generally low-risk in terms of malunion/nonunion [44]. As such, many authors advocate initial conservative management consisting of rest, pain control, and modified weightbearing. Conservative management is usually the preferred treatment method in younger individuals with sacral insufficiency fracture and return to sport has been reported between 3 and 6 months in distance runners with stress fracture, many of whom exhibited characteristics consistent with the female athlete triad [48, 49]. Resolution of symptoms can be a lengthy and risky process with conservative management in older individuals; therefore, surgical treatment techniques are sometimes the preferred treatment method. Surgical management is usually only considered in younger athletes after failed conservative management. A variation to vertebroplasty, sacroplasty, is one possible surgical treatment method which involves injection of polymethylmethacrylate cement under fluoroscopic guidance into the fracture. The benefits of sacroplasty as compared to conservative management include earlier mobilization and symptom relief as well as reduction of risks associated with prolonged immobilization [47, 50, 51]. Techniques of this procedure vary per surgeon as posterior approach, long axis approach, and midline approach have been advocated. The most significant complication is extravasation of cement outside of the fracture which may cause neurological sequelae [52].

#### Spine

Spinal insufficiency fractures can occur in various regions of the spine and in different areas of the vertebrae but are most often associated with the vertebral body. While spinal insufficiency fractures such as wedge or burst fractures are a common cause of back pain in the elderly with osteoporosis, literature is lacking in terms of vertebral insufficiency fractures in the younger athlete. Theoretically any condition that leads to decreased bone density in an athlete could predispose an athlete to sustain spondylolysis or a vertebral insufficiency fracture similar to an elderly osteoporotic patient. As such, care must be exercised in evaluating an athlete with back pain and risk factors for decreased bone density.

#### **Hip and Femur**

Insufficiency fractures of the femur can occur at several locations including the femoral head, femoral neck, femoral diaphysis, and femoral condyles. An insufficiency fracture of the hip can be a catastrophic injury in an athlete and as with other insufficiency fractures prevention and treatment of underlying risk factors are of monumental importance in minimizing time-lost to these injuries. Patients presenting with stress fractures in the hip often report pain in the anterior groin and pain with internal and external rotation of the hip [53, 54]. Radiographs and oftentimes MRI are of great importance in distinguishing these conditions because hip pathologies such as femoroacetabular impingement often present with similar anterior groin pain and pain with hip internal rotation.

One possible insufficiency fracture of the hip is subchondral insufficiency fracture of the femoral head. Initially, radiographs may be negative until callus formation is viewed in resolving cases [55]. In patients that progress to collapse of the femoral head, a fracture line termed a "crescent sign" may be observed. Because subchondral insufficiency fracture of the femoral head often escapes early detection by radiographs, MRI is necessary in suspected cases. Bone marrow edema and a low-signal intensity line on T1 images parallel to the subchondral bone are often observed [37]. Subchondral insufficiency fracture is oftentimes confused with osteonecrosis of the femoral head. A history of corticosteroid use or alcohol abuse may raise suspicion of osteonecrosis while a history of osteoporosis may raise suspicion of subchondral insufficiency fracture [56]. Radiographic appearance and MR findings may be similar between subchondral insufficiency fracture and osteonecrosis. While not always reliable, a high signal intensity of the proximal segment divided by the fracture line on a T2 or gadolinium enhanced image suggests subchondral insufficiency fracture while in osteonecrosis, before healing occurs, the subchondral bone segment proximal to the low intensity band is of lower signal intensity. Histopathology is the diagnostic confirmatory test to discern the two entities.

It has been suggested that if femoral head collapse is not present conservative management may be utilized. In cases of anterosuperior femoral head collapse in young patients a transtrochanteric rotational osteotomy may be a treatment option while hemiarthroplasty or total hip arthroplasty is utilized in elderly patients [57].

Another possible insufficiency fracture of the hip occurs at the femoral neck. On MRI, fracture lines and bone marrow edema are often appreciated, especially on coronal images [43]. It is important to distinguish the affected area of the femoral neck as insufficiency fractures occurring at the superior femoral neck (tension side) are at significant risk for malunion, nonunion, fracture migration across the femoral neck with subsequent displacement, and consequently avascular necrosis [53]. While strict non-weightbearing may successfully treat these fractures, their propensity for displacement oftentimes leads to cancellous lag screw fixation of the femoral neck [58]. In addition, these patients require 6 weeks of non-weightbearing followed by 6 weeks of partial weightbearing postoperatively. In contrast, an insufficiency fracture occurring at the inferior femoral neck (compression side) is at significantly lower risk for nonunion and displacement



**Fig. 16.1** Bilateral insufficiency fractures on the compression (inferior) side of the femoral neck in a patient with suspected osteomalacia. Fractures indicated by *arrows*. Image courtesy of Joanna Costello, M.D.



**Fig. 16.2** T2 sagittal MRI image of a subchondral insufficiency fracture with subchondral collapse of the medial femoral condyle in a previously active 64-year-old male. Note the significant bone marrow edema and subchondral collapse indicated by the *arrow*. Image courtesy of Joanna Costello, M.D.

and can most often be treated non-operatively [53, 59] (Fig. 16.1).

It should be noted that subchondral insufficiency fracture can also occur in the femoral condyles and may be termed spontaneous osteonecrosis of the knee (SONK or SPONK) (Fig. 16.2). These insufficiency fractures have also been associated with low bone mineral

density [60]. Subchondral insufficiency fractures in this region are initially treated with pain management, protected weightbearing, and possibly bisphosphonates [61] with refractory cases necessitating high tibial osteotomy [62], unicompartmental arthroplasty [63], or total knee arthroplasty.

Insufficiency fracture can also occur at any point along the femoral diaphysis. Clinical presentation often includes pain in the thigh with weight bearing. These fractures tend to have a lower risk of nonunion and displacement so management typically consists of conservative measures.

### Tibia

The tibia is a common location of both insufficiency and fatigue stress fractures in the athlete. These stress fractures tend to present in distance runners and can be a source of continuous pain and debilitation. A 2005 study of imaging findings in early tibial stress injuries in active young patients found that 21/50 of the tibiae studied were osteopenic [64]. This finding highlights the fact that many tibial stress fractures can be classified as insufficiency fractures rather than fatigue fractures due to the presence of underlying abnormality. This also highlights the fact that prevention of osteopenia in young patients is of significant importance in terms of fracture prevention.

Due to the possibility of nonunion and extension of the fracture line, anterior tibial diaphyseal stress fractures are classified as high-risk. Patients often present with complaints of pain with weightbearing activities and tenderness over the anterior tibia [53]. Oftentimes, tibial stress fractures may be appreciated on plain radiographs as a cortical thickening or in persistent fractures, a transverse fissure line in the cortex [65] (Fig. 16.3a). On occasion, multiple fissures corresponding to more than 1 stress fracture may be observed. If left untreated these small defects can progress to a complete fracture through both cortices. With inconclusive radiographs in highlevel athletes, MRI is a standard imaging technique. T2 and STIR MRI sequences most often confirm cortical abnormalities, a round or oval area of high signal intensity and a hyperintense line through the cortex.

Athletes that fail a period of non-weightbearing with crutches and possible pneumatic bracing may necessitate surgical treatment. Successful union, resolution of pain, and return to play at as early as 4 months has been reported with intramedullary

Fig. 16.3 (a) Anterior tibial diaphyseal stress fracture in a 19-year-old gymnast with risk factors for the female athlete triad. Fracture indicated with arrow. (b) Postoperative radiograph demonstrating successful treatment with an antegrade intramedullary nail following a failed course of non-operative management as well as calcium and vitamin D supplementation. The fracture line is less visible 2.5 months postoperatively. Location of original fracture line indicated with arrow. Images courtesy of Joanna Costello, M.D.



nailing (Fig. 16.3b) [66], anterior tension band plating [67–70], or drilling with bone grafting of the cortical defect [71, 72].

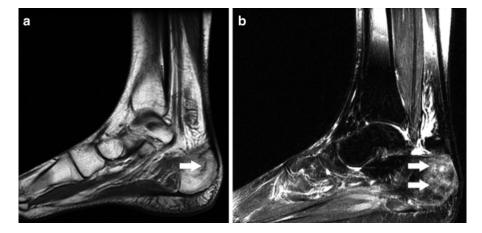
#### Foot

Stress fractures of the foot have been reported in every bone except the lesser toes. Commonly affected sites include the calcaneus, talus, navicular, and metatarsals. Because of the large number of bones in the foot, localization of the potential fracture may be difficult. Insufficiency fractures of the foot are especially common in the neuropathic foot associated with diabetes mellitus and in the elderly with osteoporosis though they may occur in younger athletes with underlying bone density issues.

Like other stress fractures, calcaneal stress fractures are more likely to be fatigue fractures in young patients or insufficiency fractures in the elderly though they do occur as insufficiency fractures in younger athletes. Insufficiency fractures of the calcaneus commonly present with pain and tenderness, especially in the posterior superior region [44]. The pain may mimic

Achilles tendinitis/bursitis so proper imaging is necessary to differentiate between the two. Radiographs may be useful in the imaging of these lesions and the fractures are usually viewed as a sclerotic line that lies in a vertical-type orientation [44]. MRI is also useful in confirming these fractures and differentiation from Achilles pathology and more severe lesions such as tumor. Marrow edema is often present on T2 sequencing and a vertically oriented fracture line may be appreciated [73]. Calcaneal stress fractures are classified as low-risk for nonunion, therefore they may successfully heal with nonoperative management which can include nonweightbearing progressed to a boot and subsequently adequate heel lift and padding. Fracture healing may be affected if underlying medical pathologies are not treated as well. Figure 16.4a, b presents a case of insufficiency fracture of the calcaneus in a 26-year-old female runner that healed after prolonged conservative and medical management.

The talus is another hindfoot bone that may experience insufficiency fracture. These fractures are associated with decreased bone density [74] and rheumatoid arthritis [75]. Talar stress fractures



**Fig. 16.4** (a) T1 weighted sagittal MRI image of a curvilinear calcaneus insufficiency fracture in an active 26-year-old runner with progestin only birth-control induced amenorrhea, vitamin D deficiency and DEXA confirmed osteopenia. Fracture line indicated by *arrow*. (b) T2 weighted sagittal MRI image demonstrating bone marrow edema (indicated by *arrows*) in the area of the

fracture. The patient was treated with estrogen containing contraceptive medication, calcium and vitamin D supplementation, extended non-weightbearing and was eventually transitioned to a boot and full weightbearing. Two-year follow-up DEXA scanning demonstrated improvement in bone density to Z-scores within normal ranges. Images courtesy of Joanna Costello, M.D. can occur in the talar body, talar neck, or lateral process with the majority occurring in the talar head in young patients [76]. Patients may complain of pain in various areas of the foot, making these fractures difficult to localize [74]. Standard radiographs may not appreciate these lesions therefore MRI and/or CT are usually necessary. On MR imaging, bone marrow edema is often observed on T2 weighted imaging and a subchondral linear fracture line may be viewed on T1 weighted images. In contrast to stress fractures of the calcaneus, talar insufficiency fractures are at high-risk for nonunion and often necessitate surgical treatment though healing with protected weightbearing and casting has been reported [53, 74].

Metatarsal stress fractures, specifically 5th metatarsal stress fractures are a well-studied injury in athletes. While metatarsal stress fractures are most often of the fatigue type, insufficiency fractures have been reported. According to the most common anatomical classification system, fifth metatarsal stress fractures are found at the proximal diaphysis and are not to be confused with the avulsion (Zone 1) fracture or Jones (Zone 2) fracture. As opposed to the Jones fracture which is located at the metaphysealdiaphyseal junction and enters the 4th and 5th metatarsal articulation, the stress fracture lies distal to the 4th–5th articulation [77, 78]. Patients often report pain exacerbated by inversion and tenderness over the lateral foot. These fractures can most often be observed on standard radiographs as a radiolucent line making further imaging unnecessary in most cases. Second metatarsal base and fifth metatarsal fractures are considered high-risk and are prone to nonunion and refracture after conservative management making intramedullary screw placement the preferred treatment method [77–79]. A 2014 study of 5th metatarsal fractures, foot stress fractures, and ankle fractures found that 47 % of the patients studied had a vitamin D level below the recommended level, suggesting that a number of these fractures may have a component of insufficiency rather than solely being fatigue fractures [80].

#### Upper Extremity

Upper extremity stress fractures are relatively uncommon injuries. Most literature reports examining upper extremity stress fractures explore fatigue type injuries rather than the less common insufficiency fracture in a younger athlete. Sites of reported stress fracture include the shoulder girdle, humerus, ulna, radius, scaphoid, and metacarpals. Throwing athletes, swimmers, gymnasts, weight lifters, and rowers are the more commonly reported athletic populations that sustain upper extremity stress injury [81]. In general upper extremity stress fractures are considered low-risk for nonunion and can be successfully treated with non-operative management [44]. Li and colleagues reported the case of a 12-year-old male baseball pitcher with secondary hyperparathyroidism and vitamin D deficiency that sustained a proximal humeral stress fracture and subsequently was diagnosed with a proximal ulnar stress fracture as well as spondylolisthesis. Interestingly, the athlete had a Z-score of 2.76 consistent with significantly elevated bone mineral density. The authors concluded that this increased bone mineral density may have translated into bone changes similar to osteopetrosis. The athlete was treated non-operatively and with medical management of his vitamin D levels and subsequently returned to baseball participation [82]. Being that a stress fracture associated with abnormal bone is an insufficiency fracture, it is clear that upper extremity insufficiency fractures do occur in athletes and at times may be misclassified as fatigue fractures. As with other stress fractures, underlying medical diagnoses must be ruled out before concluding the presence of fatigue fracture rather than insufficiency fracture in the young athlete.

#### Summary

Insufficiency type stress fractures are less commonly reported than fatigue type stress fracture but do in fact occur in athletes. At particular risk are aging athletes with osteopenia or osteoporosis and younger athletes with decreased bone density as a result of underlying causes such as the female athlete triad. Recognition of the causes of abnormal bone predisposing an athlete to insufficiency fracture is essential in the prevention of this type of injury. Proper imaging, correction of underlying medical pathologies, and possible surgical intervention in refractory and high-risk cases are necessary in order to maximize an athletes' potential to return-to-play.

#### References

- Pentecost RL, Murray RA, Brindley HH. Fatigue, insufficiency, and pathologic fractures. JAMA. 1964; 187(13):1001–4.
- Soubrier M, Dobost JJ, Boisgard S, Sauvezie B, Gaillard P, Michel JL, et al. Insufficiency fracture. A survey of 60 cases and review of the literature. Joint Bone Spine. 2003;70(3):209–18.
- Behrens SB, Deren ME, Matson A, Fadale PD, Monchik KO. Stress fractures of the pelvis and legs in athletes: a review. Sports Health. 2013;5(2):165–74.
- Lappe J, Cullen D, Haynatzki G, Recker R, Ahlf R, Thompson K. Calcium and vitamin D supplementation decreases incidence of stress fractures in female navy recruits. J Bone Miner Res. 2008;23(5):741–9.
- Lim LS, Hoeksema LJ, Sherin K. ACPM prevention practice committee. Screening for osteoporosis in the adult US population: ACPM position statement on preventative practice. Am J Prev Med. 2009;36(4): 366–75.
- Brown JP, Josse RG. 2002 clinical practice guidelines for the diagnosis and management of osteoporosis in Canada. CMAJ. 2002;167(10 Suppl):S1–34.
- Malabanan AO, Rosen HN, Vokes TJ, Deal CL, Alele JD, Olenginski TP, et al. Indications of DXA in women younger than 65 yr and men younger than 70 yr: the 2013 official positions. J Clin Densitom. 2013; 16(4):467–71.
- Schousboe JT, Shepherd JA, Bilezikian JP, Baim S. Executive summary of the 2013 ISCD position development conference on bone densitometry. J Clin Densitom. 2013;16(4):455–67.
- Gordon CM, Leonard MB, Zemel BS. 2013 pediatric position development conference: executive summary and reflections. J Clin Densitom. 2014;17:219–24.
- Ross AC, Manson JE, Abrams SA, Aloia JF, Brannon PM, Clinton SK, et al. The 2011 report on dietary reference intakes for calcium and vitamin D from the Institute of Medicine: what clinicians need to know. J Clin Endocrinol Metab. 2011;96(1):53–8.
- Ward KD, Klesges RC. A meta analysis of the effects of cigarette smoking on bone mineral density. Calcif Tissue Int. 2001;68(5):259–70.

- Bacon L, Stern JS, Keim NL, Van Loan MD. Low bone mass in premenopausal chonric dieting obese women. Eur J Clin Nutr. 2004;58(6):966–71.
- Tudor-Locke C, McColl RS. Factors related to variation in premenopausal bone mineral status: a health promotion approach. Osteoporos Int. 2000;11(1):1–24.
- Khan SN, Craig L, Wild R. Osteoporosis: therapeutic guidelines. Guidelines for practice management of osteoporosis. Clin Obstet Gynecol. 2013;56(4): 694–702.
- Yates J. A meta-analysis characterizing the doseresponse relationships for three oral nitrogencontaining bisphosphonates in postmenopausal women. Osteoporos Int. 2013;24(1):253–62.
- 16. Johnston Jr CC, Bjarnason NH, Cohen FJ, Shah A, Lindsay R, Mitlak BH, et al. Long-term effects of raloxifene on bone mineral density, bone turnover, and serum lipid levels in early postmenopausal women: three-year data from 2 double-blind, randomized, placebo-controlled trials. Arch Intern Med. 2000; 160(22):3444–50.
- Neer RM, Arnaud CD, Zanchetta JR, Prince R, Gaich GA, Reginster JY, et al. Effect of parathyroid hormone (1–34) on fractures and bone mineral density in postmenopausal women with osteoporosis. N Engl J Med. 2001;344(19):1434–41.
- 18. Brown JP, Prince RL, Deal C, Recker RR, Kiel DP, de Gregorio LH, et al. Comparison of the effect of denosumab and alendronate on BMD and biochemical markers of bone turnover in postmenopausal women with low bone mass: a randomized, blinded, phase 3 trial. J Bone Miner Res. 2009;24(1):153–61.
- Bone HG, Chapurlat R, Brandi ML, Brown JP, Czerwinski E, Krieg MA, et al. The effect of three of six years of denosumab exposure in women with postmenopausal osteoporosis: results from the FREEDOM extension. J Clin Endocrinol Metab. 2013;98(11): 4483–92.
- Behre HM, Kliesch S, Leifke E, Link TM, Nieschlag E. Long-term effect of testosterone therapy on bone mineral density in hypogonadal men. J Clin Endocrinol Metab. 1997;82(8):2386–90.
- 21. Levis S, Theodore G. Summary of AHRQ's comparative effectiveness review of treatment to prevent fractures in men and women with low bone density or osteoporosis: update of the 2007 report. J Manag Care Pharm. 2012;18(4 Suppl B):S1–15.
- 22. Orwoll ES, Scheele WH, Paul S, Adami S, Syversen U, Diez-Perez A, et al. The effect of teriparatide [human parathyroid hormone (1–34)] therapy on bone density in men with osteoporosis. J Bone Miner Res. 2003;18(1):9–17.
- 23. Orwoll E, Teglbjaerg CS, Langdahl BL, Chapurlat R, Czerwinski E, Kendler DL, et al. A randomized, placebo-controlled study of the effects of denosumab for the treatment of men with low bone mineral density. J Clin Endocrinol Metab. 2012;97(9):3161–9.
- DeSouza MJ, Nattiv A, Joy E, Misra M, Williams NI, Mallinson WJ, et al. 2014 female athlete triad coalition

consensus statement on treatment and return to play of the female athlete triad: 1st international conference held in San Francisco, CA, May 2012, and 2nd international conference held in Indianapolis, IN, May 2013. Clin J Sport Med. 2014;24(2):96–119.

- Barrack MT, Gibbs JC, DeSouza MJ, Williams NI, Nichols JF, Rauh MJ, et al. Higher incidence of bone stress injuries with increasing female athlete triadrelated risk factors: a prospective multisite study of exercising girls and women. Am J Sports Med. 2014; 42:949–58.
- Gibbs JC, Nattiv A, Barrack MT, Williams NI, Rauh MJ, Nichols JF, DeSouza MJ. Low bone density risk is higher in exercising women with multiple triad risk factors. Med Sci Sports Exerc. 2014;46(1):167–76.
- Feingold D, Hame SL. Female athlete triad and stress fractures. Orthop Clin North Am. 2006;37(4):575–83.
- Cobb KL, Bachrach LK, Greendale G, Marcus R, Neer RM, Nieves J, et al. Disordered eating, menstrual irregularity, and bone mineral density in female runners. Med Sci Sports Exerc. 2003;35(5):711–9.
- 29. Crabtree NJ, Arabi A, Bachrach LK, Fewtrell M, El-Hajj Fuleihan G, Kecskemethy HH, et al. Duelenergy X-ray absorptiometry interpretation and reporting in children and adolescents: revised 2013 ISCD pediatric official positions. J Clin Densitom. 2014;17(2):225–42.
- Kalkwarf HJ, Abrams SA, Dimeglio LA, Koo WW, Specker BL, Weller H. Bone densitometry in infants and young children: the 2013 ISCD pediatric official positions. J Clin Densitom. 2014;17(2):243–57.
- Saghafi M, Azarian A, Hashemzadeh K, Sahebari M, Rezaieyzdi Z. Bone densitometry in patients with osteomalacia: is it valuable? Clin Cases Miner Bone Metab. 2013;10(3):180–2.
- 32. The Society for Adolescent Health and Medicine. Recommended vitamin D intake and management of low vitamin D status in adolescents: a position statement of the society for adolescent health and medicine. J Adolesc Health. 2013;52(6):801–3.
- Dawson-Hughes B, Mithal A, Bonjour JP, Boonen S, Burckhardt P, Fuleihan GEH, et al. IOF position statement: vitamin D recommendations for older adults. Osteoporos Int. 2010;21:1151–4.
- Ralston SH. Pathogenesis of Paget's disease of bone. Bone. 2008;43:819–25.
- Ralston SH, Langston AL, Reid IR. Pathogenesis and management of Paget's disease of bone. Lancet. 2008;372:155–63.
- Pacheco R, Stock H. Effects of radiation on bone. Curr Osteoporos Rep. 2013;11:299–304.
- Krestan C, Hojreh A. Imaging of insufficiency fractures. Eur J Radiol. 2009;71:398–405.
- Arendt EA, Griffiths HJ. The use of MR imaging in the assessment and clinical management of stress reactions of bone in high-performance athletes. Clin Sports Med. 1997;16(2):291–306.
- Zwas ST, Elkanovitch R, Frank G. Interpretation and classification of bone scintigraphic findings in stress fractures. J Nucl Med. 1987;28:452–7.

- 40. 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(4):472–81.
- 41. Nattiv A, Kennedy G, Barrack MT, Abdelkerim A, Goolsby MA, Arends JC, et al. Correlation of MRI grading of bone stress injuries with clinical risk factors and return to play: a 5-year prospective study in collegiate track and field athletes. Am J Sports Med. 2013;41(8):1930–41.
- Miller C, Major N, Toth A. Pelvic stress injuries in the athlete: management and prevention. Sports Med. 2003;33(13):1003–12.
- Cabarrus MC, Ambekar A, Lu Y, Link TM. MRI and CT of insufficiency fractures of the pelvis and proximal femur. Am J Roentgenol. 2008;191(4):995–1001.
- 44. Boden BP, Osbahr DC, Jimenez C. Low-risk stress fractures. Am J Sports Med. 2001;29(1):100–11.
- Krestan CR, Nemec U, Nemec S. Imaging of insufficiency fractures. Semin Musculoskelet Radiol. 2011; 15:198–207.
- 46. Gotis-Graham I, McGuigan L, Diamond T, Portek I, Quinn R, Sturgess A, et al. Sacral insufficiency fractures in the elderly. J Bone Joint Surg Br. 1994; 76(6):882–6.
- Lyders EM, Whitlow CT, Baker MD, Morris PP. Imaging and treatment of sacral insufficiency fractures. Am J Neuroradiol. 2010;31(2):201–10.
- Fredericson M, Salamancha L, Beaulieu C. Sacral stress fractures: tracking down nonspecific pain in distance runners. Phys Sportsmed. 2003;31(2):31–42.
- Klossner D. Sacral stress fracture in a female collegiate distance runner: a case report. J Athl Train. 2000;35(4):453–7.
- 50. Eichler K, Zangos S, Mack MG, Marzi I, Vogl TJ. Outcome of long-axis percutaneous sacroplasty for the treatment of sacral insufficiency fractures with a radiofrequency-induced, high-viscosity bone cement. Skeletal Radiol. 2014;43:493–8.
- Garant M. Sacroplasty: a new treatment for sacral insufficiency fracture. J Vasc Interv Radiol. 2002; 13:1265–7.
- Frey ME, Depalma MJ, Cifu DX, Bhagia SM, Carne W, Daitch JS. Percutaneous sacroplasty for osteoporotic sacral insufficiency fractures: a prospective, multicenter, observational pilot study. Spine J. 2008; 8(2):367–73.
- Boden BP, Osbahr DC. High-risk stress fractures: evaluation and treatment. J Am Acad Orthop Surg. 2000;8(6):344–53.
- Yamamoto T. Subchondral insufficiency fractures of the femoral head. Clin Orthop Surg. 2012;4(3):173–80.
- 55. Kim JW, Yoo JJ, Min BW, Hong SH, Kim HJ. Subchondral fracture of the femoral head in healthy adults. Clin Orthop Relat Res. 2007;464: 196–204.
- 56. Ikemura S, Yamamoto T, Motomura G, Nakashima Y, Mawatari T, Iwamoto Y. The utility of clinical features for distinguishing subchondral insufficiency

fracture from osteonecrosis of the femoral head. Arch Orthop Trauma Surg. 2013;133:1623–7.

- Yamamoto T, Iwasaki K, Iwamoto Y. Transtrochanteric rotational osteotomy for a subchondral insufficiency fracture of the femoral head in young adults. Clin Orthop Relat Res. 2010;468(12):3181–5.
- Aro H, Dahlstrom S. Conservative management of distraction-type stress fractures of the femoral neck. J Bone Joint Surg Br. 1986;68:65–7.
- DeFrano MJ, Recht M, Schills J, Parker RD. Stress fractures of the femur in athletes. Clin Sports Med. 2006;25(1):89–103.
- Akamatsu Y, Mitsugi N, Hayashi T, Kobayashi H, Saito T. Low bone mineral density is associated with the onset of spontaneous osteonecrosis of the knee. Acta Orthop. 2012;83(3):249–55.
- Jureus J, Lindstrand A, Geijer M, Roberts D, Tagil M. Treatment of spontaneous osteonecrosis of the knee (SPONK) by a bisphosphonate. Acta Orthop. 2012;83(5):511–4.
- 62. Saito T, Kumagai K, Akamatsu Y, Kobayashi H, Kusayama Y. Five- to ten-year outcome following medial opening-wedge high tibial osteotomy with rigid plate fixation in combination with an artificial bone substitute. Bone Joint J. 2014;96-B(3):339–44.
- Bruni D, Iacono F, Raspugli G, Zaffagnini S, Marcacci M. Is unicompartmental arthroplasty an acceptable option for spontaneous osteonecrosis of the knee? Clin Orthop Relat Res. 2012;470(5):1442–51.
- 64. Gaeta M, Minutoli F, Scribano E, Ascenti G, Vinci S, Bruschetta D, et al. CT and MR imaging findings in athletes with early tibial stress injuries: comparison with bon scintigraphy findings and emphasis on cortical abnormalities. Radiology. 2005;235(2):553–61.
- 65. Orava S, Hulkko A. Stress fracture of the mid-tibial shaft. Acta Orthop Scand. 1984;55(1):35–7.
- 66. Varner KE, Younas SA, Lintner DM, Marymont JV. Chronic anterior midtibial stress fractures in athletes treated with reamed intramedullary nailing. Am J Sports Med. 2005;33(7):1071–6.
- Borens O, Sen MK, Huang RC, Richmond J, Kloen P, Jupiter JB, et al. Anterior tension band plating for anterior tibial stress fractures in high-performance athletes: a report of 4 cases. J Orthop Trauma. 2006; 20:425–30.
- Cruz AS, deHollanda JP, Duarte Jr A, Hungria Neto JS. Anterior tibial stress fractures treated with anterior tension band plating in high-performance athletes. Knee Surg Sports Traumatol Arthrosc. 2013;21(6): 1447–50.

- Liimatainen E, Sarimo J, Hulkko A, Ranne J, Heikkila J, Orava S. Anterior mid-tibial stress fractures. Results of surgical treatment. Scand J Surg. 2009;98(4):244–9.
- Merriman JA, Villacis D, Kephart CJ, Rick Hatch 3rd GF. Tension band plating of a nonunion anterior tibial stress fracture in an athlete. Orthopedics. 2013; 36(7):534–8.
- Beals RK, Cook RD. Stress fractures of the anterior tibial diaphysis. Orthopedics. 1991;14(8):869–75.
- Miyamoto RG, Dhotar HS, Rose DJ, Egol K. Surgical treatment of refractory tibial stress fractures in elite dancers: a case series. Am J Sports Med. 2009; 37(6):1150–4.
- Fredericson M, Jennings F, Beaulieu C, Matheson GO. Stress fractures in athletes. Top Magn Reson Imaging. 2006;17(5):309–25.
- 74. Long NM, Zoga AC, Kier R, Kavanagh EC. Insufficiency and nondisplaced fractures of the talar head: MRI appearances. AJR Am J Roentgenol. 2012;199:W613–7.
- Bischoff MJ, Reininga IH, van Raaij TM. Atraumatic bilateral insufficiency fractures of the talar neck in a rheumatoid patient. J Foot Ankle Surg. 2013; 52(2):231–4.
- Sormaala MJ, Niva MH, Kiuru MJ, Mattila VM, Pihlajamaki HK. Bone stress injuries of the talus in military recruits. Bone. 2006;39:199–204.
- Rosenberg GA, Sferra JJ. Treatment strategies for acute fractures and nonunions of the proximal fifth metatarsal. J Am Acad Orthop Surg. 2000;8:332–8.
- Torg JS, Balduini FC, Zelko RR, Pavlov H, Peff TC, Das M. 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.
- O'Malley MJ, Hamilton WG, Munyak J, DeFranco MJ. Stress fractures at the base of the second metatarsal. Foot Ankle Int. 1996;17(2):89–94.
- Smith JT, Halim K, Palms DA, Okike K, Bluman EM, Chiodo CP. Prevalence of vitamin D deficiency in patients with foot and ankle injuries. Foot Ankle Int. 2014;35(1):8–13.
- Miller TL, Harris JD, Kaeding CC. Stress fractures of the ribs and upper extremities: causation, evaluation, and management. Sports Med. 2013;43(8):665–74.
- Li X, Heffernan MJ, Mortimer ES. Upper extremity stress fractures and spondylolysis in an adolescent baseball pitcher with an associated endocrine abnormality: a case report. J Pediatr Orthop. 2010;30:339–43.