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Case Study

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A CASE REPORT OF PELVIC STRESS FRACTURE AND LITERATURE REVIEW

Rishat Ibtisham[1], Jiang Letao[2], Tauhid Sajjad[3], Du Jianwei*[4], Wu Shang[5], Quan Kai[6]

[1]MBBS, MS (Orthopedic Surgery), Medical College of Yangzhou University, Yangzhou, 225009 P. R. China Department of Orthopedics, Affiliated Hospital of Yangzhou University, Yangzhou, 225000 P. R. China.
[2]Department of Orthopedics, Affiliated Hospital of Yangzhou University, Yangzhou, 225000 P. R. China.
[3]Medical College of Yangzhou University, Yangzhou, 225009 P. R. China.
[4]Department of Orthopedics, Affiliated Hospital of Yangzhou University, Yangzhou, 225000 P. R. China.
[5]Department of Orthopedics, Affiliated Hospital of Yangzhou University, Yangzhou, 225000 P. R. China.
[6]Department of Orthopedics, Affiliated Hospital of Yangzhou University, Yangzhou, 225000 P. R. China.

***Corresponding Author: Dr. Du Jianwei**

Department of Orthopedics, Affiliated Hospital of Yangzhou University, Yangzhou, 225000 P. R. China.

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BACKGROUND

Stress fracture is a kind of continuous and repeated cyclic stress acting on the bone under the premise of bone fatigue, which is less than the limit of bone strength. Microscopic damage occurs in the bone and the microscopic damage eventually leads to fatigue fractures. Under normal circumstances, living bone can pass through Self-reconstruction to repair micro-damage, make the bone have good anti-fatigue performance. Therefore, bone is not prone to fatigue fracture. But When abnormal conditions occurs, such as long-term fatigue or weight-bearing of bones, a large number of microscopic damages may occur in the bones. When the micro-damages continue to accumulate, resulting in When bone repair cannot counteract this, stress fractures are induced.[1] The most common site for stress fractures is the tibia, followed by the tarsals, metatarsals, femur, and pelvis.[2] This article reports a case of a female patient with pelvic stress fracture caused by long-term physical labor and reviews relevant literature in order to provide reference for clinical diagnosis and treatment.

CASE REPORTS

A 69-year-old female patient was admitted to the hospital due to bilateral lumbar and lumbosacral pain for more than 1 month. She had no history of trauma and had a working history of squatting and moving heavy objects for more than 10 years. She had no underlying diseases. Examination showed bilateral lumbar and lumbosacral percussion pain, VAS score was 6 points, no obvious abnormality in muscle strength and tension of lower limbs, normal physiological reflexes and no positive pathological signs. Combined with the previous medical history and physical examination, the patient was considered to have a pelvic stress fracture caused by long-term stress concentration. CT, PET/CT, and MRI examinations (Figure 1-4) confirmed the existence of bilateral ilium fractures. Bone density tests showed that the patient Combined with osteoporosis. For this patient, we carried out non-surgical conservative treatment, including bed rest, symptomatic analgesia, injection of teriparatide, calcium supplementation and vitamin D. After 10 days, the patient's pain was significantly relieved compared with the admission, and the VAS score was 2 points. After discharge the patient, bed rest, oral calcium and vitamin D were continued. At a follow-up visit 6 weeks after discharge, the patient complained that the pain had been completely relieved.

DISCUSSION

Microscopic bone damage is a pathological change that widely exists in cortical bone and cancellous bone. The types of microscopic bone damage in histology mainly include: diffuse damage, neural network cracks and linear cracks.[3] There are two mechanisms that cause microscopic damage to bone: One is chronic fatigue injury caused by continuous load and the other is external impact. Stress damage caused by mechanical damage such as prosthesis or screw implantation. At the same time, muscle contraction and physiological load also act on the bone. The resulting tension can lead to more microscopic damage. Stress fractures occur when microscopic damage accumulates and the bone's ability to repair itself, that is bone remodeling, fails to offset the damage. OSTLIE et al.[4] formally proposed after research: Under the continuous and repeated action of small stress, the bone trabecular structure is destroyed. When these damages accumulate, once the reconstruction capacity of the bone is exceeded, it can lead to fractures at the damaged site. The most common types of microscopic bone damages are diffuse microscopic damage and linear microscopic damage. Linear damage is composed of linear cracks and diffuse micro-damage is composed of a large number of fine cracks cross-stacked,[5] but diffuse micro-damage is not generated on

the basis of linear micro-damage, they are two different types damage. Different stress conditions can cause bone microscopic damage in different shapes. Linear microscopic damage is mainly generated in the bone under pressure, while diffuse microscopic damage is usually generated in the bony unit under tension.[6] Different types of microscopic damage have different effects on the bio-mechanical properties of bone. Diffuse microscopic damage can prolong the life of bone, have a stronger ability to dissipate energy and minimize the damage to bone caused by stress.[7] Conversely, linear microscopic damage reduces bone strength and stiffness. SEREF-FERLENGEZ et al.[8] proved that diffuse microscopic damage can be directly repaired without bone reconstruction.

Stress fracture is responsible for approximately 20% of all sports medicine injuries and runners who run an average of more than 40 km per week are considered to be at higher risk for stress fractures[9] Due to the repetitive nature of military training, stress fractures are common among military personnel with an incidence of stress fractures in recruits ranging from 3% to 9%.[10] The most common stress fractures in descending order of incidence are the tibia (23.6%), tarsal (17.6%), metatarsal (16.2%), femur (6.6%), and pelvis (1.6%).[11] Due to lower Incidence rate, stress fractures of the pelvis are more likely to be overlooked in clinical practice. The clinical manifestations of pelvic stress fractures are atypical, often manifested as pain in the groin and hip joints and it is difficult to differentiate from adductor muscle strain, osteitis pubis or sacroiliitis. Patients often come to see a doctor with pain in areas such as the hip joint.

Plain X-rays are usually the first-choice examination to rule out fractures. However, it is difficult to find the lucent fracture line in the early stage of stress fracture on plain X-ray films[12] and usually it is not until 3 weeks after the fracture that abnormalities can be shown.[13] The most common radiographic abnormalities include shallow linear sclerosis (usually perpendicular to the trabeculae), periosteal thickening or sclerosis, cortical changes and lateral callus formation or endosteal thickening and narrowing of the marrow cavity. According to the recommendations of the American College of Radiology (ACR) for patients with suspected stress fractures, MRI is the gold standard for diagnosis when the X-ray results are negative. MRI has extremely high sensitivity and specificity in the detection of occult bone injury cases. It can show linear fracture lines with low signal on both T1W and T2W sequences, and can show adjacent bone marrow and adjacent bones on T2W sequences. Hyperintensity in soft tissue, the latter suggesting edema of bone and soft tissue.[14] Early diagnosis by MRI allows patients to avoid weight bearing as early as possible to prevent the trabecular bone in the fracture area from being compressed due to force, resulting in persistent pain or sequelae such as osteoarthritis.

According to local blood supply and load conditions at different locations, the predilection sites of stress fractures can be divided into three categories: low-risk, intermediate-risk, and high-risk.[15-16] If high-risk stress fractures occur in areas with high tensile load and insufficient blood supply (such as the tension side of the femoral neck, patella, anterior tibia, medial malleolus, talus, navicular, proximal fifth metatarsal, etc.), if not properly treated, Complications such as delayed fracture, complete fracture and nonunion may occur. Early identification and timely treatment of such high-risk stress fractures are crucial to improving the prognosis.[12] For stress fractures in high-risk areas, surgical treatment is an option when the risk of progression to nonunion is high or the patient cannot afford a longer recovery time for non-surgical treatment.[17-19] For most stress fractures with poor or failed non-surgical treatment, surgical treatment has a higher rate of symptom relief, faster recovery of exercise capacity, and better results. Low-risk stress fractures can be found in the posterior tibia, 2nd to 4th metatarsals, femur, superior and inferior pubic ramus, sacrum, and fibula and a relatively good prognosis can often be obtained with rest and conservative treatment.

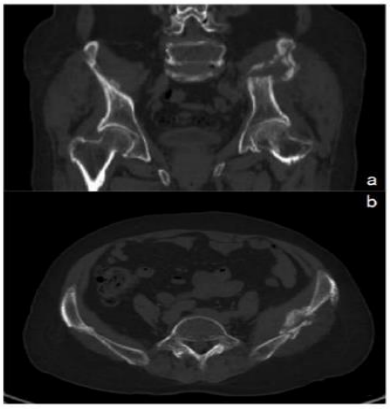
Biotherapy is a treatment method that uses various biological materials that can promote fracture healing, including bone marrow mesenchymal stem cell technology, platelet-rich plasma technology, recombinant human parathyroid hormone technology etc. which have emerged in recent years. Although the application of biological therapy to promote fracture healing has a good application prospect, due to its immature development and few human trials, more clinical trials are still needed to verify its practical application. Bone marrow mesenchymal stem cells play a key role in bone repair and regeneration.[20-21] Bone marrow mesenchymal stem cells have the ability of immunoregulation and osteogenic differentiation and have broad application prospects in the treatment of refractory fractures, but there are still some uncertainties that may limit their application. For example, their use in the repair of bone damage caused by tumor surgery is still unknown there is controversy. In addition, the number and timing of bone marrow mesenchymal stem cell transplantation still need further study.[22] Mousaei et al.[23] conducted experimental verification in rat nonunion models and confirmed that transplanting cancellous bone and chitosan hydrogel can promote the osteogenesis process by inducing bone marrow mesenchymal stem cells to secrete a variety of growth factors, which is the basis for cell growth. Provides a good environment for attachment and growth. At present, there are still few studies on bone marrow mesenchymal stem cells, and most of them are based on animal models. There are few high-quality clinical trials for humans, especially cost-effectiveness studies of treatment. Platelet-rich plasma has good prospects in promoting fracture healing.[24] Guzel et al[25] experimentally confirmed that the application of platelet-rich plasma can promote fracture histological

healing and improve the biomechanical properties of fracture healing in rat femoral fracture model. Roffi[26] and other studies believe that although the current studies all suggest the effectiveness of platelet-rich plasma in bone healing, more high-quality research evidence is still needed. Parathyroid hormone is an important hormone involved in bone mineral homeostasis and various metabolic processes in the human body. It can promote the generation of osteoblasts, stimulate bone formation and accelerate bone healing.[27] Teriparatide is a 1-34 amino acid fragment of the N-terminal of the parathyroid molecule produced by recombinant DNA technology. Baillieul et al[28] used teriparatide to treat a 36-year-old long-distance runner diagnosed with sacral stress fracture and achieved good results. Bakr et al.[29] conducted a randomized controlled trial on fracture model rats and the results showed that intracorporeal injection of parathyroid hormone can increase the production of osteoclasts in the second week of stress fracture. Therefore, teriparatide can be used in the treatment of stress fractures but the dosage and timing of application still need further research to confirm.

Bisphosphonates can inhibit osteoclast-mediated bone resorption, so they may play a role in the treatment and prevention of stress fractures. However as of now, there is no conclusive evidence that bisphosphonates have any positive effects on stress fracture healing in humans, so further trials are needed to confirm their efficacy and safety.[30-31] Miller et al[32] put forward a holistic strategy for the treatment of stress fractures, that is, individualized treatment plans, including nutrition, endocrine, etc. Psychology professional biomechanical equipment and physical therapy, etc. to achieve the balance between the generation and repair of bone microcracks.

The prevention of stress fractures is mainly the prevention and correction of various risk factors that cause stress fractures. In the prevention of stress fractures, both extrinsic and intrinsic risk factors must be considered. Extrinsic risk factors include previous physical inactivity, sudden increase in training load, footwear discomfort, insufficient vitamin D/calcium intake, etc. Intrinsic risk factors Including gender, race, age, height, body mass index (BMI), hormone levels, dietary habits, low bone mineral density, previous history of stress fractures, muscle fatigue, etc.[2,14,16,33-35] The recommended daily intakes of calcium and vitamin D are 1000 mg and 800-1000 U, respectively.[36] However, some researchers believe that the intake of the two should vary from person to person and recommend a daily intake of 1200-1500 mg of calcium and 800-3000 U of vitamin D.[37-38] McCabe et al[39] suggested that risk groups should take 800-1000 U (up to 2000 U) of vitamin D and 1000-1200 mg of calcium daily to prevent stress fractures. In a study of female Navy recruits, those given 2000 mg of calcium and 800 U of vitamin D per day had a 21 percent reduction in the incidence of stress fractures compared with a control group.[40] These results strongly support calcium and vitamin D supplementation as an effective preventive measure to reduce the incidence of stress fractures. Although the preventive effect of vitamin D on stress fractures is still divided, the prevention of stress fractures by calcium and vitamin D supplementation is still supported by most studies.[34,41-42] At the same time, studies have confirmed that vitamin D supplementation alone cannot prevent fractures and vitamin D supplements taken together with calcium can effectively prevent stress fractures.[39] Low bone mineral density is an independent risk factor for prolonged healing of bone stress injuries.[43] Studies have confirmed that estrogen replacement therapy has a positive effect on bone mineral density.[44] However, since low energy utilization is a potential cause of low bone mineral density and bone stress injury, some researchers have suggested restoring energy utilization as the main intervention for the treatment of low bone mineral density.[45] The study by Hughes et al[46] showed that the risk of stress fractures in military personnel who took NSAIDs increased by 1.9 times, especially during the basic training of recruits, the risk of stress fractures occurred in those who took NSAIDs increased more than 4 times. Studies have found that the use of non-steroidal anti-inflammatory drugs is a risk factor for stress fractures.[47] Therefore, cautious use of NSAIDs is also one of the important measures to prevent stress fractures.

In summary, pelvic stress fracture is a rare type of stress fracture, which often manifests as groin and hip pain and needs to be differentiated from sacroiliitis. MRI is considered the gold standard for the diagnosis of the disease and non-surgical treatments such as bed rest and teriparatide injection can obtain a better clinical prognosis but a large number of studies are still needed to further confirm its effectiveness.



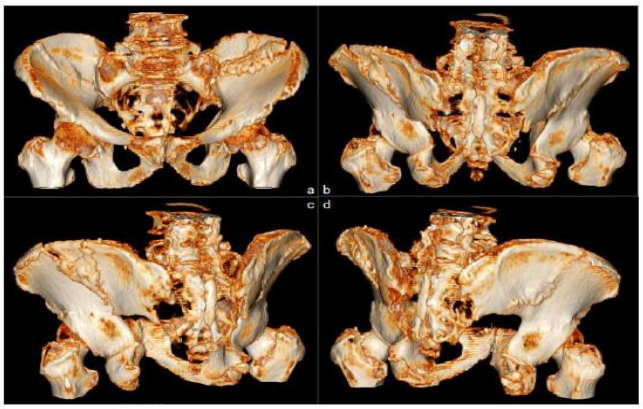
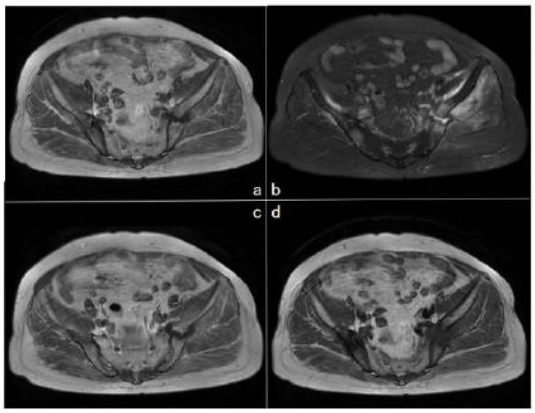


Figure 1 **Figure 2**

Figure 1: CT plain scan, a coronal view, b transverse view showing bilateral ilium fractures.
Figure 2: CT three-dimensional reconstruction shows bilateral ilium fractures.



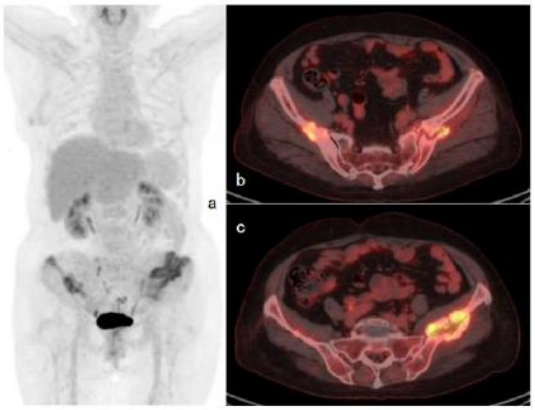


Figure 3 **Figure 4**

Figure 3: MRI bilateral ilium with high signal on T1 sequence and high signal on T2 lipid-suppressive sequence. Bilateral iliacus, gluteus, pectineus, and intermuscular soft tissue swelling, high signal on T1 and T2 sequences.
Figure 4: PET/CT showed continuous interruption of bilateral iliac bone cortex with local callus formation and abnormally increased radioactive uptake.

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