

Systemic Bone Infarction in Middle-Aged Women: A Case Report

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Abstract: Objective: Report a complete case of rare multiple bone infarction in a 42-year-old woman, including diagnosis, treatment and follow-up results, so as to improve the understanding of the diagnosis and treatment of systemic multiple bone infarction. Methods: With the help of kirschner needle, the medulla of bilateral osteonecrosis of the femoral head was decompressed, the cystic cavity and bone graft were treated and covered with cortical bone slices of corresponding size. Drilling decompression of bilateral femur and bilateral tibia. Remove the dead bone in the osteonecrosis area caused by bone infarction, reduce inflammation, and take the dead bone for pathological examination. Results: The distension of both lower limbs, the pain and discomfort basically disappeared, the food was OK, the second stool was adjusted, and the sleep was normal; physical examination: the wound dressings of the lower limbs were well bandaged, the dressings were dry, no obvious exudation was found, the local tenderness was positive, the muscle strength of both lower limbs was normal, and the peripheral blood circulation was normal. Conclusion: The operation can effectively and thoroughly cure the focus, improve the blood circulation, reduce the pressure, and provide mechanical support to prevent fracture. The new bone graft provides a scaffold for the growth of neovascularization.

1. Introduction

Bone infarction, also known as bone marrow infarction, bone fat infarction, refers to the osteonecrosis that occurs in the metaphysis and diaphysis, mostly in the lower femur, upper tibia and upper humerus, showing multiple and symmetrical changes, which is often secondary to decompression sickness, and may also be related to the extensive use of hormones and

immunosuppressant's, alcoholism, trauma, pancreatitis and so on[1]. There may be clinical manifestations such as limb numbness, dullness, joint pain, weakness and so on. Because the disease is hidden, the symptoms are not typical, and there are basically no abnormal signs in the early X-ray examination, it is not easy to diagnose accurately[2]. The incidence of bone infarction is low, accounting for about 7/10000 of the population. Systemic bone infarction is rarer. This case reports a middle-aged woman with multiple bone infarctions.

2. Materials and Methods

2.1. General Information

A 42-year-old female was admitted to hospital with pain in her left knee a week ago, which was aggravated for 2 days. Symptoms can't be relieved after the rest. Magnetic resonance imaging of the left knee joint in the hospital showed that there was a bone infarction in the left lower femur and upper tibia, and a small amount of joint cavity effusion in the left knee joint. He was admitted to hospital with "necrosis of the left femoral head". Specialist condition: there was no obvious swelling of bilateral hip joint, equal length of both lower limbs, flexion of right hip joint 30°, passive flexion and extension, adduction and abductor movement disturbance, flexion and extension of left hip joint about 90°-20°, internal and external rotation about 10°-20°, passive range of motion was obviously limited, and left lower limb longitudinal axis percussion pain was positive. Bilateral hip flexion test (+), Patrick sign(+), no deformity of left knee joint, slight swelling, normal skin color, positive medial tenderness of knee joint, normal skin temperature, positive floating patellar test, limited movement of left knee joint and normal peripheral blood circulation of left lower limb. There was no obvious tenderness in both knee joints, floating patellar test (-), the skin of both lower limbs felt normal, the muscle strength of each muscle group was normal, and the dorsalis pedis artery could pulsate. The physiological reflex of both lower limbs existed, but the pathological reflex was not induced. The patient had a history of chronic atrophic gastritis for 7 years, no history of hypertension, coronary heart disease, diabetes and other medical diseases, no history of infectious diseases such as hepatitis and pulmonary tuberculosis, no history of operation, trauma, no history of blood transfusion, drug and food allergy. Auxiliary examination: the left femur, tibiofibula positive and lateral DR showed that the bone mineral density of the lower part of the left femur was decreased, the boundary of the focus was unclear, and bone infarction was considered. (Figure 1)



Figure 1: DR: The bone mineral density of the lower part of the left femur is decreased, the boundary of the focus is unclear, and bone infarction is considered.

MRI of the left tibia and femur showed abnormal signal in the medullary cavity of the left femur and tibia, and bone infarction was considered. Avascular necrosis of the left femoral head may be caused by effusion in the cavity of the left hip joint. MRI of the right tibia and fibula indicated that the right tibia and fibula were infarcted with a small amount of effusion in the right knee joint

cavity. CT of the hip joint shows the ischium island on the left side. (Figure 2)



Figure 2: MRI: The signal in the bone marrow cavity of the left femur was abnormal and bone infarction was often considered. Avascular necrosis of the left femoral head may be caused by effusion in the cavity of the left hip joint. A small amount of effusion in the right knee joint cavity.

The diagnosis was bilateral osteonecrosis of the femoral head, multiple bone infarction, osteoarthritis of both knee joints and injury of the posterior horn of the meniscus of the right knee joint. Improve the relevant preoperative examination, after medical discussion to determine the mode of operation is bilateral femoral head necrosis medulla decompression, bilateral, bilateral femur, bilateral tibia drilling decompression.

2.2. The Procedure of the Operation



Figure 3: The skin length of the lateral incision of the distal femur on the right side is about 3cm, exposing the bone cortex, and drilling through the lateral cortex to the medullary cavity at an angle of about 45 degrees between the drill bit and the femoral neck, so that the opening reaches the site of bone infarction and the blood flow of bone marrow at the site of infarction is poor, and part of the infarcted bone marrow is removed for pathological examination.

(1) After general anesthesia was satisfied, the towels were routinely disinfected. Under the guidance of C arm, kirschner wire were opened on the lateral side of the femoral neck. Fluoroscopy showed that the position of the kirschner wire on both sides of the femoral neck was satisfactory. The skin length was about 5cm on the outside of the guide needle. The hollow drill was drilled into the femoral neck with a hollow bit and the position of re-fluoroscopy was satisfactory. (2) Under the guidance of fluoroscopy, two long guide needles were drilled into the bone marrow cavity of the bilateral femoral bone marrow cavity from the outside of the great trochanter. Make the guide needle near the osteolysis of the femur and open the femoral bone marrow cavity. (3) Under the guidance of fluoroscopy, two guide needles are inserted through the tubercle of the tibia to reach the distal end of the tibia, and the fluoroscopic guide needle is inside the bone marrow cavity. (4) Cut the skin on the outside of the distal end of the left femur to expose the bone cortex and drill the lateral cortex to the medullary cavity at an angle of about 45 degrees between the drill bit and the

femoral neck, so that the blood flow of the bone marrow is poor at the site of bone infarction and visible infarction, and part of the infarcted bone marrow is removed for pathological examination (Figure 3). (5) After washing the wound, suturing the wound, bandaging, and returning to the ward at the end of the operation.

3. Results of Postoperative Treatment and Follow-Up Evaluation

3.1. Postoperative Treatment

Return to the ward after operation, give secondary nursing after orthopedic general anesthesia, light and digestible diet, stay with others, continuous ECG monitoring, blood oxygen saturation monitoring, continuous oxygen inhalation, guided functional exercise, nutritional status assessment, manual treatment plan, function of daily living evaluation, wound dressing change every other day, treatment with propyl gallate to improve local blood circulation. Vitamin nutrition support treatment, traditional Chinese medicine smear to prevent thrombosis, after the symptoms gradually alleviated.

3.2. Results

The patient self-reported: the distension of both lower limbs, the pain discomfort basically disappeared, the intake was OK, the second stool was adjusted, and the sleep was normal; physical examination: the vital signs were stable, there was no obvious abnormality in cardiopulmonary auscultation, and no obvious abnormality in abdominal palpation. The wound dressing of the lower extremities was well bandaged, the dressing was dry, no obvious exudation was found, the local tenderness was positive, the muscle strength and muscle tension of both lower limbs were normal, and the peripheral blood circulation was normal.

4. Discussion

4.1. Clinical Features

Bone infarction refers to the osteonecrosis that occurs in the diaphysis and metaphysis. It usually occurs in the hip, knee joint and the shoulder joint. There are usually no clinical symptoms, and most of them are found by accident during imaging examination[2].

4.2. The Etiology of Bone Infarction

(1) The formation of thrombus caused by vascular blockage: in the blood supply system, the reduction of the blood supply channel and the number of circulation of bone will lead to certain blockage of blood vessels, and then the formation of thrombus. The bubbles in the thrombus will have a certain impact on bone infarction; (2) Traumatic damage of the body: due to valgus violence, excessive flexion and extension violence on the sagittal plane and axial vertical and torsion violence, the blood supply of the bone cortex of the human body is reduced, and the blood flow of collateral circulation is also less, which is also easy to cause bone infarction. (3) Excessive use of glucocorticoid drugs: studies have found that taking too much glucocorticoid will lead to bone infarction, because glucocorticoid has an inhibitory effect on blood circulation, which leads to blockage. (4) The vascular wall is damaged to a certain extent: when the vascular wall is damaged, the amount of bone blood will be reduced, resulting in local bone infarction in the patient. (5) Venous pressure blockage: if venous pressure > arterial pressure, the venous blood flow will

increase, and the arterial tissue will appear ischemia, which can easily lead to bone infarction. 6 There is a disorder in the metabolism of oxygen free radicals: some studies have shown that the disorder in the metabolism of oxygen free radicals will lead to the denaturation of proteins, which will accelerate the degradation of proteins, lead to the damage of proteins between bones, and finally, lead to bone infarction[3].

4.3. Basic Pathological Changes

The pathological process can be divided into cellular necrosis stage and bone repair stage: cellular necrosis is the interruption of blood supply of bone tissue and the death of bone cells. Bone marrow hematopoietic tissue is very sensitive to hypoxia, the first is the death of bone marrow cells (6h-12h), then the osteocytes, osteoclasts and osteoblasts (12h-48h), and finally the adipocytes necrosis of bone marrow adipocytes (2h-5d). The necrosis of bone marrow adiposity is the change at the end of bone infarction. After the occurrence of bone infarction, it enters the stage of bone repair, including vascular regeneration, granulation tissue formation, dead bone resorption and new bone formation. Angiogenesis is the beginning of bone repair, absorption of dead bone, formation of fibrous connective tissue and dense new bone is the late stage of bone infarction. There are three basic pathological changes in the evolution of bone infarction, namely, dead bone mass, resorption zone (hyperemia, edema zone) and new bone zone, which are the basis of X-ray, CT, MRI and other imaging diagnosis of bone infarction.

4.4. Diagnosis

The diagnosis of bone infarction mainly includes: (1) Clinical manifestations: the common symptoms are pain in bone diaphysis or metaphysis, mainly dull pain or distension pain. There were basically no abnormal signs in the early stage, but local deep tenderness, adjacent joint effusion and movement disturbance occurred in the late stage. Fever and redness and swelling can occur in patients with acute bone infarction. (2) Imaging examination: X-ray examination of early bone infarction has no specific signs. It is suggested that MRI examination should be performed in patients with suspected bone infarction in order to improve the accuracy of diagnosis. (3) Pathological examination: for the cases which are difficult to be diagnosed, the diagnosis can be confirmed by puncture biopsy or surgical pathological examination. Although pathological examination is not necessary for the diagnosis of most cases, it is an important method to distinguish it from some diseases. (4) Pay attention to the possibility of bilateral disease.

4.5. Imaging Features

The differential diagnosis of bone infarction in imaging is based on the results of early X-ray and CT examination. The results show that the result of bone infarction is relatively late, and the diagnosis of it can only be limited by negative results, osteoporosis and wait for the results for a long time (30 days)[4]. The results of X-ray and CT examination in the middle and late stage showed that the bone infarction in this period had certain differential characteristics, such as the shape of the bone infarction was patchy and cord-like, and the bone was calcified and the bone mineral density changed to some extent. On the other hand, the reactive new bone showed homogeneous and non-structural ossification, while there were different ossification foci in the medullary cavity in the late stage. Clinically, CT has the advantages of sensitivity, clarity and obvious structure. However, X-ray and CT examination cannot make an accurate diagnosis of early infarction. Therefore, MRI is non-invasive and sensitive, and it can be determined that bone infarction belongs to the early stage by the change of abnormal signal. On the other hand, bone

infarction in different periods will show different foci, which can be single or multiple, irregular shape and fusion trend, showing a typical "map plate", and there are often small atypical lesions around the lesions. The early manifestations of bone infarction: the lesions showed diffuse small spotted short T1 signal, and when the fat suppression sequence signal decreased, the non-traumatic bone marrow edematous changes, and there was a certain correlation between them, while the main manifestations of bone infarction in the middle and late stage were as follows: the central part of the infarction and the surrounding normal bone marrow showed moderate or slightly low signal intensity on T1WI, while the edge of the infarction was a tortuous low signal band. On T2WI, the signal intensity of the central part was still similar to or slightly higher than that of the adjacent bone marrow tissue, while the periphery showed a circuitous high signal band, while the T1WI and T2WI changes of the lesion edge signal coincided with the pathological basis of hyperemia and edema at the edge of the lesion. Moreover, in the later stage of bone infarction, the periphery of the focus showed low signal intensity on T1WI and T2WI, which indicated that fibrosclerosis or calcification appeared on the edge of the focus, and when the liquefaction necrosis occurred in the center of the focus, there would be long T1 and long T2 signals with uneven signal intensity; therefore, when MRI showed typical signs, it was generally easy to diagnose, but when it showed different typical signs, it should be distinguished from other diseases in time. The imaging findings of [5] MRI are also varied in different stages of bone infarction[6, 7].

Bone infarction is divided into acute phase, subacute phase and chronic phase, the MRI findings are as follows: (1) acute stage: the lesion center T1WI is equal to or slightly higher signal intensity than normal bone marrow, T2WI is high signal intensity, margin is long T1 signal, long T2 signal intensity; (2) subacute stage: lesion center T1WI is similar to normal bone marrow or slightly low signal intensity, T2WI is similar to normal bone marrow or slightly high signal intensity, margin is long T1, long T2 signal intensity. (3) chronic phase: both T1WI and T2WI showed low signal intensity. The above stages correspond to the early, middle and late stages of this group (Figure 4).

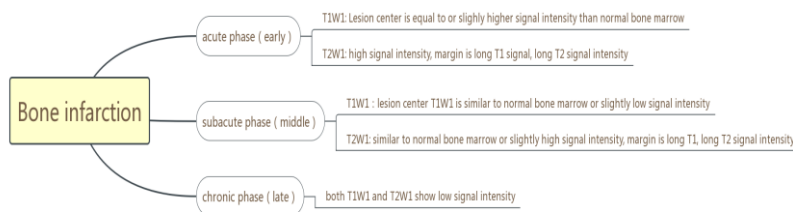


Figure 4: The Stages of bone infarction

4.6. Differential Diagnosis

It is difficult to distinguish this disease from ependymoma, and spectral analysis may be helpful to distinguish the two. The disease also needs to be differentiated from intraventricular meningioma. The CT plain scan density of intraventricular meningioma is high density, which is similar to that of this patient, but the enhanced scan is more obvious and more uniform, often causing obstructive hydrocephalus. The diagnosis of typical glioblastoma is not difficult, but it is difficult to diagnose atypical glioblastoma only by conventional CT and MRI plain scan and enhanced scan. For patients with difficult diagnosis, functional MRI sequences such as DTI and MRS can be supplemented on the basis of routine examination, which will be helpful for differential diagnosis and grading of glioblastoma, but the final diagnosis still depends on histopathology.

4.7. Treatment

4.7.1. Conservative Treatment

The treatment of patellar infarction is mainly conservative treatment, including observation and oral non-steroidal anti-inflammatory painkillers and so on. For the patients with obvious pain symptoms, poor analgesic effect of oral drugs, easy to recur after drug withdrawal and a wide range of lesions, we recommend surgical treatment[8]. Clinical pain symptoms of some patients are not obvious, but imaging examination found that the focus invaded the articular subchondral bone. If there is no surgical intervention, weight-bearing activities may lead to pathological fractures or joint dysfunction caused by articular surface collapse. In addition, a very small number of bone infarcts also have malignant transformation or are associated with the occurrence of malignant tumors, [9] Although the number of this association is small, it seems that the prognosis of this association may be more or less the same as those associated with osteosarcoma, fibrosarcoma or malignant fibrous histiocytoma, in which the survival rate is unfavorable[10, 11]. There may be a causal relationship between bone infarction and the follow-up development of osteosarcoma[12]. It should be treated by operation as soon as possible[13].

4.7.2. Surgical Treatment

It is feasible to. The focus of the femoral neck can be removed by window. The focus was thoroughly scraped, the cystic cavity was treated, and the bone graft was covered with cortical bone of the corresponding size, so as not to cause cortical bone defect in the femoral neck and reduce the strength of the femoral neck. Bone grafting is the first choice for the residual cavity. if there is no sufficient bone source, antelope-based apatite or tricalcium phosphate can be chosen, and bone cement is also a good substitute, which not only has a good supporting effect, but also the high temperature produced during its solidification can kill the residual tumor cells. Surgical treatment can achieve the following purposes: 1. Clear the focus and open the medullary cavity and remove the barrier of blood circulation. 2. Reduce the pressure in the medullary cavity. 3. Bone graft filling to repair the defective area after focus clearance to provide mechanical support. 4. The implanted bone can provide a stent for the growth of neovascularization. Through surgical treatment, we can promote the revascularization of the diseased site, accelerate the formation of new bone, fundamentally relieve pain, and restore the normal shape and strength of bone (Figure 5).

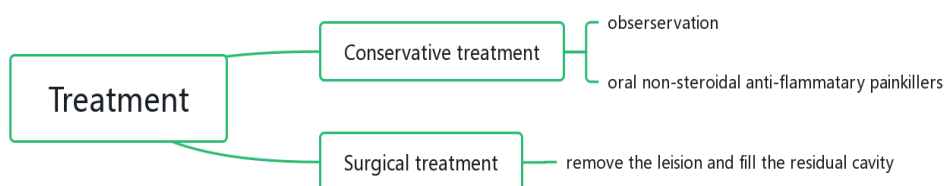


Figure 5: The Treatment of bone infarction

5. Summary

In a word, the clinical manifestations of bone infarction are not typical, the imaging findings are different in different periods, and there are even no specific signs in early diagnosis, so it is not easy to make a correct diagnosis. Familiarity with the imaging findings of bone infarction is essential to the diagnosis of bone infarction. Surgical treatment of bone infarction patients with obvious pain and the risk of fracture or articular surface collapse can achieve better results. The prognosis of bone infarction itself is very good (the risk of malignant transformation is very low), which is

usually a sign of systemic ischemic necrosis. Therefore, it is necessary to investigate the known risk factors and other ischemic necrotic foci in patients with bone infarction, which may have a threatening effect on function [13].

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