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CASE REPORT: AVASCULAR NECROSIS

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ABSTRACT

Background: Avascular necrosis (AVN) can be defined as bone cell death resulting from impaired blood flow to the bone due to traumatic or non-traumatic events. **Case Presentation**: We present a case in which a 62-year-old female came to the Orthopedic Surgery Polyclinic with complaints of pain in her groin. The pain is intermittent and worsens when the patient moves his legs. This makes it difficult for patients to sit and have pain when walking, so they need a cane as a tool. This complaint has been felt for ± 1.5 years and has been getting worse since this 1 month. During the pain, the patient said he was taking pain relievers but his complaints did not decrease. On local examination in the left Hip Joint region, it was found that on look examination there was no visible deformity, swelling, and skin color that was the same as the surrounding area. On examination of the feel found positive tenderness. On AP pelvic X-ray examination, there was a fracture of the left femoral head with superior displacement. Treatment for this patient includes non-surgical and surgical (total hip replacement). **Conclusion**: The patient was diagnosed with Avascular necrosis and treated with total hip replacement surgery. The goal is to relieve pain and allow the patient to walk normally.
Keywords: *Avascular necrosis; Total hip replacement; Hip join.*

INTRODUCTION

The death of bone cells due to a disruption in blood flow to the bones as a result of traumatic or non-traumatic occurrences is known as avascular necrosis, also known as osteonecrosis or aseptic necrosis[1]. The femoral head of the hip joint is where avascular necrosis most frequently manifests itself, although it can also occur in other anatomical sites, including the shoulder, knee, and ankle joints[2].

Despite the fact that several nations have screened their populations of people with osteonecrosis, there have been no epidemiological reports of AVN to yet. According to estimates, 20,000 new cases of osteonecrosis are detected in the US every year, and between 300,000 and 600,000 people have AVN overall. In Japan, between 12,000 to 24,000 additional cases of avascular necrosis were identified in recent years. In South Korea, the prevalence rate was 20.53 cases per 100,000 people in 2002, but by 2006, it had increased to 37.96 instances per 100,000 people, with an estimated 14,103 new cases on average year. The first comprehensive epidemiological study of nontraumatic osteonecrosis in China revealed that there were an estimated 8.12 million nontraumatic AVN patients overall, with men significantly more likely to have the disease (1,02%) than women (0.51%) and urban residents more likely to have it than rural residents. High-risk factors for nontraumatic osteonecrosis include glucocorticoids, alcohol, high blood lipid levels, obesity, certain vocations (like diving), smoking, and diabetes[3]. The median age of occurrence of osteonecrosis is 47 years, with a male-to-female ratio of 3:1[4].

Impaired subchondral microcirculation, which eventually results in bone necrosis, characterizes osteonecrosis. Because there is no bone-rebuilding mechanism, microfracture accumulation leads to the collapse of the subchondral bone. The predominant clinical symptoms are discomfort extending from the groin to the ipsilateral buttocks and knee, and they are extremely specific. Activity-induced pain rises with activity and falls with rest. The hip joint's range of motion is also restricted, particularly for hip abduction and internal rotation. It can hurt to logroll (passive internal and exterior rotation). Therefore, the current history is crucial to raise the possibility of osteonecrosis and to look into the potentially affected opposite side[3]. The purpose of this case report is to examine osteonecrosis of the femoral head as a whole to facilitate the diagnosis and treatment of this disease.

CASE REPORT

A 62-year-old woman came to the orthopedic polyclinic at Cut Meutia General Hospital with complaints of pain in her groin. The pain is like stabbing, is intermittent, and gets worse when the

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patient moves his legs. This makes it difficult for patients to sit and have pain when walking, so they need a cane as a tool. This complaint has been felt for 1.5 years and has been getting worse since one month before admission to the hospital. During the pain, the patient said he was taking pain relievers and had gone to a massage therapist, but his complaints had not diminished. denied the history of fever, denied the history of falling, and denied the history of trauma.

On physical examination, the general condition looked moderately ill: composed consciousness, blood pressure 117/69 mmHg, pulse 75 times per minute, respiratory rate 23 times per minute, and body temperature 36.5^0C. On local examination in the left hip joint region, it was found that there was no deformity, no swelling was seen, and the skin color was the same as the surrounding area. On examination, there was tenderness. On examination of the movement, we found limited ROM. On supporting examination and AP pelvic X-ray, there was good bone structure and trabeculation, smooth articular fascies, Sbenton and symmetrical skinner lines, joint space not narrowed, and a fracture of the left femoral head with displacement superiorly.

Treatment for these patients includes surgical and non-surgical. In non-surgical management, patients are given fluid therapy, antibiotics, analgesics and supportive therapy. Meanwhile, during surgery, the patient underwent a Total Hip Replacement procedure.

DISCUSSION

It was reported that the case of Mrs. JH, 62 years old, came to Cut Meutia General Hospital with complaints of groin pain. The pain is like stabbing, is intermittent, and gets worse when the patient moves his legs. This makes it difficult for patients to sit and have pain when walking, so they need a cane as a tool. This complaint has been felt for 1.5 years and has been getting worse since one month before entering the hospital. During the pain, the patient said he was taking pain relievers and had gone to a massage therapist, but his complaints had not diminished. denied the history of fever, denied the history of falling, and denied the history of trauma.

Avascular necrosis, also known as osteonecrosis or aseptic necrosis, is bone cell death caused by impaired blood flow to the bone[1]. Avascular necrosis most commonly occurs in the hip joint (femoral head), but can also occur in other anatomic locations (such as the shoulder, knee and ankle joints)[2]. This can be caused by trauma factors such as fractures or non-traumatic factors such as excessive use of steroids and alcohol, hemoglobinopathy, etc[5].

Steroid use is one of the most common nontraumatic causes of avascular necrosis[6]. Several studies have reported that prolonged use of steroids induces an estimated 9-40% incidence of avascular necrosis. However, patients usually have several other risk factors[2]. Administration of steroids can trigger vasoconstriction and cause an increase in the production of procoagulant factors. It also increases adipogenesis, decreases osteogenesis, and downregulates bone repair and remodeling[4]. Steroids can interfere with blood flow to the femoral head. The effect of steroids on blood vessels is to trigger vasoconstriction. This happens because steroids increase the response of blood vessels to endothelin-1 and decrease the response to bradykinin. Endothelin-1 functions to trigger vasoconstriction while bradykinin causes vasodilation. The vasoconstriction that occurs makes the blood supply insufficient, which then falls into an ischemic state and triggers the death of bone cells[7].

Corticosteroids and alcohol can increase the differentiation of mesenchymal stem cells into adipocytes. They also induce adipocyte hypertrophy through increased synthesis of intracellular lipids. An increase in the number and volume of adipose cells in the bone marrow can induce intraosseous hypertension in the proximal femur. The venous sinusoids are compressed due to intra-osseous hypertension, and intravascular coagulation occurs. Then, arterial blood flow is occluded, and ischemia occurs at the femoral head. Thus, Avascular necrosis Corticosteroid-and alcohol-associated femoral head is a type of intra-osseous compartment syndrome within the femoral head due to an ischemic cascade of: 1) marrow fat cell hyperplasia; 2) intra-osseous hypertension; 3) vascular compression and intravascular coagulation; 4) blood flow disorders; 5) marrow necrosis and osteocytic death; and 6) fibrovascular reparative process around the necrotic zone and necrotic marrow saponification[8].

On physical examination, the general condition looked moderately ill: conscious somnambulism, blood pressure 117/69 mmHg, pulse 75 times per

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minute, respiratory rate 23 times per minute, and body temperature 36.5°C. On local examination in the left hip joint region, it was found that on visual examination, there was no visible deformity, no visible swelling, and the skin color was the same as the surrounding area. On examination, there was tenderness. On examination of the movement, we found limited ROM. The most common complaint in patients with avascular necrosis is pain, especially localized in the hip, buttocks, or groin area. Pain worsens with activity and improves with rest. It is sometimes accompanied by knee pain and limited internal rotation of the hip[3]. The pain may be mild at first but gets worse over time. Ultimately, the pain is present at rest and worsens even at night; morning stiffness may also occur. Limited joint motion: if the joint surface is damaged, pain can increase dramatically. The pain may be severe enough to limit the patient's range of motion in the affected joint, resulting in a reduced range of motion (ROM) on physical examination with both active and passive motion. If this avascular necrosis affects the legs, the person becomes lame and the muscles become atrophic and can shorten. Movement is limited, especially abduction and internal rotation[3,9]. This is in accordance with the complaints experienced by patients.

On investigation, an AP pelvic radiograph showed good bone structure and trabeculation, smooth articular fascies, Sbenton and Skinner lines that were symmetrical, a joint space that was not narrowed, and a left femoral head fracture with superior displacement. In avascular necrosis, the joint space remains normal until the end (because avascular necrosis is not a primary disease of the joint cartilage). X-ray manifestations are usually osteosclerosis, cystic changes, and a "crescent sign" in the early stages. After collapse, there is loss of femoral head roundness and degenerative arthritis in the late stages[3,9].

X-ray imaging: Early signs of ischemia are confined to the bone marrow and cannot be detected by a plain x-ray examination. X-ray changes, when present and sometimes appearing before 3 months after ischemia, are due to reactive new bone formation at the junction of the necrotic area and trabecular failure in the necrotic segment. An area of increased radiographic density is seen in the subchondral bone; soon after, a thin tangential fracture line appears just below the joint surface—the 'crescent sign'. In advanced stages, there is more pronounced distortion of the joint surface and sclerosis, which is caused by compression of the bone in the collapsed segment. Sometimes the necrotic part separates into separate fragments. The joint space is still normal because the joint cartilage is not damaged until it is very advanced. This is what distinguishes primary avascular osteonecrosis from sclerotic and destructive osteoarthritis. The anteroposterior and frog leg positions are the basic X-ray positions used for the diagnosis of osteonecrosis of the femoral head, and X-ray manifestations are usually osteosclerosis, cystic changes, and a "crescent sign" in the early stages. After collapse, there is loss of femoral head roundness and degenerative arthritis in the late stages[5,9].

This patient's care includes both surgical and non-surgical options. The planned surgery is a total hip replacement. The patient also gets Ringer's lactate as fluid therapy, its function is to replace fluids lost during surgery and maintain hydration during hospitalization. Cefotaxime and Gentamicin as an antibiotic, to prevent surgical wound infection. Ketorolac as an analgesic, its function is to reduce pain. Ranitidine as supportive therapy.

Operative treatment modalities for avascular necrosis include core decompression, osteotomy, bone grafting, and arthroplasty. Arthroplasty is usually reserved for patients with late-stage femoral head osteonecrosis, as well as older patients and those with more advanced arthritis. Arthroplasty is a treatment that has been proven to reduce pain and restore mobility to the patient. Advances in the last two decades, with the advent of low-wear cushioning surfaces, have shown promising results when used in patients with advanced stages of necrosis. Total hip replacement or total hip arthroplasty is usually the treatment of choice for late-stage avascular necrosis or when the joint is destroyed. This procedure involves replacing damaged cartilage and bone with an artificial implant. This therapy gives excellent results by reducing pain in the long term and allowing early mobilization[3].

CONCLUSION

One case of avascular necrosis has been reported in a 62 years old woman. The diagnosis is based on anamnesis, the main complaint is pain in the

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groin. The pain is like stabbing, intermittent and gets worse when the patient moves his legs. This makes it difficult for patients to sit and have pain when walking, so they need a cane as a tool. While feeling pain the patient said he was taking pain relievers. but his complaints did not diminish. Denied history of fever, denied history of falling, denied history of trauma.

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Treatment for these patients includes surgical and non-surgical. In non-surgical management, patients are given fluid therapy, antibiotics, analgesics and supportive therapy. Meanwhile, during surgery, the patient underwent a Total Hip Replacement procedure.

REFERENCES

1. Kabra D, Jena S, Bhatted SK, Dharmarajan P. Avascular Necrosis of Femoral Head - Ayurveda Management : A Case Study. Int J Heal Sci Res. 2020;10(11):22–7.
2. Lespasio MJ, Sodhi N, Mont MA. Osteonecrosis of the Hip: A Primer. Perm J. 2019;23:1–7.
3. Zhao D, Zhang F, Wang B, Liu B, Li L, Kim SY, et al. Guidelines for clinical diagnosis and treatment of osteonecrosis of the femoral head in adults (2019 version). J Orthop Transl. 2020;21(xxxx):100–10.
4. Petek D, Hannouche D, Suva D. Osteonecrosis of the femoral head: Pathophysiology and current concepts of treatment. Efort J. 2019;4(3):85–97.
5. Gaurav S, Yogeshwari S. Avascular necrosis of femoral head: A short review. Unique J Ayurvedic Herb Med. 2015;3(4):54–7.
6. Prasad Dr. Pancham, Sandhu DPS. An Epidemiological Study of Diagnosed Avascular Necrosis of Hip Joint (AVN Hip) Cases and Exploring the Etiology and Treatment Offered in Patients Coming to Dr. Hardas Singh Orthopedic Hospital and Superspeciality Research Centre, Circular Road, Amritsar,. Sch Acad J Biosci. 2020;8(11):344–9.
7. Seamon J, Keller T, Saleh J, Cui Q. The Pathogenesis of Nontraumatic Osteonecrosis. Arthritis. 2012;1–11.
8. Hines JT, Jo WL, Cui Q, Mont MA, Koo KH, Cheng EY, et al. Osteonecrosis of the Femoral Head: an Updated Review of ARCO on Pathogenesis, Staging and Treatment. J Korean Med Sci. 2021;36(24):1–15.
9. Hu LB, Huang ZG, Wei HY, Wang W, Ren A, Xu YY. Osteonecrosis of the femoral head: Using CT, MRI and gross specimen to characterize the location, shape and size of the lesion. Br J Radiol. 2015;88(1046):1–8.