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Avascular Necrosis of the Hip Joint Due to Fracture Caput Femur: A Case Report

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Avascular necrosis, Hip joint, Femoral head fracture, Elderly patient, Conservative management

Abstract
Avascular necrosis (AVN) of the femoral head is a debilitating condition that significantly impacts patient mobility and quality of life, particularly following traumatic injuries. This case report presents a comprehensive analysis of a 72-year-old female patient who developed *avascular necrosis* secondary to femoral head fracture following a fall injury. The patient presented with a three-month history of left hip pain and mobility difficulties after sustaining a fall in the bathroom. Clinical examination, laboratory investigations, and radiological assessment using conventional X-rays were performed to establish the diagnosis. The blood supply to the head of the femur can be easily disrupted, leading to *avascular necrosis* in 15% to 50% of cases of *caput femoris* fractures. Radiological evaluation revealed grade IV *avascular necrosis* according to the Ficat and Arlet Classification, characterized by narrowing of the hip joint, deformity of the *caput femoris* with flattening, subchondral bone cysts, and secondary osteoarthritis. The patient was managed conservatively with high-dose heparin therapy aimed at improving vascular supply and preventing further thrombotic complications. Surgical intervention through total hip arthroplasty was recommended but declined by the patient and family. The goal of treatment is to relieve pain, prevent further damage, restore blood supply, and improve the function of the affected joint. This case highlights the importance of early recognition and prompt management of post-traumatic *avascular necrosis*, the challenges in treating elderly patients with multiple comorbidities, and the need for patient-centered decision-making in complex orthopedic conditions.

INTRODUCTION

Avascular necrosis, otherwise known as osteonecrosis, aseptic necrosis of bone, ischemic bone necrosis, and osteochondritis dessicans, is a condition that causes significant morbidity and impairment in the daily function of patients. It can occur due to a variety of causes, either traumatic or atraumatic in origin. These causes include fractures, dislocations, chronic steroid use, chronic alcohol use, coagulopathy, and congenital factors (Cooper et al., 2010; Elmantaser et al., 2010; Chan & Mok, 2012). One of the most common traumatic causes is femoral neck fracture or dislocation of the femoral head from the acetabulum, where the blood supply to the head of the femur can be

easily disrupted, leading to *avascular necrosis*. *Avascular necrosis* results in 15% to 50% of fractures of the neck of the femur and 10% to 25% of hip dislocations (Baig & Baig, 2018).

The epidemiological data on *avascular necrosis* shows significant geographical and demographic variations that warrant critical examination. While the incidence of *avascular necrosis* of the femoral head within the United States is estimated to occur at a rate between 20,000 to 30,000 new cases each year—contributing to 10% of the approximately 250,000 total hip arthroplasties performed annually (Moya-Angeler et al., 2015)—this data primarily reflects developed healthcare systems with robust trauma management protocols. The incidence of 0.01% found in German-speaking countries and 1.9 per 100,000 found in Japan (Hofmann et al., 2005) suggests significant underreporting in regions with limited diagnostic capabilities, particularly regarding MRI accessibility for early detection.

Previous research has predominantly focused on younger patient populations and steroid-induced *avascular necrosis*, with limited attention to post-traumatic cases in elderly patients. Studies by Ikeuchi et al. (2015), Vardhan et al. (2018), Petek et al. (2019), Barney et al. (2023), and Cowan et al. (2024) consistently report male predominance with ratios from 3:1 to 5:1. However, this demographic pattern may not accurately reflect the clinical reality in geriatric populations, where gender differences in fall risk and bone fragility create different epidemiological patterns. Furthermore, most research has concentrated on treatment outcomes in younger, healthier patients who are surgical candidates, creating a significant knowledge gap regarding optimal management strategies for elderly patients with multiple comorbidities who may not be suitable for invasive interventions.

The literature reveals a concerning bias toward surgical interventions without adequate consideration of conservative management strategies in high-risk populations. While total hip arthroplasty remains the gold standard for advanced *avascular necrosis*, the lack of comprehensive studies on conservative management protocols in elderly patients represents a significant gap that this case report aims to address. Additionally, most classification systems and treatment algorithms were developed based on younger patient cohorts, potentially limiting their applicability to geriatric populations with different healing capacities and treatment tolerances.

The incidence of *avascular necrosis* of the femoral head within the United States is estimated to occur at a rate between 20,000 to 30,000 new cases each year, contributing to 10% of the approximately 250,000 total hip arthroplasties performed annually (Moya-Angeler et al., 2015). This percentage is comparable to the incidence of 0.01% found in German-speaking countries and the incidence of 1.9 per 100,000 found in Japan (Hofmann et al., 2005). This condition is more prevalent in men than women, with studies estimating ratios from 3:1 to 5:1 (Ikeuchi et al., 2015; Vardhan et al., 2018; Petek et al., 2019; Barney et al., 2023; Cowan et al., 2024).

Avascular necrosis of the femoral head is a debilitating disease and an important condition requiring healthcare professionals to be vigilant for its presentation (Barney et al., 2023).

This case report addresses several critical clinical implications that extend beyond the individual patient presentation. For medical practitioners, this case emphasizes the importance of maintaining a high clinical suspicion for *avascular necrosis* in elderly patients presenting with hip pain following even seemingly minor trauma. The three-month delay between initial injury and diagnosis highlights the need for improved clinical awareness and earlier radiological assessment in high-risk populations. The presentation of grade IV *avascular necrosis* with secondary osteoarthritis demonstrates the progressive nature of the condition when early intervention is not implemented.

For orthopedic surgeons and geriatricians, this case illustrates the complex decision-making process required when surgical intervention is declined by elderly patients. The management challenge extends beyond technical surgical considerations to include comprehensive assessment of patient autonomy, quality of life expectations, and the role of conservative therapies in maintaining functional independence. The use of high-dose heparin therapy represents an evidence-based approach to conservative management that may serve as a model for similar cases where surgical intervention is not feasible or desired.

For healthcare systems and policymakers, this case highlights the growing population of elderly patients with traumatic injuries who require specialized geriatric orthopedic care. The increasing incidence of fragility fractures and subsequent complications like *avascular necrosis* necessitates the development of age-appropriate treatment protocols and the allocation of resources for conservative management strategies that can maintain quality of life while respecting patient preferences and medical limitations.

RESEARCH METHOD

The research employed a qualitative case report design. This approach was selected to provide a detailed, contextualized, and holistic analysis of a unique clinical presentation. The case report format is particularly suited for documenting rare conditions, unusual presentations, or complex management scenarios, allowing an in-depth exploration of clinical decision-making, diagnostic challenges, and patient-centered outcomes that may not be captured in larger quantitative studies.

The population of interest consisted of elderly patients who developed post-traumatic *avascular necrosis* (AVN) of the hip joint following a femoral head fracture. The data sample was a single, purposively selected case: a 72-year-old female patient who presented with a three-month history of left hip pain and mobility issues following a fall and was subsequently diagnosed with grade IV AVN. Purposive sampling was used, as this case illustrated the

challenges of managing advanced AVN in a geriatric patient with comorbidities who declined standard surgical intervention.

Data collection involved a comprehensive review of the patient's medical records. The primary sources were the clinical history, physical examination findings, laboratory results, and radiological assessments, specifically anterior-posterior and lateral hip X-rays. These provided the necessary data to establish the diagnosis, assess the severity of the condition using the Ficat and Arlet classification, and document the chosen conservative management approach and its outcomes.

RESULT AND DISCUSSION

CASE REPORT

Patient Presentation and History

A 72-year-old female patient presented to our hospital with a complex constellation of symptoms that had developed over a three-month period following a traumatic event. The patient's primary complaint was persistent vomiting after every meal for the two months preceding admission, during which time her oral intake was severely limited to approximately three small glasses of honeyed warm water daily. This nutritional compromise resulted in significant unintentional weight loss of approximately 10 kilograms over the two-month period, representing approximately 15% of her estimated baseline body weight.

The precipitating event occurred approximately three months prior to admission when the patient sustained a fall in the bathroom due to generalized body weakness. Following this incident, she developed persistent pain in the left hip joint that significantly impacted her mobility and ambulation capacity. There was no unilateral weakness of limbs or slurred speech. The patient initially sought medical attention at a local Community Health Center but reported no improvement in her symptoms despite treatment, ultimately leading to her presentation to our hospital for comprehensive evaluation and management.

Diagnostic Workup and Laboratory Findings

Comprehensive laboratory evaluation revealed several significant findings that guided the diagnostic workup and treatment planning. Laboratory results showed Hb 13.4g/dL, leukocyte count 10,650/µL, thrombocyte count 402,000/µL, and creatinine 1.01mg/dL. The complete blood count demonstrated adequate hemoglobin levels despite the patient's poor nutritional status, while the elevated leukocyte count suggested possible infectious or inflammatory processes. The thrombocyte count was within normal limits, and renal function appeared preserved based on serum creatinine levels.

Urinalysis results revealed significant abnormalities consistent with urinary tract infection, including yellow but cloudy appearance, +3 leukocytes, +1 urine proteins, +2 erythrocytes, and 23-25 leukocytes per high-power field,

with bacteria identified on microscopic examination. These findings confirmed the presence of a concomitant urinary tract infection that required concurrent management alongside the orthopedic condition.

Radiological Assessment and Imaging Findings

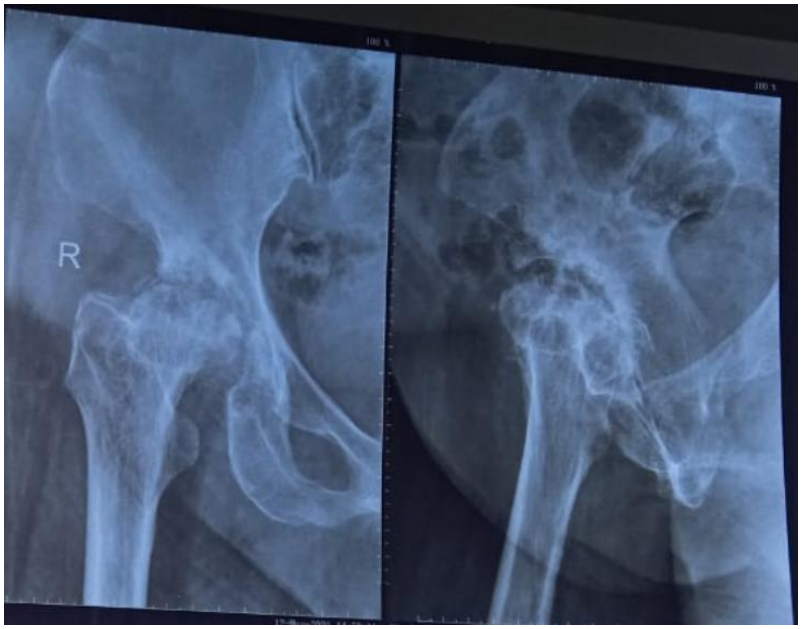

Figure 1. Radiology of right hip

Conventional radiography of the right hip joint provided definitive diagnostic information that established the underlying pathology. Radiology of right hip joint showed there was narrowing of the right hip joint and deformity of the right caput femoris with flattening and subchondral bone cyst. Also, deformity of the right acetabular roof with flattening, sclerosis, and subchondral bone cyst. These findings were pathognomonic for supporting features of avascular necrosis of the right hip joint grade IV (based on Ficat and Arlet Classification) with secondary osteoarthritis. Additional thoracic imaging revealed signs of aortosclerosis and spondylosis thoracalis, indicating generalized age-related degenerative changes consistent with the patient's advanced age.

Hospital Course and Management Approach

The patient was admitted to the hospital with a complex multisystem diagnosis including urinary tract infection, vomiting with dehydration, poor oral intake, and grade IV avascular necrosis of the hip joint. The multidisciplinary approach to management addressed both the acute medical issues and the chronic orthopedic condition. During hospitalization, patient was treated with high-dose heparin. The rationale for heparin therapy was

based on its potential to improve microcirculation, prevent further thrombotic complications, and potentially slow the progression of osteonecrosis through improved vascular perfusion of the affected bone.

Orthopedic consultation was obtained for comprehensive evaluation and consideration of surgical intervention. Patient was consulted to the orthopedic department for Total Hip Arthroplasty, but the patient refused to undergo the procedure. This decision was made after thorough discussion of the risks, benefits, and alternatives to surgical management, taking into consideration the patient's age, comorbidities, and personal preferences regarding invasive interventions.

Discussion

Hip pain is a common and disabling condition that affects patients of all ages. The differential diagnosis of hip pain is broad, presenting a diagnostic challenge. Patients often express that their hip pain is localized to one of three anatomic regions: the anterior hip and groin, the posterior hip and buttock, or the lateral hip. Lateral hip pain occurs with greater trochanteric pain syndrome. Patients often localize pain by cupping the anterolateral hip with the thumb and forefinger in the shape of a “C”, this is known as the C sign. Anterior hip and groin pain is commonly associated with intra-articular pathology, such as osteoarthritis, femoroacetabular impingement, hip labral tears, iliopsoas bursitis (internal snapping hip), occult or stress fracture, transient synovitis, septic arthritis, and avascular necrosis. Posterior hip pain is associated with piriformis syndrome, sacroiliac joint dysfunction, lumbar radiculopathy, and less commonly ischiofemoral impingement and vascular claudication. Lateral hip pain occurs with greater trochanteric pain syndrome (Wilson & Furukawa, 2014).

Avascular necrosis, otherwise known as osteonecrosis, ischemic necrosis, aseptic necrosis of bone, osteochondritis dissecans or bone death results in collapse of the normal architecture of the skeletal framework, leading to significant clinical morbidity, including progressive join pain and loss of function. The most commonly affected sites of osteonecrosis include the femoral and humeral heads, but avascular necrosis can occur elsewhere as well, including knees (femoral condyles and proximal tibia), small bones of the foot, ankle, and hands (including scaphoid and lunate), vertebrae and bony structures of the face (Powel et al., 2010).

Avascular necrosis may be silent or may clinically present with a gradual onset characterized by mild or vague pain that may worsen after ambulation and subsequently progress to severe pain when bone collapse occurs; in other cases avascular necrosis may cause a sudden onset deep joint pain that initially is elicited by movement and later is present also at rest. Avascular necrosis may be asymptomatic and not progressive when a small fragment of bone tissue is involved. In case of medium-large bone area involvement usually the

lesion progresses and eventually the necrotic tissue collapses (Caramaschi et al., 2012).

Initial physical findings are largely nonspecific and may include tenderness around the affected bone and limitation of the range of motion. Physical findings depend on the location of the affected bone. Patients with advanced avascular necrosis of the hip may present with a limp (Moghadam & Werth, 2010).

Conventional radiography is the most convenient and inexpensive first-line investigation for the diagnosis of avascular necrosis. Abnormal findings in avascular necrosis include 'crescent sign' representing subchondral collapse, cystic or sclerotic change in femoral head, abnormal contours of femoral head, collapse or secondary degenerative change. However, plain radiograph is not sensitive in detecting early avascular necrosis lesions (Chan & Mok, 2012).

This case is a 72-years-old woman with symptoms of vomiting after every meal since 2 months prior and history of pain in her right hip joint after fell in the bathroom 3 months before admission. Further, the patient is also experiencing unintended weight loss and difficulty in mobility due the pain in her hip. At first, the patient was admitted into the ward due to her dehydration that occurred after a long history of vomiting. But examination further revealed not only urinary tract infections but also avascular necrosis of the right hip joint.

Other than conventional radiography, Technetium-99m (Tc-99m) bone scanning is used in patients with suspicion of avascular necrosis where plain photographs are normal with unilateral symptoms and no risk factors. Increased bone turnover at the border of dead and reactive bone results in increased uptake surrounding the cold area, referred to as doughnut sign. However, bone scans performed on 48 patients with suspicion of avascular necrosis of the shoulder, hip, knee or ankle showed lower sensitivity compared to magnetic resonance imaging (MRI) in diagnosing avascular necrosis (Mont et al., 2008). However, bone scan is less specific for the diagnosis of avascular necrosis. Other limitations include radiation dose, poor spatial resolution and inability to quantify the lesion for prognostic purpose (Chan & Mok, 2012).

MRI is much more sensitive when compared to plain photographs or bone scans and is reported to have a sensitivity of 100% (Mont et al., 2008). The changes can already be seen in the early phase of the disease where other radiologic examinations still do not show the presence of avascular necrosis. Well-demarcated and inhomogeneous focal lesions were seen at T1W1. The earliest finding is a low density signal indicating the border between normal and ischemia bone. At T2W1 a second high density line is seen, indicating hypervascular granulation tissue, where this pathognomonic picture is called the double-line sign. This pattern reflects tissue repair around the necrotic bone. MRI can also objectively show revascularization and tissue changes in response to treatment and evaluate the avascular necrosis lesion continuously

for follow-up therapy. In comparison, Computerized Tomography Scanning (CT-Scan) can only be useful for differentiating pre-collapse avascular necrosis and early avascular necrosis (Shigemura et al., 2011).

MRI has replaced bone marrow pressure examination, venography, and bone biopsy as a means of diagnosing early-phase avascular necrosis. However, these MRI findings should be interpreted with caution, especially in asymptomatic patients. In asymptomatic patients, surgery based on MRI abnormality findings alone may lead to overtreatment in some patients. The choice of treatment modality in patients with avascular necrosis is based on the size of the lesion, location of the lesion, age of the patient, general health condition, and symptoms (Hungerford & Jones, 2004).

Histologically, lesions in avascular necrosis show accumulation of bone marrow cell debris, bone trabeculae showing lacunae and/or ghost nuclei in lacunae and an increase in bone marrow fat cells. Appositional reparative bone formation was also seen around the necrotic bone tissue, but was not significant (Sheng et al., 2013).

In patients with asymptomatic avascular necrosis, the clinical diagnosis is made if MRI or other radiological examinations support a picture of avascular necrosis and other causes of pain and bone abnormalities have been ruled out through appropriate examinations. MRI remains the gold standard for establishing a diagnosis of avascular necrosis in both asymptomatic and symptomatic patients, especially in the early phase of the disease (Amanatullah et al., 2011).

Most cases of avascular necrosis occur secondary to trauma. However, non-traumatic avascular necrosis can also occur, although the underlying pathogenesis remains unclear to date. Non-traumatic avascular necrosis is associated with a number of risk factors including the use of corticosteroids, alcohol consumption, immunosuppressive therapy, autoimmune diseases such as Systemic Lupus Erythematosus (SLE) and Rheumatoid Arthritis (RA), hematologic/thrombotic disorders, malignancy and other metabolic disorders such as diabetes mellitus and renal failure. The pathogenesis of non-traumatic avascular necrosis is thought to be multifactorial, so patients with non-traumatic avascular necrosis usually have more than one underlying risk factor (Cooper et al., 2010). In some literature, it is mentioned that avascular necrosis in the hip mostly occurs in elderly women (Suryadevara et al., 2023).

Essentially, avascular necrosis occurs through a common pathway of decreased blood flow to the bone leading to ischemia and bone death. However, the underlying mechanisms vary. Vascular occlusion can be caused by a localized thrombus, fat embolism, nitrogen bubbles or abnormally shaped red blood cells (Seamon et al., 2012).

Corticosteroids are the most common cause of non-traumatic avascular necrosis. It is reported that patients receiving steroid therapy have a 20-fold higher risk of avascular necrosis (Sakaguchi et al., 2010). Chronic corticosteroid administration promotes fat conversion of red marrow. The

increase of bone marrow fat content may elevate marrow pressure reducing arterial perfusion. Besides the aforementioned effects of corticosteroids on fat content of bone marrow, corticosteroid may impair bone integrity by inducing apoptosis of osteoblast and osteocytes (Weinstein, 2010). Through the downregulation of osteoprotegerin and the upregulation of RANKL impairing the balance between bone formation and bone resorption (Caramaschi et al., 2012).

Although the dose effect of steroid therapy on avascular necrosis is not yet known, in some studies it is said that corticosteroid doses above 25-40 mg / day are a significant risk factor for non-traumatic avascular necrosis, especially in SLE patients and post kidney transplantation (Nagasawa et al., 2005). In other literature, dexamethasone is the type of glucocorticoid that most often causes avascular necrosis (Elmantaser et al., 2010).

Common classifications that map the phases of avascular necrosis of the hip include the Ficat and Arlet and Steinberg classifications (Jawad et al., 2012; Matthews et al., 2023). Ficat and Arlet describe the four stages of disease progression based on clinical and radiographic findings. Stage 1 is the initiation of the disease without radiological findings. Stage IV is the end stage with femoral head collapse, flattening, and narrowing of the joint space. The Steinberg classification incorporates the use of MRI to detect a pre-clinical lesion and also to assess the size of the lesion (Matthews et al., 2023).

By conventional radiography, this patient revealed a result of narrowing of the right hip joint and deformity of the right caput femoris with flattening and subchondral bone cyst. Also, deformity of the right acetabular roof with flattening, sclerosis, and subchondral bone cyst. Those findings leads into a conclusion for features of avascular necrosis of the right hip joint grade IV based on Ficat and Arlet Classification. Further confirmation of examination using MRI or CT-scan wasn't conducted for this patient due to previous history of falling from anamnesa and clear findings in conventional radiography of right hip joint.

The management of avascular necrosis includes conservative non-surgical and surgical approach. Indication for surgical and non-surgical approach depends on stage of disease, size of lesion, age and co-morbidity of patients (Chan & Mok, 2012). The goal of the treatment of avascular necrosis is to preserve joint integrity by preventing bone collapse (Caramaschi et al., 2012).

Conservative management includes bed rest and reducing weight bearing by using crutches or canes or other walking aids. Non-steroidal anti-inflammatory drugs or other analgesics may be given to manage pain. However, conservative therapy alone is not enough to inhibit the progress of avascular necrosis itself (Chan et al., 2012). To date, there is no consensus on the management of patients with asymptomatic avascular necrosis. Hungerford & Jones, suggest non-surgical management in patients with lesions <15% femoral volume and surgical management in patients with

intermediate-sized lesions (15-30%) and preparation for Total Hip Arthroplasty in patients with lesions >30% (Hungerford & Jones, 2004).
The management of avascular necrosis remains controversial. There are three main therapeutic modalities, namely:

1. Conventional Therapy / Non-surgery Therapy

a. Low Molecular Weight Heparins (LMWH), given to patients with underlying coagulation disorders to inhibit the progress of avascular necrosis. Warfarin or LMWH anticoagulants, if given at an early stage before collapse, can improve the condition. In addition to correcting the coagulation abnormalities underlying avascular necrosis, LMWH may also have a direct effect on the healing process of osteonecrotic segments. It is suspected that LMWH can increase osteoclastic bone resorption and angiogenesis and can inhibit the inflammatory process. (Parsons, 2008).
b. Biphosphonate, several studies have reported the efficacy of the bisphosphonates in treatment of avascular necrosis. However, evidence is controversial. Biphosphonate increase osteoclast apoptosis and inhibit the resorptive action of the osteoclast. On the other hand, they reduce apoptosis of osteoblasts and osteocytes, thus regarding bone turnover. Currently, there is not enough evidence to recommend the routine use of bisphosphonate in patients with avascular necrosis (Chan & Mok, 2012). Agarwala et al. showed that alendronate 10 mg/day treatment for three years in hip avascular necrosis led to a significant improvement in pain and disability score than conservative therapy. The patients also have calcium therapy 500-1000 mg/day and vitamin D3 400-800 IU/day. A randomized controlled study by Lai et al. demonstrated that patients with avascular necrosis stage II or III based on University of Pennsylvania classification system had $\geq$30% caput femoral, showed that alendronate 70mg/week treatment for 25 weeks had a significant lower rate of femoral head collapse (Parsons & Steele, 2008).
c. Statins (HMG-CoA reductase inhibitors), Cui et al. reported that statins can inhibit the effect of steroids on the accumulation of fat cells in the bone marrow and maintain an osteoblastic phenotype to maintain a normal microenvironment of the bone marrow, so as to prevent the progression of avascular necrosis (Parsons & Steele, 2008).
d. Hyperbaric oxygen is another non-operative treatment option of avascular necrosis. Hyperbaric oxygen improves the oxygenation of hypoxic tissue and reduces edema by increasing the concentration of dissolved oxygen and inducing vasoconstriction (Sen, 2009).

2. Surgery

Surgery procedures are used to preserve the joint. The timing and type of surgical intervention chosen depends on the area involved and the stage of avascular necrosis. Results from a review of prospective studies on 36 patients with avascular necrosis of the caput femur, the success rate of surgical

intervention in stage I Ficat avascular necrosis was 70% compared to 20% in the conservative approach. (Chan & Mok, 2012).

a. Core decompression. This procedure is mainly indicated in early-stage avascular necrosis. It was initially used as a diagnostic tool to measure bone marrow pressure and obtain biopsy specimens. The use of decompression as a therapy is based on the consideration that increased intramedullary pressure is associated with the pathogenesis of avascular necrosis. Core decompression aims to lower the intramedullary pressure which eventually reverses the process of avascular necrosis before abnormalities appear on radiologic examination and reduces pain. However, core decompression is usually ineffective in avascular necrosis involving >25% of the femoral caput or >2/3 of the weight bearing part of the femur. (Sen, 2009).
b. Bone graft. There are various types of bone grafting, some combined with osteotomy, osteochondral grafts, muscle pedicle bone grafts, and some are vascularized grafts to improve blood flow of the bone by achieving revascularization (Chan & Mok, 2012).
c. Osteotomy. Aims to relocate the necrotic area of bone from the weight loading area of acetabulum, so as to redistribute the weight loading to articular cartilage, which is supported by healthy bone. Results of studies of osteotomy vary according to different types of osteotomy (Chan & Mok, 2012).
d. Joint replacement. Indicated in the advanced stage of avascular necrosis, once the femoral head has collapsed or the destruction process has involved the acetabulum. These include femoral resurfacing arthroplasty, hemiarthroplasty, and total hip arthroplasty. Compared with total hip replacement due to other causes like osteoarthritis, patients with avascular necrosis in general have a less favorable long-term durability or high failure rate (Chan & Mok, 2012).

This patient was consulted to the surgery department in hope to preserve her joint function thus why would improve her quality of life. After throughout discussions, both patient and the family were refusing the procedure and settled with conventional therapy.

The prognostic avascular necrosis based on stage of the disease. An early detection of the lesion and a prompt treatment are crucial to obtain optimal results. Negative prognostic factor is also the involvement of a weight-bearing area, such as the femoral head (Caramaschi et al., 2012).

CONCLUSION

Avascular necrosis should be considered a primary differential diagnosis in patients presenting with joint pain, especially in elderly populations where symptoms may be atypical or delayed, regardless of a documented trauma history. Early-stage treatment aims not only to relieve pain but also to prevent further structural damage, restore blood supply, and improve joint function. In end-stage *avascular necrosis*, individualized management based on patient

factors, comorbidities, and preferences is essential. While surgical intervention remains the most effective option for restoring joint function, conservative approaches such as anticoagulation therapy and supportive care can provide meaningful symptom relief and improve quality of life for patients who are unsuitable for or decline surgery. This case underscores the importance of early recognition, timely diagnosis, and personalized treatment planning to optimize outcomes in post-traumatic *avascular necrosis* of the hip joint. Future research should focus on developing and evaluating conservative management strategies tailored to elderly patients with advanced *avascular necrosis*, aiming to improve functional outcomes and quality of life in this growing patient population.

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