Volume 11, Issue 2, February – 2026 International Journal of Innovative Science and Research Technology
ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26feb527

Bilateral Hip Arthroplasty in Avascular Necrosis-Surgical Timing and Outcomes: A Case Report

Badhra Ajit Nair; Hamlin Joseph Antony; Manjima Sunil; Emil Siju Kuriakose

Tbilisi State Medical University, Georgia.

Corresponding Author: Badhra Ajit Nair*

Publication Date: 2026/02/20

Abstract: Avascular necrosis (AVN) is a progressive bone disorder caused by disruption of blood supply, leading to bone necrosis, structural collapse, and joint dysfunction. Early diagnosis and timely intervention are essential to prevent irreversible disability. We report the case of a 35-year-old Indian male who presented with severe left hip pain significantly affecting daily activities. Magnetic resonance imaging confirmed bilateral AVN, with Grade IV involvement of the left hip and Grade II–III involvement of the right hip. Conservative management failed to provide symptomatic relief, necessitating surgical intervention in the form of total left hip replacement and right hip core decompression. Postoperatively, the patient demonstrated clinical improvement, with pain scores on the Visual Analog Scale reducing from 8/10 to 6/10, hip flexion improving from 90° to 120°, and muscle strength increasing from grade 3/5 to 4/5. AVN is commonly associated with trauma, corticosteroid use, and systemic conditions and progresses from ischemia to joint degeneration if untreated. This case emphasizes the importance of early MRI-based diagnosis and individualized treatment strategies, as advanced AVN often requires surgical management to improve pain, mobility, and overall quality of life.

Keywords: *Avascular Necrosis; Femoral Head; Magnetic Resonance Imaging; Total Hip Replacement; Core Decompression; Hip Pain; Osteonecrosis.*

How to Cite: Badhra Ajit Nair; Hamlin Joseph Antony; Manjima Sunil; Emil Siju Kuriakose (2026) Bilateral Hip Arthroplasty in Avascular Necrosis-Surgical Timing and Outcomes: A Case Report. *International Journal of Innovative Science and Research Technology*, 11(2), 977-981. https://doi.org/10.38124/ijisrt/26feb527

I. INTRODUCTION

Bilateral AVN, or osteonecrosis, results from insufficient blood supply to bone tissue, leading to necrosis, joint deterioration, and functional impairment. When both hips are affected, mobility is severely compromised, significantly impacting daily life.[3][5]

The condition can arise from trauma, corticosteroid use, alcohol abuse, or systemic diseases such as lupus and sickle cell disease.[1][4] AVN often progresses silently, eventually causing deep joint pain, stiffness, and restricted motion. If untreated, it can lead to joint collapse and osteoarthritis, resulting in profound disability.[2].

Beyond trauma and corticosteroid use, other risk factors include dysbaric osteonecrosis (seen in divers), HIV/AIDS, chemotherapy, organ transplantation, and autoimmune vasculitis. Advanced imaging techniques, such as diffusion-weighted MRI, dynamic contrast-enhanced MRI, and SPECT, provide earlier and more precise detection of AVN than conventional methods.

Diagnosis relies on imaging, with MRI as the gold standard for early detection, revealing ischemic changes before structural collapse. X-rays identify late-stage damage, while bone scans may assess vascular impairment [7].

Avascular necrosis (AVN) is a progressive condition that can affect multiple joints, most commonly the hip. It is classified using systems such as the Ficat and Arlet and Steinberg classifications, which guide treatment decisions based on disease progression.[4][2]

Management depends on disease severity. Early-stage AVN may be managed conservatively with physical therapy, weight-bearing restrictions, and medications like bisphosphonates.[8]Surgical intervention is often required in advanced cases, with core decompression benefiting early-stage patients and total hip replacement (THR) serving as the preferred treatment for end-stage disease[10][11].

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26feb527

Emerging treatment strategies focus on regenerative medicine, including platelet-rich plasma (PRP), bone morphogenetic proteins (BMPs), and 3D-printed scaffolds for bone tissue engineering. Pharmacological advancements, such as statins, prostaglandin analogues, and hyperbaric oxygen therapy (HBOT), show promise in preventing and managing early-stage AVN.

AVN can also affect non-hip joints, such as the humeral head, knee, and talus, with similar mechanisms of ischemic bone damage. A deeper understanding of AVN's classification, risk factors, imaging techniques, and novel treatment options is crucial for improving early diagnosis and long-term joint preservation.

A multidisciplinary, patient-centered approach is essential to preserve joint function, improve mobility, and enhance overall quality of life [9].

II. CASE PRESENTATION

A 35-year-old Indian male presented with debilitating left hip pain (VAS 9/10) that began on June 27, 2024, severely impacting his daily activities, including walking, dressing, and personal care. He exhibited a limp, a short-leg appearance on the left side, and significant restriction in left hip movement, while the right hip and knees were unremarkable. MRI [Fig.1] confirmed bilateral avascular necrosis (AVN) of the femoral heads, with advanced Grade IV changes in the left hip (Kerboul angle 303°) and moderate Grade II-III changes in the right hip (Kerboul angle 298°). Despite the structural integrity of the right hip, the left hip showed collapse with soft tissue edema and mild joint effusion. The patient, with no prior medical history or substance use, underwent total left hip replacement and right hip core decompression with drilling. Postoperatively, he received five physiotherapy sessions, leading to pain reduction (VAS 8/10 to 6/10), improved left hip flexion (90° to 120°), and increased hip strength (3/5 to 4/5). A follow-up is scheduled for September 9, 2024, to assess recovery.

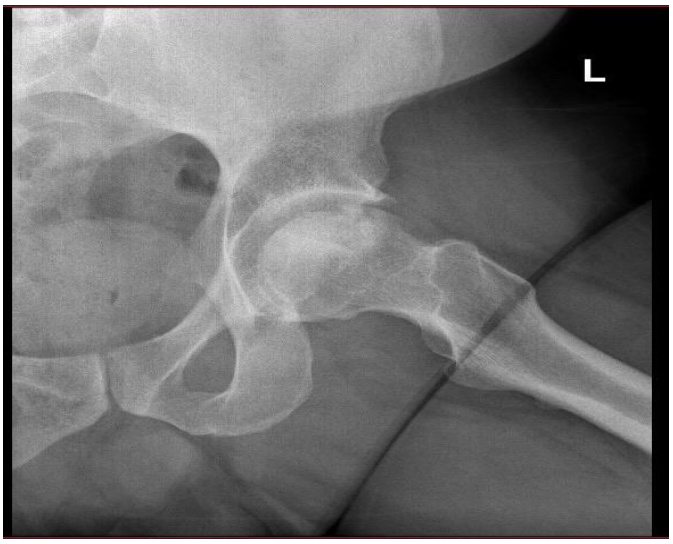

Fig.1 MRI Confirmed Bilateral Avascular Necrosis (AVN) of the Femoral Heads, with Advanced Grade IV Changes in the Left Hip (Kerboul Angle 303°) and Moderate Grade II-III Changes in the Right Hip (Kerboul angle 298°)

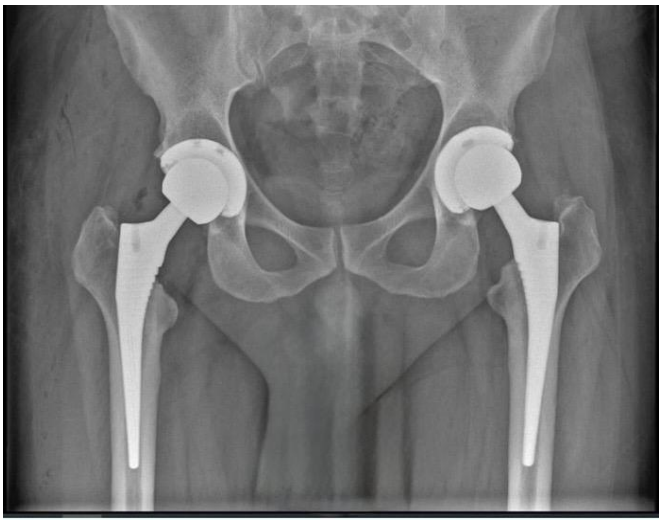

Fig.2 Patient Underwent Total Left Hip Replacement and Right Hip Core Decompression with Drilling In the Figure you can See Both Hip Bones.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26feb527

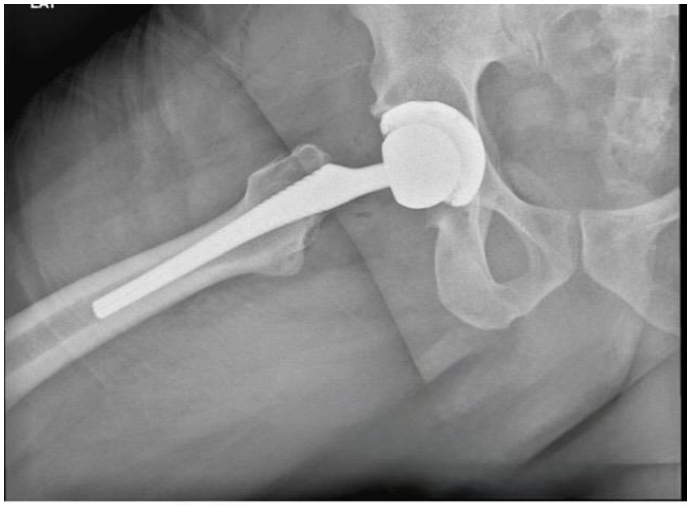

Fig.3 Patient Underwent Total Left Hip Replacement and Right Hip Core Decompression with Drilling In the Figure you can See the Right Hip Bone.

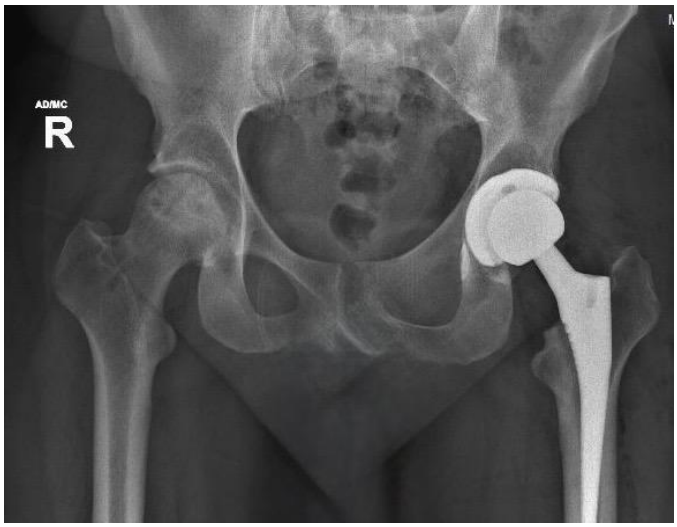

Fig.4 Patient Underwent Total Left Hip Replacement and Right Hip Core Decompression with Drilling

III. DISCUSSION

Avascular necrosis (AVN) is a progressive condition caused by impaired blood supply to bone tissue, leading to osteocyte death, structural collapse, and joint dysfunction. It commonly affects weight-bearing joints, particularly the femoral head, and can result from trauma, corticosteroid use, alcohol abuse, or systemic diseases. If untreated, AVN progresses to joint collapse and osteoarthritis, causing significant disability[3][5].

AVN develops when bone perfusion is compromised, leading to ischemia and necrosis. Trauma, such as fractures or dislocations, can disrupt blood flow, while non-traumatic causes include thrombosis, embolism, and vascular occlusion. In femoral head AVN, femoral neck fractures may damage essential blood vessels, triggering ischemia. As oxygen and nutrient deprivation continues, osteocytes die, disrupting bone remodeling. Osteoclast-driven resorption exceeds osteoblast-mediated formation, weakening the bone and increasing susceptibility to collapse. Additional factors, such as endothelial dysfunction, fat emboli from corticosteroid use or hyperlipidemia, and hypercoagulability disorders like antiphospholipid syndrome, further impair circulation and accelerate necrosis[4][7][8][9].

Trauma, particularly femoral neck fractures or hip dislocations, is a leading cause of AVN. Corticosteroid use is another major risk factor, as prolonged or high-dose exposure increases marrow fat content, leading to fat embolism and microvascular obstruction. Chronic alcohol consumption contributes to fatty infiltration of the bone marrow, vascular toxicity, and microcirculatory failure. Systemic conditions, including hypercoagulable disorders, sickle cell disease, and autoimmune diseases like lupus, further increase AVN risk. Radiation therapy can also damage vasculature, leading to bone ischemia. In some cases, AVN occurs without an identifiable cause (idiopathic AVN), often seen in younger individuals, potentially due to microvascular occlusion and intrinsic bone weakness[3][5][12].

Several conditions beyond trauma, corticosteroids, and alcohol use contribute to AVN. Dysbaric osteonecrosis, seen in scuba divers and miners, results from nitrogen emboli forming during rapid decompression. HIV/AIDS is linked to AVN due to viral effects and antiretroviral medications,

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26feb527

especially protease inhibitors. Chemotherapy and organ transplantation increase AVN risk due to prolonged corticosteroid and immunosuppressant use, with drugs like methotrexate and cyclosporine causing microvascular damage. Autoimmune vasculitis disorders such as Takayasu arteritis and Behçet's disease can also impair bone perfusion, further predisposing individuals to AVN[13][14][15][16]

Diagnosis of AVN involves clinical evaluation and imaging studies. Patients typically present with joint pain that worsens with weight-bearing activity. X-rays, the first-line imaging technique, detect late-stage changes like subchondral sclerosis and femoral head flattening, with the crescent sign indicating advanced collapse. However, MRI is the gold standard for early AVN detection, identifying bone marrow edema and ischemic changes before structural damage occurs. CT scans help assess bone structure, while bone scintigraphy can evaluate vascular impairment. In rare cases, a bone biopsy confirms necrotic tissue[6][10]

Beyond standard MRI, Diffusion-Weighted Imaging (DWI) MRI detects ischemic changes earlier, while Dynamic Contrast-Enhanced MRI (DCE-MRI) evaluates bone perfusion and microvascular integrity, aiding in disease prediction. Single Photon Emission Computed Tomography (SPECT) provides higher sensitivity in post-traumatic AVN, detecting early perfusion deficits before structural damage occurs[17][18][19]

AVN staging relies on classification systems to assess disease progression. The Ficat and Arlet Classification stages AVN from normal X-rays (Stage I) to complete femoral head collapse and osteoarthritis (Stage IV). The Steinberg Classification provides greater detail, with Stage 0 showing histological AVN despite normal imaging and Stage VI representing severe joint destruction. These classifications help guide treatment decisions based on disease severity[2][4]

In early-stage AVN, conservative treatments aim to relieve symptoms and slow disease progression. NSAIDs help manage pain and inflammation, while bisphosphonates may reduce bone resorption, though their effectiveness remains debated. Weight-bearing restrictions and assistive devices reduce stress on the joint, potentially delaying disease progression. Core decompression, a minimally invasive procedure that drills into necrotic bone to relieve pressure and stimulate blood flow, is effective in early stages. Emerging therapies, such as stem cell injections, aim to regenerate bone and restore vascular supply[11]

For advanced AVN, surgical intervention is often necessary. Osteotomy realigns the bone to redistribute weight away from necrotic areas, delaying joint replacement. Bone grafting, often combined with core decompression, provides structural support and encourages healing. For end-stage disease, total hip replacement (THR) is the definitive treatment, replacing the damaged femoral head and acetabulum with prosthetic components[4][11]

THR is indicated when AVN leads to significant joint degeneration, including femoral head collapse, cartilage destruction, and joint space narrowing. It is also considered when chronic pain remains unrelieved by medications or conservative measures. The procedure involves replacing the damaged joint with ceramic, metal, or polyethylene prosthetic components, providing durability and low friction. Post-surgery rehabilitation includes physical therapy to improve range of motion, strength, and stability, with gradual weight-bearing introduced over several months. Modern prostheses offer long-term relief, lasting 10-20 years[4][11]

Platelet-Rich Plasma (PRP) injections promote angiogenesis and bone healing, while Bone Morphogenetic Proteins (BMPs) enhance bone regeneration when combined with core decompression. 3D-printed scaffolds, often infused with stem cells or osteoinductive agents, provide structural support and stimulate new bone formation[10]

Pharmacological advancements include statins, which may prevent corticosteroid-induced AVN by reducing fat embolism formation, and prostaglandin E1 analogues, which improve microvascular circulation to slow disease progression. Hyperbaric oxygen therapy (HBOT) is also being explored to reduce intraosseous pressure and enhance vascular repair[1]

Although AVN primarily affects the femoral head, it can also occur in other weight-bearing joints. Humeral head AVN (Hass disease) is often linked to corticosteroid use or trauma, while distal femoral AVN affects the knee, either due to trauma or idiopathic causes. Talar AVN (ankle AVN) is particularly susceptible to ischemic damage due to its limited blood supply and commonly occurs after ankle fractures or vascular injury, often requiring early intervention to prevent joint collapse[16][17][18]

As AVN progresses, complications severely impact joint function. Subchondral collapse weakens necrotic bone, leading to deformity and impaired mobility. Osteoarthritis develops as joint collapse alters biomechanics, causing cartilage erosion, inflammation, stiffness, and pain. Bone marrow edema worsens symptoms due to increased intraosseous pressure. In late stages, joint degeneration leads to severe disability, making weight-bearing activities intolerable and significantly reducing quality of life[7][8][9]

IV. CONCLUSION

AVN is a progressive and potentially debilitating condition requiring early diagnosis and timely intervention. While conservative measures may slow progression in early stages, surgical management remains the standard for advanced disease. A multidisciplinary approach—combining medical, rehabilitative, and surgical strategies—is crucial to preserving joint function, improving mobility, and preventing long-term disability.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/26feb527

DECLARATIONS

➢ *Ethical Approval*
Ethical approval was not required for this study.

➢ *Consent for Publication*
Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

➢ *Availability of Data and Materials*
Available materials are all included in the manuscript

➢ *Competing Interests*
There is no conflicts of interest

➢ *Funding*
Nil

➢ *Authors' Contributions*

- BAN analyzed and interpreted the patient data regarding AVN.
- HJA researched and analyzed in depth about the surgical procedures.
- MS studied about the future possibilities of treatment AVN in a much more concise and efficient manner.
- ESK interpreted the rehabilitation possibilities and outcomes.

All authors read and approved the final manuscript."

ACKNOWLEDGMENT

We thank the authors for their valuable contribution to this project.

REFERENCES

[1]. Mont MA, Hungerford DS. Avascular necrosis of the hip: mechanisms of disease and management. J Bone Joint Surg Am. 1995;77(6):877-888. Available from: https://journals.lww.com/jbjsjournal/Abstract/1995/77060/Avascular_Necrosis_of_the_Hip__Mechanisms_of.1.aspx.
[2]. Steinberg ME, Heller S. Avascular necrosis of the femoral head: a review of current concepts. Orthop Clin North Am. 2000;31(4):673-688. Available from: https://www.orthopedic.theclinics.com/article/S0030-5898(05)00012-8/fulltext.
[3]. Mayo Clinic. Avascular necrosis: symptoms and causes. Available from: https://www.mayoclinic.org/diseases-conditions/avascular-necrosis/symptoms-causes/syc.
[4]. Ficat RP, Arlet J. Ischemia and Necrosis of Bone. Springer Science & Business Media; 1980.
[5]. National Institute of Arthritis and Musculoskeletal and Skin Diseases. Osteonecrosis. Available from: https://www.niams.nih.gov/health-topics/osteonecrosis.
[6]. National Center for Biotechnology Information. Osteonecrosis. In: StatPearls. Available from: https://www.ncbi.nlm.nih.gov/books/NBK546658/.
[7]. Arlet J, Ficat RP. Pathogenesis of nontraumatic osteonecrosis. SpringerLink. Available from: https://link.springer.com/chapter/10.1007/978-1-4613-8628-5_1.
[8]. ResearchGate. The pathogenesis of nontraumatic osteonecrosis. Available from: https://www.researchgate.net/publication/233931904_The_Pathogenesis_of_Nontraumatic_Osteonecrosis.
[9]. National Library of Medicine. Pathogenesis of osteonecrosis. PMC. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC3518945/.
[10]. National Library of Medicine. Osteonecrosis review. PubMed. Available from: https://pubmed.ncbi.nlm.nih.gov/30850073/.
[11]. National Library of Medicine. Osteonecrosis treatment advancements. PubMed. Available from: https://pubmed.ncbi.nlm.nih.gov/32800435/.
[12]. National Library of Medicine. Corticosteroid-induced osteonecrosis. PubMed. Available from: https://pubmed.ncbi.nlm.nih.gov/12690862/.
[13]. Moon RE. “Dysbaric osteonecrosis in divers and compressed-air workers.” Undersea Hyperb Med. 2014;41(6):579-590.
[14]. Carten ML, Kopp JB. “Avascular necrosis in HIV disease: epidemiology, etiology, and clinical management.” AIDS. 2003;17(1):1-9.
[15]. Assouline-Dayan Y, Chang C, Greenspan A, Shoenfeld Y, Gershwin ME. “Pathogenesis and natural history of osteonecrosis.” Semin Arthritis Rheum. 2002;32(2):94-124.
[16]. Fauchald P, Rygvold O, Oystese B. “Takayasu’s arteritis—a 5-year follow-up study.” Acta Med Scand. 1982;211(6):457-463.
[17]. Koo KH, Kim R, Ko GH, Song HR, Jeong ST, Yang JW. “Preventing collapse in early osteonecrosis of the femoral head.” Clin Orthop Relat Res. 1999;(358):221-226.
[18]. Talbot BS, Weinberg EP. “Imaging of avascular necrosis and transient osteoporosis of the hip.” Semin Musculoskelet Radiol. 2015;19(1):62-72.
[19]. Lee S, Kim JY, Han SH. “Comparison of SPECT and MRI for early detection of avascular necrosis of the femoral head.” Eur J Nucl Med Mol Imaging. 2012;39(6):1125-1133.