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Case Report

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AYURVEDIC MANAGEMENT OF AVASCULAR NECROSIS OF FEMORAL HEAD W.S.R. TO ASTHIMAJJAGATAVATA: A CASE STUDY

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ABSTRACT

Avascular necrosis (AVN) of the femoral head is a rare skeletal condition characterized by vague symptoms originating from the hip joint or lower pelvis. It involves a disruption of blood supply leading to ischemic necrosis, progressive osteocyte death, collapse of the joint surface, and loss of hip functionality. The symptoms of AVN align closely with those described as *asthimajjagatavata* in *Ayurvedic* texts by *Acharya Charaka*, manifesting as breaking pain, hip joint pain, muscle wasting, weakness in the affected joint, and persistent pain-induced insomnia. **Objective** - *Ayurvedic* Management of Avascular Necrosis of Femoral Head w.s.r. to *asthimajjagatavata*. **Materials and Methods**- A 35 year old male pt. visited our hospital with complaint of pain in bilateral hip joint (left>right) and groin region. For that *Panchatikta Ksheer basti karma* was planned along with *Sarvang snehana swedana*. The patient received *Panchatikta Ksheer Basti* for 15 days along with oral medication as part of their treatment regimen. **Result**- Better relief was found in symptoms of avascular necrosis and significant betterment in value of Hip range of motion. **Conclusion**- It was found that *panchatikta ksheer basti* along with *shamanausadh* is effective in the treatment of avascular necrosis of femoral head (*Asthimajjagatavata*).

KEYWORDS: AVN, *Asthimajjagatavata*, *Panchatikta Ksheer Basti*, *Ayurvedic* Management.

INTRODUCTION

Avascular necrosis (AVN) of the femoral head is a rare skeletal condition characterized by vague symptoms originating from the hip joint or lower pelvis. It involves a disruption of blood supply leading to ischemic necrosis, progressive osteocyte death, collapse of the joint surface, and loss of hip functionality. This condition, also known as osteonecrosis or osseous ischemia, primarily affects the femoral head but can occur in other skeletal sites like the knee, shoulder, and ankle. Risk factors include alcoholism, corticosteroid use, and medical disorders such as sickle cell disease and lupus erythematosus.[1,2]

In *Ayurveda*, musculoskeletal disorders like AVN fall under the category of *Vatavyadhi*. The pathophysiology, involving disturbances in bone remodeling pathways, angiogenesis inhibition, and coagulation abnormalities, predisposes bones to ischemia and AVN. Typically affecting individuals aged 30-50 years, males are more susceptible than females.

The symptoms of AVN align closely with those described as *Asthimajjagatavata* in *Ayurvedic* texts by *Acharya.*

Charaka, manifesting as breaking pain, hip joint pain, muscle wasting, weakness in the affected joint, and persistent pain-induced insomnia. Given the limitations of effective conservative management in modern medicine, our study aims to explore and propose safer and more effective *Ayurvedic* therapeutic approaches specifically targeting AVN of the femoral head.[3]

Disease review

According to *Ayurveda* literatures, human body is constituted of *Pancha* mahabhuta, *Tridoshas*, *Sapta dhatus* and *Trimalas*, among which *Tridoshas* (*Vata, Pitta, Kapha)* and *Sapta dhatus (Rasa, Rakta, Mamsa, Medo, Asthi, Majja, Shukra*) play a very important role in the pathogenesis of a disease and these are interconnected with each other.

Asthi-majjagata vata is an ailment under the account of *vatavyadhi* in which the morbid *vata dosha* gets lodged

in *Asthi* and *Majja* dhatu and bring about various symptoms as follows.

भेदोऽस्थिपर्वणां सन्धिशूलं मांसबलक्षयः।
अस्वप्नः सन्तता रूक च मज्जास्थि कुपितेऽनिले॥[4]

- *भेदोऽस्थिपर्वणां*(Breaking type of pain in bones)
- *सन्धिशूलं*(Pain in the joint)
- *मांसक्षय*(Muscle wasting)
- *बलक्षय*(Weakness)
- *अस्वप्नः सन्ततारुक्*(Insomnia due to continuous pain)

Nidana- No specific *nidana* is mentioned for *asthi-majjagata vata*, but as it is coming under *vatavyadhi*, so all the *nidana* is applicable. There are two major factors responsible for *vata dosha prakopa*. One is *Marga-avarodha* and another is *dhatu kshaya*.[5]

Samprapti- The *samprapti* of *Asthi-majjagata vata* can be understood from the following flow chart.

Nidan sevana

⇩

vata prakopa

⇩

Asthi and majja vaha srota dusti(kha vaigunyata)

⇩

sthana samshraya of vata dosha in Asthi and majja dhatu

⇩

vyakta lakshana

Chikitsa- *Nidana parivarjana, Vata shamaka chikitsa, Abhyantara* and *vahya snehana(maha sneha paryoga), Panchakarma, Basti, Tikta varga dravya siddha kshira* and *sarpi, Brumhana chikitsa* etc.[6]

Avascular Necrosis (AVN) which is otherwise known as Osteonecrosis/Aseptic Necrosis/Ischemic Necrosis, is defined as the bone cell death following a compromised vascular supply to the bone. It is most common in the femoral head, but can occur at other skeletal sites such as Knee, Ankle, shoulder and wrist.[7]

It affects all age groups, but more prevailing among the adults in third to fifth decades of life. True prevalence of osteonecrosis is likely quite underestimated because many patients are asymptomatic in the early stage of disease.[8]

Etiology

Etiology of AVN can be broadly classified in to two groups- i.e Traumatic and Non-traumatic.

Among non-traumatic causes, most common cause is the long-term use of corticosteroids and some often the alcohol abuse.

Non-traumatic causes include

Cellular toxicity (e.g.- chemotherapy, radiotherapy, thermal injury, smoking, alcohol)

Medication (e.g., corticosteroids, Bisphosphonates), Congenital disease (e.g., sickle cell disease, Thalassemia, Haemophilia, Gaucher disease, Congenital hip dislocation)

Orthopaedic conditions (e.g., slipped capital femoral epiphysis, developmental dysplasia of the hip), Intraosseous compression (e.g., haemorrhage, elevated bone marrow pressure), Dysbarism osteonecrosis, Lead poisoning, Electric shock etc.

Some case reports reveal that, diseases like Acute lymphoblastic leukaemia, fat embolism, pancreatitis, chronic liver disease, Gout, Hyperparathyroidism, Hyperlipidaemia, Hypercholesterolemia, Diabetes, Rheumatoid arthritis, Inflammatory bowel disease, AIDS may have the contribution as risk factors. Also, it may develop after an organ transplantation.[9]

Pathophysiology- Before understanding the pathophysiology we should know the blood supply of femoral head at least in brief. The vascularisation of femoral head includes- lateral and medial circumflex arteries which provide collateral circulation between femoral artery and internal iliac artery. The medial femoral circumflex artery and its branches supply most of the blood to the head and neck of the femur. The lateral and the medial femoral arteries wind anterolaterally and posteromedial respectively around the neck of the femur and ultimately anastomose with each other at the superolateral aspect of the femoral head. The small blood vessels, also termed as retinacular arteries, collectively give rise to epiphyseal arterial branches which supply the head and neck of the femur and believed to supply 80% of the femoral epiphyses. Compromise of this critical vascular system leads to AVN of the femoral head, which probably lead to the sequence of events is as follows.

1. Initial Infarct and Sclerosis: An area of bone tissue dies (necrosis) due to lack of blood supply, and a rim of bony thickening or sclerosis begins to form around the margins.
2. Subchondral Fractures: If the necrotic lesion is located within the weight-bearing region, fractures occur just below the cartilage surface (subchondral fractures).
3. Microfractures and Non-Healing: Repeated microfractures and continued weight-bearing prevent the initial fracture from healing completely, leading to new fractures.
4. Fracture Propagation: Secondary fractures propagate along the junction between the subchondral bone and the necrotic segment.
5. Femoral Head Collapse: Over time, these processes lead to flattening and eventual collapse of the femoral head.

6. Joint Deterioration: A nonspherical femoral head causes increased friction, erosion, and loss of cartilage within the acetabulum, resulting in degenerative changes and joint destruction. This pathological progression ultimately leads to the deterioration of the hip joint. Non-traumatic causes- Probable theories of pathogenesis of steroid induced AVN of femoral head.[10]

S. No.	Theories for the causation of AVN of FH	Effects/Pathways
1	Altered lipid metabolism	Hyperlipidemia Fat embolism Increased intraosseous-pressure Sinusoidal collapse
2	Intra-medullary fatty infiltration	Increased intramedullary adipogenesis Decreased osteogenesis Tamponade effect due to fat accumulation.
3	Inhibition of angiogenesis	Decreased vascular endothelial growth factor(VEGF) production Decreased collagen production Inhibition of physiological angiogenesis Decrease capillary growth Eventually decreased blood supply
4	Intramedullary hemorrhage	Arteriopathy
5	Primary cell death	Accumulation of lipid in the cell Compression of nucleus Cell death and necrosis
6	Apoptosis	Increased apoptosis of osteocytes Decreased number of osteoblast(decrease in bone formation and trabeculae width.) Deranged osteoclast function(reduction in bone turnover and remodeling) Disturbed process of repairing of bone
7	Cumulative stress theory	Steroids induced stress over osteocytes, make them vulnerable and they die.
8	Impaired healing process	Steroids induced osteocyte apoptosis disturbs the mechanosensory function of the osteocyte network Leading to impaired healing
9	Endothelial cell injury	Steroids cause endothelial cell injury, which further causes: Hypercoagulable state Thrombus formation Decrease in number of microvessels Increasing peripheral resistance to blood flow Increased arterial pressure increases thrombin levels, causing clot formation
10	Coagulation pathways	Decreases fibrinolytic activity by upregulating of plasminogen activator inhibitor – 1 (PAI – 1) gene Local dysregulation of coagulation at the level of FH
11	Vascular response to vasoactive substance	Decreased production of endothelial nitric oxide and prostacyclin Augmentation of endothelial-1 induced vasoconstriction of intraosseous femoral head arteries.

Other risk factors may lead to above mentioned pathways and result into this disease.

Clinical features- The early stage of AVN usually asymptomatic. Later stages may present with following symptoms-

1. Pain (Primary presenting symptom)- Typically located in the hip joint and sometimes radiate to groin, anterior thigh and knee joint. The severity of the pain depends upon the size of the infarct. The pain intensifies on the joint movement and can be persistent at rest.
2. Limitation of Range of motion (ROM) of affected hip joint.
3. Sometimes tenderness on palpation and stiffness of the affected hip joint.

Diagnostics- X-ray- It is the first line to rule out the subchondral fractures, sclerosis, bone collapse but it may be non-diagnostic at early stages.

MRI- It is the gold standard and higly sensitive. It can visualise the bone marrow changes, size and location of the necrotic area, depth of collapse, effect of acetabular

cartilage which is very helpful in deducing the prognosis and care plan.

To understand the disease progression most commonly used staging system is Steinberg staging system (on the basis of result obtained from radio imaging).[11]
Stage Features
0 Normal radiograph, bone scan, and MRI
INormal radiograph, abnormal bone scan and or magnetic resonance imaging
I A- Mild (involves less than 15% of the femoral head).
I B- Moderate (involves 15% to 30% of the femoral head)
I C- Severe (involves over 30% of the femoral head)
II Cystic and sclerotic change of the femoral head
II A- Mild (involves less than 15% of the femoral head)
II B- Moderate (involves 15% to 30% of the femoral head)
II C- Severe (involves more than than 30% of the femoral head)
III Subchondral collapse (crescent sign) without flattening of the femoral head
III A- Mild (involves under 15% of the femoral head)
III B- Moderate (involves 15% to 30% of the femoral head)
III C- Severe (involves over 30% of the femoral head)
IV Flattening of the femoral head/femoral head collapse
IV A- Mild (involves under 15% of the femoral head)
IV B- Moderate (involves 15% to 30% of the femoral head)
IV C- Severe (involves greater than 30% of the femoral head)
V Joint space narrowing and/or acetabular changes
V A- Mild
V B- Moderate
V C- Severe
VI Advanced degenerative joint disease

Management – The choice of management may be operative and conservative depending upon the staging of the lesion. In conservative management, various physical therapy, restricted weight bearing, alcohol cessation, steroid therapy discontinuation and analgesics are included. Vasodilators, statins, Bisphosphonates and anticoagulants are also used in attempt to revascularize the femoral head. Core-decompression, bone grafting, cellular therapies, osteotomy and joint replacement are preferable under operative management.[12]

Case study

- A 35-year-old male pt. Visited our hospital with complaint of pain in bilateral hip joint (left>right) and groin region. Pain aggregated during night and walking with limping on left side.
- Past history – pt. Has taken Allopathic treatment along but not get significant relief. No history of DM/HTN/Other systemic disorders.

- **Personal History**

Diet	**Vegetarian**
Appetite	**Normal**
Bowel	**Clear**
Sleep	**Disturbed due to pain**
Micturation	**Normal**

- **Family History - NAD**

Dashavidhapareeksha

Prakriti	***Pitta pradhan Kapha***
Vikriti	***Vata***
Sara	***Madhyama***
Samhanan	***Madhyama***
Pramana	***Madhyama***
Satwa	***Madhyama***
Satmya	***Madhyama***
Ahar shakti	***Madhyama***
Vyayam shakti	***Madhyama***
Vaya	***Madhyama***

- ***Ashta vidha pareeksha***

Nadi	***Vata pitta***
Mutra	***Prakrita***
Mala	***Prakrita***
Jivha	***Lipta***
Shabda	***Prakrita***
Sparsha	***Mridu***
Drishti	***Prakrita***
Akriti	***Madhyama***

- **General Examination**
- • Pallor – Absent • Icterus – Absent • Koilonychias – Absent • Lymphadenopathy – Absent • Edema – Absent

- **On MRI Hip Joint**

Impression
1. MRI study of both hip joints show bilateral Avascular Necrosis of Femoral Heads, Stage 3 on the left and Stage 2 on the right.
2. Mild to moderate left hip joint effusion.

MATERIALS AND METHODS

Treatment Plan

Shodhana chikitsa	**Doses**	**Anupan**
Panchatikta ksheer Basti –	**360ml × 15 days**	
Bahya Chikitsa- *Abhyanga – Vatashamaka Taila* *Swedana – Dashmoola Kwath*		

Abhyantar Chikitsa		
Maha manjisthadi kwath ghanavati	**500mg × Bd**	**Luke warm water**
Lakshadi Guggul	**500 mg × Bd**	**Luke warm water**

In this case, we are utilizing a refined *Panchakarma* procedure known as *Basti*, involving the rectal administration of a medicated decoction or medicated *Ghrit*. Specifically, we are employing *Panchatikta ksheer Basti*. To prepare this *Basti*, the physician requires the following ingredient

- *Guduchi* (Tinospora cordifolia)
- *Vasa* (Adhatoda vasica)
- *Neem* (Azadirachta indica)
- *Patol* (Trichosanthes dioica Roxb)
- *Kantakari* (Solanum surratense Burm.)
- *Ghrit* (medicated ghee)
- *Ksheer* (cow's milk)
- *Madhu* (honey)
- *Saindhav* (rock salt)
- *Basti* pot (enema pot)
- Rubber catheter No. 10

Preparation of *Panchatikta ksheer Basti* involves the following steps:

Mix 30 ml honey and 5 gm *Saindhav*, then add 70 ml go *ghrita*. Prepare a separate *ksheerpaka* with 30 gm *Panchatikta* by boiling it with 260 ml milk and 760 ml water, reducing it to approximately 260 ml, and then add it to the mixture of honey and ghrita.

Administration of *Panchatikta ksheer Basti*

The *Basti* is administered after *snehana swedana*, rectally with the patient positioned on their left side, with the right leg folded at the knee near the abdomen. A total of 360ml of *Panchatikta ksheer Basti* is administered to the patient.

Assessment Criteria

SUBJECTIVE CRITERIA

Bhedoasthiparvanam (Breaking pain of joints)
Mamskshaya (Muscular wasting of affected joint)
Balakshaya (weakness of affected joint)
Aswapna santata ruk (Insomnia due to pain)

1. VAS (visual analogue scale) for pain

Parameter	Criteria	Grading
Pain (vasscale)	(0)No pain	0
	(1-3) mild	1
	(4-6) Moderate	2
	(7-10) severe	3

2. Table

Parameter	Grading	Criteria
Gait	0	Normal gait
	1	Pain occasionally
	2	Walk with support, Mild pain
	3	Walk with support, Severe pain
	4	Unable to walk
Sleeplessness (*Aswapna santata ruk*)	0	Normal sound sleep
	1	Sleep disturbed 1-2 times in night
	2	Sleep disturbed 3-4 times in night
	3	Difficulty in falling asleep due to pain
	4	Difficulty in staying asleep due to continuouspain
MRC Muscle scale	0	No power
	1	Flicker of contraction only
	2	Movement with gravity eliminated
	3	Movement against gravity
	4	Movement against gravity & some resistance
	5	Normal power

OBJECTIVE CRITERIA

1. Range of movement of hip joint i.e., abduction, adduction, extension, flexion, medialrotation and lateral rotation.

S.No.	Sign & symptoms	Normal range
1.	Flexion of hip joint	110^0-120^0
2.	Extension of hip joint	10^0–15^0
3.	Abduction of hip joint	30^0-50^0
4.	Adduction	20^0-30^0
5.	Medial rotation	30^0–40^0
6.	Lateral rotation	40^0–60^0

RESULTS

Subjective Criteria

Assessment criteria	Before TreatmentRight hip	After Treatment	Before Treatment Left hip	After Treatment
Pain	1	0	4	3
Gait	1	0	2	1
Sleeplessness	1	0	1	1
Muscle strength	5	5	4	4

Objective criteria

S.No.	Objective	Normal	B.T. RT	A.T. RT	B.T. LT	A.T. LT
1.	Flexion of hip joint	110^0-120^0	100’	110’	95’	100’
2.	Extension of hip joint	10^0–15^0	20’	30’	10’	20’
3.	Abduction of hip joint	30^0-50^0	15’	20’	15’	25’
4.	Adduction	20^0-30^0	30’	35’	20’	25’
5.	Medial rotation	30^0–40^0	40’	40’	30’	25’
6.	Lateral rotation	40^0–60^0	30’	40’	20’	30’

DISCUSSION

From an *Ayurvedic* perspective, avascular necrosis (AVN) shows a predominant imbalance of *Vata Dosha* and vitiation of *Asthi Dhatu*. AVN occurs when blood supply to the femoral head diminishes due to factors like *Margavrodha* or *Abhighata*, leading to necrosis. According to commentator *Arundatta*, substances that are *Snigdha* (unctuous) and *Shoshana* (drying), and induce *Kharatwa* (roughness), promote *Asthi* (bone tissue) growth, as *Asthi* inherently has a rough nature. However, no single substance possesses both *Snigdha* and *Shoshana* properties. Therefore, milk (*Ksheer*) and ghee (*ghrut*), which are *Snigdha* in nature, are recommended to be used with bitter (*Tikta*) substances that have drying properties. The combination of *Ksheer Basti* (medicated milk enema) with *Tikta dravyas* (bitter substances) is suggested for repairing bone and cartilage degeneration, particularly in conditions affecting *Asthi*. This treatment approach aims to enhance vascularization and nourishment of bones, making it potentially beneficial for malnutrition-related bone diseases and various types of AVN.[13]

Avascular necrosis (AVN) of the hip joint typically arises from the blockage of small blood vessels that supply the femoral head, resulting in gradual necrosis due to diminished vascular circulation. This obstruction of the *Raktavaha Srota* (blood channels) is the primary factor leading to depletion of *Asthi Dhatu* in the hip joint. *Manjishtha Kwath*, characterized by its bitter (*Tikta*) and pungent (*Katu Rasa*) tastes, and its hot (*Ushna Virya*) potency, functions as a blood purifier (*Raktaprasadana*) and alleviates *Tridoshic* imbalances, potentially aiding in clearing blockages in the blood channels and enhancing blood flow to the affected area.[14]

Lakshadi Guggulu combines the anti-inflammatory properties of *Guggulu* (Commiphora wightii) and the bone healing properties of *Laksha* (Lacifer Lacca).

Guggulu is known for its ability to pacify vitiated *Vata* dosha and aid in reducing discomfort. Meanwhile, *Laksha* contributes to bone healing and wound healing, making the combination beneficial for conditions involving inflammation and fractures.[15]

CONCLUSION

Panchatikta Ksheer Basti, a therapeutic *basti* involving a combination of bitter and medicinal herbs in milk, aims to balance the vitiated *Vata dosha*, reduce inflammation, and promote healing of the femoral head.

Through this study, it becomes evident that

1. Therapeutic Efficacy: *Panchatikta Ksheer Basti*, along with other *Ayurvedic* modalities like herbal medications and dietary recommendations, has shown promising results in alleviating symptoms of avascular necrosis. By addressing the underlying pathophysiology and promoting tissue regeneration, it offers a holistic approach to treatment.

2. Complementary Therapy: Integrating *Panchatikta Ksheer Basti* into conventional treatment protocols for avascular necrosis could potentially enhance outcomes and reduce dependency on conventional medications or surgical interventions.

3. Long-term Management: *Ayurvedic* management, including regular follow-ups, lifestyle modifications, and continued adherence to treatment protocols, supports long-term management of avascular necrosis by addressing the root causes and maintaining overall health.

In conclusion, the case study underscores the role of *Panchatikta Ksheer Basti* and *Ayurvedic* management in providing a holistic, personalized approach to treating avascular necrosis of the femoral head. Further research and clinical trials would be beneficial to validate these findings and integrate *Ayurveda* more comprehensively into mainstream healthcare practices for such complex conditions.

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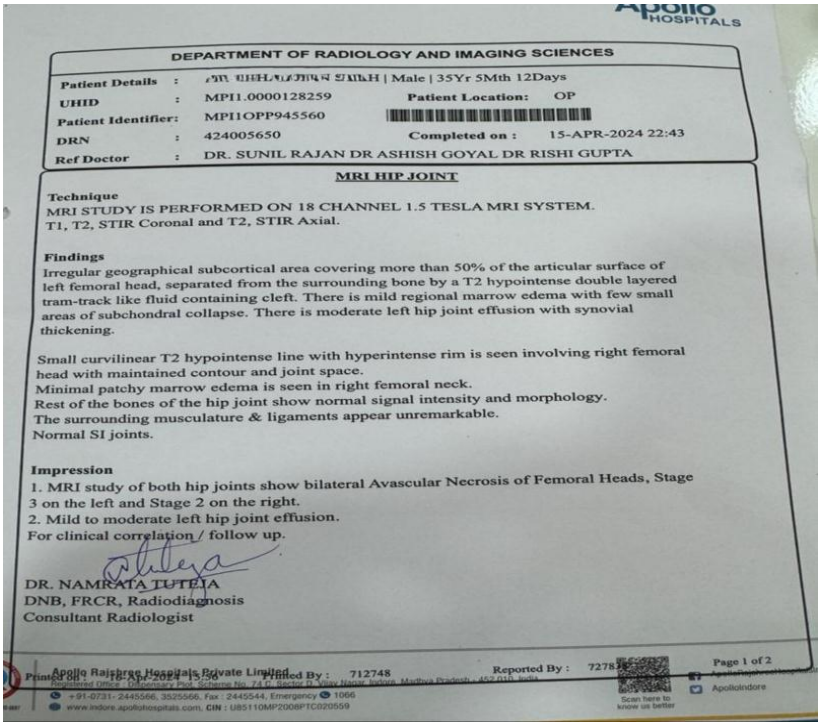

Apollo HOSPITALS

DEPARTMENT OF RADIOLOGY AND IMAGING SCIENCES

Patient Details	:	[illegible] | Male | 35Yr 5Mth 12Days	
UHID	:	MPI1.0000128259	**Patient Location:** OP
Patient Identifier:		MPI1OPP945560	
DRN	:	424005650	**Completed on :** 15-APR-2024 22:43
Ref Doctor	:	DR. SUNIL RAJAN DR ASHISH GOYAL DR RISHI GUPTA	

MRI HIP JOINT

Technique
MRI STUDY IS PERFORMED ON 18 CHANNEL 1.5 TESLA MRI SYSTEM.
T1, T2, STIR Coronal and T2, STIR Axial.

Findings
Irregular geographical subcortical area covering more than 50% of the articular surface of left femoral head, separated from the surrounding bone by a T2 hypointense double layered tram-track like fluid containing cleft. There is mild regional marrow edema with few small areas of subchondral collapse. There is moderate left hip joint effusion with synovial thickening.

Small curvilinear T2 hypointense line with hyperintense rim is seen involving right femoral head with maintained contour and joint space.
Minimal patchy marrow edema is seen in right femoral neck.
Rest of the bones of the hip joint show normal signal intensity and morphology.
The surrounding musculature & ligaments appear unremarkable.
Normal SI joints.

Impression
1. MRI study of both hip joints show bilateral Avascular Necrosis of Femoral Heads, Stage 3 on the left and Stage 2 on the right.
2. Mild to moderate left hip joint effusion.
For clinical correlation / follow up.

DR. NAMRATA TUTEJA
DNB, FRCR, Radiodiagnosis
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