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AVASCULAR NECROSIS OF THE FEMORAL HEAD AND ITS AYURVEDIC MANAGEMENT AS *ASTHIMAJJAGATA VATA*: A CONCEPTUAL REVIEW ARTICLE

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ABSTRACT

Introduction: Avascular Necrosis (AVN) of the femoral head, also known as osteonecrosis, is a progressively disabling disorder of young and middle-aged adults in which interruption of the blood supply to the femoral head leads to bone-cell death, subchondral collapse, and secondary osteoarthritis of the hip. Modern medicine has no proven pharmacological therapy that halts disease progression, and surgical options, though effective, are costly and imperfectly durable in a young, active population. Ayurveda does not describe AVN as a distinct clinical entity, but its causative process and symptomatology closely correspond to *Asthimajjagata Vata*, a *Vata*-predominant disorder in which vitiated *Vata Dosha* becomes lodged in the *Asthi* (bone) and *Majja* (marrow) *Dhatus*. **Methods:** This narrative review was compiled from the conceptual, disease-review, and drug-review chapters of a postgraduate Ayurveda dissertation, supplemented by the classical texts (*Charaka Samhita*, *Sushruta Samhita*, *Ashtanga Hridaya* and allied *Nighantus*) and contemporary orthopaedic and Ayurvedic literature cited therein. Modern-medicine sources were reviewed

for the epidemiology, pathogenesis, staging, and treatment of AVN, while classical Ayurvedic sources were reviewed for the *Nidana* (causation), *Samprapti* (pathogenesis), and *Chikitsa* (management) of *Vatavyadhi* and *Asthimajjagata Vata*, together with the composition and classical indications of *Gandha Tailam*, *Panchatikta Ghrita Guggulu*, and *Manjisthadi Ksheera Basti*. **Results:** The clinical picture of *Asthimajjagata Vata* - *Bheda Asthi Parvanam* (piercing bone-joint pain), *Sandhi Shoola* (joint pain), *Santata Ruk* (continuous pain), *Mamsabala Kshaya* (loss of muscle strength), and restricted movement - closely parallels the pain, stiffness, and progressive functional loss seen in AVN of the femoral head, supporting its use as the classical correlate of this condition. The *Samprapti* of *Asthimajjagata Vata* - channel obstruction (*Srotorodha*), *Rakta* and *Asthi Dhatu Kshaya*, and consequent *Vata* localisation in *Asthi-Majja Dhatu* - mirrors the modern description of vascular occlusion, osteocyte death, and subchondral collapse. *Gandha Tailam* and *Panchatikta Ghrita Guggulu*, both administered with *Manjisthadi Ksheera Basti*, emerge as classically indicated, pharmacologically plausible formulations combining *Vatahara* (*Vata*-pacifying), *Raktashodhaka* (blood-purifying), and *Brimhana-Rasayana* (tissue-nourishing and rejuvenating) actions relevant to arresting this pathological cascade. **Discussion:** Modern and Ayurvedic descriptions of AVN converge on a shared pathogenetic theme of impaired circulation and tissue starvation, and both systems recognise that once femoral head collapse occurs, options narrow toward joint replacement. The gap left by modern medicine's lack of a proven non-surgical disease-modifying treatment is precisely where Ayurveda's *Shodhana-Shamana* framework - combining internal oleation with *Basti* therapy - has classically been positioned, though direct clinical evidence remains limited to case reports and small comparative studies, underscoring the need for rigorously designed clinical trials.

KEYWORDS: Avascular Necrosis, Osteonecrosis of femoral head, *Asthimajjagata Vata*, *Gandha Tailam*, *Panchatikta Ghrita Guggulu*, *Manjisthadi Ksheera Basti*, *Vatavyadhi*, *Panchakarma*, Ayurveda.

1. INTRODUCTION

Avascular Necrosis (AVN) - also termed osteonecrosis, ischemic bone necrosis, or aseptic necrosis - is a serious, slowly progressive disease in which bone tissue dies because its blood supply is cut off. It most commonly affects the femoral head, though the knee, shoulder, ankle, and wrist may also be involved.[1]

The femoral head is particularly vulnerable because it depends on a small number of end-arteries with very limited collateral circulation. Once blood flow is interrupted, a well-defined cascade follows: osteocyte and marrow-cell death, subchondral microfracture, progressive collapse of the femoral head, and eventual secondary osteoarthritis of the hip.[2,3]

The causes of AVN are traditionally divided into traumatic factors (femoral neck fracture, hip dislocation) and non-traumatic factors, which include long-term corticosteroid use, chronic heavy alcohol consumption, sickle cell disease, autoimmune disorders, coagulation abnormalities, and other systemic and metabolic conditions. A substantial proportion of patients have no identifiable risk factor and are labelled idiopathic. AVN mainly affects young and middle-aged adults between 20 and 50 years of age, is more common in men, and is bilateral in more than half of affected patients.[1,3]

Symptoms are typically absent in the early stage, so diagnosis is often delayed; as the disease advances, patients develop deep groin pain, limp, hip stiffness, and progressive functional loss, ultimately requiring total hip replacement in most untreated cases.[1,5]

The global burden of AVN is considerable and appears to be rising. An estimated 10,000-20,000 new cases of femoral head osteonecrosis are diagnosed annually in the United States, accounting for close to 10% of all hip replacements performed each year, while India records an estimated 15,000-20,000 new cases annually, with long-term steroid use identified as the leading risk factor in studies from North India, followed by chronic alcohol use, trauma, and idiopathic disease.[1,4]

Because AVN strikes during the most productive years of life, it imposes a substantial physical, emotional, and economic burden on patients and health systems alike. Current treatment - analgesics, anti-inflammatory drugs, physiotherapy, restricted weight-bearing, core decompression, bone grafting, osteotomy, and total hip arthroplasty - can relieve symptoms and restore function, but no widely accepted non-surgical treatment reliably halts disease progression and preserves the native femoral head.[5,6]

Surgery remains costly, carries a risk of complications, requires prolonged rehabilitation, and the implant may not last as long in younger patients. This gap has driven interest in regenerative approaches such as mesenchymal stem cell therapy, bone marrow concentrate,

platelet-rich plasma, and growth-factor or exosome-based therapies, though questions about their long-term efficacy, accessibility, and cost persist.[6,7,8]

From an Ayurvedic perspective, AVN does not correspond to any single classically described disease, but its pathogenesis and clinical features are best understood through the concept of *Asthimajjagata Vata* - a state in which vitiated *Vata Dosha* becomes localised in the *Asthi* (bone) and *Majja* (bone marrow) *Dhatus* following *Srotorodha* (channel obstruction) arising from *Meda Dushti* (vitiation of fat tissue), disturbed *Dhatu Poshana* (tissue nourishment), and consequent *Vata* aggravation.[9]

When aggravated *Vata* settles in *Asthi* and *Majja Dhatu*, it produces *Sandhi Shoola* (joint pain), *Bheda Asthi Parvanam* (piercing bone-joint pain), *Santata Ruk* (continuous pain), *Mamsabala Kshaya* (loss of muscle strength), and restricted movement - a clinical picture that closely parallels AVN of the femoral head. This conceptual and pharmacological overlap forms the rationale for evaluating classical Ayurvedic formulations - *Gandha Tailam*, *Panchatikta Ghrita Guggulu*, and *Manjisthadi Ksheera Basti* - as a joint-preserving, non-surgical treatment strategy for this condition.[9]

2. Modern Concept of Avascular Necrosis

2.1 Epidemiology and Global Burden

The burden of AVN varies by country and is closely linked to local risk-factor prevalence. India records approximately 16,000 new cases annually; the United States around 15,000, with AVN accounting for over 10% of total hip replacements performed. In China, mean age at diagnosis is around 48 years, with male patients clustering between 30-50 years and female patients across a broader 20-60 year range. Japan records 2,500-3,300 new hip AVN cases annually, of which roughly a third are attributed to corticosteroid use, a fifth to chronic alcohol use, and over a third remain idiopathic.[10]

AVN itself is not associated with a high mortality rate, but it carries substantial long-term morbidity because it affects weight-bearing joints in young, active adults. Prosthetic joints tend to have a shorter functional life in younger patients, so most people with advanced disease eventually require more than one major surgical intervention. Apart from disease linked to sickle cell trait/disease (more common in people of African and Mediterranean descent), AVN shows no strong racial predilection; sex distribution is markedly male-predominant (approximately 8: 1) except in cases secondary to systemic lupus erythematosus,

which predominantly affects women. Peak incidence occurs in early middle age, typically the 30s and 40s, and over half of all cases are bilateral.[10,15]

2.2 Etiology and Risk Factors

AVN is a multifactorial disease that is broadly classified as traumatic or non-traumatic. Traumatic AVN follows femoral neck fracture or hip dislocation, both of which can directly shear or stretch the retinacular vessels supplying the femoral head.[11]

Non-traumatic causes account for over 80% of cases, the most common being long-term corticosteroid therapy and chronic heavy alcohol use - both of which disturb lipid metabolism, causing marrow fat-cell hypertrophy, raised intraosseous pressure, microvascular fat embolisation, and endothelial damage.[12,13]

Sickle cell disease causes vaso-occlusion through rigid, sickle-shaped erythrocytes; autoimmune conditions such as systemic lupus erythematosus are strongly linked to AVN, largely through the long-term corticosteroid therapy used to control disease flares.[14,15]

Legg-Calvé-Perthes disease is a distinct, idiopathic paediatric form of AVN (typically ages 4-10) that progresses through initial, fragmentation, reossification, and healed stages.[2]

2.3 Pathogenesis

AVN develops as a staged process. Interruption of blood flow through the femoral head's limited end-arteries - due to intraluminal blockage (thrombi, fat emboli, sickled erythrocytes) or extraluminal compression (marrow fat-cell hypertrophy raising intraosseous pressure) - causes marrow-cell death within 6-12 hours and osteocyte/osteoblast death within 24-48 hours.[3,12]

The body attempts repair at the necrotic-viable bone interface through creeping substitution, in which new capillaries and mesenchymal stem cells invade the dead area; however, osteoclastic resorption of necrotic bone typically outpaces new osteoblastic bone formation, weakening the subchondral structure. Continued weight-bearing then produces subchondral stress microfractures, delamination of bone from overlying cartilage (the radiographic crescent sign), and eventual mechanical collapse of the femoral head with secondary hip osteoarthritis.[3]

2.4 Clinical Features and Diagnosis

AVN is frequently asymptomatic in its early stages, which contributes to delayed diagnosis. As the disease advances, patients typically report deep groin or hip pain, worsened with weight-bearing, and progressive restriction of hip movement, sometimes with a limp.[16]

Physical examination may reveal tenderness, painful restriction of active and passive movement, and, in advanced disease, joint deformity and muscle wasting. Specific clinical tests include the sectoral sign, the prone instability test, the Thomas test (or measurement of limb-length discrepancy), and goniometric measurement of range of motion. Imaging - plain radiography, bone scan, CT, and MRI (the most sensitive modality for early disease) - remains central to diagnosis and staging.[16]

2.5 Staging Systems

Several classification systems guide treatment decisions in AVN, based on the natural progression of untreated disease.

System	Basis of Staging
Ficat and Arlet	Radiographic staging (Stage 1: normal X-ray; Stage 2: remodelling with cystic/sclerotic change; Stage 3: subchondral collapse/flattening; Stage 4: acetabular degeneration with joint-space narrowing). Widely used but does not quantify the extent of necrosis.
Steinberg	Adds bone scan/MRI findings and quantifies the percentage of femoral head involvement (Stages 0-6, each with mild/moderate/severe sub-grades); the most detailed system, though the full quantitative version is less used in routine practice because of its complexity.
ARCO	Combines radiography, CT, bone scan and MRI, and incorporates lesion size, crescent-sign length, percentage surface collapse, dome-depression depth, and lesion location (medial/central/lateral); proposed as the preferred system going forward (Stages 0-4).
Kerboul Necrotic Angle	Combined arc of femoral head necrosis measured on anteroposterior and lateral radiographs (or mid-sagittal/coronal MRI); outcomes are more favourable when the combined angle is below 200°.

Sources: Ficat and Arlet classification; Steinberg quantitative system; ARCO consensus classification.[17,18,19,20]

2.6 Current Management and Its Limitations

Non-operative treatment includes restricted weight-bearing (no longer regarded as adequate as a stand-alone measure), bisphosphonates (which reduce osteoclast-mediated bone turnover and may delay collapse or the need for arthroplasty), anticoagulants, statins and other

vasodilators (particularly relevant in steroid-associated AVN, where lipid-lowering may reduce marrow fat accumulation), extracorporeal shockwave therapy, pulsed electromagnetic field therapy, and hyperbaric oxygen therapy - the last of these remaining debated because of limited evidence specific to the hip.[6,21]

Hip-preserving surgery - core decompression, non-vascularised or vascularised bone grafting, porous tantalum implants, cell-based therapies (bone-marrow concentrate, platelet-rich plasma), and osteotomy - is generally reserved for pre-collapse disease, with reported success rates that vary widely by technique and stage.[21,22]

Once collapse and secondary osteoarthritis are established, total hip arthroplasty becomes the mainstay, but because AVN predominantly affects young, physically active patients, revision surgery is more often required, and reported loosening rates after primary arthroplasty in AVN patients range from 8-37%. Taken together, no medical treatment has yet been proven to reliably prevent or reverse the disease process, which sustains the case for exploring safe, affordable, joint-preserving alternatives such as those offered by Ayurveda.[6]

3. Ayurvedic Concept: *Asthimajjagata Vata*

3.1 *Vatavyadhi* and the Centrality of *Vata Dosha*

Vatavyadhi denotes the broad group of disorders that arise when *Vata Dosha* becomes vitiated. Because *Vata* governs nearly all physiological and neurological function in the body, its aggravation can affect multiple tissues and produce a wide range of clinical presentations.[23]

Vata is characterised by qualities that are dry (*Ruksha*), cold (*Sheeta*), light (*Laghu*), subtle (*Sukshma*), mobile (*Chala*), clear (*Vishada*), rough (*Khara*) and hard (*Daruna*), together with the functional properties of *Yogavahitva* (the capacity to carry and potentiate other substances), *Ashukaritva* (quick action) and *Achintya Virya* (unpredictable potency). Of its five functional subtypes - *Prana*, *Udana*, *Samana*, *Vyana* and *Apana Vayu* - *Vyana Vayu* governs circulation and the distribution of *Rasa* and *Rakta* throughout the body, and disturbance of its normal outward flow (*Anuloma Gati*) is considered central to disorders involving tissue degeneration.[24,25]

3.2 *Asthi-Majja Dhatu* and the *Ashraya-Ashrayi* Relationship

Asthi Dhatu (bone tissue) is classically regarded as the primary seat of *Vata Dosha* in the body; its hard, stable, and porous nature depends on *Vata's* own qualities, while *Majja* (marrow) fills the internal cavities of bone and remains closely dependent on *Asthi* for its nourishment.[23]

Because of this *Ashraya-Ashrayi Bhava* (mutual container-contained relationship), any disturbance in *Asthi Dhatu* affects *Vata*, and any weakening of *Asthi* in turn compromises the nourishment of *Majja*. *Acharya Charaka* describes *Dhatu Kshaya* (tissue depletion) and *Margavarana* (channel obstruction) as the two principal mechanisms of *Vata* aggravation: exposure to *Vata*-provoking *Nidanas* depletes *Asthi Dhatu*, which then impairs *Majja* nourishment through a sequential process termed *Uttarottara Dhatu Kshaya*, in which damage to one tissue propagates to the next in sequence.[23,26]

3.3 Etymology and the Concept of *Gatavata*

The term *Asthimajjagata Vata* is built from three components: *Asthi-Majja* (bone and marrow tissue), *Gata* (from the root *Gam*, meaning to move toward or become situated within), and *Vata*. Together they describe a condition in which vitiated *Vata* has travelled through the body's channels and settled (localised) within the bone-marrow complex, producing the disease's characteristic features.[24]

This falls under the broader classical category of *Gatavata*, itself a subtype of *Vatavyadhi*, which is further divided into *Ashayagata Vata* (*Vata* settling in body cavities) and *Dhatugata Vata* (*Vata* settling within one of the seven *Dhatus*). Structural weakness (*Khavaigunya*) created by ongoing tissue depletion provides the substrate in which circulating vitiated *Vata* preferentially localises - in this case, within *Asthi* and *Majja Dhatu*.[25]

Based on the mechanism of vitiation, *Asthimajjagata Vata* can be classified as *Dhatu Kshayajanya* (arising from tissue depletion due to ageing, poor nourishment, or chronic illness) or *Avaranajanya* (arising from obstruction of *Vata's* normal movement by *Kapha*, *Ama*, or other pathological substances). Based on causative factors, it may be further classified as *Nija* (primary, from internal dietary, lifestyle, metabolic, or ageing-related causes) or *Agantuja* (secondary, following trauma, repetitive occupational stress, or structural deformity). Because it primarily involves *Marma*, *Asthi*, and *Sandhi*, the condition is classified under the *Madhyama Rogamarga* (the middle disease pathway) as per the classical *Rogamarga* framework.[26]

3.4 *Samprapti* (Pathogenesis) and *Samprapti Ghataka*

Classical texts describe disease development through *Shadkriyakala*, the six sequential stages of *Sanchaya* (accumulation), *Prakopa* (aggravation), *Prasara* (spread), *Sthanasamshraya* (localisation), *Vyakti* (manifestation) and *Bheda* (complication).[27]

Applied to *Asthimajjagata Vata* and its correlation with AVN, sustained exposure to causative factors leads to progressive *Rakta Dhatu Kshaya* secondary to vascular/channel compromise, which impairs nourishment of *Asthi Dhatu* (*Asthi Dhatu Kshaya*); because *Majja* lies within *Asthi*, this is followed by *Majja Dhatu Kshaya*. This cascading tissue depletion aggravates *Vata* through the *Ashraya-Ashrayi* mechanism, and the resulting structural and functional derangement manifests clinically as *Asthimajjagata Vata*.[9,26]

Samprapti Element	Involvement
Dosha	*Vata*-predominant *Tridosha*
Dushya	*Mamsa*, *Asthi*, *Majja*, *Sandhi*
Srotas	*Asthivaha*, *Majjavaha*, *Mamsavaha*
Agni	*Vishama* (irregular)
Vyadhimarga	*Madhyama* (*Marma-Asthi-Sandhi*)
Adhisthana	*Asthi-Sandhi*
Srotodushti Prakara	*Sanga* (obstruction), *Ati Pravriti* (excess flow)
Svabhava	*Chirakari* (slow to develop)

Prognostically, *Charaka* notes that disorders of *Asthi* and *Majja Dhatu* are inherently difficult to treat because these are *Gambhira Dhatus* (deep-seated tissues); outcomes vary with disease severity, duration, and the patient's overall strength - broadly mirroring the modern observation that AVN outcome depends heavily on the stage at diagnosis and the extent of femoral head involvement at the time treatment is started.[26]

3.5 Correlating *Asthimajjagata Vata* with Avascular Necrosis

Although AVN has no exact one-to-one classical equivalent, the correspondence between its pathogenesis/symptomatology and that of *Asthimajjagata Vata* is close enough that Ayurvedic physicians and researchers widely accept the latter as its classical correlate.[9,28,29]

***Asthimajjagata Vata* (Classical features)**	**Avascular Necrosis (Clinical features)**
Bheda Asthi Parvanam - piercing/breaking pain in bone and joint	Deep, boring groin/hip pain
Sandhi Shoola - joint pain	Hip joint pain, worse on weight-bearing
Santata Ruk - continuous, unrelenting pain	Persistent pain as disease advances
Mamsabala Kshaya - loss of muscle strength	Muscle wasting and weakness in advanced disease
Aswapna - disturbed sleep from pain	Night pain / sleep disturbance in advanced cases
Sankocha / restricted movement	Progressive restriction of hip range of motion, limp
Srotorodha (channel blockage) as core pathology	Vascular occlusion / interrupted femoral head blood supply as core pathology
Asthi-Majja Dhatu Kshaya (tissue depletion)	Osteocyte death, marrow necrosis, subchondral collapse

This symptomatic and pathogenetic overlap supports treating AVN of the femoral head within the therapeutic framework developed for *Asthimajjagata Vata*, targeting *Vata* pacification, restoration of local circulation, and nourishment of depleted *Asthi* and *Majja Dhatu*.[9]

4. Procedure Review: *Panchakarma* and *Manjisthadi Ksheera Basti*

4.1 *Panchakarma* - Definition and Concept

Panchakarma, literally the fivefold action, is the flagship bio-purificatory system of Ayurveda designed to eliminate vitiated *Doshas* (*Vata*, *Pitta* and *Kapha*) from their root sites and to restore *Dhatu Samya* (tissue equilibrium). Unlike palliative (*Shamana*) measures that merely pacify aggravated *Doshas*, *Panchakarma* physically expels morbid material from the body through its natural channels of elimination, making it a radical rather than a suppressive form of treatment.[37]

Classical texts describe *Panchakarma* as the foundation of both preventive and curative Ayurveda: it is used to cleanse the body before administering *Rasayana* (rejuvenation)

therapy, to treat chronic and deep-seated (*Kostha-gata* and *Marma-gata*) disorders, and to correct the *Agni* (metabolic fire) at the level of the tissues.[37]

The five classical procedures are *Vamana* (therapeutic emesis, targeting *Kapha* seated in the chest/stomach), *Virechana* (therapeutic purgation, targeting *Pitta* seated in the small intestine/liver), *Basti* (medicated enema, targeting *Vata* seated in the colon/*Pakwashaya*), *Nasya* (nasal instillation, targeting *Doshas* above the clavicle), and *Raktamokshana* (therapeutic bloodletting, targeting vitiated *Rakta*). Among these, *Basti* occupies a unique position: because *Vata* is regarded as the principal regulator of all bodily movement and of the other two *Doshas*, and because the colon is its primary seat, *Basti* is described as *Ardha Chikitsa* - half of the entirety of Ayurvedic treatment.[26,37]

Every *Panchakarma* procedure, including *Basti*, follows a three-phase structure: *Purva Karma* (the preparatory phase of *Deepana-Pachana*, followed by *Snehana* and *Swedana* to loosen and mobilise *Doshas*), *Pradhana Karma* (the main purificatory action, administered according to the patient's *Bala*, *Koshtha* and disease status), and *Paschat Karma* (the post-procedure phase of *Samsarjana Krama*, a graded dietary regimen, together with rest and lifestyle restraint to allow *Dhatvagni* to stabilise and consolidate the therapeutic gain).[37]

4.2 *Basti* - Classification and Dosage

Acharya Charaka describes *Basti* as a treatment in which a medicine, prepared according to classical texts, is instilled into the body through the rectum, where it breaks down accumulated *Purisha* (stool) and *Dosha*, spreads its oily quality and strength through the body, and is then eliminated along with the loosened stool and *Dosha*.[34]

Basti is classified into two main types by the medicine used: *Niruha Basti* (also called *Asthapana* or *Kashaya Basti*), which mainly uses an herbal decoction; and *Sneha*-predominant *Basti*, which is further divided by the quantity of *Sneha* used into *Sneha Basti*, *Anuvasana Basti*, and *Matra Basti* - the last being a small-dose oil enema that can be given at any time without special dietary or seasonal restriction, valued for its *Balya* (strengthening), *Brimhana* (nourishing) and *Vataroganashaka* (*Vata*-disorder-relieving) actions.[35,36]

In *Kshira Basti*, milk (*Kshira*) replaces oil or ghee as the *Sneha*, administered in doses comparable to a *Matra* or *Anuvasana Basti*. Classical texts describe milk as *Guru* (heavy), *Snigdha* (unctuous), *Madhura* (sweet) and *Shita* (cooling) in quality, and *Brimhana*

(nourishing) and *Balya* (strength-promoting) in action, pacifying both *Vata* and *Pitta* - making *Kshira Basti* gentle, soothing, and well suited to patients who are weak, emaciated, or sensitive to more heating or drying forms of *Sneha*.[36]

Acharya Charaka explains the mechanism of action of *Basti* thus: medicine retained in the *Pakwashaya* (large intestine) draws out accumulated *Dosha* from every part of the body, from the feet to the head, through its *Virya* (potency) - just as the sun draws up moisture from the earth.[34]

4.3 *Manjisthadi Ksheera Basti* - Overview, Indications and Contraindications

Manjisthadi Ksheera Basti is a specialised *Tikta-Ksheera* (bitter-milk) *Basti*, a hybrid preparation that fuses the *Shodhana* (purificatory) property of bitter (*Tikta*) herbs with the *Brimhana* (nourishing) property of milk. Clinically it is regarded as a valuable intervention for *Asthi*- and *Majjavaha Sroto Vikaras* - disorders of the bone- and marrow-carrying channels - including *Sandhigata Vata* (osteoarthritis), osteoporosis, and avascular necrosis of bone.[38,39]

It is prepared from *Manjistha*, *Amalaki*, *Haritaki*, *Bibhitaki*, *Kutaki*, *Vacha*, *Daruharidra*, *Guduchi* and *Nimba Twak* processed with milk, and administered as a 360 ml enema over a course of 16 days. When designed for prolonged, low-depletion use, a similarly balanced formulation is administered as a *Yapana Basti* - a mild, life-sustaining category of enema that combines cleansing and nourishment closely enough to be given on consecutive days, without the strict alternation with plain oil-enemas required of stronger *Niruha Basti*.[38]

Indications: *Sandhigata Vata* of the hip, knee and spine; osteoporosis and generalised *Asthi-Kshaya* (bone depletion); avascular necrosis of the femoral head in the ischaemic/pre-collapse stage; and chronic *Vata*-predominant joint and lower-back disorders.[38]

Contraindications: Active diarrhoea, dysentery, or acute gastrointestinal inflammation; rectal or anal pathology (fissure, fistula, active haemorrhoids, rectal prolapse); severe debility, shock, or uncontrolled systemic illness; pregnancy (unless specifically directed by the treating physician); and administration immediately after a full meal, since *Basti* is timed to an empty or semi-empty stomach.

Manjisthadi is well known for its blood-purifying (*Rakta-Prasadana*) and channel-cleansing (*Srotoshodhana*) properties, helping correct blood-tissue vitiation, support unobstructed

circulation through the microchannels, and promote nourishment of the affected tissues, while the milk base and other ingredients pacify *Vata*, improve tissue metabolism, and nourish *Asthi* and *Majja Dhatu*. By combining the therapeutic effects of *Basti* and milk therapy, *Manjisthadi Ksheera Basti* provides nourishment (*Brimhana*), strength (*Balya*), and rejuvenation (*Rasayana*), supporting tissue repair and helping prevent further degeneration in *Asthimajjagata Vata* correlated with AVN of the femoral head.[39,40]

5. Rationale of Ayurvedic Management

5.1 The *Shodhana-Shamana* Framework

Ayurveda approaches *Vata* disorders through two complementary strategies: *Shodhana* (purification therapies that eliminate vitiated *Dosha* from its root) and *Shamana* (palliative medicines that pacify aggravated *Dosha* without expelling it). *Charaka* observed that disease treated with *Shodhana* is far less likely to recur, whereas disease managed with *Shamana* alone may relapse once medication is withdrawn - hence the classical preference for administering *Shamana Aushadhi* following a course of purification, to achieve a more durable result.[26]

In simple *Vata* disorders without *Avarana* (obstructive pathology), *Snehana* (oleation) with medicated ghee, oil, or marrow-based preparations is the primary treatment, given to counter *Vata's* dryness and roughness; this is typically followed by *Swedana* (sudation) to mobilise vitiated *Dosha*, and, where required, mild *Shodhana* measures such as *Mridu Virechana* and *Anulomana* to restore *Vata's* normal downward movement. Among all treatments for *Vata* disorders, *Basti* (medicated enema) is considered the most effective, and *Charaka* specifically recommends processing milk and ghee with bitter (*Tikta*) drugs, combined with *Basti* therapy, for disorders of *Asthi Dhatu* - the classical basis for combining internal oleation (*Gandha Tailam* or *Panchatikta Ghrita Guggulu*) with *Manjisthadi Ksheera Basti* in the management of *Asthimajjagata Vata*.[26,27]

Because *Asthimajjagata Vata* simultaneously involves depleted *Asthi-Majja* tissue and an aggravated *Vata* obstructing its nourishment, an effective formulation must work in three directions at once: pacifying *Vata* (*Vatahara*), restoring circulation so that nutrition reaches the bone (*Raktashodhaka*), and rebuilding tissue strength (*Balya*, *Brimhana*, *Rasayana*). Formulations built around *Guggulu*, *Manjistha*, and tissue-nourishing herbs are traditionally preferred for this reason, and this rationale underlies the selection of *Gandha Tailam* and

Panchatikta Ghrita Guggulu, each combined with *Manjisthadi Ksheera Basti*, for comparative evaluation in *Asthimajjagata Vata*.[28,29,30]

5.2 Drug Review

***Gandha Tailam*.** A polyherbal oil formulation containing 30 ingredients in equal parts (including *Kakolli*, *Ksheera Kakoli*, *Jeevak*, *Rishbhaka*, *Mudgaparni*, *Mashaparni*, *Guduchi*, *Manjistha*, *Ashwagandha*, *Yashtimadhu*, *Vidarikanda* and *Devadaru*, among others), administered orally at 20 ml twice daily with lukewarm water for 30 days. It is traditionally indicated in *Asthi-Bhagna* (bone fracture) and *Sandhigata* (joint) disorders and is credited with *Balya* (strengthening), *Brimhana* (nourishing), *Asthi-Sandhanakara* (bone-healing), and *Vatahara* (*Vata*-pacifying) properties, largely contributed by its *Jivaniya* and *Vayahsthapana*-group ingredients that are classically used for tissue rejuvenation and strength restoration.[30]

***Panchatikta Ghrita Guggulu*.** A classical medicated-ghee-and-*Guggulu* tablet preparation containing five bitter herbs (*Nimba Twak*, *Guduchi*, *Vasa*, *Patola*, *Kantakari*) processed with cow's ghee, combined with a *Prakshepa Dravya* group of 24 additional herbs and *Shuddha Guggulu*, administered as two 250 mg tablets twice daily with lukewarm water for 30 days. Its *Deepana* (digestive-stimulant), *Pachana* (digestive), *Vata-Kapha Shamaka* (*Vata-Kapha* pacifying), *Asthi Dhatu Poshana* (bone-nourishing), and *Rasayana* (rejuvenating) properties make it a preferred formulation for *Asthi Dhatu* disorders; experimental and clinical observations suggest it may support bone metabolism, reduce inflammation, and aid tissue repair.[31,32,33]

***Manjisthadi Ksheera Basti*.** As detailed in Section 4.3, this milk-based enema of *Manjistha*, *Amalaki*, *Haritaki*, *Bibhitaki*, *Kutaki*, *Vacha*, *Daruharidra*, *Guduchi* and *Nimba Twak* is administered as a 360 ml enema over 16 days, contributing *Vatahara*, *Raktashodhaka*, and *Asthi-Majja-poshaka* actions that complement the oral formulations.[38,39]

5.3 Probable Mode of Action

Combining oral *Sneha*-based medication with *Basti* therapy is intended to address both the systemic (*Vata* aggravation, tissue depletion) and local (channel obstruction, poor femoral head nourishment) dimensions of *Asthimajjagata Vata*.[9]

Conceptually, the *Vatahara* action of the study drugs is expected to reduce pain and stiffness by pacifying *Vata's* characteristic dryness, mobility, and roughness; their *Raktashodhaka* and *Srotoshodhana* actions are expected to improve microcirculation to the femoral head, analogous to the modern therapeutic goal of restoring blood supply to necrotic bone; and their *Balya*, *Brimhana*, and *Rasayana* properties are expected to support the nourishment and structural rebuilding of depleted *Asthi* and *Majja Dhatu* - conceptually paralleling the modern aim of encouraging creeping substitution and osteoblastic bone formation to outpace osteoclastic resorption.[28,39]

Because *Basti* therapy also acts through absorption at the level of the *Pakwashaya* (the classically described site of *Vata's* origin) and systemic distribution of its nourishing and *Vata*-pacifying constituents, it is considered to work synergistically with the oral medication rather than merely as a local rectal intervention.[34]

6. DISCUSSION

Read together, the modern and Ayurvedic literatures on AVN converge on several points. Both recognise the disease as slowly progressive, driven by a vicious cycle of impaired circulation and tissue starvation, and both acknowledge that once bone collapse occurs, options narrow sharply toward joint replacement. Modern medicine's principal gap - the absence of a proven, disease-modifying non-surgical treatment - is precisely the space in which Ayurveda's *Shodhana-Shamana* framework, and specifically the combination of *Vatahara/Raktashodhaka/Rasayana*-yielding formulations with *Basti* therapy, has classically been positioned.[6,26]

The correlation between *Asthimajjagata Vata* and AVN is symptomatic and pathogenetic rather than exact, and this is an important limitation to state plainly: *Asthimajjagata Vata* is a broader *Vyadhi* category, and its use as a classical correlate for AVN, while widely accepted in contemporary Ayurvedic literature, does not amount to a one-to-one disease equivalence.[9,28]

The proposed mechanisms by which *Gandha Tailam*, *Panchatikta Ghrita Guggulu*, and *Manjisthadi Ksheera Basti* might benefit AVN - improved microcirculation, reduced local inflammation, and enhanced tissue nourishment - are conceptually plausible and are broadly consistent with several ingredients (*Guggulu*, *Manjistha*, *Guduchi*, *Ashwagandha*) that have

documented anti-inflammatory, immunomodulatory, or bone-metabolism-related actions in the wider pharmacological literature; however, much of the direct clinical evidence specific to AVN remains limited to case reports and small studies rather than large, controlled trials.[28,29]

A rigorously conducted comparative clinical study - evaluating *Gandha Tailam* versus *Panchatikta Ghrita Guggulu*, both combined with *Manjisthadi Ksheera Basti*, in patients with radiologically confirmed AVN of the femoral head - is therefore a logical and necessary next step to generate objective, subjective, and radiological outcome data that can be compared against the natural history and modern staging systems (Ficat and Arlet, Steinberg, ARCO) described above.[17,18,19]

From a public-health perspective, the case for pursuing this line of enquiry is strong: AVN affects patients in their most economically productive years, current surgical treatment is costly and imperfectly durable in the young, and a safe, low-cost, joint-preserving adjunct or alternative would meaningfully reduce the long-term burden of repeated surgery.[4,5]

At the same time, any integrative claim needs to be tempered by acknowledging that Ayurvedic treatment is best positioned, on current evidence, for pre-collapse or early-collapse stages (comparable to Ficat-Arlet I-II or Steinberg 1-3), where the pathological process is still substantially one of impaired circulation and tissue depletion rather than established mechanical failure of the joint surface; once significant collapse and secondary osteoarthritis have occurred, surgical management remains unavoidable in both systems of medicine.[17,18]

7. CONCLUSION

Avascular Necrosis of the femoral head remains one of orthopaedics' more difficult problems: a disease with a well-understood vascular pathogenesis but no proven disease-modifying medical therapy, in which surgery, while effective, is costly and imperfectly durable in the young patients it most often affects.[6]

Ayurveda's classical concept of *Asthimajjagata Vata* offers a coherent, symptomatically and pathogenetically consistent framework within which to understand and treat this condition, built on the interlinked principles of *Vata* pacification, blood-tissue purification, channel clearance, and *Asthi-Majja* nourishment. *Gandha Tailam* and *Panchatikta Ghrita Guggulu*, each administered with *Manjisthadi Ksheera Basti*, represent classically grounded,

pharmacologically plausible treatment options whose comparative efficacy merits rigorous clinical evaluation.[9,39]

Well-designed comparative clinical studies, using standardised subjective and objective outcome measures alongside modern radiological staging, are needed to establish the place of these Ayurvedic formulations in the joint-preserving, non-surgical management of *Asthimajjagata Vata* correlated with Avascular Necrosis of the femoral head.

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