ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 11| Issue: 10| October 2025|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2025: 8.691 || ISI Value: 1.188

A COMPREHENSIVE PERSPECTIVE OF AVASCULAR NECROSIS (AVN) AND ITS CLINICAL PARALLELS WITH '*ASTHI-MAJJĀGATA VĀTA*': TRADITIONAL INSIGHTS FROM *MAHĀSNEHA* AND *SNEHANA CHIKITSĀ*

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ABSTRACT

Avascular Necrosis (AVN), or osteonecrosis, is a progressive ischemic disorder characterised by compromised vascular supply leading to necrosis of bone and marrow elements, resulting in collapse and functional impairment, most commonly affecting the femoral head. In Ayurvedic parlance, it closely correlates with Asthi-Majjāgata Vāta, a Vāta-pradhāna vyādhi described in Charaka Saṃhitā, presenting with sandhiśūla (joint pain), māṃsa-bala-kṣaya (muscle wasting), and Asthiparva bheda (bony crepitus). The underlying pathology in both systems involves tissue depletion (dhātu-kṣaya) and obstruction of microchannels (srotorodha), analogous to ischemia and necrosis in modern understanding. Ācārya Charaka prescribes Mahāsneha – a formulation comprising taila, vasā, ghṛta, majjā, and kṣīra processed with Daśamūla, Rasnā, Chitraka, and Jīvanīya gaṇa – for Sirā-Majjā-Asthi-gata Vāta, providing deep nourishment, unctuousness, and Vāta-pacifying action. Therapeutic measures such as Snehana, Svedana, Basti, and Rasāyana chikitsā counteract dryness, promote regeneration, and enhance microcirculation, aligning conceptually with angiogenic and anti-inflammatory approaches in modern regenerative medicine. This integrative correlation suggests that AVN and Asthi-Majjāgata Vāta represent a common degenerative-ischemic continuum interpreted through different epistemological frameworks. Therefore, an integrative management approach combining Ayurvedic Snehana-Basti therapy and Rasāyana principles with modern regenerative modalities may offer synergistic benefits in restoring bone vitality, preventing collapse, and improving joint function.

KEYWORDS: *Avascular Necrosis, Asthi-Majjāgata Vāta, Osteonecrosis, Dhātu-kṣaya, Mahāsneha, Snehana, Basti.*

INTRODUCTION

Avascular necrosis (AVN), also known as osteonecrosis, is a pathological condition characterised by the death of bone tissue due to compromised blood supply, leading to structural collapse, pain, and progressive joint dysfunction. It most commonly affects the femoral head but can involve other bones such as the humerus, knee, and talus. The earliest medical description of AVN dates back to Alexander Monro Primus in 1738, who first identified bone necrosis associated with vascular insufficiency [1]. Later, Jean Cruveilhier (1829–1842) provided detailed anatomical observations of femoral head deformities resulting from disrupted vascularity [2]. In the modern era, Henry J. Mankin (1962) systematically reported 27 cases of non-traumatic osteonecrosis, which established the foundation for contemporary understanding and classification of AVN [3]. The condition has since been recognised as multifactorial in origin, with causes ranging from trauma and corticosteroid use to metabolic and idiopathic factors. From an Ayurvedic perspective, the pathogenesis may be correlated with *Asthi-Majjagata Vāta*, wherein vitiation of *Vāta* leads to degeneration and necrosis of bone tissue, reflecting the intricate interplay of *Dhātu kṣaya* and *Srotorodha*. Thus, exploring AVN through both biomedical and Ayurvedic paradigms offers a comprehensive understanding for developing integrative and preventive therapeutic approaches.

Definition of AVN

Avascular Necrosis (AVN), also known as Osteonecrosis, is a progressive and degenerative bone disorder resulting from the loss of blood supply to the subchondral region of bone, leading to ischemic necrosis and eventual structural collapse. The condition primarily affects the epiphyseal ends of long bones, particularly those involved in weight-bearing joints such as the femoral head [4]. The compromised vascular perfusion causes death of osteocytes and marrow components, followed by microfracture formation and subchondral collapse, which may ultimately deform the articular surface and impair joint integrity. [5] Although the femoral head is the most frequently affected site, AVN can occur in any bone and may present as a mono- or polyostotic condition, involving one or multiple

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bones either simultaneously or sequentially. Early diagnosis and timely intervention are essential to prevent irreversible joint damage and preserve function. [6]

Avascular Necrosis (AVN), also known as Osteonecrosis, Aseptic Necrosis, Ischemic Bone Necrosis, Osteochondritis Dissecans, or Chandler's Disease, is a pathological process characterised by bone cell death due to loss of blood supply to the subchondral region. The resulting ischemia leads to necrosis of osteocytes and bone marrow elements, followed by structural collapse and subsequent joint dysfunction. [7-9] The condition most commonly involves the femoral head, but other frequent sites include the knee, shoulder, and ankle joints, where the vascular anatomy predisposes to compromised circulation. [10]

Epidemiologically, AVN has a reported annual incidence of approximately 10,000–20,000 cases in the United States, and it is most prevalent among individuals aged 20–50 years, often affecting those in their most active and productive years of life. [11-13] The disease may occur secondary to trauma, corticosteroid use, alcohol abuse, or systemic disorders, though many cases remain idiopathic. Early detection and appropriate management are critical to prevent irreversible joint destruction and maintain function.

Aetiology of Avascular Necrosis (AVN)

Avascular Necrosis (AVN) arises from multiple factors that impair bone vascularity, leading to ischemia and necrosis of osseous tissue. The causes can be broadly categorized into traumatic and non-traumatic origins.

1. Trauma:
Fractures or joint dislocations, especially around the hip, may directly damage the vascular network, reduce blood flow and initiate bone necrosis [14]. Radiation therapy may further aggravate vascular injury [15].
2. Fat Embolism and Vascular Occlusion:
Deposition of fat within small blood vessels can obstruct intraosseous circulation, resulting in cellular death [16].
3. Systemic Diseases:
Haematological and metabolic disorders such as sickle cell anaemia and Gaucher's disease may impair blood flow and predispose to AVN [17].
4. Arterial Pathology:
Inflammation, thrombosis, or arterial wall damage can block vascular channels, compromising bone perfusion [18].
5. Corticosteroid Therapy:
Long-term, high-dose corticosteroid use is a leading non-traumatic cause of AVN (≈35% of cases), primarily due to fat deposition and reduced marrow perfusion [19-20].
6. Alcohol Consumption:
Chronic alcohol intake alters lipid metabolism, causes fat emboli, and diminishes bone vascularity, leading to necrosis [21].
7. Smoking:
Nicotine induces vasoconstriction and reduces oxygen delivery to bone tissue, increasing the risk of AVN [22].
8. Medical Treatments:
Radiation and organ transplant procedures can cause vascular injury and metabolic imbalance, predisposing to osteonecrosis [23].

Pathogenesis of Avascular Necrosis

Avascular necrosis results from compromised blood supply leading to bone ischemia and subsequent osteocyte death. Traumatic causes interrupt vascular channels directly, while non-traumatic factors such as corticosteroid use and alcohol induce intravascular coagulation, fat embolism, or extravascular compression. These events culminate in decreased perfusion and ischemia, followed by osteocyte necrosis and ineffective repair. Progressive structural weakness ensues, ultimately causing subchondral collapse and joint dysfunction.

Symptoms of Avascular Necrosis

The clinical manifestations of avascular necrosis (AVN) often develop insidiously, with symptoms appearing gradually over weeks or months. Early stages present as **intermittent pain that appears and eases when pressure is applied to the affected bone and then relieved**, indicating early ischemic changes. As the condition progresses, pain becomes persistent—even at rest or during the night—accompanied by stiffness and restricted joint movement. Advanced stages lead to limping and difficulty in climbing stairs, standing, or walking, particularly when the hip or knee joints are involved. These progressive symptoms reflect the continuum from vascular compromise to structural collapse of the bone. [24]

Differential Diagnosis

Table No. 1: Differential Diagnosis for Avascular Necrosis

No.	Disease	Inclusion Criteria	Exclusion Criteria
1.	Bone marrow oedema (Transient osteopenia)	Pain, stiffness, oedema of joints, and reduced range of motion (ROM).	Increased interstitial fluid within bone marrow.
2.	Inflammatory synovitis	Joint pain, swelling, stiffness, and difficulty moving a joint.	The joint appears swollen and feels puffy or boggy to the touch.
3.	Neoplastic bone	Persistent bone pain that increases at night.	Redness or inflammation over a bone associated with a lump.
4.	Osteoarthritis	Loss of flexibility, crepitus, pain, and stiffness.	Presence of bone spurs.
5.	Osteomyelitis	Bone pain, local swelling, and night pain.	Open wound with pus, fever, and chills.
6.	Osteoporosis	Bones break easily; pain is caused by a collapsed bone.	Spinal malfunction with stooped or hunched posture.

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7.	Rheumatoid arthritis	Pain, joint swelling, and reduced ROM.	Morning stiffness, fever, and loss of appetite.
8.	Septic arthritis	Swelling, reduced ROM, and joint stiffness.	Sudden onset of pain, fever, and warmth over the joint.
9.	Soft tissue trauma	Swelling, pain, and loss of power.	Bruising and sudden onset of pain.
10.	**Avascular necrosis**	*Intermittent pain that appears and eases when pressure is applied to the bone and then is relieved.*	—

Different Sites Prone to Avascular Necrosis

Necrosis of the Hip Joint

Femoral head osteonecrosis is categorized as traumatic or atraumatic, with up to 70% of atraumatic cases being bilateral. The Ficat & Arlet system outlines four radiographic stages from early pain without imaging findings to advanced collapse with secondary osteoarthritis. The Steinberg classification incorporates MRI to detect preclinical lesions and assess necrotic extent more precisely.

Necrosis of the Knee Joint

Spontaneous osteonecrosis of the knee (SONK) predominates, often seen in women over 50, commonly affecting the medial femoral condyle. Pain mimics a meniscal tear. The Koshino classification defines four stages, from clinical symptoms without radiologic change to advanced degeneration with osteosclerosis and osteophyte formation.

Necrosis of the Shoulder Joint

Usually post-traumatic or related to prolonged corticosteroid use, humeral head osteonecrosis is staged by the Cruess classification, progressing from MRI-only lesions (Stage I) to articular collapse and glenoid involvement (Stage V).

Necrosis of the Talus

Commonly follows neck fractures with associated ankle or subtalar dislocation. Due to limited vascularity, the Hawkins classification tracks progression, where the absence of the Hawkins sign on radiographs (6–8 weeks post-injury) suggests evolving necrosis.

Necrosis of the Scaphoid

Also called Preiser's disease, it arises from trauma, steroids, alcoholism, or connective tissue disorders. It usually affects middle-aged men's dominant wrist. The Herbert–Lanzetta classification outlines four stages—from proximal pole changes to carpal collapse.

Table No. 2: Various sites prone to Avascular Necrosis

Joint Involved	Common Type / Aetiology	Key Classification System	Stage Summary / Clinical Highlights
Hip (Femoral Head)	Traumatic / Atraumatic (70% bilateral)	*Ficat & Arlet*, *Steinberg*	Stage I–IV: from pain without radiographic change → femoral collapse and osteoarthritis. MRI aids early detection.
Knee (SONK)	Spontaneous, secondary, or post-arthroscopic	*Koshino*	Four stages: from symptomatic onset → degenerative phase with sclerosis and osteophytes. Common in females, the medial femoral condyle.
Shoulder (Humeral Head)	Post-traumatic, corticosteroid-related	*Cruess*	Stage I–V: MRI-only lesion → humeral head collapse → glenoid degeneration.
Talus	Post-traumatic (neck fractures, dislocation)	*Hawkins*	Progresses with loss of Hawkins sign at 6–8 weeks; indicates avascular necrosis.
Scaphoid (Preiser's Disease)	Trauma, steroid use, alcohol abuse	*Herbert & Lanzetta*	Four stages: proximal pole changes → fragmentation → carpal collapse. Common in the dominant wrist of middle-aged men.

Treatment Protocol for Avascular Necrosis (AVN)

Conservative Management

In the early stage of AVN (pre-collapse) the focus is on symptom relief and vascular preservation. Non-steroidal anti-inflammatory drugs (NSAIDs) such as ibuprofen, naproxen, or aspirin are employed for pain control. Osteoporosis-modifying agents (e.g., raloxifene, alendronate) may slow pathological bone resorption. Lipid-lowering therapies (e.g., lovastatin, rosuvastatin, pitavastatin) help reduce intravascular lipid deposition that may obstruct bone microvasculature. Vasodilators such as iloprost may enhance regional perfusion, and anticoagulants like warfarin are considered when thrombophilia is implicated. Physical rest, restricted weight-bearing (e.g., via crutches), and guided physical-therapy exercises maintain joint mobility. Adjunctive electrical stimulation may promote osteogenesis and revascularisation in select patients.

Para-Surgical / Minimally Invasive Management

When conservative measures fail or imaging indicates early structural changes, para-surgical interventions are indicated. Core decompression removes the necrotic core, reducing intraosseous pressure and promoting angiogenesis. Bone grafting (vascularised or non-vascularised) reinforces subchondral bone and restores structural integrity. Load-shifting osteotomy (e.g., varus or valgus realignment) redistributes mechanical stress away from necrotic zones, helping preserve the joint if collapse is not yet advanced.

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Surgical Management

In advanced AVN with collapse of the subchondral bone or joint-space narrowing, surgical reconstruction or replacement becomes necessary. Joint-preserving options become less feasible as collapse progresses. Arthroplasty (total hip, total knee, shoulder replacement) restores function and relieves pain when the articular surface has failed. In joints where motion can be sacrificed for pain relief (e.g., talus), arthrodesis provides stability. Emerging reconstructive options, including stem-cell-augmented grafts, biologic scaffolds, and 3D-printed implants, are under investigation for younger patients with limited disease.

Table No. 3: Summary of Treatment/Surgery based on the site and severity of AVN

Joint Affected	Key Features	Classification System	Surgical / Treatment Approach
Hip Joint	May be traumatic or atraumatic; up to 70% bilateral involvement	Ficat & Arlet (4 stages) – from pain without radiographic change → collapse & arthritis. Steinberg – adds MRI-based lesion size.	Stage I–II: Core decompression ± bone grafting. Stage II–III: Vascularized fibular graft/osteotomy. Stage IV: Total hip arthroplasty. Emerging: Stem cell & BMP-assisted grafts.
Knee Joint (SONK)	Common in elderly females, usually the medial femoral condyle (≈94%)	Koshino (4 stages) – early radiologically silent → late osteosclerosis & osteophytes.	Early: Protected weight-bearing, decompression. Intermediate: Bone grafting / high tibial osteotomy. Advanced: Unicompartmental or total knee replacement.
Shoulder Joint	Often post-traumatic or steroid-induced; humeral head involvement	Cruess (5 stages) – MRI-detected early lesion → collapse & glenoid degeneration.	Early: Core decompression ± biologic graft. Moderate: Humeral resurfacing/hemiarthroplasty. Late: Total shoulder arthroplasty.
Talus Bone	Follows displaced talar neck fractures; vascular supply limited	Hawkins – predicts AVN risk; absence of Hawkins sign = necrosis.	Early: Immobilization, decompression, vascularized graft. Advanced: Arthrodesis (tibiotalar / tibio-calcaneal) or custom prosthesis.
Scaphoid Bone (Preiser Disease)	Idiopathic or secondary; affects dominant wrist of middle-aged males	Herbert & Lanzetta (4 stages) – proximal pole changes → carpal collapse.	Early: Vascularized bone graft, curettage. Late: Proximal row carpectomy / partial wrist fusion.

AYURVEDIC DOCTRINE OF AVASCULAR NECROSIS

In Ayurveda, Avascular Necrosis (AVN) can be conceptually understood with *Asthi-Majjagata Vata*, *Asthi Kshaya*, or *Kaphavrita Vyana Vata*, conditions arising from *Vata* aggravation and depletion of *Asthi* and *Majja Dhatus* leading to pain, degeneration, and functional impairment. The pathology involves *Srotorodha* (microvascular obstruction) and *Dhatu Kshaya* (tissue depletion). Management focuses on *Vata Shamana*, *Rasayana*, and *Asthi Dhatu Poshana* through *Basti*, *Abhyanga*, and *Ghrita Taila Prayoga*. [25-28]

1. Asthi Majjagata Vata

Charaka Samhita, Chikitsa Sthana 28/33

भेदोऽस्थिपर्वणां सन्धिशूलं मांसबलक्षयः ।
अस्वप्नः सन्तता रुक् च मज्जास्थिकुपितेऽनिले ॥
(Ch. Chi. 28/33)

Transliteration

Bhedo'sthiparvaṇāṃ sandhiśūlaṃ māṃsabalakṣayaḥ |
Asvapnaḥ santatā rūk ca majjāsthikupite'nile ||

This verse from *Charaka Samhita* explains the pathological state of *Vata* aggravation affecting the *Asthi* (bone) and *Majja Dhatu* (bone marrow). The symptoms include cracking sensations in the bones and joints (*Asthi-Parvanam Bheda*), joint pain (*Sandhi Shoola*), muscular wasting (*Mamsa Bala Kshaya*), insomnia (*Asvapna*), and persistent pain (*Santata Ruk*). These features collectively represent a degenerative bone disorder caused by *Vata Prakopa* and *Dhatu Kshaya*, leading to weakened bone structure and functional impairment. The description bears close resemblance to avascular necrosis in contemporary understanding, where interruption of nourishment parallels *Srotorodha* and *Asthi-Majja Dushti* described in Ayurveda. The condition signifies the depletion of *Asthi Dhatu Sara* and impaired *Majja Vaha Srotas*, forming the Ayurvedic basis for degenerative bone pathologies.

2. Asthi Āvṛta Vāta (अस्थिआवृतवात)

स्पर्शमस्थ्नाऽऽवृते तूष्णं पीडनं चाभिनन्दति॥६६॥
सम्भज्यते सीदति च सूचीभिरिव तुद्यते॥ (Ch. Chi. 28/66)
Sparśam asthnāvṛte tūṣṇaṃ pīḍanaṃ cābhinandati |
Sambhajyate sīdate ca sūcibhiriva tudyate || (Ch. Chi. 28/66)

In Asthi Āvṛta Vāta, described by Ācārya Charaka, Vāta Doṣa becomes obstructed by Asthi Dhātu, leading to characteristic pain manifestations such as tūṣṇaṃ pīḍanam—a dull, pressing or kneading type of pain—and sūcibhiriva tudyate—a pricking pain similar to needle sensation. This reflects the involvement of both Vāta and Asthi Dhātu, resulting in deep-seated pain and restricted movement. The description closely aligns with

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clinical presentations of degenerative or ischemic bone disorders, where Vāta prakopa and Dhātu kṣaya coexist, highlighting the classical understanding of Doṣa-Āvarana pathology.

3. Majjāvruta Vāta

मज्जावृते विनामः स्याज्जृम्भणं परिवेष्टनम् ।
शूलं तु पीड्यमाने च पाणिभ्यां लभते सुखम् । (Ch. Chi 28/67)

Transliteration:

Majjāvṛte vināmaḥ syāj jṛmbhaṇaṃ pariveṣṭanam |
śūlaṃ tu pīḍyamāne ca pāṇibhyāṃ labhate sukham ||

In *Majjāvṛta Vāta*, the obstructed *Vāta* within the *Majjā Dhātu* leads to characteristic symptoms such as *vināmaḥ* (bending or stiffness of the body), *jṛmbhaṇa* (frequent yawning), and *pariveṣṭanam* (twisting or constricting type of pain). The patient experiences *śūla* (colicky pain) that is notably relieved by manual pressure, as indicated by *pāṇibhyāṃ labhate sukham*. This verse highlights the localized vitiation of *Vāta* in the *Majjā Dhātu*, manifesting as neuromuscular discomfort and spasmodic pain, which responds favorably to warmth and pressure—features typical of *Avarana* (obstruction) type *Vāta Vyādhi*.

4. Sandhigata Vata

वातपूर्ण दृटिस्पर्शः शोषः सन्धिगतानिले ।
प्रसारणाकुञ्चनयोः प्रवृत्तिश्च सवेदनाः ॥ (च.चि. २८/३७)

Transliteration:

Vāta-pūrṇa dṛti-sparśaḥ śothaḥ sandhigate'nile |
Prasāraṇākuñchanayoḥ pravṛttiśca savedanā || (Ch. Chi. 28/37)

When Vata gets localized in the joints, it manifests as swelling that feels like an air-filled leather bag on touch (*Vata-purna druti sparsha shotha*), indicating distension and stiffness. The affected individual experiences *vedana* (pain) and restricted or painful movement (*prasārana-ākuñchana apravritti vedana*) during flexion and extension of the joint. This description aligns closely with the pathological presentation of Avascular Necrosis (AVN), where ischemic bone necrosis leads to joint stiffness, swelling, and severe pain with restricted mobility, correlating the Ayurvedic concept of *Sandhigata Vata* to AVN-like conditions.

***Samānya Cikitsā* (General Line of Management)**

सर्पिस्तैलवसामज्जसेकाभ्यञ्जनबस्तयः[३] ||
स्निग्धाः स्वेदा निवातं च स्थानं प्रावरणानि च|
रसाः पयांसि भोज्यानि स्वाद्वम्ललवणानि च||
बृंहणं यच्च तत् सर्वं प्रशस्तं वातरोगिणाम्|| (Ch.Chi 28/104-106)

Transliteration:

Sarpis-taila-vasā-majja-sekābhyañjana-bastayaḥ |
Snigdhāḥ svedā nivātaṃ ca sthānaṃ prāvaraṇāni ca |
Rasāḥ payāṃsi bhojyāni svādu-amla-lavaṇāni ca ||
Bṛṃhaṇaṃ yacca tat sarvaṃ praśastaṃ vātarogiṇām || (Ch. Chi. 28/104–106)

The general line of management in *Vātavyādhi* emphasizes therapies aimed at pacifying the *Vāta doṣa* through unctuous, nourishing, and stabilizing measures. Administration of *Sarpis* (ghee), *Taila* (medicated oil), *Vasā* (muscle fat), and *Majjā* (bone marrow) is recommended in the form of *Pāna*, *Abhyañjana*, and *Basti* to counteract *Vāta*'s dryness and lightness. *Snigdha Svedana* (unctuous sudation) aids in softening and channel opening, while residing in calm, windless environments and covering the body with warm garments help maintain *Vāta* stability. Nutritionally, the intake of meat soups, milk, and foods possessing *madhura* (sweet), *amla* (sour), and *lavaṇa* (salty) tastes — all inherently *Vāta-śāmaka* — is advocated. Collectively, these interventions serve to nourish (*bṛṃhaṇa*), lubricate, and restore the deranged *Vāta*, thus alleviating pain, rigidity, and dryness associated with degenerative conditions like *Asthi-Majjāgata Vāta* (comparable to Avascular Necrosis in modern parlance).

Asthi Majjagata Vāta Chikitsā

बाह्याभ्यन्तरतः स्नेहैरस्थिमज्जगतं जयेत्॥ (Ch. Chi. 28/93)

Transliteration

Bāhyābhyantara taḥ snehair asthi-majja-gataṃ jayet.

In *Asthi-Majjagata Vāta*, Acharya Charaka highlights *Snehana* (oleation) both *Bāhya* (external) and *Abhyantara* (internal) as the key therapy. *Vāta* vitiation in *Asthi* and *Majjā dhatus* causes degeneration, pain, and restricted movement. Oleation replenishes unctuousness, nourishes tissues, and restores mobility. *Abhyantara Sneha* with medicated *ghṛta* and *taila pāna* mitigates internal dryness, while *Bāhya Sneha* through *Abhyanga* and *Parisheka* relieves stiffness and enhances circulation. This dual approach pacifies *Vāta* and halts degenerative changes resembling avascular necrosis (AVN).

SIDHA GHRUTA

विविधान् विविधव्याधिप्रशमायामृतोपमान्|
द्रोणेऽम्भसः पचेद्भागान् दशमूलाच्चतुष्पलान्||
यवकोलकुलत्थानां भागैः प्रस्थोन्मितैः सह|
पादशेषे रसे पिष्टैर्जीवनीयैः सशर्करैः||
तथा खर्जूरकाश्मर्यद्राक्षाबदरफल्गुभिः|
सक्षीरैः सर्पिषः प्रस्थः सिद्धः केवलवातनुत्||
निरत्ययः प्रयोक्तव्यः पानाभ्यञ्जनबस्तिषु| (Ch.Chi 28/118-122)

Acharya Charaka advocates the use of *Siddha Ghṛita*, prepared with *Dashamoola*, *Yava*, *Kola*, and *Kulattha* decoction, for the alleviation of Vata-related pathologies. The decoction is further fortified with the paste of *Jivaniya Gana* drugs, *Sharkara*, and fruits like *Kharjura*, *Kashmarya*, *Draksha*, *Badara*, and *Phalgu*, cooked with ghee and milk to enhance its nutritive and unctuous properties. This *Ghṛita* acts as an *Amrita-upama* formulation—restoring vitality, nourishing *Asthi* and *Majja Dhatus*, and pacifying aggravated Vata. It is administered via *Paana*, *Abhyanga*, and *Basti*, offering a comprehensive rejuvenative and Vata-shamana effect suitable for conditions such as *Asthi-Majjagata Vata* and degenerative disorders like Avascular Necrosis.

SIDHA MAJJA

ग्राम्यानूपौदकानां तु भित्वाऽस्थीनि पचेज्जले|| १२४||
तं स्नेहं दशमूलस्य कषायेण पुनः पचेत्|
जीवकर्षभकास्फोताविदारीकपिकच्छुभिः|| १२५||
वातघ्नैर्जीवनीयैश्च कल्कैर्द्विक्षीरभागिकम्|
तत्सिद्धं नावनाभ्यङ्गात्तथा पानानुवासनात्|| १२६||
सिरापर्वास्थिकोष्ठस्थं प्रणुदत्याशु मारुतम्|

ISSN (Online): 2455-3662
EPRA International Journal of Multidisciplinary Research (IJMR) - Peer Reviewed Journal
Volume: 11| Issue: 10| October 2025|| Journal DOI: 10.36713/epra2013 || SJIF Impact Factor 2025: 8.691 || ISI Value: 1.188

ये स्युः प्रक्षीणमज्जानः क्षीणशुक्रौजसश्च ये॥१२७॥ (Ch.Chi 28/123-127)

Transliteration

Grāmyānūpaudakānāṃ tu bhitvā'sthīni pacejjale ||124||
Taṃ snehaṃ daśamūlasya kaṣāyeṇa punaḥ pacet |
Jīvakarṣabhakāsphotāvidārīkapi-kacchubhiḥ ||125||
Vātaghnair jīvanīyaiśca kalkair dvikṣīra-bhāgikam |
Tatsiddhaṃ nāvanābhyaṅgāt tathā pānānuvāsanāt ||126||
Sirāparvāsthi-koṣṭhasthaṃ praṇudatyāśu mārutam |
Ye syuḥ prakṣīṇa-majjānaḥ kṣīṇa-śukra-ojasashca ye ||127||

Bones of domesticated (Grāmya), marshy-land (Anūpa), and aquatic (Audhaka) animals are crushed and cooked in water. The decoction is then processed again with *Daśamūla kaṣāya* and a paste (kalka) of *Jīvaka* (*Malaxis acuminata*), *Ṛṣabhaka* (*Manilkara hexandra*), *Asphota* (*Jasminum angustifolium*), *Vidārī* (*Pueraria tuberosa*), *Kapikacchu* (*Mucuna pruriens*), along with *Vātaghna* and *Jīvanīya* herbs. This mixture is cooked with double the quantity of milk. The resulting medicated *Sneha* is beneficial for *Nāvana*, *Abhyanga*, *Basti*, and *Pāna*, effectively pacifying *Vāta* located in *Sirā*, *Parva*, *Asthi*, and *Koṣṭha*. It is especially indicated for conditions with depletion of *Majjā*, *Śukra*, and *Ojas*.

SIDDHA VASA

तद्वत्सिद्धा वसा नक्रमत्स्यकूर्मचुलूकजा॥१२८॥
प्रत्यग्रा विधिनाऽनेन नस्यपानेषु शस्यते॥

Transliteration

Tadvat siddhā vasā nakra matsya kūrma culūkajā,
pratyagrā vidhinānena nasyapāneṣu śasyate.

In the management of *Asthi-Majjagata Vāta*, *Acharya Charaka* advises the use of *Siddha Vasa*—a preparation of muscle fat obtained from aquatic and amphibian sources such as *Nakra* (crocodile), *Matsya* (fish), *Koorma* (tortoise), and *Ulluka* (owl). These fats, processed as per classical procedure, serve as potent *Snehana dravyas* for *Nasya* (nasal therapy) and *Pāna* (internal oleation). The unctuous, heavy, and strengthening qualities of *Vasa* deeply nourish the *Asthi* and *Majjā dhātus*, thereby pacifying *Vāta* and alleviating degenerative and neurological symptoms associated with bone and marrow depletion. This formulation highlights the classical understanding of lipid-based rejuvenation therapy in chronic degenerative conditions.

MAHA SNEHA

प्रस्थः स्यात्त्रिफलायास्तु कुलत्थकुडवद्वयम्॥
कृष्णगन्धात्वगाढक्योः पृथक् पञ्चपलं भवेत्
रास्नाचित्रकयोर्द्वे द्वे दशमूलं पलोन्मितम्॥
जलद्रोणे पचेत् पादशेषे प्रस्थोन्मितं पृथक्
सुरारनालदध्म्लसौवीरकतुषोदकम्॥
कोलदाडिमवृक्षाम्लरसं तैलं वसां घृतम्
मज्जानं च पयश्चैव जीवनीयपलानि षट्॥
कल्कं दत्त्वा महास्नेहं सम्यगेनं विपाचयेत्
सिरामज्जास्थिगे वाते सर्वाङ्गैकाङ्गरोगिषु॥ (Ch.Chi 28/129-133)

Prasthaḥ syāt triphalāyāstu kulattha-kuḍavadvayam |
Kṛṣṇagandhā-tvak-āḍhakyoḥ pṛthak pañca-palaṁ bhavet ||
Rasnā-citrakayor dve dve daśamūlaṁ palonmitam |
Jala-droṇe pacet pāda-śeṣe prasthonmitaṁ pṛthak ||
Surāraṇāla-dadhy-amla-sauvīraka-tuṣodakam |
Kola-dāḍima-vṛkṣāmla-rasaṁ tailaṁ vasāṁ ghṛtam |
Majjānaṁ ca payaś caiva jīvanīya-palāni ṣaṭ ||
Kalkaṁ dattvā mahāsnehaṁ samyag enaṁ vipācayet |
Sirā-majjāsthi-ge vāte sarvāṅgai-kāṅga-rogiṣu ||

In the management of *Asthi-Majjāgata Vāta*, *Acharya Charaka* recommends *Mahāsneha*—a potent polyherbal and poly-fatty formulation prepared through *Sneha Kalpana*. It involves decocting *Triphala*, *Kulattha*, *Kṛṣṇagandha*, *Ādhakī*, *Rasnā*, *Chitraka*, and *Daśamūla* in water, then mixing the reduced decoction with *Sura*, *Āranāla*, *Dadhyāmla*, *Sauvīraka*, *Tuṣodaka*, *Kola rasa*, *Dāḍima rasa*, and *Vṛkṣāmla rasa*. To this, *Taila*, *Vasā*, *Ghṛta*, *Majjā*, and *Kṣīra* are added along with *Jīvanīya gaṇa* paste to prepare *Mahāsneha*.

This formulation deeply nourishes the *Asthi* and *Majjā dhātus*, pacifies *Vāta*, restores lubrication, and strengthens the musculoskeletal system. It is especially indicated in *Sirā-Majjā-Asthi-gata Vāta* and both localised and generalised *Vāta* disorders, correlating with modern degenerative bone and marrow diseases such as osteoarthritis and avascular necrosis.

DISCUSSION

Avascular Necrosis (AVN) or Osteonecrosis is a progressive ischemic disorder of bone resulting from loss of blood supply, leading to necrosis of osteocytes and marrow elements, structural collapse, and articular dysfunction. The femoral head is most commonly affected due to its terminal vascularity, making it prone to ischemic injury following trauma, corticosteroid use, alcohol intake, or metabolic disturbances. Clinically, it manifests as deep-seated pain, stiffness, and restricted movement. In Ayurveda, AVN closely correlates with *Asthi-Majjāgata Vāta*, a *Vāta-pradhāna vyādhi* described by *Ācārya* Charaka (Ch. Chi. 28/33), marked by *Sandhiśūla* (joint pain), *Māṃsa-bala-kṣaya* (muscle wasting), *Asthiparva bheda* (crepitus), and *Ruk* (pain), arising from *Vāta prakopa* and depletion of *Asthi* and *Majjā dhātus*. The underlying pathology involves *Dhātu-kṣaya* (tissue depletion) and *Srotorodha* (microchannel obstruction), comparable to ischemic compromise in modern pathology. *Āvaraṇa* of *Vāta* by *Asthi* or *Majjā dhātu* (*Asthyāvṛta* or *Majjāvṛta Vāta*) explains the deep, pricking pain typical of AVN.

Ācārya Charaka advocates *Snehana* (oleation), *Svedana* (fomentation), and *Bṛṃhaṇa* (rejuvenation) therapies to alleviate *Vāta* and nourish depleted tissues. Among these, *Mahāsneha*—a formulation of *Taila, Vasā, Ghṛta, Majjā,* and *Kṣīra* processed with *Daśamūla, Rasnā, Chitraka,* and *Jīvanīya gaṇa dravyas*—is indicated in *Sirā-Majjā-Asthi-gata Vāta*. It provides deep nourishment, enhances microcirculation, and pacifies aggravated *Vāta*, addressing both degenerative and ischemic pathology. Conceptually, *Snehana* and *Basti karma* improve vascular perfusion and lubrication, aligning with the angiogenic and anti-inflammatory effects recognised in regenerative medicine. Thus, *Avascular Necrosis* and *Asthi-Majjāgata Vāta* represent a shared pathological framework—modern science views it as ischemia and bone collapse, while Ayurveda interprets it through *Vāta prakopa*, *Dhātu kṣaya*, and *Srotorodha*. Integrative management combining *Snehana*, *Basti*, *Rasāyana chikitsā*, and modern regenerative strategies may offer synergistic benefits in restoring bone vitality and joint function.

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CONCLUSION

Avascular Necrosis (AVN) closely resembles *Asthi-Majjāgata Vāta*, characterised by *Vāta prakopa*, *Dhātu-kṣaya*, and *Srotorodha*. Ayurvedic management through *Snehana*, *Basti*, and *Bṛṃhaṇa*—particularly using *Mahāsneha*—offers deep nourishment, enhances circulation, and restores *Asthi-Majjā* integrity. This integrative approach provides a holistic and regenerative solution for managing degenerative bone disorders like AVN.

ACKNOWLEDGEMENT

The author expresses sincere gratitude to **Dr. Bharathi D. Anvekar**, Guide, for her constant guidance, encouragement, and valuable support throughout every milestone of this work. Heartfelt thanks are also extended to **Dr. Manjunath**, Co-guide, for his insightful suggestions; to **Dr. Shankar**, Head of the Department, for his guidance; and to the esteemed faculty members **Dr. Parvathy S.** and **Dr. Monisha C. J.** for their continuous support and motivation.

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