



# 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 sandhīśūla (joint pain), māṃsa-bala-ksaya (muscle wasting), and Asthiparva bheda (bony crepitus). The underlying pathology in both systems involves tissue depletion (dhātu-ksaya) and obstruction of microchannels (srotorodha), analogous to ischemia and necrosis in modern understanding. Ācārya Charaka prescribes Mahāsneha – a formulation comprising taila, vasā, ghr̥ta, 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-ksaya, 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 <sup>[1]</sup>. Later, Jean Cruveilhier (1829–1842) provided detailed anatomical observations of femoral head deformities resulting from disrupted vascularity <sup>[2]</sup>. 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 <sup>[3]</sup>. 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-Majjāgata Vāta, wherein vitiation of Vāta leads to

degeneration and necrosis of bone tissue, reflecting the intricate interplay of Dhātu ksaya 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 <sup>[4]</sup>. 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. <sup>[5]</sup> 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



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

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. <sup>[7-9]</sup> 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. <sup>[10]</sup>

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. <sup>[11-13]</sup> 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 <sup>[14]</sup>. Radiation therapy may further aggravate vascular injury <sup>[15]</sup>.

**2. Fat Embolism and Vascular Occlusion:**

Deposition of fat within small blood vessels can obstruct intraosseous circulation, resulting in cellular death <sup>[16]</sup>.

**3. Systemic Diseases:**

Haematological and metabolic disorders such as sickle cell anaemia and Gaucher’s disease may impair blood flow and predispose to AVN <sup>[17]</sup>.

**4. Arterial Pathology:**

Inflammation, thrombosis, or arterial wall damage can block vascular channels, compromising bone perfusion <sup>[18]</sup>.

**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 <sup>[19-20]</sup>.

**6. Alcohol Consumption:**

Chronic alcohol intake alters lipid metabolism, causes fat emboli, and diminishes bone vascularity, leading to necrosis <sup>[21]</sup>.

**7. Smoking:**

Nicotine induces vasoconstriction and reduces oxygen delivery to bone tissue, increasing the risk of AVN <sup>[22]</sup>.

**8. Medical Treatments:**

Radiation and organ transplant procedures can cause vascular injury and metabolic imbalance, predisposing to osteonecrosis <sup>[23]</sup>.

**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. <sup>[24]</sup>

**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.



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.	<b>Avascular necrosis</b>	<i>Intermittent pain that appears and eases when pressure is applied to the bone and then is relieved.</i>	—

**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
<b>Hip (Femoral Head)</b>	Traumatic / Atraumatic (70% bilateral)	<i>Ficat &amp; Arlet, Steinberg</i>	Stage I–IV: from pain without radiographic change → femoral collapse and osteoarthritis. MRI aids early detection.
<b>Knee (SONK)</b>	Spontaneous, secondary, or post-arthroscopic	<i>Koshino</i>	Four stages: from symptomatic onset → degenerative phase with sclerosis and osteophytes. Common in females, the medial femoral condyle.
<b>Shoulder (Humeral Head)</b>	Post-traumatic, corticosteroid-related	<i>Cruess</i>	Stage I–V: MRI-only lesion → humeral head collapse → glenoid degeneration.
<b>Talus</b>	Post-traumatic (neck fractures, dislocation)	<i>Hawkins</i>	Progresses with loss of Hawkins sign at 6–8 weeks; indicates avascular necrosis.
<b>Scaphoid (Preiser’s Disease)</b>	Trauma, steroid use, alcohol abuse	<i>Herbert &amp; Lanzetta</i>	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.



### 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 *Ghruta Taila Prayoga*.<sup>[25-28]</sup>

#### 1. Asthi Majjagata Vata

*Charaka Samhita, Chikitsa Sthana 28/33*

*भेदोऽस्थिपर्वणां सन्धिःशूलं मांसबलक्षयः ।*

*अस्वप्नः सन्तता रुक् च मज्जास्थिकुपितेऽनिले ॥*

*(Ch. Chi. 28/33)*

#### Transliteration

*Bhedo 'sthiparvaṇāṃ sandhiśūlaṃ māmsabalakṣayaḥ |*

*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 Āvrta Vāta (अस्थि आवृतवात)

*स्पर्शमस्थान्ऽऽवृते तूष्णं पीडनं चाभिनन्दति ॥६६॥*

*सम्भज्यते सीदति च सूचीभिरिव तुद्यते ॥ (Ch. Chi. 28/66)*

*Sparśam asthnāvṛte tūṣṇaṃ pīdanaṃ cābhinandati |*

*Sambhajyate sīdate ca sūcibhiriva tudyate || (Ch. Chi. 28/66)*

In *Asthi Āvrta Vāta*, described by Ācārya Charaka, *Vāta Doṣa* becomes obstructed by *Asthi Dhātu*, leading to characteristic pain manifestations such as *tūṣṇaṃ pīdanaṃ*—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



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

### 3. Majjāvṛta Vāta

मज्जावृते विनामः स्याज्जम्भणं परिवेष्टनम् ।

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

#### Transliteration:

Majjāvṛte vināmaḥ syāj jṛmbhaṇam pariveṣṭanam |  
śūlam tu pīdyamāne ca paṇibhyāṃ 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 paṇibhyāṃ 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 Āvarana (obstruction) type Vāta Vyādhi.

### 4. Sandhigata Vata

वातपूरणं दृष्टिस्पर्शः शोषः सन्धिगतानिले ।

प्रसारणाकुञ्चनयोः प्रवृत्तिश्च सवेदनाः ॥ (च.चि. २८/३७)

#### Transliteration:

Vāta-pūrṇaṃ dr̥ṣṭi-sparśaḥ śoṣaḥ sandhigate'nile |  
Prasāraṇākuñchanayoḥ pravṛttiśca savedanāḥ || (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 (Vāta-pūrṇa druti sparsha shotha), indicating distension and stiffness. The affected individual experiences vedana (pain) and restricted or painful movement (prasāraṇa-ākuñchana apravṛtti 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 ।<sup>13</sup> ॥

स्निग्धाः स्वेदा निवातं च स्थानं प्रावरणानि च ।

रसाः पर्यासि भोज्यानि स्वाद्मल्लवणानि च ॥

बृंहणं यच्च तत् सर्वं प्रशस्तं वातरोगिणाम् ॥ (Ch. Chi 28/104-106)

#### Transliteration:

Sarpis-taila-vasā-majja-sekābhyanjana-bastayah |  
Snigdhaḥ svedā nivātam ca sthānam prāvaraṇāni ca |  
Rasāḥ payāmsi bhojyāni svādu-amlā-lavaṇāni ca ||  
Bṛmhanam yacca tat sarvaṃ praśastam vataroginām || (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-sāmaka — is advocated. Collectively, these interventions serve to nourish (bṛmhaṇa), 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 स्नेहैरस्थिमज्जागतं 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 ghr̥ta 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 विविधव्याधिप्रशमयामृतोपमान्

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 Ghrita, 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 Ghrita 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

ग्राम्यानुपौदकानां तु भित्वाऽस्थीनि पचेज्जले ॥ १२४ ॥

तं स्नेहं दशमूलस्य कषायेण पुनः पचेत्

जीवकर्षभकास्फोताविदारीकपिकच्छुभिः ॥ १२५ ॥

वातघ्नैर्जीवनीयैश्च कल्कैर्द्विक्षीरभागिकम्

तत्सिद्धं नावनाभ्यङ्गात्तथा पानानुवासनात् ॥ १२६ ॥

सिरापर्वीस्थिकोष्ठस्थं प्रणुदत्याशु मारुतम्



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

#### Transliteration

Grāmyānūpaudakānām tu bhityā'sthīni pacejjale ||124||  
Taṃ snehaṃ daśamūlasya kaṣāyena punaḥ pacet |  
Jīvakaṣabhakāśphotāvidārikapi-kacchubhiḥ ||125||  
Vātaghnair jīvanīyaisca kalkair dvikṣīra-bhāgikam |  
Tatsiddhaṃ nāvanābhyaṅgāt tathā pānānūvāsanaṭ ||126||  
Sirāparvāsthi-koṣṭhasthaṃ praṇudatyāśu mārutam |  
Ye syuḥ prakṣīna-majjānaḥ kṣīna-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*), Rṣabhaka (*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 नक्रमत्स्यकूर्मचूलकजा || १२८ ||  
प्रत्यग्रा विधिनाऽनेन नस्यपानेषु शस्यते ||

#### Transliteration

Tadvat siddhā vasā nakra matsya kūrma culūkajā,  
pratyagrā vidhinānena nasyapāneṣu śasyate.  
In the management of Asthi-Majjāgata 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 |  
कृष्णगन्धात्वाढक्योः पृथक् पञ्चपलं भवेत् |  
रास्नाचित्रकयोर्द्वे द्वे दशमूलं पलोन्मितम् |  
जलद्रोणे पचेत् पादशेषे प्रस्थोन्मितं पृथक् |  
सुरारनालदध्यम्लसौवीरकतुषोदकम् |  
कोलदाडिमवृक्षाम्लरसं तैलं वसां घृतम् |  
मज्जानं च पयश्चैव जीवनीयपलानि षट् |  
कल्कं दत्त्वा महास्त्रेहं सम्यगेनं विपाचयेत् |  
सिरामज्जास्थिगे वाते सर्वाङ्गैकाङ्गरोगिषु | (Ch.Chi 28/129-133)  
Prasthaḥ syāt triphalāyāstu kulattha-kuḍavadvayam |  
Kṛṣṇagandhā-tvak-ādhakyoḥ pṛthak pañca-palaṃ bhavet ||  
Rasnā-citrakayor dve dve daśamūlāṃ palonmitam |  
Jala-droṇe pacet pāda-śeṣe prasthonmitam pṛthak ||  
Surāraṅāla-dadhy-āmla-sauvīraka-tuṣodakam |  
Kola-dāḍima-vṛkṣāmla-rasaṃ tailaṃ vasāṃ ghr̥tam |

Majjānam ca payas caiva jīvanīya-palāni ṣaṭ ||  
Kalkam dattvā mahāsneham samyag enaṃ 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, Aranāla, 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), Māmsa-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 Bṛmhaṇa (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.



## CONCLUSION

Avascular Necrosis (AVN) closely resembles *Asthi-Majjāgata Vāta*, characterised by *Vāta prakopa*, *Dhātu-kṣaya*, and *Srotorodha*. Ayurvedic management through *Snehana*, *Basti*, and *Bṛmhāṇa*—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.

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