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MEDA VIMARGAGAMANA IN ASTHI-MAJJA DHATU AND POSTERIOR INTERVERTEBRAL DISC BULGE: AN INTEGRATIVE CONCEPTUAL REVIEW

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ABSTRACT

Background: Intervertebral disc degeneration is a major contributor to chronic low back pain and radiculopathy and remains a leading source of disability worldwide. Although mechanical loading remains central to disc pathology, growing evidence indicates that metabolic dysfunction, obesity-related inflammation, adipokine dysregulation, and vertebral marrow fat may also contribute to disc matrix failure and impaired repair. In Ayurvedic physiology, tissue homeostasis is understood through the integrated relationships among *Dosha*, *Dhatu*, *Agni*, and *Srotas*, and pathological disruption of these channels is described as *Srotodushti*. **Objective:** To develop an integrative conceptual framework for *Meda Vimargagamana* within the *Asthi-Majja* axis and to examine its theoretical correspondence with immunometabolic and degenerative mechanisms implicated in intervertebral disc pathology.

Materials and Methods: A narrative conceptual review was conducted using classical Ayurvedic sources, including the *Caraka Saṃhitā*, *Suśruta Saṃhitā*, and *Aṣṭāṅga Hṛdaya*,

together with contemporary biomedical literature retrieved from indexed databases on intervertebral disc degeneration, adipokine biology, marrow adiposity, and immunometabolic spinal mechanisms. **Results:** Classical Ayurvedic literature describes *Meda Dhatu* as functionally interconnected with *Asthi* and *Majja* and recognizes *Meda* as a root of the *Asthivaha Srotas*, supporting a systemic relationship between lipid homeostasis and deeper structural tissues. Contemporary evidence similarly suggests that intervertebral disc degeneration is not solely a mechanically driven disorder but also involves inflammatory and metabolic imbalance, including adipokine-mediated signaling, oxidative stress, extracellular matrix catabolism, and altered nutrient exchange across the cartilaginous endplate. Studies of adjacent vertebral marrow further report associations between increased vertebral bone marrow fat and biochemical changes in neighboring discs, supporting a plausible disc-endplate-bone marrow interaction. Within this integrative model, *Meda Vimargagamana* may be interpreted as a conceptual analogue of pathological lipid deviation and local metabolic obstruction contributing to structural disc failure. **Conclusion:** Posterior intervertebral disc bulge can be framed conceptually not only as a localized biomechanical event but also as a downstream structural expression of broader immunometabolic disturbance. This interpretation is hypothesis-generating and integrative rather than clinically validated, and it requires direct translational and longitudinal investigation.

KEYWORDS: *Vimargagamana*, Intervertebral Disc Bulge, Disc Bulge, Immunometabolism.

INTRODUCTION

The lumbar intervertebral disc (IVD) is an avascular fibrocartilaginous structure optimized to distribute axial loads while preserving multi-axial spinal mobility. Structurally composed of a hydrophilic Nucleus Pulposus (NP) encapsulated by the dense collagenous lamellae of the Annulus Fibrosus (AF), its metabolic homeostasis is understood to depend predominantly on passive nutrient diffusion across the porous Cartilaginous Endplates (CEPs) [Adams & Roughley, 2006].

Within the framework of Ayurvedic physiology, the structural maintenance and longevity of these deep connective tissues involve a functional continuum between *Meda Dhatu* (lipid matrix) and *Asthi Dhatu* (osseous and deep cartilaginous structures). This physiological relationship is anchored by the classical principle identifying *Meda* as a primary operational root (*Mula*) of the *Asthivaha Srotas* (Caraka Saṃhitā, Vimānasthāna 5/8).

When lipid homeostasis undergoes qualitative or quantitative impairment (*Meda Dushti*), a pathodynamic cascade may emerge wherein dysfunctional tissue elements deviate into inappropriate anatomical domains. This process is defined as *Vimargagamana*—the movement of constituents through anomalous or unorthodox pathways (Caraka Saṃhitā, Vimānasthāna 5/24).

From a contemporary pathophysiological perspective, the authors propose that this systemic metabolic spillover shares theoretical parallels with the expansion of yellow Bone Marrow Adipose Tissue (BMAT) within the vertebral bodies and the concurrent elevation of pro-inflammatory, fat-derived adipokines (Leptin, Resistin, $TNF-\alpha$, IL-6) infiltrating the spinal microenvironment [Inoue and Espinoza Orias, 2006]. This review explores the hypothesis that the phenomenon of *Meda Vimargagamana* into the *Asthi Dhatu* matrix may function as an upstream contributor to structural disc failure.

By evaluating this systemic-to-local progression, this paper aims to project a conceptual model bridging ancient bio-channel pathodynamics with modern molecular immunometabolism, thereby re-evaluating Prolapsed Intervertebral Disc (PIVD) and chronic Low Back Ache (LBA) not merely as isolated mechanical injuries, but as potential structural consequences of chronic metabolic disruption.

NEED OF THE STUDY

The clinical necessity for a broader pathogenetic model is underscored by the escalating global burden of chronic Low Back Ache (LBA) and Prolapsed Intervertebral Disc (PIVD). Data from the Global Burden of Disease (GBD 2021) study indicates that prevalent chronic low back pain cases have crossed 628.8 million worldwide, reflecting a 62.59% increase in absolute volume since 1990, with projections estimated to exceed 843 million by 2050 [Foster et al., 2018; GBD Collaborators, 2023].

Epidemiological metrics demonstrate a distinct gender-based skew, with women displaying higher age-standardized prevalence rates (ASPR) and increased years lived with disability (YLDs) due to PIVD, particularly in the post-menopausal phase [GBD Collaborators, 2023]. Biomedical frameworks often associate this post-menopausal vulnerability with estrogen depletion—which is suggested to downregulate proteoglycan synthesis within disc cells—alongside concurrent alterations in lumbar lordosis.

Despite these documented clinical associations, contemporary therapeutics often remain polarized, balancing between short-term symptomatic management (NSAIDs, epidural injections) and late-stage surgical interventions (discectomy, spinal fusion), frequently without directly targeting potential metabolic drivers. Similarly, traditional Ayurvedic clinical protocols commonly address these spinal disorders during their terminal phase, managing them as localized *Vata-vyadhi* (neurological or mechanical excitation) or *Asthi Kshaya* (bone matrix depletion) after macroscopic structural damage has occurred.

Consequently, exploring an upstream conceptual framework that links systemic metabolic anomalies (*Meda Dushti*) with structural spinal alterations offers an opportunity to evaluate early, preventative, metabolic-focused therapeutic strategies (*Langhana*, *Rukshana*, *Lekhana Basti*). These interventions are theoretically designed to address early-stage degeneration before irreversible mechanical structural failure takes place.

REVIEW OF AYURVEDIC LITERATURE

Concept of Srotas and the Pathodynamics of Srotodushti

Ayurvedic biology models the living organism as an intricate network of specialized bio-channels termed *Srotas*, defined as functional pathways for the transformation, transport, and continuous distribution of bodily constituents (Caraka Saṃhitā, Vimānasthāna 5/3). The functional integrity of these channels is understood to maintain homeostatic balance.

Pathological variations within these pathways are classically categorized into four functional and structural expressions of *Srotodushti* (channel dysfunction) (Caraka Saṃhitā, Vimānasthāna 5/24):

- ***Atipravritti:*** Excessive, hyper-functional flow or increased discharge.
- ***Sanga:*** Localized obstruction, stasis, or structural blockade.
- ***Siragranthi:*** Dilation or the formation of structural nodules within the channels.
- ***Vimargagamana:*** The diversion of flow into abnormal or unorthodox pathways.

Etymologically, *Vimargagamana* denotes "entering an improper or unintended path." Within the context of this review, the authors interpret this principle at a micro-pathological level as the potential ectopic localization, aberrant biological expression, or pathological overflow of a tissue precursor or metabolic element into a foreign *Srotas* domain, where its hyper-presence may alter the host microenvironment.

The Meda-Asthi Continuum and Meda Dushti

The *Medovaha Srotas* regulates the synthesis, processing, and systemic management of *Meda Dhatu* (lipid tissue matrices), and is described as being rooted anatomically within the *Vrikka* (kidneys) and *Vapavahana* (peritoneum/omentum) (Caraka Saṃhitā, Vimānasthāna 5/7). The prolonged overconsumption of *Guru* (heavy) and *Snigdha* (slimy/unctuous) foods, paired with a sedentary lifestyle (*Avyayama*), is understood to downregulate systemic *Jatharagni* and localized *Dhatvagni*.

This cascade induces *Agnimandya* (metabolic deceleration), yielding *Meda Dushti*. The resulting lipid matrix is characteristically defined as *Abaddha* (loosely structured or unbound) and *Aparipakwa* (incompletely metabolized), potentially transforming a physiological tissue constituent into a circulating pathogenetic factor.

Within this conceptual framework, this impaired metabolic state is hypothesized to influence the *Asthivaha Srotas*, which governs the osseous and deep cartilaginous matrices (*Asthi Dhatu*). This inter-tissue relationship is anchored by the classical operational dictum: *"Asthivahānāṃ srotasāṃ medomūlaṃ jaghanaṃ ca"* (Caraka Saṃhitā, Vimānasthāna 5/8). In the sequential tissue metabolic cascade (*Dhatu-Parampara*), *Meda Dhatu* functions as the immediate nutritional precursor that undergoes transformation by *Asthyagni* to generate *Asthi Dhatu*.

Hence, the authors propose that qualitative impairment in *Meda* (such as the *Abaddha* state) may structurally compromise the downstream density and mechanical resilience of the *Asthi* matrix, theoretically rendering it more susceptible to premature structural failure under normal mechanical loading.

BIOMEDICAL ANALYSIS OF THE DISC MATRIX & METABOLIC DECAY

Biomechanics and Biology of Intervertebral Disc Degeneration

The physiological function of the healthy IVD is understood to depend predominantly on osmotic balance and structural containment. The central Nucleus Pulposus possesses a highly concentrated proteoglycan matrix—chiefly aggrecan—bound to hydrophilic glycosaminoglycans (GAGs), which generates the hydrostatic swelling pressure required to resist axial compression [Roughley, 2006]. This core is enclosed by the Annulus Fibrosus, which consists of highly organized, concentric lamellae of Type I and Type II collagen fibers designed to withstand multi-axial tensile strain.

Disc degeneration represents an aberrant, cell-mediated response to cumulative micro-structural alterations [Adams & Roughley, 2006]. The initial phase is frequently characterized by microscopic fissures or calcification within the porous Cartilaginous Endplates, a state that can disrupt the passive diffusion of vital nutrients (such as glucose and oxygen) into the avascular disc core.

Starved of essential substrates, the resident disc cells shift toward an anaerobic, catabolic state, upregulating matrix-degrading enzymes including Matrix Metalloproteinases (MMPs) and A Disintegrin and Metalloproteinase with Thrombospondin Motifs (ADAMTS). This enzymatic activity gradually degrades the aggrecan-collagen architecture, lowering water-binding capacity, reducing hydrostatic pressure, and shifting mechanical stresses onto the annulus fibrosus, which potentially culminates in structural failure and visible disc bulging [Roughley, 2006].

The Immunometabolic Shift: Obesity, Marrow Adiposity, and Adipokines

Recent molecular profiling indicates that chronic disc decay is significantly influenced by systemic immunometabolic pathways. Visceral adipose expansion is understood to function as an active endocrine disruptor, continuously secreting pro-inflammatory cytokines and adipokines. In metabolic syndrome states, circulating levels of Leptin and Resistin are frequently elevated. Because human disc cells express functional leptin receptors, chronic hyperleptinemia has been shown to activate a signaling cascade that may upregulate inflammatory markers within the disc matrix, potentially accelerating cellular apoptosis and matrix breakdown.

Concurrently, advanced magnetic resonance spectroscopy highlights the phenomenon of Vertebral Marrow Adiposity, characterized by the expansion of Bone Marrow Adipose Tissue (BMAT). Under systemic lipid stress, multipotent mesenchymal stem cells within the vertebral bodies appear to shift their differentiation pathway, favoring yellow, fatty adipocytes over red, vascularized osteogenic lines. This fatty accumulation within the subchondral bone spaces alters local microvascular perfusion and induces subchondral bone remodeling and endplate sclerosis, thereby further restricting nutrient transport into the avascular disc matrix, largely independent of mechanical loading.

AN INTEGRATIVE CONCEPTUAL MODEL: *MEDA VIMARGAGAMANA* AS THE UPSTREAM DRIVER OF IVD BULGE

The authors hypothesize that an intervertebral disc bulge and its secondary clinical manifestations represent the macroscopic culmination of a long-standing, systemic pathodynamic process characterized by *Meda Dhatu Vimargagamana* into *Asthi Dhatu* territories. This integrative synthesis maps out the transition from systemic metabolic overload to localized mechanical structural failure:

[Systemic Phase]

Nidana Sevana & Jatharagni Mandya

▼

Meda Dushti & Meda Vriddhi (Unbound, circulating lipid accumulation)

▼

[Pathological Deviation]

Meda Dhatu Vimargagamana (Ectopic shift into spinal structures)

├── Vertebral Marrow Adiposity (Encroachment into internal Asthi-Majja)

└── Systemic Adipokine Infiltration (Flooding of spinal matrix)

▼

[Local Channel Obstruction]

Asthivaha Srotodushti via Localized Sanga (Endplate sclerosis / Nutrient choking)

▼

[Structural Degradation]

Asthi-Poshana Vaigunya & Local Asthi Kshaya (Dehydrated disc matrix decay)

▼

[Mechanical Tipping Point]

Vata Prakopa (Desiccation & Instability) ⟶ PIVD / Disc Bulge ⟶ Chronic LBA / Gridhrasi

1. **Systemic Initiation:**From Nidana to Meda Dushti.

Chronic *Nidana Sevana* (etiological factors) induces *Agnimandya* (metabolic deceleration), arresting the optimal conversion of lipids and generating *Abaddha* (unbound or poorly consolidated) *Meda Dhatu*. This state is interpreted by the authors as a theoretical parallel to systemic hyperlipidemia, visceral obesity, and elevated circulating free fatty acids.

2. **Pathological Deviation:**The Mechanism of Vimargagamana.

As physiological adipose storage pathways reach operational saturation, the circulating impaired lipid matrix deviates from its regulatory channel networks. Hypothesized to be driven by a latent imbalance in *Vata* (the principal governor of bodily movement), it undergoes *Vimargagamana*, shifting ectopically into the spinal domain via two concurrent pathways:

- **Vertebral Marrow Adiposity:** The physical expansion of yellow fat cells within the vertebral bodies, representing an encroachment of *Meda* into the structural domains of *Asthi* and *Majja*.
- **Systemic Adipokine Infiltration:** The chronic influx of fat-derived inflammatory markers into the spinal column, serving as a potential biochemical expression of *Meda*'s pathological influence invading the osseous matrix.

3. **Local Blockade and Structural Failure:**From Sanga to Asthi Kshaya.

Because *Meda* is a primary operational root of the *Asthivaha* network, this ectopic shift is proposed to induce *Asthivaha Srotodushti*. The local, low-grade chronic inflammatory response initiates a secondary channel pathology defined as *Sanga* (obstruction or stasis).

The authors suggest that *Sanga* represents a conceptual parallel to cartilaginous endplate (CEP) sclerosis and microvascular calcification. The physical hardening of the endplate restricts passive nutrient transit, leading to *Asthi-Poshana Vaigunya* (defective tissue nourishment) and subsequent *Asthi Kshaya* (matrix degradation, proteoglycan loss, and narrowing of intervertebral disc height).

4. **Mechanical Failure and Pain Manifestation:**Vata Prakopa to PIVD and Chronic LBA.

The progression of *Asthi Kshaya* within the disc space provokes a distinct accentuation of *Vata Prakopa* inside the localized joint segment (*Sandhi*), manifesting through the attributes of *Rukshata* (extreme structural desiccation and dehydration of the nucleus pulposus) and *Khara-Chala Gati* (mechanical joint instability).

Under ordinary axial loading, this weakened, dehydrated segment undergoes structural failure, potentially causing the nucleus pulposus to bulge through the compromised fibers of the annulus fibrosus (PIVD). This structural displacement mechanically compresses and chemically irritates adjacent nerve roots, presenting clinically as *Katigraha* (functional stiffness), chronic low back ache, and radiating neuro-inflammatory pain (*Gridhrasi*).

COMPREHENSIVE INTEGRATIVE MAPPING

Table 1: Comparative Pathophysiology of Spinal Disc Decay.

Ayurvedic Pathological State (Samprapti)	Biomedical Correlate & Molecular Mechanism	Clinical Presentation / Diagnostic Imaging
Nidana Sevana* & *Jatharagni Mandya	Chronic positive energy balance, insulin resistance, and downregulated systemic metabolic rate.	Subclinical weight gain, elevated fasting blood glucose, and metabolic fatigue.
Meda Dushti* & *Meda Vriddhi	Visceral adipose tissue expansion, dyslipidemia, and rise in circulating free fatty acids.	Elevated serum triglycerides, altered LDL/HDL ratios, and metabolic syndrome criteria.
Meda Dhatu Vimargagamana	Ectopic lipid accumulation, bone marrow adipose tissue (BMAT) expansion, and systemic adipokine hypersecretion.	High fat fraction on lumbar spine MRI; elevated serum Leptin and Resistin levels.
Asthivaha Srotodushti* & Local *Sanga	Pro-inflammatory signaling, subchondral bone remodeling, and cartilaginous endplate (CEP) calcification.	CEP sclerosis on X-ray and compromised passive nutrient diffusion into the disc space.
Asthi-Poshana Vaigunya	Hypoxic disc microenvironment, chondrocyte starvation, and downregulated aggrecan synthesis.	Early signs of disc desiccation and loss of high T2-weighted signal intensity on MRI.
Local Asthi Kshaya	Accelerated extracellular matrix (ECM) degradation via upregulation of catabolic enzymes (MMPs, ADAMTS).	Reduction in disc height and progressive narrowing of the intervertebral joint space.
***Vata Prakopa* leading to PIVD & LBA**	Total structural dehydration, mechanical instability, macroscopic herniation, and nerve root compression.	Annular tears or visible PIVD on MRI; clinical Low Back Ache, positive SLR test, and radiculopathy (*Gridhrasi*).

DISCUSSION

The integrative framework developed in this review proposes a conceptual link between systemic immunometabolic pathways and localized spinal structural failure. Historically, both traditional Ayurvedic clinical models and early contemporary orthopedics have evaluated intervertebral disc disorders predominantly through a late-stage mechanical lens. Contemporary frameworks have long categorized disc herniation as an isolated wear-and-tear event driven primarily by cumulative axial loading and physical trauma [Adams & Dolan, 2012].

Correspondingly, traditional Ayurvedic protocols typically address these presentations under the classification of *Vata-vyadhi* (such as *Gridhrasi* or *Katigraha*) or *Asthi-Majja Kshaya*, focusing therapeutic efforts on pacifying *Vata Prakopa* after macroscopic structural collapse has already taken place.

While these mechanical and neurological interventions effectively target the symptomatic terminal phase of the condition, they leave a critical analytical gap regarding the mechanisms by which systemic metabolic disorders and lipid perversion may accelerate structural disc breakdown largely independent of physical spinal workload [Inoue and Espinoza Orias, 2006].

Reinterpreting the Meda-Asthi Continuum via Molecular Biology

By analyzing the classical principle establishing *Meda Dhatu* as the functional root of the *Asthivaha Srotas* (Caraka Saṃhitā, Vimānasthāna 5/8), this study introduces a systemic-to-local pathogenetic model. In Ayurvedic physiology, tissue nutrition follows a structured sequential cascade (*Dhatu-Parampara*), where healthy *Meda* serves as the immediate metabolic precursor for the synthesis and structural integrity of *Asthi Dhatu*.

When systemic metabolic dysregulation is driven by chronic *Nidana Sevana* and *Jatharagni Mandya*, the lipid matrix undergoes qualitative impairment, transforming into *Abaddha* (unbound) and *Aparipakwa* (immature) *Meda Dhatu*. This state corresponds theoretically to the contemporary biomedical profile of visceral obesity, circulating hyperlipidemia, and elevated free fatty acids.

The subsequent shift where this perverted lipid matrix leaks into foreign anatomical spaces represents a pathological manifestation of *Meda Dhatu Vimargagamana*. In the lumbar spine, this metabolic deviation may manifest via the structural expansion of yellow BMAT replacing hematopoietically active marrow within the vertebral bodies, alongside the chronic, low-grade systemic infiltration of fat-derived adipokines into the avascular microenvironment of the intervertebral disc.

The Sanga-Sclerosis Conundrum: Mechanics of Nutrient Starvation

A core insight of this review is the mapping of *Srotodushti* expressions operating in a multi-tiered sequence. The biochemical flooding (*Vimargagamana*) of inflammatory markers is hypothesized to alter local subchondral bone remodeling, triggering a secondary localized

channel pathology defined as *Sanga* (obstruction). This conceptual model aligns with the biomedical mechanics of Cartilaginous Endplate (CEP) sclerosis and microvascular calcification.

Because the adult intervertebral disc is the largest avascular structure in the human body, its metabolic survival is understood to depend predominantly on the passive diffusion of essential nutrients through the microscopic pores of the CEP [Adams & Roughley, 2006]. When *Meda Vimargagamana* drives endplate calcification, these vital pores are physically compromised. This matches the classical definition of *Srotorodha* or *Sanga,* leading to *Asthi-Poshana Vaigunya* (defective tissue nourishment).

Starved of nutrients, the chondrocytes inside the Nucleus Pulposus shift to an anaerobic, catabolic survival mode, upregulating matrix-degrading enzymes (MMPs and ADAMTS) while downregulating the synthesis of water-binding aggrecan molecules. The resulting loss of hydrophilic glycosaminoglycans potentially accelerates rapid disc desiccation—the modern biochemical equivalent of *Rukshata*—reducing the disc's height and its mechanical capacity to handle axial stress..

The Feedback Loop Between Vata Prakopa and Metabolic Failure

This review suggests that macroscopic Prolapsed Intervertebral Disc (PIVD) and its primary clinical manifestation, Low Back Ache (LBA), are not merely sudden, isolated mechanical events, but rather the structural tipping point of a long-standing metabolic crisis. As localized *Asthi Kshaya* degrades the cartilage matrix, the structural containment fails under routine mechanical strain, causing the Nucleus Pulposus to herniate through the compromised fibers of the Annulus Fibrosus. This mechanical displacement is proposed to cause a distinct flare-up of *Vata Prakopa* within the *Sandhi* (joint segment), compressing or chemically irritating adjacent nerve roots to produce the severe radiculopathy characteristic of *Gridhrasi* (Sciatica).

Crucially, a bilateral feedback loop may be established: the neuro-inflammatory signaling of *Vata Prakopa* further increases local tissue stress, while the chronic pain and subsequent sedentary lifestyle (*Avyayama*) of the patient exacerbate *Jatharagni Mandya,* locking the individual into a cycle of metabolic decline and progressive spinal degeneration.

Translational Clinical Implications: Shifting the Treatment Paradigm

The clinical value of this integrative paradigm lies in its potential to transform therapeutic strategies. Current contemporary medicine often remains highly polarized, offering short-term symptomatic management for LBA (NSAIDs, epidural steroid injections) or invasive surgical interventions for late-stage PIVD (discectomy, spinal fusion) without directly addressing upstream metabolic drivers.

By validating *Meda Vimargagamana* as a plausible upstream contributor, this model suggests that spinal disc therapies must incorporate systemic interventions. Instead of waiting for advanced mechanical collapse and managing it solely with late-stage local oil applications (*Snehana*) or localized anti-inflammatory drugs, early therapeutic models should focus on clearing channel blockages and regulating systemic lipids using metabolic therapies:

- ***Langhana* & *Deepana-Pachana*:** Targeted fasting protocols and bio-cleansing agents administered to correct *Jatharagni Mandya* and eliminate *Aparipakwa Meda* (circulating lipotoxins).
- ***Rukshana* & *Kledahara Upakrama*:** Drying and moisture-depleting therapies designed theoretically to counter vertebral marrow fat accumulation and downregulate systemic adipokine expression.
- ***Lekhana Basti*:** Medicated, depleting enemas specifically indicated in classical texts to clear *Srotas* obstructions (*Sanga*) and restore proper nutrient pathways to the *Asthi Dhatu* matrix before irreversible mechanical structural failure occurs.

Ultimately, this integrative model highlights that managing an intervertebral disc bulge successfully requires evaluating the spine not as an isolated mechanical column, but as a dynamic structure deeply connected to the body's systemic immunometabolic health.

CONCLUSION

This integrative conceptual review proposes that a Prolapsed Intervertebral Disc (PIVD) and chronic Low Back Ache (LBA) are not merely isolated mechanical wear-and-tear events, but rather structural consequences of a long-standing, systemic immunometabolic crisis. By re-evaluating the classical *Meda-Asthi* continuum, this study positions the qualitative impairment of lipids (*Meda Dushti*) and its subsequent ectopic deviation into the spinal domain (*Vimargagamana*) as a plausible upstream driver of disc matrix decay.

This pathological overflow—characterized by vertebral marrow adiposity and systemic adipokine flooding—is proposed to trigger a localized channel blockade (*Sanga*) across the cartilaginous endplates, potentially restricting vital nutrient diffusion to the resident disc cells.

The resulting cellular starvation leads to disc desiccation (*Asthi Kshaya*), culminating in mechanical collapse and severe neuro-inflammatory excitation (*Vata Prakopa*).

Ultimately, this novel framework introduces a shift in clinical management, encouraging healthcare providers to move beyond reactive, late-stage symptomatic care and employ preventative, upstream Ayurvedic metabolic interventions—such as *Langhana*, *Rukshana*, and *Lekhana Basti*—to stabilize lipid homeostasis and preserve spinal structural integrity before irreversible mechanical breakdown occurs.

FUTURE SCOPE

- **Biochemical Profiling:** Clinical trials are required to evaluate the statistical correlation between traditional *Meda Dushti* phenotypes and circulating serum biomarkers (Leptin, Resistin, and $TNF-\alpha$) in patients diagnosed with PIVD and chronic LBA.
- **Advanced Radiological Trials:** Designing MRI-based protocols to evaluate the potential relationship between vertebral bone marrow adipose tissue (BMAT) fractions, paraspinal muscle fatty infiltration, and the clinical severity of *Asthivaha Srotodushti* and macroscopic disc herniation.
- **Targeted Clinical Trials:** Conducting controlled clinical trials to assess the efficacy of upstream Ayurvedic metabolic interventions (*Langhana*, *Rukshana*, *Deepana-Pachana*) in preserving disc height and slowing mechanical disc bulging (PIVD) in early-stage metabolic syndrome patients presenting with mild LBA.

LIMITATIONS

- This study is a conceptual narrative review and requires direct clinical and translational validation to confirm these proposed theoretical correlations.
- Traditional Ayurvedic pathophysiological concepts like *Vimargagamana* and *Dhatvagni Vaigunya* currently lack universally standardized, objective measurement metrics in contemporary clinical research.
- The contemporary biomedical data used in this synthesis is largely associative, derived from cross-sectional epidemiological frameworks; establishing definitive causal pathways requires long-term, longitudinal molecular studies.

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