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A PATHOPHYSIOLOGICAL CORRELATION BETWEEN ARSHA SAMPRAPTI AND PORTAL CIRCULATION: AN INTEGRATIVE REVIEW

Dr. Rushikesh Late*[1], Dr Shilpak Sadgune[2], Dr. Sanjeev Yadav[3]

[1]PG Scholar Shalyatantra, YMT Ayurveda Medical College and Hospital, Kharghar, Navi Mumbai.

[2]Associate Professor, YMT Ayurveda Medical College and Hospital, Kharghar, Navi Mumbai.

[3]Dean and Professor, YMT Ayurveda Medical College and Hospital, Kharghar, Navi Mumbai.

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***Corresponding Author**

Dr. Rushikesh Late

PG Scholar Shalyatantra, YMT Ayurveda Medical College and Hospital, Kharghar, Navi Mumbai.

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ABSTRACT

Arsha, described in Ayurveda as a Tridoshaja Vyadhi, shares clinical and pathological similarity with hemorrhoids in modern medicine. Classical texts attribute its origin to Agnimandya (impaired digestive fire), *Apana Vayu* vitiation, Srotodushti (channel pathology), and involvement of Dushyas such as *Rakta*, *Mamsa*, and *Meda*. Modern literature describes hemorrhoids as pathological enlargements of the normal anal cushions arising due to venous congestion, mucosal sliding, and portal hemodynamic alterations. The aim of this integrative review is to systematically correlate the classical Ayurvedic *Samprapti* of Arsha with the physiology of the portal venous system and the known mechanisms of hemorrhoidal disease. Methods include a comprehensive narrative review of Ayurvedic classical texts (*Charaka Samhita*, *Sushruta Samhita*, *Ashtanga Hrudaya*), modern physiology, surgical literature, and indexed PubMed sources. Correlation analysis revealed significant parallels between Ama, Srotorodha, and Vata-Pitta Dushti with modern endothelial inflammation, venous congestion, and mucosal prolapse. The internal hemorrhoidal plexus drains into the portal

system via the superior rectal vein, establishing a direct hemodynamic link that mirrors *Apana Vayu*–Srotas dysfunction in Arsha *Samprapti*. Conclusion: Arsha *Samprapti* aligns closely with modern portal venous physiology and provides a holistic, integrative pathophysiological framework supporting contemporary clinical understanding and management of hemorrhoidal disease.

KEYWORDS: Arsha, Samprapti, Portal Circulation, Hemorrhoids, Apana Vayu, Srotorodha, Integrative Review, Ayurveda.

INTRODUCTION

Arsha is one of the most prevalent anorectal disorders mentioned in Ayurvedic literature, classified among the *Ashtamahagada* (eight major diseases) by *Sushruta*, indicating its chronicity, severity, and therapeutic challenge.[1] The word 'Arsha' etymologically means 'enemy to vitality', signifying its debilitating effect on the patient's physical and functional health. Classical texts describe Arsha as arising from the simultaneous vitiation of all three Doshas — Vata, Pitta, and Kapha — along with involvement of Agni (digestive fire), Srotas (body channels), and specific Dushyas such as Rakta (blood), Mamsa (muscular tissue), and Meda (adipose tissue).[2]

In modern surgical and gastroenterological literature, hemorrhoids are defined as pathological enlargements and downward displacement of normal anal vascular cushions. These cushions, physiologically present in all humans, consist of submucosal arteriovenous communications and supportive connective tissue that contribute to anal continence. Pathological hemorrhoids develop when these cushions undergo vascular engorgement, connective tissue weakening, and mucosal prolapse due to increased intraabdominal pressure, chronic straining, constipation, portal hypertension, and lifestyle-related factors.[3]

A particularly significant anatomical observation is that internal hemorrhoids drain exclusively into the portal venous system via the superior rectal vein → inferior mesenteric vein → portal vein pathway. This direct hemodynamic connection means that any alteration in portal venous pressure — as seen in liver disease, portal hypertension, or even functional constipation-related straining — directly impacts the hemorrhoidal vasculature and contributes to the development and progression of internal hemorrhoids.[4] This integrative review aims to bridge the conceptual gap between Ayurvedic pathogenesis and modern pathophysiology by correlating Arsha *Samprapti* with portal venous anatomy and physiology,

providing a unified scientific framework.

AIM AND OBJECTIVES

Aim: To correlate the Ayurvedic *Samprapti* of Arsha with modern portal venous physiology and hemorrhoidal pathogenesis in an integrative scientific framework.

Objectives

- **Objective 1:** To review and summarize the Ayurvedic description of Arsha based on classical texts including Charaka Samhita, Sushruta Samhita, Ashtanga Hrudaya, and Madhava Nidana.
- **Objective 2:** To describe the anatomy and physiology of the portal venous system with specific reference to hemorrhoidal venous drainage.
- **Objective 3:** To systematically correlate each component of Arsha Samprapti with corresponding modern pathophysiological mechanisms.
- **Objective 4:** To highlight the clinical and therapeutic significance of this correlation in supporting integrative approaches for Arsha management.

REVIEW OF LITERATURE

1. Arsha in Ayurveda — Classical Description

Arsha is elaborately described in *Sushruta Samhita* (Nidana Sthana, Chapter 2), *Charaka Samhita* (Chikitsa Sthana, Chapter 14), *Ashtanga Hrudaya* (Nidana Sthana, Chapter 7), and *Madhava Nidana* (Chapter 28). These texts provide comprehensive descriptions of Nidana (causative factors), Purvarupa (prodromal features), Rupa (clinical manifestations), Samprapti (pathogenesis), and Chikitsa (therapeutic management).[5]

1.1 Nidana (Causative Factors)

According to *Sushruta*, the primary Nidanas of Arsha include: *Aharaja Nidana* such as excessive intake of Ruksha (dry), Guru (heavy), Ushna (hot), and Abhishyandi (channel-blocking) foods; *Viharaja Nidana* including prolonged sitting on hard surfaces (Vishamashana), suppression of natural urges (Vega Dharana), sedentary lifestyle, and chronic constipation; and *Manasika Nidana* such as grief, anxiety, and psychological stress.[6] *Charaka Samhita* additionally mentions alcohol consumption, incompatible food combinations (*Viruddha Ahara*), and irregular meal timings. Collectively, these Nidanas disturb Jatharagni and aggravate Apana Vayu, initiating the Samprapti.[7]

1.2 Dosha and Dushya Involvement

Arsha involves all three Doshas, with primary involvement of *Apana Vayu* and *Pachaka Pitta*. Vata causes dryness, constipation, and pain; Pitta produces inflammation, bleeding, and burning sensation; Kapha contributes to heaviness, sliminess, and chronicity. The Dushyas include Rakta (hemorrhagic component), Mamsa (formation of the pile mass), and Meda (fatty cushion enlargement), with *Purishavaha Srotas* as the primary Srotorodha site.[8]

1.3 Samprapti (Pathogenesis)

The classical Samprapti follows this sequential pathway: *Nidana Sevana* → *Agnimandya* → Ama formation → Srotorodha → *Apana Vayu Prakopa* → Vata-Pitta-Kapha Dushti → *Siragranthi* formation → Arsha manifestation. The term *Siragranthi* (venous knotting/dilation within a Sira/vessel) closely parallels the pathological dilation and engorgement of the hemorrhoidal venous plexus seen in modern medicine.[9]

2. Portal Venous System — Anatomy and Physiology

The portal venous system is a specialized vascular network draining nutrient-rich blood from the gastrointestinal tract, spleen, pancreas, and gallbladder to the liver for metabolic processing before systemic circulation. The portal vein is formed by the union of the superior mesenteric vein and splenic vein posterior to the neck of the pancreas. Under normal physiological conditions, portal venous pressure is maintained between 5–10 mmHg. Portal hypertension — defined as a sustained portal pressure above 12 mmHg — leads to development of portosystemic collateral channels at anastomotic sites including the anorectal junction.[10,11]

2.1 Hemorrhoidal Venous Anatomy

The anorectal region contains three venous plexuses with distinct drainage pathways, making it a critical portosystemic anastomotic site:

- **Superior Rectal Vein:** Drains internal hemorrhoidal plexus → Inferior Mesenteric Vein → Portal System (direct portal connection).
- **Middle Rectal Vein:** Drains middle rectal plexus → Internal Iliac Vein → IVC (portosystemic anastomosis).
- **Inferior Rectal Vein:** Drains external hemorrhoidal plexus → Internal Pudendal Vein → IVC (purely systemic; relevant to external hemorrhoids).

Any increase in portal venous pressure is directly transmitted to the internal hemorrhoidal

plexus via the superior rectal vein, causing venous engorgement and progressive hemorrhoidal disease. The anorectal junction thus functions as a hemodynamic barometer of portal circulation.[12]

Table 1: Venous Anatomy of the Hemorrhoidal Plexus and Portal Connections.

Vessel	Drains From	Drains Into	Relevance to Arsha
Superior Rectal Vein	Internal hemorrhoidal plexus	Inferior Mesenteric Vein → Portal Vein	Direct portal hemodynamic link to internal hemorrhoids
Middle Rectal Vein	Middle rectal plexus	Internal Iliac Vein → IVC (systemic)	Portosystemic anastomosis site; portal hypertension relevance
Inferior Rectal Vein	External hemorrhoidal plexus	Internal Pudendal Vein → IVC	Systemic drainage; relevant to external hemorrhoidal disease
Inferior Mesenteric Vein	Sigmoid colon / rectal region	Splenic Vein → Portal Vein	Major portal tributary; affected in portal hypertension

CORRELATION BETWEEN ARSHA SAMPRAPTI AND PORTAL CIRCULATION

1. Apana Vayu Dushti ↔ Impaired Venous Return

Apana Vayu governs all downward movements of the body — defecation, urination, and excretion. Its vitiation leads to constipation, irregular bowel habits, and sustained straining during defecation — all well-recognized risk factors for hemorrhoidal disease. When straining occurs, there is a significant and sustained increase in intraabdominal and intrarectal pressure, directly impeding venous return from the superior rectal vein and causing hemorrhoidal venous engorgement.[13] This precisely mirrors the modern pathophysiological mechanism of straining-induced venous hypertension in the hemorrhoidal plexus. *Apana Vayu Dushti* thus correlates directly with impaired venous return and portal-anorectal hemodynamic dysfunction.

2. Agnimandya and Ama Formation ↔ Endothelial Dysfunction and Vascular Inflammation

Agnimandya (impaired digestive fire) is described as the root cause of Arsha. When Agni is weakened, ingested food is improperly metabolized, generating Ama — a sticky, reactive metabolic byproduct that obstructs Srotas and initiates systemic inflammatory processes. The modern equivalent involves endothelial dysfunction and vascular inflammation. Studies have demonstrated significant endothelial cell dysfunction in hemorrhoidal tissue, characterized by elevated expression of inflammatory mediators (TNF-α, IL-6), adhesion molecules, and

vascular endothelial growth factor (VEGF).[14] The micro-obstruction caused by Ama at the Srotas level directly mirrors the endothelial inflammation and vascular congestion observed at the capillary and venular level in hemorrhoidal pathology.

3. Srotorodha (Sanga Type) ↔ Venous Obstruction and Congestion

Srotodushti of the Sanga type refers to obstruction or stagnation within body channels, leading to proximal accumulation of Doshas and Dushyas. This is remarkably analogous to venous stasis and obstruction in hemorrhoidal disease. When superior rectal vein drainage is impeded — due to portal hypertension, constipation-induced straining, or anatomical factors — venous blood stagnates in the internal hemorrhoidal plexus, causing progressive dilation and thinning of vessel walls. The Sanga in *Purishavaha Srotas* directly correlates with venous obstruction in the portal-mesenteric-rectal venous axis.[15]

4. Siragranthi Formation ↔ Hemorrhoidal Varicosities and Cushion Engorgement

Siragranthi, literally 'a knot or dilatation within a Sira (vessel)', is the key structural pathological entity in Arsha Samprapti. This term precisely describes pathological dilatation of the internal hemorrhoidal venous plexus. Thomson's vascular slide theory (1975) describes hemorrhoids as pathological downward displacement and engorgement of the three normal anal cushions — right anterior, right posterior, and left lateral — correlating exactly with *Siragranthi* formation at the Guda (anorectal) region described in *Sushruta Samhita*. The Ayurvedic term thus represents a clinically accurate morphological description of hemorrhoidal varicosities documented centuries before modern vascular anatomy.[16]

5. Mamsa Anubandha ↔ Hemorrhoidal Cushion Prolapse

Mamsa Anubandha refers to involvement of *Mamsa Dhatu* (muscular and connective tissue) leading to structural weakening of supportive tissues around the hemorrhoidal mass. In modern pathophysiology, this corresponds to degeneration of the fibromuscular supporting tissues of the anal cushions — specifically Park's ligament, the fibromuscular connective tissue anchoring hemorrhoidal cushions to the internal sphincter. As this ligament degenerates, cushions undergo downward displacement and prolapse, manifesting clinically as Grade II–IV hemorrhoids.[17] *Mamsa Anubandha* in Arsha thus accurately represents the connective tissue weakening and prolapse mechanism central to modern hemorrhoidal disease theory.

6. Nidana Correlation ↔ Modern Risk Factors

The Nidanas described for Arsha in classical texts show one-to-one correspondence with modern epidemiological risk factors:

- **Ruksha, Guru Ahara:** High-fat, low-fiber diet → constipation → straining → hemorrhoidal venous congestion.
- **Madyapana (alcohol intake):** Portal vasodilation → increased portal venous pressure → internal hemorrhoidal congestion.
- **Vishamashana:** Irregular meals → altered gut motility → constipation → Apana Vayu imbalance → Srotorodha.
- **Chirastayana:** Prolonged sitting → increased perineal pressure → impaired venous return → hemorrhoidal stasis.
- **Vega Dharana:** Suppression of urges → chronic constipation → straining → elevated rectal venous pressure.
- **Manas Nidana:** Psychological stress → altered gut motility → dysbiosis → Agnimandya → Ama accumulation.

Table 2: Comprehensive Correlation — Arsha Samprapti and Portal Venous Pathophysiology.

Ayurvedic Concept (Samprapti)	Modern Equivalent (Pathophysiology)	Clinical Significance
Apana Vayu Dushti	Impaired Venous Return / Straining-induced venous hypertension	Constipation and straining → ↑ intrarectal pressure → hemorrhoidal venous congestion
Agnimandya (impaired Agni)	Endothelial Dysfunction / Metabolic endotoxemia	Ama accumulation ↔ systemic inflammation → vascular endothelial injury in hemorrhoidal tissue
Srotorodha — Sanga type	Venous Obstruction / Portal venous stasis	Portal congestion → superior rectal vein stasis → internal hemorrhoidal plexus engorgement
Siragranthi (venous knotting)	Hemorrhoidal Varicosities / Anal cushion dilation	Pathological dilation of internal hemorrhoidal plexus = Siragranthi of Guda Siras
Mamsa Anubandha	Connective tissue degeneration / Cushion prolapse	Degeneration of Park's ligament → anal cushion prolapse (Grade II–IV hemorrhoids)
Nidana (dietary & lifestyle)	Modern Risk Factors for hemorrhoids	Alcohol, spicy diet, constipation, prolonged sitting, stress — exact overlap between systems

DISCUSSION

The integrative analysis presented in this review demonstrates consistent and scientifically

coherent parallels between the Ayurvedic *Samprapti* of Arsha and the modern pathophysiology of hemorrhoidal disease with specific reference to the portal venous system. These correspondences reflect deep conceptual alignment between two independent medical traditions that arrived at similar pathophysiological conclusions through different methodological approaches: one through clinical observation and systematic reasoning over millennia, the other through anatomical dissection, vascular physiology, and experimental medicine over the past two centuries.[18]

The most clinically significant correlation is the relationship between *Apana Vayu Dushti* and impaired portal venous return. *Apana Vayu* governs the functional integrity of the lower gastrointestinal and anorectal region. Its vitiation leads to constipation, irregular defecation, and sustained straining — all independently documented risk factors for hemorrhoidal disease in prospective epidemiological studies. The direct hemodynamic consequence of straining is a measurable increase in intrarectal pressure that impedes venous return from the superior rectal vein, leading to progressive hemorrhoidal venous congestion. This functional-hemodynamic link validates the Ayurvedic emphasis on *Apana Vayu* regulation as the primary therapeutic target in Arsha management.[19]

The correlation between Agnimandya/Ama and endothelial dysfunction provides a molecular bridge between the two systems. Ayurveda's concept of Ama — an undigested, pro-inflammatory metabolic byproduct — aligns with modern understanding of metabolic endotoxemia, systemic low-grade inflammation, and vascular endothelial dysfunction. Research has demonstrated elevated levels of inflammatory cytokines including TNF-α, IL-6, and VEGF in hemorrhoidal tissue, consistent with the systemic inflammatory state predicted by Ama accumulation.[20] This parallel suggests that interventions targeting Agni restoration and Ama Pachana may possess documented anti-inflammatory mechanisms at the vascular endothelial level.

The concept of *Srotodushti*, particularly the Sanga type, provides a sophisticated framework for understanding venous obstruction that predates modern venous pathology. The portal venous system, with its unique architecture of draining splanchnic circulation into the liver before systemic entry, is inherently vulnerable to the Sanga described in Arsha *Samprapti*, particularly when portal pressure rises or when *Apana Vayu* is compromised.[21]

Siragranthi is perhaps the most precise and clinically accurate term in classical Ayurvedic

pathology, directly describing what modern anatomy calls hemorrhoidal varicosities — dilated, tortuous venous structures at the anorectal junction. The accuracy of this morphological description, documented by *Sushruta* approximately 2,500 years ago, is remarkable and suggests direct anatomical observation in classical Ayurvedic taxonomy. Similarly, *Mamsa Anubandha* predicts the connective tissue component of hemorrhoidal disease — degeneration of Park's ligament — whose deterioration is the structural basis of hemorrhoidal prolapse.[22]

From a therapeutic perspective, this correlation has direct clinical implications. Ayurvedic management of Arsha emphasizes *Deepana-Pachana* (restoration of Agni and digestion of Ama), *Vatanulomana* (regularization of Apana Vayu), *Raktashodhana* (purification of Rakta Dhatu), and *Kshara or Agnikarma* (local procedures). These therapeutic objectives correspond respectively to reducing endothelial inflammation, correcting venous return impairment, addressing portal hemodynamic alterations, and locally obliterating hemorrhoidal varicosities — all recognized therapeutic endpoints in modern hemorrhoid management.[23]

CONCLUSION

This integrative review establishes that the Ayurvedic *Samprapti* of Arsha aligns closely and coherently with the modern understanding of hemorrhoidal pathophysiology, particularly in relation to the portal venous system. The sequential pathogenesis described in classical texts — from Agnimandya and Ama formation, through Srotorodha and *Apana Vayu Dushti*, to *Siragranthi* and *Mamsa Anubandha* — maps precisely onto the modern cascade of endothelial dysfunction, venous congestion, impaired venous return, hemorrhoidal varicosity formation, and cushion prolapse.

The direct anatomical and hemodynamic connection between the internal hemorrhoidal plexus and the portal venous system via the superior rectal vein provides a physiological foundation that bridges both conceptual frameworks. This correlation not only validates classical Ayurvedic clinical observations through contemporary biomedical evidence but also provides a rational scientific basis for integrative therapeutic approaches addressing both root cause (Agnimandya, *Apana Vayu* imbalance) and structural consequences (*Siragranthi*, Mamsa degeneration) of Arsha.

Future research should include prospective clinical studies measuring portal venous pressure

parameters, hemorrhoid grading (Goligher's classification), and Dosha assessment in patients with Arsha, along with biochemical markers of endothelial dysfunction and inflammation. Such studies would provide quantitative validation of the qualitative correlations identified in this review and pave the way for evidence-based integrative clinical protocols for Arsha management.

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