

THORACIC DUCT AND LYMPHATIC CONGESTION IN CARDIOVASCULAR DISEASE: AN INTEGRATED AYURVEDIC AND BIOMEDICAL LITERATURE REVIEW

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Article Info: Received: 18 July 2026, Revised: 08 August 2026, Accepted: 28 August 2026

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DOI: <https://doi.org/10.5281/zenodo.22210445>



Citation:

¹Dr. S. V. Emmi, ²Dr. Vishal Patil. (2026). Thoracic Duct And Lymphatic Congestion In Cardiovascular Disease: An Integrated Ayurvedic And Biomedical Literature Review. International Journal of Clinical and Pharmaceutical Innovations, 1(6), 1-12.

ABSTRACT

Background: Lymphatic dysfunction and thoracic duct congestion are emerging as critical yet underappreciated pathomechanisms in cardiovascular disease (CVD). Classical Ayurvedic medicine offers an elaborate conceptual framework — centered on Rasavaha Srotas, Hridaya Marma, and Medo Dhatu — that closely parallels contemporary biomedical understanding of the lymphatic-cardiac axis. This literature review synthesises the biochemical, physiological, pharmacological, and pathophysiological evidence linking thoracic duct dysfunction to coronary artery disease, heart failure, and atherosclerosis, with concurrent integration of Ayurvedic classical textual references. **Methods:** A systematic literature search was conducted across PubMed, Scopus, Ayush Research Portal, and classical Sanskrit textual databases (Charaka Samhita, Sushruta Samhita, Ashtanga Hridayam, Sharangadhara Samhita) for studies published up to 2024. Keywords included: lymphatic congestion, thoracic duct, cardiovascular disease, VEGF-C, LYVE-1, Rasavaha Srotas, Hridroga, Srotorodha, and cardioprotective phytochemicals. One hundred references were critically reviewed and synthesized. **Results:** Robust experimental and clinical evidence confirms that impaired cardiac lymphangiogenesis accelerates post-infarction inflammation, myocardial fibrosis, and pericardial pathology. Cholesterol-laden chylomicron reflux from a congested thoracic duct contributes measurably to coronary atherogenesis. The Ayurvedic construct of Rasavaha Srotorodha (channel obstruction) maps mechanistically to lymphatic congestion, while Medo Dhatu Dushti corresponds to dyslipidaemia. Eight key medicinal plants — Punarnava, Arjuna, Guggulu, Pippali, Devadaru, Haritaki, Varun, and Shilajatu — demonstrate lymphagogue, anti-atherogenic, anti-inflammatory, and cardioprotective activities at biochemical and molecular levels. **Conclusion:** The lymphatic-cardiac interface represents a convergence zone where classical Ayurvedic pathophysiology and modern biomedical science achieve remarkable congruence. Polyherbal Ayurvedic formulations targeting this axis hold significant therapeutic promise that warrants rigorous clinical evaluation.

KEYWORDS: Thoracic duct; Lymphatic congestion; Cardiovascular disease; Rasavaha Srotas; Hridroga; Srotorodha; Ayurvedic cardiology; VEGF-C; Guggulu; Arjuna; Punarnava; Medo Dhatu Dushti.

1. INTRODUCTION

The lymphatic system - long regarded primarily as a drainage apparatus is now recognised as an integral regulator of cardiovascular homeostasis. The thoracic duct, the largest lymphatic vessel of the human body, drains approximately 75% of total body lymph, including intestinal chylomicron-rich chyle, into the left subclavian vein at the thoracic inlet.^[41, 42] Disruption of this drainage pathway produces wide-ranging cardiovascular consequences, from myocardial oedema and pericardial effusion to accelerated atherogenesis consequences that were, remarkably, anticipated in classical Ayurvedic texts composed over two millennia ago.

Emerging biomedical literature has compellingly demonstrated that: (i) cardiac lymphangiogenesis - the formation of new lymphatic vessels- is impaired following myocardial infarction, thereby perpetuating inflammation and fibrosis;^[44,48,49] (ii) elevated interstitial pressure consequent to lymphatic stasis promotes cardiomyocyte necrosis and replacement fibrosis;^[45,46] (iii) cholesterol-laden lymph from the congested thoracic duct seeds atherosclerotic plaques;^[51,52,53,54] and (iv) pericardial lymphatic insufficiency is causally linked to recurrent pericarditis and constrictive pericardiopathy.^[43,59]

In Ayurvedic medicine, the Charaka Samhita (Vimana Sthana, Chapter 5) elaborately describes Rasavaha Srotas — the channels conveying Rasa (plasma and lymph) — and their central role in the maintenance of Hridaya (cardiac) function.^[4] Obstruction of these channels (**Srotorodha**), vitiation of **Medo Dhatu** (adipose and lipid fraction), and impairment of **Agni** (digestive and metabolic fire) are posited as the foundational pathological triad underpinning Hridroga (cardiac disease).^[1,3,8] This conceptual convergence between Ayurvedic and biomedical frameworks provides an outstanding opportunity for integrative pharmacological research.

The present review comprehensively examines the biochemical basis, physiology, pharmacology, pharmacokinetics, pharmacodynamics, kinetic action, and pathophysiological effects of thoracic duct dysfunction on cardiovascular diseases, with concurrent Ayurvedic classical textual correlation and evidence for phytomedicinal interventions targeting the lympho-cardiac axis.

2. HISTORICAL BACKGROUND AND CLASSICAL AYURVEDIC PERSPECTIVES

2.1 Ayurvedic Conceptualisation of the Lymphatic-Cardiac Axis

The Charaka Samhita, in its Vimana Sthana (Chapter 5 — Srotovimaniya Adhyaya), provides the most comprehensive classical description of bodily channels and their pathological variations.^[4] Srotavidushti (channel vitiation) is described as occurring through four mechanisms: **Atipravrutti** (excessive flow), **Sanga**

(obstruction), **Vimarga Gamana** (flow in wrong direction), and **Siragranthi** (nodular dilatation). Each of these modes has precise modern biomedical correlates in lymphatic pathophysiology — **hyperlymphorrhoea**, **lymphoedema**, **chylous reflux**, and **lymphangiomatosis** respectively.

Rasavaha Srotas is explicitly described in Charaka Samhita (Vimana Sthana 5.8) as having its *Moola* (root) in Hridaya (heart) and Dasha Dhamani (ten great vessels).⁴ The correspondence between Rasa (plasma-lymph fraction), the primordial nourishing fluid, and lymph carrying absorbed nutrients from the intestine through the thoracic duct is well established in contemporary Ayurvedic scholarship.^[5,71,72] Acharya Charaka further states that vitiation of Rasavaha Srotas manifests as Praseka (excessive salivation/fluid accumulation), Aruchi (anorexia), Chardi (nausea), Gaurava (heaviness), Jwara (fever), and Pandu (pallor) — symptoms that collectively represent the syndrome of cardiac lymphatic insufficiency with anasarca.^[4]

Sushruta Samhita (Sharira Sthana, Chapter 9 — Marma Prakarana) identifies Hridaya as a Sadya Pranahara Marma — a vital anatomical nexus whose injury is immediately fatal.^[6] Sushruta's description of the Hridaya as a ten-fingered breadth structure, pendant and cone-shaped, with pulsatile Siras (vessels) and Dhamani (arteries), reflects sophisticated cardiovascular anatomical knowledge.^[2,9] The Nibandhasangraha commentary of Dalhana on Sushruta Samhita elaborates that the Hridaya is continuously bathed in lymph-like fluid (Rasa) and that its proper function depends on unrestricted Rasa circulation.^[9]

Vagbhata in Ashtanga Hridayam (Nidana Sthana, Chapter 12 — Shotha Nidana) provides a detailed pathogenetic account of Shotha (oedema), describing how Rasavaha Srotorodha leads to Sthana-Samshraya (localisation) of vitiated Doshas in the interstitium, producing Shotha with cardinal features of pitting oedema, heaviness, and reduced mobility.^[3] This mechanistic description is congruent with modern understanding of cardiac lymphoedema arising from elevated capillary filtration with insufficient lymphatic drainage.^[45,47]

2.2 Hridroga — Classical Classification and Biomedical Correlation

Table 1: Classical Ayurvedic classification of Hridroga (Charaka Samhita, Chikitsa Sthana, Chapter 26) with modern biomedical correlation.^[1,71,72]

Hridroga Type	Classical Description (Charaka Samhita Ch. 26)	Doshas	Modern Biomedical Correlation
Vataja Hridroga	Hrid Shoola (chest pain), Palpitation, Anxiety, Tachycardia	Vata Pradhanata	Coronary artery spasm, Arrhythmia, Hypertensive heart disease
Pittaja Hridroga	Burning, Fever, Sweating, Pallor, Thirst	Pitta Pradhanata	Inflammatory cardiomyopathy, Myocarditis, Pericarditis
Kaphaja Hridroga	Heaviness, Cough, Oedema, Lethargy, Dyspnoea	Kapha Pradhanata	Congestive heart failure, Dilated cardiomyopathy, Pericardial effusion
Tridoshaja Hridroga	All above symptoms combined; grave prognosis	Sannipata	Advanced CVD with multi-system involvement
Krimija Hridroga	Unbearable pain, Itching, Infection signs, Rapid deterioration	Krimija (infective)	Infective endocarditis, Rheumatic heart disease, Chagas disease

3. ANATOMY AND PHYSIOLOGY OF THE THORACIC DUCT AND CARDIAC LYMPHATICS

3.1 Gross Anatomy of the Thoracic Duct

The thoracic duct (Ductus thoracicus) originates at the Cisterna chyli (L1-L2 level), ascends through the aortic hiatus of the diaphragm, traverses the posterior mediastinum, crosses the midline at T4-T5, and terminates at the left venous angle (junction of left internal jugular and subclavian veins).^[41,46] It measures 38-45 cm in length with a diameter of 1-5 mm, contains approximately 8-10 bicuspid valves to prevent retrograde flow, and transports 2-4 litres of lymph daily under normal conditions.^[42,46] The cardiac lymphatics arise from subendocardial, myocardial, and subepicardial plexuses, draining into collecting vessels that converge on mediastinal nodes and ultimately into the thoracic duct.^[46,48]

Miller (1982) provided the seminal anatomical mapping of cardiac lymphatics, demonstrating that the subendocardial network is the most extensive, with dense capillary loops in the papillary muscles and interventricular septum — regions most vulnerable to ischaemia.^[46] Klotz et al. (2015) subsequently demonstrated using lineage-tracing studies that cardiac lymphatics are heterogeneous in embryological origin — derived from both venous endothelium and extra-vascular progenitors — and respond differentially to injury.^[48]

3.2 Ayurvedic Anatomical Parallel: Dasha Dhamani and Rasayani

Charaka Samhita (Sharira Sthana, Chapter 7) describes Hridaya as the origin of Dasha Dhamani (ten great vessels) including Rasayani — vessels specifically carrying Rasa (plasma-lymph).^[5] Sushruta Samhita (Sharira Sthana, Chapter 9) elaborates on Sira (veins and lymphatics) as a 700-vessel network, with specific Siras

termed 'Stanya' (milky/chylous) that converge in the thoracic region.^[6] The term 'Ksheera-Vaha Sira' (milk-bearing vessel) in Sushruta's text almost certainly refers to the chyle-bearing thoracic duct and lacteals, reflecting direct observation of the lymphatic system's chylomicron content.^[2,71]

3.3 Physiological Functions of Cardiac Lymphatics

The cardiac lymphatic system performs four cardinal physiological functions: (i) maintenance of myocardial interstitial fluid balance, preventing oedema and compressive injury to cardiomyocytes;^[45,47] (ii) immune surveillance through transport of antigen-presenting cells and immune mediators from the myocardium to lymph nodes;^[83,84] (iii) clearance of macromolecules, metabolic waste products, and lipoproteins from the myocardial interstitium;^[51,52] and (iv) facilitating reverse cholesterol transport (RCT) — the efflux of excess cholesterol from peripheral tissues via lymph → thoracic duct → HDL recycling.^[52,53]

Drake et al. (1989) demonstrated experimentally that myocardial lymph flow increases linearly with cardiac output and inversely with lymphatic outflow resistance.^[47] When this flow is impeded — as in thoracic duct obstruction or elevated central venous pressure — interstitial fluid accumulates, producing myocardial oedema that mechanically impairs systolic contraction and diastolic relaxation.^[45,50] Mehlhorn et al. (2001) confirmed that post-cardiac surgery myocardial oedema, a major contributor to post-operative cardiac dysfunction, is primarily mediated by lymphatic insufficiency.^[45]

4. BIOCHEMICAL BASIS OF LYMPHATIC-CARDIAC INTERACTION

4.1 Lymphangiogenic Signalling: VEGF-C / VEGFR-3 Axis

Vascular Endothelial Growth Factor-C (VEGF-C) is the principal lymphangiogenic cytokine, acting through its receptor VEGFR-3 (Flt-4) on lymphatic endothelial cells (LECs) to drive lymphatic vessel formation, maturation, and maintenance.^[81,82] Under physiological conditions, VEGF-C is constitutively expressed in the myocardium, maintaining the integrity of subepicardial and subendocardial lymphatic networks.^[48,84] Following myocardial infarction, VEGF-C expression is transiently upregulated during the first 48-72 hours, but this endogenous lymphangiogenic response is markedly insufficient to prevent pathological lymphatic regression in the infarct zone.^[44,49,84]

Henri et al. (2016) demonstrated in a pivotal experimental study that selective stimulation of cardiac lymphangiogenesis using AAV9-VEGF-C administration following experimental MI in mice resulted in a 40% reduction in myocardial oedema, 35% reduction in inflammatory infiltration, and significant improvement in left ventricular ejection fraction at 4 weeks.^[49] LYVE-1 (Lymphatic Vessel Endothelial Hyaluronan Receptor-1) is a specific surface marker of lymphatic endothelial cells that mediates hyaluronan uptake and fluid clearance, making it a reliable biomarker for lymphatic endothelial integrity and function.^[83,94]

4.2 NT-proBNP and Cardiac Stress Biomarkers

N-terminal pro-brain natriuretic peptide (NT-proBNP) is released by cardiomyocytes under wall stress and volume overload — both of which are precipitated by lymphatic stasis-induced myocardial oedema.^[50,57] Paluszynska et al. (2022) demonstrated a significant positive correlation between serum LYVE-1 levels and NT-proBNP in patients with chronic heart failure, establishing a direct biochemical link between lymphatic endothelial dysfunction and cardiac stress.^[94] High-sensitivity C-reactive protein (hs-CRP), a sensitive marker of systemic inflammation, is consistently elevated in states of lymphatic stasis — reflecting the pro-inflammatory milieu generated by impaired immune cell clearance from cardiac tissue.^[55,92]

4.3 Lipid Biochemistry and Chylomicron-Mediated Atherogenesis

The thoracic duct transports post-prandial chylomicrons — large triglyceride-rich particles (50-1000 nm diameter, containing ApoB48) synthesised in intestinal enterocytes following dietary fat absorption.^[53,54] Under normal conditions, chylomicrons are efficiently cleared by lipoprotein lipase in peripheral tissues and by hepatic lipoprotein receptors. When the thoracic duct is congested, retrograde pressure impairs chylomicron transit, promoting their accumulation in the mediastinal and pericardial lymphatics.^[54] Von zur Muhlen et al. (2019) demonstrated in an experimental thoracic duct

ligation model that duct obstruction resulted in a 60% increase in plasma VLDL and chylomicron remnants, accelerated atherosclerotic plaque formation in the aortic root, and elevated sdLDL (small dense LDL) — the most atherogenic lipoprotein fraction.^[54]

Martel et al. (2013) provided compelling evidence that lymphatic vasculature is the primary route of macrophage reverse cholesterol transport — the mechanism by which foam cells within atherosclerotic plaques return excess cholesterol via lymph to the circulation for hepatic clearance.^[51] Lim et al. (2013) extended these findings, demonstrating that SR-BI-mediated HDL transport through lymphatics accounts for 30-40% of peripheral cholesterol efflux.^[52] Lymphatic insufficiency therefore creates a dual atherogenic burden: increased chylomicron delivery and impaired cholesterol efflux from plaques.^[51,52,53]

In Ayurvedic terms, this biochemical dysregulation corresponds precisely to Medo Dhatu Dushti — pathological vitiation of the lipid/adipose tissue fraction — as described in Charaka Samhita (Sutra Sthana, Chapter 28).^[8] Charaka states that Medo Dushti manifests as Atisthoulya (central obesity), Shosha (wasting), Ayasaj shwasa (exertional dyspnoea), and Alasya (fatigue) — a clinical constellation that maps to metabolic syndrome and cardiac cachexia.^[1,8] Bhavamishra in Bhavaprakasha Nighantu further elaborates that Medovaha Srotas (the channels of lipid transport, correlating with the lymphatic-chylomicron pathway) when obstructed, produce Hridayavyatha (cardiac pain) and Shwasa (dyspnoea).^[18]

5. PATHOPHYSIOLOGY OF CARDIAC DISEASE THROUGH LYMPHATIC DYSFUNCTION

5.1 Lymphatic Stasis and Myocardial Inflammation

Cardiac lymphatic vessels are not passive tube but active immunological participants. They express CCL21 and CXCL12 chemokines that actively recruit dendritic cells and T-lymphocytes from the myocardium.^[83,84] When lymphatic drainage is impaired, this immune cell clearance function fails, leading to accumulation of pro-inflammatory macrophages (M1 phenotype) in the myocardial interstitium.^[44,89] Kataru et al. (2020) demonstrated in a murine MI model that genetic deletion of cardiac lymphatic vessels led to a 3-fold increase in myocardial macrophage infiltration, 2.5-fold elevation of IL-6 and TNF-alpha, and significantly worsened cardiac remodelling at 4 weeks post-infarction.^[44]

5.2 Myocardial Fibrosis and Remodelling

Chronic lymphatic stasis generates sustained interstitial hypertension, activating cardiac fibroblasts through TGF-beta1 and CTGF signaling pathways to produce excessive extracellular matrix collagen deposition — the hallmark of myocardial fibrosis.^[88,89,90] Rienks et al. (2014) demonstrated that the myocardial ECM undergoes continuous remodelling under lymphatic pressure signals, and that disruption of these signals

triggers pathological fibrosis indistinguishable from that seen in pressure-overload cardiomyopathy.^[88] Sievers et al. (2019) provided direct evidence for a lympho-cardiac connection in myocardial fibrosis by demonstrating that VEGF-C overexpression reduced cardiac collagen deposition by 45% in a chronic lymphatic insufficiency model.^[90]

Prabhu and Frangogiannis (2016) systematically reviewed the biological basis of cardiac repair after MI, establishing that the transition from inflammatory to fibrotic phase is critically regulated by lymphatic clearance of inflammatory debris.^[89] Failure of this clearance — as in lymphatic congestion — prolongs the inflammatory phase and shifts myocardial healing toward fibrous scar rather than functional regeneration.^[84,89]

From an Ayurvedic pathogenetic perspective, this cascade corresponds to Samprapti of Kaphaja Hridroga as described in Charaka Samhita (Chikitsa Sthana, Chapter 26): Kapha (anabolic, fibrotic) and Vata (catabolic, arrhythmogenic) become simultaneously vitiated, leading to Sanga (obstruction) of Rasavaha Srotas, Margavarodha (channel blockade), and eventually Hridaya Daurbalya (cardiac weakness).^[1,72]

5.3 Pericardial Lymphatics and Pericardial Disease

The pericardial space is drained by a specialised network of lymphatic sinuses within the fibrous and serous pericardium, ultimately draining into right and left cardiophrenic lymph nodes and thence to the thoracic duct.^[43,46,59] Obstruction or insufficiency of pericardial lymphatics results in pericardial effusion — initially transudative but progressing to exudative with protein-rich, fibrin-laden fluid as lymphatic permeability increases.^[59] Faber et al. (2020) reviewed evidence from cardiac surgery patients and animal models demonstrating that iatrogenic thoracic duct injury during mediastinal dissection produces pericardial effusion in 12-18% of cases, strongly implicating lymphatic drainage in pericardial fluid homeostasis.^[59]

5.4 Atherosclerosis as a Lympho-Vascular Disease

The vascular wall possesses an intrinsic lymphatic network — vasa vasorum lymphatics — that drains the adventitia and outer media of large vessels including the aorta and coronary arteries.^[40,43] Atherosclerosis is associated with progressive lymphatic regression in the vascular wall — a process termed 'lymphatic dropout' — that impairs the clearance of lipid-laden macrophages (foam cells) from developing plaques.^[51,53] Siebert et al. (2017) provided epidemiological and mechanistic evidence that lymphatic vessel dysfunction is causally, not merely associatively, linked to coronary artery disease — establishing lymphatic health as a novel therapeutic target.^[40]

The Ayurvedic parallel to this pathomechanism is found in the concept of **Raktavrita Vata** — Vata dosha

obstructed by vitiated Rakta (blood with lipid overload), described in both Charaka Samhita and Ashtanga Hridayam as producing Hrid Shoola (coronary chest pain), Shwasa (dyspnoea), and Murcha (syncope).^[1,3,72] Dalhana, in his Nibandhasangraha commentary, describes Sira-Sanga (vascular obstruction) mediated by Medo Vikara (lipid disorders) as the fundamental mechanism of Hridroga in Sthula Prakriti (obese constitution) individuals.^[9]

6. PHARMACOLOGY OF TRIAL DRUG COMPONENTS: LYMPHO-HRIDYA YOGA

6.1 Comprehensive Phytochemical and Pharmacological Profile

The eight-herb formulation Lympho-Hridya Yoga is designed on Ayurvedic principles of Srotovishodhana (channel purification), Lekhana (lipolytic scraping), Hridya (cardioprotection), and Kapha-Vata Shamana (dosha pacification).^[1,19,73] Each component demonstrates molecular-level evidence for activity relevant to lympho-cardiac pathophysiology:

6.1.1 Punarnava (*Boerhavia diffusa* L. — Root)

Boerhavia diffusa contains the alkaloid punarnavine, rotenoids (boeravinones A-H), flavonoids (quercetin, kaempferol), sterols (beta-sitosterol), and the unique alkaloid lirioidendrin.^[11,36] Trivedi et al. (2019) demonstrated significant lymphagogue activity of Punarnava root extract in experimental models — increasing lymphatic vessel contractility by 28% and lymph flow rate by 35% at 200 mg/kg dose.^[25] *Boerhavia diffusa* exerts diuretic activity through inhibition of renal Na⁺/K⁺-ATPase and aquaporin-2 upregulation, reducing pre-load and providing cardiac decongestant effects.^[11,36] Kong et al. (2022) demonstrated anti-fibrotic effects of *Boerhavia diffusa* root extract in experimental cardiac fibrosis through TGF-beta1 pathway inhibition.^[58] The Ayurvedic classical indication Srotoshodhana (channel purification) and Shothahara (oedema reduction) aligns precisely with these mechanistic findings.^[73]

6.1.2 Arjuna (*Terminalia arjuna* — Bark)

Terminalia arjuna bark is exceptionally rich in triterpene glycosides (arjunic acid, arjunolic acid, arjungenin, arjunetin), flavonoids (luteolin, quercetin), tannins (arjunin, punicalcorin-D), calcium salts, and sitosterol.^[12,66] Bharani et al. (2002) conducted a landmark double-blind, placebo-controlled crossover study demonstrating that *Terminalia arjuna* bark extract (500 mg TDS) was equivalent to isosorbide mononitrate in reducing angina frequency and improving treadmill exercise tolerance in stable angina.^[62] Mechanistically, arjunic acid inhibits LDL oxidation, reduces platelet aggregation through thromboxane A2 pathway suppression, and enhances myocardial contractility through positive inotropic effects.^[33,34] Kapoor et al. (2014) demonstrated endothelial-protective effects of *Terminalia arjuna* via NO-synthase upregulation and ICAM-1 downregulation, directly addressing atherogenic endothelial dysfunction.^[34]

6.1.3 Guggulu (*Commiphora mukul* — Oleo-gum-resin)

Shuddha Guggulu contains guggulsterones Z and E as principal bioactive steroidal compounds, along with guggulipid (a fraction of guggulipid), ferulic acid, and myrrh-related terpenoids.^[14,23,65] The hypolipidaemic mechanism of guggulsterones was definitively elucidated by Urizar et al. (2002) in Science, establishing that guggulsterones act as antagonist ligands for the Farnesoid X Receptor (FXR) — the nuclear receptor that governs bile acid and lipid homeostasis thereby upregulating hepatic LDL-receptor expression and reducing serum LDL-C.^[14] Mehra et al. (2019) further demonstrated NF- κ B inhibitory activity of guggulsterones, conferring anti-inflammatory effects relevant to the cytokine storm of myocardial infarction and heart failure.^[32] Satyavati et al. (1969) published the seminal clinical evidence for Guggulu's hypocholesterolaemic effect, demonstrating 24% LDL reduction in a clinical series.^[65]

6.1.4 Pippali (*Piper longum* — Fruit)

Piper longum contains piperine, piperlongumine, methyl piperine, sesamin, sylvatin, and multiple piperidal alkaloids.^[13,28] Piperine is a potent bioavailability enhancer — inhibiting CYP3A4 and P-glycoprotein efflux transporters in the intestinal epithelium, thereby increasing oral bioavailability of co-administered phytochemicals by 30-154%.^[28,69] This property — termed Yogavahi (carrier and potentiator) in Ayurvedic texts — is critical in polyherbal formulations, ensuring systemic absorption of cardioactive compounds.^[1,8,69] Pingale et al. (2011) demonstrated significant anti-hyperlipidaemic activity of *Piper longum* extract with 22% reduction in TC and 18% reduction in LDL in a rat model of high-fat diet-induced hyperlipidaemia.^[69]

6.1.5 Devadaru (*Cedrus deodara* — Heartwood)

Cedrus deodara heartwood yields himachalol, deodarin (a biflavanone), alpha- and beta-himachalene sesquiterpenes, himachalol esters, and phenolic acids.^[16] Sharma and Julka (2014) characterised the anti-inflammatory activity of *Cedrus deodara* constituents through COX-1/2 inhibition — comparable in potency to diclofenac in carrageenan-induced paw oedema models.^[16] The Kapha-Vata Hara and Lekhana (fat-scraping) properties assigned in classical texts correspond to vasodilatory and anti-atherogenic activities demonstrated in vitro.^[16,73]

6.1.6 Haritaki (*Terminalia chebula* — Fruit Rind)

Table 2: Phytochemical constituents, mechanisms of action and lympho-cardiac targets of the eight components of Lympho-Hridaya Yoga.^[11,12,13,14,15,16,20,25,26,27,28,32,33,34,37,58,62,65,67,68,69,79]

Herb	Key Phytoconstituents	Primary Mechanism	Lympho-Cardiac Target	Ref
Punarnava	Punarnavine, Boeravinones A-H, Quercetin	Lymphagogue, Diuretic, Anti-fibrotic	Lymphatic contractility, TGF- β 1 inhibition	11,25,58
Arjuna	Arjunic acid, Arjunolic	Cardioprotective, Anti-	Endothelial protection,	12,33,34,62

Terminalia chebula is among the richest known plant sources of hydrolysable tannins — including chebulinic acid, chebulagic acid, corilagin, and gallic acid — alongside flavonoids, terpenoids, and anthraquinone glycosides.^[15,37] Mallick et al. (2020) demonstrated in a comprehensive in vitro and in vivo study that standardised Haritaki extract produced significant lipid-lowering effects (TC reduced by 31%, LDL by 28%) and strong anti-oxidant activity via DPPH radical scavenging (IC₅₀: 12.8 μ g/mL), with anti-inflammatory activity mediated through IL-6 and TNF-alpha suppression.³⁷ Bhakta et al. (1999) confirmed diuretic activity of *Terminalia chebula* — relevant to cardiac fluid offloading in congestive heart failure.^[27]

6.1.7 Varun (*Crataeva nurvala* — Bark)

Crataeva nurvala contains lupeol, betulinic acid, friedelin, phytol, beta-sitosterol, and glucosinolates including glucocapparin.^[26] Sini et al. (2011) reviewed the diuretic and lymphatic decongestant properties of Varun, noting its traditional use in Shotha (oedema) management and Srotoshodhana.^[26] Jain and Vyas (1975) observed improvement in lymph vessel contractility in patients treated with *Crataeva nurvala* bark decoction in a preliminary clinical series.^[67] Lupeol, a major constituent, has demonstrated NF- κ B pathway inhibition and anti-inflammatory effects in cardiac models.^[26,68]

6.1.8 Shilajatu (*Asphaltum punjabianum* — Exudate)

Shilajatu contains fulvic acids (40-85%), humic acids, dibenzo-alpha-pyrones, triterpenes, and more than 84 ionic minerals including iron, zinc, copper, and selenium.^[20,79] Chandola and Tripathi (2013) demonstrated in a pilot clinical study that Shilajatu supplementation over 90 days produced significant reductions in total cholesterol (18%), LDL (22%), and hs-CRP (31%), while improving mitochondrial ATP production markers in cardiac patients.^[79] Fulvic acid — the principal active constituent — acts as a potent mitochondrial electron transport chain facilitator, improving cellular energy production in hypoxic cardiac tissue.^[20] The classical Sarvarogahara (universal remedy) designation of Shilajatu in Ashtanga Hridayam reflects its broad metabolic and Rasayana (rejuvenating) properties.^[7,35]

Herb	Key Phytoconstituents	Primary Mechanism	Lympho-Cardiac Target	Ref
	acid, Luteolin	platelet, eNOS upregulation	Coronary vasodilation	
Guggulu	Guggulsterone Z&E, Guggulipid	FXR antagonism, NF- κ B inhibition	Hypolipidaemia, Anti-atherogenesis	14,32,65
Pippali	Piperine, Piperlongumine	CYP3A4 inhibition (bioenhancer), Anti-hyperlipidaemic	Bioavailability enhancement, Lipid clearance	13,28,69
Devadaru	Himachalol, Deodarin, Alpha-himachalene	COX-1/2 inhibition, Vasodilation	Anti-inflammatory, Anti-atherogenic	16
Haritaki	Chebulinic acid, Chebulagic acid, Gallic acid	Anti-oxidant, IL-6/TNF- α suppression	Lipid reduction, Lymphoprotection	15,27,37
Varun	Lupeol, Betulinic acid, Friedelin	NF- κ B inhibition, Lymph vessel tone	Lymphatic decongestant, Anti-oedema	26,67,68
Shilajatu	Fulvic acids (40-85%), Dibenzo- α -pyrones	Mitochondrial ETC facilitation, Anti-oxidant	Cardiac energy metabolism, Vasculoprotection	20,79

7. PHARMACOKINETICS AND KINETIC ACTION

7.1 Absorption and Bioavailability

The oral bioavailability of phytochemicals in polyherbal Ayurvedic formulations is governed by multiple factors: intestinal solubility, membrane permeability (passive diffusion and active transport), first-pass hepatic metabolism, and efflux pump interactions.^[77] Guggulsterones Z and E, the principal bioactive compounds of Guggulu, demonstrate poor aqueous solubility (log P = 3.8) but are substantially absorbed as micellar solubilisates in the chylomicron-rich intestinal lymph — meaning that thoracic duct patency directly influences their systemic delivery.^[14,23] Arjunic acid, the primary triterpene of Terminalia arjuna, reaches peak plasma concentration (T_{max}) at 2.5 hours following oral administration with absolute bioavailability of approximately 14%, which is significantly enhanced (to ~45%) by co-administration with piperine.^[22,28]

The Ayurvedic concept of Anupana (vehicle/adjuvant) parallels modern pharmacokinetic enhancement strategies. The classical specification of Ushna Jala (warm water) as Anupana for cardiac formulations is pharmacologically rational — warm water increases gastric motility and dissolution rates, while Arjuna Ksheerapaka (Terminalia arjuna bark decoction in milk) provides a lipid-rich vehicle that enhances absorption of fat-soluble phytoconstituents including guggulsterones and himachalol.^[1,19,73]

7.2 Distribution and Tissue Targeting

Guggulsterones demonstrate preferential distribution to hepatic tissue (liver-to-plasma ratio: 8:1) and to the myocardium (myocardium-to-plasma ratio: 3:1),

consistent with their primary therapeutic targets.^[23,29] Fulvic acid (from Shilajatu) demonstrates unique intracellular penetration through all biological membranes including the inner mitochondrial membrane, enabling direct delivery of minerals and electron transport chain mediators to cardiac mitochondria.^[20] Punarnavine from Boerhavia diffusa distributes preferentially to lymphatic endothelial cells and renal tubular epithelium — consistent with its dual lymphagogue and diuretic pharmacological profile.^[25,36]

7.3 Metabolism and Elimination

Guggulsterones undergo extensive phase-I (CYP3A4-mediated hydroxylation) and phase-II (glucuronidation and sulfation) hepatic biotransformation, with elimination primarily through bile (70%) and urine (30%), half-life approximately 4-6 hours.^[29] Chebulinic acid and chebulagic acid from Haritaki are hydrolysed by intestinal microbiota to gallic acid, which is subsequently absorbed and undergoes rapid methylation to syringic acid — a metabolite with potent anti-oxidant and vascular protective activity.^[21,37] The piperine-mediated inhibition of CYP3A4 and P-glycoprotein (MDR1) effectively extends the elimination half-lives of co-administered compounds by 30-60%, prolonging their therapeutic action — a kinetic synergy that validates the classical polyherbal formulation strategy.^[28]

8. PANCHAKARMA INTERVENTIONS IN LYMPHO-CARDIAC DISEASE: MECHANISTIC BASIS

8.1 Snehana (Oleation Therapy)

Internal oleation (Abhyantara Snehapana) with medicated oils including Dhanwantaram Taila initiates the therapeutic process by mobilising lipid-soluble toxic

metabolites from tissues into the gastrointestinal tract for elimination.^[75,78] Agarwal et al. (2010) reported preliminary clinical evidence that Snehapana in cardiac rehabilitation significantly reduced serum cholesterol and improved functional class.^[78] Mechanistically, internal lipid loading paradoxically stimulates hepatic LDL-receptor upregulation through transient feedback inhibition of HMG-CoA reductase — a mechanism that parallels the 'reverse loading' hypothesis of lipid-lowering therapy.^[75,78]

8.2 Virechana (Therapeutic Purgation) and Basti (Medicated Enema)

Virechana with Trivrit Leha (Operculina turpethum preparation) achieves Pitta-Kapha elimination through intestinal secretion enhancement, reducing the chylomicron burden in intestinal lymphatics by accelerating their clearance.^[1,75] Basti (medicated enema) with Dashmoola Ksheera Basti addresses Vata Anulomana — restoration of normal downward flow of Apana Vayu — which in cardiovascular terms corresponds to normalisation of autonomic tone, venous return, and lymphatic propulsion.^[75,76] Kumar et al. (2012) reviewed the cardiovascular indications of Basti Karma, noting significant improvements in NYHA class, heart rate variability (a marker of cardiac autonomic regulation), and oedema scores following Yoga Basti protocol in heart failure patients.^[75]

9. CLINICAL EVIDENCE AND RESEARCH GAPS

9.1 Clinical Trials of Individual Herb Components

Dwivedi and Jauhari (1997) reported the first randomised comparison of Terminalia arjuna with placebo in post-infarction patients, demonstrating significant improvements in LVEF, effort tolerance, and frequency of anginal episodes at 3 months.^[61] The subsequent RCTM trial by Bharani et al. (2002) established equivalence with isosorbide mononitrate — a standard anti-anginal agent.⁶² Yadav and Sarkar (2016) conducted

a systematic review of Ayurvedic management of Hridroga vis-à-vis coronary artery disease, identifying 12 clinical studies with consistent evidence for Terminalia arjuna, Guggulu, and Punarnava in reducing cardiac symptoms and improving biomarkers.^[76] Frishman (2021) reviewed current evidence for phytomedicines in cardiac conditions, including guggulsterones, quercetin, and piperine, concluding that evidence quality warrants Phase II-III controlled trials.^[80]

9.2 Research Gaps and Future Directions

Despite the extensive evidence base for individual components, no published clinical trial has specifically investigated an Ayurvedic polyherbal formulation targeting the lympho-cardiac axis with lymphatic biomarkers (VEGF-C, LYVE-1) as primary endpoints.^[40,84,94] The novice research protocol will address this critical gap — being the first RCT to systematically evaluate lymphatic biomarkers in an Ayurvedic cardiac trial with concurrent echocardiographic and inflammatory biomarker assessment. Patwardhan and Mashelkar (2009) advocated for traditional medicine-inspired drug discovery approaches, specifically recommending polyherbal multi-target strategies as superior to single-compound extraction for complex diseases like cardiovascular disease.^[77]

Future research directions include: (i) mechanistic studies of Ayurvedic lymphagogues on VEGF-C / VEGFR-3 signalling in human lymphatic endothelial cell cultures; (ii) metabolomic profiling of polyherbal formulations using LC-MS/MS to identify synergistic bioactive compounds; (iii) investigation of Prakriti-based pharmacogenomic variations in treatment response; and (iv) integration of Panchakarma sub-protocols into cardiac rehabilitation programs with lymphatic imaging endpoints.^[77, 81, 82]

10. INTEGRATED PATHOGENESIS: AYURVEDIC AND BIOMEDICAL CONVERGENCE

Table 3: Integrated pathogenesis matrix: Ayurvedic Samprapti (pathogenesis) of Hridroga and biomedical correlates in lympho-cardiac disease.^[1,2,3,4,6,43,44,50,57,71,72]

Ayurvedic Pathological Event	Classical Text Reference	Biomedical Equivalent	Measurable Biomarker
Rasavaha Srotorodha (channel obstruction)	CS Vimana 5.8; AH Nidana 12	Lymphatic vessel congestion / thoracic duct obstruction	VEGF-C ↓, LYVE-1 ↓, Lymph flow ↓
Medo Dhatu Dushti (lipid vitiation)	CS Sutra 28; BP Nighantu Haritakyadi Varga	Dyslipidaemia / chylomicron overload / sdLDL elevation	LDL ↑, TG ↑, AIP ↑, sdLDL ↑
Kapha Sanga in Hridaya (cardiac channel blockage)	CS Chikitsa 26; SS Uttara 43	Myocardial oedema / pericardial effusion / intimal lipid deposition	NT-proBNP ↑, Echo: pericardial effusion
Raktavrita Vata (blood-obstructed Vata)	AH Nidana 12; CS Chikitsa 26	Coronary atherosclerosis / vasospasm / endothelial	hs-CRP ↑, LDL-C ↑, Troponin I ↑, Echo LVEF ↓

Ayurvedic Pathological Event	Classical Text Reference	Biomedical Equivalent	Measurable Biomarker
		dysfunction	
Shotha (oedema)	AH Nidana 12; CS Vimana 5	Interstitial myocardial oedema / bilateral pedal oedema	NT-proBNP ↑, Clinical: pitting oedema, 6MWT ↓
Hridaya Daurbalya (cardiac weakness)	CS Chikitsa 26; SS Uttara 43	Systolic / diastolic heart failure	LVEF ↓, E/A ratio abnormal, NYHA class ↑
Srotovishodhana (channel purification — treatment)	CS Sutra 28; SS Chikitsa	Lymphagogue / anti-atherogenic therapy	VEGF-C ↑, LYVE-1 ↑, LDL ↓, NT-proBNP ↓

11. CONCLUSIONS

The present review establishes, through convergent lines of classical Ayurvedic textual scholarship and contemporary biomedical evidence, that thoracic duct dysfunction and cardiac lymphatic congestion constitute a pivotal pathomechanism in the genesis and progression of coronary artery disease, heart failure, atherosclerosis, and pericardial disease.

The Ayurvedic conceptual architecture — Rasavaha Srotorodha, Medo Dhatu Dushti, Kaphavrita Vata, and Hridroga Samprapti — achieves remarkable congruence with the modern lympho-cardiac pathophysiological model, validating the prescient observational acuity of classical Ayurvedic physicians.^[1,2,3,4,71,72] The eight-herb Lympho-Hridya Yoga formulation, combining Punarnava, Arjuna, Guggulu, Pippali, Devadaru, Haritaki, Varun, and Shilajatu, addresses the lympho-cardiac axis through complementary and synergistic mechanisms: lymphangiogenesis stimulation, lipid clearance, endothelial protection, anti-inflammation, and mitochondrial support.^[11,12,13,14,15,16,20,25,26,34,37,58,62,65,79]

The proposed RCT (Protocol DGM/RSHA/2025-26/001) represents a scientifically rigorous and clinically significant study that will, for the first time, test Ayurvedic herbal intervention against validated lymphatic biomarkers (VEGF-C, LYVE-1) alongside established cardiac endpoints.^{95,96,97} A positive outcome would not only validate a novel therapeutic strategy for cardiovascular disease but would also contribute landmark evidence to the integration of classical Ayurvedic medicine within the framework of evidence-based cardiology.

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