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SHONITA ABHISHYANDA AND DYSLIPIDEMIA: AN INTEGRATIVE AYURVEDIC REVIEW ON PATHOPHYSIOLOGY AND BASTI-BASED MANAGEMENT

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Abstract: The 21st century's technological boom has paradoxically fueled lifestyle disorders like dyslipidemia—a major cardiovascular risk factor characterized by abnormal blood lipid levels. Ayurveda, India's ancient health science, views health as complete well-being beyond mere absence of disease. Classical texts describe ***Shonita Abhishyanda***—excess *Kapha* and *Medodhatu* in *Rasa–Rakta*—as a *Santarpanajanya Vyadhi* matching dyslipidemia's clinical profile.

Arising from *Viruddhahara* and overnourishment, *Shonita Abhishyanda* causes *Srotorodha* and progresses to *Sthaulya, Hridshula*, and *Margavarana* complications, paralleling hyperlipidemia's atherosclerotic sequelae. This review correlates their etiology, pathophysiology, and clinical features, offering an integrative Ayurvedic framework for early detection and holistic lipid disorder management.

Index Terms- Dyslipidemia, *Shonita Abhishyanda*, Lipid, *Virudhaahara, Santarpanjanya vyadhi.*

I. INTRODUCTION

Dyslipidemia is a metabolic disorder involving abnormal lipoprotein metabolism, characterized by elevated total cholesterol, LDL-C, triglycerides, or reduced HDL-C levels in blood.[1] These lipids, absorbed from the intestine and transported via lipoproteins, are vital for energy production, hormone synthesis, and bile acid formation. However, their dysregulation constitutes a major risk factor for atherosclerotic cardiovascular disease. [2]

Classical Ayurvedic texts lack a single precise term for dyslipidemia, likely viewing it as a metabolic derangement rather than an independent disease. Various scholars have proposed equivalents such as *Shonita Abhishyanda, Rasagata Sneha Vriddhi, Rasa Raktagata Sneha Vriddhi, Medovriddhi, Medoroga, Medodosha, Prameha,* and *Atisthoulya*[3] to describe this condition.

Acharya Charaka emphasizes *Ahara* as the supreme determinant of bodily constitution—essential not only for survival but also for maintaining *Bala* (strength), *Varna* (complexion), and *Upachaya* (growth). Modern lifestyle patterns dominated by junk foods, processed/packaged meals, late-night eating, incompatible food combinations (*Viruddhahara*), and sedentary habits fueled by industrialization have triggered a global pandemic of lifestyle disorders. Ayurveda classifies these as ***Santarpanajanya Vyadhis***, primarily involving *Kapha Dosha* and *Medodhatu* vitiation through excessive nourishment. Ayurveda conceptualizes this as increased *Sneha Guna* (unctuousness) leading to *Raktadushti* or disrupted *Apa–Tejo Mahabhuta* balance within blood circulation.

Shonita Abhishyanda, described by *Acharya Charaka* as arising from *Santarpana Nidana* and *Viruddha Ahara*, manifests as pathological accumulation of *Kapha* and *Medas* within *Shonita Dhatu*. This leads to *Dhamani Pratichaya* (vascular thickening) and *Margavarana* (channel obstruction), closely paralleling **(Dys)hyperlipidemia**—where abnormal lipoprotein metabolism predisposes individuals to obesity, diabetes mellitus, hypertension, ischemic heart disease, and atherosclerosis.[4,5,6]
Management of *Shonita Abhishyanda* follows *Apatarpana* (depletion), *Virukshana* (drying), and *Chedana* (excisions) principles through integrated *Shodhana* (purification), *Shamana* (pacification), and *Rasayana* (rejuvenation) approaches. This analysis establishes their conceptual and clinical convergence, providing an integrative Ayurvedic framework for prevention and holistic management of contemporary lipid metabolic disorders.

II. OBJECTIVES

To collect and evaluate textual references supporting the understanding and management of dyslipidemia in Ayurveda.

III. MATERIALS AND METHODS

An effort was made to collect various references about integrative ayurvedic approach for understanding and management of Santarpanajanya vikara, Shonita Abhishyanda, Medoroga with special reference to dyslipidemia.

IV. ORGAN CORRELATION

Fat digestion begins minimally in the stomach through the action of gastric lipase. The main site of fat digestion lies in the small intestine, where the hormone cholecystokinin is released and stimulates the breakdown of dietary fats. The liver and pancreas contribute by secreting bile and pancreatic lipase respectively. These two secretions mix in the common bile duct and then enter the small intestine, facilitating efficient fat emulsification and digestion.

The absorption of fat occurs primarily through the absorptive epithelial cells lining the duodenum and jejunum, after which the processed lipids enter the liver and are further metabolized within the circulatory system[7,8] Thus, the key organs involved in the digestion, absorption, and metabolism of fat include the *Amashaya* (a seat of *Pitta*), the *Yakrit* (liver) and *Pleeha* (spleen) as roots of *Raktavaha Srotas*, and various Dhamanis (vessels) as roots or sites of *Rasavaha Srotas*.[9,10]

From an Ayurvedic perspective, this indicates that dyslipidemia is closely associated with dysfunction or imbalance of *Raktavaha and/or Rasavaha Srotas,* and therefore involves *Rakta and/or Rasa Dhatu* at the tissue level.

V. ETIOLOGICAL CORRELATION

Modern Medicine Perspective

From the modern viewpoint, dyslipidemia is classified into:

1. **Primary dyslipidemia**: Arising from genetic mutations that affect mechanisms of lipid synthesis, transport, or clearance.
2. **Secondary dyslipidemia**: Caused by lifestyle factors, metabolic disorders (e.g., diabetes, obesity), endocrine abnormalities (e.g., hypothyroidism), and certain medications, all of which are modifiable at least in part. Among adults, secondary causes contribute to a large proportion of dyslipidemia cases. A sedentary lifestyle combined with excessive intake of saturated fat, cholesterol, and trans fats is the prime preventable cause. Other contributing factors include diabetes mellitus, alcohol overuse, hypothyroidism, and cholestatic liver disease.The condition is further aggravated by high-calorie intake (over-consumption of carbohydrates and fats) and physical inactivity.[11]

Ayurvedic Perspective

The Ayurvedic understanding of *Shonita Abhishyanda* runs parallel to these modern mechanisms. The causative factors described in the classics include:

1. Excessive intake of fatty, sweet, oily, and heavy foods (*Guru–Snigdha–Madhura Ahara*), which can be linked to *Atisnigdha–Madhura–Abhishyandi–Pichchila* items, *Snigdha–Ushna* or *Vidahi* substances, and *Viruddhahara* (opposing food combinations).
2. Regular consumption of foods with *Viruddha* properties.
3. Sedentary lifestyle (*Avyayama*), including features like *Cheshta Dwesha*, *Sayya Sukha*, and *Asana Sukha*.[12]
4. Over-sleeping (*Ati Nidra*).
5. Genetic susceptibility (*Beeja Dosha*).
6. Psychological-stress-related factors (Atichintanam), including stress-induced overeating, Anxiety and excessive alcohol consumption.

In the context of *Shonita Abhishyanda*, the classics explicitly mention *Viruddhahara* and *Santarpanajanya Nidana* as key etiological factors, highlighting its multifactorial nature. Under *Viruddhahara,* combinations such as Matsya with *Payas, Sarshapataila-bhrista Ahara* with *Payas,* and *Chilichima Matsya with Payas* are implicated in the pathogenesis.[13,14,15]Among *Santarpanajanya Nidanas,* the condition is associated with *Madhura rasa* foods such as *Panasa*, *Masha*, *Godhuma* with *Navaneeta*, *Navaanna*, *Snigdha Ahara* (oily foods), *Ksheera vikaras*, and *Mahisha dugdha*. It is also linked to *Viruddhahara*-type items such as junk foods, milkshakes, ice creams, non-vegetarian foods rich in saturated fat and cholesterol, and overall *Santarpana-pradhan Ahara* patterns.[16]

Among *Viharaja Nidanas, Diwaswapna, Kalatiswapna, and Avyayama*—hallmarks of a modern sedentary life—are recognized as significant contributors to *Shonita abhishyanda*, as they promote *Agnimandya*, *Kapha–Meda vriddhi*, and obstruction of *Raktavaha Srotas.*[17]

Notably, a high intake of saturated fats, trans fats, and cholesterol, along with physical inactivity and chronic stress, can raise lipid levels even in individuals with normal BMI, reinforcing the idea that lifestyle-induced metabolic derangements are central in both modern dyslipidemia and the Ayurvedic entity *Shonita Abhishyanda*.

Both paradigms converge on the concept that lifestyle-related imbalance—through diet, inactivity, and psychological stress—is a major driver of lipid and lipoprotein abnormalities.

VI. PATHOPHYSIOLOGY

Modern Perspective

The pathophysiology of dyslipidemia involves several interrelated mechanisms:

1. **VLDL overproduction by the liver-** Excess free fatty acids released from adipose tissue serve as substrate for hepatic triglyceride synthesis. High intake of carbohydrates and alcohol stimulates hepatic lipogenesis and VLDL secretion. Insulin resistance further promotes VLDL overproduction by enhancing free fatty acid flux and hepatic triglyceride synthesis.
2. **Reduced LDL catabolism-** Genetic mutations, such as those in familial hypercholesterolemia, reduce LDL-receptor activity and retards LDL clearance from the circulation. Insulin resistance and receptor downregulation, often compounded by high saturated-fat intake, decrease the efficiency of LDL catabolism.
3. **Loss of HDL protective function-** Obesity, systemic inflammation, and hypertriglyceridemia impair reverse cholesterol transport, leading to dysfunctional HDL that loses its atheroprotective capacity.
4. **Endothelial injury and plaque formation-** Oxidized LDL injures the vascular endothelium, promoting monocyte recruitment, foam-cell formation, and progressive plaque development. This underlies the transition from dyslipidemia to atherosclerotic cardiovascular disease.
5. **Insulin resistance producing lipid abnormalities-** Insulin resistance increases adipose-tissue lipolysis, reduces triglyceride clearance, and enhances hepatic lipogenesis, creating a mixed pattern of elevated triglycerides, high LDL, and low HDL.
6. **Hormonal and metabolic secondary causes-** Hypothyroidism, nephrotic syndrome, and diabetes mellitus exacerbate lipid abnormalities by altering synthesis, transport, and clearance pathways, making them important secondary contributors.

Ayurvedic Perspective

The modern entity dyslipidemia can be correlated with *Shonitabhishyanda—Abhishyanda* (abnormal, slimy flow) in *Shonita* (blood).

The classical condition *Shonitabhishyanda* (also termed *Shonitabhishyanda*) is described by *Acharya Charaka* in the context of *Viruddhahara*, where regular consumption of *Poushkara, Rohinikashaka,* or pigeon meat roasted or fried in mustard oil along with *Madhu–Payas* (honey–milk) is said to cause this state.[15] *Shonita* refers to blood, and *Abhishyanda* denotes excessive, slimy, morbid oozing of vitiated *Rakta* through the *Raktavaha Srotas*.

The Ayurvedic pathophysiology of dyslipidemia is best understood through the framework of *Shonitabhishyanda*, with the following components:

1.Kapha Dosha Prakopa

1.1. Excessive *Kapha* increases *Guru* (heaviness) and *Snigdha* (unctuousness) guna which further increases meda, promoting abnormal fat deposition in *Rakta* and *Rasa* and predisposing to *Santarpanajanya* derangements.

1.2. Foods that smear or pollute the channels transporting the essence of food to body parts are termed *Abhishyandi*. In the *Mamsa Varga* classification, *Anupa Mamsa*—meats from aquatic, marshy, or domestic animals—is described as *Mahabhishyandi*, defined as foods that cause excessive moisture in the channels of *Dosha, Dhatu, and Mala*, and capable of forming a thread between two fingers (*Tantumat*)—clinical correlates of hyperviscous,

1.3. *Vagbhata* states that vitiated blood itself causes *Abhishyanda*, emphasizing that *Abhishyandi* is a *property* of food and activities that disturb flow in the channels. Various Ayurvedic scholars define *Abhishyandi* as any substance whose *Snigdha*, *Guru*, or *Klama-kara* (difficult-to-digest) quality obstructs the *Siras* carrying *Rasa* from the *Hridaya*, causing dullness, sluggishness, or difficulty in flow.

1.4. Blood vitiated by *Kapha Dosha* is described as *Ishat Pandu* (pallid), *Pichilam* (slimy), *Ghanam* (thick), atherogenic blood.

2.Asthayi Poshaka Dhatu

2.1.The *Poshaka Medo Dhatu* being *Asthayi* in nature circulates along with the *Rasa and Rakta* to nourish the *Sthayi Medo Dhatu*. The *Sthayi or Poshya Medo Dhatu* is derived from the *Poshaka Medo Dhatu* by the action of *Medodhatvagni* (Lipoprotein lipase).

2.2.The lipoproteins synthesized by the liver can be correlated with *Asthayi Medodhatu or the Poshaka Medo Dhatu*. They can also be referred to the "*Abaddha Medas*" which can be said as unbound or freely circulating fat.

3.*Mandagni* (Weak Digestive Fire)

3.1.Impaired *Jatharagni* and *Bhutagni* lead to incomplete transformation of *Anna Rasa* into *Rasadhatu*, resulting in *Aama* formation and abnormal *Medodhatu* production.

4.*Ama* Formation and *Srotorodha*

4.1.Sticky, heavy *Ama* obstructs *Srotas*, especially *Raktavaha* and *Rasavaha Srotas*, paralleling impaired lipid clearance and endothelial injury in modern physiology.

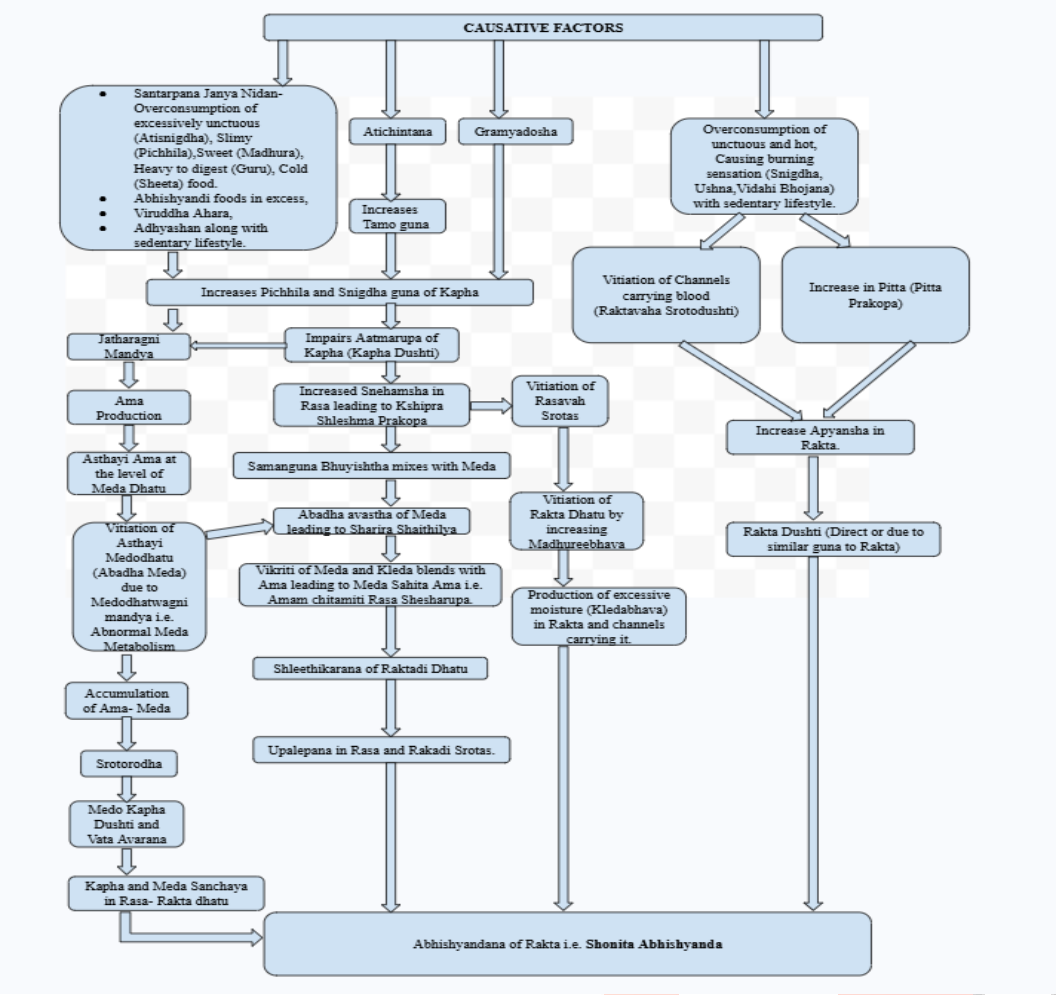
5.Vicious Cycle: *Meda-Vriddhi with Meda Dhatvagni Mandya*

5.1.Excess *Meda* impairs *Meda Dhatvagni*, reducing fat metabolism and creating a self-perpetuating cycle of further *Medodhatu* accumulation and *Srotorodha*.

5.2.When these dietary factors are combined with a sedentary lifestyle, they promote *Kapha–Meda* vitiation, *Aama* formation, *Raktavaha* obstruction, and the progressive development of *Shonitabhishyanda*, which clinically and pathophysiologically corresponds to dyslipidemia and its vascular sequelae.

Samprapti Ghataka* of Dyslipidemia in *Ayurveda

1.*Dosha: Kapha* and *Pitta, asthayi meda dhatu*
2.*Dushya: Rakta* and/or *Rasa*
3.*Srotas*: *Raktavaha* and/or *Rasavaha Srotas*
4.*Srotodushti: Atipravritti* (excessive flow) and *Sanga* (obstruction)
5.*Udbhava Sthana: Amashaya*
6.*Sancharasthana: Rasa–Raktavaha Srotas*
7.*Vyaktisthana: Dhamani* (vessels)
8.*Rogamarga: Abhyantara* (internal)
9.*Agni: Jatharagni* or *Bhutagni Mandya*



Modern Medicine	Ayurveda	Integrated Interpretation
Insulin resistance	*Mandagni*	Impaired metabolic capacity
High LDL, triglycerides	*Meda(Asthayi/ Poshaka) Vriddhi*	Excess/ abnormal fat accumulation in arteries or blood vessels
Oxidized LDL, inflammation	*Aama*	Toxic metabolic by-products
Atherosclerosis	*Srotorodha*	Channel obstruction
Low HDL	*Dhatu Kshaya*	Loss of protective mechanism

Fig.:Integrative Interpretation

VII. MANAGEMENT

Modern Treatment

Dyslipidemia is a key modifiable risk factor for cardiovascular disease. Its management aims to lower cardiovascular risk through lifestyle changes and, when needed, drug therapy.

1.Lifestyle Modifications

1.1.Reduce saturated and trans fats; increase soluble fiber and omega-3 fatty acids.
1.2.Exercise At least 150 minutes per week of moderate-intensity aerobic activity.
1.3.Maintain a healthy BMI.
1.4.Complete cessation of tobacco.
1.5.Limit Alcohol intake to recommended limits.

2.Pharmacological Therapy

2.1.**Statins** (e.g., atorvastatin, rosuvastatin, simvastatin): Inhibit HMG-CoA reductase, reduce cholesterol synthesis, up-regulate LDL receptors, and lower LDL-C; first-line for high LDL and secondary CAD prevention. Side effects include myopathy, elevated liver enzymes, and rare rhabdomyolysis.
2.2.**Ezetimibe**: Blocks intestinal cholesterol absorption; lowers LDL-C; often added to statins in high-risk patients.
2.3.**PCSK9 inhibitors** (evolocumab, alirocumab): Monoclonal antibodies that protect LDL receptors from degradation, causing marked LDL-C reduction; used in familial hypercholesterolemia or statin-intolerant high-risk individuals; injectable and costly.
2.4.**Niacin** (Vitamin B3): Suppresses hepatic VLDL production, lowers LDL and triglycerides, and raises HDL; use is now limited due to flushing, hyperglycemia, and hepatotoxicity.
2.5.**Omega-3 fatty acids** (e.g., icosapent ethyl): Reduce hepatic VLDL synthesis and lower triglycerides; used as adjunct therapy in severe hypertriglyceridemia.

Ayurvedic Approach

1.***Shodhana* (purificatory therapies)**: Since it's a *Santarpanottha, Shleshma–Medo pradhana* condition, treatment employs *Katu, Tikta, Kashaya, Tikshna, Ushna, and Ruksha* qualities—specifically *Ruksha-Teekshana Basti, Vamana, Virechana, and Udwartana* as these help reduce *Kapha* and reset *Meda Dhatu* metabolism.
2.***Shamana* (palliative therapies)**: *Medohara* herbs like *Guggulu, Triphala, Vacha, Haridra, and Musta,* along with *Agni*- and *Aama*-oriented formulations, support healthier fat metabolism.
3.***Ahara–Vihara* (diet and lifestyle)**: *Kapha*-reducing, low-fat diet rich in whole grains, barley, legumes, and leafy greens; regular physical activity, yoga, pranayama; and balanced sleep and stress regulation.

Action of Basti in Shonita Abhishyanda

Agnideepana, Amapachana, Kapha–Medo–Vatahara, and Srotoshodhana therapies are highly beneficial in managing this disease. Ayurveda aims at "*Swasthasya Swasthya Rakshanam and Aturasya Vikara Prashamanam*," which applies equally to *Santarpanajanya* disorders; treatment mainly includes *Samshodhana and Apatarpana Chikitsa*. Among *Panchakarma* procedures, *Basti* stands out as the most effective *Samshodhana* modality, acting as a rapid *Aptarpana* therapy that removes excess *Meda* when prepared with *Aptarpaka* formulations.

Basti is administered via the anal route; the *Basti Dravya* is absorbed through the superior hemorrhoidal veins in the rectal mucosa, reaches the duodenum, then enters systemic and portal circulation, helping normalize

liver metabolism, support bile-salt production, and promote fat emulsification. This process reduces abnormal fat deposition in the liver and blood, correcting lipid metabolism and supporting overall metabolic health.

Basti plays a central role in managing ***Shonita Abhishyanda***-type dyslipidemia by acting on multiple pathophysiological levels, from *Dosha and Dushya to Srotas* and systemic metabolism. It is not merely a bowel-cleansing enema; its therapeutic effect arises from **rectal absorption of medicated *Basti Dravya*** into the portal and systemic circulation, influencing liver function, lipid metabolism, and vascular health.

Mechanism of Basti in Shonita Abhishyanda

Basti—especially ***Niruha, Anuvasana, Rukshana, Teekshana and Lekhana*** types—**breaks *Samprapti* at the *Dosha–Dushya* level** by eliminating vitiated *Kapha–Pitta and Medovaha* through ***Grahana* (absorption)** and ***Shodhana* (cleansing)** actions, while Rooksha and Lekhana properties prevent ***Margavarana*** and lipid-related *Srotorodha*. It is particularly indicated in ***Rakta-related Srotorodha***, as it influences the ***Mutaravaha–Raktavaha* convergence** and peripheral vasculature via *Apana Vata* modulation.

Clinically, Basti achieves ***Samprapti Vighatana*** by:

1. ***Sanchaya–Prakopa Bhanga***: *Rooksha-Tikshna Basti Dravyas (e.g.,Vacha, Haridra, Musta, Ativisha,Triphala, Guduchi)* pacify *Kapha–Pitta* accumulation in the *Pakvashaya*, preventing their spread into *Rasa and Rakta.*
2. ***Dushya Shodhana****: Grahana and Lekhana* actions remove ***Pichhila Medas*** from ***Rakta***, normalizing blood texture and flow and reducing lipid-like deposits in vessels.
3. ***Srotorodha Nivritti:*** *Anupravana and Vata-shamana* effects clear *Sanga* in *Dhamanis*, restoring *Pravahana* and preventing *Atipravritti* and *Margavarana.*
4. ***Adhistana* Correction**: By targeting *Yakrit-Pleeha via Srotomula, Basti* supports healthier *Rakta* formation and prevents pathological localization in *Vyaktisthana.*

Basti's Mode of Action in Lipid and Vascular Pathways

Basti Dravya, absorbed via the superior hemorrhoidal veins and rectal mucosa, reaches the duodenum, portal circulation, and then systemic circulation. It:

1. Modulates hepatic lipid metabolism, supports bile-salt synthesis, and enhances fat emulsification, thereby reducing fatty accumulation in the liver and blood cells.
2. Exerts anti-inflammatory, hypolipidemic, and antioxidant effects on liver and blood vessels, influencing cholesterol synthesis and uptake and reducing lipid peroxides linked to *Raktavaha-srotodushti*.
3. Balances *Apana Vata* and improves micro-circulation, reducing *Sandrata* (thickness/viscosity) of plasma and improving *Rasa–Raktavaha-srotas* flow, especially in cases of sluggish blood, heaviness, and early vascular stiffness even without marked obesity.

In *Lekhana Basti,* particularly *Kshara*-based regimens, the *Lekhana* (scraping) action is more pronounced, targeting circulating lipids and vascular debris, with studies showing better-directed reductions in total cholesterol and LDL-C and improved *Raktavaha*-related symptoms. This makes *Kala Basti* especially suitable for cholesterol-LDL-dominant, vascular-stiffness-prone, or moderate *Shonita Abhishyanda,* including frail or *Vata*-prone patients, where gentler yet sustained lipid-modulating and vascular-cleaning effects are required.

VIII. DISCUSSION AND CONCLUSION

This article demonstrates that dyslipidemia and its Ayurvedic counterpart *Shonita Abhishyanda* represent convergent models of lipid-metabolic derangement arising from lifestyle-induced *Santarpana, Viruddhahara, and Agnimandya.* Both paradigms identify similar etiological roots—excessive intake of *Snigdha, Guru, and Abhishyandi* foods, sedentary habits, and stress-related behavioral patterns—leading to *Kapha-Meda-Pitta* imbalance, *Aama* formation, and *Raktavaha/Rasavaha Srotorodha.*

Pathophysiologically, modern dyslipidemia (VLDL overproduction, impaired LDL clearance, HDL dysfunction, and endothelial injury) finds a coherent Ayurvedic correlate in *Shonita Abhishyanda*, characterized by *Pichhila-Ghana Rakta, Medovriddhi, and Dhamani Pratichaya-Margavarana.* The concept of *Asthayi Poshaka Medo Dhatu / Abaddha Medas* as circulating lipoproteins provides a useful integrative bridge between hepatic lipid metabolism and the *Ayurvedic Srotos-Dhatu-Agni* framework.

Management aligns around two core principles: *Apatarpana–Samshodhana* and lifestyle-driven correction of root causes. In modern medicine, this translates into statins, fibrates, ezetimibe, PCSK9 inhibitors, and omega-3-based pharmacotherapy, supported by diet, exercise, weight control, and abstinence from tobacco and excess alcohol. In *Ayurveda*, the same intent is fulfilled through *Basti-centered Samshodhana, Virechana, Vamana, Udwartana, Medohara Shamana, and Agni-Aama-targeted formulations,* along with low-fat, *Kapha*-reducing *Ahara–Vihara.*

Within this integrative framework, *Basti* emerges as a pivotal modality in *Shonita Abhishyanda,* not only as an *Aptarpana therapy for Medovriddhi* but also as a systemic, *Srotoshodhaka,* and *Vata*-balancing intervention. Its rectal-absorption-based action on hepatic lipid metabolism, bile-salt-mediated fat emulsification, and *Lekhana*-driven removal of *Pichhila-Medas-Rakta* helps normalize blood viscosity, prevent early atherogenic changes, and reduce cardiovascular risk. By harmonizing classical *Ayurvedic* concepts with contemporary lipid physiology, this synthesis supports a holistic, evidence-informed approach to prevention and management of dyslipidemia—promoting both ***Swasthasya Swasthya Rakshanam and Aturasya Vikara Prashamanam*** in the era of *Santarpanajanya* lifestyle disorders.

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