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Review Article

Atherosclerotic Vascular

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The role of Rasaushadhies in the management of Srotosanga w.s.r to Atherosclerotic Vascular disease

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The rapid pace of modern living, coupled with poor lifestyle choices and environmental factors, has contributed significantly to the growing prevalence of Srotorodha (atherosclerotic vascular disease). In Ayurveda, maintaining a balance of Dosha, Dhatu, Mala and Agni is key to a healthy life. This balance is achieved through the continuous transport and circulation of these elements via the Srotas, the body's internal transport system. These channels, which differ in size, are linked to various parts of the body and causes diseases when blocked. Atherosclerosis is a disease that is characterized by the accumulation of lipids, fibrous elements, and calcification within the large arteries. This process is initiated by endothelium activation, followed by a cascade of events, which implies the vessel narrowing and activation of inflammatory pathways leading to atheroma plaque formation. Atherosclerosis, the leading cause of cardiovascular diseases, primarily affects the heart and brain, resulting in ischemic heart disease (IHD) and ischemic stroke, which are the first and fifth leading causes of death worldwide, respectively. Srotosanga mentioned under Rasavaha Sroto Vikara is analogous to atherosclerotic vascular disease in modern medicine. In Ayurvedic practice the Rasaushadhies has been considered as more effective and beneficial due to lesser therapeutic doses, enhancement of action of other ingredients of formulation, more shelf life, quicker action and palatability as compared to herbal preparation. Rasaushadas which possess Kapha Medho hara, Lekhana Karma, Hridya, Ushna, Teekshna, Sukshma, Sara etc properties are useful in the management of atherosclerosis.

Keywords: Srotosanga, Atherosclerosis, Rasaushadhies

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Introduction

Atherosclerosis significantly contributes to various cardiovascular diseases, including coronary artery and peripheral arterial disease. It is marked by artery thickening due to plaques composed of fatty acids, cholesterol, calcium, fibrin, and cellular debris. These plaques cause arterial stenosis, potentially leading to complete blood flow blockage and resulting in hypoxia in vital organs. As plaques grow, they may rupture, causing blood clots that further block veins or arteries, a condition known as thrombosis.[1] Ischaemic heart disease and stroke, both primarily caused by atherosclerosis, are leading causes of death worldwide. Ischaemic heart disease alone accounts for 13% of global deaths, Stroke responsible for approximately 10% of total deaths, became the third leading cause of death.**[2]** Primary therapeutic interventions for atherosclerosis includes lifestyle modifications, such as regular exercise, a healthy diet, smoking cessation, and management of hypertension, diabetes, and high cholesterol levels.**[3]** Lifestyle changes can prevent early-stage atherosclerosis and restore health in some patients, but once the disease advances to intimal thickening, pharmacological interventions are essential for management.

According to *Charaka*, *Srotas* are defined as the transporting passage of dhatus undergoing transformations. Srotas may be considered as the channels (micro or macro) on the basis of morphology. These channels functions as the medium through which the biological materials, nutrients and waste products. It refers both to the gross major channels like respiratory tract, gastrointestinal tract, genito-urinary tract etc. Micro channels like vessels, capillary, lymphatics, etc and also to the molecular channels like the permeability of membranous pores of cell membrane etc.**[4]** In this article we have correlated *Srotas* as Arteries (blood vessels). *Srotodushti* (vitiation of system) can be seen by four signs i.e., *Atipravrutti* (increase of the contents of the system), *Sanga* (non-flow of the contents of the system), *Siragranthi* (Reduction of lumen of the system) and *Vimargagamana* (diverted movement of the contents of the channels. Among these *Sanga*/*Rodha* and *Siragranthi* can be included under obstruction of system. *Sanga* is a cause of functional obstruction of system and *Siragranthi* is a cause of structural as well as functional obstruction of system.

In an atherosclerotic vessel, initial stages show no obstruction, but endothelial damage triggers a cascade of reactions leading to vessel blockage. *Sanga* represents a functional obstruction, correlating with the progression of atherosclerosis, while *Siragranthi* involves both structural and functional obstructions, akin to cysts, tumors, and aneurysms. Thus, Sanga aligns with the functional aspects of atherosclerosis, but *Siragranthi* encompasses more extensive structural changes.

Aims and Objectives

1. To briefly explore the concept of *Srotosanga*, atherosclerosis.
2. To investigate the potentials of *Rasaushadhies* in addressing atherosclerosis.
3. To explore the pharmacological properties and potential mechanisms of action of *Rasaushadhies*.

Materials and Methods

For this study, *Ayurveda* classical text like *Charaka Samhita* etc has been used to evaluate the concepts. Various related websites, journals have been searched.

Conceptual Study

The importance of Srotas in disease manifestation: When *Srotas* are healthy, the formation of *Dosa*, *Dhatu*, and mala is balanced. However, if *Srotas* are vitiated it leads to imbalanced state leading to disease in the body. *Srotas* act as a transportation system, *Dhatus* transported through these *Srotas* are constantly subjected to metabolism. Without *Srotas*, no body part can grow, develop, or degenerate. *Srotovaigunya* plays a vital role in the *Samurchhana* of *Dosha* and *Dusyas* at a particular site, resulting in disease manifestation within the body.**[5]**

Srotovaigunya is a condition of *Srotas* which is susceptible for pathological changes or favourable movement to produce a disease. *Dalhana* comments that *Prakupita Doshas* will be moving in the body, produces disease after lodging in *Srotovaigunya* and *Dosha-Dushya Sammurchana* takes place. *Srotovaigunya* stands for some inherent weakness or lack of natural immunity to some particular type of disease or susceptibility to disease formation process and loss of resistance to disease producing factor.

Srotovaigunya does not necessarily cause disease on its own without *Dosha-Dushya Sammurchana* (the interaction of imbalanced doshas with bodily tissues). However, for the manifestation of disease, *Srotovaigunya* is essential and cannot be ignored.

In atherosclerosis, despite a cascade of reactions involving lipid metabolism, immune responses (such as macrophages and platelets), and other factors, no symptoms are present in the early stages which can be compared to *Srotovaigunya Avastha*. Symptoms appear only when the vessel blockage becomes significant (*Dosha Dusya Samurchana* occur). *Sroto Dushti* means impairment or vitiation of the body's channels. Foods (Ahara) and lifestyles (*Vihara*) that aggravate the *Doshas* and have opposite properties to the *Dhatus* can cause *Sroto Dushti*.**[6]** *Sroto Sanga*, one of the four types of *Sroto Dushti*, refers to blockages in these channels.

Acharya Charak identified thirteen major *Srotas* essential for the body's normal function, while *Acharya Susruta* described eleven pairs of *Srotas*. *Charaka* mentioned *Srotorodha* as a *Lakshana* of *Rasavaha Sroto Dushti*, indicating an obstruction in the channels carrying rasa (nutrients). *Charaka* mentioned the *Mulasthana* (root) of *Rasavaha Srota* as *Hridaya* and *Dasha Dhamani*. *Sushruta* on the other hand mentioned *Hridaya* and *Rasavaha Dhamani* as *Mulasthana*.

Rasavahanini Dhamani means channels carrying nutritive juices to every part of body from heart, so here the word *Rasavahini Dhamani* can be compared with arteries. Combining the opinions of both master *Charaka* and *Sushruta*, the roots of *Rasa* transporting channels can be said to be the heart and the blood vessels i.e. aorta and its branches taking their origin from the heart. Master *Charaka's* opinion of 10 arteries probably indicate the bigger branches of the aorta which further divide and re-divide into many branches and supply nutrition to every part of the body.

Pathogenesis

Atherosclerosis is not caused by a single etiologic factor but is a multifactorial process whose exact pathogenesis is still not known. Since the times of Virchow, a number of theories have been proposed. Currently, pathogenesis of atherosclerosis is explained on the basis of the following two theories: 1. Reaction-to-injury hypothesis 2. Monoclonal theory.

However, following is the generally accepted role of key components involved in atherogenesis.

I) Endothelial injury: It is the initial event in atherosclerosis development. Endothelial dysfunction, rather than complete denudation, triggers this process. In humans, hypertension and chronic dyslipidemia are major risk factors. Hemodynamic stress, particularly at blood vessel bifurcations where shear stress is highest, further supports the formation of atheromatous plaques**.**

Ii) Intimal smooth muscle cell proliferation: Endothelial injury leads to the adherence and aggregation of platelets and infiltration by inflammatory cells at the site of exposed connective tissue. This triggers the proliferation of smooth muscle cells and the production of extracellular matrix, stimulated by cytokines like IL-1 and TNF-α from monocytes and activated platelets. These cytokines promote the synthesis of growth factors such as PDGF and FGF, which encourage smooth muscle cells to move from the media to the intima. TGF-β and IFN-γ from activated T lymphocytes regulate collagen synthesis by smooth muscle cells. The proliferation is also facilitated by nitric oxide and endothelin from endothelial cells, with matrix proteins like collagen, elastic fibres, and proteoglycans being synthesized.

Iii) Role of blood monocytes: When LDL (low-density lipoprotein) enters the inner layer of an artery (intima), it undergoes oxidation. This oxidized LDL attracts and activates monocytes, causing them to proliferate and immobilize. The oxidized LDL is then taken up by scavenger receptors on monocytes, transforming them into lipid-laden foam cells. Additionally, oxidized LDL is toxic to endothelial cells. When foam cells die through apoptosis, they release lipids, which accumulate and form the lipid core of atherosclerotic plaque. This process contributes significantly to the development and progression of atherosclerosis.

Iv) Role of dyslipidemia: chronic dyslipidaemia in itself may initiate endothelial injury and dysfunction by causing increased permeability. High LDL promotes foam cell formation, contributing to atherosclerosis, while high HDL has a protective effect.

V) Thrombosis: Endothelial injury exposes subendothelial tissue,

Leading to the formation of small platelet aggregates and smooth muscle cell proliferation. This causes a mild inflammatory reaction, which, along with foam cells, gets incorporated into the atheromatous plaque. The plaque enlarges by attaching fibrin and blood cells, making thrombus a part of the atheromatous plaque.

Treatment

The basic aim of treatment is to make the movement of *Vayu* in *Srotas* normal so that all the physiological activities shall come to normalcy, स्रोतःसु च विशुद्धेषु चरत्यविहतोऽनिलः (च. चि.१७/७६).

Basic *Gunas* which form baseline principles to remove *Srotorodha*

***Laghuguna*:** It removes *Srotorodha* created by *Guruguna*. Clinical application: *Langhana* is *Laghutvakaraka* hence advised in *Ajirna* due to consumption of *Gurudravyas*.

***Tikshnaguna*:** Helps in *Śodhana* by deeply penetrating and piercing *Sandhibandha*. It effectively removes *Srotorodha* caused by combinations of *Guru*, *Ruksha*, *Snigdhagunas*. Clinical application: *Tikshnataila Pana* is advised where *Mamsa* and *Medodhatu Srotas* has been obstructed by *Snigdha* and *Guru Kapha*.

***Ushnaguna*:** Removes *Srotorodha* due to *Stambhana* by *Sitaguna*. Clinical application: in *Samavata* pain occur due to *Stambana* of *Vayu* due to *Rodha* by *Ama*.

***Vishadaguna*:** Helps in removal of *Srotorodha* caused by *Picchila Guna*. Clinical application: *Guggulu* is *Vishada* in nature which can be used to remove the *Pichilabhava* in *Srotas*.

Basic *Karmas* which form baseline principles to remove *Srotorodha*.

Anulomana, Pramathi, Sramsana, Bhedana, Rechana, Sodhana, Chedana, Lekhana, Grahi, Sukshma.

Basic treatment procedures which for removing *Srotorodha*.

Snehaprayoga, Svedaprayoga, mechanical removal of *Srotorodha – Sukshmaguna Vridhi Srotorodha* (dilatation), *Atyanta Guru Kapharodha-Apaharanam*.

Some basic drugs which remove *Srotorodha*: *Dahimandam, Annamandam, Takram*, Warm water, *Madyam, Erandataila, Suska Kustumbari, Yava, Haritaki* etc

Management with *Rasaushadas*

Deficiency of micro minerals in the human body can lead to various diseases, and *Rasaushadhas,* which are enriched with these essential minerals, not only act as powerful *Rasayanas* by rejuvenating and promoting overall well-being, but also possess therapeutic properties that address and cure specific ailments, making them more than just supplements.

Each *Rasa Dravya* has a specific composition which contain one or more minerals, metals etc. Following description helps us to know how these *Rasa Dravyas* play a role in *Srotosanga*

***Abharaka* (Mica)[7-10]**

Lipid peroxidation involves the oxidative degradation of lipids, leading to the formation of reactive oxygen species (ROS), which contribute to atherosclerosis by damaging endothelial cells and promoting atherosclerotic plaque formation. Glutathione, a crucial antioxidant, neutralizes ROS and reduces oxidative stress, thus playing a protective role against lipid peroxidation and atherosclerosis. *Abhrak Bhasma*, has been shown to modulate oxidative stress parameters, influencing the activity of antioxidant enzymes like superoxide dismutase and catalase, as well as glutathione levels, providing potential protection against oxidative damage and the development of atherosclerosis.

Vascular oxidative stress contributes to endothelial dysfunction, arterial stiffness, and hypertension. This is caused by an imbalance between reactive oxygen species (ROS) production and the body's antioxidant defence. Excessive ROS levels damage macromolecules like proteins, DNA, RNA, and cell membranes. Increased oxidative stress reduces nitric oxide (NO) bioavailability, impairing endothelium-dependent vasodilation and resulting in higher vascular tone and arterial stiffness, leading to hypertension. Reducing oxidative stress may improve arterial function and prevent hypertension complications in the elderly.**[11]**

Abhraka bhasma has an immunomodulatory effect, *Abhrak bhasma* may be able to modulate the production of nitric oxide, a key signalling molecule involved in the immune response.**[12]**

***Swarna Makshika* (Copper Pyrite)[13-15]**

LOX (Lysyl oxidase) activity is crucial for the stability of atherosclerotic plaques, particularly through the crosslinking of collagen fibers that form the fibrous cap over the lipid core.

Low LOX activity can lead to weak collagen crosslinking, weakening the fibrous cap, and making it more prone to rupture. This instability can result in acute complications like myocardial infarction due to thrombus formation on a vulnerable plaque. Essentially, LOX downregulation can compromise plaque stability, making it susceptible to degradation and clinical complication.

LOX is a copper-dependent enzyme essential for the crosslinking of collagen and elastin, maintaining the structural integrity of blood vessels, and playing a crucial role in the progression of atherosclerosis, from endothelial dysfunction to plaque formation and vascular stiffness. *Swarna Makshika*, a *Rasa Dravya* rich in copper, can potentially enhance LOX activity due to its copper content, thereby supporting vascular stability and repair.

This synergy between LOX and the therapeutic properties of *Swarna Makshika*, particularly its ability to improve cardiovascular health and mitigate the progression of atherosclerosis, underscores the significant role of *Swarna Makshika* in maintaining healthy arterial walls and preventing atherosclerotic disease.

***Vimala* (Iron Pyrite) [16]**

Iron supplementation has demonstrated significant antilipidemic and antiatherosclerotic potential. Studies show that iron reduces body weight gain and hepatic lipid accumulation in mice on a high fat diet, indicating its role in combating obesity, related lipid issues. Furthermore, iron enhances mitochondrial function by upregulating genes involved in mitochondrial respiration and betaoxidation, crucial for efficient energy metabolism in liver and skeletal muscle. It also increases the expression of genes related to heme and iron sulphur cluster synthesis, essential for mitochondrial energy processes. Importantly, iron supplementation lowers plasma total cholesterol and glucose levels, improving lipid metabolism and reducing hepatic steatosis. These actions collectively underscore iron's potential in managing lipid levels and preventing atherosclerosis.

***Shilajit* (Black Bitumen)**

Shilajit is composed of 60–80% humic substances, such as humic and fulvicacids, it is rich in nutrients, such as mineral salts, amino acids, and other organic components.

Fulvic acid and Homocysteine[17]

Homocysteine is thought to be an independent risk factor for atherosclerosis in human beings. Multifactorial mechanisms such as oxidative stress and inflammation have been found to play a role in hyperhomocysteinemia - induced atherogenesis. In addition, previous study suggested that elevated levels of homocysteine may not directly induce atherogenesis, but may instead accelerate atherosclerotic lesion development in combination with other cardiovascular risk factors. The production of pro-inflammatory mediators such as COX-2 in monocytes plays an important role in atherogenesis. However, the mechanism by which homocysteine regulates COX-2 gene expression of monocytes remains unclear. Fulvic acid is one of the most interesting Phyto-complex molecules and is reported to have several nutraceutical properties with potential anti-oxidant and anti-inflammatory activities. A study demonstrates for the first time that Fulvicacid can exert inhibitory effects on homocysteine-induced COX-2 expression in monocytes, thereby possibly serving anti-inflammatory and Athero-protective functions. This inhibitory effect was mediated by the ERK/JNK and NF-κB signaling pathways based on several lines of evidence.**[17]**

Humic Acid - Platelet Aggregation[18]

Lan HT, Zheng YT *et al.* findings demonstrate the therapeutic potential of native humic acids on Venous thromboembolism. when GPIIb/IIIa is activated, then it can bind to fibrinogen and connect to adjacent platelets through fibrinogen, causing platelets to aggregate and form early thrombosis. HA inhibits platelet activation by targeting GPIIb/IIIa, reducing thrombus formation. This effect is achieved by inhibiting the EMP-PDI and GPIIb/IIIa signaling pathways, and decreasing plasma P-selectin levels.

***Chapala* (Selenium)**

Selenium (Se) is an essential trace element that is essential for various metabolic processes, protection from oxidative stress and proper functioning of the cardiovascular system.

Se deficiency has long been associated with multiple cardiovascular diseases, including endemic Keshan's disease, Heart failure, coronary heart disease, myocardial infarction and atherosclerosis.**[19]**

Evidence from animal studies suggests that selenium and selenoproteins, such as glutathione peroxidases, thioredoxin reductase 1, selenoprotein P, and selenoprotein S, might prevent experimental atherosclerosis. This can be attributed to selenium's molecular and cellular effects, including inhibiting oxidative stress, modulating inflammation, suppressing endothelial dysfunction, and protecting vascular cells against apoptosis and calcification. However, the benefit of selenium supplementation in atherosclerosis prevention is not well-documented. Future studies should consider factors like baseline selenium status, dosage, forms of supplementation, and selenoprotein genotype to confirm selenium's role and underlying mechanisms in preventing atherosclerosis.**[20]**

Se is an essential trace element in the body. The Se-containing protein glutathione peroxidase exhibits central roles in regulating the physiological antioxidant status and plays additional roles in the thyroid metabolism and regulation of the immune response.**[21]**

Rasaka **(Calamine) - Zinc[22]**

Zinc plays a crucial role in the synthesis of nitric oxide (NO) in endothelial cells (ECs). NO, produced via endothelial NO synthase (eNOS), is a key vasodilator that significantly influences EC function. It provides several anti-atherosclerotic benefits, including vasodilation, inhibition of vascular smooth muscle cell (VSMC) proliferation, and prevention of leukocyte and platelet adhesion and aggregation. Reduced NO availability, due to lower eNOS expression or activity, is a significant factor in the development of atherosclerosis. Zhuang et al. further found that zinc supplementation effectively enhances intracellular NO production by elevating the expression and enzymatic activity of eNOS.

Secondly, atherosclerosis is considered a chronic inflammatory disorder, while zinc exhibits anti-inflammatory effects in ECs. Activated ECs could trigger chemokine secretion and recruit the circulating monocytes to the vascular wall, known as monocyte-EC interaction, which is a key event in the formation of atherosclerotic lesions.

ZEB1, a key member of the zinc finger-homeodomain family, inhibits monocyte-endothelial cell interactions, thereby reducing atherosclerosis. Zinc plays a vital role in preventing atherosclerosis by reducing VCAM-1 and ICAM-1 levels, which trigger monocyte attachment to endothelial cells. Zinc deficiency exacerbates chronic inflammation by increasing the expression of inflammatory cytokines and activating NF-κB, a factor involved in all stages of atherosclerosis development. Zinc supplementation, on the other hand, suppresses NF-κB activity. Additionally, zinc influences the expression of KLF family transcription factors and PPARs, both of which have anti-inflammatory properties.

Zinc plays a crucial role in immune cell function. It influences macrophages, acts as an intracellular messenger, and affects signalling pathways. Zinc deficiency can increase oxidative stress, promote pro-inflammatory cytokines, and disrupt the balance of T and B lymphocytes. While direct interactions with immune cells in atherogenesis aren't fully confirmed, zinc supplementation helps reduce inflammation and supports immune function, indicating its potential role in mitigating atherosclerosis. Zinc supplementation can reverse abnormal lipid metabolism and lower cholesterol and triglyceride levels. However, the effects of zinc on lipid-conjugated molecules remain mixed, with studies showing both inverse and direct associations with cholesterol levels. Zinc also affects transcription factors like ZEB1 and KLF2, regulating cholesterol metabolism and reducing lipid accumulation in macrophages, underscoring its atheroprotective role.

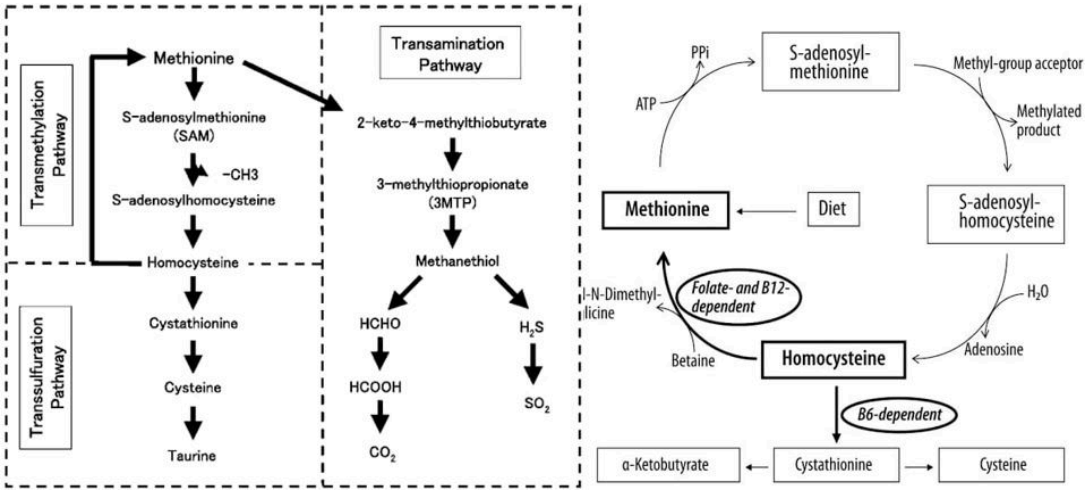
Wang Y et al.**[23]** had developed a 3D arterial wall model using GelMA hydrogel, incorporating HUVECs and SMCs, to study atherosclerosis (AS) progression and zinc ion evaluation. This model mimics the arterial intima-media and shows key AS features when treated with ox-LDL, TNF-α, and IL-1β. Zinc ions showed a biphasic effect, with optimal cell promotion at 20.0 μm and better evaluation accuracy (IC50 116.2 μm) compared to 2D models. Zinc inhibited DNA synthesis in hyperlipidemic and inflammatory conditions by blocking cell cycle transition. The 3D model is a promising tool for studying cardiovascular diseases and evaluating zinc ions and drugs, supporting zinc-based biomaterials in AS therapy.

Zinc plays a critical role in combating oxidative stress, a major risk factor for cardiovascular diseases (CVDs). It stabilizes proteins, promotes antioxidant synthesis, competes with redox-active metals to prevent lipid oxidation, and acts as a cofactor for superoxide dismutase, protecting against oxidative damage. Additionally, zinc supports nitric oxide availability, crucial for vasodilation, and participates in redox signaling, making it essential for mitigating CVD progression. **[24]**

***Gandaka* (Sulphur)**

Methionine is an essential amino acid that is converted into homocysteine through transmethylation.

Elevated levels of homocysteine, known as hyperhomocysteinemia, are associated with an increased risk of atherosclerosis due to oxidative stress and endothelial dysfunction. Cysteine, produced from homocysteine via the transsulfuration pathway (which requires vitamin B6), plays a crucial role in detoxification and antioxidant defence. Vitamin B12 is essential for the remethylation of homocysteine back to methionine. A deficiency in vitamin B12 can lead to elevated homocysteine levels, exacerbating oxidative stress and endothelial damage, both of which are key factors in the development of atherosclerosis. Thus, the balance and proper metabolism of methionine, homocysteine, cysteine, and vitamin B12 are critical in preventing atherosclerosis and maintaining cardiovascular health.



*Images showing the relation between sulphur, Vitamin B12, B6 in transsulfuration, remethylation pathways.

Angiogenesis is a regulated process of microvascular growth that can re-vascularize ischaemic tissue. H_2S induces angiogenesis by increasing endothelial cell proliferation and migration. Exogenous H_2S (NaHS) increases cell growth, migration and the formation of tube-like structures in cultured endothelial cells. H_2S and its synthesizing enzymes, including cystathionine γ-lyase, can protect against atherosclerosis H_2S regulates various pathophysiological functions via interaction with nitric oxide, activation of molecular signalling cascades, post-translational modification of proteins and control of redox-dependent responses.**[25]**

Seneff S *et al.***[26]** argue that the accumulation of cholesterol in the artery wall is a reflection of impaired cholesterol transport due to low bioavailability of Sulfate to the vasculature.

This paper hypothesizes that impaired cholesterol sulfate supply to the heart is key in atherosclerosis. Sulfates are vital for vascular health, and disruptions in enzyme activity by environmental toxins affect sulfate synthesis, leading to cholesterol buildup in arteries. Inflammation and platelet activation are crucial for regenerating sulfate at injury sites, enhancing vascular flow. Lifestyle changes, such as consuming sulfur-rich foods and avoiding toxins, are suggested to improve vascular health.**[26]**

***Gairika* / *Loha* / Iron**

Targeted therapies focusing on iron metabolism have proven effective in treating atherosclerosis and other cardiovascular diseases.**[27]** Iron is a necessary element for life; however, excess iron leads to oxidative stress by the Fenton reaction.

Iron deficiency is prevalent in patients with heart failure, while iron overload is associated in the pathogenesis of atherosclerosis. These findings suggest the "iron paradox" in cardiovascular diseases. Iron binds to transferrin in the circulation. In conditions where iron exceeds the carrying capacity of transferrin, non-transferrin bound iron circulates and promotes organ damage such as vascular endothelial cell and smooth muscle cell dysfunction. These also support the need to consider cautiously the extent and duration of iron repletion in patients with cardiovascular diseases. **[28]**

***Spatika* (Alum)**

Wigren M et al. findings demonstrate that treatment of*Apoe* mice with Alum results in an increase of regulatory T cells and suggest that these are activated by tolerogenic antigen-presenting cells presenting oxidized LDL antigens.

These findings provide improved mechanistic understanding of the atheroprotective properties of aluminum hydroxide adjuvants but also point to the importance of determining if hypercholesterolemia may compromise the efficacy of Alum-containing vaccines used clinically today.**[29]**

***Haratala / Manashila / Gouripashana* - Arsenic compounds**

Arsenic does not have anti-atherosclerosis properties. In fact, chronic exposure to arsenic is associated with an increased risk of atherosclerosis and other cardiovascular diseases. Arsenic exposure can lead to oxidative stress, endothelial dysfunction, and inflammation, all of which contribute to the development and progression of atherosclerosis.

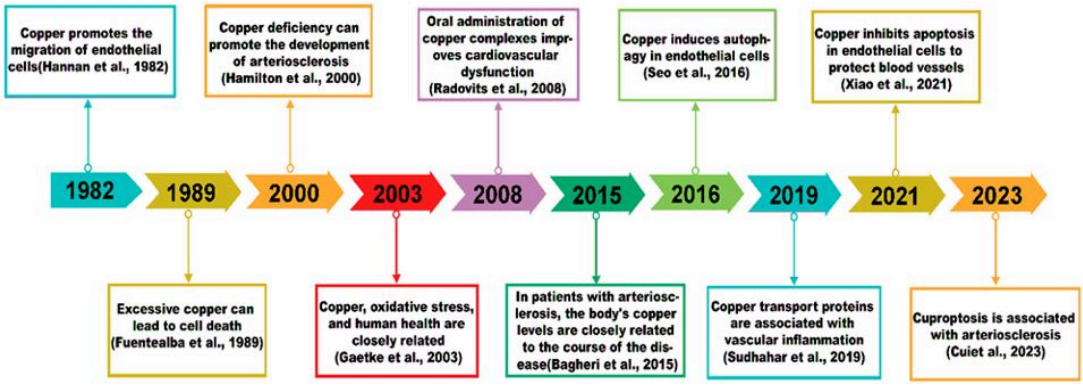
***Swarna* (Gold)**

Nanno-intervention with the delivery of silica–gold NPs has been proven to reduce total atheroma volume more in patients who have CAD with target lesions than in those who accepted stent implantation.**[30]**

***Rajata* (Silver)**

The elevated levels of serum lipids such as total cholesterol, triglycerides, LDL and VLDL were reduced after the treatment of silver (Ag) nanoparticles whereas the level of HDL increased. *Periyasamy Karuppannan* et al., concluded that the biosynthesized silver (Ag) nanoparticles possess antihyperlipidemic activity against STZ induced hyperlipidemic condition in diabetic rats which might be useful for the treatment of cardiovascular diseases after advanced clinical evaluation.**[31]**

***Tamra* (Copper)**



*The timeline outlines significant historical milestones in the research advancements regarding copper's association with Atherosclerosis.**[32]**

Disturbances in copper homeostasis can harm the cardiovascular system by promoting oxidative stress and endothelial dysfunction, leading to atherosclerosis. Excess copper increases ROS production, activates NF-κB, and upregulates adhesion molecules, facilitating inflammation. Elevated copper levels activate XIAP, further driving inflammation and affecting apoptosis,

Which impacts plaque stability and disease progression. Meanwhile copper impacts the KEAP1-NRF2 complex by allowing NRF2 to activate antioxidant responses under oxidative stress, mitigating cellular damage. It also serves as a cofactor for HIF-1α, essential for stability and activity regulation in hypoxic conditions, influencing genes related to angiogenesis, vasoconstriction,

And antioxidative stress. Copper deficiency disrupts these processes, highlighting its importance in maintaining cardiovascular health.**[32]** Copper (Cu) is an essential mineral nutrient that participates in cellular metabolism and function as a component of a number of cuproenzymes, an integrated structural element, and a regulatory agent.**[33-35]** However, Cu also catalyses the production of highly reactive oxygen species (ROS), which have the potential to cause oxidative damage to lipids, proteins, DNA and other molecules.**[36-38]** Therefore, either Cu deficiency or excess can lead to diseases or affect the progression of diseases including atherosclerosis. Understanding the complexity of the role of Cu in vascular homeostasis is helpful in designing targeted therapies for reversal of atherosclerosis.**[39]**

Targeted Cu Delivery: A novel ultrasound-assisted Cu-albumin microbubble (Cu-MB-US) delivery method has been developed to target Cu-deficient atherosclerotic lesions, showing promising results in reducing plaque size without increasing plasma Cu levels.**[40]** ***Vanga*** **(Tin)**

Vanga comes under the category of *Putiloha* whose *Bhasma* is mainly indicated in *Prameha*. Besides this it has been also used for various disorders including *Meha, Medoroga* and *Krimi*.**[41]**

Dr. Lalitha M. Vatar *et al.* demonstrated *Vanga Bhasma* at high dose and medium dose owing to its *Rasadi Gunas* and *Lekhana*, *Medhopaha Karma* helps in *Samprapti Vighatana* (breaking the Pathogenesis) of *Medoroga* which is due to impaired *Kapha* and *Vata Dosha* and helps in reducing the biochemical parameters and regulation of metabolism, as no physiological or adverse behavioural changes were noticed in the experimental animals, so it is considered as a safer drug for Hyperlipidaemic condition.**[42]**

Naga **(Lead (Pb))**

Lead (Pb), known as *Naga*, is one of the oldest metals used for medicinal purposes in human beings to treat many disease entities such as diabetes, obesity, joint disorders, eye diseases, skin diseases, anaemia, sexual disorders, diseases of old age group, etc. *Naga Bhasma* increases *Kanti, Virya, Ayu*, alleviates diseases, *Trishna, Ama, Sotha, Shula, Arsha, Kustha, Pandu, Meha*, etc. It is *Guru, Caksusya*, *Medo Hara*, and *Vata Hara*.**[43]**

Kamsya **(Bronze)**

Kamsya is another important *Misra Loha*, an alloy of Copper and Tin known since the period of *Samhita Kala*. Therapeutic dose of *'Kamsya Bhasma'* range from 60 mg to 120 mg. Appropriately prepared *'Kāmsya Bhasma'* possesses *Kaşāya* and *Tikta Rasa*; *Usṇa Vīrya; Lekhana Guṇa*. It does *Netra Prasādana*. It also a posesses *Rūkṣya, Sara* and *Viśada Guṇa*. Its judicious use cures *Udara Krimi* (intestinal worms), *Kustha Roga* (skin diseases). It mitigates vitiated *Kapha, Pitta* and *Vāta Dosa* and is agoodappetizer.**[44]**

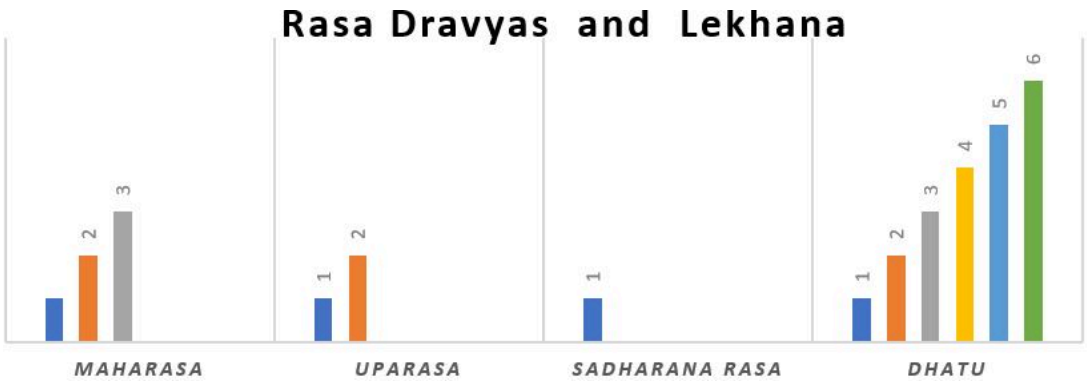
Pittala **(Brass)**

Pittala is an important *Misra Loha*, an alloy of Copper and Zinc. The therapeutic dose of *'Pittala Bhasma'* range from 60 mg to 120 mg. *Acharyas* have mentioned *Pittala* to be *Tridoshahara*. It has tremendous potential for combating a broad spectrum of ailments. Some therapeutic use of *Pittala* mentioned in Classical texts is *Shodhana, Panduroghara, Krimighna, Na Atilekhana, Pliharoga Nashaka, Raktapittanut, Lekhana, Bala-Virya-Ayuvardhana, Jantughna, Kushthagna, Basti Vishodhana*. These therapeutic purposes illustrate that *Pittala* was utilized and is currently used as a remedy for an extensive spectrum of maladies.**[45]**

Kshara **and** ***Medoroga***

Dyslipidemia which contributes to the development of atherosclerosis is considered under the umbrella term *Santarpanajanya Vikara*. Conditions like *Medoroga, Sthoulya, Shonita Abhishyanda* are caused due to *Santarpana Nidana*. Among *Shamana* formulations *Kashaya, Guggulu, Lauha Kalpanas* are used widely in the management of dyslipidemia. As Lauha has affinity towards blood, *Lauha Kalpanas* may have upper hand in managing dyslipidemia on comparing with other formulations.**[46]**

Generally, *Kshara* has properties like *Chedana* (excision), *Bhedana* (incision), *Lekhana* (scraping), *Krimighna* (anti-helmenthic), *Shodhana* (cleaning), *Ropana* (healing), *Pachana* (digestive).**[47]** These properties help in reducing atherosclerosis. *Chedana*: The action by which dosha adhered to channels are removed from its root is known as *Chedana*. In dyslipidemia, there is an increase in *Meda* in *Rasa* and *Raktavaha Srotas*. *Chedana Dravya* like *Kshara* (alkalies), *Maricha* and *Shilajith* are helpful in removal of excessive lipid from blood. **[46]**



Graph showing the *Lekhana* property of *Rasa dravya

Lekhana: The action which dries up and scrapes the abnormally deposited tissues or waste products in the body is known as *Lekhana*. *Kshoudra* (honey), *Ushnajala* (hot water), *Vacha* and *Yava* are best among *Lekhana Dravya*.**[47]**

***Sikata Varga* (Silicon)**

Silicon has been shown to have anti-atherogenic effects by influencing lipid metabolism. In studies on rabbits, those on an atherogenic diet with added silicon showed significantly fewer atherosclerotic plaques compared to those without silicon. Silicon supplementation lowered the plasma concentration of mono and polyunsaturated fatty acids, reducing the formation of toxic peroxides that damage arteries. In rats, silicon increased HDL-cholesterol and HDL-phospholipids while decreasing triglycerides and LDL-cholesterol. These findings suggest that silicon may help mitigate atherosclerosis by improving lipid profiles and reducing harmful lipid oxidation.**[48]**

Discussion

Pharmacology has transformed over the centuries, evolving from rudimentary herbal remedies to sophisticated biopharmaceuticals. This evolution highlights humanity's quest to understand and manipulate natural substances for healing. *Rasashastra* is a sophisticated system that involves the use of metals, minerals, plant, and animal derivatives to create medicines easily assimilated by the body. These essential elements must be processed and purified for safe and effective absorption. Micronutrients, including vitamins and minerals, are required by the body in tiny amounts but are crucial for health. Deficiencies in these micronutrients can lead to severe and even life-threatening conditions.

To break the pathogenesis of *Srotosanga*, various *Rasaushadies* has diverse functions as seen in the following table

Action	Rasaushadhies
Endothelial protection	Tamra
Anti-inflammatory Properties	Abraka, Tamra.
Anti atherosclerotic	Tamra, Loha
Antioxidants	Abhraka, Yasada Swarna
Lipid profile modulation	Tamra
Improving blood circulation	Loha Vanga, Tamra, Abhraka
Detoxification	Rasa Sindura, Tamra
Rejuvenation and tissue repair	Swarna, Yasada.
Vasodilation	Abhraka

Some of the compound preparations of *Rasa Aushadhies* useful in atherosclerosis are

Kharaliya Rasa	**Hridayarnavaras, Trinetraras, Vadavanalaras, Vadavagniras, Medodwamsiras, Trimurtiras, Rasamanikyaras, Shilajitvadiloha, Trivikramaras, Yogendra Ras etc.**
Kupipakwa Rasa	Rasa Sindura, Tamra Sindura, Malla Sindura etc.
Pottali Rasa	Tamragarbha Pottali, Rajatagarbha Pottali, Hemagarba Pottali, Mallagarbha Pottali, Loha Garbha Pottali etc.
Bhasmas	Abhraka, Swarnamakshika, Yasada, Loha, Tamra, Vimala, Kaseesa, Heeraka, Vaikranta, Rajata Bhasmas etc.

Conclusion

Rasaushadhies comprises diverse bioactive constituents such as fulvic and humic acids in *Shilajit*, copper in *Tamra*, etc. and various microminerals in other formulations possess therapeutic potential to modulate the pathogenesis of *Srotosanga* (atherosclerosis) due to their anti-inflammatory, antioxidant, endothelial protection, cardioprotective properties etc.

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