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Review Article

Ayurveda Section

Role of *Virechana Karma* as a Cardioprotective Modality: A Narrative Review

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ABSTRACT

Cardiovascular disorders lead to many deaths worldwide due to strokes and heart attacks, with dyslipidaemia being the main risk factor. This condition is similar to *Medo Rog* (disorder of fat metabolism) in Ayurveda. Other recognised metabolic risk factors for CVDs include diabetes, Hypertension (HTN), obesity, and Non-alcoholic Fatty Liver Disease (NAFLD). The present review aimed to assess the effectiveness of *Virechana Karma* (therapeutic purgation) as a cardioprotective method by examining its impact on key metabolic risk factors. *Virechana* Karma demonstrates significant potential to regulate lipid imbalances, serve as a primary therapy for eliminating vitiated *Pitta* and *Kapha Doshas*, and improve Agni. This holistic biopurification approach could be an effective alternative or supplementary therapy for managing the main pathophysiological elements of CVDs, acting as a cardioprotective strategy.

Keywords: Heart disease risk factors, *Medo Roga*, Metabolic syndrome, *Panchakarma*

INTRODUCTION

"Cardio-protection" refers to a wide range of treatments or techniques used to reduce the incidence of cardiovascular manifestations [1], among which Atherosclerotic Cardiovascular Diseases (ASCVD) are key concerns [2]. Even after significant development in the medical field, the number of ASCVD is increasing, in the influence of sedentary lifestyle, and ageing demographic, and escalating prevalence of contributory factors including dyslipidaemia, diabetes, HTN, obesity, and sedentary behaviour [3].

Dyslipidaemia is an altered lipoprotein metabolism condition characterised by elevated cholesterol levels or elevated triglyceride levels, Low Density Lipoprotein (LDL) cholesterol, and decreased levels of HDL, occurring singly or in combination [4]. This abnormal lipid profile promotes atherosclerosis, a leading cause of ASCVD. LDL cholesterol, in particular, is primary cause of damage to the intima of the endothelium and is a key player in both the formation and progression of atherosclerotic plaques, which initiate a cascade of inflammatory responses, oxidative stress, and foam cell formation. Given that LDL is the prime source of damage to the intima of the endothelium, it is highly deserving of attention in the context of atheroma-induced cardiovascular events and sequelae [5].

Epidemiological evidence has suggested the relation between NAFLD and subclinical atherosclerosis, including increased carotid intimal thickness that results in clinical manifestations of CVD [6,7]. The development of ASCVD is largely influenced by obesity, a significant modifiable risk factor. Furthermore, individuals suffering from obesity have more chances to develop concomitant conditions such as metabolic syndrome, diabetes, and HTN, all of which raise the risk of atheroma-induced cardiovascular events [8]. HTN, in particular, accelerates endothelial dysfunction and promotes vascular remodelling by exerting undue pressure on the inner lining of the vessel wall, which may increase the permeability of the vessel wall for lipoproteins [9].

Treatment of dyslipidaemia has gained prominence as a strategy for preventing atherosclerotic plaque formation. As far as contemporary science is concerned, standard protocol for dyslipidaemia includes statins, fibrates, and others. These lipid-lowering medications are usually given on a long-term basis and occasionally show adverse drug reactions [10]. A meta-analysis suggested that a reduction in cholesterol levels, specifically LDL in the blood, reduces the possibilities of atheroma formation and further progression of ASCVD. Incidents of heart attack, of revascularisation, and of ischaemic strokes reduce significantly with the reduction in the levels of LDL cholesterol. Every 1 mmol/L drop in blood cholesterol lowers the risk of these major vascular events by 20% [11].

In Ayurveda classical texts, *Santarpanajanya Vyadhi* refers to disorders caused by overnutrition and excessive accumulation of *Kapha*, *Pitta*, and *Meda*, leading to metabolic and systemic imbalances. The imbalance of *Kapha* leads to an overnourished state, resulting in the deposition of excess *meda* (fat tissue), contributing to obesity, insulin resistance, and many more. Most of the mentioned contributory factors for CVD can be put under the umbrella of *Santarpanajanya Vyadhi*. Excessive accumulation of *meda* is a hallmark of Santarpanajanya Vyadhi [12].

The causes of *medovaha sroto dushti* (disruption in fat metabolism channels) include *Avyayama* (insufficient exercise), *Diwaswapna* (sleeping during the day), *Atibhakshana* of *Medovardhaka Ahara* (consuming too much saturated fat), drinking *Varuni* (a fermented beverage made from date and palm trees), and consuming too much *Madhura* (sweet), *Snigdha* (unctuous), or other foods that vitiate *Kapha Dosha* [13]. In essence, *Medovaha Srotodushti* explains the same risk profile that contemporary medicine sees as contributing to metabolic and cardiovascular disorders, which includes a sedentary lifestyle, a bad diet, and excessive sleep during the day. Likewise, excessive daytime napping (exceeding 30 minutes) has been associated with a 32 % increased risk of NAFLD [14]. The causes of *Medovaha strotodushti* align well with current risk factors for CVD, implying an integrative and comprehensive approach to prevention and treatment.

Most of the primary causes of *medovaha sroto dushti* have similarity with those of *santarpan-janya vyadhi*, and in treating *santarpan-janya vyadhi*, the *Virechana* procedure is recommended. Fundamentally, Ayurveda states that metabolism is controlled by balanced *Doshas* and *Agni* (digestive fire) [15]. This suggests that metabolic dysfunction may arise due to an imbalance between *Agni* and *Pitta*, which might further cause hindrance in fat metabolism. Impaired lipid metabolism leads to dyslipidaemia, HTN, insulin resistance, NAFLD, and obesity, all of which are known contributory factors for ASCVD [16].

Virechana karma, with its potential of a holistic approach to influence the major risk factors of atherosclerosis, can act as a cardioprotective modality: it is a *Samshodhan chikitsa* (purification

therapy) used in *Bahudoshavastha* (multiple *dosha* imbalance) for eliminating vitiated *doshas* [17]. It may contribute to improve liver function and related metabolic pathways by normalising *Agni*. Therefore, the present review aimed to evaluate the efficacy of *Virechana Karma* as a cardioprotective modality, focusing on its effects on key cardiovascular risk factors- dyslipidaemia, insulin resistance, HTN, obesity, and NAFLD by consolidating evidence from contemporary clinical and preclinical studies.

REVIEW OF LITERATURE

Relevant studies published between January 2000 and March 2025 were identified by searching electronic databases (PubMed/MEDLINE, Scopus, Google Scholar) and Ayurveda platforms along with a thorough analysis of classical literature, contemporary literature, and the Ayurvedic Samhitas. This review bridges the gap between modern evidence and traditional practice by integrating both classical Ayurvedic wisdom and contemporary scientific literature, including clinical trials, experimental models, and mechanistic studies, to ensure thorough coverage.

Key factors in Cardiovascular Diseases

Dyslipidaemia: Evidence suggests that heart disease is not limited to middle-aged and elder adults in the present period. A study reported a high prevalence of lipid abnormalities in young ST-segment Elevation Myocardial Infarction (STEMI) patients (<45 years), with increased triglyceride levels being the most common, suggesting a key role of triglyceride in early-onset myocardial infarction [18]. Research advocates that raised triglyceride levels are a serious contributor to coronary artery disease on their own [19]. Along with that, LDL is accused of causing damage to the endothelium of the intima, making it more permeable and enabling more LDL to enter the intima, promoting platelet adhesion, which can lead to plaque rupture and an acute cardiovascular event [20]. This can further manifest as MI, limb ischaemia, stroke, angina pectoralis, and other severe complications [21]. In addition to that, research also demonstrates that reduced HDL cholesterol is a unique marker for atherosclerosis-induced CVD [22].

Insulin resistance and DM: Excess of fatty acid derivatives interferes with the liver's insulin signalling mechanisms. This impairs glucose homeostasis, adding to insulin resistance [23]. Increased atherogenic lipoproteins and decreased HDL are linked to insulin resistance, which encourages fat buildup in the arterial walls, lipid accumulation in the cardiomyocytes, and myocardial fibrosis, which leads to diastolic dysfunction [24].

Hypertension (HTN): Hypertensive patients having dyslipidaemia had an 18.1-fold increased risk of developing coronary artery disease compared to individuals without the condition. A case-control study suggests that adults with dyslipidaemia only had a 2.5-fold increased risk of developing coronary heart disease without the existing condition of HTN [25]. With HTN, the intima of the endothelium undergoes repeated and chronic injuries due to sheer stress, initiating a chain of inflammatory responses and plaque development via LDL penetration [9].

Obesity: Obesity also increases the prevalence of non-traditional risk factors. The hallmark of obesity is the overabundance of adipose tissue, particularly in the form of visceral fat, which serves as a source of chemokines (such as MCP-1) and pro-inflammatory cytokines (such as IL-6, TNF-α). These elements cause long-term systemic inflammation, which in turn encourages oxidative stress, vascular inflammation, and endothelial dysfunction all of which are critical stages in atherogenesis [26].

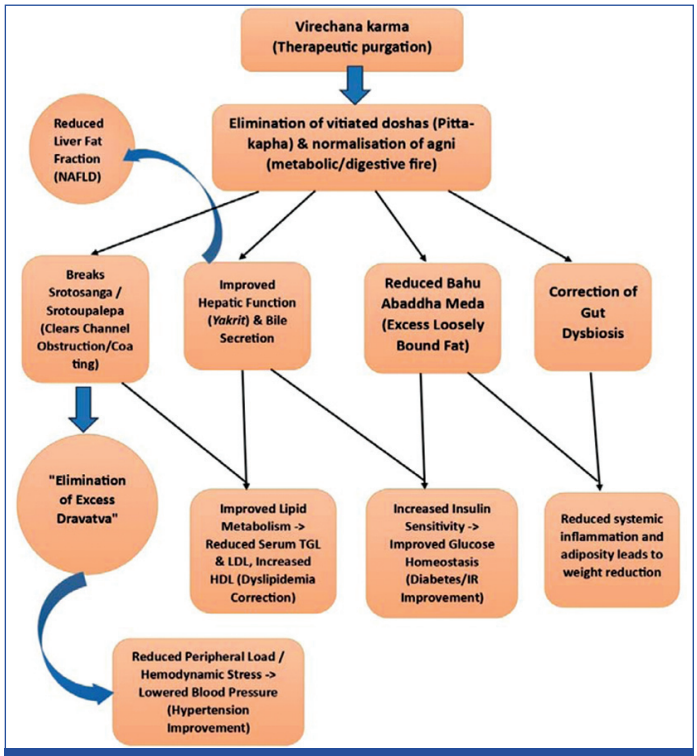
Non-alcoholic fatty liver diseases: The NAFLD has been connected to increased thickness of the intima and media of carotid arteries, a well-accepted indicator of subclinical atherosclerosis. Several epidemiological studies showed that cardiovascular events, both fatal and non-fatal, are more commonly occurring in patients with NAFLD [27-29]. It contributes to atherosclerosis via accumulated lipotoxic metabolites, a prothrombotic state, and its fundamental link to dysregulated lipid metabolism and insulin resistance [24].

Virechana Therapy and Its Probable Mode of Action as a Cardioprotective Agent

According to Ayurveda, *Bahudrava Shleshma* (excessively liquified kapha) and *Bahu Abaddha Meda* (excess accumulation of loosely bound fat) are the primary pathological factors for *Prameha Roga*, comparable to excess fatty acid derivatives in the context of insulin resistance [30]. *Medo Dushti* Lakshana is the prime factor in the *Samprapti* (pathogenesis) of *Prameha Roga*. The *Purvaroop* (prodromal symptoms) of *Prameha*, such as *Hrid, Netra, Jihva*, and *Shravan Upadeha*, indicate the deposition of undesired substances in the *Hridaya Pradesha* (cardiac region). This concept might have similarities with the pathogenesis of insulin resistance-induced atherogenic activities, which could eventually result in the emergence of heart-related disorders [31].

Virechna therapy works by disrupting the pathophysiology through its macro and microcirculatory purification ability. Since *Virechana* primarily acts at the site of Pitta, it exerts its effect on the liver and pancreas, either by promoting insulin secretion or by reducing insulin resistance [32].

It aids in the removal of *Doshas* from the entire body, not just from the *Aamashaya* (stomach) and *Pakwashaya* (large intestine). Additionally, *Virechana* helps in reducing and intensifying conditions like *Agnimandhya* (impaired digestive fire), and breaks *Srotosanga* (channel obstruction) and *Srotoupalepa* (coating of physiological channel) [Table/Fig-1].



[Table/Fig-1]: Mechanism of action of *Virechana*.

Through the *Ushna* (hot) and *Tikshna* (sharp) properties of *Virechana Dravyas*. These infiltrative qualities may reduce the peripheral load and show improvement in HTN [33]. *Virechana* may also eliminate excess *Dravatva* from the body and help in reestablishing the functional integrity between the three *Doshas*, ultimately leading to the calming of vitiated *doshas*. Regular, seasonal administration of *Virechana therapy* may act as adjuvant therapy and reduce haemodynamic stress on the endothelium [34]. It helps in ceasing the growth of aerobic bacteria following the procedure. In follow-ups, it was evident that the dysbiosis of gut flora had been corrected,

thereby initiating the body's weight reduction process and showing improvement in the symptoms of obesity [35].

NAFLD results from changes in the physiology of the liver (*Yakrit*), which is predominantly governed by *Pitta Dosha* as per the concept of *Ashraya-Ashrayee Bhava* in Ayurveda. Through the *Virechana procedure*, the vitiated *Pitta* and *Kapha Doshas*, aggravated in NAFLD, can be eliminated from the system. This leads to the *Prakritisthapan* (restoration of normal state) of *Pitta* and *Kapha*, which in turn regulates metabolic function at the cellular level. *Virechana* therapy is thought to improve metabolism and circulation while reducing the liver burden. According to recent research, it may reduce the liver fat fraction, making it a promising strategy for restoring liver health. The excess fatty content in NAFLD can be excreted from the body by boosting bile production and secretion [36].

Free fatty acid buildup, hepatic lipid overload, and endothelial lipid infiltration are the initial events in the pathophysiology of these disorders. This imbalance drives systemic inflammation, reduces vascular tone, and promotes organ-specific dysfunctions such as insulin resistance in diabetes, increased vascular resistance in HTN, and hepatic steatosis in NAFLD [35].

Literature on The Role of Virechna Therapy in Metabolic Disorders

Virechana procedure has a consistent and multifaceted effect on the key cardiovascular risk factors. It had shown an established and statistically significant impact on dyslipidaemia, particularly in reducing serum triglycerides and increasing serum HDL (along with positive results in other lipid profile parameters [Table/Fig-2] [37-47].

These findings underscore the potential of *Virechana* as a cardioprotective intervention through its ability to modify key metabolic risk factors that contribute to atherosclerosis and cardiovascular disease [Table/Fig-2]. While the results are promising,

Study	Title of study	Author name	Sample size (n)	Intervention	Key parameters	Mean changes/% improvement
Study 1 [37]	A standard controlled clinical study on *Virechana Karma* and *Lekhana Basti* in the management of dyslipidaemia (*Medoroga*)	Pooja BA and Bhatted SK	90	*Virechana Karma* in Group-A Atorvastatin Group-C (with 90-Days follow-up)	Lipid profile	Serum Triglyceride - ↓40.5% (256.89 to152.78) in Virechana group and ↓31.1%(232 to 159.67) in atorvastatin group Serum HDL-↑9.9% (51.71 to 56.87) in virechan group and ↓1.3% (50.37 to 49.70) by atorvastatin Serum cholesterol-↓17%(240.84 to 199.87) in virechan group and ↓27%(303.33 to 223.33) in atorvastatin group.
Study 2 [38]	Role of *virechana* therapy (purgation by herbs) in the management of hyperlipidemia: a clinical trial	Bohra M and Sharma UK	15 patients of hyperlipidemia	2 spells of *virechana* at gap of 15 days	Lipid profile	Serum cholesterol ↓26.03% (248.94 to 184.13) Serum Triglyceride ↓28.9% (218.89 to 155.62) Serum Ldl ↓30.35% (164.06 to 114.26)
Study 3 [39]	Comparative study of *Vamana* and *Virechana karma* in controlling blood sugar levels in diabetes mellitus	Jindal N and Joshi NP	10 known case of DM 2	*Virechana karma*	Blood Sugar	FBS ↓ 13.07% (167.5mg/dL to 145.6mg/dL) PPBS ↓ 17.46% (254.9mg/dL to 210.4mg/dL)
Study 4 [40]	Effect and Mechanism of *Virechana Karma* (Therapeutic Purgation) Over Fructose-induced Metabolic Syndrome: An experimental study	Chaturvedi A et al.,	Fructose induced metabolic syndrome in an experimental study using albino rats" with "16 albino rats underwent Virechana in two groups	*Virechana karma*	Fatty acid storage, Insulin sensitivity	Decreased fatty acid storage in the liver, kidney, heart and adipose tissue, increased insulin sensitivity, number and size of Langerhans' islets decreased
Study 5 [41]	Management of uncontrolled type 2 diabetes mellitus associated with peripheral neuropathy through *Virechana karma*: A case report	Yadav U and Bhatted SK	Case report	*Virechana karma*	HbA1c	64-year-old diabetic patient who received *Virechana* treatment was able to secure a remarkable change in the HbA1c from 7.4 to 6.5
Study 6 [42]	Effect of Triphaladi *Virechana karma* in the management of Essential Hypertension (HTN)	Hivale US and Santoshkumar Bhatted	15 patients of essential HTN	*Virechana Karma*	Blood pressure	The mean SBP ↓ by 17.84% (160.67 to 132) Mean DBP ↓ by 18.32% (105.3 to 86)
Study 7 [43]	Efficacy of *Virechana Karma* in the management of *Uccha Rakta Chapa* (Essential HTN) - a clinical Study	Patel RK and Patel A	15 Days follow-up	*Virechana karma*	Blood pressure	SBP 13.22% (145.79 to 126.50) DBP 6.76% (94.57 to 81.71)
Study 8 [44]	Efficacy of *Virechana* and *Basti Karma* with *Shamana* therapy in the management of essential HTN: A comparative study	Shukla G et al.,	33 patients with essential HTN	Virechan karam followed by arjunadi ghanvati	Blood Pressure	The *Virechana* group showed marked improvement (43.75%) compared to the Basti (29.41%) as the overall result of the therapies on systolic and diastolic blood pressure
Study 9 [45]	Role of *Virechana Karma* in metabolic syndrome-Clinical trial	Sharma P et al.,	22 patients	Two sittings of *virechana* Duration – 60 days	Lipid profile	Serum Triglyceride ↓26.09% (118.42 to 87.528) Serum Ldl ↓21.21% (117.25 to 92.37) Serum HDL -↑14.8% (32.90 to 37.77)
					Blood pressure	SBP ↓3.62% (129.80 to 125.10) DBP ↓3.90% (84.700 to 81.400)
					Obesity measures	Mean weight ↓7.05% (80.800 kg to 75.100 kg) waist circumference ↓4.51% (107.2 to 102.37)
					Blood sugar (F)	↓2.83% (105.94 to 102.94)
Study 10 [46]	Effect of *Virechana Karma* on *Sthaulya w.s.r.* to Obesity	Rajan NM and Bhatted SK	30 patients	*Virechana* procedure	Lipid profile	Serum cholesterol ↓7.7% (194.30 to 179.20) Serum Triglyceride ↓10.1% (159.47 to 143.330) Serum Hdl -↑0.35% (57.7 to 57.9)
					Blood sugar	FBS↓ 6.26% (80.93 to 75.87) ↓PPBS 97.91 to 91.96

					Obesity measures	Chest circumference ↓3.18% (105.97 to 102.60) Abdomen circum.↓3.01% (107.37 to 104.13) Hip circum.↓4.5% (112.13 to 107.63)
Study 11 [47]	Clinical Evaluation of *Virechana* in the Management of Metabolic Syndrome-An Observational, Clinical Trial	Chaudhary N, et al.,	30 patients	*Virechana karma*	Lipid profile	Serum Triglyceride ↓ 30.56% (184.5 to 128.1) Serum HDL ↑42.40% (34.2 to 48.7)
					Blood pressure	SBP ↓ 8.41% (143.8 to 131.7) DBP ↓ 9.95% (93.5 to 84.2)
					Weight measures	Weight ↓ 6.55% 90.1kg to 84.2kg Waist circum. ↓ 9.61% (96.8 to 87.5cm)

[Table/Fig-2]: Clinical and experimental evidence on the therapeutic effects of *Virechana Karma* across metabolic disorders [37-47].

the current body of evidence primarily consists of small-scale studies, observational trials, and case reports. To establish the clinical utility of *Virechana*, future research should include rigorously designed randomized controlled trials that compare *Virechana Karma* combined with structured lifestyle interventions to standard pharmacological treatments, such as atorvastatin. Longer follow-up studies are necessary to assess lipid profiles, carotid intima-media thickness, and inflammatory biomarkers. This will be essential to validate its cardioprotective potential and to integrate *Virechana* into evidence-based guidelines for managing metabolic and cardiovascular diseases.

CONCLUSION(S)

Virechana Karma demonstrates consistent benefits across metabolic disorders, including improvements in lipid profile, glycaemic control, blood pressure, and obesity measures. Experimental and clinical data support its role in enhancing insulin sensitivity and reducing cardiovascular risk factors, underscoring its potential as a cardioprotective intervention.

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