

AYURVEDIC MANAGEMENT OF DYSLIPIDEMIA (MEDOROGA): A CASE REPORT

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ABSTRACT

Dyslipidaemia, characterised by an abnormal elevation of total cholesterol, triglycerides, and LDL-cholesterol along with reduced HDL-cholesterol, is a major modifiable risk factor for atherosclerosis and coronary artery disease. In Ayurveda, this condition correlates closely with *Medoroga*, arising from *Agnimandya* (impaired digestive fire) and accumulation of *Ama* and vitiated *Meda Dhatu* due to faulty dietary habits (*Ahara*) and sedentary lifestyle (*Vihara*). This case report presents a 47-year-old male patient with generalised weakness, fatigue, and a lipid profile showing raised total cholesterol (273 mg/dl), triglycerides (294 mg/dl), and LDL-C (158 mg/dl) with low HDL-C (56 mg/dl), diagnosed as *Medoroga*. The patient was managed with a combination of *Shamana* (palliative) and mild *Shodhana* (purificatory) Ayurvedic therapy, comprising *Arogyavardhini Vati*, *Triphala Guggulu*, *Medohar Guggulu*,

and *Arjunarishta*, along with *Nityam Virechana* using *Triphala Churna* for the initial fifteen days, supported by *Pathya- Apathya* (dietary and lifestyle regulation) for a total duration of sixty days. After treatment, a marked improvement was observed: total cholesterol reduced to 128 mg/dl, triglycerides to 151 mg/dl, and LDL-C to 47.8 mg/dl, while HDL-C improved to 50 mg/dl. Symptomatic relief in fatigue and heaviness of the body was also noted, with no

adverse effects reported. This case supports the efficacy of a classical Ayurvedic *Medohara-Lekhana* protocol in the management of dyslipidemia and highlights the need for larger controlled studies to validate these findings.

KEYWORDS: Dyslipidemia, *Medoroga*, *Guggulu*, *Lekhana Chikitsa*, *Virechana*.

INTRODUCTION

Dyslipidaemia refers to an abnormality in the concentration of lipids and lipoproteins in the blood, typically presenting as elevated total cholesterol, triglycerides, and low-density lipoprotein cholesterol (LDL-C), together with decreased high-density lipoprotein cholesterol (HDL-C). It is one of the principal modifiable risk factors contributing to atherosclerosis, coronary artery disease, and stroke, and its prevalence is rising steadily due to sedentary lifestyles, calorie-dense diets, obesity, and stress.^[1] Conventional management relies largely on statins and fibrates, which, despite their efficacy, are frequently associated with myopathy, hepatotoxicity, and other adverse effects, along with the need for long-term or lifelong use.^[2]

Ayurveda does not describe dyslipidaemia as an independent clinical entity, but its pathophysiology and clinical presentation align closely with the classical concept of *Medoroga*, a disorder of *Meda Dhatu* (adipose/fat tissue).^[3] According to Ayurvedic principles, excessive intake of *Guru* (heavy), *Snigdha* (unctuous), and *Madhura* (sweet) foods, combined with *Avyayama* (lack of physical exercise) and *Diva Swapna* (day sleep), leads to *Agnimandya* (weakened metabolic fire) at the level of *Medo Dhatvagni*.^[4] This results in the formation of *Ama* (improperly metabolised toxic byproducts) and abnormal accumulation of *Meda* within *Srotas* (channels), obstructing normal physiology and manifesting as *Sthaulya* (obesity), *Angagaurava* (heaviness of the body), *Alasya* (lethargy), and *Klama* (fatigue) symptoms that parallel the clinical picture frequently seen in dyslipidaemia patients. Management in Ayurveda is therefore aimed at *Agnideepana* (kindling digestive fire), *Amapachana* (digestion of toxins), *Medohara* (fat-reducing) and *Lekhana* (scraping) therapy, together with *Srotoshodhana* (channel clearance), most classically achieved through *Virechana* (therapeutic purgation).^[5]

This report documents a case of dyslipidaemia managed successfully with a classic combination of *Medohara-Lekhana* Ayurvedic formulations and mild *Shodhana* therapy, intending to illustrate a reproducible, low-cost, and low-toxicity treatment approach.

CASE REPORT

Patient Information

A 47-year-old male, a sedentary office worker, presented to the outpatient department with complaints of generalised weakness, easy fatigability, heaviness of the body, and occasional breathlessness on exertion for the past six months. He had no history of diabetes mellitus, hypertension, or coronary artery disease, and was a non-smoker and occasional alcohol consumer. His family history was positive for coronary artery disease (father, myocardial infarction at age 58).

Clinical Findings

- General examination: BMI 27.8 kg/m² (overweight), pulse 78/min, BP 128/84 mmHg, no pallor/icterus/cyanosis/lymphadenopathy/oedema.
- Systemic examination: Cardiovascular, respiratory, and abdominal examinations were within normal limits.
- *Ashtavidha Pariksha* (Ayurvedic Eightfold examination): *Nadi* (pulse) - *Kapha-Pittaja*; *Mala* (stool) - irregular, *Malavrodha* (mild constipation); *Mutra* (urine) - normal; *Jihva* (tongue) - coated (*Ama Lakshana*); *Shabda*, *Sparsha*, *Drik*, *Akriti* - *Madhyama Bala* (moderate constitution), *Sthula* physique.

Investigations (Before Treatment)

Fasting lipid profile revealed: Total Cholesterol 273 mg/dl, Triglycerides 294 mg/dl, LDL-C 158 mg/dl, HDL-C 56 mg/dl, VLDL-C 58.8 mg/dl. Fasting and post-prandial blood glucose, renal function tests, and liver function tests were within normal limits, ruling out secondary causes of dyslipidaemia. Thyroid function was also within normal range.

Ayurvedic Diagnosis

Based on *Nidana Panchaka* (five-fold diagnostic assessment), including *Nidana* (aetiology: *Guru-Snigdha Ahara*, *Avyayama*, *Diva Swapna*), *Purvarupa*, *Rupa* (*Angagaurava*, *Alasya*, *Klama*), *Samprapti* (*Agnimandya* leading to *Meda Dhatu Dushti* and *Ama* formation), and *Upashaya*, the case was diagnosed as *Medoroga* (*Sthaulyajanya Medodushti*) with associated *Amavastha*.

Therapeutic Intervention

The patient was managed on an outpatient basis with a combination of *Shamana Aushadhi* (palliative internal medicines) and mild *Shodhana* (*Nityam Virechana*) for a total duration of 60 days, as summarised in Table

Table 1: Ayurvedic treatment protocol administered.

Drug	Dose	Anupana (Vehicle)	Frequency	Duration
<i>Arogyavardhini Vati</i>	250 mg	Lukewarm water	Twice daily, after food	60 days
<i>Triphala Guggulu</i>	500 mg	Lukewarm water	Twice daily, after food	60 days
<i>Arjunarishta</i>	15 ml	Equal water	Twice daily, after food	60 days
<i>Medohar Guggulu</i>	500 mg	Lukewarm water	Twice daily, before food	60 days
<i>Nityam Virechana (mild)-Triphala Churna</i>	5 g	Warm water, at bedtime	Once daily	First 15 days

Pathya-Apathya (Diet and Lifestyle Advice)^[6]

- *Pathya* (advised): Barley (Yava), old rice (*Shastika/Purana Shali*), green gram (*Mudga*), bitter gourd, drumstick, honey with warm water, *Trikatu*-spiced buttermilk, and regular brisk walking/*Surya Namaskara* for 30–40 minutes daily.
- *Apathya* (restricted): Curd, dairy fat, fried and processed foods, excessive sweets, red meat, day sleep (*Diva Swapna*), and sedentary habits.
- Follow-up: Clinical assessment every 15 days; lipid profile repeated at the end of 60 days.

RESULTS AND DISCUSSION

At the end of 60 days of treatment, the patient reported substantial subjective improvement in the resolution of fatigue and body heaviness, improved exercise tolerance, and regularisation of bowel movements. Repeat lipid profile showed a favourable shift across all parameters, as shown in Table 2.

Table 2: Comparative lipid profile before and after Ayurvedic treatment.

Parameter	Before Treatment	After Treatment (60 days)	Reference Range
Total Cholesterol (mg/dl)	273	128	< 200
Triglycerides (mg/dl)	294	151	< 150
LDL-C (mg/dl)	158	47.8	< 100
HDL-C (mg/dl)	56	50	> 40
VLDL-C (mg/dl)	58.8	30.2	< 30
TC/HDL Ratio	5.89	2.56	< 5

METROPOLIS			
Investigation	Observed Value	Unit	Biological Reference Interval
Remarks: In blood, Urea is usually reported as BUN and expressed in mg/dL. BUN mass units can be converted to urea mass units by multiplying by 2.14.			
Uric Acid, Serum (Serum/Uricacid)	4.0	mg/dL	3.5-7.2
Phosphorus, Serum (Serum/Phosphate (P))	4.02	mg/dL	Adult: 2.5-4.5 Children: 4.0-7.5
Calcium, Serum (Serum/Arsenazo II)	9.5	mg/dL	Adult: 8.6-10.2
Lipid Profile - 2 (Mini-Fastinet)			
Cholesterol Total, Serum (Serum/Cholesterol oxidase peroxidase (CHOD-PAP))	128	mg/dL	Desirable: < 200 Borderline: 200-239 High > 239 Child Desirable: < 170 Child Borderline: 170-199 Child High > 199
Triglycerides, Serum (Serum/Glycerol-3-phosphate peroxidase chromogenic method (GPO-PGD))	151	mg/dL	Normal < 151 High: 151-199 Hypertripleydenic: 200-499 Very High > 499
HDL Cholesterol Direct (Serum/Direct Homogeneous method)	50	mg/dL	Major risk factor for heart disease: < 40 Negative risk factor for heart disease: > 60
Non HDL Cholesterol (Serum/Calculated)	78.00	mg/dL	Optimal: < 130 Desirable: 130-159 Borderline high: 159-189 High: 189-220 Very High > 220
LDL Cholesterol (Serum/Calculated)	47.8	mg/dL	Optimal: < 100 Near Optimal: 100-129 Borderline high: 130-159 High: 160-189 Very High > 190

METROPOLIS			
Investigation	Observed Value	Unit	Biological Reference Interval
Remarks: In blood, Urea is usually reported as BUN and expressed in mg/dL. BUN mass units can be converted to urea mass units by multiplying by 2.14.			
HDL Cholesterol Direct (Serum/Direct Homogeneous method)	56	mg/dL	Major risk factor for heart disease: < 40 Negative risk factor for heart disease: > 60
Non HDL Cholesterol (Serum/Calculated)	217.20	mg/dL	Optimal: < 130 Desirable: 130-159 Borderline high: 159-189 High: 189-220 Very High > 220
LDL Cholesterol (Serum/Calculated)	158.4	mg/dL	Optimal: < 100 Near Optimal: 100-129 Borderline high: 130-159 High: 160-189 Very High > 190
VLDL Cholesterol (Serum/Calculated)	58.8	mg/dL	Optimal: < 30 High: 30-60
LDL/HDL Ratio (Serum/Triglycerides)	2.84		2.5-3.5
Cholesterol / HDL Ratio (Serum/Triglycerides)	4.89		3.5-5
Electrolytes, Serum			
Sodium, Serum (Serum/Ion Selective Electrode (ISE))	142	mmol/L	136-149
Potassium, Serum	4.4	mmol/L	3.8-5.0
Chloride, Serum	101	mmol/L	98-106

Before Treatment

After Treatment

Fig 1: Before and After Reports.

The observed improvement can be explained on the basis of the individual and combined pharmacological actions of the formulations used. *Arogyavardhini Vati*, containing *Guggulu*, *Katuki*, and mineral ingredients, is known for its *Deepana-Pachana* (digestive-stimulant) and hepato-protective properties, aiding correction of *Agnimandya* and *Ama*. *Guggulu*-based formulations such as *Triphala Guggulu* and *Medohar Guggulu* possess classical *Lekhana* (scraping) and *Medohara* properties, attributed in contemporary pharmacological studies to *guggulsterones* that modulate hepatic cholesterol and bile-acid metabolism.^[7] *Arjuna* (*Terminalia arjuna*) is well documented for its cardioprotective, antioxidant, and lipid-lowering actions, supporting cardiovascular protection in dyslipidaemia patients.^[8] *Mild Virechana* with *Triphala Churna* facilitated *Srotoshodhana* and *Ama-pachana*, addressing the root pathology of *Medo Dhatu Dushti* rather than merely the resulting lipid abnormality.^[9] The dietary regulation (*Pathya-Apathya*) and incorporation of daily physical activity further supported metabolic correction, consistent with both Ayurvedic and contemporary lifestyle-medicine principles for dyslipidaemia.^[10]

This case illustrates that a classical, multi-pronged Ayurvedic approach combining *Deepana-Pachana*, *Medohara-Lekhana* formulations, mild *Shodhana*, and *Pathya-Apathya* can produce clinically meaningful improvement in lipid parameters over a relatively short duration, with a favourable safety profile.^[11] However, being a single-case observation, these

findings cannot be generalised, and the possibility of natural variation or placebo effect cannot be entirely excluded in the absence of a control group.

CONCLUSION

This case highlights the potential of an integrated Ayurvedic protocol comprising *Deepana-Pachana*, *Medohara-Lekhana Aushadhis*, mild *Virechana*, and structured *Pathya-Apathya* in achieving significant improvement in lipid profile and associated symptoms in a patient with dyslipidaemia (*Medoroga*), without any reported adverse effects. These findings, while encouraging, warrant validation through larger sample-size clinical trials with appropriate controls and longer follow-up to establish reproducibility and long-term safety.

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