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DYSLIPIDEMIA WITH AN AYURVEDIC VIEW

Silpa Surendran[1], Harikrishnan G [2]

[1]Final year Pg scholar, Department of Kayachikitsa, Sree Narayana Institute of Ayurvedic Studies and Research, Pangode, Puthoor, Kollam.
[2]Associate Professor, Department of Kayachikitsa, Sree Narayana Institute of Ayurvedic Studies and Research, Pangode, Puthoor, Kollam.

Corresponding Author: Silpasurendran1990@gmail.com

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ABSTRACT

Dyslipidemia is an important modifiable risk factor for the onset of Atherosclerosis and Cardiovascular disease. According to the World Health Organization, Dyslipidemia contributes to over 50% of global ischemic heart disease cases and results in more than 4 million deaths annually. Approximately 80% of lipid disorders are linked to diet and lifestyle, while the remaining cases are hereditary.

In Ayurveda, symptoms of Dyslipidemia correspond to *Medodushti*, a condition related to *Kapha Dosha* and *Agnimandya* at the level of *Dhathu.* It is seen as an antecedent stage of *Medoroga. Sthoulyahara chikitsa* can be implemented for Dyslipidemia, encompassing *Deepana, Pachana, Lekhana, Rookshana, Vathanulomana, and Kapha Medohara karmas.*

Keywords: Dyslipidemia, *Agnimandya, Medodushti*

INTRODUCTION

Dyslipidemia is a disorder of lipoprotein metabolism, including lipoprotein overproduction or deficiency. Elevating total cholesterol, low-density lipoprotein cholesterol, triglycerides, and a decrease in high-density lipoprotein cholesterol may manifest. Elevated cholesterol promotes atherosclerosis due to greater

concentrations of low-density lipoprotein and remnant lipoproteins and reduced levels of high-density lipoprotein.[1] Although several new risk factors have been introduced to increase the accuracy of predicting chance of coronary heart disease (CHD) events, only four conditions – Dyslipidemia, Hypertension, cigarette smoking, and/or Diabetes -account for increased CHD risk in 80% to 90% of patients.[2]

Fast foods, lack of exercise, stress, and various addictions are some of the factors which are adversely impacting the lifestyle of man in the 21st century. The incidence of Dyslipidemia is increasing in many developing countries due to dietary and other lifestyle changes. Studies have reported a higher prevalence of lipid abnormalities among Asians compared with non-Asians.[3]

Dyslipidemia is divided into two types: primary and secondary. Primary dyslipidemia results from genetic mutations that impact lipid metabolism. Secondary dyslipidemia is acquired due to lifestyle factors or other medical conditions that influence lipid levels.

Primary Dyslipidemia

This form of dyslipidemia arises from genetic mutations that disrupt lipid metabolism. Primary dyslipidemia can be inherited in autosomal dominant, autosomal recessive, or X-linked patterns. Examples include familial hypercholesterolemia, familial hypertriglyceridemia, familial combined hyperlipidemia, and familial dysbetalipoproteinemia.

Familial hypertriglyceridemia results from mutations in the LPL or apo C-II gene, which hinder the breakdown of triglycerides in chylomicrons and very low-density lipoproteins (VLDL), causing elevated triglyceride levels and increasing the risk of pancreatitis. Familial combined hyperlipidemia is caused by the liver's overproduction of apo B-containing lipoproteins, such as VLDL and LDL, leading to elevated cholesterol and triglyceride levels and insulin resistance. Familial dysbetalipoproteinemia stems from mutations in the apo E gene, impairing the clearance of chylomicron and VLDL particles from the blood and resulting in high cholesterol and triglyceride levels and xanthoma formation.

Secondary Dyslipidemia[4]

Secondary dyslipidemia is a lipid disorder that develops due to external factors such as lifestyle, underlying medical conditions, or medication use rather than genetic causes. Common causes include:

- Obesity
- Diabetes mellitus
- Thyroid disease
- Renal disorders
- Liver disorders
- Alcohol
- Cushing syndrome
- Glycogen storage diseases
- Estrogen
- Drugs

Screening [5]

The **NCEP ATP III Cholesterol Guidelines** classify lipids as follows: Total cholesterol <200 mg/dL is desirable, 200–239 mg/dL is borderline high, and ≥240 mg/dL is high. LDL cholesterol <100 mg/dL is optimal, 100–129 mg/dL near optimal, 130–159 mg/dL borderline high, 160–189 mg/dL high, and ≥190 mg/dL very high. HDL cholesterol <40 mg/dL is low, ≥60 mg/dL is protective, and triglycerides <150 mg/dL are typical, 150–199 mg/dL borderline high, 200–499 mg/dL high, and ≥500 mg/dL very high.

Treatment

Lipid-lowering therapies are key in the secondary and primary prevention of cardiovascular diseases. Therapeutic lifestyle changes, including dietary intervention, moderate exercise, and weight loss, are the first line of treatment for hyperlipidemia and may be sufficient for mild dyslipidemias in low-risk patients.

HMG-CoA Reductase inhibitors, nicotinic acid, fibrates, bile acid sequestrants(resins), omega-3 fatty acids, ezetimibe, and PCSK9 inhibitors are lipid-lowering medications, and the choice of a specific drug depends on the patient's lipid profile, Cardiovascular risk etc. Combination drug therapy is used when a single medication fails to achieve LDL-C targets, manage hypertriglyceridemia and hypercholesterolemia, or address high LDL-C levels with low HDL-C. LDL apheresis is the recommended treatment for hypercholesterolemia, which is unrespon-

sive to medication. Partial ileal bypass is currently reserved for patients with severe hypercholesterolemia, normal triglyceride levels, intolerance to lipid-lowering drugs, and no access to LDL apheresis.

AIM

• To study the *Nidana Panchaka* of *Medodushti* and equate them with Dyslipidaemia.

MATIERALS AND METHODS: The classical *Ayurvedic samhitas*, such as Charaka Samhita, Susrutha Samhita, Ashtanga Hridaya, Madhava Nidana, etc., were analysed critically to better understand the *Nidana Panchaka* and *Chikitsa* of the disease.

Dyslipidemia and *Ayurveda*

Dyslipidemia is not explicitly mentioned and does not directly correspond to any particular disease described in classical texts. However, it may be regarded as a potential stage in the development of *Samprapti* or a pathological state that could lead to disease, although it has not yet progressed to a fully developed disease. It can be analysed as *Medodushti*, which is symptomatically silent as a disease. According to Madavanidana, Madhukosha commentary has described *Medodushti* as a precursor to *Sthoulya*.[6] So, Dyslipidemia can be considered as an antecedent stage of *Medo roga* or *Sthoulya*. Acharya Dalhana has quoted three main etiological factors of *Medovridhi*[7]

Table no-1

Manasika nidana

Sl.no	*Manasika nidana*	Ch	Su	A. S	A.H	M.N	B. P	Y. R	Ba.Raj
1	*Achinthanath*	+	-	+	+	-	-	-	-
2	*Harsha nithyatvath*	+	-	+	+	-	-	-	-
3	*Manasonivrithi*	+	-	+	+	-	-	-	-
4	*Priyadarshana*	+	-	-	-	-	-	-	-
5	*Saukyena*	+	-	-	+	-	-	-	-

Table no-2

Anya nidana

Sl.no	Anya nidana	Ch	Su	A. S	A.H	M. N	B. P	Y. R	Ba.Raj
1	Ama rasa	-	+	-	-	-	-	-	-
2	Beejadosha swabhavath	+	-	-	-	-	-	-	-
3	Snigdhamadura vasthi sevana	+	-	+	+	-	-	-	-
4	Snigdha udwarthana	+	-	-	-	-	-	-	-
5	Taila abhyanga	+	-	+	+	-	-	-	-

a. ADRISHTAVASHATH

a. *Vishishtaharavashath* (includes the use of specific dietary, lifestyle and psychological factors)
b. *Adrishtavashath* (a factor which cannot be seen directly or assessed directly)
c. *Medasavritha margathwath* (*Avarana* of the *Marga* by *Medodhathu*).

1. Aharaja Nidana

Guru, Madhura, Sheeta, Snigdha, Shleshmala, Atipichila, and *Abhishyandi* are the *Gunas* in *Dravyas,* dominant of *Prithvi* and *Ap Mahabhuta*. Because of the same *Bhautika* composition, a direct increase in *Kapha Dosha* and *Medo Dhatu* is seen.

Nava madya, Gramya Rasa, Audaka Rasa, Mamsa Sevana, Paya Vikara, Dadhi, Sarpi, Ikshu Vikara, Guda Vikara, Shali, Godhuma, Masha, Varuni causes *Kapha* and *Medavruddhi*.

2. *Viharaja Nidana*

Avyayama, Avyavaya, Divaswapna, Asyasukha, Swapnasukha, Bhojanottara Snana, Bhojanottara Nidra etc leads to *Kapha* and *Medovruddhi*. A decreased metabolic rate during rest is a significant factor in the onset of excessive fat accumulation. *Viharatmaka Nidana*, like *Divaswapna,* has an *Abhishyandi* property that causes *Srothorodha,* specifically in the *Medovaha Srotas.*

3. Manasa Nidana[8]

Physical factors and psychological elements are essential to manifest *Medo roga* or *Sthoulya.*

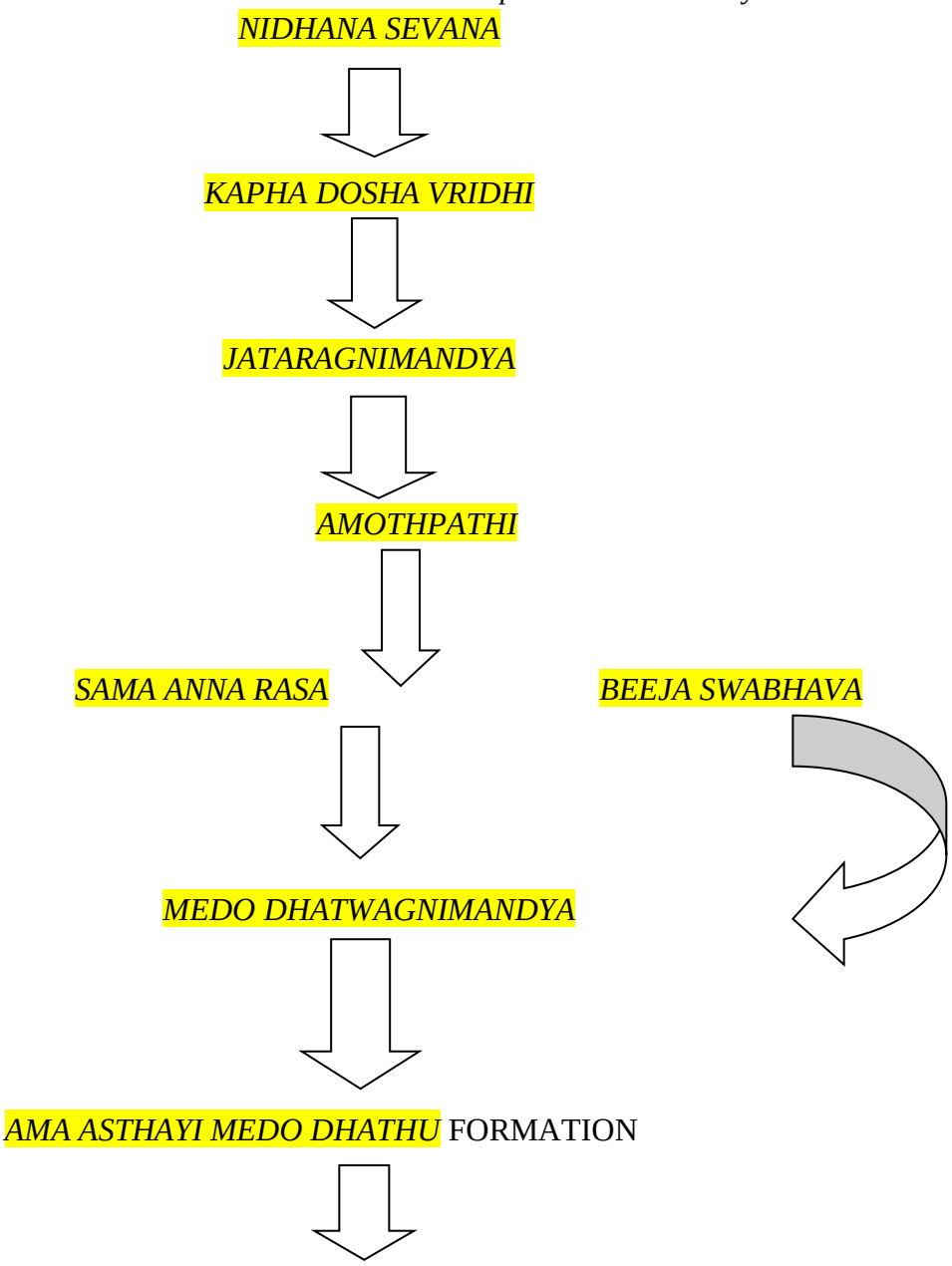
Adrishtavashath is a factor that cannot be directly seen or assessed. However, it can be correlated with hereditary or genetic predisposition.

MEDASAVRITA MARGATVAT

According to Dalhana, *Vata Vikaras* developed due to the Medo Dhatu's *Avarana* of the Marga, which refers to secondary reasons contributing to *Medoroga* and where Vata plays a prominent part in pathogenesis. An overabundance of Medo Dhatu impedes the usual Gati of Vata Dosha, producing additional pathogenesis.

SAMPRAPTHI

Kaphavardhaka ahara viharas cause *Jadaragnimandhya*, which leads to the formation of *Sama rasa*. This *Rasadushti* leads to *Medodhatwagnimandya*, resulting in *Ama asthayi medodhathu* and manifesting as Dyslipidemia. Suppose a *Medodushti* person continues to follow the same improper regimen. In that case, there will be *Sanga* of *Ama asthayi medodhathu* in *Srothas* leading to *Margavarodhajanya vatha prakopa, Jadaragni sandhushana* and formation of *Upadravas* like *Sthoulya.*



MEDODUSHTI

SAMPRAPTHI GHATAKAS

Dosha : Tridosha

- *Pitta - Pachaka,*
- *Vata - Samana and Vyana*
  Kapha – Kledaka,
- *Dushya : Rasa and Meda Dhatu*
- *Agni : Jatharagni, Medodhatwagni*
- *Srotasa : Medovaha, Mamsa,*
- *Rasavaha, Swedavaha Srotasa*
- *Srotodushti : Sanga*
- *Adhisthana : Sarvanaga*
- *Udbhavasthana : Amashaya*
- *Prasara : Rasayani*
- *Roga Marga : Bahya*

Medodushti occurs when the *Meda dhatvagni* is weak and unable to nourish the next dhatu in sequence properly. This leads to the increase of *Abaddha medodhatu*, a condition that can be associated with dyslipidemia due to similar causes, disease progression, and clinical features. If the underlying etiological factors continue, this *Medodushti* can develop into *Medoroga.*

CHIKITSA

Medoroga is *Santharpanajanya vyadhi, so Apatarpana* measures have been applied. Nidhanaparivarjana and Samprapthivighatana can attain it.

The treatment should focus on restoring Agni's balance and promoting the *Pachana* (digestion) of accumulated *Samamedas*. The management aims to enhance Agni's functional efficiency at various levels and employ *Lekhana* therapy to clear *Srotorodha* (obstruction) in the vessels. *Pachana dravyas* are predominant in *Agneya Mahabhuta*, opposite Ama's Saumya properties. *Samana chikitsa* followed by proper *Sodhana* will give better results.

Samsodhana is administered in patients who have *Bahudosha* and *Adhikabala* to withstand *Samsodhana. Swedana of Sthula rogi is not recommended, but if required, Mrudu Sweda is indicated. Ruksha, Ushna vasthi, lekhana vasthi* are indicated. Acharya Sushruta mentions *Nasya* with *Triphaladi taila* for the therapeutic approach of *Medovriddhi.*

Pathya

The diet must include *Kapha Medoharadravyas*. *Katu, Tiktha, Kashaya rasa* have *Meda upasoshana* karma. So *Katu, Tiktha, Kashaya rasa* can be used. *Laghu guna aharas* is indicated because of its predominant *Akasha Vayu Mahabhutha*. The *Vishesha guna* principle reduces the *Prithivi, Jala* predominant *Kapha* and *Medus in Sareera*. Regular *Vyayama*, according to *bala*, provides *Agni Deepthi* and strength to the body.

Apathya

Aharas having *Guru, Pichila, and Snigha Gunas* are undesirable. The *Samana guna* principle increases the *Kapha* and *Medus*. *Abhishyandi aharas* produces *Srothorodha, leading* to *Ama* formation, so it should be avoided. All *Kaphamedokara aharas* are unsuitable in this condition. *Aharas-Viharas* that lead to *Agnimandya*, such as *Adhyasana, Samasana, Vishamasana*, and *Virudhahara*, should be avoided.

Summary

There is no specific term for Dyslipidemia in the Ayurvedic classics. *Medodushti* can be linked to dyslipidemia and is considered a precursor to *Medoroga*, a condition primarily associated with agnimandya at the level of *Dhathu*. Various factors, such as *Aharaja, Viharaja, Mansika* and *Beejadosha*, are responsible for its development. *Medo dhathu has* two types: *Badha /Poshaka* and *Abadha/Poshya medo dhathu*. *Badha medo dhathu* is immobile in nature. *Abadha medo dhatu* is *Asamhatham, which* means mobile in nature, which circulates throughout the body and nourishes the *Sthayi medo dhathu.*

Early intervention during the initial stages of *Kriyakala* can help prevent progression to advanced stages, such as coronary artery disease (CAD) and stroke.

CONCLUSION

Medodushti occurs when the *Meda dhatvagni* becomes weak and cannot adequately nourish the subsequent dhatu in the sequence. This leads to the accumulation of *Abaddha Medodhatu.* Due to similar etiological factors, disease progression, and clinical manifestations, this stage of pathogenesis can be

compared to dyslipidemia. Over time, with the persistence of these causative factors, *Medodushti* progresses into *Medoroga*.

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