Ayurvedic Understanding Of Dyslipidemia – A Conceptual Analysis

Dr Yadu Gopan¹, Dr Shrilatha Kamath T.²

¹Assistant professor, Dept. of PG studies in Kayachikitsa, SDM College of Ayurveda and Hospital, Hassan
²Professor & HOD, Dept. of PG and PhD studies in Kayachikitsa, SDM College of Ayurveda, Udupi

ABSTRACT

Dyslipidemia is a disorder of lipoprotein metabolism, which can include overproduction or deficiency of lipoproteins or both. Increased levels of atherogenic lipoproteins (especially LDL, but also IDL) contribute to the development of atherosclerosis. This in turn can lead to coronary artery disease (CAD), cerebrovascular diseases (CVD), peripheral vascular diseases (PVD) etc. The Indian Council of Medical Research (ICMR) surveillance project reported a prevalence of dyslipidemia of 37.5% among adults aged between 15 to 64 years, with an even higher prevalence of dyslipidemia (62%) among young male industrial workers. Dyslipidemia remains asymptomatic and undetected until a complication occur such as myocardial infarction due to early atherosclerosis.

In Ayurveda, dyslipidemia is correlated with medoroga, a condition arises due to medovridhi which in turn restricts the nourishment of other dhatus. Medoroga, prameha, shaulya are interrelated conditions in which the main culprit is vitiated medo dhatu. Hence these conditions possess similarity in management also. Ayurvedic management of medoroga includes rukshana, udvartana, ruksha-ushna basti, virechana and shamana prayogas like guggulu, shilajithu, guduchi etc.

Keywords: Dyslipidemia, Medoroga, Ayurveda
I. INTRODUCTION

Cholesterol in our body is usually obtained from food sources (animal) or synthesized in the liver. All cells depend on cholesterol as building blocks to make many membranes. Fats (lipids) are insoluble in plasma and therefore cannot be transported directly in the blood. It is now recognized that lipids (fats/cholesterol) in different forms are transported by lipoproteins.

Dyslipidemia is a disorder of lipoprotein metabolism, which may include lipoprotein overproduction or deficiency, or both. This disorder may manifest as elevated plasma cholesterol, TG or both, or a low plasma concentration of high-density lipoprotein or all three contribute to the development of atherosclerosis. The most common types of dyslipidemia include high levels of low-density lipoprotein (LDL) cholesterol, high levels of triglycerides, and low levels of high-density lipoprotein. When LDL cholesterol levels are high, fatty plaques can build up in the blood vessels that, over time, create atherosclerosis and lead to peripheral artery disease, coronary heart disease or stroke.

Dyslipidemia is correlated with medoroga in ayurveda. Kapha-medo prakopaka nidanas cause kapha prakopa in turn leading to medo vridhi and dushana resulting in medoroga. Here the abnormality in medodhatu is both quantitative and qualitative. It produces various lakshanas as well as upadravas too. Based on the etiology, clinical features and complications; dyslipidemia can be well correlated with medoroga.

II. OBJECTIVES

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

III. MATERIALS AND METHODS

An effort was made to collect and evaluate various references regarding dyslipidemia and medoroga in order to assess their similarities in the etiopathogenesis and clinical features.

3.1. Medodhatu

Medo dhatu is formed from mamsa dhatu when the later is acted upon by meda dhatvagni. During this process medoposhaka bhaga is formed which in turn nourishes the medodhatu. Medo dhatu has snigdha and guru guna predominantly. It is provides bala and causes brimhana of the body. It nourishes the asthi dhatu and also causes snigdhata of gatra and netra. Medo dhatu is of two forms; asthayi and sthayi medo dhatu. Asthayi medas is the poshaka medo dhatu and sthayi medas is poshya medo dhatu. Poshaka medodhatu circulates through the medovaha srotas in the whole body and gives nourishment to the poshya medodhatu. Poshya medodhatu is located in sites like udara, anvasthi, sphik, sthana etc. It is also found in mamsa as vasa.
3.2. Medo pradoshaja vikara

Medo dhatu can undergo different types of pathological changes. Medo vridhi, kshaya, dushti etc are some examples. Vridha medo dhatu can cause avarana to vata dosha and srotorodha. This in turn will lead to different disorders. Origin of any type of medo pradoshaja vikara is from the medo vaha srotodushti. Avyayama (lack of physical exercise, divaswapna (day time sleep), ati medya bhakshana (excessive fat intake), ati varuni sevana (excessive alcohol intake) are causes for medo vaha srotodushti. This will lead to diseases like sthoulya and prameha. Kapha dosha plays vital role in causing medodushti. Because of the similar properties and etiological factors, kapha dosha vitiates medo dhatu. Prakupita kapha dosha can cause vardhana and dushana of medo dhatu ie, quantitative and qualitative vitiation.

3.3. Santarpana janya vikara

Charaka classified diseases in to two broad types; santarpana janya vikara and apatarpana janya vikara. Ati santarpana and ati apatarpana are the causes for these diseases. Santarpana janya vikaras are caused by dietary habits like excessive intake of foods having properties like snigdha, madhura, guru, pichila such as navanna, nava madya, anupa-varija mamsa etc. Cheshta dwesha, divaswapna, shayya sukha, asana sukha etc are the viharaja nidanas. Ati santarpana leads to kapha vridhi and dushana of rasa and medo dhatus mainly. This in turn leads to diseases like prameha, sthoulya, srotolepa, kandu etc. Dyslipidemia can be considered as a santarpana janya vyadhi based on the similar etiological factors.

Apatarpana is the chikitsa for santarpana janya vikaras based on the vishesa siddhanta. Drugs used for apatarpana chikitsa possess opposite qualities of the santarpana nidanas.

3.4. Medoroga

Medoroga is explained as a separate chapter in madhavanidana. Charaka explains medoroga in terms of sthoulya. Sushruta in sutrasthana explained medo vridhi nidana, lakshana and chikitsa which are similar to the other textbooks. Kapha medo prakopaka nidanas are the causative factors for the development of medoroga. Though the medo roga occurs due to medo dhatu dushti, the actual pathology starts at the jataragni level. Medoroga samprapti explained by Charaka and Sushruta gives importance to jataragni.

3.5. Medo roga v/s Dyslipidemia

3.5.1. Nidana

Charaka explains ati madhura- guru- snigdha- medya anna sevana (excessive intake of foods rich in fat and carbohydrates), avyayama- avyavaya- harshanitya– achintana (lack of physical exercise and sedentary lifestyles) as medoroga nidanas. He also includes beejaswabhava as a nidana. Dyslipidemia is mainly of two types based on the etiology; primary and secondary. Primary dyslipidemia is due to genetic defects in lipoprotein metabolism or genetic defects which are precipitated due to several environmental factors such as diet, substance use, steroids, contraceptives, etc. Secondary dyslipidemia is caused by life style factors and...
underlying medical conditions.\textsuperscript{12} Beejaswabhava can be correlated with the genetic predisposition for primary dyslipidemia. Excessive fat intake and lack of physical exercise can lead to the secondary dyslipidemia which is similar to the aharaja and viharaja nidanas causing medo roga.

3.5.2. Samprapti

The prime causative factor for medoroga is kapha dosha and medodhatu. It is evident from the nidanas of medoroga. As kapha and medas are having similar properties, the chances for their association and vitiation are more. Similar process is behind the development of sthoulya and prameha also. Other than kapha and medas, involvement of vata and pitta are also explained by acharyas. Prakupita medas causes srotorodha to vata and as a result of that the vata stays in the koshta and in turn increases the agni. This will lead to excessive food intake and medovridhi. Here the agnivridhi is contributed by pitta dosha also.\textsuperscript{13} Though the agni is increased, ahara rasa formed will be ama rasa due to the “adhyashana sheela” (excessive food intake).\textsuperscript{14} This type of anna rasa does not contribute to the nourishment of body but increases the meda.

Samprapti of medoroga can be explained in two ways.

In the first type of samprapti, kapha medo vardhaka nidanas like ati sampurana, ati madhura-guru-snigdha ahara sevana, adhyashana etc directly increase the kapha and medas. As a result of the digestion of this kind of food articles and after they attain avastha paka and vipaka, it will lead to the atimadhura bhava. This “madhura praya anna rasa” increases snehabhava in shareera. Hence the circulating sneha in the shareera through the rasavaha srotas also is more than the required quantity. This will lead to medoroga.

In the second type of samprapti, kapha medo vardhaka nidanas like avyayama, avyavaya, divaswapna, achintana etc not only cause kapha medo vridhi but also reduce the utilization of already accumulated meda. Since vyayama, vyavaya, chintana etc increases rukshata and which is opposite to meda, they are of great importance in the utilization of sthayi meda for energy production. Role of dhatwagnimandya and dhatugata ama is also can be considered here. Kapha medo vardhaka viharas can affect medodhatwagni than the jatharagni. This will lead to the medodhatu gata ama and in turn pachana of excess meda.

Medo dhatu undergoes two types of pathological changes in the development of medoroga; medovridhi (quantitative increase) and medodushti (qualitative vititation). Kapha dosha and medo dhatu possess ashrayashrayi bhava due to which the same nidanas cause vitiation of both. Prakupita kapha gets mixed with medas and lead to medovridhi and medodushti. Medovridhi leads to the srotorodha in various srotas and lead to specific symptoms. Srotorodha in shukravaha srotas leads to alpavyavayata and in koshta leads to samana vata prakopa and ati kshudha. Srotorodha also restricts the uttarottara dhatu poshana which in turn lead to dourbalya. Medodushti leads to changes in the quality of medodhatu which in turn affects the normal functioning of medodhatu. This leads to symptoms like pipasa, swedabadha, dourgandhya, javoparodha etc.
3.5.3. Lakshana

Medoroga can lead to localized and generalized symptoms. Excessive fat deposition in the sphik (buttock), sthana (breast), udara (abdomen), parshwa (waist) etc is seen in the medo roga. These kinds of symptoms are seen in the obesity associated dyslipidemia. Ati kshut (excessive appetite) and ati pipasa (excessive thirst) are caused by the agnivridhi due to the samana vata prakopa. Though the agni is increased, the ama annarasa is formed which does not contribute to the nourishment. Energy production is not possible when the increased cholesterol level leads to insulin resistance which makes the person feel more hungry and thirsty. Kshudra swasa (dysnea of exertion) is seen mainly in obese patients. Atisweda (excessive perspiration) and dourbalya (fatigue) are also the lakshanas of medoroga.

3.5.4. Shonita abhisyanda

Shonita abhisyanda is a condition mentioned by Charaka. Abhisyanda is the property which causes srotorodha (obstruction in the channels). Obstruction to the channels of blood is termed as shonita abhisyanda. Intake of vegetables like pushkara, rohinika and meat fried in sarshapa taila along with madhu is told as a specific nidana for shonita abhisyanda. Santarpana janya vikaras leads to the excessive madhura bhava of rasadhatu which increases the sneha bhava in shareera. This leads to shonita abhisyanda.

3.5.5. Upadrava

Complications of medoroga are explained by Charaka under the name “ashta sthoulya dosha”. First among the complications is ayusho hrasa ie, reduced life expectancy. Dyslipidemia has increased risk of cardiovascular diseases and cerebro vascular accidents which are the major causes for death worldwide. Javapardha (lack of enthusiasm), dourbalya (fatigue), dourgandhya (foul body odor), swedabadha (excessive sweating) are also seen dyslipidemia. Krichra vyavayata is also included under sthoulya dosha. It is found that elevated serum cholesterol and reduced HDL cholesterol levels are associated with an increased risk of erectile dysfunction. Ati kshut and ati pipasa are also sthoulya doshas which can be correlated with polyphagia and polydypsia. Excessive fat accumulation can lead to peripheral insulin resistance which in turn can reduce the energy production from the fat. This energy demand leads to excessive hunger and thirst.

3.5.6. Medoroga chikitsa

Pathya and apathy are given more in the management of all type of medopradoshaja vikaras. Kapha-Meda-Vata hara anna pana and viharas like vyayama, langhana etc are advised in medoroga chikitsa. This can be used as a preventive measure also. Modern medicine also advises non pharmacological managements like lifestyle modification, dietary changes and physical exercise. Charaka explains “guru cha atarpanam” as the first line of management for medoroga, providing ahara or oushadha which have guru guna but do not cause tarpana. Honey is considered to be this kind of dravya. This can be correlated with the concept of low fat low carbohydrate high protein diet used in the non pharmacological management of dyslipidemia. Vata shleshma medo hara anna pana is also advised and treatment procedures like ruksha ushna teeksha basti and ruksha udvartana also are used.
According to Sushruta, virukshana and chedana are the main two lines of management of medoroga. Virukshana is meant for medoharana and chedana is for srotoshodhana. These two therapies are required for the removal of excessive lipid as well as to regulate the normal lipid metabolism. Modern medicine advises statins as the first line of management for dyslipidemia. Statins are HMG-CoA inhibitors that limit the rate of cholesterol synthesis. When cholesterol production is reduced, number and activity of LDL receptors are upregulated and this stimulates removal of circulating LDL, IDL and VLDL cholesterol. Fibrates are the next choice of drugs which enhance catabolism of plasma lipoproteins. The concept of virukshana or medoharana is similar to the catabolism of lipoproteins which allows the removal of excessive level of circulating lipoproteins. LDL receptor stimulation leading to the removal of LDL, IDL and VLDL can be correlated with the chedana and srotoshodhana. These two functions are required in a balanced manner in order to maintain normal lipoprotein level in the blood. Other than this, shamana oushadhis like guggulu, shilajithu, lohabhasma, triphala, kshara etc are also useful in the effective management of dyslipidemia.

IV. DISCUSSION
Dyslipidemia is a risk factor for the development of atherosclerosis which further leads to serious illnesses like cardiovascular disease and cerebrovascular accidents. Increased levels of atherogenic lipids like TG, LDL-C, VLDL-C and decreased levels of HDL-C are the causes for this. Dyslipidemia and its complications can be prevented to a higher extend by adopting proper life style and diet modifications. Pharmacological managements will also be required once the disease has set in. In ayurveda, medo dhatu is correlated with lipid. By evaluating the textual references, it can be understood that medo dhatu and lipid possess similarity in their structural and functional aspects. Dyslipidemia is correlated with medoroga and these two conditions have similarity in their etiopathogenesis, clinical features and complications also. Treatment of dyslipidemia is mainly intended at reducing the cholesterol production and enhancing the removal of excessive level of circulating cholesterol. Ayurvedic methods like virukshana and chedana are also similar to this. Medoharana can be done by virukshana and srotoshodhana can be done by chedana chikitsa.

V. CONCLUSION
Dyslipidemia is widely managed by Ayurvedic medicines and treatment procedures. Understanding the theoretical concepts in detail by evaluating the classical references of medoroga will help in enhancing the efficacy of the treatment. This conceptual study contains the information about dyslipidemia and its ayurvedic counterpart ie, medoroga. Similarities between dyslipidemia and medoroga in their etiopathogenesis, clinical features, complications and management are discussed in this article.
REFERENCES


