

## Pathophysiology of Dyslipidemia in Modern Medicine and Its Correlation in Unani Literature

Sana Taufiq<sup>1</sup>, Huma Noor<sup>2</sup>

<sup>1</sup>Assistant Professor, Department of Amraz-e-Jild wa Zohrawiya, AKTC, AMU, Aligarh

<sup>2</sup>Assistant Professor, Department of Kullyat, SUMCH, Sanskriti University, Mathura

Corresponding Author: Sana Taufiq

### ABSTRACT

Dyslipidemia is defined as a disorder of lipoprotein metabolism, which includes the deficiency or overproduction of lipoproteins or can be a combination of both. The manifestation of the disorder can be seen as an elevation of plasma cholesterol or triglycerides or both or a low HDL level and sometimes a combination of all three together contributing to the development of atherosclerosis. The Pathophysiology includes any defect in the lipid metabolism either in the form of overproduction of a lipoprotein or a decrease in its catabolism i.e. *LDL clearance defect*, *Defect in Lipolysis and Remnant removal defects* etc. In Unani literature, no disease with the name of Dyslipidemia could be found, but going through the treatise one could get familiar with a disease *Siman-e-mufrat* which actually corresponds to dyslipidemia in many ways. Various eminent scholars have given detailed account of the disease and also, they were aware with the concept of *Dasoomat-fid-dam* (presence of fats in the blood). With the liability of all of them in the disease, the full-length paper will discuss in detail the pathophysiology in Modern Medicine and pathological co-relation with the diseased process of dyslipidemia in Unani Medicine.

**Key Words:** Unani, Dyslipidemia, *Siman-e-Mufrat*, Pathophysiology

### INTRODUCTION

Dyslipidemia is defined as a disorder of lipoprotein metabolism, which includes the deficiency or overproduction of lipoproteins or can be a combination of both.<sup>1</sup> The manifestation of the disorder can be seen as an elevation of plasma cholesterol or triglycerides or both or a low

HDL level and sometimes a combination of all three together contributing to development of atherosclerosis.<sup>[1,2]</sup>

**PATHOPHYSIOLOGY IN MODERN SYSTEM OF MEDICINE:** Any defect in the lipid metabolism may leads to development of the diseased condition. Defects can be either overproduction of a lipoprotein or a decrease in its catabolism. Following possible mechanism may contribute to the causation of the disease which are summarized below:

- *LDL clearance defect:*<sup>[3]</sup> Deficiency or reduction of apo B-100/E receptors leads to defective hepatic clearance of VLDL remnants (IDL), and then more IDL is converted into LDL. Also clearance of LDL is reduced resulting in increased in LDL concentrations.
- *Defect in Lipolysis:*<sup>[3]</sup> There are two factors responsible for defective lipolysis:
  - i. A reduction in the availability of Lipoprotein Lipase (LPL), such patients have marked elevation of TG-rich lipoproteins especially Chylomicrons.
  - ii. There could be abnormality in the lipoprotein themselves. Marked triglyceridemia could be seen in patients having defect in the composition of apoprotein of triglycerides rich lipoprotein, which is best illustrated by the patients who have a congenital absence of apo C-II.
- *Remnant removal defects:*<sup>[3]</sup> Apo E plays a crucial role in the removal of

remnants of Triglyceride rich lipoprotein. The chylomicrons receptors recognize Apo E, whether they are present on chylomicron remnant or on large LDL. Apo E also appears to promote removal of smaller VLDL remnants via LDL receptor. Every person inherits two genes for apo E and there are around six genotypes. People with E-2/2 genotype tend to accumulate remnants leading to the diseased condition.

- *Overproduction of lipoproteins:* [3] Overproduction of lipoproteins particularly VLDL is the causation of many types of dyslipidemia. Increased production of VLDL can be primary or secondary. The possible reason for primary type could be excessive synthesis of apo B without a concomitant overproduction of VLDL-TG leading to increased secretion of VLDL. For secondary causes, diet rich in carbohydrates can stimulate synthesis of VLDL-TG.

Overproduction of VLDL also leads to conversion of VLDL to LDL. The over production of VLDL-apo B when associated with excessive synthesis of VLDL-TG, it results in the production of endogenous hyperlipidemia.

When hypersecretion of VLDL is combined with apo E 2/2 genotype, there is a marked increase in LDL remnants or beta VLDL explaining how a catabolic defect can accentuate hyperlipidemia in a patient with overproduction of VLDL. Hyperalphalipoproteinemia is usually the result of a mild defect in clearance of LDL linked with the production of VLDL.

Familial combined hyperlipidemia is explained by combination of VLDL and one or more catabolic defects defining lipoprotein phenotypes which can occurs in different family members at different times.

**PATHOPHYSIOLOGY IN UNANI SYSTEM OF MEDICINE:** In Unani literature, no disease with the name of dyslipidemia could be found, but going through the treatise one could get familiar

with a disease Siman-e-mufrat which actually corresponds to dyslipidemia in many ways. Various eminent scholars have given detailed account of the disease and also, they were aware with the concept of Dasoomat-fid-dam (presence of fats/ Shaham in the blood) which is congruent to lipids in present era.

Regarding Pathophysiology in Unani literature, the concept of dasoomat-fid-dam and Siman-e-mufrat has been already discussed. With the liability of all of them in the disease, here is a pathological correlation being made with the diseased process of dyslipidemia.

According to Ibn Nafees, *Dasoomat* is of two types: *Lateef type* (Lighter one); which produces Tabai hararat (energy) into Lahmi Aza (Muscular organs) and *Kaseef type* (Heavier one); which penetrates into Barid Aza (cold organs) or Agshiya (membranes) and here they are deposited after getting solidified into shaham. [4] The reason by which dasoomat gets solidify is excessive Buroodat, therefore Barid Ratab (Cold and Wet) person has more accumulation of fat in the body. [5]

In normal being, the vessels remain dilated and there is a proper and constant supply of nutrition to various parts of the body. But excessive fat in the body leads to the narrowing of the blood vessels, which in turn are not able to fulfill the demands of Rooh-e-Haiwani and thus causes diminution of Hararat-e-ghareezia. There is also a risk of rupture to the blood vessels due to narrowing and compression. [6-10]

## CONCLUSION

Thus it can be summarized from the above discussion that morbid matter (*shaham*) when gets accumulated leads to narrowing and hardness of blood vessels, hence blood flow is hampered, there will be disturbance in flow and penetration of Rooh into the organs. When Rooh is inadequate, the supply of blood to various organs is hindered especially when Heart and Brain are affected, there are chances of syncope or stroke or sometimes death may also occurs.

[4] These changes can be co-related with pathological changes arising due to atherosclerosis, for which dyslipidemia plays a contributing role in its development.

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