

# Farte Tadassum Fid Dam (Dyslipidemia): A Comparative Review of Unani and Modern Perspective

Momin Arsheen<sup>1</sup>; Zaibunissa Begum<sup>2</sup>

<sup>1</sup>P.G. Scholar, <sup>2</sup>HOD & Professor

<sup>1,2</sup>Department of Ilmul Advia (Pharmacology), Govt. Nizamia Tibbi College, Charminar, Hyderabad, Telangana, India.

**Abstract :** A dysbalance of blood lipids that may involve an increase in levels of cholesterol, triglycerides, or low-density lipoproteins (LDL) and reduce the levels of high-density lipoproteins (HDL) is called Dyslipidemia, or *Farte Tadassum fid Dam* in Unani terms. According to Unani view, these lipid imbalances are directly connected with derangement in *Balgham* (phlegm), especially when it is abnormal (*Balgham-e-Ghair Tabai*). Unani scholars have always argued that *Balgham* is very crucial in ensuring the integrity of blood (*Dam*) and vascular health. Excess or qualitative alteration of *Balgham*, usually through sedentary life, cold, and moist eating habits, or suppressed digestion (*Zo'f-e-Hazm*), causes stagnation of the blood circulation, constriction of vessels and inappropriate humoral transformation, which corresponds to the contemporary lipid deposition and atherosclerosis. The goal of the review is to combine the classical Unani literature with modern biomedical evidence regarding dyslipidemia and identify commonalities between the pathological functions of abnormal *Balgham* and lipid dysfunction. It also examines the conventional Unani practices of treating this condition with regimented therapy (*Ilaj bil Tadbeer*), with herbal medicine (*Ilaj bil Dawa*), and through dietary correction (*Ilaj bil Ghiza*), in a time-tested and comprehensive system of managing dyslipidemia

**IndexTerms** - Dyslipidemia, Farte Tadassum fid Dam, Unani Medicine, Balgham, Humoral Theory, Phlegmatic imbalance

## 1. INTRODUCTION

In Unani medicine, dyslipidemia is called *Farte Tadassum fid Dam*, which is the pathological deposition or disproportion of lipids in the blood, such as increased total cholesterol, triglycerides, low-density lipoproteins (LDL), and reduced levels of high-density lipoproteins (HDL).<sup>1,2,12</sup> Dyslipidemia is currently considered a significant cardiovascular disease risk factor, an obesity risk factor, a type 2 diabetes risk factor, and a metabolic syndrome risk factor in contemporary biomedical sciences.<sup>12,13</sup> Yet, even well before the development of modern lab diagnostics, the Unani scholars theorized about the lipid abnormalities in terms of humoral imbalance, especially the *Balgham* (phlegm).<sup>1-5</sup>

According to the Unani philosophy, there are four basic humors that rule the human body: *Dam* (blood), *Balgham* (phlegm), *Safra* (yellow bile), and *Sauda* (black bile). Humors have certain qualities and functions and the balance between their quantity and quality provides health. *Balgham* is of cold, wet nature, and is important in nutrition, in the lubrication of the human body, and in the preservation of the internal environment of the body.<sup>1,3,4,26-28</sup> But this extreme or changed quality of *Balgham* may result in stagnation in the metabolism, impaired digestion (*Zo'f-e-Hazm*), and vessel stasis—processes that are virtually synonymous with the pathophysiology of dyslipidemia and atherosclerosis in the contemporary idiom.<sup>3-5,14,15</sup>

The symptoms of heaviness, fatigue, vessel blockage, and slow circulation resembling those of modern dyslipidemia were described by the Unani scholars like *Ibn Sina* (Avicenna) and *Zakariya Razi*.<sup>1,2</sup> They were commonly associated with chilly and damp eating habits, sedentary behaviour and excessive or incorrect production or conversion of *Balgham*.<sup>3,4</sup> Moreover, the deposition of pathological *Balgham* was thought to make the blood thick, and to retard the blood flow, like what we now know is an increased blood viscosity and lipid deposits in arterial walls.<sup>5,7,9,14</sup>

This review will attempt to bridge the gap between classical thinking of the Unani and modern biomedical knowledge by discussing the concept of *Farte Tadassum fid Dam* in relation to abnormal *Balgham*. It also critiques therapeutic interventions in Unani medicine by relying on both traditional knowledge and current studies, this paper aims to advance an integrative and interdisciplinary approach to the treatment of dyslipidemia.<sup>6,8,13</sup>

## 2. METHODOLOGY

Electronic databases: PubMed, Scopus, Google Scholar, ScienceDirect, ResearchGate. Classical Unani literature: *Al-Qanoon fil Tibb* (Ibn Sina), *Kitab al-Hawi* (Razi), *Makhzan al-Mufradat*, National Formulary of Unani Medicine (NFUM) and other canonical works. Contemporary manuals: *Principles of Internal Medicine* by Harrison, *Medicine* by Davidson, *Textbook of medical physiology* by Guyton and Hall, *Pharmacology* by Goodman and Gilman etc.

### 3. MODERN PERSPECTIVE ON HYPERLIPIDEMIA:

#### A. Dyslipidemia / Hyperlipidemia (*Farte Tadassum Fiddam*)

Dyslipidemia is a general classification of unhealthy levels of lipids and lipoproteins in the blood. Either in the form of increased total cholesterol, low density lipoprotein (LDL-C), triglycerides (TG) or decreased high density lipoprotein (HDL-C).<sup>12,13</sup> The term hyperlipidemia denotes in particular high levels of plasma lipids, and dyslipidemia focuses on the qualitative changes of lipid fractions.<sup>13</sup> There are two most common clinical abnormalities: hypercholesterolemia (elevated LDL-C and total cholesterol) and hypertriglyceridemia (elevated TG-rich lipoproteins).<sup>12,13</sup> The two conditions have a close relationship with cardiovascular risk and atherosclerosis.<sup>14,15</sup>

#### B. Physiology and Lipid Transport.

Lipids are important in the maintenance of cell structure and energy. These may be classified into element constant lipids, including structural phospholipids that do not break down when one gets hungry, and ingredient variable lipids, including triglycerides, which are energy stores and are synthesized and degraded equally.<sup>16,17</sup>

One of the large body lipid stores is the depot fat, which comprises approximately 12 percent of body weight of a healthy adult. This olefin accumulates primarily in the subcutaneous tissue, in mesentery, omentum, perirenal tissues, and in muscle.<sup>16</sup> The depot fat chemically consists of triglycerides, which are formed by fatty acids such as oleic, palmitic, stearic acid, small quantities of lecithin, cholesterol and polyunsaturated fatty acids.<sup>16,17</sup> In pathological states, lipids can gather in abnormal tissues and result in atherosclerosis, xanthomas, and cholesteroses.<sup>14,15</sup>

#### C. Cholesterol Metabolism

Cholesterol is an essential lipid molecule which is needed to maintain the integrity of cell membranes, bile acid production and to produce steroid hormones. It is insoluble in water and is carried by plasma in the shape of lipoproteins—chylomicrons, VLDL, IDL, LDL, and HDL.<sup>18,19</sup>

Dietary cholesterol, whether free or anesterified is hydrolyzed by cholesterol esterase and emulsified by bile salts and then is absorbed in the small intestine. It is absorbed into blood as chylomicrons that pass-through lymphatics to the systemic blood and ultimately to the liver.<sup>19</sup>

**Biosynthesis:** The biosynthesis of endogenous cholesterol occurs by synthesizing acetyl-CoA in the liver into cholesterol. The multi-step route is through the production of HMG-CoA, to mevalonate, then isoprenoids, squalene and eventually cholesterol. The rate-limiting enzyme HMG-CoA reductase is an important control valve, blocked by cholesterol itself, and acted upon by statins.<sup>20</sup>

**Regulation:** The liver is the center stage in cholesterol homeostasis. It releases VLDL that is broken down to IDL and finally LDL. Cholesterol is transported to peripheral tissues by LDL and reversed transportation of cholesterol to the tissues is performed by HDL. Increased intracellular cholesterol leads to three feedback processes, including: inhibition of HMG-CoA reductase (to slow down synthesis), activation of ACAT (to increase storage as cholesterol esters), and down-regulation of LDL receptors (to slow down uptake).<sup>18-20</sup>

#### D. Atherosclerosis and lipoproteins.

The effects of different lipoproteins on atherosclerosis are different:

LDL-C is the principal vehicle of plasma cholesterol and is regarded as extremely atherogenic. Smaller, dense LDL particles are especially harmful since they can more easily penetrate the wall of the arteries, are more likely to be oxidatively altered, and they are quickly absorbed by the macrophages to create foam cells, which leads to the formation of plaques.<sup>14,15,20</sup>

VLDL and Triglycerides are pro-atherogenic, as well. High TG levels play a part in endothelial dysfunction and promote the development of foam cells.<sup>19,21</sup>

HDL-C, in turn, is protective. It facilitates reverse cholesterol transport, in which excess cholesterol is returned to the liver to be removed. It also has antioxidant and anti-inflammatory action, prevents LDL oxidation, platelet aggregation, and also increases bile acid production.<sup>19,21</sup>

Oxidative stress: This is of central importance in the aetherogenesis. FOAM cell and endothelial damage are centered on oxidized LDL. Vitamin C and E, superoxide dismutase and catalase are antioxidants that alleviate this process.<sup>21</sup>

#### E. Obesity and Dyslipidemia

Obesity is closely related to dyslipidemia and increases the rate of atherosclerosis. The presence of excess adipose tissue leads to the increased secretion of free fatty acids into the circulation, increasing the production of hepatic VLDL, increasing the amount of triglyceride in the blood and decreasing the amount of HDL cholesterol in the blood. Furthermore, insulin resistance, secretion of

pro-inflammatory cytokines (TNF- $\alpha$ , IL-6), leptin resistance, and high C-reactive protein have been linked to obesity and lead to vascular inflammation and the formation of plaques.<sup>22,23</sup> On the other hand, protective molecules like adiponectin and nitric oxide are depleted in obesity and this further contributes to endothelial dysfunction.<sup>22</sup>

### F. Causes of Dyslipidemia

The etiology of dyslipidemia may be generally divided into:

- ✓ Primary (genetic) causes: e.g. familial hypercholesterolemia (or defective LDL receptors), familial hyperchylomicronemia (or deficiency of Apo-CII or lipoprotein lipase).<sup>24</sup>
- ✓ Secondary causes: These are usually related to other medical conditions such as diabetes mellitus, hypothyroidism, nephrotic syndrome, liver disease, alcoholism, and some medications (steroids, oral contraceptives, beta-blockers, diuretics).<sup>24,25</sup>
- ✓ Lifestyle factors: Unhealthy diets rich in fats, obesity, lack of exercise, smoking, and alcohol consumption are major causes of dyslipidemia.<sup>25</sup>
- ✓ Other causes: Heredity, sex and age also have a modifying role in lipid metabolism.<sup>24,25</sup>

### G. Pathophysiology

Complex metabolic disturbances are involved in the development of dyslipidemia. In primary forms, genetic defects of lipoprotein metabolism are inherited and cause the lack of clearance or excess production of lipoproteins. Indicatively, Apo-CII or lipoprotein lipase deficiency inhibits the breakdown of triglycerides, leading to hypertriglyceridemia. In secondary dyslipidemia, systemic diseases such as diabetes cause VLDL production to increase whereas LDL clearance decreases due to hypothyroidism.<sup>24,25</sup>

The most significant pathophysiological disease of dyslipidemia is atherosclerosis. The LDL particles penetrate the endothelium of the arteries, are oxidized and cause the recruitment of macrophages. Such macrophages absorb oxidized LDL and form fatty streaks and foam cells. These eventually develop into fibrous plaques, which constrict the lumen of the vessels, and obstruct blood circulation. Rupture of the plaque can cause acute thrombotic events: myocardial infarction, stroke, peripheral artery disease, and sudden cardiac death.<sup>14,15,20,21</sup>

### H. Investigations<sup>12,13</sup>

Dyslipidemia is assessed mainly by a lipid profile, ideally with fasting to measure the triglycerides. The main parameters include:

- Total cholesterol (TC): Healthy less than 200 mg/dL; Borderline less than 200 and 239; Healthy less than 240.
- The levels are as below: LDL-C: Ideal <100mg/dL; Ideal 100-129; Ideal >160.
- HDL-C: Protection  $\geq$ 60mg/ dL; Low <40mg/ dL.
- Triglycerides (TG): Norm <150 mg/dl; Borderline 150-199; High 200-499; Very high 500 and above.
- Non-HDL cholesterol: This is computed as TC-HDL with the target normally 30 mg/dL above LDL targets.

### I. Management

Dyslipidemia can be treated by lifestyle changes as well as pharmacological therapy.

Lifestyle interventions comprise changes in diet (lowering saturated, trans fats, and cholesterol levels, increasing the intake of fiber, weight loss, exercise, avoiding the use of tobacco and alcohol excessively). These actions are the key to treatment and make drugs more effective.<sup>12,25</sup>

Pharmacological treatment is based on lipid abnormality and cardiovascular risk profile:

- a. The first-line agents are statins (HMG-CoA reductase inhibitors). They lower the LDL-C, increase the HDL-C moderately, and decrease triglycerides. These are atorvastatin, rosuvastatin, and simvastatin.<sup>20</sup>
- b. Cholestyramine is a bile acid sequestrants which binds the salts in the bile and thus decreases cholesterol by increasing the secretion of bile.<sup>19</sup>
- c. Fibrates (gemfibrozil, fenofibrate) are especially effective in hypertriglyceridemia because they stimulate lipoprotein lipase and increase the breakdown of TG.<sup>24</sup>
- d. Nicotinic acid (Niacin) decreases VLDL and increases HDL, but it is poorly tolerated owing to flushing.<sup>24</sup>
- e. Ezetimibe prevents the absorption of intestinal cholesterol and is usually combined with statins.<sup>19</sup>

The therapeutic intervention is to reduce the LDL-C to target levels, increase HDL-C, and prevent the complications of atherosclerosis.

#### 4. UNANI LITERATURE ON FART-E-TADASSUM FID-DAM (HYPERLIPIDEMIA)

##### A. Humoral Theory and Health

Unani medicine relies on the Humoral Theory according to which health is determined by the correct balance and content of four humors (*al-akhlat*): blood (*dam*), yellow bile (*safra*), phlegm (*balgham*), and black bile (*sauda*). Once this balance is disrupted, either quantitatively or qualitatively, the temperament (*mizaj*) of the body is disturbed, and disease then develops. The aetiology and pathology of diseases in Unani medicine is therefore described in terms of an imbalance of humors.<sup>1,2,31</sup>

##### B. Al-Akhlat (Humors)

The moist and fluid products of food during digestion and metabolism are known in the Unani terminology as *khelt* and are referred to as *suarat-e-nauiyah*. They are the source of nutrition, growth and repair and the source of energy to maintain life. Hippocrates, the father of medicine says that it is only when the four humors are in a state of equilibrium (homeostasis) that a state of health is attained. Any change in either quantity or quality results in disease.<sup>26-28,30</sup>

Unani writers put all the colourless body fluids into *balgham* (phlegm). Fat is also in the colourless, moist category since it is both colourless and moist. So, any pathological alteration of *balgham* leads to an abnormality. Thereby, hyperlipidemia (*fart-e-tadassum fid-dam*) is a known phlegmatic disease.<sup>31-33</sup>

##### C. Balgham (phlegm) temperament

*Balgham* is not one, but a combination of colourless body fluids of various properties. Therefore, it is improperly scientifically to attribute to all *balgham* one temperament (*mizaj*).<sup>31-33</sup> According to *Ibn Nafis*, and most physicians, its temperament is *barid-ratab* (cold and moist), as the signs of most phlegmatic diseases indicate coldness (*burudat*) and moistness (*rutubat*).<sup>31</sup> Not all types of phlegm are the same though. An example is *balgham malih* (saline phlegm) is hot and some doctors like *Ali Ibn al-Abbas* and *Abu Sahl al-Masihi* said that sweet phlegm was hot (*har*).<sup>32,33</sup> *Ibn Sina* (Avicenna) and others were not consistent in classifying all phlegm as cold. Therefore, although the overall temperament of phlegm is cold and moist, some of them can be hot.<sup>32-34</sup>

##### D. Abnormal Phlegm and Disease

Phlegm is said to be abnormal either when its quality (*kaifiyat*) is not in its proper state or when it is combined with other humors to produce a different temperament. These abnormalities express themselves as disease symptoms, which may also be identified during clinical and laboratory observations. Excessive and sticky phlegm, such as that, can block the vascular or cardiac system, thus disrupting metabolism. Therefore, we can explain diseases such as hyperlipidemia as diseases caused by abnormal phlegm in Unani medicine.<sup>31-33</sup>

#### ➤ Types of Ghair Tabai Balgham<sup>53,55,56</sup>

##### 1. According to taste:

- ✓ *Balgham Hamiz* – sour, cold & dry.
- ✓ *Balgham Masikh* – tasteless, extremely cold and unripened.
- ✓ *Balgham Afis* – gallic, cold & dry.

- ✓ *Balgham Hulu* – sweet, hot & moist.

## 2. *he Consistency (Qiwam)*

- ✓ *Balgham Mai* – thin, serous.
- ✓ *Balgham Jassi* – very thick.
- ✓ *Balgham Fijji* – raw.
- ✓ *Balgham Mukhati* – mucous.
- ✓ *Balgham Zujaji* – vitreous-like.

## 3. *According to Odour (Bu')*

- ✓ There is one form called, *balgham Muntin*, (fetid phlegm), which was caused by infection and putrefaction.

### E. *Hyperlipidemia (Fart-e-Tadassum Fid-Dam) Unani concept*

Classical Unani thinkers had known about the existence of fatty substances (*dasoomat*) in blood and their toxicity when they occur in excess. Such an amount was commonly referred to as obesity (*saman-e-mufrat*).<sup>1,2,53</sup> Modern science can use the term *dasoomat* to refer to cholesterol and triglycerides. An abnormal increase of these in the blood is named *fart-e-tadassum fid-dam* (hyperlipidemia).<sup>1,2,53,54</sup>

It was seen as healthy to gain weight and moderate levels of fat but beyond that, excess fat was identified as bad. In Unani thinking, *fart-e-tadassum fid-dam* is majorly a phlegmatic illness (*balghami marz*), because phlegm is the dominating kind in the body of obese people. Excessive consumption and overeating of fatty substances were perceived as predisposing factors. Famous doctors like *Razi* and *Ibn Sina* cautioned that oily and fatty food worsens the situation and has dangerous health effects.<sup>1,2</sup>

### F. *Physiology of Fat (Shaham)*

Unani scholars have described fat (*shaham*) in some detail and connected it with health and disease. Fat was a soft and white material that was directly around the fascia and nerves. It has a cold temper and may create heat when metabolized. *Ibn Rushd* regarded fat as a by-product (*fuzla*) of well-formed blood, and thus normal levels of fat were a sign of good health, and an excess of fat was a predisposing factor to disease, implying immaturity of blood.<sup>53,55</sup>

*Ibn Sina* elaborated on the fact that the watery and greasy constituent of the blood is coagulated by cold and that this is the reason why fat can be dissolved by heat. Other thinkers like *Allama Mehmood Aamli* and *Allama Kabeeruddin* explained that fat is naturally cool, but it can be used as a source of energy (*hararat*) and give a lubricating effect and protection to organs.<sup>1,53</sup>

Fat was also reduced into two major categories:<sup>53,55</sup>

- ✓ *Sameen*- Malleable. Soft, semisolid and not likely to solidify.
- ✓ *Shaham* – fat that is thick and hard and can be stuck on organs such as kidneys.

Functions of fat mentioned in Unani literature are that it provides energy, keeps organs moist, aids digestion, shields organs against external stresses and allows organs to retain heat longer because of its oily nature.<sup>53,55</sup>

### G. *Pathophysiology of Fart-e-Tadassum Fid-Dam in Unani*

Pathophysiology of *fart-e-tadassum fid-dam* is also extensively explained in Unani medicine in the framework of obesity (*saman-e-mufrat*). Overabundance of fat spoils the *hararat-e-ghareezia* (innate heat) because coldness (*barudat*) and moistness (*rutubat*) prevail. At the same time, vessel constrictions (*tangi urooq*) also constrict the flow of *rooh* (vital spirit) and nutrition (*ghiza*) to the organs further. This two-fold mechanism of vasoconstriction and impaired natural heat is a vicious circle, as the level of vitality gradually decreases, and the natural state of death occurs.<sup>1-5,31-34</sup>

Obesity also produces a condition of *imtilai kaifiyat* (vascular engorgement), whereby the excessive amount of phlegm and fat raises the viscosity of the blood, raises the blood pressure, and decreases vasodilation. This hinders circulation, reduces oxygen and nutrient supply and hastens the deterioration of essential heat, thus predisposing to cardiovascular disease and early mortality.<sup>1-5,31-34</sup>

### H. *Usool-e-Ilaj*<sup>1-4,35-37</sup>

#### 1. *Tadeel-e-Mizaj (Correction of Temperament)*

- ✓ Restoration of balance between *Dam*, *Balgham*, *Safra*, and *Sauda*.
- ✓ Lifestyle modification: moderate physical activity, balanced diet, avoidance of cold-moist foods.

#### 2. *Tanqiya-e-Mawad (Evacuation of Morbid Matter)*

- ✓ *Munzij wa Mushil therapy*: Administration of *Munzij* (concoctive drugs) to prepare morbid matter, followed by *Mushil* (purgatives) for evacuation.
- ✓ Examples: *Ispaghul*, *Turanjabeen*, *Sana makki*.

### 3. *Tahleel-e-Riyah wa Shahm (Resolution of Fat & Lipids)*

- ✓ Use of *Muhallil-e-Shaham* (lipolytic) and *Muhallil-e-Riyah* drugs to reduce fat deposition and dissolve thick humors.
- ✓ Examples: *Zanjabeel* (*Zingiber officinale*), *Zeera* (*Cuminum cyminum*), *Ikli-ul-Malik* (*Tribulus terrestris*).

### 4. *Islah-e-Dam (Correction and Purification of Blood)*

- ✓ Use of *Musaffi-e-Dam* (blood purifiers) to improve quality of blood and prevent atherogenic deposition.
- ✓ Examples: *Unnab* (*Ziziphus jujuba*), *Gul-e-Surkh* (*Rosa damascena*), *Chiraita* (*Swertia chirata*).

### 5. *Taqwiyat-e-Aaza (Strengthening of Vital Organs)*

- ✓ *Taqwiyat-e-Qalb* (strengthening of heart): Drugs like *Arjun* (*Terminalia arjuna*), *Sandal safed* (*Santalum album*).
- ✓ *Taqwiyat-e-Kabid* (strengthening of liver): Drugs like *Kasni* (*Cichorium intybus*), *Berge-Kasni*.
- ✓ *Taqwiyat-e-Medda* (support of digestive system) to enhance metabolism.

### 6. *Ilaj bil Ghiza (Dietotherapy)*

- ✓ Adoption of *Ghiza-e-Latif* (light diet) that is low in saturated fats and rich in fibrous content.
- ✓ Avoidance of *Ghiza-e-Saqeel* (heavy, cold, moist foods) like mutton, oily and fried items.
- ✓ Encouragement of barley, oats, legumes, fruits, and vegetables.

### 7. *Ilaj bil Dawa (Pharmacotherapy)*

- ✓ Use of hypolipidemic and hepatoprotective Unani drugs such as:

- *Arjun* (*Terminalia arjuna*)
- *Asgandh* (*Withania somnifera*)
- *Lehsan* (*Allium sativum*)
- *Ajwain* (*Trachyspermum ammi*)
- *Hanzal* (*Citrullus colocynthis*)

### 8. *Ilaj bil Tadbeer (Regimenal Therapy)*

- ✓ *Hammam* (Turkish bath/steam bath)
- ✓ *Riyazat* (exercise) for weight reduction and improved circulation
- ✓ *Hijama* (cupping) for *Tanqiya-e-Dam*
- ✓ *Dalak* (massage) with stimulating oils

Table no 1: **DRUGS USED IN FARTE TADASSUM FID – DAM (DYSLIPIDEMIA)** <sup>38-52</sup>

| Unani Name              | Botanical Name                   | Reported Action   |
|-------------------------|----------------------------------|---|
| Arjun                   | <i>Terminalia arjuna</i>         | Cardio-protective, reduces TC, LDL, TG; raises HDL        |
| Asl-us-Soos (Liquorice) | <i>Glycyrrhiza glabra</i>        | Antioxidant, hepatoprotective, regulates lipid metabolism |
| Berge Neem              | <i>Azadirachta indica</i>        | Antihyperlipidemic, anti-atherosclerotic                  |
| Berge Kasoos            | <i>Cuscuta reflexa</i>           | Improves lipid profile, hepatoprotective                  |
| Chiraita                | <i>Swertia chirayita</i>         | Bitter tonic, reduces lipid accumulation                  |
| Guggul                  | <i>Commiphora mukul</i>          | Potent hypolipidemic, reduces TC, LDL, TG                 |
| Hulba (Methi)           | <i>Trigonella foenum-graecum</i> | Decreases TC, TG; increases HDL                           |
| Ispaghul                | <i>Plantago ovata</i>            | Soluble fiber lowers LDL & TC                             |
| Kalonji                 | <i>Nigella sativa</i>            | Reduces TC, TG, LDL; raises HDL                           |
| Lasun (Lehsun)          | <i>Allium sativum</i>            | Hypocholesterolemic, anti-atherosclerotic                 |
| Basal (Onion)           | <i>Allium cepa</i>               | Reduces TC, TG; antioxidant                               |
| Luffah                  | <i>Luffa cylindrica</i>          | Lipid-lowering, antioxidant                               |
| Sarpagandha             | <i>Rauwolfia serpentina</i>      | Cardioprotective, antihypertensive                        |
| Sana Makki              | <i>Cassia angustifolia</i>       | Laxative, reduces cholesterol absorption                  |
| Zanjabeel (Ginger)      | <i>Zingiber officinale</i>       | Reduces TG, TC, LDL; antioxidant                          |

Table no 2: **COMPARATIVE UNDERSTANDING OF FARTE TADASSUM FIDDAM (DYSLIPIDEMIA) IN UNANI AND MODERN MEDICINE**

| Aspect                              | Unani Medicine (Fart-e-Tadassum Fid-Dam) <sup>1-5,28,29,31-34,53,55,56</sup>   | Modern Medicine (Hyperlipidemia) <sup>10-13,18,19,24</sup>  |
|-------------------------------------|--|---|
| Definition                          | Excessive fatness ( <i>dasoomat</i> ) of blood due to dominance of <i>balgham</i> (cold & moist humor), causing heaviness and vascular obstruction ( <i>sudda</i> ).   | Elevated levels of plasma lipids: cholesterol, triglycerides, LDL-C, and/or reduced HDL-C; a major risk factor for CVD.   |
| Etiology                            | Improper diet ( <i>ghiza musammina</i> – oily, fatty, cold, moist foods), sedentary lifestyle, weak <i>hararat-e-ghareezia</i> (digestive/metabolic heat), heredity, stress.   | Genetic (familial hypercholesterolemia, ApoB/LDL receptor defects); secondary causes (diabetes, hypothyroidism, CKD, obesity, alcohol, drugs, diet rich in saturated fats, physical inactivity).  |
| Pathophysiology                     | Imbalance of humors ( <i>su-e-mizaj</i> ); excess <i>balgham</i> and <i>dasoomat</i> in blood → vascular engorgement ( <i>imtila</i> ), narrowing ( <i>tangi urooq</i> ), and obstruction ( <i>sudda</i> ). Weak metabolism due to reduced <i>hararat-e-ghareezia</i> .  | Excess LDL-C enters vascular intima → oxidation → foam cell formation → fatty streaks → atherosclerotic plaque → narrowing, rupture, thrombosis. Linked with insulin resistance and inflammation.   |
| Clinical Features                   | Heaviness, fatigue, sluggish digestion ( <i>zo'f-e-hazm</i> ), palpitations ( <i>khafaqan</i> ), breathlessness, fullness of vessels ( <i>imtila</i> ), lethargy, sudden obstruction ( <i>sudda qalb</i> ).  | Often asymptomatic early. Signs: xanthomas, xanthelasma, corneal arcus. Advanced: hypertension, angina, MI, stroke, pancreatitis (in hypertriglyceridemia).   |
| Complications                       | <i>Sudda qalb</i> (cardiac obstruction), <i>za'f quwwat</i> (weakening of vital organs), respiratory distress, premature aging, sudden death.  | Atherosclerosis, ischemic heart disease, cerebrovascular accidents, metabolic syndrome, type 2 diabetes, pancreatitis, premature CVD mortality.   |
| Preventive & Therapeutic Principles | <b>Ilaj-bil-ghiza</b> (dietotherapy): avoid fatty, oily foods; use light, warm, dry diet (barley, lentils, vinegar, garlic). <b>Ilaj-bil-dawa</b> (pharmacotherapy): hypolipidemic herbs ( <i>guggul, lehsan, isabgol, zanjabeel, turmeric</i> ). <b>Ilaj-bil-tadbeer</b> (regimenal therapy): exercise ( <i>riyazat</i> ), massage, steam bath, hijamah (cupping), fasd (venesection). <b>Ilaj-bil-nafsiyat</b> (psychotherapy): moderation, stress control, lifestyle balance. | <b>Lifestyle modifications:</b> low-fat diet, exercise, weight control, smoking/alcohol restriction. <b>Drugs:</b> statins (first-line), fibrates (for triglycerides), bile acid sequestrants, ezetimibe, niacin, PCSK9 inhibitors, omega-3 fatty acids. <b>Advanced:</b> LDL apheresis, gene therapies for familial cases. |
| Conceptual Correlation              | <i>Balgham</i> ↔ Plasma lipids <i>Dasoomat fid-Dam</i> ↔ Hyperlipidemia <i>Imtila / Tangi urooq</i> ↔ Atherosclerosis <i>Hararat-e-ghareezia</i> ↔ Basal metabolism <i>Sudda</i> ↔ Vascular obstruction  | Explained in molecular, biochemical, and pathophysiological terms (lipid metabolism, vascular inflammation, oxidative stress).  |

## I. DISCUSSION

According to Unani medicine, fatness of blood in excess is referred to as *Fart-e-Tadassum Fid-Dam*; it is mainly caused by excess of *balgham* (cold and moist humor), weak *hararat-e-ghareezia* (innate metabolic heat), and improper diet and lifestyle. The state leads to heaviness, blockage of the veins or arteries of the body (*sudda*), slow digestion and palpitations and, ultimately, fatigue of the body organs. Prevention and treatment methods include diet control (*Ilaj-bil-Ghiza*), light, warm and dry food control, pharmacotherapy (*Ilaj-bil-Dawa*) of hypolipidemic herbs, garlic, guggul, isabgol and regimenal therapies (*Ilaj-bil-Tadbeer*); exercise, massage, hijamah, and fasd.

In medicine today, dyslipidemia is described as an increase in total cholesterol, LDL-C, triglycerides or a decrease in HDL-C, which is caused by both inherited or secondary factors such as diabetes, hypothyroidism, obesity, alcohol and sedentary lifestyle. It is mostly asymptomatic during the initial stages and develops into xanthomas, atherosclerosis, ischemic heart disease, cerebrovascular disease and metabolic complication.

Management involves lifestyle change interventions and pharmacological interventions including statins, fibrates, bile acid sequestrants, and PCSK9 inhibitors.

In theory, Unani terminology such as *balgham* (plasma lipids), *dasoomat fid-dam* (hyperlipidemia), *imtila* (vascular obstruction and atherosclerosis), and *hararat-e-ghareezia* (basal metabolism) can be compared. In that way, both systems acknowledge the key role of lipid imbalance in blood and its impact on cardiovascular health, albeit with different language.

## J. CONCLUSION

Excessive fat in the blood is the leading cause of cardiovascular disease by both Unani and modern medicine though by different paradigms. Although Unani finds the cause of *Fart-e-Tadassum Fid-Dam* as imbalance of humor, poor metabolism and improper diet, contemporary science attributes dyslipidemia because of lipid metabolism and inflammation of the vascular system. Both put stress on dietary and lifestyle as two important preventive steps, which are enhanced by pharmacological medications in modern medicine and herbal and regimenal medicines in Unani. A composite intervention based on both systems incorporating evidence-based practices could offer more comprehensive care of dyslipidemia that can be safely managed.

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