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THERAPEUTICAL APPRAISAL OF BADRANJBOYA (MELISSA OFFICINALIS) IN FASADE TASHAHHUM FID DAM(DYSLIPIDAEMIA) IN COMPARISON TO ATORVASTATIN-A CLINICAL STUDY

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ABSTRACT
There is no doubt that dyslipidaemia is one of the most common risk factor of cardio vascular diseases. This vulnerable disease is essentially an abnormal concentration of lipids or lipoproteins in blood. Increased level of cholesterol is responsible for atherogenesis, which ultimately leads to development of cardiovascular, cerebro vascular and peripheral vascular diseases. This disease has a significant contribution towards mortality and morbidity rates and also poses economic downfall among patients. These days the treatment of dyslipidaemia is lipid lowering agents with life style intervention, while lipid lowering agents are producing various side effects. In Unani system of medicine several drugs are being used as lipid lowering agents, which are comparatively safe. However, such drugs are still not validated on scientific parameters. Two different drugs were selected and present study contemplated as "Therapeutic evaluation of Bādranjboya (Melissa officinalis) In Fasāde Tashahhum Fid Dam (Dyslipidaemia) In Comparison to Atorvastatin" Thus, a clinical trial was conducted with the objective of providing safe and effective drug in the management of dyslipidaemia.

KEYWORDS: Dyslipidaemia; Melissa officinalis; Bādranjboya; Quwate Tabaiya.

I. INTRODUCTION OF DYSLIPIDAEMIA
Dyslipidaemia is a metabolic disorder of lipid and lipoproteins, as well as increased concentration of total cholesterol, triglycerides, LDL cholesterol and decreased concentration of HDL cholesterol.[1] Lipids are a group of heterogeneous metabolically active substances constantly moving in the circulation and existing in estate of dynamic equilibrium between peripheral tissue, gastrointestinal tract and liver.[2] Almost all the lipoproteins are formed in the liver; the liver has active enzyme system for synthesizing triacylglycerol, phospholipids, cholesterol, plasma lipoproteins and for converting fatty acid to ketone bodies. In the process of metabolism LPL (Lipoprotein lipase) enzyme, secreted from liver cells is responsible for catabolic activity upon lipid and lipoprotein to maintain the equilibrium of lipid concentrations. In other words, Metabolic disorders were one of the great challenge of 20th and now in 21th century and it is directly related with the changes of life style pattern of human being in the era of computerisation, electronics as well as machine, where humans are bound around it. The metabolic disorders are slow developing and mostly silent killers and their prognosis leads to vital organ damage and multi organ-system failure at the end of disease. The Dyslipidaemia is not a single morbid condition but it invites several others to make a derangement at the level of vascular changes, visceral changes including derangement in carbohydrate, protein, fat even of minerals and water. Dyslipidaemia is considered as an iceberg of many metabolic disorders including metabolic syndrome, as a result, Non-alcoholic Fatty liver disease (NAFLD), Diabetes mellitus, Hypertension, CAD may emerge as a co-morbid factor.[3,4] Simane mufrit is another well-known disease since Greco Arab period and was first described by Buqrat (Hippocrates), later on other Unani physicians like Jalinoos,[5] Ibn-e-Sina,[6] Zakariya Razi,[7] Rabban Tabri[8] etc, mentioned Simane Mufrit in their treatises.

They defined etiological factors, symptoms, signs, and complications of Simane Mufrit expansively. Ibn-e-Sina especially pointed out that obese people are more prone to develop cardiac and cerebral complications like stroke, syncope, coma, palpitation, breathlessness, concealed haemorrhage and sudden death.[6,9] As per the Unani philosophy, Simane Mufrit develops due to increased rutoobat and buroodat leading to imbalance of humours in the body and increases tendency of accumulation of Akhlate fasida particularly Maddae balghamia.

Objectives of study

- To evaluate the safety of Bādranjboya in the management of Dyslipidaemia on modern Scientific parameters in comparison to "Atorvastatin"
- To validate the effect of Bādranjboya (Melissa officinalis) in Fasāde Tashahhum Fid Dam (Dyslipidaemia) in the management of Dyslipidaemic patients.

II. Unani concept of disease

Dyslipidaemia has been categorized under Amraze Balghamiya. Because in Dyslipidemic persons, Balgham (Phlegm) is more than blood proportionally, hence they have Barid Mizaj.[47] Unani physicians have also mentioned that excessive Ratoobat and Baroodat (Wet and Cold), Increase the Sameen and Shaham in the body Presence of Maayeat and Dasumat in the blood. Which is more Ratab than blood. Described Maddi causes of sameen and shaham. This Dasumat (Fatty particles) is solidified by excessive Baroodat (Coldness) in body, therefore Barid Ratab (Cold and Wet) person has more shaham and sameen. Generally fat deposits on abdomen. Dyslipidaemia causes narrowing of vessels, and due to this, vessels transport less amountof Naseem (Oxygen) to the tissues, leading to decrease Hararate Ghariziya of the body which may result in Fasade Mizaj and infection. If Hararate Ghariziya is completelylost it may cause death.

The word dyslipidaemia is derived from Greek word the etymology; Dys, Difficult and haima, blood, so dyslipidaemia is defined as any abnormality in or abnormal amount of lipid and lipoprotein in blood. Any defect in lipoprotein metabolism, e.g. increased cholesterol, triglyceride, LDL cholesterol and decreased HDL cholesterol.[1] Elevation in one or more of the lipoprotein fractions constitutes hyperlipoproteinemia, some authors used the term hyperlipidaemia or dyslipidaemia instead of hyperlipoproteinemia.

III. Asbab (Etiology)

Following are the causes of Fasāde Tashahhum Fid Dam (Dyslipidaemia) which have been described by the various Unani Physicians.

- Khilqi and Mauroosi (Hereditary and Congenital)
- Kasrate Farhat wa Musarrat (Excess of joy)
- Rahat wa Sakoon (Excessive rest and lack of exercise)
- Excessive use of Martoob wa Duhuniyat (excessive use of fatty diet and oils)
- Kasrate ghiza (excessive eating)
- Baroodate Mizaj (Cold Temperament)
- Kasrate Sharab noshi (excessive consumption of alcohol)
- Excessive sleeping.

Some various causes, which produce buroodat, are as follows

- Excessive activity disperses the innate heat
- Excessive repose which produce cold by suppressing the innate heat
- Cold food and drinks
- Marked reduction in food
- Cold medicine
- Occupation which produce cold
- Excessive depletion by involving loss of the material for innate heat and dispersionof vital force
- Cooling application
- Severe prolong compression blocking the passage of innate heat
- Excessive dilatation of pores of body causing dispersion of innate heat
- Immaturity of akhlat results in dominance of balgham

IV. Introduction of drug melissa officinalis (Badranjboya)

The drug Bādranjboya consist of dried leaves of Melissa officinalis Linn. It is aperennial plant, which belongs to the family of Labiate (Lamiaceae). It is mostly cultivated in Mediterranean region and native to Europe, Northern Africaand West Asia. In India the plant is found in hilly areas of Punjab, Kashmir, Bengal, Bihar, Kumaon, Rajasthan, Deccan, and Konkan. It occurs during winter season. It is called lemon balm, bee balm, Melissa, sweet balm. It has a lemony flavourand fragrance. Traditionally this herb was used for longevity, Dyslipidaemia, healing wound, relaxing the heart, treating tooth ache, nowadays it is used in anxiety, mild depression, restlessness, irritability, indigestion, acidity, nausea, bloating and colicky pains, and cold sores. It is also called as a hormonal herb due to its anti-thyroid activity.

V. METHODOLOGY

A. Criteria for selection of subject

Inclusion criteria

- Diagnosed patients of Dyslipidaemia.
- Patients irrespective of gender
- Total Cholesterol $\geq$ 240mg /dl
- Triglycerides < 499 mg / dl (High)
- LDL 160-189 mg / dl
- HDL < 40 mg / dl in men and <50 in woman
- Age group between 20-50 years of age
- Patients able to participate in the study who follow the protocol
- Known cases of DM type -II with Dyslipidaemia

Fasting blood sugar (FBS) $\geq$ 126 mg/dl - < 150mg/dl

Post Prandial blood sugar (PPBS) $\geq$ 140 mg/dl - < 250mg/dl[3]

- Normotensives (< 130 – 80 mm of Hg)
- Patients who follow the protocol

Exclusion criteria

Known cases of Dyslipidaemia, with disease history of > 3yrs

»	Total Cholesterol	≤ 240 mg / dl
»	Triglycerides	> 499 mg / dl (High)
»	LDL	> 189 mg / dl
»	HDL	> 70 mg / dl

B. Subjective parameters:*

Palpitation * Breathlessness *Joint pain

C. Objective parameters

- Lipid Profile
- Total Cholesterol
- Triglycerides
- Low Density Lipoprotein (LDL)
- High Density Lipoprotein (HDL)

D. Study design: An Open labelled, Randomized, Comparative, clinical study

Sample size

- 40 patients.
- 20 patients in test group
- 20 patients in control group

E. Method of collection of data

Collection of data was made through clinical history, physical examination andlaboratory investigations which were recorded in the CRF.

F. Allocation of subjects

The 40 patients were randomly allocated by using computer generated table into two groups 20 for test (Group A) and 20 for standard control (Group B) respectively.

G. Duration of protocol therapy

The treatment period in both test and control groups was determined for 45 days.

H. Criteria for selection of drugs

A single drug Bādranjboya (Mellisa Officinalis) was selected, having functions as: Muffarah (Exhilarent), Muqawwi Qalb (Cardiotonic), Muffateh Sudad (Deobstruent), Muqawwi Dimagh (Brain tonic), Muqawwi Meda (Stomachic),

I. Method of Preparation, Dosage and Mode of administrationof test drug

The Leaves of test drug Bādranjboya (Mellisa Officinalis) were used in the form of Joshānda (Decoction) to test group patients. Drug was provided by the Unani pharmacy of Regional Research Institute of Unani Medicine (RRIUM) Srinagar. Before preparing the Joshanda, the drug was properly identified to determine its originality, and cleaned, then 25-gram Bādranjboya leaves was soaked in 200 ml of water throughout night, laterthe next morning water containing soaked leaves was boiled till the water remained halfof its quantity. Then the semi tepid decoction was advised to take in an empty stomach orally in the morning.25 grams of dried leaves packed in zip packed polythene bags were given to the patientsfor 15 days in each follow up.

J. Administration of control drug

The control drug was Atorvastatin 10 mg (prepared by certified company LUPIN LTD. Bhasmey Block, Duga ilaka, East Sikkim – 737132, INDIA given dose was) one Tablet Twice a day, after meal for the period of 45 days was given.

K. Follow up during treatment

Duration of 45 days study was divided into 4 visits of follow up including baseline. i.e. $0, 15^{th}$, 30^{th}, 45^{th} days. At every visit, patients were enquired about the progression or regression in their symptoms and were subjected to examination for the assessment of clinical findings.

L. Criteria for safety evaluation

Any adverse event or reaction appearing during the study either in test or control group was recorded.

VI. RESULT AND OBSERVATIONS

Table 1: Showing Age distribution of Test and Control group.

Age (years)	Test		Control		P-value
	No.	% age	No.	% age	
<35	4	20	2	10	0.081
35-39	6	30	3	15	
40-44	3	15	5	25	
45-49	6	30	8	40	
50	1	5	2	10	
Total	20	100	20	100	
Mean ±S.D	40±7.02		44.10±7.75		

Table No. 01 We observe that there is an insignificant difference between Test and Control group with respectto Age group of patients because the p-value is 0.081

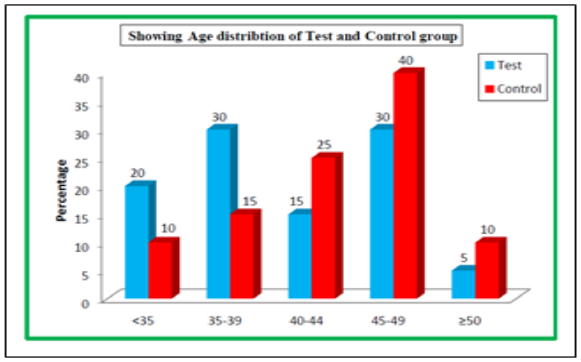

Table 2: Showing distribution of patients as per Sex.

Sex	Test		Control		p value
	No.	% age	No.	% age	
Male	17	85	11	55	0.0820
Female	3	15	9	45	
Total	20	100	20	100	

Table 2, Displays the Gender distribution of patients in Test and Control group. Since p value is 0.082, which means the difference is insignificant.

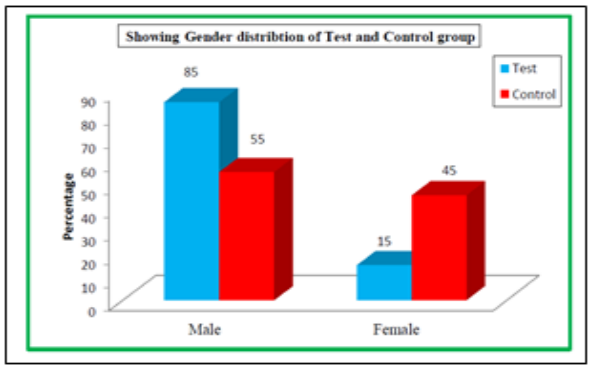

Table 3: Showing distribution of patients as per Marital status.

Maritalstatus	Test		Control		p value
	No.	% age	No.	% age	
Married	18	90	20	100	0.4870
Unmarried	2	10	0	0	
Total	20	100	20	100	
Fisher Exact Test					

Table 3, Shows the distribution of Marital status among Test and Control group. Clearly there is no significant difference between Test and Control group with respect to Marital status.

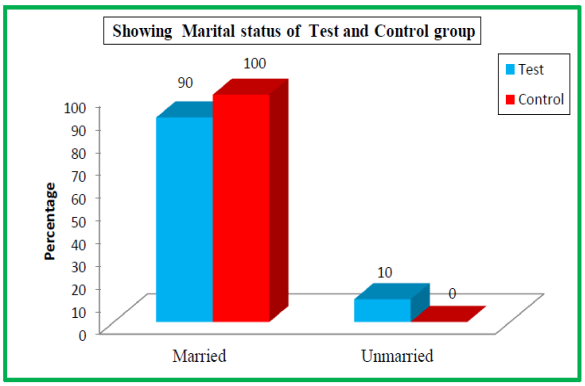

Table 4: Showing distribution of patients as per Socio economic status.					
SES	**Test**		**Control**		**p-value (Monte Carlo)**
	No.	**%age**	**No**	**%age**	
Upper Class	0	0	1	5	0.6790
Upper Middle	5	25	2	10	
Lower Middle	8	40	10	50	
Upper Lower Class	4	20	5	25	
Lower Class	3	15	2	10	
Total	20	100	20	100	

From table 4, we observe that there is no significant difference between Test and Control group with respect to Socio-economic status of patients.

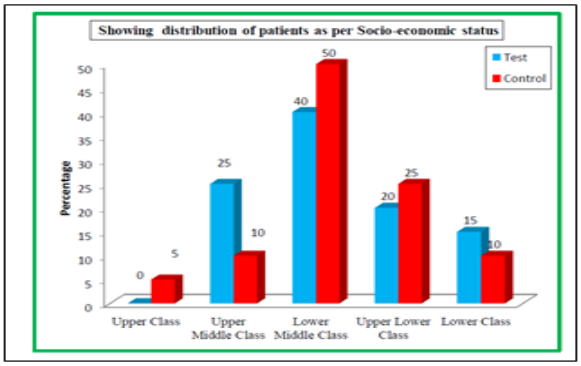

Table 5: Showing distribution of patients with respect to Family history.					
Familyhistory	**Test**		**Control**		**P-value**
	No.	**% age**	**No.**	**% age**	
Present	7	35	6	30	0.7360
Absent	13	65	14	70	
Total	20	100	20	100	
Test applied; Pearsonian Chi-square					

Table 5, Displays that there is no significant difference (p value is 0.736) between Test and Control group with respect to Family history of subjects.

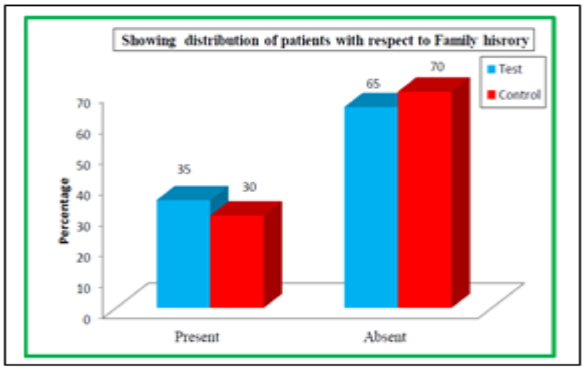

Table 6: Showing distribution of patients as per Dietary habit.					
Dietary habit	**Test**		**Control**		**p-value**
	No.	**%age**	**No.**	**%age**	
Mixed	17	85	20	100	0.2310
Vegetarian	3	15	0	0	
Total	20	100	20	100	
Test applied: Fisher exact test					

Table 6: Shows the distribution of Dietary habit among patients, we observe that there is nosignificant difference between the two groups because p-value is 0.231

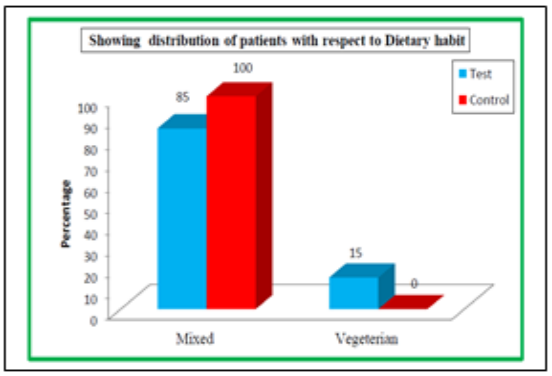

Table 7: Showing distribution of patients with respect to Mizaj.					
Mizaj	**Test**		**Control**		**p value**
	No	**%age**	**No.**	**%age**	
Damvi	6	30	8	40	0.5970
Balgami	7	35	8	40	
Safravi	5	25	4	20	
Saudavi	2	10	0	0	
Total	20	100	20	100	
Test applied: Fisher exact test					

Table 7, Reveals an insignificant difference between Test and Control group with respect toMizaj of patients since p value is 0.597

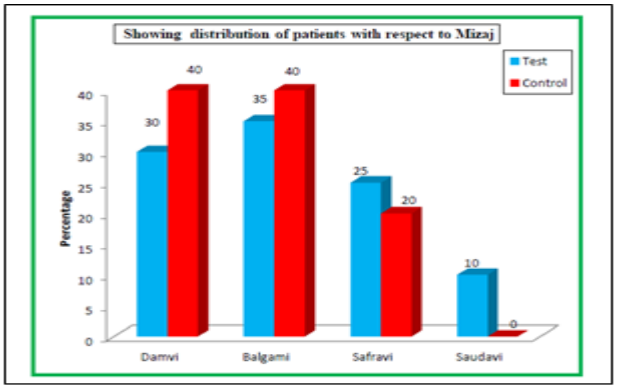

Table 8: Showing distribution of patients as per Palpitation in Test and Control group

Status of Palpitation	Test				Control				P-value between Groups
	BT		AT		BT		AT		
	No.	%age	No.	%age	No.	%age	No.	%age	
Normal	0	0	18	90	0	0	17	85	0.6326
Mild	0	0	2	10	0	0	3	15	
Moderate	4	20	0	0	2	10	0	0	
Severe	16	80	0	0	18	90	0	0	
Total	20	100	20	100	20	100	20	100	
P-value within Groups	<0.001*				<0.001*				

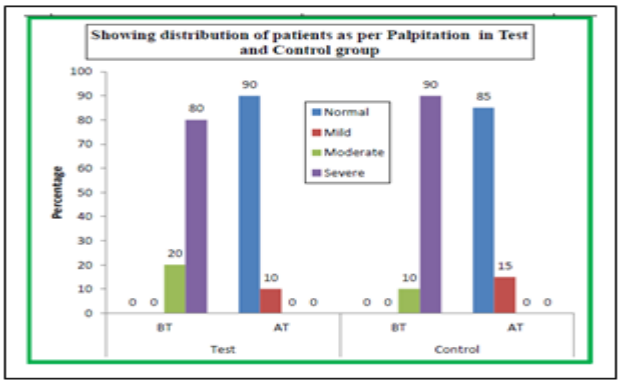

Table 09: Comparison of Joint pain before and after the treatment with in Test and Control group.

Group	Follow-up	Mean	N	Std. Deviation	p value
Test	BT	4.35	20	1.08942	<0.001
	AT	1	20	0	
Control	BT	4.05	20	0.88704	<0.001
	AT	1	20	0	

The above table displays within group comparison in Test and Control group. We observe that there is strong significant difference before and after the treatment in both the groups, implying The both the treatments perform excellently equally well in curing the Joint pain.

Table 10: Comparison between Test and Control group with respect toObjective parameters.

Objective Parameters	Group	N	Mean	Std. Deviation	p value
Sr.Chol BT	Test Group	20	174.95	48.63	0.745
	Control Group	20	179.50	38.79	
Sr.Chol AT	Test Group	20	182.30	55.34	0.255
	Control Group	20	162.55	52.70	
Sr. T.G BT	Test Group	20	261.08	198.39	0.934
	Control Group	20	256.77	116.42	

Sr. TG.AT	Test Group	20	223.91	81.76	0.74
	Control Group	20	214.97	87.61	
Sr. HDL BT	Test Group	20	51.11	9.31	0.413
	Control Group	20	53.65	10.08	
Sr. HDL AT	Test Group	20	51.52	8.00	0.705
	Control Group	20	52.63	10.18	
Sr. LDL BT	Test Group	20	144.74	29.39	0.821
	Control Group	20	142.64	29.09	
Sr. LDL AT	Test Group	20	137.51	34.75	0.011
	Control Group	20	106.92	37.76	

Table 10, Shows descriptive statistics of Objective parameters for Test and Control group at base line and last follow up. We observe that both the treatments are almost equally effective because there is no significant difference between the two groups as p value is >0.05.

Table 11: Comparison of Objective parameters before and after thetreatment with in Test group.

Objective parameters		Mean	N	Std. Deviation	P value
Sr. Chol (mg/dl)	BT	174.9500	20	48.63016	0.650
	AT	182.3000	20	55.33829	
Sr. TGL (mg/dl)	BT	261.0750	20	198.38891	0.378
	AT	223.9100	20	81.75849	
Sr. HDL (mg/dl)	BT	71.3050	20	89.13655	0.331
	AT	51.5200	20	7.99701	
Sr. LDL (mg/dl)	BT	144.7400	20	29.39453	0.391
	AT	137.5050	20	34.74641	

Table 11, Shows that there is an insignificant difference before and after the treatment with respect to Objective parameters with in the Test group since p value <0.001

Table 12: Comparison of Objective parameters before and after the treatment within Control group.

Objective parameters		Mean	N	Std. Deviation	P value
Sr. Chol (mg/dl)	BT	179.5000	20	38.78755	0.162
	AT	162.5500	20	52.70022	
Sr. TGL (mg/dl)	BT	256.7700	20	116.41720	**0.090**
	AT	214.9650	20	87.60699	
Sr. HDL (mg/dl)	BT	53.6450	20	10.08472	0.695
	AT	52.6250	20	10.17566	
Sr. LDL (mg/dl)	BT	142.6350	20	29.08594	0.002
	AT	106.9200	20	37.76355	

Table 12, Shows that there is an insignificant difference before and after the treatment with in the Control group with respect to Objective parameters except for Sr. LDL.

Table 13: Comparison of Safety parameters before and after the treatmentwith in Test group.

Safety parameters		Mean	N	Std. Deviation	p-value
Hb%	BT	14.8000	20	1.67929	.02344
	AT	15.6550	20	1.39867	
WBC	BT	6376.0215	20	2648.94002	0.72491
	AT	6141.0000	20	2042.84327	
N	BT	61.0000	20	10.24952	0.05695
	AT	56.2000	20	8.30726	
L	BT	35.3000	20	9.69590	0.06847
	AT	39.7500	20	8.28997	
M	BT	1.8000	20	1.28145	.76630
	AT	1.9000	20	.30779	
ESR	BT	24.8500	20	17.59867	0.14900
	AT	18.5500	20	14.24402	
FBS	BT	103.1650	20	40.27536	0.18800

	AT	94.0600	20	16.31288	
PPBS	BT	126.4250	20	40.19502	0.08100
	AT	137.2550	20	52.57357	
TSH	BT	10.4400	20	34.75920	0.33210
	AT	3.1505	20	1.84779	
B.UREA(mg/dl)	BT	32.5400	20	7.56108	0.92500
	AT	32.2900	20	11.08840	
Sr. CREAT(mg/dl)	BT	.9385	20	.14521	.77900
	AT	.9310	20	.16889	
Sr. Bil (mg/dl)	BT	.8505	20	.47452	.96100
	AT	.8455	20	.46225	
SGOT(U/L)	BT	33.4900	20	20.55040	0.03000
	AT	26.2750	20	9.95611	
SGPT(U/L)	BT	118.7650	20	267.45996	0.17700
	AT	37.5950	20	17.41855	
A. PHOS (U/L	BT	115.2500	20	43.39400	0.02200
	AT	102.0500	20	33.72251	

Table 13; Shows that there is an insignificant difference before and after the treatment with in theTest group with respect to Safety parameters

Table 14: Comparison of safety parameters before and after thetreatment with in control group.

Safety parameters		Mean	N	Std.Deviation	p-value
Hb%	BT	5698.242	20	3519.75849	.11600
	AT	6759.450	20	2265.80567	
WBC	BT	58.2500	20	10.37647	0.91800
	AT	58.0500	20	8.02939	
N	BT	38.1500	20	9.89032	0.95900
	AT	38.0500	20	8.06209	
L	BT	1.9000	20	1.48324	0.55100
	AT	1.7000	20	.57124	
M	BT	.2000	20	.52315	.10400
	AT	0.0000	20	0.00000	
ESR	BT	40.4000	20	32.38811	0.00300
	AT	20.3000	20	20.26431	
FBS	BT	116.8500	20	43.66636	0.54400
	AT	111.1650	20	39.98413	
PPBS	BT	164.7150	20	74.37135	0.31600
	AT	149.6050	20	68.02778	
TSH	BT	3.8730	20	1.84169	0.42000
	AT	4.2335	20	1.91397	
B.UREA (mg/dl)	BT	30.7550	20	9.35895	0.17200
	AT	27.8700	20	8.30536	
Sr. CREAT (mg/dl)	BT	.8805	20	.20930	.18200
	AT	.8550	20	.22421	
Sr. Bil (mg/dl)	BT	.8600	20	.45046	.23320
	AT	.9625	20	.57100	
SGOT(U/L)	BT	32.4050	20	20.69537	0.91200
	AT	32.0300	20	18.15889	
SGPT(U/L)	BT	53.2700	20	41.91539	0.60700
	AT	49.9050	20	38.38637	
A. PHOS (U/L	BT	129.8000	20	49.61176	0.32900
	AT	123.7500	20	60.26509	

Table 14; Shows that there is an insignificant difference before and after the treatment with in the Control group with respect to safety parameters.

VII. DISCUSSION

The clinical study was conducted to evaluate the efficacy of Bādranjboya (Melissa officinalis) in Dyslipidaemia. This was an open labelled, randomized, Comparative,

pre and post clinical study, with 40 patients (20 in test group and 20 in control group) belonging to 20-50 years of age, irrespective of gender. out of 47 patients 40 completed, 45 days protocol, 7 patients (3 in test and 4 in control) were dropped out. The test group was treated with Joshandae (Decoction) Bādranjboya (25 gm of dried leaves) empty stomach in the morning once a day orally, whereas control group was managed, with one tablet of Atorvastatin 10 mg twice a day orally for 45 days. Subjective parameter (joint pain) were assessed based on Visual Analogue Scale (VAS) on every 15th day of follow up and Objective parameters were carried out before and after treatment in each group. This study stretched from July 2019 to December 2020. In this study a total no of 40 patients participated, we observed that there is an insignificant difference between test and control group with respect to age distribution. However, the maximum number of patients about 40% were falling in the age group of (45-49) years in control group while as in test group the age group (35-39) and (45- 49) were most prevalent. This finding supports the description shows that more prevalence of Dyslipidaemia found in age group of 45-49 years, second high incidence was found in age group of 35-39 years (Table No.1, Figure No.1). This is suggested by A.M. Sawami et al (2008) that the prevalence of Dyslipidaemia is high in 45-49 years of patients[20] Prevalence over the age 60 years is also high suggested by M Estariet al (2009).[21]

This study reveals that Dyslipidaemia is more common in male patients, as they were 28 (70%) and only 12 (30%) patients were female (Table No. 2, Figure No 2). Present data correlates with the observation of David C. Goff et al, A.M. Sawami et al (2008), M Estari et al (2009) and IC Health New Delhi Data base (2004).[20,21,22]

The association of disease with reference to marital status concern, this study Discussion more evidence A total no of 38 (95%) patients were married and 2(5%) unmarried, (Table No. 3, Figure No 3). This finding is in consideration with the etiological concept as mentioned by various Unani authors as Fasāde Tashahhum Fid Dam (Dyslipidaemia) is higher due to highly intake of oily diets after married ultimately more married people will be sufferers as is evident from this study.

The highest number 18 (45%) patients were from lower middle class (III), 9 (22.5%) belonged to Upper lower class (IV), rest 7 (17.5%) belonged to upper middle class (II), 5 (12.5%) patients were from lower class (V) and 1 (2.5%) belonged to Upper class (I) respectively in socio economic strata (Table No. 4, Figure No 4). Present data may be influenced due to more patients belonging to lower middle class visited to RRIUM Hospital. Apart from that we observed that there is no significant difference between test and control group with respect to socio-economic status of patients.

According to family history of Dyslipidaemia, out of 40 patients 27 (67.5%) had no family history and 13 (32.5%) have positive family history (Table No. 5, Figure No 5). This data shows that the incidence of Positive family history (primary Dyslipidaemia) is fewer. Present data may be interpreted as incidence of primary Dyslipidaemia is varying according to type of gene involved in mutation. The conditions are found worldwide and are generally diagnosed in childhood.Familial dysbetalipoproteinemia occurs in approximately one in 10,000 persons and is found worldwide. Familial hypercholesterolemia is found worldwide. Heterozygous familial hypercholesterolemia occurs in about one in 500 persons worldwide. Homozygous familial hypercholesterolemia occurs with a frequency of about one in a million person worldwide. Familial combined hyperlipidaemia occurs in about one in 200 persons worldwide, an estimated 15% of patients with premature coronary artery disease have FCHL. Polygenic hypercholesterolemia is relatively common, occurring up to 5% of the general population. Homozygotes familial dysbetalipoproteinemia approximately 0.5% of the general population.

According to dietary distribution 37 (92.5%) patients had mixed diet habit and only 3 (7.5%) patients were vegetarian (Table No. 6, Figure No 6). Consuming more fatty dietis a factor of Dyslipidaemia. The distribution of dietary habit among patients, we observed that there is no significant difference between the two groups because p-value is 0.2310 It is suggested that more fatty diet causes lipoprotein disorders by Stephen J Mc Phee and Maxine A. Papadakis (2010).

Out of 40 patients 15 (37.5%) have Balghami Mizaj while 14 (35%) patients were of Damvi Mizaj, 9 (22.5%) patients were of Safravi Mizaj and 2 (0.5%) patients were of Saudavi Mizaj. (Table No. 7, Figure No 7). It reveals an insignificant difference betweentest and control group with respect to mizaj of patients since p value is 0.5970.

It is mentioned that Baroodate Jigar produces more Khilte Balgham, which provides nutrition to all organs. Ultimately temperament of organs becomes Balghami (Phlegmatic).The nature of disease is progressive, and asymptomatic. It may be diagnosed accidentally or during routine investigations. It also depends upon the awareness of individual. Palpitation and breathlessness was assessed on the severity of disease and extent of involvement were assessed by using (Arbitrarily Scale) graded as severe, moderate, mild and absent/normal and was coded as 3+, 2+, 1+ and 0 respectively. Chi-square test was employed for inter group comparison of categorical variables and for intra group analysis of categorical variable with more than two levels we applied McNemar- Bowker's test. It was evident that there is a no significant difference between the groupswith respect to palpitation and breathlessness, however; there is a strong significant difference before and after the treatment in both test and control group because the

severity of palpitation and breathlessness among patients in both the groups significantly improves to normality after the treatment. For the significance of pain in test and control group we applied paired t-test was applied for intra-group analysis. We observed that both the treatments are almost equally effective because there is a clear significant difference before and after the treatment within both test and control group as p value is <0.05. To analyse the difference between test and control group with respect pain we used students independent test and observed that both the treatments are insignificantly equally effective.

Joints pain was assessed on the severity of disease and extent of involvement were assessed by using VAS (Visual Analogue Scale) consists of a line, often 10 cm long, which was graded as severe, moderate, mild and absent. The mean score of joints pain was calculated in both control and test group on 0^{th}, 15^{th}, 30^{th} and 45^{th} day as Comparison between test and control group at base line and after the treatment with respect to subjective parameters (Table No. 8, Figure No 8). The mean scores of both groups were compared statistically using Student's independent t-test for inter group. (p value is >0.346).

Paired t-test was applied for intra-group analysis. It was found that the descriptive statistics of subjective parameter like pain for test and control group at base line and last follow up. We observed that both the treatments are almost equally effective because there is no significant difference between the two groups as p value is >0.05 at 45^{th} day.Comparison of subjective parameters before and after the treatment within test group (Table No 9), In test group there is a significant difference before and after the treatment with respect to subjective parameters within the test group since (p value <0.001) at 45^{th} day with respect to test day 0, and also significant difference (p value <0.001) at 45^{th} day with respect to test day 15.

Comparison of subjective parameters before and after the treatment within control group (Table No 10), In control group there is a significant difference before and after the treatment with respect to subjective parameters within the control group since p value is <0.001 at 45^{th} day with respect to control day 0, and also significant difference (p value <0.001) at 45^{th} day with respect to control day 15.

Present result on palpitation, breathlessness and joints pain reveals that the both treatments are almost equally effective because there is no significant difference between the two groups as p value is >0.05 at 45^{th} day on all subjective parameters. High level of lipoproteins develops cardiovascular disease symptoms and peripheral vascular symptoms.

Serum Cholesterol was assessed before and after treatment in both test and control group. The Mean ± SD score of test group was (174.95 ± 48.63) on base line and (182.30 ± 55.34) on 45^{th} day. In control group Mean ± SD score was (179.50 ±38.79) on baseline and (162.55 ± 52.70) on 45^{th} day respectively (Table No. 11). We analysedintra group data comparison by Paired-t test for inter group comparison we applied student independent t-test. We observed that Serum Cholesterol level in both the groups at base line and found that they were comparable with a p-value of 0.745. However, we also observed that there was an insignificant difference between the two groups with respect to the effect on Cholesterol levels after the treatment as the p-value was >0.05.

Serum Triglyceride was assessed before and after treatment in both test and control group. The Mean ± SD score of test group was (261.08 ± 198.39) on base line and (223.91 ± 81.76) on 45^{th} day. In control group Mean ± SD score was (256.77 ± 116.42) on baseline and (214.97 ± 87.61) on 45^{th} day respectively (Table No. 11). We analysedintra group data comparison by Paired-t test for inter group comparison we applied student independent t-test. We observed that Serum Triglyceride level in both the groups at base line and found that they were comparable with a p-value of 0.934. However, we also observed that there was an insignificant difference between the two groups with respect to the effect on Triglyceride levels after the treatment as the p- value was >0.05.

HDL cholesterol was assessed before and after treatment in both test and control group. The Mean ± SD score of test group was (51.11 ± 9.31) on base line and (51.52 ± 8.00)on 45^{th} day. In control group Mean ± SD score was (53.65 ± 10.08) on baseline and (52.63 ± 10.18) on 45^{th} day respectively (Table No. 11). We analysed intra group data comparison by Paired-t test for inter group comparison we applied student independent t-test. We observed that HDL. Cholesterol level in both the groups at base line and found that they were comparable with a p-value of 0.413. However, we also observed that there was an insignificant difference between the two groups with respect to the effect on HDL. cholesterol levels after the treatment as the p-value was >0.05.

LDL cholesterol was assessed before and after treatment in both test and control group. The Mean ± SD score of test group was (144.74 ± 29.39) on base line and (137.51 ± 34.75) on 45^{th} day. In control group Mean ± SD score was (142.64 ± 29.09) on baselineand (106.92 ± 37.76) on 45^{th} day respectively (Table No. 11). We analysed intra group data comparison by Paired-t test for inter group comparison we applied student independent t-test. We observed that LDL. cholesterol level in both the groups at baseline and found that they were comparable with a p-value of 0.821. However, we also observed that there was an insignificant difference between the two groups with respect to the effect on LDL. cholesterol levels after the treatment as the p-value was>0.05.

In this study Bādranjboya has effect on most of the parameters which is evident from statistical analysis, since the p-value is <0.001* for almost all the parameters including palpitation, breathlessness and joint pain, however, there was an insignificant difference before and after the treatment with respect to some parameters like Cholesterol, Triglyceride and HDL in both test and control group except for LDL showed some improvement in control group. In case of safety parameters, we observed they remained within normal range before and after the treatment which indicates that both the treatments are safe to patients. However, haemoglobin level among patients in test group improved which is evident from the statistical analysis as the p value is <0.001*.

A study has reported that essential oil extracted from Melissa officinalis leaves contributes to a lipid lowering action in cholesterol-fed rabbit.A study has reported protective effect of Melissa officinalis extract on liver of hyperlipidaemic effect. The result showed that Melissa officinalis extract decreased total cholesterol, total lipid ALT, AST and ALP levels in serum and LPO (tissue lipidperoxidation) levels in liver tissue. As a result, it was suggested that Melissa officinalis L. extract exerted an hypolipidemic effect and showed a protective effect on the liver of hyperlipidaemic rats. From the above scientific studies.

VIII. SUMMARY

This study entitled "Therapeutic evaluation of Bādranjboya (Melissa officinalis) In Fasāde Tashahhum Fid Dam (Dyslipidaemia). In Comparison to Atorvastatin" was conducted in Regional Research Institute of Unani Medicine Hospital after obtaining permission from institutional ethical committee. 40 diagnosed patients were enrolled in the study. Patients between the age group of 20 to 50 years from both sexes were registered for the study and made to complete study as per protocol. Diagnosis was made on the basis of history, clinical examination and investigations. The severity of disease and extent of involvement was assessed either by arbitrarily scale or by using VAS (Visual Analogue Scale). Diagnosed patients were divided in two groups, group A, group B. Group A patients were treated with Joshandae Bādranjboya, (Melissa officinalis) (25 gm of dried leaves) empty stomach in the morning once a day orally, whereas control group was managed, with one tablet of Atorvastatin 10 mg twice a day orally for 45 days. all groups of patients were treated for 15 days. Subjective and objective parameters were assessed and noted in Case Report Proforma.After completion of study, the result was analysed and observed that both the treatments are almost equally effective because there is no significant difference between the two groups as p value is >0.05. Overall, improvement was observed in test group, without any clinically and statistically significant side effects or toxicity. The compliance to the treatment was found good. These results conclude that the test drug is quite safe in thetreatment of Dyslipidaemia.

Cases were selected on the basis of clinical diagnosis, inclusion and exclusion criteria in the research protocol. The protocol duration was 45 days. Cases were randomly assigned in two groups; test group comprising 20 patients, while control group consisting 20 patients. The efficacy of the both test and control drugs were assessed on the basis of clinical examination and laboratory investigations. Findings of effectiveness of both drugs were recorded in CRF and the inference was made by appropriate statistical analysis. Summary of demographic data, effects of treatment on different subjective and objective parameters are as follows:

IX. Demographic data

A. Age

In this study a total no of 40 patients participated, we observed that there is aninsignificant difference between test and control group with respect to age distribution. However, the maximum number of patients about 40% were falling in the age group of(45-49) years in control group while as in test group the age group (35-39) and (45-49)were most prevalent.

B. Sex

The incidence of Dyslipidaemia with 28 (70%) was observed in male patients and 12 (30%) was observed in female patients in both groups.

C. Dietary habit

According to dietary distribution 37 (92.5%) patients had mixed diet habit and only 3 (7.5%) patients were vegetarian.

D. Marital status

38 (95%) patients were married. 2 (5%) of unmarried in both groups.

E. Socio-Economic status

1 (2.5%) were belonged with Upper class (I) 7 (17.5%) were belonged with Upper middle class (II) 18 (45%) patients were from lower middle class (III) 9 (22.5%) patients from Upper lower class (IV) 5 (12.5%) patients from lower class (V) espectively in socio economic Strata in both groups.

F. Family history

According to family history of Dyslipidaemia, out of 40 patients 27 (67.5%) had no family history and 13 (32.5%) have positive family history.

G. Mizaj

15 (37.5%) Maximum patients were found to be having Balghami Mizaj 14 (35%) patients were found to be having Damvi Mizaj, 9 (22.5%) patients were found to be having Safravi Mizaj (5%) patients were found to be having Saudawi Mizaj with respectively.

X. SUBJECTIVE PARAMETERS

A. Effect on Palpitation and Breathlessness

Palpitation was categorised in severity, moderate, mild and normal class using arbitrarily scale Chi-square test was employed for inter group comparison of categorical variables and for intra group analysis of categorical variable with more than two levels we applied McNemar-Bowker's test. It was evident that there is a no significant difference between the groups with respect to palpitation and breathlessness (p-value >0.05), however; there is a strong significant difference before and after the treatment in both test and control group (P-value<0.001*) because the severity of palpitation among patients in both the groups significantly improves to normality after the treatment.

B. Effect on joints pain

The mean scores of both groups were compared statistically, it was found that both the groups were comparable at 0 day. However, there was a significant decline in average VAS value after the treatment (45th day) in both the groups because the p value is (<0.001*) in each group. Evidently both the treatments are equally effective in respectof effect on joints pain.

XI. OBJECTIVE PARAMETERS

A. Effect on serum cholesterol

We analysed the Serum Cholesterol level in both the groups at base line and observed that they were comparable with a p-value of 0.745. However, we also observed that there was an insignificant difference between the two groups with respect to the effect on Cholesterol levels after the treatment as the p-value was >0.05.

B. Effect on triglyceride

We analysed the Triglyceride level in both the groups at base line and observed that they were comparable with a p-value of 0.934. However, we also observed that there was an insignificant difference between the two groups with respect to the effect on Triglyceride levels after the treatment as the p-value was >0.05.

C. Effect on HDL

We analysed the HDL level in both the groups at base line and observed that they were comparable with a p-value of 0.413. However, we also observed that there was an insignificant difference between the two groups with respect to the effect on HDL levels after the treatment as the p-value was >0.05.

D. Effect on LDL

We analysed the LDL level in both the groups at base line and observed that they were comparable with a p-value of 0.821. However, we also observed that there was an insignificant difference between the two groups with respect to the effect on LDL levels after the treatment as the p-value was >0.05.

E. Safety assessment

The safety and toxicity of both drugs was evaluated by complete routine blood, LFT, KFT, blood sugar, ECG and urine investigation. Investigations done were Hb%, TLC, DLC, ESR, Total bilirubin, S. Urea, S. Creatinine, FBS, PPBS, ECG and urine forroutine and microscopic examinations. No adverse effects were observed after the study in both groups. Otherwise this study proved that both test and control drugs are safe for the patients. It is evident from the above described observations the Bādranjboya is found significantly effective in improving some subjective and some objective parameters assame as the control drug. With all these qualities, the Unani drug may be proclaimed as a safe and effective in the treatment of Dyslipidaemia.

XII. CONCLUSION

This study entitled "Therapeutic evaluation of Bādranjboya (Melissa officinalis) in Fasāde Tashahhum Fid Dam (Dyslipidaemia) in Comparison to Atorvastatin" was conducted at RRIUM, Hospital, University of Kashmir, Srinagar. The study was a randomized, comparative, pre and post clinical in nature aimed to evaluate the efficacy of drug in the management of Dyslipidaemia. The scientifically chosen sample size 40 was divided in two groups; 20 patients were randomly allocated to test group and 20 patients were randomly allocated in control group. Test group was treated with Joshandae Bādranjboya (25 gm of dried leaves) empty stomach in the morning once a day orally, whereas control group was managed, with one tablet of Atorvastatin 10 mg twice a day orally for 45 days. All patients who qualified the inclusion criteria were included in the study. Treatment protocol was followed for 15 days in both groups, subjective and objective parameters were recorded in each follow up i.e., 0, 15th, 30th and 45th day, The severity of disease and extent of involvement was assessed either by arbitrarily scale or by using VAS (Visual Analogue Scale).

The overall effect of the Bādranjboya was found quite encouraging in the treatment of Dyslipidaemia. Drastic improvement in subjective parameters like palpitation, breathlessness and joints pain was seen in patients placed in both test and control group as the same is evident from statistical analysis, however, some parameters like; Cholesterol, Triglyceride, HDL and LDL did not show significant improvement in either groups. In conclusion we observed that both the treatments are almost equally effective on parameters like palpitation, breathlessness, joint pain and equally not effective on Cholesterol, Triglyceride, HDL and LDL. However, for test group patients there was a significant improvement in haemoglobin level of patients since the p-value corresponding to haemoglobin level (before and after) is <0.001*. Interestingly, we observed that in test group, safety parameters remained under normal range after the administration of Bādranjboya which rules out any possible side effects or toxicity of the drug. The compliance to the treatment was found good. These

results conclude that the test drug is quite safe in the treatment of Dyslipidaemia. However, long term study on larger sample size is required for furtherexploration of the effects of Bādranjboya, and also to determine their mechanism of action with modified methodology.

REFERENCES

1. Concise dictionary of modern medicine: Mc Graw Hill Companies, 2002.
2. Siddharth N. Shah. API Textbook of Medicine. Mumbai: The Association of Physicians of India, 2008; 8, 2: 951-959.
3. Jonathan Q. Purnell, M.D. ACP Medicine, 2005; 604-615.
4. Rushad Patell 1, Rupal Dosi2, et al. Non-alcoholic fatty liver disease in Dyslipidaemia Journal of clinical and Diagnostic Research, 2014; 8(1): 62-66.
5. Qamari AMH. Ghina Muna (Urdu translation Minhaj-ul- Ilaj). New Delhi: Central Council for Research in Unani Medicine, 2008; 384-90.
6. Ibne Sina Al Qānun Fit Tib, (Urdu translated by Kantoori GH). New Delhi: IdaraKitabul Shifa, 2007; 1, 4: 124,145-146, 216, 755, 765, 1445.
7. Razi AMBZ. Kitabul Mansoori (Urdu translation). New Delhi: Central Council for Research in Unani Medicine. Ministry of Health and Family Welfare, 1991; 132, 133, 136, 171, 391, 200.
8. Tabari A R. Firdausul Hikmat. New Delhi: Idara Kitabul Shifa, 2010; 112-13.
9. Ismail Jurjani. Zakheera Khawarzam Shahi. New Delhi: Idara Kitabul Shifa, 2010; 8: 24-25.
10. Colledge NR, Walker BR, Ralston SH. Davidson's principles & practice of Medicine. Landon: Churchill Livingston, 2010; 21, 518: 577-579.
11. Leadingham JGG, Warrel DA. Concise Oxford Textbook of Medicine. USA: Oxford University Press, 2000; 1: 47-49.
12. Warnell DA, Cox TM, Firth JD. Oxford Text of Medicine. London: Oxford press, 2010; 4, 15.1.2:2.798-2.800.
13. Natalya M. Ananyeva, Diana V. Kouiavskaia, Midori Shima and Evgueni L. Saenko.Intrinsic pathway of blood coagulation contributes to thrombogenicity of atherosclerotic plaque. Blood, 2002; 15, 99: 4475-4485.
14. Humes HD. Kelly's Textbook of Internal Medicine. USA: Lippincott Williams & Wilkins, 2000; 4: 99-113.
15. Brunton Laurence L. Goodman & Gilman's the pharmacological basis of therapeutics. USA: Mc Graw Hill, 2001; 10: 984-994.
16. Eric J. Topol. Textbook Cardiovascular Medicine. Edition. Lippincott Williams & Wilkins, 2007; 3: 68-72.
17. Brunton Laurence L. Goodman & Gilman's the pharmacological basis of therapeutics. USA: Mc Graw Hill, 2001; 10: 984-995.
18. Ahmad SI. Al Umoor Al Tabi'yah. New Delhi: Central Council for Research in Unani Medicine, 1980; 162-167, 174-175.
19. Majoosi A I A. Kāmilus Sanāah. New Delhi: Idara Kitabush Shifa, 2010; 1,2: 336-37, 441.
20. Sawami AM, Shetty Dhanashri, Mankeshwar R, Ashvaid Tester F. Prevalence of Dyslipidaemia in young adult Indian population. JAPI, 2008; 56: 99-102.
21. Estari M, Reddy AS, Bhikshapati T, Satyanarayan J, Venkanna L, Reddy MK. The Investigation of serum lipids and prevalence of Dyslipidaemia in urban adult population of Warangal district, Andhra Pradesh, India. Biology and Medicine, 2009; 1(2): 61-65.
22. Anonymous. National cardio vascular disease database. Ministry of Health &Family welfare, Govt. of India and WHO. SE/04/233208.
23. Mooradian Arshag D. Dyslipidaemia in type 2 diabetes mellitus. Endocrinology and metabolism, 2009; 5, 3: 150-157.
24. Longe Jacquelinel and Blanchfield Deirdres. The Gale encyclopaedia of medicine USA: Farmington Hills, 2002; 2, 2: 1709.
25. Altaf Rabia, Asmawi Mohammad Zaini, Dewa Aidiahmad and Umar Muhammad Ihtisham. Sources and possible mechanisms of action of important phytoconstituents with cardiovascular properties. African Journal of Pharmacy and Pharmacology, 2012; 6(9): 563-580.
26. Majoosi. Kamilus sanah New Delhi: Idara Kitabul Shifa, 2010; 1, 2: 42, 54, 176-177, 373-377.
27. Kabeeruddin M. Tarjuma wa sharah Kulliyate Qanoon. Delhi: Barqi press, 1930; 1, 11, 192: 354-356.
28. Kabeeruddin M. Kulliyate Nafeesi. New Delhi Idara Kitabul Shifa, 1954; 12, 60, 62, 67,78, 114, 453.
29. Razi AMBZ. Al Hawi fit Tib. (Urdu translation by Central Council for Research in Unani Medicine. Vol-6 New Delhi: Ministry of Health and Family Welfare, Govt. of India, 1997; 9-45, 187-188, 191, 195.
30. A Jalinoos, 'Fusool-e-Buqrat ma Talkhees-e-Jalinoos' Translated by Ghulam Husain kintoori, Munshi Nawal Kishore, Lucknow, 1903; 5-6, 16, 44.
31. Al Qamri Abul Mansoor A, 'Ghina muna ma Tarjuma Minhajul ilaj' 1255H PP, 316-322.
32. Tabri R, Firdausal Hikmat, Idara kitab-us-Shifa, kucha chelan Darya Ganj, New Delhi, 2010; 112-113.
33. Razi M. Bin Zakariya, 'Alhawi-fit-Tibb', Central Council for Researchin Unani Medicine, New Delhi, 1999; 6: 183-239.
34. Majūsi AA. Kamil al-Sanaah, Lucknow: Munshi Naval Kishore, 1889; 2, 42: 176-177.
35. Ibne zuhr. Kitabut Taisir Fil Mudawat wa Tadbir. New Delhi: Central Council for Research in Unani Medicine (CCRUM), Ministry of Health and Family Welfare, 1986; 53-54.
36. Ibne Hubul.M.A.A.A Al Mukhtarat Fit Tib. New Delhi: Central Council for Research in Unani Medicine, Ministry of Health and Family Welfare, 2007; 1, 2: 51, 85, 122-23, 118, 138, 185-86, 292.

37. Ibne Rushd. Kitabul Kulliyat. New Delhi: Central Council for Research in Unani Medicine, Ministry of Health and Family Welfare, 1987; 2, 147-150, 220-221, 232, 277, 294, 301, 304, 320.
38. Nafees I. Moa'lajate Nafeesi. NM ed: Lucknow: Munshi Naval Kishore; 1324 Hijri: New Delhi: Elsevier Inc, 2004; 1258-1267, 537-538.
39. Jurjani S. Ismail 'Zakheera Khawarzam Shahi', Translated by Hakeem Hadi Hasan, Idara Kitab-us-Shifa, Kucha chelan Darya Ganj New Delhi, 2010; 8: 24-31.
40. Iqsarai Jamaluddin, 'Sharah-e-Mojizul Qanoon' Translated by Hakeem Ayoob Israili, Munshi Nawal Kishore, Lucknow, 1907; 572-576.