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AN EXPLORATORY STUDY OF BADRANJBOYA (MELISSA OFFICINALIS) IN FASADE TASHAHHUM FID-DAM (DYSLIPIDAEMIA) IN COMPARISON TO ATORVASTATIN-A RESEARCH ARTICLE

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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.

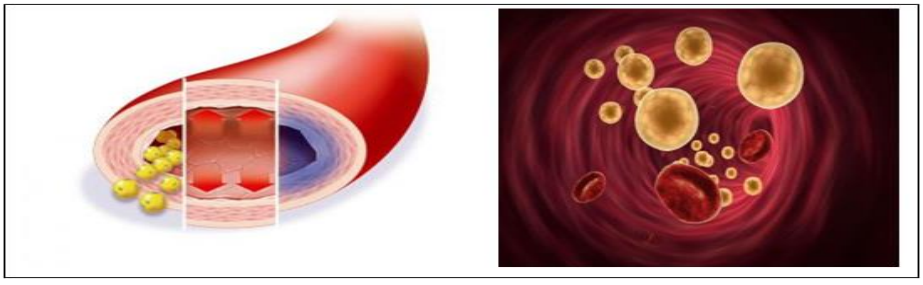

Fig. 01: Anatomy of blood vessels in dyslipidaemia.

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. It is an established fact that hyperlipidaemia is associated with Simane Mufrit and Atherosclerosis.[6] The conventional view of atherosclerosis is of a slow and progressive process consisting of lipid deposition and accumulation in large and medium-sized arteries. This lipid accumulation will form what is called an atherosclerotic plaque or lesion. The process develops over decades and leads to progressive reduction of the arterial lumen that ultimately might lead to ischemia of the irrigated organs. The possible final consequencebeing Myocardial and Cerebral infarction, Peripheral vascular diseases and stroke. Although still recognized by the low-density lipoprotein receptor, minimally modified low-density lipoprotein can stimulate the release of macrophage colony-stimulating factor and monocyte chemoattractant protein-1 from endothelial cells, these facilitate monocyte recruitment and their differentiation into tissue macrophages. Minimally modified low-density lipoprotein adheres to matrix proteins of the arterial wall, where it undergoes more extensive oxidation.[10]

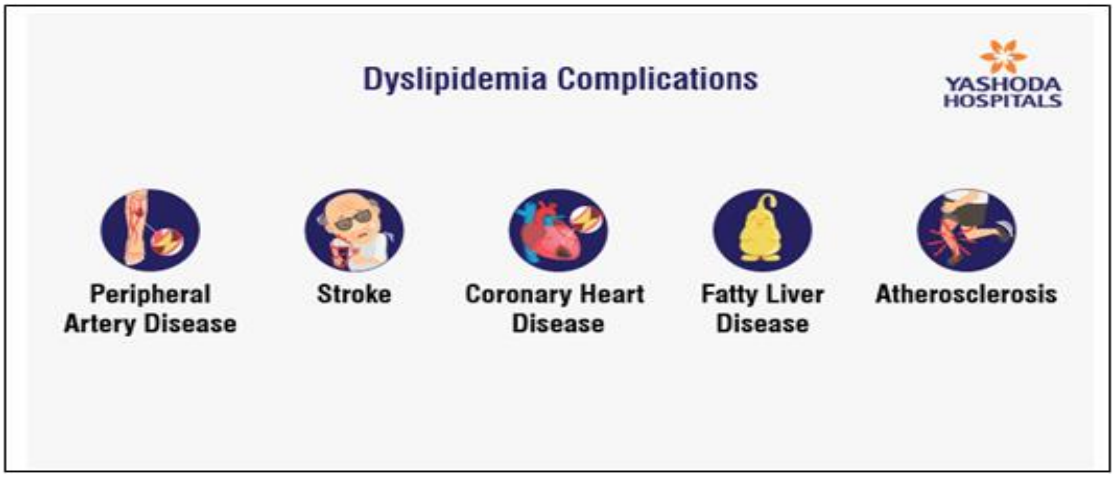

Fig. 02: Complications of dyslipidemia.

Free radicals are produced from macrophages and from nitric oxide derived from endothelial cells. This is compounded by the products of tobacco smoke and by homocysteine. Highly oxidized/modified low-density lipoprotein is characterized by changes not only of the lipid but also of the protein portion of low-density lipoprotein, leading to loss of recognition by the low-density lipoprotein receptor. Thus, oxidized low-density lipoprotein becomes the major legend for the scavenger receptor family expressed in the macrophages accumulating at the site of the injury to the vessel wall. This shift in receptor recognition leads to uptake of oxidized low-density lipoprotein by receptors, not regulated by the cholesterol content of the cell. The result is massive accumulation of cholesteryl esters in the macrophages, giving the cytoplasm its characteristic foamy appearance and transforming the macrophage into a foam cell.[11] Hypertension accelerates atherogenesis by activating genes in response to increased sheer stress, the products of which perturb vascular tone and promote the accumulation of smooth muscle cells. Hypertension also increases the formation of hydrogen peroxide and free radicals that worsen oxidative damage, reduces the formation of nitric oxide by the endothelium, and increases leucocytes adhesion. In Diabetes mellitus, Hyperglycaemia may promote non-enzymatic glycation of low-density lipoprotein, which may initiate atherosclerosis in the same way as oxidative modified low-density lipoprotein. A high plasma homocysteine concentration is toxic to endothelium, decreases the availability of nitric oxide, and has prothrombotic activity. Current strategies for the management of Dyslipidaemia include diet, exercise, drug therapy (hypolipidaemic, antithrombolytic and antioxidant drugs).[12,13]

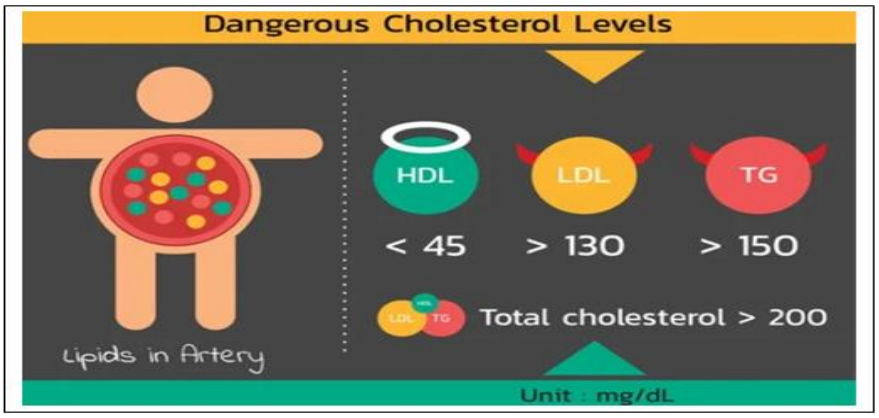

Fig. 03: Range of blood cholesterol in dyslipidemia.

The aim of treatment of atherosclerosis in Dyslipidaemia is to reduce body weight and risk factor modification that can be achieved by non- pharmacological therapy such as decreasing daily calorie intake, increase in physical activity and indeed, life style modification is helpful for most patients but in several circumstances pharmacological management of atherosclerosis is inevitable. nowadays many treatment options are available for lowering cholesterol, such as Lovastatin, Atorvastatin, Simvastatin, Clofibrate, Bezafibrate and Niacin etc. are widely prescribed drugs in mainstream Medicine. But long-term use of these drugs produces several side effects, such as Hepatotoxicity, Myopathy, Dyspepsia, Renal failure and Cholelithiasis.[11,14] Currently, available anti hyperlipidaemic drugs or lipid lowering agents; Atorvastatin, Simvastatin, pravastatin and lovastatin etc. are modestly effective in some subjects but administration of these pharmacological agents produces several life-threatening adverse effects or side effects along with antilipidemic activity.[15,16] These are hepatotoxicity, myopathy, renal dysfunction, dyspepsia, bloating, constipation, flushing, pruritus of the face and upper trunk, skin rashes, acanthosis nigricans, urticaria, hair loss, myalgias, fatigue, headache, impotence, and anaemia. Hence, there is pressing need for novel therapeutic agents for better management to prevent from adverse effect of conventional therapy. As a result, there is a growing need to develop effective, safe and well-tolerated drugs to reduce atherosclerotic complications in Dyslipidaemia.[17]

Dyslipidemia occurs when someone has abnormal levels of lipids in their blood. While the term describes a wide range of conditions, the most common forms of dyslipidemia involve:

- High levels of low-density lipoproteins (LDL), or bad cholesterol
- Low levels of high-density lipoproteins (HDL), or good cholesterol
- High levels of triglycerides
- High cholesterol, which refers to high LDL and triglyceride levels

Lipids, or fats, are building blocks of life and provide energy to cells. Lipids include:

- **LDL cholesterol**, which is considered bad because it can cause plaques to form in the blood vessels.
- **HDL cholesterol**, which is regarded as good because it can help to remove LDL from the blood.
- **Triglycerides**, which develop when calories are not burned right away and are stored in fat cells.

Healthy blood lipid levels naturally vary from person to person. However, people with high levels of LDL and triglycerides or very low HDL levels tend to have a higher risk of developing atherosclerosis. Atherosclerosis develops when hard, fatty deposits called plaques accumulate in blood vessels, making it difficult for blood to flow.

II. SIGN AND SYMPTOMS

Unless it is severe, most people with dyslipidemia are unaware that they have it. A doctor will usually diagnose dyslipidemia during a routine blood test or a test for another condition. Severe or untreated dyslipidemia can lead to other conditions, including coronary artery disease (CAD) and peripheral artery disease (PAD). Both CAD and PAD can cause serious health complications, including heart attacks and strokes. Common symptoms of these conditions include:

- Leg pain, especially when walking or standing
- Chest pain
- Tightness or pressure in the chest and shortness of breath
- Pain, tightness, and pressure in the neck, jaw, shoulders, and back
- indigestion and heartburn
- Sleep problems and daytime exhaustion
- Dizziness
- Heart palpitations
- Cold sweats
- Vomiting and nausea
- Swelling in the legs, ankles, feet, stomach, and veins of the neck
- Fainting

These symptoms may get worse with activity or stress and get better when a person rests.

Talk with a doctor about chest pain, especially any of the above symptoms accompany it.

Anyone who experiences severe chest pain, dizziness, and fainting, or problems breathing should seek emergency care.

Fig. 04: Melissa officinalis.

III. INTRODUCTION OF BĀDRANJBOYA

The drug Bādranjboya consists 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.

IV. METHODOLOGY

The present clinical study entitled as "Therapeutic evaluation of Badranjboya (Mellisa Officinalis) In Fasade Tashahhum Fid Dam (Dyslipidaemia) in Comparison to Atorvastatin" has been conducted at the Department of Moalajat in Regional Research Institute of Unani Medicine, University of Kashmir, Srinagar. Apart from clinical examination with detailed history of the disease and necessary haematological, biochemical investigations, Clinical symptoms, history and investigations were recorded on the prescribed Case Report Form (CRF) designed for the study with specific inclusion criterion. After the ethical clearance, clinical study was started by enrolling eligible patients into test and control groups by randomisation with the help of computer generated method from the OPD of Regional Research Institute of Unani Medicine Hospital.

V. CRITERIA FOR SELECTION OF SUBJECT

A. Inclusion criteria

- Diagnosed patients of Dyslipidaemia.
- Patients irrespective of gender
- Total Cholesterol> 240mg /dl
- Triglycerides< 499 mg / dl (High)
- LDL160-189 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)> 126 mg/dl - < 150mg/dl
- Post Prandial blood sugar (PPBS)> 140 mg/dl - < 250mg/dl[3]
- Normotensives (< 130 – 80 mm of Hg)
- Patients who follow the protocol

B. Subjective parameters

- Palpitation
- Breathlessness
- Joint pain

C. Objective parameters

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

VI. RESULT AND DISCUSSION

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

Age (years)	Test		Control		P-value
	No.	%	No.	%	
<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		

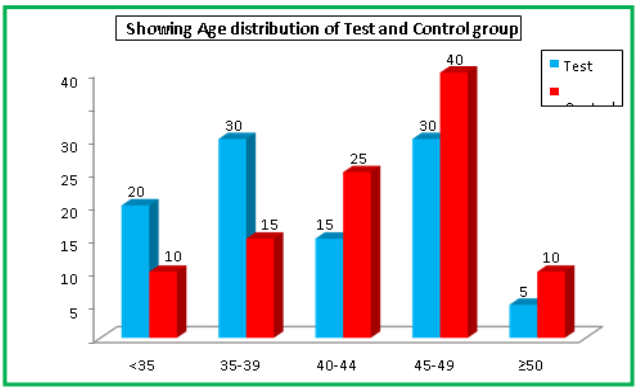

Table 2: Showing distribution of patients as per Sex.					
Sex	Test		Control		p value
	No.	%	No.	%	
Male	17	85	11	55	0.0820
Female	3	15	9	45	
Total	20	100	20	100	

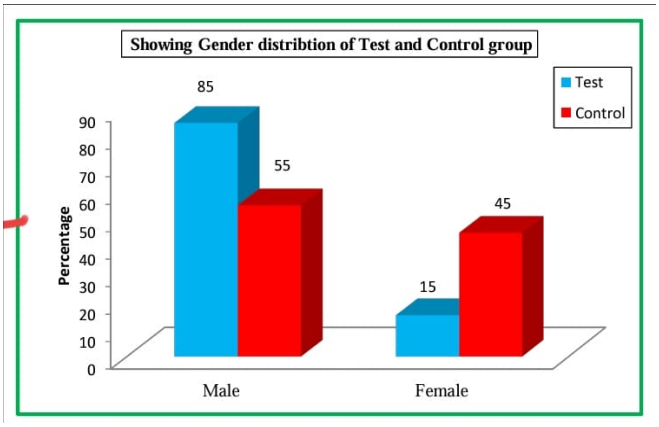

Table 3: Showing distribution of patients as per Marital status.					
Maritalstatus	Test		Control		p value
	No.	%	No.	%	
Married	18	90	20	100	0.4870
Unmarried	2	10	0	0	
Total	20	100	20	100	
Fisher Exact Test					

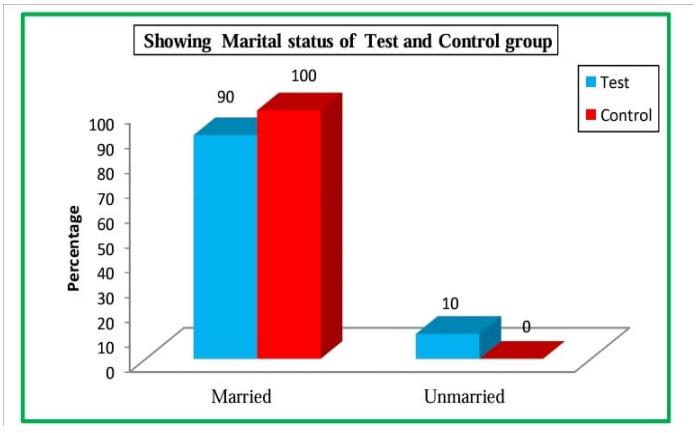

Table 4: Showing distribution of patients as per Socio economic status.					
SES	Test		Control		p-value (Monte Carlo)
	No.	%	No	%	
Upper Class	0	0	1	5	0.6790
Upper Middle Class	5	25	2	10	
Lower Middle Class	8	40	10	50	
Upper Lower Class	4	20	5	25	
Lower Class	3	15	2	10	
Total	20	100	20	100	

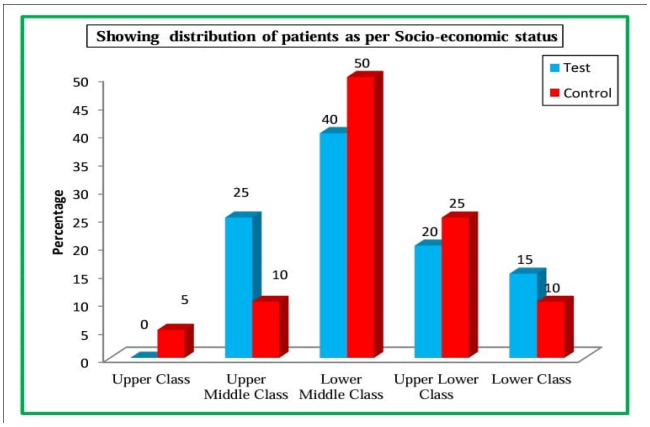

Table 5: Showing distribution of patients with respect to Family history.					
Family history	Test		Control		P-value
	No.	%	No.	%	
Present	7	35	6	30	0.7360
Absent	13	65	14	70	
Total	20	100	20	100	

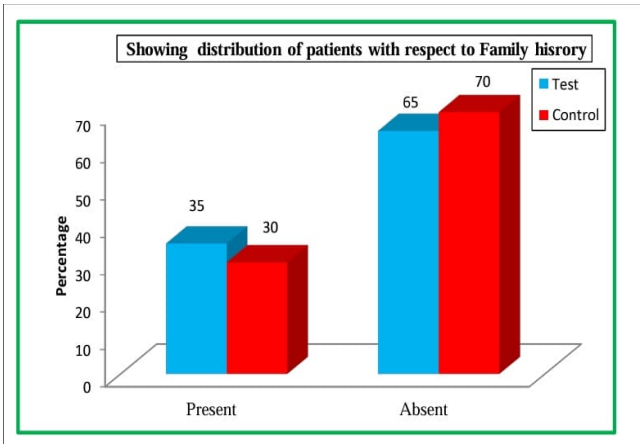

Table 6: Comparison of Safety parameters Before and After the treatmentwith in Test group.					
Safety parameters		Mean	N	Std. Deviation	p-value
Hb %	BT	14.8000	20	1.67929	.02344
	AT	15.6550	20	1.39867	
WBC	BT	6376.0215	20	2648.94002	0.72491
	AT	6141.0000	20	2042.84327	
N	BT	61.0000	20	10.24952	0.05695
	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 7: Comparison of safety parameters Before and After thetreatment with in control group.

Safety parameters		Mean	N	Std. Deviation	p-value
Hb %	BT	5698.2425	20	3519.75849	.11600
	AT	6759.4500	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	

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. 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. 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).

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. 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. 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. (These areas; familial chylomicron syndromes are relatively rare). The conditions are found worldwide and are generally diagnosed in childhood.[51] 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.

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*. 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.

The present study was an open labelled, randomized, Comparative, pre and post analysisconducted in RRIUM, Hospital, University of Kashmir, Srinagar to evaluate the efficacy of test drug Bādranjboya, (Melissa officinalis) in the management 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.

VIII. CONCLUSION

In this study 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 followup i.e., 0, 15^{th}, 30^{th} and 45^{th} 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.

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