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A Clinical Study to Determine the Efficacy of Pramehhara Kwatha in the Management of Madhumeha W.S.R. to Type II Diabetes Mellitus

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ABSTRACT

The present is an era of rapid modernization. Due to sedentary lifestyle and faulty dietary habits, humans are becoming more vulnerable to several metabolic disorders. Preameha is one of them which is caused by association of vitiated tridoshas and morbid Meda, Rakta, Shukra, Ambu, Vasa, Lasika, Majja, Rasa, Oja and Mamsa dhatus. In modern science, it resembles the symptomatology of type II diabetes mellitus which is caused by inadequate insulin production, improper insulin action, or both. In 20th century Dr. Gananath Sen and others have expounded about the theory that Madhumeha is Dhatwagni mandya vikara which can be correlated to diabetes mellitus. Type 2 diabetes mellitus may be included under the broad spectrum of prameha described in Ayurvedic classics. Prameha is considered as Mahagada by Ayurvedic Acharayas. Mahagada are diseases having chronicity with involvement of Tridoshas, multiple Dhatus and Strotases affecting vital organs and thus rendering them difficult to treat. From Ayurvedic point of view, Prameha seems to stem from metabolic derangements at different levels caused either due to genetic factor or due to Apathya Ahara and Vihara. The prevalence of diabetes in high socio-economic population of rural areas is more and in rural areas of all states of India, diabetes was more prevalent in individuals of higher SES. In the next 20 years, it is expected that the prevalence of diabetes in adults-of which type 2 DM is becoming more prevalent. A large portion of this rise will take place in developing nations, where the majority of patients are between the ages of 45 and 64. In 2011, 366 million individuals were expected to have DM; by 2030, this number will have increased to 552 million. It is a potential signal for several comorbidities like hypertension, coronary artery disease, renal diseases etc.

Keywords: Madhumeha, Pramehhara, Metformin, Diabetes mellitus

INTRODUCTION

The metabolic and endocrine disorders are either increasing across the globe or may have been observed with more focus in recent years. Diabetes mellitus is one of the most commonly encountered endocrine disease in clinical practice [1].

Due to a competitive lifestyle, urbanisation, and industrialization, the current period is marked by stress and strain. Diabetes mellitus is one of the many metabolic illnesses that have increased in prevalence as a result and are currently of major concern. Diabetes mellitus is a clinical syndrome characterized by hyperglycaemia due to absolute

or relative deficiency of insulin. Over a period of time, it can damage the blood vessels and nerves as well as many other body systems which consequently cause life threatening complications [2].

In developing nations, diabetes mellitus is increasingly becoming a major public health issue. According to the World Diabetes Federation, there have been worldwide periodic increases in the number of persons with diabetes. India is known as the "Diabetes Capital of the World" since it has the highest percentage of diabetes patients worldwide. According to the first WHO global report on diabetes from 2016, there are 422 million people worldwide who have diabetes, nearly quadrupling since 1980. Diabetes is expected to overtake heart disease as the seventh biggest cause of death by 2030. Yet, the disease related consequences, which raise morbidity and death, actually burden the population. According to these calculations, diabetes was a significant contributor to heart disease and stroke [3,4].

Diabetes mellitus has gained gigantic disgrace in recent times as it is rapidly becoming world's largest silent killer. WHO has projected India, as the country with fastest growing population of diabetic patients hence called it the 'diabetes capital' of the world. According to recent study by Union Ministry of Family and Health Welfare (MoFHW) published on 6th January 2021, among Indians over 45 years of age 11.5% were diagnosed with diabetes. The prevalence was higher in senior citizens (14%) than in those who aged 45-59 years that is 11.5%. Therefore, the prevalence of diabetes mellitus is increasing and placing burden on health care resources [5].

So, a better, safer, and long-lasting therapy is needed for the present scenario and now it is a demand of time to search the management for this type of ailment through the heritage of Ayurveda. Thus, here an attempt is made to treat Madhumeha W.S.R. to type II diabetes mellitus with herbal medicines after compiling all available references from classical authentic texts [6].

A very scientific and elaborated description of diabetes is available in Ayurvedic literature as Prameha. Ayurvedic classics have laid importance upon the etiological factors, their role in vitiations of Doshas and Dushayas which manifest disease conditions. Besides this, Prameha Roga is also considered due to Beeja Dushti (genetic) or Sahaj (congenital) and also Santarpanoth (due to overeating and sedentary life style). For the purpose of clinical management, patients of Prameha have been divided into two categories that is, Sthula (obese patients) and Krisha (emaciated patients). Ayurveda through its armamentarium can become a potential source of better management of diabetes that may be relatively safe, significantly potent without untoward effects and can improve quality of life [7,8].

Aims and Objectives

Primary objective: To evaluate the clinical efficacy of an Ayurvedic formulation Pramehhara Kwatha in the management of Madhumeha W.S.R. to type II diabetes mellitus.

- To evaluate the clinical efficacy of an Ayurvedic formulation Pramehhara Kwatha as an add on therapy to Metformin in the management of Madhumeha W.S.R. to type II diabetes mellitus.

Secondary objective: To assess the clinical safety of Pramehhara Kwatha in management of Madhumeha W.S.R. to type II diabetes mellitus.

- To assess the clinical safety of Pramehhara Kwatha as an add on therapy to metformin in the management of Madhumeha W.S.R. to type II diabetes mellitus [9].

MATERIAL AND METHODS

Selection of the patient

The proposed work was a clinical trial on willing volunteers. Patients fulfilling the diagnostic and inclusion criteria were selected randomly from OPD/IPD of department of Kayachikitsa, R.G.G.P.G. Ayurvedic Hospital, Paprola. A sample of 45 patients-15 patients in each group was assessed in the clinical study [10].

Study design

- Study type–Randomized clinical trial

- Masking–Single blind
- Timing–Prospective
- Number of patients–45 (15 in each group)
- No of groups–3
- Duration of trial–12 weeks
- Follow up visit–After 2 weeks, 4 weeks, 8 weeks and at the completion of trial.

Diagnostic criteria

Selected study subjects were diagnosed on the basis of:

- Fasting plasma glucose ≥ 126 mg/dl
- PPBS (2-hour plasma glucose level) ≥ 200 mg/dl
- HbA1C ≥ 6.5%

Inclusion criteria

- Study subjects of either gender aged between 20-70 yrs.
- Study subjects of type II diabetes mellitus having:
    - Fasting plasma glucose-126 mg/dl to 200 mg/dl
    - PPBS (2-hour plasma glucose level)-200 to 400 mg per decilitre of blood.
    - HbA1C- 6.5-9 %
    - Study subjects willing to participate in the trial.

Exclusion criteria

- Study subjects below the age of 20 yrs. and above the age of 70 yrs.
- Study subjects not willing to participate in the trial.
- Study subjects having FBS >200 mg/dl and/or PPBS >400 mg/dl and/or HbA1c >9%.
- Study subjects having major medical illness like cancer, concurrent infection like tuberculosis.
- Study subjects with uncontrolled hypertension.
- Study subjects with established diagnosis of CAD or any other clinically significant cardiovascular disease.
- Renal dysfunction (defined by eGFR <60 ml/min calculated by MDRD Calculator).
- Subjects who have completed participation in any other clinical trial during the past 3 months.
- Any other condition which the investigator thinks may compromise the safety of the subject.
- Subjects with haemoglobin percentage <9 gm% for males and <8% for females.
- Subject of type-1 DM or type-II DM on insulin/OHA's other than Metformin/any other medications for glycaemic control in the last 3 months.
- Subjects suffering from the complications of diabetes mellitus *viz.,* diabetic neuropathy, diabetic nephropathy, diabetic retinopathy etc. which require an urgent treatment.
- Subjects with concurrent hepatic dysfunction (defined as AST and/or ALT >3 times of the upper normal limit)
- Uncontrolled pulmonary dysfunction requiring inhalational or systemic steroids.
- Pregnant/lactating women.
- Subjects on systemic or inhaled steroids, oral contraceptives pills or oestrogen replacement therapy [11,12].

Investigations

- **Haematological:** Hb%, TLC, DLC, ESR
- **Biochemistry:** FBS, PPBS, HbA1C

Blood urea, serum creatinine, serum lipid profile, SGOT, SGPT

- **Urine:** Routine and microscopic.

Grouping of patients

Study was conducted randomly on 45 patients in three groups (15 patients in each group). Group I was administered with Pramehhara Kwatha 50 ml twice a day, group II was administered with tab. Metformin 1000 mg twice a day while Group III was given Pramehhara Kwatha 50 ml twice a day along with tab. Metformin 1000 mg twice a day [13].

Trial drug

- Pramehhara Kwatha
- **Dose**–50 ml twice a day (50 gm dry coarse powder of Pramehhara Kwatha was dissolved in 800 ml of water. It was reduced to 100 ml and taken in two equally divided doses *i.e*., 50 ml BD) [14].
- **Route of administration**-Oral

Trial drug composition

Trial drug content described in Table 1.

Table 1 Contents of Pramehhar Kwatha

Sr. no	Ingredients	Botanical name	Family	Parts used	Part
1	Haritaki	*Terminalia chebula* (Retz.)	Combretaceae	Pericarp	1 part
2	Vibhitaki	*Erminalia bellerica* (Roxb)	Combretaceae	Pericarp	1 part
3	Amalaki	*Mblica officinalis* (Gaertn)	Euphorbiacea	Pericarp	1 part
4	Daruhridra	*Berberis aristata* (DC.)	Berberidaceae	Root	1 part
5	Mustak	*Cyperus rotundus* (Linn.)	Cyperaceae	Rhizome	1 part

Criteria of assessment

- Subjective parameters were assessed before and after the treatment as per grade score.

Objective criteria:

Haematological: Hb%, TLC, DLC, ESR

Biochemistry: FBS, PPBS, HbA1C

Blood urea, Serum creatinine, Serum lipid profile, SGOT, SGPT

- **Urine:** Routine and microscopic

Grading of subjective criteria

Table 2 describe the grading of subjective criteria.

Table 2 Grading of subjective criteria

Prabhuta Mutrata (Polyuria)	**Grade**
3-5 times per day, rarely at night	0
6-8 times per day, 1-2 times per night	1
9-11 times per day, 3-4 times per night	2
>11 times per day, >4 times per night	3
Avila Mutrata (Turbidity)	
Clear	0
Faintly cloudy or smoky (turbidity barely visible)	1
Turbidity clearly present	2
Highly turbid	3
Pipasa-Adhikya (Increased Thirst)	
Feeling of thirst (7-9 times/24 hrs.) and relieved by drinking water	0
Feeling of moderate thirst (>9-11 times/24 hrs.) and relieved by drinking water	1
Feeling of excess thirst (>11-13 times/24 hrs.) not relieved by drinking water	2
Feeling of severe thirst (>13 times/24 hrs.) not relieved by drinking water	3
Kshuda-Adhikya (Increased Appetite)	
As usual/Routine	0
Slightly Increased (1 meal extra with routine diet)	1
Moderate Increased (2 meals extra with routine diet)	2
Markedly Increased (3 meals extra with routine diet)	3
Karapada Daha (Burning sensation in hand and feet)	
No Daha	0
Karapada Daha found occasionally, mild bearable	1
Karapada Daha continuous but bearable and not severe	2
Karapada Daha continuous and severe and unbearable	3
Swedadhikya (Perspiration)	
Sweating after heavy work and fast movement or in hot weather	0
Profuse sweating after moderate work and movement	1
Sweating after little work and movement (stepping ladder etc.)	2

Profuse sweating after little work and movement	3
Galatalushosha (Dryness of palate and throat)	
None	0
Mild	1
Moderate	2
Severe	3
Madhurasyata (Feeling sweetness in mouth)	
None	0
Mild	1
Moderate	2
Severe	3
Karapadasuptata (Numbness)	
No Karapadasuptata	0
Hasta-Pada-Tala Daha found occasionally, mild bearable	1
Hasta-Pada-Tala Daha continuous but bearable and not severe	2
Hasta-Pada-Tala Daha continuous and severe and unbearable	3
Shithilangata	
None	0
Mild	1
Moderate	2
Severe	3

Grading of urine routine and microscopic

Table 3 describe grading of urine routine and microscopic.

Table 3 Grading of urine routine and microscopic

Urine sugar	**Grade**
Nil	0
+	1
++	2
+++	3
Pus cells	
Nil	0
1-2	1

3-4	2
5-6	3
Epithelial cells	
Nil	0
1-2	1
3-4	2
5-6	3

Final assessment of results statistical analysis: Data obtained during the trial was tabulated and statistically analysed using Student Paired 't' Test. The results were considered significant or insignificant on the basis of value of 'p'.

- Insignificant at $p>0.05$.
- Significant at $p<0.05$.
- Highly significant at $p<0.001$.

RESULTS

Among all the registered study subjects the incidence of Madhumeha was highest in age group 51- 60 years (*i.e*., 45.09%), males (*i.e.,* 50.9%) and Hindus (*i.e*., 100%). Majority of the study subjects were household workers (*i.e.,* 39.21 %) and maximum were matriculate (*i.e*., 33.33%) and majority of study subjects *i.e*., 66.66% were above poverty line and belonged to rural area (*i.e*., 68.62%).

Based on the constitutional profile maximum study subjects *i.e.,* 68.62% were living with sedentary lifestyle. Appetite (*i.e.,* 58.82 %) and thirst (*i.e*., 62.74%) was increased in majority of study subjects. Bowel habit was normal and regular in 74.50 % study subjects and frequency of micturition was increased in majority of the study subjects (*i.e*., 62.74%). Most of the study subjects *i.e.,* 68.62 % were taking mixed diet and majority of the study subjects were non addicted to smoking or alcohol (*i.e*., 62.74%). The incidence of Madhumeha was more in study subjects with Pitta-Kaphaj Prakriti (*i.e*., 47.05%) (Table 4). In the present clinical study frequency of signs and symptomatology observed in registered study subjects is as follows:

- Prabhuta Mutrata in 32 study subjects (62.74%).
- Pipasadikya in 32 study subjects (62.74%).
- Karapadasuptata in 31 study subjects (60.78%).
- Kshudhadikya in 30 study subjects (58.82%).
- Shithilangata in 30 study subjects (58.82%).
- Karapada daha in 29 study subjects (56.86%).
- Swedadikya in 28 study subjects (54.90%).
- Galatalusosha in 26 study subjects (50.98%).
- Avila Mutrata in 25 study subjects (49.01%).
- Madhurasyata in 7 study subjects (13.72%).

Table 4 Incidence of signs and symptoms of type 2 diabetes mellitus in registered study subjects

Sr. no.	Symptoms	Gr. I		Gr. II		Gr. III		Total	
		N	% age	N	% age	N	% age	N	% age
1	Prabhuta Mutrata	10	58.82	11	68.75	11	61.11	32	62.74
2	Avila Mutrata	8	47.05	7	43.75	10	55.55	25	49.01

3	Pipasa-Adhikya	10	58.82	11	68.75	11	61.11	32	62.74
4	Kshudha-Adhikya	9	52.94	10	62.5	11	61.11	30	58.82
5	Karapadadaha	9	52.94	9	56.25	11	61.11	29	56.86
6	Swedadikya	10	58.82	9	56.25	9	50	28	54.9
7	Galatalushosha	10	58.82	8	50	8	44.44	26	50.98
8	Madhurasya	1	5.88	4	25	2	11.11	7	13.72
9	Karapadasuptata	12	70.58	9	56.25	10	55.55	31	60.78
10	Shithilangata	10	58.82	9	56.25	11	61.11	30	58.82

Effect of therapy based on subjective criteria

The effect of therapy on various assessment criteria was obtained after statistical analysis of the data and is presented in Tables 5 and 6.

Table 5 Effect of therapy on subjective parameters

S. no.	Symptoms	Group	N	Mean			% relief	SD ±	SE ±	't'	p value	Significance
				BT	AT	Diff.						
1	Prabhuta Mutrata	G-I	15	0.8	0.267	0.533	66.62↓	0.516	0.133	4	0.001	S
		G-II	15	1.133	0.467	0.667	58.78↓	0.724	0.187	3.568	0.003	S
		G-III	15	0.933	0.066	0.867	92.81↓	0.915	0.236	3.666	0.003	S
2	Avila Mutrata	G-I	15	0.733	0.267	0.467	63.57↓	0.743	0.192	2.432	0.029	S
		G-II	15	0.533	0.133	0.4	75.04↓	0.632	0.163	2.449	0.028	S
		G-III	15	0.867	0.133	0.733	84.65↓	0.594	0.153	4.785	<0.001	HS
3	Pipasa-Adhikya	G-I	15	0.867	0.267	0.6	69.20↓	0.828	0.214	2.806	0.014	S
		G-II	15	1	0.333	0.667	66.70↓	0.617	0.159	4.183	<0.001	HS
		G-III	15	0.867	0.066	0.8	92.38↓	0.775	0.2	2.827	0.001	S
4	Kshuda-Adhikya	G-I	15	0.8	0.333	0.467	58.37↓	0.64	0.165	2.824	0.014	S
		G-II	15	0.933	0.2	0.733	78.56↓	0.704	0.182	4.036	0.001	S
		G-III	15	0.867	0.066	0.8	92.38↓	0.775	0.2	4	0.001	S
5	Karapadadaha	G-I	15	0.733	0.333	0.4	54.57↓	0.632	0.163	2.449	0.028	S
		G-II	15	0.8	0.267	0.533	66.62↓	0.516	0.133	4	0.001	S
		G-III	15	0.733	0.2	0.533	72.71↓	0.64	0.165	3.228	0.006	S
6	Swedadikya	G-I	15	0.867	0.4	0.467	53.86↓	0.743	0.192	2.432	0.029	S
		G-II	15	0.867	0.333	0.533	61.59↓	0.516	0.133	4	0.001	S
		G-III	15	0.733	0.2	0.533	72.71↓	0.743	0.192	2.779	0.015	S

7	Galatalushosha	G-I	15	0.733	0.467	0.267	36.28↓	0.458	0.118	2.256	0.041	S
		G-II	15	0.8	0.467	0.333	41.62↓	0.617	0.159	2.092	0.055	IS
		G-III	15	0.6	0.2	0.4	66.66↓	0.507	0.131	3.055	0.009	S
8	Madhurasya	G-I	15	0.066	0.066	0	0	0	0	0	1	IS
		G-II	15	0.267	0.133	0.133	50.18↓	0.352	0.09	1.468	0.164	IS
		G-III	15	0.133	0.066	0.066	50.37↓	0.258	0.066	1	0.334	IS
9	Karapadasuptata	G-I	15	0.933	0.6	0.333	35.69↓	0.488	0.126	2.646	0.019	S
		G-II	15	0.733	0.267	0.467	63.57↓	0.516	0.133	3.5	0.004	S
		G-III	15	0.867	0.333	0.533	61.59↓	0.516	0.133	4	0.001	S
10	Shithilangata	G-I	15	0.533	0.2	0.333	62.47↓	0.488	0.126	2.646	0.019	S
		G-II	15	0.533	0.133	0.4	75.04↓	0.507	0.131	3.055	0.009	S
		G-III	15	0.667	0.066	0.6	90.10↓	0.507	0.131	4.583	<0.001	HS
Note: ↑: Increase, ↓: Decrease, IS: Insignificant, S: Significant, HS: Highly Significant												

Table 6 Intergroup comparison of subjective criteria

Comparisno	**% age relief**		**Diff. of % age relief**	**SD**	**SE**	**'t'**	**p value**	**Significance**
Prabhuta Mutrata								
GP I *vs.* II	G-I	66.62%	7.84%	0.65	0.24	-0.53	0.566	IS
	G-II	58.78%						
GP II *vs.* III	G-II	58.78%	-34.03%	0.85	0.32	-0.61	0.512	IS
	G-III	92.81%						
GP I *vs.* III	G-I	66.62%	-26.19%	0.77	0.29	-1.14	0.23	IS
	G-III	92.81%						
Avila Mutrata								
GP I *vs.* II	G-I	63.57%	-11.47%	0.71	0.27	0.24	0.793	IS
	G-II	75.04%						
GP II *vs.* III	G-II	75.04%	-9.61%	0.63	0.24	-1.38	0.148	IS
	G-III	84.65%						
GP I *vs.* III	G-I	63.57%	-21.08%	0.69	0.26	-1	0.287	IS
	G-III	84.65%						
Pipasa-Adhikya								
GP I *vs.* II	G-I	69.20%	2.50%	0.75	0.28	-0.23	0.804	IS
	G-II	66.70%						
GP II *vs.* III	G-II	66.70%	-25.60%	0.72	0.27	-0.48	0.606	IS

	G-III	92.38%						
GP I *vs.* III	G-I	69.20%	-23.18%	0.83	0.31	-0.63	0.5	IS
	G-III	92.38%						
Kshuda-Adhikya								
GP I *vs.* II	G-I	58.37%	-20.19%	0.69	0.26	-1	0.287	IS
	G-II	78.56%						
GP II *vs.* III	G-II	78.56%	-13.82%	0.76	0.29	-0.22	0.807	IS
	G-III	92.38%						
GP I *vs.* III	G-I	58.37%	-34.01%	0.73	0.27	-1.19	0.209	IS
	G-III	92.38%						
Karpada Daha								
GP I *vs.* II	G-I	35.69%	-27.88%	0.59	0.22	-0.58	0.532	IS
	G-II	63.57%						
GP II *vs.* III	G-II	66.62%	-6.09%	0.6	0.22	0	1	IS
	G-III	72.71%						
GP I *vs.* III	G-I	35.69%	-37.02%	0.66	0.25	-0.53	0.571	IS
	G-III	72.71%						
Swedadikya								
GP I *vs.* II	G-I	53.86%	-7.73%	0.66	0.25	-0.26	0.778	IS
	G-II	61.59%						
GP II *vs.* III	G-II	61.59%	-11.12%	0.66	0.25	0	1	IS
	G-III	72.71%						
GP I *vs.* III	G-I	53.86%	-18.85%	0.66	0.25	-0.26	0.778	IS
	G-III	72.71%						
Galatalushosha								
GP I *vs.* II	G-I	36.28%	-5.34%	0.56	0.21	-0.31	0.739	IS
	G-II	41.62%						
GP II *vs.* III	G-II	41.62%	-25.04%	0.58	0.22	-0.3	0.749	IS
	G-III	66.66%						
GP I *vs.* III	G-I	36.28%	-30.38%	0.5	0.18	-0.7	0.456	IS

	G-III	66.66%						
Madhurasya								
GP I *vs.* II	G-I	0%	-50.18%	0.24	0.09	-1.46	0.153	IS
	G-II	50.18%						
GP II *vs.* III	G-II	50.18%	-0.19%	0.32	0.12	0.54	0.559	IS
	G-III	50.37%						
GP I *vs.* III	G-I	0%	-50.37%	0.18	0.07	-0.92	0.326	IS
	G-III	50.37%						
Karpadasuptata								
GP I *vs.* II	G-I	35.69%	-27.88%	0.52	0.19	-0.67	0.473	IS
	G-II	63.57%						
GP II *vs.* III	G-II	63.57%	1.98%	0.53	0.2	-0.32	0.726	IS
	G-III	61.59%						
GP I *vs.* III	G-I	35.69%	-25.9%	0.52	0.19	-1.01	0.285	IS
	G-III	61.59%						
Shithilangata								
GP I *vs.* II	G-I	62.47%	-9.57%	0.51	0.19	-0.34	0.716	IS
	G-II	75.04%						
GP II *vs.* III	G-II	75.04%	-15.06%	0.52	0.19	-1	0.289	IS
	G-III	90.10%						
GP I *vs.* III	G-I	62.47%	-27.63%	0.51	0.19	-1.36	0.153	IS
	G-III	90.10%						

Effect of therapy based on objective criteria

Tables 7-10 describe the effect of therapy based on objective criteria.

Table 7 Effect of therapy on objective parameters

Objective criteria	Group	N	Mean			% relief	SD ±	SE ±	't'	p value	Significance
			BT	AT	Diff.						
FBS	G-I	15	154.26	140.46	13.80	8.94↓	8.64	2.23	6.18	<0.001	HS
	G-II	15	169.66	135.46	34.20	20.15↓	7.80	2.01	16.97	<0.001	HS
	G-III	15	165.73	125.46	40.26	24.29↓	20.42	5.27	7.63	<0.001	HS
PPBS	G-I	15	222.66	197.93	24.73	11.10↓	10.08	2.60	9.49	<0.001	HS
	G-II	15	229.00	161.06	67.93	29.66↓	25.86	6.67	10.17	<0.001	HS
	G-III	15	225.33	145.53	79.80	35.41↓	21.87	5.64	14.13	<0.001	HS
HbA1C	G-I	15	7.85	7.67	0.18	2.29↓	0.17	0.04	3.91	0.002	S
	G-II	15	7.19	6.51	0.68	9.45↓	0.43	0.11	6.01	<0.001	HS
	G-III	15	7.24	6.45	0.78	10.87↓	0.43	0.11	7.07	<0.001	HS

Blood urea	G-I	15	28.06	26.66	1.40	4.98↓	5.98	1.54	0.90	0.380	IS
	G-II	15	26.80	27.40	-0.60	-2.23↑	3.77	0.97	-0.61	0.548	IS
	G-III	15	34.26	26.80	0.60	1.75↓	5.71	1.47	0.40	0.556	IS
Serum creatinine	G-I	15	0.77	0.74	0.03	3.89↓	0.17	0.04	0.60	0.689	IS
	G-II	15	0.80	0.77	0.0267	3.25↓	0.16	0.04	0.63	0.535	IS
	G-III	15	0.810	0.81	0.00	0.00	0.110	0.029	0.000	0.103	IS
SGOT	G-I	15	30.33	28.33	2.00	6.59↓	2.10	0.543	3.681	0.002	S
	G-II	15	36.93	29.00	7.93	21.47↓	15.34	3.96	2.003	0.065	IS
	G-III	15	29.60	28.13	1.46	4.95↓	2.06	0.53	2.75	0.016	S
SGPT	G-I	15	27.93	25.66	2.26	8.11↓	2.65	0.68	3.30	0.005	S
	G-II	15	35.20	30.60	4.60	13.06↓	10.86	2.80	1.64	0.123	IS
	G-III	15	31.00	28.53	2.46	7.90↓	3.87	0.99	2.46	0.027	S
Serum cholesterol	G-I	15	189.20	188.66	0.53	0.28↓	10.44	2.69	0.19	0.846	IS
	G-II	15	202.73	197.20	5.53	2.72↓	13.51	3.49	1.58	0.135	IS
	G-III	15	190.60	184.60	6.00	3.14↓	10.96	2.83	2.12	0.052	IS
Serum triglycerides	G-I	15	176.13	166.66	9.46	5.37↓	34.30	8.85	1.06	0.303	IS
	G-II	15	196.60	190.66	5.93	3.01↓	17.75	4.58	1.29	0.217	IS
	G-III	15	191.00	187.06	3.93	2.05↓	10.21	2.63	1.49	0.158	IS
Serum LDL	G-I	15	111.00	104.13	6.87	6.18↓	18.77	4.84	1.41	0.175	1S
	G-II	15	120.40	115.40	5.00	4.15↓	14.22	3.67	1.36	0.195	1S
	G-III	15	121.40	119.73	1.66	1.37↓	5.38	1.38	1.20	0.250	1S
Serum HDL	G-I	15	41.13	43.66	-2.53	-6.15↑	6.25	1.61	-1.56	0.139	IS
	G-II	15	46.86	54.86	-8.00	-17.07↑	15.57	4.02	-1.98	0.067	IS
	G-III	15	42.06	46.53	-4.46	-10.60↑	12.06	3.11	-1.43	0.174	IS
Haemoglobin	G-I	15	12.53	12.69	-0.16	-1.28↑	0.82	0.21	-0.84	0.414	IS
	G-II	15	13.22	13.37	-0.15	-1.13↑	0.64	0.16	0.15	0.876	IS
	G-III	15	12.01	11.98	0.03	0.24↓	0.50	0.13	0.25	0.803	IS

TLC	G-I	15	7120	7046	74	1.04↓	249.18	64.34	0.16	0.872	IS
	G-II	15	7540	7413	127	6.25↓	465.16	120.10	1.26	0.228	IS
	G-III	15	7926	7320	606	7.65↓	1864.50	481.40	2.37	0.235	IS
Neutrophils	G-I	15	63.61	56.73	6.88	10.81↓	11.86	3.06	2.24	0.141	IS
	G-II	15	59.43	61.41	-1.98	-3.33↑	5.63	1.45	-1.36	0.195	IS
	G-III	15	68.41	65.38	3.03	4.42↓	5.981	1.54	1.96	0.070	IS
Lymphocytes	G-I	15	33.86	32.14	1.72	5.07↓	8.078	2.08	0.97	0.348	IS
	G-II	15	33.13	31.08	2.05	6.18↓	7.158	1.84	1.13	0.275	IS
	G-III	15	27.70	27.60	0.1	0.36↓	4.155	1.07	0.09	0.922	IS
Mixed cells	G-I	15	7.38	7.50	-0.12	-1.62↑	3.27	0.84	-0.14	0.889	IS
	G-II	15	8.56	8.10	0.46	5.37↓	1.36	0.35	1.31	0.211	IS
	G-III	15	7.72	7.42	0.3	3.88↓	1.336	0.34	0.85	0.409	IS
ESR	G-I	15	21.73	15.46	6.27	28.80↓	12.85	3.32	1.88	0.08	IS
	G-II	15	15.80	11.73	4.07	25.75↓	6.307	1.62	2.49	0.126	IS
	G-III	15	29.40	16.46	12.94	44.01↓	17.42	4.49	2.87	0.062	IS
Urine Sugar	G-I	15	0.33	0.26	0.066	20.12↓	0.25	0.06	1.00	0.33	IS
	G-II	15	0.40	0.06	0.33	83.25↓	0.61	0.15	2.09	0.05	IS
	G-III	15	0.40	0.06	0.33	83.25↓	0.72	0.18	1.78	0.09	IS
Note: ↑ Increase, ↓: Decrease, IS: Insignificant, S: Significant, HS: Highly Significant											

Table 8 Inter group comparison of effect of therapy on biochemical parameters

Comparison	**% age relief**		**Diff. of % age relief**	**SD**	**SE**	**'t'**	**p value**	**Significance**
FBS								
GP I *vs.* II	G-I	8.94%	-11.21%	8.38	3.11	-6.54	<0.001	HS
	G-II	20.15%						
GP II *vs.* III	G-II	20.15%	-4.14%	15.74	5.85	-1.03	0.292	IS

	G-III	24.29%						
GP I *vs.* III	G-I	8.94%	-15.35%	15.96	5.93	-4.45	<0.001	HS
	G-III	24.29%						
PPBS								
GP I *vs.* II	G-I	11.10%	-18.56%	19.99	7.42	-5.81	<0.001	HS
	G-II	29.66%						
GP II *vs.* III	G-II	29.66%	-5.75%	24.55	9.12	-1.37	0.166	IS
	G-III	35.41%						
GP I *vs.* III	G-I	11.10%	-24.31%	17.34	6.44	-8.54	<0.001	HS
	G-III	35.41%						
HbA1C								
GP I *vs.* II	G-I	2.29%	-7.16%	0.34	0.12	-3.95	<0.001	HS
	G-II	9.45%						
GP II *vs.* III	G-II	9.45%	-1.42%	0.44	0.16	-0.64	0.507	IS
	G-III	10.87%						
GP I *vs.* III	G-I	2.29%	-8.58%	0.33	0.12	-4.86	<0.001	HS
	G-III	10.87%						
Blood urea								
GP I *vs.* II	G-I	4.98%	7.21%	5.09	1.89	1.05	0.283	IS
	G-II	-2.23%						
GP II *vs.* III	G-II	-2.23%	-3.98%	4.93	1.83	-0.65	0.503	IS
	G-III	1.75%						
GP I *vs.* III	G-I	4.98%	3.23%	5.96	2.21	0.36	0.711	IS
	G-III	1.75%						
Serum creatinine								
GP I *vs.* II	G-I	3.89%	0.64%	0.16	0.06	0	1	IS
	G-II	3.25%						
GP II *vs.* III	G-II	3.25%	3.25%	0.14	0.05	0.5	0.606	IS
	G-III	0.00%						
GP I *vs.* III	G-I	3.89%	3.89%	0.14	0.05	0.48	0.619	IS

	G-III	0.00%						
SGOT								
GP I *vs.* II	G-I	6.59%	-14.88%	11.15	4.14	-1.43	0.149	IS
	G-II	21.47%						
GP II *vs.* III	G-II	21.47%	16.52%	11.14	4.14	1.56	0.117	IS
	G-III	4.95%						
GP I *vs.* III	G-I	6.59%	1.64%	2.12	0.78	0.67	0.489	IS
	G-III	4.95%						
SGPT								
GP I *vs.* II	G-I	8.11%	-4.95%	8.05	2.99	-0.77	0.426	IS
	G-II	13.06%						
GP II *vs.* III	G-II	13.06%	5.16%	8.304	3.08	0.69	0.48	IS
	G-III	7.90%						
GP I *vs.* III	G-I	8.11%	0.21%	3.38	1.25	-0.15	0.87	IS
	G-III	7.90%						
Serum cholesterol								
GP I *vs.* II	G-I	0.28%	2.44%	8.05	2.99	-0.77	0.426	IS
	G-II	2.72%						
GP II *vs.* III	G-II	2.72%	0.42%	8.3	3.08	0.69	0.48	IS
	G-III	3.14%						
GP I *vs.* III	G-I	0.28%	2.86%	3.38	1.25	-0.15	0.87	IS
	G-III	3.14%						
Serum triglycerides								
GP I *vs.* II	G-I	5.37%	2.35%	27.81	10.33	0.34	0.726	IS
	G-II	3.02%						
GP II *vs.* III	G-II	3.02%	0.95%	14.75	5.48	0.36	0.708	IS
	G-III	2.06%						
GP I *vs.* III	G-I	5.37%	3.31%	25.77	9.57	0.57	0.554	IS
	G-III	2.06%						
LDL								

GP I *vs.* II	G-I	6.18%	2.03%	16.95	6.3	0.29	0.761	IS
	G-II	4.15%						
GP II *vs.* III	G-II	4.15%	2.78%	10.94	4.06	0.81	0.403	IS
	G-III	1.37%						
GP I *vs.* III	G-I	6.18%	4.81%	14.06	5.22	0.99	0.311	IS
	G-III	1.37%						
HDL								
GP I *vs.* II	G-I	-6.15%	-10.92%	12.08	4.49	1.261	0.218	IS
	G-II	-17.07%						
GP II *vs.* III	G-II	-17.07%	-6.47%	14.18	5.27	-0.695	0.493	IS
	G-III	-10.60%						
GP I *vs.* III	G-I	-6.15%	-4.45%	9.78	3.63	0.551	0.586	IS
	G-III	-10.60%						

Table 9 Intergroup comparison of effect of therapy on urine sugar

Comparison	**% age relief**		**Diff. of % age relief**	**SD**	**SE**	**'t'**	**p value**	**Significance**
GP I *vs.* II	G-I	20.12%	-63.13%	0.48	0.17	-1.48	0.134	IS
	G-II	83.25%						
GP II *vs.* III	G-II	83.25%	0%	0.68	0.25	0	1	IS
	G-III	83.25%						
GP I *vs.* III	G-I	20.12%	-63.13%	0.55	0.2	-1.29	0.19	IS
	G-III	83.25%						

Table 10 Inter group comparison of effect of therapy on hematological parameters

Comparison	**% age relief**		**Diff. of % age relief**	**SD**	**SE**	**'t'**	**p value**	**Significance**
Haemoglobin								
GP I *vs.* II	G-I	-1.28%	-0.15%	0.75	0.28	-0.73	0.453	IS
	G-II	-1.13%						
GP II *vs.* III	G-II	-1.13%	-0.89%	0.59	0.22	-0.03	0.975	IS
	G-III	0.24%						
GP I *vs.* III	G-I	-1.28%	-1.52%	0.69	0.25	-0.82	0.402	IS

	G-III	0.24%						
TLC								
GP I *vs.* II	G-I	1.04%	-5.21%	2542.47	944.81	-0.5	0.603	IS
	G-II	6.25%						
GP II *vs.* III	G-II	6.25%	-1.40%	1670.51	620.78	-0.38	0.692	IS
	G-III	7.65%						
GP I *vs.* III	G-I	1.04%	-6.61%	2376.9	883.28	-0.81	0.406	IS
	G-III	7.65%						
Neutrophils								
GP I *vs.* II	G-I	10.81%	14.14%	9.45	3.51	2.52	0.014	S
	G-II	-3.33%						
GP II *vs.* III	G-II	-3.33%	-37.75%	5.91	2.19	-2.27	0.026	S
	G-III	4.42%						
GP I *vs.* III	G-I	10.81%	6.39%	9.56	3.55	1.08	0.271	IS
	G-III	4.42%						
Lymphocytes								
GP I *vs.* II	G-I	5.07%	-1.11%	0.28	4.601	-1.973	0.059	IS
	G-II	6.18%						
GP II *vs.* III	G-II	6.18%	5.82%	0.31	0.312	0.977	0.337	IS
	G-III	0.36%						
GP I *vs.* III	G-I	5.07%	4.71%	0.48	3.234	-1.746	0.092	IS
	G-III	0.36%						
Mixed cells								
GP I *vs.* II	G-I	-1.62%	-6.99%	2.54	0.94	0.43	0.654	IS
	G-II	5.37%						
GP II *vs.* III	G-II	5.37%	1.49%	2.55	0.94	-0.61	0.532	IS
	G-III	3.88%						
GP I *vs.* III	G-I	-1.62%	-5.50%	1.37	0.51	-0.32	0.737	IS
	G-III	3.88%						
ESR								

GP I *vs.* II	G-I	28.80%	3.05%	10.31	3.83	0.57	0.557	IS
	G-II	25.75%						
GP II *vs.* III	G-II	25.75%	-18.26%	13.34	4.95	-1.78	0.074	IS
	G-III	44.01%						
GP I *vs.* III	G-I	28.80%	-15.21%	15.59	5.79	-1.15	0.19	IS
	G-III	44.01%						

Effect of therapy on subjective criteria signs and symptoms

In the present clinical study Prabhuta Mutrata (Polyuria) occurs due to osmotic diuresis which in turn is due to hyperglycaemia. There was 92.81 % reduction in mean score of Prabhuta Mutrata, which was statistically significant ($p<0.05$) in group III. In group I and II there was reduction of 66.62% and 58.78% in mean score of Prabhuta Mutrata respectively, which was also statistically significant ($p<0.05$). On intergroup comparison, there was statistically insignificant difference ($p>0.05$) between group I, II and III [15].

Avila-Mutrata (Turbid urine) occurs due to the presence of sugar or protein in the urine and sometimes due to urinary tract infection. In the present study there was 84.65% reduction in mean score of Avila Mutrata which was statistically highly significant ($p<0.001$) in group III. Whereas in group I and II there was reduction of 63.57% and 75.04% in mean score of Avila Mutrata respectively, which was statistically significant ($p<0.05$). On intergroup comparison, there was statistically insignificant difference ($p>0.05$) between group I, II and III [16].

Pipasa-Adhikya (Polydipsia) is due to Pittavriddhi and Udaka Kshaya *i.e.* loss of Udaka Dhatu through Mutra. The intense thirst appears because of obligatory renal water loss combined with hyper osmolarity resulting from the increased level of glucose in the blood tending to deplete the intra cellular water, triggering the osmo-receptors in the thirst center of the brain. There was 66.7% reduction in mean score of Pipasa Adhikya which was statistically highly significant ($p<0.001$) in group II. In group I and III there was reduction of 69.20% and 92.38% in mean score of Pipasa Adhikya respectively, which was statistically significant (*i.e.,* $p<0.05$, $p=0.001$ respectively). On intergroup comparison, there was statistically insignificant difference ($p>0.05$) between group I, II and III [17-19].

In uncontrolled diabetes where blood glucose level remains abnormally high, glucose from the blood cannot enter the cells due to either a lack of insulin or insulin resistance. So, the body can't convert the food into energy. The lack of energy causes an increase in hunger *i.e.,* Kshuda-Adhikya. In the present study there was 92.3%, 78.56% and 58.3% reduction in mean score of Kshuda-Adhikya which was statistically significant in group III ($p=0.001$), II ($p=0.001$) and group I ($p<0.05$) respectively. On intergroup comparison, the result was statistically insignificant between group I, II and III ($p>0.05$) [20].

Karapada Daha is a Purvarupa of Prameha which continue even in Rupa stage. Pitta and Vata Dosha plays a dominant role in causation of the symptom. The interventional trial drug contains Pitta-Vatahara drugs which cause the alleviation of Pitta as well as Vata Dosha leading to the subsidence of Karapada Daha. In modern science, it is explained that this symptom is a feature of Diabetic neuropathy.

In the present study there was 72.71%, 66.62% and 54.57% reduction in mean score of Karapada Daha which was statistically significant in group III ($p<0.05$), II ($p=0.001$) and group I ($p<0.05$) respectively. On intergroup comparison, the result was statistically insignificant between group I, II and III ($p>0.05$) [21].

Swedadikya is due to Pitta Prakopa and Medomala Vriddhi in the study subjects of Santarpanottha Prameha. In the present study there was 72.71%, 61.59% and 53.86% reduction in mean score of Swedadikya which was statistically significant in group III ($p<0.05$), II ($p=0.001$) and group I ($p<0.05$) respectively. On intergroup comparison, the result was statistically insignificant between group I, II and III ($p >0.05$).

Gala-Talushosha (Dryness in oral cavity) is a symptom follows from the feature Pipasa (polydipsia) and is caused by excessive loss of water through urine. In the present study there was 66.66% and 41.62% reduction in mean score of Gala-Talushosha which was statistically significant in group III ($p<0.05$) and group I ($p<0.05$) respectively. In

group I it was statistically insignificant. On intergroup comparison, the result was statistically insignificant between group I, II and III ($p > 0.05$).

Madhurasyata (Sweetness in Mouth) is felt due to excess glucose in blood causing persistent salivation of the mouth. This symptom is found most frequent in all diabetics. In the present study only few (*i.e.,* 7) study subjects in all three groups presented with the feature of Madhurasyata and all three groups showed statistically insignificant ($p>0.05$) results. On intergroup comparison, the result was statistically insignificant between group I, II and III ($p > 0.05$).

Karapada Suptata (Numbness) is also a feature of hyperglycemia induced neuropathy. In the present study there was 61.59%, 63.57% and 35.69% reduction in mean score of Karapada Suptata which was statistically significant in group III ($p=0.001$), II ($p<0.05$) and group I ($p<0.05$) respectively. On intergroup comparison, the result was statistically insignificant between group I, II and III ($p > 0.05$).

Excessive production of Shareera Kleda leads to the manifestation of Shithilangata. This is the most common symptom found frequently in almost all diabetics resulting from excessive loss of electrolytes through urine and also due to less glucose supply to the cells and thus causing fatigue and lethargy in the diabetic study subjects. In this study there was 90.10% reduction in mean score of Shithilangata which was statistically highly significant ($p<0.001$) in group III. Whereas in group II and I, there was reduction of 75.04% and 62.47% in mean score of Shitilangata respectively, which was statistically significant (*i.e.,* $p<0.05$). On intergroup comparison, the result was statistically insignificant between group I, II and III ($p>0.05$).

Effect of therapy on objective criteria

On F.B.S., P.P.B.S., HbA1C and Urine sugar: Fasting blood sugar: In the present clinical study the mean score of FBS in group I, II and III before treatment was 154.26, 169.66, 165.73 mg/dl and after treatment it was 140.46, 135.46 and 125.46 mg/dl giving 8.94%, 20.15%, 24.29% reduction in mean score respectively, which was statistically highly significant ($p<0.001$) in group I, II and III. The FBS levels significantly decreased in all groups but maximum decrease was found in group III (24.29%) after three months of treatment. On intergroup comparison, the result was statistically highly significant ($p<0.001$) between group I-II and group I-III and was statistically insignificant between group II-III ($p>0.05$).

Post prandial blood sugar: In the present clinical study the mean score of PPBS in group I, II and III before treatment was 222.66, 229.0, 225.33 mg/dl and after treatment it was 197.93, 161.06 and 145.53 mg/dl giving 11.10%, 29.66% and 35.41% reduction in mean score respectively, which was statistically highly significant ($p<0.001$) in group I, II and III. The PPBS level significantly decreased in all groups but maximum decrease found in group III (35.41%) after three months of treatment. On intergroup comparison, the result was statistically highly significant ($p<0.001$) between group I-II and group I-III and was statistically insignificant between group II-III ($p>0.05$).

Glycosylated haemoglobin/HbA1C: In the present clinical study the mean score of HbA1c in group I, II and III before treatment was 7.85, 7.19, 7.24 and after treatment it was 7.67, 6.51 and 6.45 giving 2.29%, 9.45% and 10.87% reduction in mean score respectively, which was statistically highly significant ($p<0.001$) in group II and III and statistically significant in group I ($p<0.05$). On intergroup comparison, the result was statistically highly significant ($p<0.001$) between group I-II and group I-III and was statistically insignificant between group II-III ($p>0.05$). The HbA1c level significantly decreased in all groups but maximum decrease was found in group III (*i.e.,* 10.87%). This shows the significant effect of Pramehhara Kwatha in reducing the HbA1c level.

Urine sugar: In the present clinical study only a few enrolled study subjects had glycosuria, the mean score of urine sugar in group I, II and III before treatment was 0.33, 0.40 and 0.40 and after treatment it came to 0.26, 0.06 and 0.066 giving 20.12%, 83.25 % and 83.25% reduction in mean score respectively which was statistically insignificant ($p>0.05$) in all three groups. On intergroup comparison, there was a statistically insignificant difference ($p>0.05$).

Effect of Therapy on other Haematological and Biochemical Parameters

In the present study, no considerable change was noticed in Hb, TLC, DLC, ESR, FBS, blood urea and serum creatinine after treatment in both the groups. But significant change was noticed in SGOT and SGPT level after treatment.

SGOT: In the present clinical study the mean score of SGOT in Group I, II and III before treatment was 30.33 IU/L, 36.93 IU/L, 29.60 IU/L and after treatment it was 28.33 IU/L, 29.00 IU/L and 28.13 IU/L giving 6.59%, 21.47% and 4.95% reduction in mean score respectively which was statistically significant in group I and III ($p<0.05$) and was statistically insignificant in group II ($p>0.05$). Reduction in the levels of SGOT suggests that there must be some hepatoprotective effects of Pramehhara Kwatha also. On intergroup comparison, there was statistically insignificant difference between group I, II and III ($p>0.05$).

SGPT: In the present clinical study the mean score of SGPT in group I, II and III before treatment was 27.93 IU/L, 35.20 IU/L, 31 IU/L and after treatment it was 25.66 IU/L, 30.60 IU/L and 28.53 IU/L giving 8.11%, 13.06% and 7.9% reduction in mean score respectively which was statistically significant in group I and III ($p<0.05$) and was statistically insignificant in group II ($p>0.05$). Reduction in the levels of SGPT suggests that there must be some hepatoprotective effects of Pramehhara Kwatha also. On intergroup comparison, there was statistically insignificant difference between group I, II and III ($p>0.05$).

DISCUSSION

Probable mode of action of Pramehhara Kwatha can be explained on the following basis: As per the fundamental principles of Ayurveda the mode of action of every drug is determined by the dominant pharmacodynamic factor in that particular drug and that may be Rasa, Guna, Veerya, Vipaka or Prabhava.

In Ayurvedic classics, it has been clearly mentioned that Kapha and Vata play important role in the pathogenesis of Madhumeha. In Sushruta Samhita while describing the principle of management of Madhumeha it has been mentioned that the drugs which have Tikta, Katu, and Kashaya Rasa, Katu Vipaka and Ushna Veerya with Soshaka and Chedana actions are useful in the treatment of Madhumeha. As vitiated Kapha is initiating Dosha in Prameha or Madhumeha and Dushyas are also of the same nature, so the drugs used in Madhumeha treatment must possess the above-mentioned properties in order to maintain equilibrium between Vata and Kapha. In the present study, a combination of 5 herbs was used to assess its efficacy in the management of Madhumeha. Majority of the drugs in the formulation are having Katu and Tikta Rasa. Katu Rasa is Agni Mahabhuta predominant. It has got Deepana, Pachana, Srothoshodhana, Kapha Shamana action and causes Shoshana of Kleda and Meda. Since the Samprapti begins with Kapha Prakopa and Mala Sanchaya hence, Kapha Shamanatva and Srothoshodhana are absolutely essential for Samprapati Vighattan. Also majority of the ingredients of trial formulation possess Laghu and Ruksha Guna which is dominant in Akasha, Agni and Vayu Mahabhuta. It is Laghavakara, Kaphagna and Lekhana. This results in alleviation of aggravated Kapha and causes Apatarpana. It also aids to Deepana and Pachana. Ruksha Guna which is dominant in all the ingredients of trial formulation has Sroto Shodhaka property. Ruksha Guna helps in cleaning all Srotas, therefore, decreases Prabhuta Mutrata as it possesses Dravansh Shoshaka property. Sheeta Guna which is dominant in Amalaki and Mustaka pacifies Pitta which further subsides symptoms like Pipasadhikya, Kshudhadhikya, Kara Pada Daha, Gala Talu Shosha, Swedadhikya and Shithilangata. Ushna Virya of majority of drugs leads to Kapha Shamana. which aids in Srothoshodhana, Anulomana, correction of Agni, Ama Pachana and prevents Kapha and Malasanchaya.

Methanolic extract and chloroform extract of *T. chebula* decrease the blood sugar level. Principal constituents of Vibhitaki are ß- sitosterol, gallic acid leads to increase in the plasma insulin, C-peptide and glucose tolerance levels. Amalaki has high amount of vitamin C content in its fruit which reduces the sugar level in blood. It stimulates the islets of Langerhans *i.e.,* the isolated Group of cells which secrete hormone insulin. The major chemical constituents present in *B. Aristata* plant are alkaloids and the main one *i.e*., isoquinoline berberine which has been shown to affect blood glucose levels. Berberine has also effects on blood glucose level similar to the drug Metformin in improving insulin resistance. The major chemical constituents present in cyperus rotundus are Cyperen 1 and 2, eplerenone, isopatchoulenone significantly lowered blood sugar levels in alloxan- induced hyperglycemic rats.

CONCLUSIONS

After the careful review of the results obtained from the clinical study following conclusion can be drawn:

- The trial drug *i.e.,* Pramehhara Kwatha showed statistically significant results on subjective parameters *i.e.,* Prabhuta Mutrata, Avila Mutrata, Pipasa-Adhikya, Kshuda-Adhikya, Karapada Daha, Swedadikya, Galatalusosha, Madhurasyata, Karapadasuptata and Shithilangata.

- There was statistically highly significant decrease of FBS, PPBS and HbA1c levels in all three groups but maximum decrease was observed in group III, wherein the patients were managed with Pramehhara Kwatha as well as tab. Metformin. This shows the synergistic action of Pramehhara Kwatha along with tab. Metformin in the management of type II diabetes mellitus.
- Various haematological and biochemical parameters *i.e.,* Hbgm%, TLC, DLC, blood urea, serum creatinine and serum lipid profiles remained within the normal limits in all three groups during and after the completion of clinical trial. However, statistically significant reduction of SGOT and SGPT levels after the completion of the trial in group-I and group-III.
- No untoward effects of Pramehhara Kwatha were observed during the entire trial period.
- Thus, on the basis of present clinical study, it may be concluded that Pramehhara Kwatha is efficient in management of Madhumeha and also possess significant hypoglycemic activity.

REFERENCES

[1] American Diabetes Association. Diagnos is and Classification of Diabetes Mellitus. *Diabetes Care* 37(Supplement_1), 2014, S81–S90.

[2] Olokoba AB, et al. Type 2 diabetes mellitus: A review of current trends. *Oman Medical Journal*. Vol. 27, No. 4, 2012, pp. 269.

[3] Piero MN, et al. Diabetes mellitus-a devastating metabolic disorder. *Asian Journal of Biomedical and Pharmaceutical Sciences*. Vol. 4, No. 40, 2015, pp. 1–7.

[4] Deshpande AD, et al. Epidemiology of diabetes and diabetes-related complications. *Physical Therapy.* Vol. 88, No. 11, 2008, 1254-1264.

[5] Lindberg O, et al. Impacts of components of the metabolic syndrome on health status and survival in an aged population. *European Journal of Epidemiology.* Vol. 13, 1997, pp. 429-434.

[6] Zelber-Sagi S, et al. Nutrition and physical activity in NAFLD: An overview of the epidemiological evidence. *World Journal of Gastroenterology: WJG.* Vol. 17, No. 29, 2011, pp. 3377.

[7] Anderson JW, et al. Carbohydrate and fiber recommendations for individuals with diabetes: a quantitative assessment and meta-analysis of the evidence. *Journal of the American College of Nutrition*. Vol. 23, No. 1, 2004, pp. 5-17.

[8] Manson JE, et al. The escalating pandemics of obesity and sedentary lifestyle. A call to action for clinicians. *Archives of Internal Medicine.* Vol. 164, No. 3, 2004, pp. 249-258

[9] Kumar KJ. Effect of geographical variation on contents of tannic acid, gallic acid, chebulinic acid and ethyl gallate in Terminalia chebula. *Natural Products.* Vol. 2, No. (3-4), 2006, pp. 170-175.

[10] Dodke PC, et al. Ayurvedic and modern aspect of Terminalia chebula Retz. Haritaki an overview. *International Journal of Ayurvedic and Herbal Medicine*. Vol. 7, No. 2, 2017, pp. 2508-2517.

[11] Tasduq SA, et al. Terminalia chebula (fruit) prevents liver toxicity caused by sub-chronic administration of rifampicin, isoniazid and pyrazinamide in combination. *Human and Experimental Toxicology*. Vol. 25, No. 3, 2006, pp. 111-118.

[12] Sabu MC, et al. Anti-diabetic activity of medicinal plants and its relationship with their antioxidant property. *Journal of Ethnopharmacology.* Vol. 81, No. 2, 2002, pp. 155-160.

[13] Rao NK, et al. Antidiabetic and renoprotective effects of the chloroform extract of Terminalia chebula Retz. seeds in streptozotocin-induced diabetic rats. *BMC Complementary and Alternative Medicine.* Vol. 6, 2006, pp. 1-6.

[14] Kannan VR, et al. Anti-diabetic activity on ethanolic extracts of fruits of *Terminalia chebula* Retz. Alloxan induced diabetic rats. *American Journal of Drug Discovery and Development.* Vol. 2, No. 3, 2012, pp. 135-142.

[15] Senthilkumar GP, et al. Biochemical studies on the effect of *Terminalia chebula* on the levels of glycoproteins in streptozotocin-induced experimental diabetes in rats. *Journal of Applied Biomedicine.* Vol. 6, No. 2, 2008, pp. 105-115.

[16] Amrithpal Singh. Medicinal Plants of the World, Published by Mohan Primlani for Oxford and IBH Co. Pvt,New Delhi. 2006, 26.

[17] Latha RC, et al. Parameters in Streptozotocin diabetic rats. *International Journal of Pharmacology.* Vol. 6, No. 2, 2010, pp. 89-96.

[18] Jadon A, et al. Protective effect of Terminalia belerica Roxb. and gallic acid against carbon tetrachloride induced damage in albino rats. *Journal of Ethnopharmacology*. Vol. 109, No. 2, 2007, pp. 214-218.

[19] Thilakchand KR, et al. Hepatoprotective properties of the Indian gooseberry (Emblica officinalis Gaertn): A review. *Food and Function.* Vol. 4, No. 10, 2013, pp. 1431-1441.

[20] Kumar KS, et al. Recent trends in potential traditional Indian herbs Emblica officinalis and its medicinal importance. *Journal of Pharmacognosy and Phytochemistry.* Vol. 1, No. 1, 2012, pp. 24-32.

[21] Kim HJ, et al. Influence of amla (*Emblica officinalis* Gaertn.) on hypercholesterolemia and lipid peroxidation in cholesterol-fed rats. *Journal of Nutritional Science and Vitaminology.* Vol. 51, No. 6, 2005, pp. 413-418.