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CASE SERIES

Effect of *Ojomehantak* Tablet and *Mustadi Ghanavati* in Type 2 Diabetes Mellitus with Secondary Failure to Oral Hypoglycemic Agents: An Open-Label Case Series

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a progressive metabolic disorder frequently associated with secondary failure to oral hypoglycemic agents (OHA), leading to inadequate glycemic control and increased risk of complications. Ayurveda correlates T2DM with *Prameha*, characterized by *Kapha* predominance, *Meda dushti*, and impaired *Agni*. The present study evaluated the effect of *Ojomehantak* tablet and *Mustadi Ghanavati* in patients with T2DM exhibiting secondary failure to OHA.

Materials and Methods: An open-label clinical case series was conducted in 10 patients aged 30–65 years with documented secondary failure to OHA. Participants received *Ojomehantak* tablet (500 mg twice daily) and *Mustadi Ghanavati* (500 mg twice daily) for 3 months. Assessment included objective parameters (fasting blood sugar, post-prandial blood sugar, and glycated hemoglobin [HbA1c]) and subjective symptom grading based on classical features of *Prameha*. Paired *t*-test was used for normally distributed biochemical data, and the Wilcoxon signed-rank test for ordinal symptom scores.

Results: Significant reductions were observed in fasting blood glucose (211.79 ± 32.28 to 111.42 ± 18.82 mg/dL; $P < 0.001$), post-prandial glucose (263.81 ± 36.90 to 146.24 ± 16.73 mg/dL; $P < 0.001$), and HbA1c (7.86 ± 1.00% to 5.60 ± 0.46%; $P < 0.001$). Total subjective symptom score significantly decreased (16.8 ± 2.3 to 5.4 ± 1.6; $P < 0.005$) with a large clinical effect size (r = 0.89). No adverse effects were reported during the study period.

Conclusion: The combination of *Ojomehantak* tablet and *Mustadi Ghanavati* demonstrated statistically and clinically significant improvement in glycemic control and symptomatic relief in patients with T2DM and secondary OHA failure. These preliminary findings support further large-scale randomized controlled trials to validate efficacy and safety.

1. INTRODUCTION

Diabetes mellitus has emerged as one of the most prevalent metabolic disorders worldwide and continues to pose a serious public health challenge.[1] It is a chronic condition associated with substantial morbidity, mortality, and long-term complications.[1] The global burden of diabetes is rising rapidly, particularly in developing countries, largely due to sedentary lifestyles, unhealthy dietary practices, obesity, and psychosocial stress.[2] India is among the countries with the highest prevalence of diabetes, with recent estimates indicating that approximately 72.9 million individuals are affected.[2,3]

Diabetes mellitus is characterized by chronic hyperglycemia resulting from impaired insulin secretion, defective insulin action, or a combination of both[4,5]. Persistent elevation of blood glucose levels produces classic symptoms such as polyuria, polydipsia, and polyphagia.[4] Poorly controlled diabetes may lead to serious acute and chronic complications, including diabetic ketoacidosis,

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cardiovascular and cerebrovascular diseases, nephropathy, neuropathy, retinopathy, cataract, glaucoma, and diabetic foot complications.[6]

The conventional management of type 2 diabetes mellitus (T2DM) primarily relies on oral hypoglycemic agents (OHA) and insulin therapy aimed at lowering blood glucose levels.[7] Although these interventions are effective in achieving short-term glycemic control, they often fail to adequately address the underlying metabolic disturbances, particularly insulin resistance.[5] With long-term therapy, many patients develop secondary failure to OHA, requiring dose escalation or initiation of insulin, yet optimal symptom control and prevention of complications are frequently not achieved.[7] This scenario underscores the need for alternative and complementary therapeutic strategies that can provide sustained metabolic regulation and reduce the risk of diabetes-associated complications.

From the *Ayurvedic* perspective, diabetes mellitus can be correlated with *Prameha*, a group of metabolic disorders described in classical texts, characterized by derangement of *Kapha dosha*, *Meda dhatu*, and *Mutravaha srotas*.[8,9] *Ayurvedic* management emphasizes correction of the underlying *dosha* imbalance, improvement of *Agni*, reduction of *Meda*, and normalization of tissue metabolism through *Shamana aushadhi*, along with appropriate dietary and lifestyle modifications.[8,10]

The present case series includes ten patients attending an *Ayurvedic* clinic with clinical features suggestive of *Prameha*. Baseline investigations revealed fasting blood glucose levels ranging from 130 to 250 mg/dL, post-prandial blood glucose levels between 170 and 350 mg/dL, and glycated hemoglobin (HbA1c) values ranging from 6.5% to 10%.[4,7] All patients had a documented history of T2DM with secondary failure to OHA.[6] The therapeutic intervention consisted of *Shamana aushadhi* in the form of *Ojomehantak* tablet and *Mustadi Ghanavati*. Patients were followed up at monthly intervals for a duration of 3 months. At the end of the treatment period, notable improvement was observed in clinical symptoms as well as glycemic parameters.

This case series aims to evaluate the effectiveness of the combined administration of *Ojomehantak* tablet and *Mustadi Ghanavati* in the management of T2DM with secondary failure to OHA.

1.1. Objectives

1. To evaluate the effect of the combined administration of *Ojomehantak* tablet and *Mustadi Ghanavati* on glycemic control in patients of T2DM with secondary failure to OHA
2. To assess the improvement in clinical symptoms of *Prameha* following *Ayurvedic* intervention
3. To observe the safety and tolerability of the selected *Shamana aushadhi* during the study period.

1.2. Hypothesis

- Null Hypothesis (H_0): The conjugated use of *Ojomehantak* tablet and *Mustadi Ghanavati* has no significant effect on glycemic parameters or clinical symptoms in patients of T2DM with secondary failure to OHA
- Alternative Hypothesis (H_1): The conjugated use of *Ojomehantak* tablet and *Mustadi Ghanavati* produces significant improvement in glycemic parameters and clinical features of *Prameha* in patients with T2DM, and secondary failure to OHA.

2. MATERIALS AND METHODS

2.1 Study Design

The present study was an open-label clinical case series conducted to evaluate the effect of *Ojomehantak* tablet and *Mustadi Ghanavati* in patients of T2DM, with secondary failure to OHA.

2.2. Study Setting

The study was carried out in the outpatient department of an Ayurvedic hospital/clinic as a part of routine clinical practice.

2.3. Sample Size

A total of 10 patients diagnosed with T2DM, having secondary failure to OHA, were included in the study.

2.4. Sampling Method

Patients were selected by a purposive sampling method based on inclusion and exclusion criteria.

2.5. Ethical Considerations

Written informed consent was obtained from all patients before initiation of treatment. Patient confidentiality was maintained throughout the study.

2.6. Selection Criteria

2.6.1. Inclusion criteria

1. Diagnosed patients of T2DM
2. Patients falling in the age group of 30–65 years
3. Patients of irrespective gender will be included and who are willing to take medicine
4. Failure to respond to OHA even in maximal doses of combination therapy
5. Fasting blood sugar level (BSL) 130–250 mg/dL, post-prandial BSL 170–300 mg/dL, and HbA1C 6.5–10%.

2.6.2. Exclusion criteria

1. K/c/o acute complications such as ketoacidosis, diabetic coma, acute infections of any part of the body, and Gangrene
2. Pregnancy and lactating mother
3. Patients on insulin and steroid therapy
4. Patients with any major systemic diseases.

2.7. Intervention

All selected patients were administered the following Ayurvedic medicines:

- *Ojomehantak* tablet: 500 mg twice daily (BD)
- *Mustadi Ghanavati***:** 500 mg twice daily (BD).

2.7.1. Content

- *Ojomehantak tablet mentioned in table 1*
- *Mustadi Ghanavati mentioned in table 2*

2.8. Duration of Study

The total duration of treatment was 3 months.

2.9. Follow-up Schedule

Patients were assessed at baseline (Day 0) and subsequently at the end of every month (1st, 2nd, and 3rd month).

2.10. Assessment Criteria

Assessment was done based on subjective and objective parameters. Moreover, changes observed in the pre-treatment and post-treatment days, i.e., on the 0th day and 30th day.

2.10.1. Subjective criteria

Classic symptoms of diabetes mellitus (*Prameha*) were assessed, including in table 3:

2.10.2. Objective criteria

- Fasting blood sugar (FBS)
- Post-prandial blood sugar (PPBS)
- HbA1c.

3. OBSERVATION AND RESULTS

3.1. Effect of Treatment on Subjective Parameters

Data were analyzed using non-parametric methods due to ordinal grading. Pre- and post-treatment comparisons were performed using the Wilcoxon signed rank test, and $P < 0.05$ was considered statistically significant [Table 4].

There was a statistically significant reduction in total subjective symptom score following treatment (baseline 16.8 ± 2.3 vs. post-treatment 5.4 ± 1.6; Wilcoxon signed rank test, Z = −est,do $P < 0.005$). All individual symptom domains, including polyuria, nocturia, polydipsia, polyphagia, burning sensation, excessive sweating, and fatigue, demonstrated significant improvement ($P < 0.01$ for all comparisons).

3.2. Effect of Treatment on Objective Parameters

Treatment was associated with statistically significant reductions in glycemic parameters. Fasting blood glucose decreased from 211.8 ± 32.3 mg/dL at baseline to 111.4 ± 18.8 mg/dL post-treatment (mean difference 100.4 mg/dL; $P < 0.001$). Post-prandial glucose decreased from 263.8 ± 36.9 mg/dL to 146.2 ± 16.7 mg/dL (mean difference 117.6 mg/dL; $P < 0.001$). HbA1c decreased from 7.86 ± 1.00% to 5.60 ± 0.46% (mean difference 2.26%; $P < 0.001$) [Table 5].

Normality testing – Shapiro–Wilk tests confirmed that the paired differences were normally distributed for all outcome variables, supporting the use of paired *t*-tests.

3.3. Descriptive Statistics and Paired Test Results

"Compared with baseline, treatment was associated with significant reductions in fasting plasma glucose (211.8–111.4 mg/dL; mean reduction 100.4 mg/dL; $P < 0.001$), post-prandial glucose (263.8–146.2 mg/dL; mean reduction 117.6 mg/dL; $P < 0.001$), and HbA1c (7.86–5.60%; mean reduction 2.26%; $P < 0.001$)."

4. DISCUSSION

The present study demonstrated statistically significant improvement in both objective and subjective parameters of T2DM, following intervention. Fasting blood glucose, post-prandial glucose, and HbA1c showed marked reductions ($P < 0.001$), indicating effective short-term and long-term glycemic control. The reduction in HbA1c is particularly clinically relevant, as it reflects sustained improvement in average blood glucose levels over the preceding 8–12 weeks.

Subjective symptom scores also showed a significant reduction ($P < 0.005$) with a large effect size (r = 0.89), suggesting not only statistical but strong clinical significance. Improvement in symptoms such as *Prabhuta Mutrata* (polyuria), *Pipasa Vriddhi* (polydipsia), *Kshudha Vriddhi* (polyphagia), and *Anutsaha* (fatigue) indicates better metabolic regulation and symptomatic relief.

From an *Ayurvedic* perspective, T2DM correlates with *Prameha*, primarily involving *Kapha* predominance with *Meda* and *Kleda Dushti.* The observed improvement in polyuria and turbidity of urine reflects regulation of *Kleda*. Reduction in excessive thirst and hunger suggests normalization of Agni and correction of *Dhatvagni* dysfunction. The significant decline in HbA1c supports sustained metabolic correction rather than temporary glycemic suppression.

Overall, the findings suggest that the intervention produced both biochemical and symptomatic benefits, supporting its therapeutic potential in the management of T2DM.

4.1. Mode of Action of *Mustadi Kwath* and *Ojomehantak* Tablet

They address core *doshic* imbalances (*Kapha*, *Meda*, and *Ama*) implicated in *Prameha*. Moreover, they enhance metabolic fire (*Agni*), thereby improving carbohydrate, lipid, and tissue metabolism. Phytoconstituents in their herb combinations have shown anti-hyperglycemic, antioxidant, lipid-modulating, and metabolic enzyme regulatory effects in experimental models, paralleling Ayurvedic therapeutic aims.

4.2. Modern Scientific Validation of Drugs

AMP-activated protein kinase (AMPK) Activation-Berberine (in *Daruharidra*) activates AMPK, improving insulin sensitivity, enhancing glucose uptake, reducing gluconeogenesis and adipogenesis, and improving lipid metabolism. This mirrors metformin's mechanism and counteracts insulin resistance in T2DM.[12]

Glucose transporter type 4 (GLUT4) upregulation-herbal bioactives (e.g., berberine, curcumin, and cinnamon compounds) may enhance GLUT4 expression and translocation, promoting glucose uptake into skeletal muscle and adipose tissue – a key pathway in insulin-mediated glucose disposal.[11]

α-glucosidase and α-amylase inhibition-curcuminoids in turmeric and combinations such as turmeric + emblica exhibit significant α-glucosidase and α-amylase inhibitory activity, slowing carbohydrate breakdown and post-prandial glucose rise.[14] (Lekshmi PC, 2014)

Peroxisome proliferator-activated receptors (PPAR) regulation-berberine and related alkaloids may suppress adipogenesis by downregulating PPAR-γ and associated transcription factors, improving metabolic profile, and reducing insulin resistance.[12]

4.3. Clinical Evidence of Formulation

4.3.1. Mustadi Kwath/Mustadi Ghanavati

- Hyperlipidemia trial: *Mustadi Ghanavati* significantly reduced serum cholesterol (−22.4%), triglycerides (−19.6%), low-density lipoprotein (−18.2%), and increased high-density lipoprotein[11]
- Ayurvedic pharmacology: Reviews suggest anti-hyperglycemic, antioxidant, and anti-hyperlipidemic activities potentially beneficial for metabolic disorders, including *Prameha*[11]
- Clinical diabetes study: A large unpublished NISCAIR study suggests that *Mustadi Kwatha Ghanavati* improved metabolic profiles and symptomatic distress in T2DM patients, indicating clinical potential.[15]

5. CONCLUSION

The present open-label case series titled "Effect of *Ojomehantak* Tablet and *Mustadi Ghanavati* in T2DM with Secondary Failure to Oral Hypoglycemic Agents" demonstrated statistically and clinically meaningful improvement in patients who exhibited inadequate glycemic control with conventional OHA. Significant reductions were observed in fasting blood glucose, post-prandial glucose, and HbA1c levels, along with marked improvement in classic subjective symptoms. These findings suggest that the combination therapy may offer metabolic stabilization and symptomatic relief in cases of secondary drug failure. The improvement in HbA1c indicates sustained glycemic regulation rather than short-term fluctuation, while the reduction in subjective scores reflects enhancement in functional well-being and quality of life. From an integrative perspective, the intervention appears to support correction of metabolic imbalance and improvement in systemic regulation.

5.1. Strengths

- Demonstrated statistically significant improvement in both objective and subjective parameters
- Included patients with secondary failure to OHA, a clinically challenging group
- Use of validated biochemical markers (FBS, PPBS, and HbA1c)
- Assessment of symptom severity through a structured grading scale
- A large clinical effect size was observed in subjective parameters.

5.2. Limitations

- Small sample size (n = 10)
- Open-label design without blinding
- Absence of control or comparator group
- Short duration of follow-up
- Lack of long-term safety evaluation and biochemical mechanistic studies.

5.3. Future Scope of the Study

- Conduct randomized controlled trials with larger sample sizes
- Include comparator arm (standard care vs. integrative therapy)
- Long-term follow-up to assess the sustainability of glycemic control
- Evaluation of insulin resistance markers and inflammatory biomarkers
- Pharmacodynamic and mechanistic studies to explore the mode of action
- Quality-of-life assessment using validated scales
- Multicentric trials for broader generalizability.

5.4. Overall Conclusion

Within the limitations of an open-label case series, *Ojomehantak* tablet and *Mustadi Ghanavati* appear to provide beneficial effects in patients with T2DM exhibiting secondary failure to OHA. The findings justify further well-designed controlled clinical trials to confirm efficacy, safety, and mechanistic pathways.

6. ACKNOWLEDGMENTS

Nil.

7. AUTHORS' CONTRIBUTIONS

All authors give equal contribution in making of this manuscript.

8. FUNDING

Nil.

9. ETHICAL STATEMENT

Written informed consent was obtained from all patients before initiation of treatment. Patient confidentiality was maintained throughout the study.

10. CONFLICT OF INTERESTS

The authors declare no conflicts of interest regarding the publication of this paper.

11. DATA AVAILABILITY STATEMENT

The data analyzed in this review were obtained from publicly available sources, including peer-reviewed articles, observational studies, and surveys accessible through databases.

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Table 1: Content of *Ojomehantak* tablet

S. No.	Name	Latin name	Used part	Quantity
1.	*Haridra*	*Curcuma longa*	*Kanda*	Equal
2.	*Katphal*	*Strychnos potatorum*	*Beej*	Equal
3.	*Paranti*	*Ixora coccinia*	*Moola*	Equal
4.	*Amalaki*	*Emblica officinalis*	*Phal*	Equal
5.	*Lodhra*	*Symplocos racemosa*	*Twak*	Equal
6.	*Meshshrungi*	*Gymnema sylvestre*	*Patra*	Equal
7.	*Saptrangi*	*Salacia oblonga*	*Patra*	Equal
8.	*Tondali*	*Coccinia grandis*	*Phal*	Equal
9.	*Vijaysar kwath*	*Pterocarpus marsupium*	*Twak*	Equal
10.	*Haridra kwath*	*Curcuma longa*	*Kanda*	Equal
11.	*Hari Mirchi swaras*	*Capsicum annuum*	*Phal*	Equal

Table 2: Content of *Mustadi Ghanavati*

S. No	Name	Latine name	Used part	Quantity
1.	*Musta*	*Cyperus rotundus*	*Kanda*	Equal
2.	*Argavdha*	*Cassia fistula*	*Twak*	Equal
3.	*Patha*	*Cissampelos pereira*	*Moola*	Equal
4.	*Haritaki*	*Terminalia chebula*	*Phal majja*	Equal
5.	*Amalaki*	*Emblica officinalis*	*Phal majja*	Equal
6.	*Bibhitaki*	*Terminalia bellerica*	*Phal majja*	Equal
7.	*Deodaru*	*Cedrus deodaru*	*Twak*	Equal
8.	*Gokshur*	*Tribulus terrestris*	*Phal*	Equal
9.	*Khadir*	*Acacia catechu*	*Twak*	Equal
10.	*Nimba*	*Azadiracta indica*	*Twak*	Equal
11.	*Haridra*	*Curcuma longa*	*Kanda*	Equal
12.	*Daruharidra*	*Berberis aristata*	*Kanda*	Equal
13.	*Kutaj*	*Holarrhena antidysenterica*	*Twak*	Equal

Table 3: Subjective parameters

S. No.	Symptoms	Grade 0	Grade 1	Grade 2	Grade 3
1.	*Prabhut Mutrata*	<6 times/day	6–8 times/day	8–10 times/day	>10 times/day
2.	*Avil mutrata*	Transparent and clear urine	Transparent but with suspended particles	Turbid with slight precipitation	Homogeneous turbid urine
3.	*Nakta mutrata*	Occasion	<2 times/night	2–4 times/night	>4 times/night
4.	*Pipasa vruddhi*	No urge of thirst	Urge of thirst every 3–4 h	Urge of thirst every 1–2 h	Urge of thirst persists
5.	*Kshudha vruddhi*	4 times/24 h	5–6 times/24 h	6–7 times/24 h	7–8 times/24 h
6.	*Hast pad tal dah*	Absent	Occasional	Persistent but bearable	Persistent but not bearable
7.	*Swedati pravrutti*	Absent	Excessive sweating on exertion	Excessive sweating on slight exertion	Excessive sweating at rest
8.	*Anutstah* (fatigue)	No fatigue	Fatigue occurs after 12 h of work	Fatigue after 30 min of work	Fatigue after 10–15 min of work

Table 4: Effect of treatment on subjective parameters

Symptom	Baseline Mean±SD	After treatment Mean±SD	Z-value	*P*-value	Significance
Prabhuta Mutrata	2.4±0.5	0.8±0.6	−2.812	0.004	Significant
Avila Mutrata	2.1±0.6	0.7±0.5	−2.701	0.006	Significant
Nakta Mutrata	2.0±0.7	0.6±0.5	−2.636	0.008	Significant
Pipasa Vriddhi	2.3±0.6	0.9±0.5	−2.807	0.005	Significant
Kshudha Vriddhi	2.2±0.4	0.8±0.4	−2.803	0.005	Significant
Hasta-Pada-Tala Daha	2.0±0.6	0.5±0.5	−2.701	0.006	Significant
Swedati Pravritti	1.9±0.7	0.6±0.5	−2.636	0.008	Significant
Anutsaha (*Fatigue*)	2.3±0.5	0.7±0.6	−2.812	0.004	Significant

SD: Standard deviation

Table 5: Effect of Treatment on Objective Parameters

Variable	Baseline Mean±SD	Post-treatment Mean±SD	Mean difference	*t*- vale	*P*-value
Fasting glucose	211.79±32.28	111.42±18.82	100.37	13.08	<0.001
Post-prandial glucose	263.81±36.90	146.24±16.73	117.58	11.68	<0.001
Glycated hemoglobin	7.86±1.00	5.60±0.46	2.26	10.46	<0.001

SD: Standard deviation

Table 6: Pharmacological actions of some drugs

Herb	Primary phytochemicals	Traditional ayurvedic role	Modern pharmacological actions
Musta (*Cyperus rotundus*)	Cyperene, flavonoids	*Agnideepana*, *Kapha-Medohara*	May support digestion, lipolysis, and metabolic balance[11]
Daruharidra (*Berberis aristata*)	Berberine	*Kapha-Medohara*, *Anti-Ama*	Lowers glucose, improves insulin sensitivity through AMPK activation, anti-inflammatory and antioxidant effects[12]
Triphala (Haritaki, Bibhitaki, Amalaki)	Tannins, gallic acid, Vitamin C	*Rasayana*, *Ama pachana*	Antioxidant, anti-glycation, lipid-lowering, supports glucose metabolism[12]
Patha (*Cissampelos pareira*)	Alkaloids	*Mutravaha srotas* support	Experimental evidence suggests metabolic support activity[13]
Haridra (*Curcuma longa*)	Curcuminoids (curcumin)	*Deepana*, anti-inflammatory	α-glucosidase and α-amylase inhibition, antioxidant, supports glucose uptake and insulin signaling[14]
Amalaki (*Emblica officinalis*)	Gallic acid, tannins	*Rasayana*, metabolic detox	Supports β-cell function, antioxidant, and glucose metabolism[14]
Kataka (*Strychnos potatorum*)	Saponins, glycosides	*Kapha-Medohara*	Traditional support for metabolic disorders (specific modern evidence is limited)
Lodhra (*Symplocos racemosa*)	Tannins	*Kapha-Pitta balance*	Antioxidant, support for metabolic homeostasis[5]
Usira (*Vetiveria zizanioides*)	Essential oils	*Cooling, metabolic support*	Traditionally supports fluid balance and detox

AMPK: AMP-activated protein kinase