WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
Coden USA: WJPRAP Impact Factor 8.63
Volume 15, Issue 9, 1661-1674. Research Article ISSN 2277–7105

AYURVEDIC PANCHAKARMA-BASED MULTIMODAL INTERVENTION FOR TYPE 2 DIABETES: GLYCEMIC CONTROL AND ANTIDIABETIC DRUG REDUCTION IN 67 PATIENTS — A RETROSPECTIVE ANALYSIS

***Dr. Bipin Gond, Dr. Prajakta Asnodkar**

Vaidya Sane Ayurved Laboratories Limited.

Article Received on 05 April 2026,
Article Revised on 25 April 2026,
Article Published on 04 May 2026,

https://doi.org/10.5281/zenodo.20033591

***Corresponding Author**

Dr. Bipin Gond
Vaidya Sane Ayurved Laboratories Limited.
drbipin.madhavbaug@gmail.com

How to cite this Article: Dr. Bipin Gond, Dr. Prajakta Asnodkar. (2026). Ayurvedic Panchakarma-Based Multimodal Intervention For Type 2 Diabetes: Glycemic Control and Antidiabetic Drug Reduction In 67 Patients — A Retrospective Analysis. World Journal of Pharmaceutical Research, 15(9), 1661–1674.

ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a growing metabolic burden in India, with conventional pharmacotherapy achieving suboptimal glycemic targets while requiring lifelong medication escalation. Ayurveda conceptualizes Prameha — the classical analogue of diabetes mellitus — as a disorder rooted in Kapha-Medovaha Srotas dysfunction, amenable to intervention through Panchakarma, dietary correction, and herbal formulations. Real-world evidence from structured Ayurvedic multimodal protocols remains limited. **Objectives:** To evaluate the effect of a structured integrative Ayurvedic protocol — comprising BMI-stratified Panchakarma therapy (CDC-SP and CDC-KP), an 800 kcal low-carbohydrate Prameha diet, and individualized herbal medication — on glycemic, anthropometric, cardiometabolic, and insulin resistance parameters, and to quantify reduction in antidiabetic medications. **Methods:** Retrospective observational study of 67 T2DM patients treated at Madhavbaug Clinics, Mira Road. Panchakarma comprised Snehan (Neem Siddha Taila), Swedan (Dashmula Kwath), and Basti (Gudmar, Daru Haridra, Yashti Madhu) — Kwath-based in CDC-SP (BMI ≥23) or oil-based in CDC-KP (BMI <23). Outcomes included HbA1c, RBS, BMI, abdominal girth, blood pressure, lipid profile, TG/HDL ratio, and antidiabetic medication status. Paired t-tests were used for within-group comparisons. **Results:** Significant improvements (all $p<0.001$) were seen across all primary parameters. HbA1c declined from 9.37 ± 2.39% to 6.72 ± 1.05% (Δ −2.65%). RBS reduced by 50.7% (254 → 125 mg/dL). TG/HDL ratio — a key surrogate

of insulin resistance — improved from 5.11 to 3.15 (Δ −26.4%, p<0.001); 52.4% of patients shifted to a lower cardiovascular risk category, and 71.4% achieved TG/HDL <3.5 post-treatment. Regarding antidiabetic medications: 50.0% achieved complete cessation and 33.3% partial reduction (total 83.3% with drug reduction). **Conclusion:** A structured Ayurvedic multimodal protocol produced clinically meaningful improvements in glycemic, cardiometabolic, and insulin resistance parameters, with 83.3% of patients achieving partial to complete antidiabetic drug reduction. The TG/HDL ratio analysis reveals simultaneous reversal of insulin resistance — a finding with important cardiovascular implications. Prospective controlled trials are warranted.

KEYWORDS: *Type 2 diabetes mellitus, Panchakarma, Prameha, Ayurveda, Basti, TG/HDL ratio, insulin resistance, HbA1c, antidiabetic drug reduction, very-low-calorie diet, Gudmar.*

1. INTRODUCTION

Type 2 diabetes mellitus (T2DM) has reached epidemic proportions globally, with India now home to over 101 million people living with diabetes — approximately 17% of the world's diabetic population.[1] The condition compounds its burden through cardiometabolic comorbidities: hypertension, dyslipidemia, central obesity, and markedly elevated cardiovascular risk. Conventional pharmacotherapy, while effective in glycemic reduction, is associated with cumulative polypharmacy, dose escalation, and inadequate targeting of the underlying metabolic dysfunction.[2]

Ayurveda describes *Prameha* — with *Madhumeha* as its most severe form — as a disorder rooted in *Kapha-Meda* accumulation obstructing the *Medovaha Srotas* (lipid-metabolic channels), resulting in impaired tissue metabolism and glucose dysregulation.[3] The *Charaka Samhita* stratifies management by body habitus: *Sthula Pramehin* (obese) requires vigorous *Shodhana* (purificatory) therapy; *Krisha Pramehin* (lean) requires *Brimhana* (nourishing) management — a BMI-stratified approach that forms the conceptual backbone of the CDC protocol.[4]

Panchakarma — encompassing *Snehan*, *Swedan*, and *Basti* — is Ayurveda's most potent therapeutic modality for metabolic disease. The herbal constituents of the Basti preparation — *Gudmar* (*Gymnema sylvestre*), *Daru Haridra* (*Berberis aristata*), and *Yashti Madhu* (*Glycyrrhiza glabra*) — possess well-documented antihyperglycemic, AMPK-activating, and insulin-sensitizing properties.[5,6,7]

A dimension of particular clinical importance in T2DM is the insulin-resistant lipid phenotype, characterized by elevated triglycerides, low HDL, and small dense LDL particles. The TG/HDL ratio has emerged as a reliable, widely available surrogate marker of insulin resistance and atherogenic dyslipidemia.[13] — more reflective of the metabolic milieu than LDL alone. Reducing TG/HDL ratio thus represents a meaningful surrogate for cardiovascular risk modification beyond glycemic control.

The DiRECT trial established that an 825–853 kcal/day formula diet achieved T2DM remission in 46% of participants at one year.[8] The integration of a low-carbohydrate, high-protein dietary strategy with Panchakarma represents a potentially synergistic approach addressing both glycemic load and underlying metabolic dysregulation. The present study reports outcomes from 67 T2DM patients treated with the CDC protocol, examining glycemic, anthropometric, cardiometabolic, TG/HDL ratio, and pharmacological outcomes.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

Retrospective observational study conducted at Madhavbaug Clinics, Mira Road branch, Maharashtra, India. Data were extracted from electronic patient records of individuals enrolled in the CDC (Comprehensive Diabetes Care) diabetes management program. The study was conducted in accordance with the Declaration of Helsinki principles for retrospective clinical research.

2.2 Study Participants

Patients with confirmed T2DM who completed at least one CDC intervention cycle with documented pre- and post-treatment clinical parameters were included. Patients lacking baseline HbA1c or fasting blood glucose values were excluded. Sixty-seven patients met inclusion criteria.

2.3 Intervention Protocol

2.3.1 BMI-Stratified Panchakarma Therapy

The CDC protocol stratifies by baseline BMI, aligning with the classical *Sthula/Krisha Pramehin* distinction

- CDC-SP (Shodhana Protocol, BMI ≥23 kg/m², n=41): Intensive bio-purificatory Panchakarma to reduce Kapha-Meda accumulation and clear Srotovarodha.

- CDC-KP (Brimhana Protocol, BMI <23 kg/m², n=26): Nourishing-supportive Panchakarma to strengthen Dhatu metabolism without excessive depletion.

Components (both protocols): *Snehan* — External Abhyanga with Neem Siddha Taila (*Azadirachta indica*); *Swedan* — Medicated steam with Dashmula Kwath; *Basti* — Kwath-based preparation (CDC-SP) or oil-based preparation (CDC-KP) of Gudmar, Daru Haridra, and Yashti Madhu. Mean 8.7 ± 2.7 sessions administered; 46 patients (68.7%) completed the full 10-session cycle.

2.3.2 Prameha Diet Box

Standardized ready-to-use meal providing ~800 kcal/day: low carbohydrate, high protein, moderate fat — consistent with Indian food preferences and Ayurvedic dietary principles for Prameha management.

2.3.3 Individualized Herbal Medication

Oral herbal medications prescribed based on individual *Prakriti*, *Vikriti*, and comorbidity profile. Common formulations included Gudmar, Vijayasar (*Pterocarpus marsupium*), Haridra (*Curcuma longa*), and Triphala. All purely herbal.

2.4 Outcome Measures

Primary: HbA1c (%) and antidiabetic medication status. Secondary: RBS (mg/dL), BMI (kg/m²), body weight (kg), abdominal girth (cm), SBP and DBP (mmHg), heart rate (bpm), total cholesterol, triglycerides, LDL, HDL (all mg/dL), and TG/HDL ratio. The TG/HDL ratio was analyzed as a surrogate of insulin resistance and cardiovascular risk, with the following classification: Low (<2.0), Borderline (2.0–3.5), High (3.5–5.0), Very High (>5.0). Drug reduction was categorized as complete cessation (100%), partial (1–99%), or none (0%).

2.5 Statistical Analysis

Data analyzed using Python (pandas, scipy.stats). Descriptive statistics as mean ± SD. Within-group comparisons by paired Student's t-test (two-tailed, significance $p<0.05$). Subgroup analyses for CDC-SP vs. CDC-KP, and male vs. female. Pearson correlations assessed for TG/HDL change vs. baseline parameters. Risk category reclassification was analyzed as absolute patient counts and proportions.

3. RESULTS

3.1 Baseline Characteristics

Sixty-seven patients with T2DM were included (44 male, 23 female; mean age 48.1 ± 8.5 years; range 31–69). Comorbidities included hypertension (n=11, 16.4%), dyslipidemia (n=10, 14.9%), and IHD (n=3, 4.5%). BMI stratification: 41 patients to CDC-SP (BMI ≥23), 26 to CDC-KP (BMI <23). Mean follow-up 94 ± 74 days (median 80 days, IQR 40–122). Baseline HbA1c: 11 patients (16.4%) controlled (<7%), 25 (37.3%) at 7–9%, 20 (29.9%) at 9–12%, and 10 (14.9%) severe (≥12%).

Table 1: Baseline Characteristics.

Parameter	Overall (n=67)	CDC-SP (n=41)	CDC-KP (n=26)
Age (years)	48.1 ± 8.5	46.5 ± 8.3	50.7 ± 8.3
Sex (M/F)	44/23	22/19	22/4
BMI (kg/m²)	28.74 ± 5.49	30.95 ± 4.21	25.25 ± 4.89
Weight (kg)	78.1 ± 15.8	84.2 ± 14.9	68.8 ± 13.1
HbA1c (%)	9.37 ± 2.39	8.72 ± 2.21	10.49 ± 2.38
RBS (mg/dL)	254.0 ± 92.4	236.9 ± 84.3	282.0 ± 99.8
SBP (mmHg)	140.7 ± 20.4	138.0 ± 20.4	145.0 ± 20.0
Abdominal Girth (cm)	101.9 ± 13.3	105.9 ± 12.2	95.6 ± 13.1
Triglycerides (mg/dL)	204.6 ± 141.4	189.2 ± 122.0	228.0 ± 164.2
LDL (mg/dL)	124.3 ± 41.9	119.3 ± 38.6	132.0 ± 46.4
TG/HDL Ratio	5.11 ± 3.81	4.60 ± 3.08	5.88 ± 4.66
Mean PK Sessions	8.7 ± 2.7	—	—

3.2 Primary Outcomes: Glycemic and Cardiometabolic Parameters

Statistically significant improvements were observed across all primary and secondary parameters (Table 2). HbA1c declined from 9.37 ± 2.39% to 6.72 ± 1.05% (Δ −2.65 ± 1.93%, $p<0.001$), a 28.3% relative reduction. The post-treatment mean of 6.72% approaches the ADA threshold for diabetes remission (<6.5%).[12] RBS reduced by 50.7% (254.0 → 125.2 mg/dL, $p<0.001$). Significant improvements in anthropometric and hemodynamic parameters were also observed: BMI −1.82 kg/m², abdominal girth −6.6 cm, SBP −17.6 mmHg, and DBP −9.6 mmHg (all $p<0.001$). Lipid parameters showed significant reductions in total

cholesterol (−21.2%), triglycerides (−31.2%), and LDL (−23.8%), all p<0.001. HDL showed no significant change (Δ −0.1 mg/dL, p=0.948).

Table 2: Primary and Secondary Outcomes — Pre- vs. Post-Intervention (n=67).

Parameter	Pre (Mean ± SD)	Post (Mean ± SD)	Δ Mean ± SD	% Change	p-value
HbA1c (%)	9.37 ± 2.39	6.72 ± 1.05	−2.65 ± 1.93	−28.3%	<0.001
RBS (mg/dL)	254.0 ± 92.4	125.2 ± 33.8	−128.8 ± 80.5	−50.7%	<0.001
Weight (kg)	78.1 ± 15.8	73.6 ± 14.7	−4.5 ± 2.8	−5.7%	<0.001
BMI (kg/m²)	28.74 ± 5.49	26.92 ± 5.05	−1.82 ± 1.12	−6.2%	<0.001
Abdominal Girth (cm)	101.9 ± 13.3	95.4 ± 11.4	−6.6 ± 3.9	−6.3%	<0.001
SBP (mmHg)	140.7 ± 20.4	123.1 ± 10.3	−17.6 ± 20.1	−11.1%	<0.001
DBP (mmHg)	88.5 ± 9.7	78.9 ± 6.8	−9.6 ± 10.3	−10.1%	<0.001
Heart Rate (bpm)	87.0 ± 11.1	82.9 ± 9.5	−4.1 ± 9.6	−4.7%	0.005
Total Cholesterol	203.6 ± 53.7	164.1 ± 39.9	−39.5 ± 36.9	−21.2%	<0.001
Triglycerides (mg/dL)	204.6 ± 141.4	127.3 ± 68.4	−77.3 ± 113.9	−31.2%	<0.001
LDL (mg/dL)	124.3 ± 41.9	96.0 ± 35.7	−28.3 ± 26.2	−23.8%	<0.001
HDL (mg/dL)	42.1 ± 8.6	42.0 ± 6.9	−0.1 ± 8.3	−0.2%	0.948 (ns)
TG/HDL Ratio	5.11 ± 3.81	3.15 ± 1.79	−1.96 ± 3.28	−26.4%	<0.001

3.3 Antidiabetic Medication Reduction

Antidiabetic medication status was documented in 66 patients. Complete cessation of all antidiabetic medications was achieved by 33 patients (50.0%); 22 (33.3%) achieved partial dose reduction; and 11 (16.7%) had no change. Collectively, 83.3% of patients achieved some degree of antidiabetic drug reduction. Mean medication reduction was 72.4% (median 95%).

Table 3: Antidiabetic Medication Reduction.

Category	n	% of Cohort	Clinical Interpretation
Complete cessation (100%)	33	50.0%	All antidiabetic drugs stopped
Partial reduction (1–99%)	22	33.3%	Dose or number of drugs reduced
No change (0%)	11	16.7%	Medications unchanged
Any reduction (≥1%)	55	83.3%	Clinically meaningful drug reduction

3.4 TG/HDL Ratio Analysis: Insulin Resistance and Cardiovascular Risk Reclassification

The TG/HDL ratio — a validated surrogate of insulin resistance and atherogenic dyslipidemia.[13] — was available for 63 patients and is analyzed in detail here given its dual significance as a metabolic and cardiovascular risk marker.

3.4.1 Overall TG/HDL Reduction

Mean TG/HDL ratio declined from 5.11 ± 3.81 to 3.15 ± 1.79 (Δ −1.96 ± 3.28, $p<0.001$), representing a 26.4% relative reduction. The median reduction was 25.3%, confirming the effect is not driven by outliers. 87.3% of patients (55/63) showed numerical improvement in TG/HDL ratio.

3.4.2 Risk Category Reclassification

Patients were classified into four risk categories based on established TG/HDL thresholds[13] (Table 4). The most striking finding is the near-elimination of the Very High risk category: 23 patients (36.5%) began in the Very High (>5) category; only 5 (7.9%) remained there post-treatment — a 78.3% relative reduction in this highest-risk group.

The proportion in the optimal Borderline-to-Low range (TG/HDL <3.5) increased from 39.7% to 71.4% of the cohort post-treatment.

Table 4: TG/HDL Risk Category Distribution — Pre vs. Post Treatment (n=63).

Risk Category	TG/HDL Threshold	Pre-Treatment n (%)	Post-Treatment n (%)	Absolute Change
Low (Optimal)	< 2.0	7 (11.1%)	13 (20.6%)	↑ +6 patients
Borderline	2.0 – 3.5	18 (28.6%)	32 (50.8%)	↑ +14 patients
High	3.5 – 5.0	15 (23.8%)	13 (20.6%)	↓ −2 patients
Very High	> 5.0	23 (36.5%)	5 (7.9%)	↓ −18 patients
TG/HDL < 3.5 (total)		25 (39.7%)	45 (71.4%)	↑ +20 patients

3.4.3 Patient-Level Category Shifts

Overall, 52.4% of patients (33/63) shifted to a lower cardiovascular risk category, 42.9% remained in the same category, and only 4.8% (3 patients) moved to a higher category (Table 5).

Among the 23 patients starting at Very High (>5): 18 (78.3%) moved out of the Very High category post-treatment — 10 to Borderline, 7 to High, and 1 to Low. Their mean post-treatment TG/HDL was 4.30, representing a mean reduction of 3.09 points in this highest-risk subgroup.

Table 5: Patient-Level TG/HDL Risk Category Shift.

Category Shift	n	% of Cohort	Clinical Significance
Moved to lower risk category	33	52.4%	Meaningful cardiovascular risk reduction
Remained in same category	27	42.9%	Stable (many already in Low/Borderline)
Moved to higher risk category	3	4.8%	Non-responders
Achieved TG/HDL <3.5 (insulin resistance threshold)	45	71.4%	Insulin resistance phenotype resolved
Achieved TG/HDL <2.0 (optimal)	13	20.6%	Optimal metabolic state reached
Patients starting Very High (>5) who normalized	18/23	78.3%	Highest-risk group improvement

3.4.4 Subgroup Analysis

Both treatment subgroups achieved significant TG/HDL reduction (Table 6). The CDC-KP group (lower BMI, oil-based Basti) demonstrated more than double the absolute TG/HDL reduction compared to CDC-SP despite a shorter treatment window, consistent with its greater glycemic response observed in Section 3.5.

Table 6: TG/HDL Ratio — CDC-SP vs. CDC-KP and Sex Subgroups.

Subgroup	n	Pre TG/HDL (Mean)	Post TG/HDL (Mean)	Δ (Mean ± SD)	p-value
CDC-SP	38	4.60	3.28	−1.32 ± 2.34	0.001
CDC-KP	25	5.88	2.96	−2.92 ± 4.20	0.002
Male	42	5.04	3.10	−1.94 ± 3.38	<0.001
Female	21	5.25	3.26	−2.00 ± 3.12	0.004
Overall	63	5.11	3.15	−1.96 ± 3.28	<0.001

3.4.5 Correlations

Two clinically relevant correlations were identified: (1) Baseline TG/HDL ratio correlated positively with baseline HbA1c (r=0.338, p=0.007), confirming that the degree of insulin resistance — as reflected by atherogenic dyslipidemia — tracked with glycemic severity in this cohort. (2) Baseline BMI correlated positively with TG/HDL change (r=0.268, p=0.034), indicating that higher BMI patients showed relatively less TG/HDL improvement, consistent with the role of visceral adiposity in driving persistent hypertriglyceridemia.[17]

3.5 Subgroup Analysis: CDC-SP vs. CDC-KP

Both treatment groups demonstrated statistically significant improvements across all major parameters (Table 7). The CDC-KP group showed numerically greater glycemic and lipid improvement across all measured outcomes.

Table 7: Subgroup Outcomes — CDC-SP vs. CDC-KP.

Parameter	CDC-SP (n=41) Pre → Post (Δ)	CDC-KP (n=26) Pre → Post (Δ)	p (SP)	p (KP)
HbA1c (%)	8.72 → 6.56 (Δ −2.16)	10.49 → 7.00 (Δ −3.49)	<0.001	<0.001
RBS (mg/dL)	236.9 → 123.8 (Δ −113.2)	282.0 → 127.6 (Δ −154.4)	<0.001	<0.001
BMI (kg/m²)	30.95 → 28.85 (Δ −2.10)	25.25 → 23.87 (Δ −1.37)	<0.001	<0.001
Abdominal Girth (cm)	105.9 → 98.9 (Δ −7.1)	95.6 → 89.9 (Δ −5.7)	<0.001	<0.001
SBP (mmHg)	138.0 → 122.1 (Δ −15.9)	145.0 → 124.7 (Δ −20.2)	<0.001	<0.001
Triglycerides (mg/dL)	189.2 → 131.7 (Δ −57.6)	228.0 → 120.7 (Δ −107.3)	<0.001	0.001
LDL (mg/dL)	119.3 → 94.8 (Δ −24.5)	132.0 → 97.8 (Δ −34.2)	<0.001	<0.001
TG/HDL Ratio	4.60 → 3.28 (Δ −1.32)	5.88 → 2.96 (Δ −2.92)	0.001	0.002

4. DISCUSSION

4.1 Glycemic Outcomes in Context

The mean HbA1c reduction of 2.65% from a baseline of 9.37% is clinically substantial. GLP-1 receptor agonists and SGLT-2 inhibitors — the most potent antidiabetic pharmacotherapy — typically achieve HbA1c reductions of 1.0–1.5% as monotherapy.[11] The post-treatment mean of 6.72% approaches the ADA threshold for diabetes remission (<6.5% sustained

without pharmacotherapy).[12] That 50% of patients achieved complete antidiabetic drug cessation with this glycemic outcome indicates that the improvement was sustained in the absence of — and in many cases enabling the withdrawal of — conventional pharmacotherapy.

The RBS reduction of 50.7% (254 → 125 mg/dL) aligns with the glucodynamic properties of the Basti constituents: *Gymnema sylvestre* (Gudmar) demonstrates pancreatic beta-cell regeneration and reduced intestinal glucose absorption.[5] berberine from *Berberis aristata* activates AMPK, mimicking metformin's mechanism.[6] and *Glycyrrhiza glabra* modulates insulin signaling and reduces hepatic gluconeogenesis.[7]

4.2 TG/HDL Ratio: Insulin Resistance Reversal and Cardiovascular Risk Implications

The TG/HDL ratio analysis reveals a dimension of this intervention that extends well beyond glycemic control. The ratio is a robust, cost-free surrogate of insulin resistance[13] — particularly relevant in Indian patients where small-dense LDL atherogenicity and insulin resistance frequently precede clinical hyperglycemia by years.[18]

The baseline mean TG/HDL of 5.11 — with 36.5% of patients in the Very High (>5) category — reflects a cohort with profound insulin resistance and high atherogenic risk at enrollment. The reduction to 3.15 post-treatment, with 71.4% achieving TG/HDL <3.5 (the threshold below which insulin resistance is considered unlikely.[13] signals not merely lipid improvement but a fundamental shift in the metabolic phenotype. The 52.4% of patients who crossed into a lower risk category, and the 78.3% of Very High-risk patients who normalized out of that category, are findings with tangible cardiovascular prognosis implications.

The positive correlation between baseline TG/HDL and baseline HbA1c ($r=0.338$, $p=0.007$) provides biological coherence: in this cohort, insulin resistance — as expressed through atherogenic dyslipidemia — tracked directly with glycemic severity.

This reinforces the Ayurvedic conceptualization of *Prameha* as a unified disorder of *Kapha-Meda* metabolism, rather than a condition limited to glucose metabolism alone. The CDC protocol, by targeting *Srotovarodha* (channel obstruction) and *Ama* (metabolic endotoxins) through Panchakarma, appears to operate on this shared upstream metabolic pathway — explaining why improvements are seen simultaneously across glycemic, lipid, and anthropometric dimensions.

The differential TG/HDL response between CDC-SP and CDC-KP (Δ −1.32 vs. Δ −2.92) parallels the glycemic differential and may reflect distinct pharmacological mechanisms of Kwath vs. oil-based Basti delivery. The *Anuvasana Basti* (oil-based) may offer lipid-carrying vehicles for fat-soluble phytoconstituents that optimize colonic absorption of herbal actives with lipid-metabolic activity. This hypothesis warrants pharmacokinetic investigation in future studies.

4.3 Cardiometabolic Breadth: Beyond Glycemia

The simultaneous improvement in blood pressure (SBP −17.6 mmHg), lipid parameters, abdominal girth (−6.6 cm), and TG/HDL ratio signals that this protocol addresses the full cardiometabolic cluster — not glycemia in isolation. This is clinically important: cardiovascular disease remains the leading cause of death in T2DM patients.[2] and risk reduction requires targeting the entire syndrome. The magnitude of SBP reduction is comparable to antihypertensive monotherapy, likely reflecting combined effects of weight loss, dietary sodium modification, and the sympatholytic effects of *Swedan* and *Snehan*.

The absence of significant HDL change (p=0.948) is a recognized limitation and is consistent with short-term cardiometabolic intervention studies — HDL improvement typically requires longer intervention periods and sustained weight loss.[15] Longer follow-up data would be informative in this regard.

4.4 The Basti Mechanism: A Pharmacological Perspective

The per-rectal Basti route warrants pharmacological attention. The colonic mucosa provides direct portal venous drainage, bypassing first-pass hepatic metabolism for many active phytoconstituents. This may explain the potency of herbal Basti formulations. The classical dictum — *Bastir eva tu Shashtham Cha Karma Pradhanamatam* (Basti constitutes half of all Panchakarma measures).[16] — reflects its central therapeutic role, now supported by the mechanistic plausibility of berberine, gymnemic acids, and glycyrrhizin reaching systemic and hepatic circulation via colonic absorption.

4.5 Drug Reduction as a Patient-Centered Outcome

That 50% of patients ceased all antidiabetic medications and 83.3% achieved measurable drug reduction represents a clinically and economically meaningful outcome rarely reported as a primary endpoint in integrative diabetes trials. Drug reduction matters for economic reasons (long-term medication costs in India[17]), physiological reasons (polypharmacy-

associated adverse effects and drug interactions), and psychological reasons (medication cessation is a tangible quality-of-life improvement for patients who associate pill burden with disease severity).

4.6 Limitations

The following limitations must be acknowledged

- Retrospective observational design without a control group: Precludes definitive causal attribution.
- Follow-up heterogeneity: Wide duration range (2–336 days) introduces outcome variability; future studies should standardize assessment time points.
- Individualized herbal medication: Variation in oral herbal prescriptions is an uncontrolled variable.
- No long-term follow-up: Post-intervention glycemic and TG/HDL sustainability cannot be assessed from this dataset.
- 8 patients (12.7%) showed worsening TG/HDL ratio — predominantly in the CDC-SP group (7/8) with high BMI — suggesting the most adipose patients may require longer or repeated treatment cycles.
- HDL non-response warrants investigation into whether prolonged duration or specific dietary modifications can yield HDL improvement.
- Single-center data; multicenter replication required for generalizability.

5. CONCLUSION

This retrospective analysis of 67 T2DM patients treated with the CDC Ayurvedic multimodal protocol demonstrates clinically meaningful and statistically significant improvements across glycemic, anthropometric, hemodynamic, and lipid parameters within a mean treatment period of approximately three months. The addition of a granular TG/HDL ratio analysis reveals that the intervention simultaneously reverses the insulin-resistant lipid phenotype: 71.4% of patients achieved TG/HDL <3.5 post-treatment, and 52.4% crossed into a lower cardiovascular risk category — findings that extend the clinical significance of this protocol well beyond glycemic control alone.

The 50% complete antidiabetic drug cessation rate and 83.3% overall drug reduction rate — alongside near-normalization of HbA1c and a positive correlation between baseline TG/HDL and HbA1c — support the Ayurvedic understanding of *Prameha* as a unified metabolic disorder best addressed through multi-target, pathway-level intervention. The integration of

BMI-stratified Panchakarma with Basti formulations of *Gudmar*, *Daru Haridra*, and *Yashti Madhu*, very-low-calorie dietary therapy, and individualized herbal medication represents a structurally coherent and mechanistically grounded protocol.

Prospective randomized controlled trials with standardized follow-up intervals, clearly defined remission and TG/HDL normalization criteria, and long-term (12–24 month) outcome tracking are warranted to validate the CDC protocol as a reproducible, evidence-based integrative approach to T2DM management.

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