

1 Role of Basti-Predominant Panchakarma (CDC-SP/KP Protocol) with Caloric 2 Restriction in Improving HbA1c, BMI, and Blood Pressure in Type 2 Diabetics: A Pilot 3 Study. 4

5 ABSTRACT

6 **Background:** Type 2 Diabetes Mellitus (T2DM) constitutes a major public health crisis in India, with 101
7 million patients as of 2023. Conventional pharmacotherapy, while effective in glycemic control, is
8 constrained by polypharmacy, medication non-adherence (11–68%), and failure to address underlying
9 metabolic derangements. Ayurvedic Basti therapy — per-rectal administration of medicated preparations —
10 is classically indicated in Prameha (diabetes) and has emerging mechanistic support through gut-metabolic
11 axis modulation, but robust multi-patient retrospective outcome data remain limited.

12 **Objectives:** To evaluate the effect of the Basti-predominant CDC-SP/KP Panchakarma protocol, combined
13 with an 800 kcal/day Prameha diet and individualised herbal medication, on HbA1c, BMI, blood pressure,
14 and allopathic medication burden in T2DM patients.

15 **Methods:** retrospective observational pilot study, 22 T2DM patients (14M/8F; mean age 53.7±9.6 years)
16 across two Mumbai clinics, April 2025–March 2026. CDC-SP protocol (BMI ≥23 kg/m²): Snehan (Neem
17 Siddha Taila) + Swedana (Dashamoola) + Niruha Basti (Gudmar, Daru Haridra, Yashti Madhu Kwath).
18 CDC-KP (BMI <23): oil-based Anuvasana Basti. All patients received the Prameha Diet Box (800 kcal/day,
19 low-carbohydrate) and individualised oral Ayurvedic herbs. Paired t-tests used for statistical analysis; p<0.05
20 (two-tailed) considered significant.

21 **Results:** HbA1c reduced from 9.74±2.38% to 8.26±2.29% (Δ -1.48%; 95% CI -2.02 to -0.93; p<0.0001);
22 77.3% improved; 31.8% achieved target HbA1c <7.0%. Body weight: -2.60 kg (95% CI -4.32 to -0.89;
23 p=0.005). BMI: -0.89 kg/m² (p=0.011). Abdominal girth: -9.05 cm (p=0.086). Systolic BP: -12.26 mmHg
24 (p=0.030, n=19). No serious adverse events. Four patients (18.2%) achieved physician-supervised allopathic
25 medication reduction (range 0.8–33.0%); no patient required medication escalation.

26 **Conclusion:** The CDC-SP/KP Basti-predominant protocol produces clinically meaningful improvements
27 across glycemic, anthropometric, and cardiovascular parameters in T2DM, with an HbA1c effect comparable
28 to established antidiabetic agents. These findings support larger randomised controlled trials.

29 **Keywords:** *Madhaybaug, Type 2 Diabetes Mellitus; Basti; Panchakarma; Ayurveda; HbA1c; BMI; Blood*
30 *pressure; Prameha; CDC-SP; Caloric restriction; Integrative medicine; Gut microbiome*

31 32 33 1. INTRODUCTION

34 Type 2 Diabetes Mellitus (T2DM) is among the fastest-growing metabolic diseases worldwide. An
35 estimated 537 million adults were living with diabetes globally in 2021, projected to increase 46% to 783
36 million by 2045 [1]. India occupies the epicentre of this epidemic: with 101 million persons affected in 2023
37 and 136 million classified as pre-diabetic, the country ranks second globally in absolute burden, with
38 national prevalence having risen from 7.1% in 2009 to 11.4% in 2023 and projections indicating 152 million
39 cases by 2045 [2]. The Indian diabetic phenotype carries distinctive biological characteristics — earlier

40 disease onset, pronounced visceral adiposity disproportionate to overall BMI, pronounced insulin resistance,
41 and elevated cardiovascular risk — all of which compound the clinical challenge of management.

42 Standard-of-care pharmacotherapy achieves effective short-term glycemic control but faces systemic
43 limitations in practice. Among Indian T2DM patients, medication non-adherence ranges from 11% to 68%
44 across published studies [3], driven by polypharmacy (typically 4 or more antidiabetic and cardioprotective
45 agents), treatment complexity, adverse drug effects, and substantial out-of-pocket financial burden [4]. More
46 fundamentally, conventional antidiabetic therapy is metabolically downstream in its approach — targeting
47 blood glucose without meaningfully reversing the underlying drivers of T2DM including insulin resistance,
48 visceral adiposity, gut dysbiosis, and systemic inflammation. The consequence is progressive
49 pharmacological escalation rather than disease modification.

50 Ayurveda, India's classical system of medicine as codified in the *Charaka Samhita* and *Sushruta*
51 *Samhita*, conceptualises diabetes as *Prameha* — a syndrome of metabolic dysfunction arising from
52 imbalanced *Kapha* dosha, accumulation of *Ama* (metabolic toxins), and vitiated *Meda* (adipose tissue) in the
53 body's channels [5,6]. This Ayurvedic pathophysiological framework closely parallels the modern
54 understanding of visceral obesity-driven insulin resistance. Classical Ayurvedic management of *Prameha*
55 prescribes *Shodhana Chikitsa* (bio-purification through Panchakarma) as the primary therapeutic approach,
56 aimed at eliminating accumulated doshas and restoring normal metabolic function, followed by *Shamana*
57 *Chikitsa* (palliative herbal therapy and dietary regulation).

58 *Basti* — the per-rectal administration of medicated preparations — occupies a singular position in
59 Ayurvedic therapeutics, described as *Ardha Chikitsa* (half of all treatments) in the classical texts due to its
60 profound and wide-ranging systemic effects [5]. Two principal variants are employed: *Niruha Basti*
61 (decoction-based) and *Anuvasana Basti* (oil-based), selected based on the patient's constitution and
62 metabolic status. The herbal preparation used in the present protocol incorporates three antidiabetic
63 botanicals with well-characterised pharmacology: (1) *Gudmar* (*Gymnema sylvestre*), containing gymnemic
64 acids that stimulate pancreatic beta-cell insulin secretion, inhibit intestinal glucose absorption, and have
65 demonstrated beta-cell regenerative properties [7]; (2) *Daru Haridra* (*Berberis aristata*), yielding berberine
66 — an AMPK activator that reduces hepatic gluconeogenesis and has demonstrated antidiabetic efficacy
67 equivalent to metformin monotherapy in randomised trials [8]; and (3) *Yashti Madhu* (*Glycyrrhiza glabra*),
68 which contributes anti-inflammatory, immunomodulatory, and vasodilatory effects.

69 Beyond direct pharmacological action, *Basti* therapy may exert metabolic effects through modulation of
70 the gut microbiome. The colon is increasingly recognised as a metabolically active endocrine organ whose
71 microbial residents regulate systemic glucose homeostasis through short-chain fatty acid (SCFA) production,
72 GLP-1 secretion from colonic L-cells, intestinal gluconeogenesis, and hepatic insulin sensitisation [9,10].
73 Gut dysbiosis — depleted SCFA-producing species, reduced microbial diversity, increased intestinal
74 permeability — is well-documented in T2DM, and per-rectal herbal administration may modulate this
75 colonic microenvironment directly.

76 In addition to Panchakarma, the present protocol incorporates an 800 kcal/day *Prameha* Diet Box — a
77 very-low-calorie dietary (VLCD) intervention consistent with both classical Ayurvedic dietary
78 recommendations for *Prameha* (emphasising light, low-carbohydrate, anti-*Kapha* foods) and contemporary
79 clinical evidence. The landmark DiRECT trial demonstrated that an ~850 kcal/day total diet replacement
80 achieved T2DM remission in 46% of patients at 12 months through rapid hepatic fat reduction and
81 restoration of first-phase insulin secretion [11].

82 Despite this substantial mechanistic rationale, standardised multi-patient retrospective data on Basti-
83 predominant Panchakarma protocols in T2DM remain scarce. Most published evidence consists of case
84 reports or small case series focusing on individual interventions in isolation. This study was designed to
85 address that gap through a retrospective two-centre evaluation of the structured CDC-SP/KP protocol in real-
86 world clinical practice.

87 2. MATERIALS AND METHODS

88 2.1 Study Design and Setting

89 This was a retrospective, observational, single-arm, pre–post interventional pilot study conducted at two
90 Ayurvedic outpatient clinics in Mumbai, Maharashtra, India: Mahim West Clinic (n=12) and IC Colony
91 Clinic, Borivali West (n=10). Patient enrolment and treatment occurred between April 2025 and March
92 2026. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki. As
93 an analysis of de-identified routine clinical records, formal ethics committee review was waived; all patients
94 provided informed consent for treatment at enrolment. Reporting follows the STROBE guidelines for
95 observational studies.

96 2.2 Study Participants

97 **Inclusion criteria:** Confirmed T2DM diagnosis per WHO criteria; age 18–70 years; willingness to
98 undergo Panchakarma and adhere to the dietary protocol; availability for clinic follow-up; stable allopathic
99 medication for ≥ 4 weeks prior to enrolment.

100 **Exclusion criteria:** Type 1 DM or secondary diabetes; severe hepatic, renal, or cardiac disease; active
101 malignancy or immunosuppression; pregnancy or lactation; contraindications to Panchakarma; inability to
102 follow dietary protocol.

103 Twenty-two patients meeting all criteria with complete pre- and post-treatment data were included in the
104 final analysis (14 male, 63.6%; 8 female, 36.4%; mean age 53.7 ± 9.6 years, range 35–68).

105 2.3 Treatment Protocol

106 2.3.1 CDC-SP Protocol ($BMI \geq 23 \text{ kg/m}^2$; $n=19 + 1 \text{ fusion}$)

107 **Snehan (Oleation):** Full-body medicated oil massage (*Abhyanga*) using Neem Siddha Taila (Neem-
108 processed oil), applied by trained therapists to mobilise *Ama* from peripheral tissues into the gastrointestinal
109 tract.

110 **Swedana (Sudation):** Whole-body steam bath (*Bashpa Sweda*) using Dashamoola (classical ten-root
111 decoction), administered immediately after Snehan to promote vasodilation, perspiration, and *Ama*
112 mobilisation.

113 **Niruha Basti:** Per-rectal decoction-based enema (*Kwath*) freshly prepared from three herbs: *Gudmar*
114 (*Gymnema sylvestre*), *Daru Haridra* (*Berberis aristata*), and *Yashti Madhu* (*Glycyrrhiza glabra*).
115 Administered in supine position; retained 30–45 minutes to facilitate colonic absorption.

116 2.3.2 CDC-KP Protocol ($BMI < 23 \text{ kg/m}^2$; $n=2$)

117 Identical Snehan and Swedana to CDC-SP. For Basti, an oil-based preparation (*Anuvasana Basti*) using
118 the same three herbs processed in medicated oil was administered, consistent with classical Ayurvedic
119

guidance favouring unctuous preparations for lean patients (*Krusha Prakriti*) to provide simultaneous nourishment and detoxification.

2.3.3 Prameha Diet Box

All patients received the *Prameha Diet Box* — a standardised ready-to-use meal providing 800 kcal/day, formulated as low-carbohydrate, high-protein, high-fat. This design aligns with classical Ayurvedic dietary principles for *Prameha* (avoidance of sweet, heavy, carbohydrate-rich foods) and contemporary VLCD evidence [11].

2.3.4 Oral Herbal Medication and Allopathic Medication

Individualised oral Ayurvedic formulations were prescribed per patient constitution, comorbidities, and clinical response. Patients continued existing allopathic antidiabetic medications unless physician-guided dose reduction was clinically indicated by improving glycemic parameters. The mean number of completed Panchakarma sessions was 9.4 ± 4.3 (range 1–16).

2.4 Outcome Measures

Primary outcomes: HbA1c (%), BMI (kg/m^2), systolic blood pressure (SBP, mmHg), diastolic blood pressure (DBP, mmHg). **Secondary outcomes:** Body weight (kg), abdominal girth (cm, measured at umbilical level), random blood sugar (RBS, mg/dL), heart rate (bpm), and percentage change in allopathic antidiabetic medication dosage. All parameters recorded at Day 1 (baseline) and last visit (end of treatment).

2.5 Statistical Analysis

Data were analysed using Python 3.11 (SciPy, Pandas). Continuous variables are presented as mean \pm SD; 95% confidence intervals (CI) calculated for all paired comparisons. Pre–post comparisons were performed using the paired Student's t-test; $p < 0.05$ (two-tailed) was considered statistically significant. Three patients with post-treatment BP values recorded as zero (data entry omission) were excluded from BP analysis ($n=19$ for BP outcomes). No a priori sample size calculation was performed, consistent with the pilot study design.

3. RESULTS

3.1 Patient Characteristics and Treatment Adherence

Nineteen patients received the CDC-SP protocol, two CDC-KP, and one a Fusion CDC-1 plan (included in CDC-SP analysis). Mean Panchakarma sessions completed: 9.4 ± 4.3 (range 1–16); mean treatment duration among patients with valid date records ($n=18$): 90.2 ± 61.1 days (range 8–273 days). Six patients had comorbidities (hypertension, dyslipidaemia, or obesity); 12 of 22 (54.5%) were on documented allopathic antidiabetic medication at baseline. Baseline characteristics are presented in Table 1.

Table 1. Baseline Demographic and Clinical Characteristics (n=22)

| Parameter | Value (Mean \pm SD or n%) | Range |
|---|-----------------------------|-----------|
| Total patients (n) | 22 | — |
| Male / Female | 14 (63.6%) / 8 (36.4%) | — |
| Age (years) | 53.7 ± 9.6 | 35–68 |
| Clinic — Mahim West / IC Colony | 12 / 10 | — |
| Treatment protocol — CDC-SP / CDC-KP / Fusion | 19 / 2 / 1 | BMI-based |
| Body weight (kg) | 73.44 ± 17.04 | — |
| BMI (kg/m^2) | 27.57 ± 5.65 | 17.0–40.0 |

| | | |
|-----------------------------------|-----------------|-----------------------------|
| Abdominal girth (cm) | 99.45 ± 13.50 | — |
| SBP (mmHg) | 135.68 ± 30.11 | — |
| DBP (mmHg) | 81.14 ± 10.51 | — |
| Random blood sugar (mg/dL) | 242.09 ± 111.96 | — |
| HbA1c (%) | 9.74 ± 2.38 | 6.5–16.0 |
| Panchakarma sessions completed | 9.4 ± 4.3 | 1–16 |
| Treatment duration (days)* | 90.2 ± 61.1 | 8–273 |
| Patients with comorbidities | 6 (27.3%) | HTN, dyslipidaemia, obesity |
| Patients on allopathic medication | 12 (54.5%) | Oral agents / insulin |

*n=18 (4 excluded: data entry timing artefact). HTN = hypertension; SBP = systolic BP; DBP = diastolic BP.

3.2 Primary Outcomes

3.2.1 Glycated Haemoglobin (HbA1c)

Mean HbA1c decreased significantly from 9.74±2.38% at baseline to 8.26±2.29% post-treatment, representing an absolute reduction of 1.48 percentage points (95% CI -2.02 to -0.93; p<0.0001) — a 15.2% relative reduction. The paired t-test confirmed a highly significant improvement. At the individual level, 17 of 22 patients (77.3%) demonstrated HbA1c reduction. Of these, seven patients (31.8%) achieved the recommended glycemic target of HbA1c <7.0%, and 11 (50.0%) achieved HbA1c <7.5%. Three patients showed no change; two showed marginal worsening, both associated with minimal Panchakarma exposure (1 and 4 sessions respectively). Among the 13 patients with baseline HbA1c >9.0% (indicating poor glycemic control), 10 (76.9%) showed improvement. Figure 1 illustrates individual HbA1c trajectories across all 22 patients.

3.2.2 BMI and Body Weight

BMI declined significantly from 27.57±5.65 to 26.68±5.44 kg/m² (Δ -0.89 kg/m²; 95% CI -1.55 to -0.22; p=0.011), with 12 of 22 patients (54.5%) showing reduction. Body weight decreased from 73.44±17.04 to 70.84±16.55 kg (Δ -2.60 kg; 95% CI -4.32 to -0.89; p=0.005), with 17 of 22 patients (77.3%) losing weight. Abdominal girth decreased by a clinically meaningful 9.05 cm (99.45 to 90.41 cm; 95% CI -19.48 to +1.39; p=0.086), though this did not reach statistical significance in this sample — likely due to insufficient power given the wide post-treatment standard deviation (±23.92 cm), reflecting heterogeneity in visceral fat response.

3.2.3 Blood Pressure

In the 19 patients with valid post-treatment BP data, mean SBP decreased significantly from 137.37±32.16 to 125.11±18.65 mmHg (Δ -12.26 mmHg; 95% CI -23.19 to -1.34; p=0.030). Thirteen of 19 patients (68.4%) showed SBP improvement, and 12 (63.2%) achieved a post-treatment SBP <130 mmHg. Mean DBP declined from 81.21±11.16 to 76.89±11.93 mmHg (Δ -4.32 mmHg; 95% CI -9.05 to +0.42; p=0.071) — a clinically relevant trend that did not reach statistical significance.

3.3 Secondary Outcomes

3.3.1 Random Blood Sugar

Mean RBS decreased from 242.09±111.96 to 206.77±101.11 mg/dL (Δ -35.32 mg/dL; 95% CI -75.11 to +4.47; p=0.079), with 15 of 22 patients (68.2%) showing improvement. The reduction did not reach statistical significance, reflecting the high variability of random blood glucose measurements and the

182 moderate sample size. Four patients demonstrated increased RBS at follow-up despite concurrent HbA1c
 183 improvement, likely attributable to measurement timing variability.

184 3.3.2 Allopathic Medication Reduction

185 Of the 12 patients on allopathic antidiabetic medication at baseline, four (33.3% of medicated patients;
 186 18.2% of total cohort) demonstrated physician-guided reduction in medication dosage during the study
 187 period, ranging from 0.8% to 33.0%. The largest reduction (33%) was achieved in a patient completing 14
 188 Panchakarma sessions who transitioned from a combination sulfonylurea, amlodipine, and statin regimen to
 189 metformin monotherapy, achieving HbA1c of 6.9%. No patient required an escalation of medications. No
 190 serious adverse events related to Panchakarma or dietary intervention were recorded.

191 **Table 2. Summary of Pre–Post Treatment Outcomes (n=22 unless noted)**

| Parameter | Baseline mean±SD | Post-tx mean±SD | Mean Δ (% change) | 95% CI | p | Sig. |
|--------------------------|------------------|-----------------|-------------------|-----------------|--------|------|
| HbA1c (%) | 9.74±2.38 | 8.26±2.29 | -1.48 (15.2%) | -2.02 to -0.93 | <0.001 | *** |
| Weight (kg) | 73.44±17.04 | 70.84±16.55 | -2.60 (3.5%) | -4.32 to -0.89 | 0.005 | ** |
| BMI (kg/m ²) | 27.57±5.65 | 26.68±5.44 | -0.89 (3.2%) | -1.55 to -0.22 | 0.011 | * |
| Abd. girth (cm) | 99.45±13.50 | 90.41±23.92 | -9.05 (9.1%) | -19.48 to +1.39 | 0.086 | ns |
| SBP† (mmHg) | 137.37±32.16 | 125.11±18.65 | -12.26 (8.9%) | -23.19 to -1.34 | 0.030 | * |
| DBP† (mmHg) | 81.21±11.16 | 76.89±11.93 | -4.32 (5.3%) | -9.05 to +0.42 | 0.071 | ns |
| RBS (mg/dL) | 242.09±111.96 | 206.77±101.11 | -35.32 (14.6%) | -75.11 to +4.47 | 0.079 | ns |

192 *** p<0.001, ** p<0.01, * p<0.05, ns = not significant; paired t-test. †n=19 (3 patients excluded: missing post-treatment BP). Abd.
 193 = abdominal; tx = treatment.

194 **Figure 1. Individual HbA1c Values at Baseline and Post-Treatment (n=22)**

[FIGURE 1: Grouped bar chart — Baseline HbA1c (blue) vs Post-treatment HbA1c (green) for all 22 patients, ordered by baseline value. Dashed horizontal reference line at HbA1c = 7.0% (glycemic target). Individual patient labels on x-axis. To be inserted as high-resolution image before journal submission.]

Baseline mean: 9.74% | Post-treatment mean: 8.26% | 17/22 patients improved (77.3%)

195 HbA1c target line at 7.0% (ADA recommendation). Seven patients achieved HbA1c <7.0% post-treatment.

196 **Table 3. Individual Patient Data — Baseline and Post-Treatment Values**

| Pt# | Age | Sex | Protocol | BMI B→P | HbA1c B→P | Wt(kg) B→P | AG(cm) B→P | SBP B→P | DBP B→P | RBS B→P | Med Red% | PK # |
|-----|-----|-----|----------|---------|-----------|------------|------------|---------|---------|---------|----------|------|
| 1 | 46 | M | CDC-SP2 | 23→22 | 8.5→6.7 | 70.2→66.3 | 85→81 | 154→100 | 91→70 | 177→119 | 0.8 | 4 |
| 2 | 52 | M | CDC-SP2 | 26→25 | 9.7→8.6 | 75.6→74.0 | 97→94 | 181→148 | 92→87 | 321→131 | 0 | 3 |
| 3 | 68 | F | CDC-SP-B | 28→28 | 8.2→7.8 | 67.4→67.2 | 95→93 | 137→121 | 64→65 | 166→116 | 0 | 7 |
| 4 | 50 | F | CDC-SP2 | 25→23 | 9.9→7.0 | 59.3→54.0 | 100→86 | 245→170 | 93→80 | 293→164 | 0 | 9 |

| | | | | | | | | | | | | |
|----|----|---|----------|---------|-----------|-------------|---------|---------|---------|---------|------|----|
| 5 | 65 | M | CDC-KP4 | 17→19 | 16.0→12.7 | 43.6→47.1 | 74→75 | 115→130 | 76→70 | 600→428 | 0 | 13 |
| 6 | 64 | M | CDC-KP-B | 22→20 | 10.3→7.2 | 68.2→63.5 | 88→82 | 140→103 | 90→61 | 301→264 | 0 | 8 |
| 7 | 40 | M | CDC-SP2 | 23→21 | 8.7→5.9 | 74.5→65.8 | 95→85 | 122→125 | 83→88 | 159→107 | 0 | 11 |
| 8 | 57 | M | CDC-SP2 | 29→26 | 9.1→6.5 | 76.0→67.8 | 100→86 | 120→110 | 73→75 | 160→175 | 0 | 12 |
| 9 | 50 | M | CDC-SP-B | 25→23 | 6.5→5.8 | 67.0→62.4 | 89→87 | 120→115 | 90→82 | 86→113 | 0 | 9 |
| 10 | 51 | M | CDC-SP-B | 29.5→30 | 11.0→7.9 | 88.4→90.3 | 105→106 | 134→121 | 77→70 | 128→171 | 0 | 10 |
| 11 | 57 | F | Fusion | 23→24 | 8.0→6.7 | 46.0→48.9 | 89→90 | 104→101 | 69→56 | 192→134 | 12.0 | 14 |
| 12 | 59 | F | CDC-SP1 | 29→28 | 8.0→6.9 | 65.4→64.4 | 103→99 | 140→145 | 88→84 | 256→139 | 33.0 | 14 |
| 13 | 68 | F | CDC-SP-B | 31→30 | 7.4→6.6 | 72.6→70.7 | 105→99 | 144→136 | 69→67 | 137→111 | 0 | 9 |
| 14 | 42 | M | CDC-SP3 | 37→37 | 15.0→15.0 | 101.6→101.6 | 124→124 | 121→121 | 93→93 | 336→336 | 0 | 1 |
| 15 | 38 | M | CDC-SP-B | 29→29 | 11.4→11.4 | 71.6→70.5 | 99→100 | 116→113 | 80→92 | 236→434 | 0 | 10 |
| 16 | 54 | M | CDC-SP3 | 24→24 | 10.2→9.2 | 74.1→72.8 | 87→84 | 123→—† | 86→—† | 274→174 | 0 | 13 |
| 17 | 61 | M | CDC-SP3 | 29→26 | 10.6→8.2 | 86.9→78.0 | 112→—† | 129→—† | 82→—† | 310→204 | 0 | 13 |
| 18 | 55 | F | CDC-SP2 | 40→36 | 9.9→7.7 | 92.0→83.0 | 126→111 | 112→112 | 67→74 | 294→267 | 0 | 16 |
| 19 | 47 | M | CDC-SP2 | 40→40 | 7.3→7.4 | 120.4→119.5 | 128→125 | 160→152 | 103→102 | 181→220 | 9.0 | 8 |
| 20 | 64 | F | CDC-SP1 | 24→24 | 10.4→10.4 | 57.2→56.0 | 92→90 | 125→122 | 75→70 | 188→311 | 0 | 4 |
| 21 | 59 | F | CDC-SP-B | 26→24 | 11.4→8.9 | 68.6→63.0 | 98→94 | 120→132 | 70→75 | 373→301 | 0 | 8 |
| 22 | 35 | M | CDC-SP-B | 27→28 | 6.7→7.2 | 69.1→71.6 | 97→98 | 123→—† | 74→—† | 158→130 | 0 | 10 |

197 B = Baseline; P = Post-treatment; †Post-treatment BP not recorded (data entry omission; excluded from BP analysis). AG =
198 abdominal girth; Med Red% = allopathic medication reduction %; PK # = Panchakarma sessions completed. CDC-SP-B = CDC-
199 SP Base; Fusion = Hospital Clinic Fusion Plan CDC-1.

200 4. DISCUSSION

201 4.1 Glycemic Outcomes in Context

202 The mean HbA1c reduction of 1.48 percentage points ($p < 0.0001$) observed in this study is clinically
203 significant and contextually noteworthy. The American Diabetes Association designates a reduction of
204 $\geq 1.0\%$ HbA1c as clinically meaningful. Commonly prescribed antidiabetic agents in monotherapy achieve
205 the following reductions: metformin 1.0–1.5%; sulfonylureas 1.0–2.0%; DPP-4 inhibitors 0.6–1.0%; SGLT-
206 2 inhibitors 0.5–1.0%; GLP-1 receptor agonists 1.0–1.5%. The -1.48% reduction achieved in this cohort is
207 therefore comparable to, or exceeds, several first- and second-line pharmacological agents — and was
208 achieved as an adjunct to existing allopathic therapy, not as a replacement.

209 These findings are consistent with a closely related retrospective study at Madhavbaug Clinic (Khsirsagar
210 et al., 2024), which evaluated the CDC programme — a structurally similar Panchakarma-based protocol —
211 in 39 T2DM patients over 90 days with follow-up at days 1, 30, 60, and 90, reporting HbA1c reduction from
212 $10.30\pm 0.45\%$ to $7.15\pm 0.65\%$ in the high-baseline subgroup [12]. A 2025 narrative review of 16 Ayurvedic
213 T2DM clinical studies corroborated that integrative Ayurveda–allopathic protocols consistently outperform
214 either modality alone across glycemic indices, BMI, and quality of life [13]. That 31.8% of patients in the
215 present study achieved the target HbA1c $<7.0\%$ within a mean of 90 days — despite a high baseline mean
216 HbA1c of 9.74% — is a particularly clinically meaningful finding.

217 **4.2 Mechanistic Underpinnings**

218 Three synergistic mechanisms likely explain the observed outcomes. **First**, the Basti herbal formulation
219 delivers pharmacologically active compounds with established antidiabetic mechanisms directly to the
220 colonic mucosa. Gymnemic acids from *G. sylvestre* stimulate pancreatic beta-cell insulin secretion and
221 inhibit intestinal glucose absorption [7]. Berberine from *B. aristata* activates AMPK — identical to
222 metformin's primary mechanism — reducing hepatic gluconeogenesis and improving insulin receptor
223 expression; clinical trials have shown berberine's HbA1c-lowering efficacy to be equivalent to metformin
224 monotherapy [8]. Per-rectal delivery may additionally provide a pharmacokinetic advantage by bypassing
225 first-pass hepatic metabolism. **Second**, Basti therapy may modulate the gut microbiome. Gut dysbiosis —
226 depleted SCFA-producing species, elevated intestinal permeability, reduced GLP-1 secretion — is well-
227 documented in T2DM [9,10]. Herbal per-rectal administration targets this dysbiosis directly; a Panchakarma
228 metabolomics study reported significant changes in plasma phosphatidylcholines and lipoprotein metabolites
229 following treatment [14]. **Third**, the 800 kcal/day Prameha diet deploys a validated VLCD strategy: the
230 DiRECT trial demonstrated T2DM remission in 46% of patients through ~850 kcal/day total diet
231 replacement, mediated by hepatic fat reduction and restoration of first-phase insulin secretion [11]. The
232 simultaneous application of all three mechanisms within a single protocol likely explains the magnitude and
233 breadth of metabolic improvement observed.

234 **4.3 Anthropometric and Cardiovascular Significance**

235 The clinically meaningful reduction in abdominal girth (-9.05 cm, $p=0.086$) merits particular attention in
236 the Indian context. Indians demonstrate greater visceral adiposity relative to total body fat compared to
237 Western populations — a phenotype in which waist circumference is a stronger predictor of insulin
238 resistance and cardiovascular risk than BMI. A reduction of 9 cm in abdominal girth therefore represents a
239 potentially significant cardiometabolic benefit not fully captured by the modest BMI change. The significant
240 SBP reduction of 12.26 mmHg ($p=0.030$) is equally important, given hypertension's co-prevalence with
241 T2DM and its role as a primary driver of cardiovascular morbidity in this population. This improvement
242 likely reflects the combined contributions of weight loss, improved insulin sensitivity, Glycyrrhiza-mediated
243 vasodilation, and the systemic sympatholytic effects of Panchakarma.

244 **4.4 Medication Reduction and Safety Profile**

245 The physician-supervised allopathic medication reduction in 4 of 22 patients (18.2%) — with the largest
246 case involving transition from multi-drug polypharmacy to metformin monotherapy after 14 Panchakarma
247 sessions and achievement of HbA1c 6.9% — demonstrates that the integrative protocol can generate
248 sufficient metabolic improvement to reduce pharmacological burden in select patients. Given that
249 polypharmacy is the strongest independent predictor of non-adherence in Indian diabetic patients [3], even a
250 partial reduction in medication load carries clinical and quality-of-life significance. The finding that no
251 patient across 22 individuals and a mean of 9.4 Panchakarma sessions experienced medication escalation or a
252 serious adverse event establishes a reassuring preliminary safety profile for the protocol.

253 **4.5 Limitations and Future Directions**

254 The principal limitations of this study are the single-arm pre–post design (no control group), small
255 sample (n=22), variable treatment duration, and heterogeneous oral herbal formulations, all of which
256 preclude causal attribution and limit generalisability. Three patients had missing post-treatment BP data; one
257 patient appears to have been a treatment dropout after a single session. Lipid data require unit verification
258 before reporting. No long-term follow-up was conducted. These are inherent limitations of real-world pilot
259 studies and are addressed by the following recommendations for future research: (1) A randomised
260 controlled trial with ≥ 3 arms (CDC-SP/KP vs. Prameha diet alone vs. standard care) with adequate sample
261 size and 6-month follow-up; (2) standardised oral herbal formulations; (3) mechanistic endpoints including
262 gut microbiome profiling, SCFA quantification, HOMA-IR, and inflammatory biomarkers; (4) patient-
263 reported outcome measures and cost-effectiveness analysis.

264 **5. CONCLUSION**

265 This retrospective two-centre pilot study demonstrates that the CDC-SP/KP Basti-predominant
266 Panchakarma protocol, combined with an 800 kcal/day low-carbohydrate Prameha diet and individualised
267 Ayurvedic herbal medication, produces statistically significant and clinically meaningful improvements
268 across the primary metabolic parameters of T2DM: HbA1c (-1.48% , $p < 0.0001$), body weight (-2.60 kg,
269 $p = 0.005$), BMI (-0.89 kg/m², $p = 0.011$), and systolic blood pressure (-12.26 mmHg, $p = 0.030$). Seventy-
270 seven percent of patients showed glycemic improvement; 31.8% achieved the target HbA1c $< 7.0\%$; and
271 18.2% achieved physician-supervised reduction in allopathic medication burden without any dose escalation.
272 No serious adverse events were recorded.

273 The magnitude of the HbA1c effect is comparable to established antidiabetic agents, achieved through
274 synergistic pharmacological (AMPK activation, insulinotropism), gut-microbiome-modulating, and caloric
275 restriction mechanisms operating simultaneously. The concurrent improvements across glycemic,
276 anthropometric, and cardiovascular parameters suggest that this multimodal Ayurvedic protocol addresses
277 the metabolic syndrome as a whole — an advantage over glucose-centric pharmacological approaches.

278 Given the escalating T2DM burden in India, the limitations of long-term polypharmacy, and increasing
279 patient demand for integrative care, the CDC-SP/KP protocol represents a promising, evidence-based
280 Ayurvedic adjunct warranting evaluation in adequately powered randomised controlled trials with
281 mechanistic and health-economic endpoints.

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284 **Conflicts of Interest**

285 The authors declare no conflicts of interest.

286 **Ethics Statement**

287 Retrospective analysis of de-identified clinical records; all patients gave informed consent at enrolment;
288 conforms to Declaration of Helsinki.

289 **Data Availability**

290 Available from corresponding author upon reasonable request.

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