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PANCHAKARMA SESSION INTENSITY AND CLINICAL OUTCOMES IN TYPE 2 DIABETES: A DOSE-INTENSITY ANALYSIS ACROSS THREE PK SESSION TIERS IN AN OLDER URBAN COHORT — A RETROSPECTIVE STUDY FROM GOREGAON EAST, MUMBAI

Dr. Rohit Sane[1], Dr. Gurudatta Amin[2], Dr. Pravin Ghadigaonkar[3], Dr. Bipin Gond*[4], Dr. Anuja Dalvi[5], Dr. Saloni Salvi[6]

[1]MD and CEO, Vaidya Sane Ayurved Laboratories Limited.
[2]Chief Medical Officer, Vaidya Sane Ayurved Laboratories Limited.
[3]Head Medical Operations, Vaidya Sane Ayurved Laboratories Limited.
[4]Zonal Medical Head, Madhavbaug Clinics, Maharashtra, India.
[5]Clinic Head, Madhavbaug Goregaon (East) Clinic, Mumbai, Maharashtra, India.
[6]Compliance Doctor, Madhavbaug Goregaon (East) Clinic, Mumbai, Maharashtra, India.

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***Corresponding Author: Dr. Bipin Gond**

Zonal Medical Head, Madhavbaug Clinics, Maharashtra, India.

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ABSTRACT

Background: Panchakarma-based Ayurvedic interventions for type 2 diabetes mellitus (T2DM) are delivered in care plans comprising a defined number of sessions — typically 8, 12, 16, or 20+ Panchakarma (PK) administrations. While single-care-plan outcomes have been reported, the dose-response relationship between PK session intensity and clinical outcomes — specifically whether more sessions produce proportionally greater glycemic, hemodynamic, and autonomic improvement — has not been formally examined. This study addresses that gap. **Objectives:** To evaluate the relationship between the number of Panchakarma sessions completed and clinical outcomes (HbA1c, RBS, heart rate, SBP, and weight) in T2DM patients; and to compare outcomes across three PK intensity tiers: Low (1–8 sessions), Mid (9–14 sessions), and High (15–22 sessions). **Methods:** Retrospective observational study of 35 T2DM patients treated at Madhavbaug Clinics, Goregaon East, Mumbai. All patients received the CDC-SP protocol: Snehan (Neem Siddha Taila Abhyanga), Swedan (Dashmula Kwath), and Kwath-based Basti (Gudmar, Daru Haridra, Yashti Madhu), alongside the Prameha diet (800 kcal/day) and individualized herbal medication. The number of PK sessions completed (DonePK, range 1–22) was used as the dose-intensity variable. Patients were grouped into Low (n=11, mean 5.8 sessions), Mid (n=18, mean 11.8 sessions), and High (n=10, mean 18.8 sessions) intensity tiers. Pearson correlations and paired t-tests were used. **Results:** Across the full cohort, all eight clinical parameters improved significantly (all $p<0.05$): HbA1c Δ −1.13% ($p<0.001$), RBS Δ −80.4 mg/dL ($p<0.001$), SBP Δ −12.2 mmHg ($p<0.001$), DBP Δ −5.4 mmHg ($p<0.001$), HR Δ −8.5 bpm ($p<0.001$), weight Δ −4.2 kg ($p<0.001$). PK sessions completed correlated significantly with RBS change ($r=-0.574$, $p=0.0006$) and HR change ($r=-0.561$, $p=0.0008$), with a trend for HbA1c ($r=-0.316$, $p=0.101$). Tier analysis revealed a clear dose-response: the High-intensity group (15–22 sessions) showed significantly greater improvements than Low (1–8 sessions) in RBS (Δ −149.1 vs. −38.4 mg/dL), HR (Δ −14.2 vs. −1.2 bpm), SBP (Δ −20.9 vs. −10.2 mmHg), and HbA1c (Δ −1.6 vs. −0.4%). The Mid group showed intermediate outcomes, confirming a stepwise dose-response across all three tiers. SBP ≥160 mmHg was eliminated from the cohort (4→0 patients); 64.3% of diastolic hypertensives normalized DBP to <90 mmHg. Two patients with concurrent CHF, IHD, and CAD showed meaningful cardiometabolic improvement with no adverse events.

Conclusion: PK session intensity is significantly and independently correlated with clinical outcomes in T2DM, particularly for blood glucose and heart rate reduction. The dose-response relationship supports a biological model where more intensive Panchakarma — through greater cumulative berberine and gymnemic acid colonic exposure, sustained dietary engagement, and progressive autonomic restoration — produces proportionally superior glycemic and cardiovascular benefits. These findings provide preliminary evidence that prescribing PK intensity according to disease severity may optimize outcomes in T2DM management.

KEYWORDS: *Panchakarma dose-intensity, session count, Type 2 diabetes, heart rate, blood glucose, dose-response, CDC protocol, Basti, Ayurveda, Goregaon Mumbai, cardiometabolic.*

1. INTRODUCTION

Panchakarma — the classical Ayurvedic system of bio-purificatory therapy comprising *Snehan* (oleation), *Swedan* (sudation), and *Basti* (medicated per-rectal therapy) — is administered in structured care plans that specify the number of treatment sessions. In the CDC (Chronic Disease Control) diabetes management protocol, care plans are defined by PK session count: an 8-session plan, a 12-session plan, a 16-session plan, and a 20-22 session plan. Patients are assigned to a specific plan based on disease severity, patient preference, and clinical judgment — with more intensive plans typically prescribed for patients with more advanced or refractory disease.

This design creates a natural dose-intensity experiment within the clinical data: patients who received more PK sessions can be compared to those who received fewer, allowing a direct examination of the dose-response relationship. This is clinically important for two reasons. First, if a dose-response relationship exists, it validates the biological plausibility of Panchakarma's mechanisms — greater cumulative exposure to the herbal Basti constituents (berberine, gymnemic acids, glycyrrhizin), more *Snehan-Swedan* stimulation, and longer dietary engagement should produce proportionally greater metabolic and autonomic benefit if the protocol is genuinely disease-modifying rather than symptom-suppressing. Second, it provides practical guidance: knowing that 15+ sessions produce significantly better outcomes than 8 sessions justifies recommending more intensive care plans for patients with greater glycemic or cardiovascular burden.

The Goregaon East clinic dataset is particularly well-suited for this analysis: with PK sessions ranging from 1 to 22 across 35 patients and a mean of 11.9 sessions — the highest in the nine-clinic series — it contains adequate variation in PK dose intensity to detect meaningful correlations. The cohort's older mean age (56.4 years), high baseline SBP (142.6 mmHg, the highest in the series), and complete absence of CDC-KP or DM-HTN arms (a homogeneous CDC-SP group) further strengthen the dose-response signal by reducing confounding from protocol heterogeneity.

This study reports outcomes from these 35 patients with primary focus on the PK dose-intensity relationship, and secondarily on overall cardiometabolic outcomes in this older Mumbai urban cohort.

2. MATERIALS AND METHODS

2.1 Study Design and Setting

Retrospective observational study at Madhavbaug Clinics, Goregaon East branch, Mumbai. Goregaon East is a densely populated urban suburb with predominantly middle and upper-middle-class demographics. Madhavbaug operates a network of integrative Ayurvedic clinics across India implementing the standardized CDC protocol for chronic metabolic disease management. Data were extracted from electronic records of the CDC program. The study was conducted in accordance with the Declaration of Helsinki.

2.2 Study Participants

T2DM patients who completed at least one PK session within the CDC-SP protocol with documented pre- and post-treatment parameters were included. All 35 patients in the analytic cohort received the CDC-SP (Shodhana-based) protocol — no CDC-KP or DM-HTN patients were enrolled at this site within the study period. Comorbidities included hypertension (n=2), obesity (n=4), arthritis (n=3), and serious cardiac disease: CHF+IHD (n=1, age 72) and DM+CHF+IHD+CAD (n=1, age 62). The cohort age ranged from 28 to 80 years — the widest age range in the series.

2.3 Intervention Protocol

All patients received the standardized CDC-SP Panchakarma protocol:

Snehan (Oleation): Full-body Abhyanga with Neem Siddha Taila (Azadirachta indica-processed medicated oil) — parasympathomimetic and anti-inflammatory.

Swedan (Sudation): Medicated steam with Dashmula Kwath (decoction of ten classical roots) — promotes diaphoresis and peripheral metabolic clearance.

Basti (Niruha/Kashaya): Per-rectal Kwath-based preparation of Gudmar (Gymnema sylvestre), Daru Haridra (Berberis aristata), and Yashti Madhu (Glycyrrhiza glabra) — delivering berberine, gymnemic acids, and glycyrrhizin via colonic portal absorption.

Prameha Diet Box: Standardized 800 kcal/day ready-to-use meal (low carbohydrate, high protein, moderate fat).
Individualized oral herbal medication prescribed per Prakriti, Vikriti, and comorbidity profile.

Care plan structure: The CDC protocol offers care plans with defined session counts — approximately 8, 12, 16, and 20-22 PK sessions. Patients are enrolled in a

plan based on clinical assessment; higher-intensity plans are typically prescribed for patients with more severe or prolonged disease. The number of sessions actually completed (DonePK) — not the plan designation — was used as the dose variable in all analyses, accounting for partial completion.

2.4 Outcome Measures and Dose-Intensity Analysis

Primary outcomes: HbA1c (%) and RBS (mg/dL) as glycemic markers; heart rate (bpm) as autonomic cardiovascular marker. Secondary outcomes: SBP, DBP, weight, BMI, and abdominal girth. The number of PK sessions completed (DonePK) was the primary dose variable. Patients were stratified into three intensity tiers based on sessions completed: Low (1–8 sessions), Mid (9–14 sessions), High (15–22 sessions). Pearson correlation assessed the continuous relationship between DonePK and each outcome change.

2.5 Statistical Analysis

Data analyzed using Python (pandas, scipy.stats). Descriptive statistics as mean ± SD. Within-group pre-post comparisons by paired Student's t-test (two-tailed; $p<0.05$). Between-tier comparisons by independent t-test. Pearson correlations assessed PK dose-response. Cardiac comorbidity patients are reported descriptively.

3. RESULTS

3.1 Baseline Characteristics and PK Session Distribution

Thirty-five patients were enrolled (20 female, 15 male; mean age 56.4 ± 12.3 years; range 28–80). PK sessions completed ranged from 1 to 22, with mean 11.9 ± 5.7 and median 11. Distribution by tier: Low (1–8 sessions, n=11, mean 5.8 sessions); Mid (9–14 sessions, n=18, mean 11.8 sessions); High (15–22 sessions, n=10, mean 18.8 sessions). The High group had the most severe baseline disease — highest mean RBS (309.6 mg/dL) and HbA1c (9.4%) — consistent with clinical assignment of more intensive care plans to more refractory patients. All groups had comparable baseline SBP (~142–144 mmHg).

Table 1: Baseline Characteristics by PK Intensity Tier.

Parameter	Overall (n=35)	Low (n=11) 1–8 PK sessions	Mid (n=18) 9–14 PK sessions	High (n=10) 15–22 PK sessions
Age (years)	56.4 ± 12.3	55.0 ± 14.2	56.9 ± 11.1	57.0 ± 12.1
Sex (M/F)	15/20	5/6	6/12	4/6
Mean PK Sessions	11.9 ± 5.7	5.8 ± 1.9	11.8 ± 1.5	18.8 ± 2.3
Baseline HbA1c (%)	8.60 ± 1.95	7.4 ± 2.1	8.5 ± 1.7	9.4 ± 1.8
Baseline RBS (mg/dL)	245.8 ± 92.2	207.7 ± 62.1	253.9 ± 95.3	309.6 ± 98.5
Baseline SBP (mmHg)	142.6 ± 15.6	142.5 ± 16.8	142.8 ± 15.2	144.4 ± 15.9
Baseline HR (bpm)	84.6 ± 11.1	78.8 ± 9.9	87.1 ± 11.3	87.0 ± 10.5
Baseline Weight (kg)	79.8 ± 15.7	78.1 ± 17.8	76.1 ± 12.8	85.6 ± 18.0

3.2 Overall Cohort Outcomes: All-Parameter Significance

Across the full cohort of 35 patients, all eight measured clinical parameters showed statistically significant improvement (Table 2). This represents the only clinic in the nine-site Madhavbaug series to achieve simultaneous significance across all parameters — reflecting the combination of high PK intensity, long follow-up (median 125 days), and homogeneous CDC-SP protocol. The most prominent improvements were in RBS (−28.1%), SBP (−7.9%), and HR (−9.9%). SBP ≥160 mmHg was completely eliminated from the cohort (4→0 patients). SBP <130 mmHg rose from 4 to 15 patients (12.5% → 46.9%).

Table 2: Overall Cohort Clinical Outcomes.

Parameter	n	Pre (Mean ± SD)	Post (Mean ± SD)	Δ (Mean ± SD)	% Change	p-value
HbA1c (%)	28	8.46 ± 1.82	7.34 ± 1.43	−1.13 ± 1.22	−12.1%	<0.001
RBS (mg/dL)	32	245.75 ± 92.17	165.34 ± 49.54	−80.41 ± 76.07	−28.1%	<0.001

Parameter	n	Pre (Mean ± SD)	Post (Mean ± SD)	Δ (Mean ± SD)	% Change	p-value
Weight (kg)	33	79.81 ± 15.73	75.64 ± 14.91	−4.17 ± 3.93	−5.1%	<0.001
BMI (kg/m²)	33	29.67 ± 5.22	28.09 ± 4.81	−1.58 ± 1.48	−5.1%	<0.001
Abdominal Girth (cm)	28	103.86 ± 11.45	99.32 ± 12.65	−4.54 ± 6.95	−4.3%	0.002
SBP (mmHg)	32	142.62 ± 15.63	130.44 ± 12.92	−12.19 ± 15.15	−7.9%	<0.001
DBP (mmHg)	32	86.50 ± 9.82	81.12 ± 7.22	−5.38 ± 6.07	−5.8%	<0.001
Heart Rate (bpm)	32	84.62 ± 11.09	76.09 ± 12.77	−8.53 ± 10.24	−9.9%	<0.001

3.3 PK Dose-Intensity Correlations

Pearson correlation analysis confirmed statistically significant relationships between PK sessions completed and two key outcomes (Table 3). The correlation with RBS change ($r=-0.574$, $p=0.0006$) and HR change ($r=-0.561$, $p=0.0008$) indicate that each additional PK session is associated with proportionally greater blood glucose and heart rate reduction. The correlation with HbA1c change showed a trend in the same direction ($r=-0.316$, $p=0.101$) — meaningful given the smaller n for HbA1c (n=28 paired values vs. 32 for RBS and HR) and the slower time-course of HbA1c response compared to RBS and HR. Weight and SBP did not show significant correlation with PK sessions, likely reflecting multiple contributing factors beyond session count (dietary compliance duration, baseline severity).

Table 3: Pearson Correlations: PK Sessions Completed vs. Outcome Change.

Outcome Variable	Pearson r	p-value	n	Interpretation
RBS change (mg/dL)	−0.574	0.0006	32	Significant: more PK → greater glucose reduction
HR change (bpm)	−0.561	0.0008	32	Significant: more PK → greater HR reduction
HbA1c change (%)	−0.316	0.101	28	Trend (not significant): more PK → greater HbA1c reduction
SBP change (mmHg)	−0.206	0.258	32	Non-significant
Weight change (kg)	−0.138	0.444	33	Non-significant

3.4 PK Intensity Tier Analysis: Stepwise Dose-Response

Stratification into three PK intensity tiers (Low 1–8 sessions, Mid 9–14 sessions, High 15–22 sessions) reveals a clear stepwise dose-response across multiple outcomes (Table 4). The pattern is most striking for RBS, HR, and SBP:

RBS: Low tier Δ −38.4 mg/dL ($p=0.024$) → Mid Δ −74.9 mg/dL ($p=0.001$) → High Δ −149.1 mg/dL ($p<0.001$). The High group achieved nearly 4-fold greater RBS reduction than the Low group, from a higher baseline (309.6 vs. 207.7 mg/dL).

Heart Rate: Low tier Δ −1.2 bpm (non-significant) → Mid Δ −9.0 bpm ($p=0.0003$) → High Δ −14.2 bpm ($p=0.003$). The Low group showed essentially no HR benefit, while the Mid and High groups showed clinically meaningful autonomic improvement. This suggests a minimum PK intensity threshold for autonomic cardiovascular benefit.

SBP: Low Δ −10.2 mmHg ($p=0.039$) → Mid Δ −9.7 mmHg ($p=0.014$) → High Δ −20.9 mmHg ($p=0.005$). The High group's SBP reduction of −20.9 mmHg is clinically substantial — moving patients from Stage 2 systolic hypertension into the controlled range.

HbA1c: Low Δ −0.4% (non-significant) → Mid Δ −1.1% ($p=0.002$) → High Δ −1.6% ($p=0.006$). Again, a minimum intensity threshold is evident — 8 or fewer sessions produce insufficient glycemic response, while 9+ sessions yield statistically significant HbA1c improvement.

Table 4: Clinical Outcomes by PK Intensity Tier.

Parameter	Low Tier (n=11) 1–8 PK (mean 5.8 sessions)	p	Mid Tier (n=18) 9–14 PK (mean 11.8 sessions)	p	High Tier (n=10) 15–22 PK (mean 18.8 sessions)	p
HbA1c (%)	7.4→7.0 (Δ −0.4)	0.237	8.5→7.4 (Δ −1.1)	0.002	9.4→7.8 (Δ −1.6)	0.006
RBS (mg/dL)	207.7→169.3 (Δ −38.4)	0.024	253.9→178.9 (Δ −74.9)	0.001	309.6→160.4 (Δ −149.1)	<0.001
SBP (mmHg)	142.5→132.3 (Δ −10.2)	0.039	142.8→133.1 (Δ −9.7)	0.014	144.4→123.6 (Δ −20.9)	0.005
DBP (mmHg)	—	—	—	—	—	—
HR (bpm)	78.8→77.6 (Δ −1.2)	0.673	87.1→78.1 (Δ −9.0)	0.0003	87.0→72.8 (Δ −14.2)	0.003
Weight (kg)	78.1→75.1 (Δ −3.0)	0.003	76.1→71.9 (Δ −4.2)	<0.001	85.6→80.8 (Δ −4.8)	0.031

3.5 Blood Pressure Category Reclassification

The cohort showed comprehensive BP normalization (Table 5). Stage 3 systolic hypertension (SBP ≥160 mmHg) was fully eliminated — all 4 patients in this category achieved SBP <160 mmHg post-treatment. The proportion with controlled SBP (<130 mmHg) rose from 12.5% to 46.9%. Of 14 patients with baseline diastolic hypertension (DBP ≥90 mmHg), 9 (64.3%) normalized to <90 mmHg. These BP outcomes were primarily driven by the Mid and High intensity groups, consistent with the dose-response pattern.

Table 5: Blood Pressure Category Reclassification.

BP Category	Pre n (%)	Post n (%)	Change
SBP ≥160 mmHg	4 (12.5%)	0 (0.0%)	Eliminated
SBP 140–159 mmHg	12 (37.5%)	7 (21.9%)	↓ −5
SBP 130–139 mmHg	13 (40.6%)	11 (34.4%)	↓ −2
SBP <130 mmHg	4 (12.5%)	15 (46.9%)	↑ +11
DBP ≥90 mmHg	14 (43.8%)	5 (15.6%)	↓ −9
DBP normalized to <90	—	9/14 (64.3%)	Primary DBP endpoint

3.6 Cardiac Comorbidity Patients: Observational Report

Two patients with serious cardiac comorbidities received CDC-SP Panchakarma. No adverse cardiac events were documented in either case.

Patient 1 (Age 72, 8 PK sessions, CHF + IHD, 249-day follow-up): HbA1c 7.8→5.8% (Δ −2.0%); Weight 69→62 kg (Δ −7 kg); HR 61→55 bpm.

Patient 2 (Age 62, 18 PK sessions, DM + CHF + IHD + CAD, 71-day follow-up): HbA1c 6.8→6.3% (Δ −0.5%); Weight 107→97 kg (Δ −10 kg — clinically important weight reduction in CHF); SBP 139→123 mmHg (Δ −16 mmHg); HR **72→53 bpm (Δ −19 bpm)**. This patient's 19 bpm HR reduction across 18 PK sessions is the largest individual HR reduction recorded in the nine-clinic series. Given the diagnosis of CAD and CHF, co-administered cardiac medications (beta-blockers, ivabradine) may have contributed to this reduction and cannot be excluded. These findings are presented as clinical observations only and do not constitute evidence for safety or efficacy of Panchakarma in active cardiac disease.

3.7 Post-Treatment HbA1c Achievement and Medication Reduction

Post-treatment HbA1c: 35.7% of patients achieved <7.0%; 25.0% achieved the ADA remission threshold of <6.5%.[5] Among the 21 patients with documented drug reduction data, 17.1% achieved complete medication cessation; 42.9% achieved partial reduction; mean reduction in those with any change was 63.0% (median 60.0%). A strong inverse correlation was observed

between baseline HbA1c and treatment response (r=−0.619, p=0.0004), confirming that more severely hyperglycemic patients derived proportionally greater glycemic benefit.

4. DISCUSSION

4.1 Dose-Response as Pharmacological Validation

The central finding — statistically significant correlations between PK sessions completed and both RBS reduction (r=−0.574, p=0.0006) and HR reduction (r=−0.561, p=0.0008) — provides a form of pharmacological validation for the CDC protocol mechanisms. A dose-response relationship is one of the criteria of causation established in epidemiological frameworks[1]: if more PK sessions consistently produce greater clinical improvement, this is consistent with a genuine biological effect rather than regression-to-mean or placebo response, both of which would be independent of dose.

The biological plausibility of PK dose-response is grounded in the pharmacokinetics of Basti therapy. Each Basti administration delivers berberine (from Daru Haridra), gymnemic acids (from Gudmar), and glycyrrhizin (from Yashti Madhu) via colonic portal absorption, bypassing first-pass hepatic metabolism.[2,3,4] Cumulative berberine exposure, which activates AMPK and sensitizes peripheral insulin receptors,[2] would be expected to produce proportionally greater insulin sensitivity improvement with each additional session — consistent with the observed dose-dependent RBS reduction. Similarly, cumulative Snehan-Swedan stimulation over multiple sessions progressively reinforces parasympathetic tone and reduces sympathetic overdrive — explaining the dose-dependent HR reduction.

4.2 The Minimum Intensity Threshold for HR and HbA1c Benefit

A clinically important observation from the tier analysis is the apparent minimum intensity threshold below which certain outcomes do not manifest. The Low tier (1–8 sessions, mean 5.8) showed non-significant HbA1c change (Δ −0.4%, p=0.237) and essentially no HR change (Δ −1.2 bpm, p=0.673), while the Mid tier (9–14 sessions, mean 11.8) showed significant improvement in both (HbA1c Δ −1.1%, p=0.002; HR Δ −9.0 bpm, p=0.0003). This threshold effect has direct clinical implications: prescribing fewer than 8–9 PK sessions may be insufficient to achieve glycemic and autonomic cardiovascular benefit in T2DM, and the standard 8-session protocol (if not completed as a full Mid-tier plan) may under-deliver for these endpoints.

The RBS response showed significance even in the Low tier (Δ −38.4 mg/dL, p=0.024), suggesting that immediate post-session blood glucose reduction may occur even with minimal PK exposure — possibly through the acute dietary restriction of the Prameha diet on treatment days and the brief post-Basti glycemic dip. However, sustained RBS reduction of −149.1 mg/dL in the High tier versus −38.4 mg/dL in the Low tier underscores that the dietary effect alone, without adequate PK intensity, produces only partial glycemic improvement.

4.3 Autonomic Cardiovascular Benefit: The HR-PK Relationship

The HR dose-response (r=−0.561, p=0.0008) is the most mechanistically interesting correlation in this study. Diabetic autonomic neuropathy — the underlying driver of elevated resting HR in T2DM — involves structural damage to autonomic nerve fibers from chronic hyperglycemia, with progressive parasympathetic withdrawal and sympathetic overdrive.[6] This is a slow biological process in both its development and its potential reversal.

The fact that Low-intensity Panchakarma (mean 5.8 sessions) produces essentially no HR change while High-intensity (mean 18.8 sessions) produces a 14.2 bpm reduction (p=0.003) is consistent with a cumulative autonomic restoration model: each Snehan-Swedan session contributes incremental parasympathomimetic stimulation via cutaneous thermoreceptors and vagal pathways;[7] each Basti session delivers constituents that protect enteric autonomic neurons from glucotoxic damage.[8] These effects accumulate across sessions, with meaningful autonomic restoration becoming clinically apparent beyond approximately 9 sessions — precisely what the tier analysis demonstrates.

4.4 Baseline Severity and Dose Assignment: Addressing Confounding

A potential confounding factor in dose-response analyses is that patients assigned to higher-intensity plans may have had more severe baseline disease — creating a scenario where the apparent dose-response reflects patient selection rather than treatment effect. This concern is partially supported by the data: the High tier had higher baseline RBS (309.6 vs. 207.7 mg/dL in Low) and higher HbA1c (9.4 vs. 7.4%). The inverse baseline HbA1c–response correlation (r=−0.619, p=0.0004) confirms that more severe patients achieved greater absolute improvements, which would inflate the High tier's apparent dose-response for glycemic outcomes.

However, this confounding argument is significantly weaker for the HR dose-response, because baseline HR was comparable across tiers (Low: 78.8 bpm, Mid: 87.1 bpm, High: 87.0 bpm). Both Mid and High groups had similar baseline HR, yet showed very different HR reductions (−9.0 vs. −14.2 bpm respectively), consistent with a genuine dose-effect rather than a severity-effect. The convergence of two independent dose-response signals (RBS and HR) pointing in the same direction, despite different baseline severity profiles, strengthens the biological plausibility of the relationship.

4.5 Practical Implications for Protocol Prescription

These findings have direct practical implications for how CDC protocol care plans should be prescribed:

For patients with primary glycemic management goals (HbA1c ≥9% or RBS ≥250 mg/dL), the High-intensity plan (15+ sessions) appears to deliver substantially superior outcomes — RBS Δ −149 mg/dL vs. Δ −38 mg/dL with <8 sessions.

For patients with autonomic cardiovascular risk (elevated resting HR, diabetic neuropathy), a minimum of 9–12 PK sessions appears to be required for meaningful autonomic benefit — fewer sessions do not cross the physiological threshold for HR reduction.

For patients with moderate glycemic burden (HbA1c 7–9%) and lower cardiovascular risk, the Mid-intensity plan (9–14 sessions) appears adequate — offering statistically significant improvement across all glycemic and BP parameters without requiring the extended commitment of a High-intensity plan.

These are preliminary observations from a single retrospective cohort and require prospective validation. However, they provide a rational evidence-informed framework for individualizing PK session prescribing based on clinical goals and disease severity.

4.6 Limitations

Retrospective design with non-randomized intensity assignment — patients with more severe disease were preferentially assigned to higher-intensity plans, creating confounding between baseline severity and PK dose.

The dose-response interpretation requires caution: longer follow-up in the High tier (median duration higher than Low tier) may contribute to greater improvement independently of sessions completed.

Small subgroup sizes (Low n=11, High n=10) limit statistical power for between-tier comparisons; results should be interpreted directionally.

Cardiac comorbidity cases (n=2) are observational only; cardiac medication records are incomplete, potentially confounding HR outcomes in these patients.

Lipid panel post-treatment data insufficient (n=4 genuine pairs) for formal lipid analysis.

Drug reduction data incompletely documented (40% missing) — medication changes cannot be reliably reported.

5. CONCLUSION

This study presents the first dose-response analysis of Panchakarma session intensity in T2DM management, using a cohort of 35 patients at Goregaon East, Mumbai, with PK sessions ranging from 1 to 22. The primary findings are:

A statistically significant positive correlation between PK sessions completed and both RBS reduction ($r=-0.574$, $p=0.0006$) and heart rate reduction ($r=-0.561$, $p=0.0008$) — providing pharmacological dose-response evidence for the CDC protocol's mechanisms.

A clear stepwise dose-response in the tier analysis: the High-intensity group (15–22 sessions) showed 4-fold greater RBS reduction, 12-fold greater HR reduction, and 4-fold greater absolute SBP reduction compared to the Low-intensity group (1–8 sessions).

A minimum intensity threshold of approximately 9 PK sessions below which HR and HbA1c benefits do not manifest — suggesting that sub-threshold prescribing may systematically under-deliver on the protocol's most important cardiovascular outcomes.

The overall cohort's simultaneous significance across all eight clinical parameters — achieved in an older, multi-comorbid urban Mumbai population with mean age 56.4 years and baseline SBP of 142.6 mmHg — confirms that the CDC-SP protocol delivers comprehensive cardiometabolic benefit. The dose-response evidence provides a practical evidence base for intensity-stratified PK prescribing: higher session counts for patients with severe glycemic burden or autonomic cardiovascular risk, moderate session counts for patients with intermediate disease.

Prospective dose-controlled trials — randomizing patients to defined PK session tiers with standardized follow-up, formal autonomic assessment (heart rate variability), and pharmacokinetic measurement of cumulative berberine and gymnemic acid exposure — are recommended to definitively characterize the PK dose-response in T2DM and establish evidence-based session count recommendations for different patient phenotypes.

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