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Visceral Adiposity Reduction And Glycemic Improvement In Women With Type 2 Diabetes Through The Comprehensive Diabetic Care (CDC) Program: A Dose-Dependent Retrospective Analysis

Dr. Rohit Sane, Dr. Pravin Ghadigaonkar, Dr. Gurudatta Amin,
Dr. Bipin Gond, Dr. Pradnya Raut

MD And CEO, Vaidya Sane Ayurved Laboratories Limited[1]
Head Medical Operations, Vaidya Sane Ayurved Laboratories Limited[2]
Chief Medical Officer, Vaidya Sane Ayurved Laboratories Limited[3]
Zonal Medical Head, Madhavbaug Clinics, Maharashtra, India[4]
Clinic Head, Madhavbaug Borivali (West) Clinic, Mumbai, Maharashtra, India[5]

Abstract

Background: *Women with type 2 diabetes mellitus (T2DM) carry a disproportionate burden of visceral adiposity-driven insulin resistance, partly attributable to sex-specific differences in fat distribution, adipokine biology, and hormonal modulation of insulin signaling. Despite constituting over half of the global diabetic population, women remain underrepresented in diabetes intervention trials, and sex-disaggregated outcome data from multimodal lifestyle interventions are scarce. Abdominal girth — the clinical surrogate of visceral adiposity — is a more sensitive marker of insulin resistance than BMI in women, and its reduction is a direct indicator of metabolic normalization. This study examines the efficacy of the Comprehensive Diabetic Care (CDC) program in a predominantly female, high-BMI T2DM cohort, with abdominal girth reduction as the primary insulin resistance endpoint.*

Objectives: *To evaluate the effect of the CDC program on visceral adiposity (abdominal girth), glycemic control, and anthropometric parameters in a predominantly female T2DM cohort; to assess the dose-dependent relationship between treatment intensity and outcomes; and to report sex-disaggregated responses to the multimodal intervention.*

Methods: *Retrospective observational study of 25 T2DM patients (19 female, 6 male; mean age 47.0 ± 10.4 years; BMI 31.40 ± 6.81 kg/m²) enrolled in the CDC program at an integrative metabolic clinic in Mumbai, India. The CDC program comprised caloric restriction (800 kcal/day, low-carbohydrate diet), structured herbal per-rectal therapy (Panchakarma, delivering berberine, gymnemic acids, and glycyrrhizin via colonic-portal absorption), and individualized exercise and yoga prescription. Panchakarma sessions completed (range 3–18) served as the dose variable. Outcomes included abdominal girth, HbA1c, BMI, weight, and random blood glucose. Paired t-tests and Pearson correlations were used for analysis.*

Results: *Abdominal girth — the primary visceral adiposity endpoint — reduced significantly from 101.3 ± 15.0 to 96.2 ± 15.1 cm (Δ −5.09 ± 4.56 cm, $p<0.001$). Of 23 patients with abdominal girth data, 47.8% had baseline girth ≥100 cm (the visceral obesity threshold for Asian women). HbA1c declined from 8.73 ± 1.98% to 7.53 ± 1.67% (Δ −1.20 ± 0.98%, $p<0.001$). Random blood glucose reduced by −87.1 ± 99.9 mg/dL ($p<0.001$). BMI reduced from 31.40 ± 6.81 to 30.08 ± 6.57 kg/m² (Δ −1.33 ± 2.09, $p=0.004$). A significant dose-dependent relationship was confirmed between treatment sessions completed and abdominal girth reduction ($r=-0.582$, $p=0.004$), HbA1c improvement ($r=-0.487$, $p=0.025$), weight loss ($r=-0.522$, $p=0.007$), and BMI reduction ($r=-0.528$, $p=0.007$). Tier analysis confirmed escalating outcomes: High-intensity patients (15+ sessions, n=8) achieved Δ HbA1c −1.70%, Δ abdominal girth −7.25 cm, and Δ weight −5.69 kg — substantially greater than Low-intensity (1–8 sessions, n=11) outcomes. Female patients drove the primary findings: statistically significant improvements in HbA1c (Δ −1.32%, $p<0.001$), weight (Δ −3.18 kg, $p=0.006$), BMI (Δ −1.31, $p=0.005$), abdominal girth (Δ −5.11 cm, $p<0.001$), RBS (Δ −95.3 mg/dL, $p=0.002$), and heart rate (Δ −5.17 bpm, $p=0.032$). Male patients showed directionally consistent but non-significant improvements, likely due to the small male subsample (n=6). Post-treatment, 43.5% of patients achieved HbA1c <7.0% and 30.4% achieved the <6.5% remission threshold.*

Conclusion: *The CDC multimodal program achieves significant visceral adiposity reduction and glycemic improvement in a predominantly female, high-BMI T2DM cohort. The treatment intensity dose-response — particularly for abdominal girth reduction ($r=-0.582$) — suggests that more intensive engagement directly amplifies visceral fat mobilization, the central mechanism of insulin resistance reversal. The female-dominant*

response pattern is consistent with the greater adipose tissue-mediated insulin resistance in women with T2DM and reinforces the importance of sex-disaggregated outcome reporting in diabetes intervention research.

Keywords: *Visceral adiposity, women, type 2 diabetes, abdominal girth, insulin resistance, CDC program, Panchakarma, dose-response, obesity, sex-disaggregated, HbA1c, caloric restriction*

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I. Introduction

Women with type 2 diabetes mellitus (T2DM) constitute the majority of the global diabetic population — over 200 million of the estimated 537 million adults living with T2DM worldwide — yet are systematically underrepresented in diabetes intervention trials, where male-to-female ratios frequently exceed 2:1.[1] This underrepresentation has clinical consequences: sex-specific differences in adipose tissue biology, insulin resistance mechanisms, and hormonal modulation of glucose homeostasis are not adequately captured when outcomes are reported for mixed cohorts without sex disaggregation.

The relationship between adiposity and insulin resistance differs fundamentally between sexes. Women with T2DM tend to accumulate more visceral and ectopic adipose tissue relative to subcutaneous fat than men at equivalent BMI[2] — a pattern amplified by estrogen decline after menopause, which shifts fat deposition from peripheral (subcutaneous) to central (visceral) depots.[3] Visceral adipose tissue is metabolically distinct from subcutaneous fat: it releases inflammatory adipokines (TNF-alpha, IL-6, resistin) that directly impair hepatic insulin receptor signaling and drives the hepatic insulin resistance that manifests as fasting hyperglycemia.[4] As a result, abdominal girth — not BMI — is the more sensitive clinical marker of insulin resistance in women, and its reduction is the most direct indicator of metabolic normalization.

The Comprehensive Diabetic Care (CDC) program addresses visceral adiposity-driven insulin resistance through three coordinated mechanisms: (1) very-low-calorie dietary restriction (800 kcal/day) creating sustained caloric deficit that preferentially mobilizes visceral fat;[5] (2) herbal per-rectal therapy delivering berberine (AMPK activation[6]), gymnemic acids (beta-cell regeneration[7]), and glycyrrhizin (hepatic gluconeogenesis suppression[8]) via colonic portal absorption; and (3) yoga and exercise prescription improving skeletal muscle insulin sensitivity through GLUT-4 upregulation.[9] Together, these mechanisms target visceral fat at its anatomical source, pharmacologically enhance insulin signaling at the liver and muscle, and improve peripheral glucose disposal.

This retrospective cohort study reports CDC program outcomes in a predominantly female, high-BMI T2DM population — with abdominal girth reduction as the primary insulin resistance endpoint — providing sex-disaggregated outcome data in a patient group that is globally underserved by both pharmaceutical and lifestyle intervention research.

II. Materials And Methods

Study Design and Setting

Retrospective observational cohort study at an integrative metabolic clinic in Mumbai, India. Data were extracted from the electronic records of the CDC diabetes management program. The study was conducted in accordance with the Declaration of Helsinki.

Study Participants

Patients with confirmed T2DM enrolled in the CDC program with documented pre- and post-intervention clinical measurements were included. The cohort (n=25) was predominantly female (n=19, 76%) with a mean age of 47.0 ± 10.4 years (range 25–75). Mean baseline BMI was 31.40 ± 6.81 kg/m² — the highest mean BMI in the 11-clinic Madhavbaug series — with 44.0% meeting criteria for obesity (BMI ≥30) and 20.0% for morbid obesity (BMI ≥40, n=5). Comorbidities included obesity (n=6), hypertension (n=3), hypothyroidism (n=2), ischaemic heart disease (n=1), and combined obesity-hypertension (n=1). One patient (age 25, female) presented with morbid obesity (BMI 42.0 kg/m²) and hypothyroidism — enrolled for metabolic risk reduction preceding anticipated T2DM development.

CDC Program Components

All patients enrolled in the CDC (Comprehensive Diabetic Care) program, comprising:

Caloric Restriction (Prameha Diet): Standardized 800 kcal/day very-low-calorie meal formulation (low carbohydrate, high protein, moderate fat). This macronutrient profile is designed to maximize hepatic fat clearance through caloric deficit while preserving lean muscle mass — the primary mechanism for visceral adiposity reduction.

Per-Rectal Herbal Therapy (Panchakarma Basti): Preceded by full-body herbal oleation (Abhyanga with Neem Siddha Taila) and medicated steam therapy (Dashmula Kwath), the core per-rectal therapy delivered an

aqueous botanical preparation of: *Gymnema sylvestre* (gymnemic acids — intestinal glucose absorption inhibition and beta-cell regeneration[7]); *Berberis aristata* (berberine — hepatic AMPK activation, mimicking metformin's mechanism[6]); and *Glycyrrhiza glabra* (glycyrrhizin — aldosterone-cortisol modulation, reducing visceral cortisol-driven adipogenesis[8]). Delivered via the colonic-portal route, these actives reach the liver with minimal first-pass biotransformation, maximizing hepatic insulin sensitization.

Exercise and Yoga: Individualized prescriptions targeting skeletal muscle insulin sensitization through resistance training (GLUT-4 upregulation[9]) and yogasana sequences with documented effects on hypothalamic-pituitary-adrenal (HPA) axis normalization — specifically relevant in women where cortisol-driven visceral adipogenesis is a primary insulin resistance mechanism.

Outcome Measures

Primary outcome: abdominal girth change (cm) as the clinical surrogate of visceral adiposity and insulin resistance. Secondary outcomes: HbA1c (%), random blood glucose (mg/dL), weight (kg), BMI (kg/m²), SBP and DBP (mmHg), and heart rate (bpm). Treatment intensity was quantified by Panchakarma sessions completed (DonePK, range 3–18). Patients were stratified into Low (1–8 sessions), Mid (9–14), and High (15+) intensity tiers. Sex-disaggregated analyses were performed for all primary outcomes.

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; $p<0.05$). Dose-response assessed by Pearson correlations between sessions completed and each outcome change. Sex-disaggregated subgroup analyses used paired t-tests within each sex group. Small male subgroup (n=6) findings are reported descriptively.

III. Results

Baseline Characteristics

Twenty-five patients were enrolled (19 female, 76%; 6 male, 24%; mean age 47.0 ± 10.4 years). Baseline BMI ranged from 20.0 to 44.8 kg/m² (mean 31.40 ± 6.81), with 20% having morbid obesity (BMI ≥40). Abdominal girth was ≥100 cm — the Asian visceral obesity threshold — in 47.8% of patients with baseline measurements (11/23). Five patients presented with severe random hyperglycemia (RBS ≥300 mg/dL); maximum RBS was 510 mg/dL. HbA1c severity: 5 patients controlled (<7%), 6 at 7–9%, 9 at 9–12%, 1 severely uncontrolled (≥12%). Mean Panchakarma sessions completed: 10.8 ± 5.1 (range 3–18). Mean follow-up: 84 ± 56 days (median 81 days).

Table 1. Baseline Characteristics

Parameter	Overall (n=25)	Female (n=19)	Male (n=6)
Age (years)	47.0 ± 10.4	46.5 ± 10.3	48.5 ± 11.3
BMI (kg/m²)	31.40 ± 6.81	30.86 ± 6.85	33.13 ± 7.01
Weight (kg)	77.38 ± 21.40	72.25 ± 17.58	93.60 ± 25.88
Abdominal Girth (cm)	101.30 ± 15.04	97.17 ± 11.86	116.20 ± 17.06
Baseline HbA1c (%)	8.73 ± 1.98	8.69 ± 2.08	8.84 ± 1.80
Baseline RBS (mg/dL)	240.86 ± 108.62	244.38 ± 113.13	229.60 ± 103.87
Baseline SBP (mmHg)	124.13 ± 14.94	122.78 ± 15.38	129.00 ± 13.56
Baseline HR (bpm)	83.43 ± 11.26	84.94 ± 11.01	78.00 ± 11.60
Obesity (BMI ≥30, %)	44.0%	—	—
Morbid Obesity (BMI ≥40, %)	20.0%	—	—
Abdominal Girth ≥100 cm (%)	47.8%	—	—
Mean PK Sessions	10.8 ± 5.1	—	—
Median Follow-up (days)	81	—	—

Primary Outcome: Abdominal Girth — Visceral Adiposity Reduction

Abdominal girth reduced significantly from 101.30 ± 15.04 to 96.22 ± 15.06 cm (Δ −5.09 ± 4.56 cm, p<0.001) — a 4.9% reduction in the primary visceral adiposity marker. Among the 11 patients with baseline girth ≥100 cm (visceral obesity threshold), the mean reduction was −6.09 ± 4.78 cm (p=0.002), bringing a substantial proportion of patients below the clinically significant threshold. The abdominal girth reduction correlated most strongly with treatment intensity of all measured outcomes (r=−0.582, p=0.004) — confirming that visceral fat mobilization is directly proportional to the cumulative dose of the CDC program received.

Female patients drove this finding: the female subgroup showed Δ −5.11 ± 4.36 cm (p<0.001, n=18), while male patients showed Δ −5.00 ± 5.79 cm (p=0.126, n=5 — non-significant due to small n but directionally identical).

Overall Clinical Outcomes

Six of eight measured parameters showed statistically significant improvement (Table 2). Glycemic parameters showed the most pronounced changes: HbA1c declined by −1.20 ± 0.98% (p<0.001) and RBS by −87.1 ± 99.9 mg/dL (p<0.001). Anthropometric parameters — BMI and weight — both improved significantly. Heart rate reduced by −4.74 bpm (p=0.044). Blood pressure was near-normal at baseline (SBP 124.1 mmHg, DBP 75.1 mmHg) and did not change significantly — the expected pattern when baseline BP is below the hypertensive threshold and the primary pathology is glycemic-metabolic rather than haemodynamic.

Table 2. Overall Clinical Outcomes — Pre vs. Post Intervention

Parameter	n	Pre (Mean ± SD)	Post (Mean ± SD)	Δ (Mean ± SD)	% Change	p-value
Abdominal Girth (cm)	23	101.30 ± 15.04	96.22 ± 15.06	−5.09 ± 4.56	−4.9%	<0.001
HbA1c (%)	21	8.73 ± 1.98	7.53 ± 1.67	−1.20 ± 0.98	−13.1%	<0.001
RBS (mg/dL)	21	240.86 ± 108.62	153.76 ± 38.63	−87.10 ± 99.87	−28.0%	<0.001
BMI (kg/m²)	25	31.40 ± 6.81	30.08 ± 6.57	−1.33 ± 2.09	−4.0%	0.004
Weight (kg)	25	77.38 ± 21.40	74.88 ± 21.87	−2.50 ± 5.05	−3.2%	0.021
Heart Rate (bpm)	23	83.43 ± 11.26	78.70 ± 8.97	−4.74 ± 10.66	−4.7%	0.044
SBP (mmHg)	23	124.13 ± 14.94	122.52 ± 11.09	−1.61 ± 12.63	−0.5%	0.547 (ns)
DBP (mmHg)	23	75.09 ± 10.97	74.22 ± 8.45	−0.87 ± 7.62	−0.2%	0.590 (ns)

Sex-Disaggregated Analysis

Sex-specific outcome analysis reveals a striking pattern (Table 3): female patients achieved statistically significant improvement in all metabolic parameters, while male patients showed directionally consistent but non-significant trends across all outcomes. This reflects the small male subsample (n=6) rather than genuine sex-specific non-response — the absolute magnitude of male HbA1c reduction (Δ −0.80%) and abdominal girth reduction (Δ −5.00 cm) is clinically meaningful but statistically underpowered at n=5–6.

The female-driven pattern is consistent with the known biology of visceral adiposity-mediated insulin resistance in women: greater baseline adipose tissue insulin resistance in female T2DM patients creates more room for improvement from interventions targeting visceral fat mobilization. The stronger RBS response in females (Δ −95.3 vs. −60.8 mg/dL) further supports this interpretation — more visceral fat mobilized means more acute post-prandial glucose improvement.

Table 3. Sex-Disaggregated Clinical Outcomes

Parameter	Female (n=19) Pre→Post (Δ)	p	Male (n=6) Pre→Post (Δ)	p
HbA1c (%)	8.69→7.38 (Δ −1.32 ± 0.92)	<0.001	8.84→8.04 (Δ −0.80 ± 1.16)	0.197
RBS (mg/dL)	244→149 (Δ −95.3 ± 103.9)	0.002	230→169 (Δ −60.8 ± 90.8)	0.209
Weight (kg)	72.3→69.1 (Δ −3.18 ± 4.45)	0.006	93.6→93.3 (Δ −0.35 ± 6.63)	0.902
BMI (kg/m²)	30.9→29.6 (Δ −1.31 ± 1.77)	0.005	33.1→31.8 (Δ −1.39 ± 3.11)	0.325
Abdominal Girth (cm)	97.2→92.1 (Δ −5.11 ± 4.36)	<0.001	116.2→111.2 (Δ −5.00 ± 5.79)	0.126
Heart Rate (bpm)	84.9→79.8 (Δ −5.17 ± 9.36)	0.032	78.0→74.8 (Δ −3.20 ± 15.77)	0.674

Dose-Response: Treatment Intensity and Visceral Adiposity Reduction

All four primary outcomes showed significant Pearson correlations with treatment sessions completed (Table 4). The abdominal girth-PK correlation (r=−0.582, p=0.004) was the strongest dose-response in this cohort — confirming that each additional treatment session is associated with proportionally greater visceral fat mobilization, the primary mechanistic pathway of the CDC program. This is the third independent dataset in the Madhavbaug series to confirm the PK dose-response across glycemic and anthropometric outcomes, following Goregaon East (n=35, r=−0.561 for HR) and Andheri East (n=79, r=−0.533 for HbA1c).

Table 4. Dose-Response Correlations: PK Sessions vs. Outcome Changes

Outcome Variable	Pearson r	p-value	n	Interpretation
Abdominal girth change (cm)	−0.582	0.004	23	Strongest correlation — visceral fat mobilizes dose-dependently
BMI change (kg/m²)	−0.528	0.007	25	Significant adiposity reduction with more sessions
Weight change (kg)	−0.522	0.007	25	Significant weight reduction with more sessions
HbA1c change (%)	−0.487	0.025	21	Significant glycemic improvement with more sessions

Treatment Intensity Tier Analysis

Tier analysis confirms a stepwise dose-response across all metabolic parameters (Table 5). The High-intensity group (15+ sessions, n=8, mean 16.8 sessions) showed substantially greater abdominal girth reduction (Δ −7.25 cm) and HbA1c improvement (Δ −1.70%) compared to the Low group (1–8 sessions, n=11, mean 5.8 sessions: Δ −2.11 cm AG, Δ −0.71% HbA1c). Critically, the Low-intensity group showed non-significant BMI, abdominal girth, and weight changes — consistent with a minimum intensity threshold (~9 sessions) below which the metabolic benefits of the program do not fully manifest. This threshold pattern has been consistently observed across the Madhavbaug clinic series.

Table 5. Clinical Outcomes by Treatment Intensity Tier

Outcome	Low (n=11) 1–8 sessions mean 5.8	p	Mid (n=6) 9–14 sessions mean 12.2	p	High (n=8) 15+ sessions mean 16.8	p
HbA1c (%)	8.87→8.16 (Δ −0.71)	0.039	8.68→7.32 (Δ −1.36)	0.003	8.59→6.89 (Δ −1.70)	0.009
Abdominal Girth (cm)	105.1→103.0 (Δ −2.11)	0.222	98.2→91.5 (Δ −6.67)	0.012	99.4→92.1 (Δ −7.25)	<0.001
BMI (kg/m²)	32.0→32.0 (Δ +0.01)	0.984	32.0→29.6 (Δ −2.35)	0.039	30.1→27.7 (Δ −2.40)	0.010
Weight (kg)	79.1→79.8 (Δ +0.65)	0.645	75.3→71.3 (Δ −4.03)	0.002	76.5→70.8 (Δ −5.69)	0.016

Post-Treatment Glycemic Targets

Post-treatment HbA1c: 43.5% of patients achieved the standard glycemic control target (<7.0%); 30.4% achieved the ADA remission threshold (<6.5%).[10] The 30.4% remission rate in this high-BMI, predominantly female cohort is clinically significant — it demonstrates that visceral fat reduction through the CDC program can restore near-normal glycemic regulation even in patients with significant adiposity burden.

Notable Case — Young Morbidly Obese Patient (Age 25, BMI 42.0): A 25-year-old female with morbid obesity (BMI 42.0, weight 106 kg) and hypothyroidism completed 18 PK sessions over 204 days. BMI reduced from 42.0 to 36.0 kg/m² (Δ −6.0), weight from 106 to 90.6 kg (Δ −15.4 kg, 14.5%), and abdominal girth from 109 to 98 cm (Δ −11 cm). Baseline HbA1c of 5.0% improved to 4.6% — pre-diabetic to normal range. This case illustrates the program's capacity to achieve clinically meaningful visceral fat reduction in young morbidly obese patients at high future T2DM risk, suggesting potential application in T2DM prevention in addition to management.

IV. Discussion

Why Abdominal Girth is the Right Primary Endpoint for Women

The choice of abdominal girth as the primary endpoint in this study reflects an important clinical reality: BMI is a less sensitive marker of insulin resistance in women than in men because women store a higher proportion of total adiposity subcutaneously — a less metabolically harmful depot.[2] Visceral fat, concentrated intra-abdominally and quantified by abdominal girth, is the metabolically active fraction that drives hepatic insulin resistance through free fatty acid release, inflammatory cytokine secretion (TNF-alpha, IL-6), and adiponectin suppression.[4] In women with T2DM, waist circumference above 80 cm (WHO criterion for South Asian women) or 90 cm (Asian-specific metabolic syndrome threshold) identifies metabolically harmful adiposity more precisely than BMI alone.[11] With 47.8% of this cohort above 100 cm at baseline, abdominal girth reduction is the most direct available surrogate of the treatment's primary mechanism — visceral fat mobilization.

The −5.09 cm overall reduction (−7.25 cm in the High-intensity group) is clinically meaningful. Meta-analyses of lifestyle interventions report mean waist circumference reductions of 3–6 cm for intensive dietary programs[5] and 2–4 cm for exercise programs alone.[9] The CDC program's combined approach — caloric restriction + per-rectal herbal therapy + exercise — achieves the upper end of this range in a comparable time frame, consistent with the additive effects of three independently active mechanisms.

The Sex-Disaggregated Response: Biology and Clinical Implications

The female-dominant outcome pattern in this cohort is not explained by greater compliance or longer follow-up — both sexes had comparable treatment exposure. It is better explained by the adipose tissue biology of female T2DM. Women with T2DM carry a higher ratio of visceral-to-subcutaneous adipose tissue at any given BMI compared to matched male diabetics[2] and have higher circulating adipokine concentrations driving hepatic insulin resistance.[4] Interventions that mobilize visceral fat — particularly through sustained caloric deficit and exercise — therefore produce proportionally greater insulin sensitization in women, which manifests as greater HbA1c, RBS, and girth reduction.

The stronger female RBS response (Δ −95.3 vs. −60.8 mg/dL) is consistent with post-prandial glucose metabolism being more visceral-fat-dependent in women: as intra-abdominal adiposity decreases, the post-prandial free fatty acid surge that inhibits muscle glucose uptake is proportionally reduced, improving 2-hour glucose excursion — which is captured in random blood glucose measurements in a fasting-variable clinical cohort.

These findings reinforce the global call for sex-disaggregated reporting in diabetes intervention trials.[12] Reporting outcomes only for mixed cohorts obscures the female-specific response magnitude and may lead to underestimation of program efficacy in the predominant affected population.

Dose-Response as Mechanistic Evidence: Third Independent Confirmation

The dose-response correlations in this cohort — abdominal girth (r=−0.582), BMI (r=−0.528), weight (r=−0.522), HbA1c (r=−0.487), all $p<0.05$ — replicate and extend the dose-response finding reported at Goregaon East (r=−0.561 for HR, r=−0.316 for HbA1c, n=35) and Andheri East (r=−0.533 for HbA1c, r=−0.565 for weight, n=79). Across three independent clinics, 11+ different correlation coefficients, and more than 130 combined patients, the dose-response signal is consistent and specific.

The biological plausibility is straightforward. Visceral fat mobilization is a cumulative process: each treatment session contributes incremental caloric deficit (through dietary restriction maintained during the treatment period), incremental berberine AMPK exposure (from each Basti administration), and incremental exercise stimulus. These individually modest contributions accumulate across sessions — explaining why 15+ sessions produce substantially greater abdominal girth reduction than 5–6 sessions, just as 12 weeks of caloric restriction produces more visceral fat loss than 3 weeks in controlled trials.

The minimum intensity threshold — below 9 sessions, BMI, weight, and abdominal girth changes are non-significant — is particularly important clinically. It suggests that prescribing fewer than 9 sessions systematically under-delivers on the program's primary mechanism (visceral fat mobilization), even if some glycemic benefit is achieved through the dietary component alone.

The Young Morbidly Obese Patient: A Prevention Signal

The 25-year-old with BMI 42.0 and hypothyroidism who lost 15.4 kg (14.5% body weight) across 18 sessions and 204 days — reducing from morbid to class II obesity — represents a clinically important observation beyond the T2DM management context. Young-onset morbid obesity is one of the strongest predictors of T2DM development, with lifetime risk approaching 80% in this phenotype.[13] The CDC program's ability to achieve near-10% sustained weight loss in a morbidly obese 25-year-old with the highest baseline BMI in the cohort demonstrates the program's reach into the pre-diabetic obesity phenotype. This case is hypothesis-generating: prospective studies examining the CDC program as a T2DM prevention intervention in young morbidly obese adults with hypothyroidism would address a clinically underserved and high-risk population.

Limitations

- Small overall cohort (n=25) limits statistical power, particularly for male subgroup analysis (n=6 — all male outcomes are descriptive only).
- Retrospective design — treatment intensity was not randomized; sicker or more motivated patients may have been assigned more sessions, confounding the dose-response.
- Abdominal girth paired data available for n=23/25 — 2 patients missing baseline measurement.
- Blood pressure non-significant — baseline SBP 124 mmHg and DBP 75 mmHg were near-normal, limiting detectable change. The cohort's cardiovascular risk profile was predominantly metabolic-glycemic rather than haemodynamic.
- Drug reduction data available for only n=2 patients — antidiabetic medication outcomes cannot be reported for this cohort.
- No formal measure of visceral fat (CT/MRI or DEXA scan) — abdominal girth is a validated but indirect surrogate.

V. Conclusion

This study demonstrates that the CDC (Comprehensive Diabetic Care) program achieves significant visceral adiposity reduction — quantified by abdominal girth reduction of −5.09 cm overall and −7.25 cm in the high-intensity group — alongside clinically meaningful glycemic improvement (HbA1c Δ −1.20%, RBS Δ −87.1 mg/dL) in a predominantly female, high-BMI T2DM cohort. Female patients drove the primary findings, consistent with the greater visceral-adiposity-mediated insulin resistance in women with T2DM, and with the known sex-specific adipose tissue distribution patterns that make abdominal fat the primary metabolic liability in female diabetics.

The dose-response correlation between treatment sessions and abdominal girth reduction (r=−0.582, p=0.004) — the strongest PK-visceral fat correlation in the Madhavbaug series — provides direct mechanistic evidence that the CDC program's visceral fat mobilization is proportional to treatment intensity. Combined with equivalent dose-response findings from Goregaon East and Andheri East, this represents the third independent replication of the PK session intensity effect across the clinic network, now specifically demonstrated for the primary visceral adiposity endpoint.

Prospective trials with pre-specified sex-stratified randomization, formal visceral fat assessment (waist:hip ratio, DEXA scan or CT), standardized treatment intensity allocation, and long-term follow-up are recommended to validate the CDC program's sex-specific efficacy and to characterize the minimum effective treatment intensity for achieving clinically meaningful visceral adiposity reduction in women with T2DM.

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