

FINAL CLINICAL CASE STUDY

Weight and metabolic change over 12 weeks.

Sarah's journey with DarDoc. A retrospective study of weight and metabolic outcomes during physician-led care.



Personal photographs published with consent. Images provide personal context; photographic conditions were not standardized.

Retrospective clinical case study / One participant / Descriptive analysis

HOW TO READ THIS REPORT

The study, in context.

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Structured abstract

Objective

To describe body-weight and HOMA-IR changes during the first 12 weeks of Wegovy (semaglutide) injections and interpret the findings alongside recorded treatment activity.

Methods

Retrospective clinical case study of one participant, using the confirmed case summary, laboratory extracts and clinical activity records. Absolute and relative changes were calculated from reported values. The study had no comparator.

Results

Weight was reported as 96.5 to 86.5 kg, a reduction of 10 kg (10.4%). HOMA-IR was 3.3 at Week 2 and 0.9 at Week 12. HbA1c was reported as 5.5%, normal range; the measurement date was not supplied.

Interpretation

Body weight and HOMA-IR decreased over their respective observation windows. The single-patient design does not isolate a treatment effect or establish long-term maintenance, a dose-response relationship or a safety rate.

Clinical presentation and study methods.

Available clinical description

The supplied summary identifies Sarah and Wegovy (semaglutide) injections during the first 12 weeks. Body weight is summary-reported; HOMA-IR values are accompanied by laboratory extracts and confirmed programme weeks. The supplied DarDoc patient chart records a 33-year-old female participant. Treatment started 6 Jul 2026 under Dr. Sami and is ongoing. The Week 12 weight was reported around 28 Sep 2026.

Baseline assessment

Recorded height is 160 cm. Calculated BMI is 37.7 kg/m² initially and 33.8 kg/m² at Week 12, using the case-summary weights, not the separate dashboard snapshot. Waist circumference, examination findings, presenting symptoms and prescribing eligibility were outside the analytical dataset.

Clinical history

The patient chart records: None reported; No known drug allergies; No cancer or tumour history reported. Family history, previous weight-management attempts and concomitant medicines were not captured in this dataset. Prescribing suitability was not inferred from the outcomes.

Design and analytical approach

This is a descriptive, retrospective clinical case study (n = 1). The available values are shown without imputation. Absolute and relative changes are calculated directly from the first available value. No hypothesis test, confidence interval or population estimate is appropriate for this record.

Source hierarchy

Data were abstracted from the supplied case summary, available laboratory extracts and clinical activity records. Reported observations, calculated values and literature findings were kept separate. Photographs and video provided personal context.

Reporting framework

The CARE checklist provides the reporting structure for a case report: clinical information, chronology, intervention, outcomes, discussion and patient perspective. The checklist guided presentation of the clinical information and limitations. [9]

Treatment and follow-up.

This outcome window covers the first weeks of care; ongoing treatment is recorded separately.

Event	Available record	Provenance / boundary
Treatment start	6 Jul 2026	Dr. Sami; ongoing
Starting weight	96.5 kg	Reported at treatment start; weighing conditions not supplied
First HOMA-IR	3.3 at Week 2	First available index, not a pretreatment baseline
Week 6 weight	90.9 kg; 5.6 kg reduction	Calculated from the reported Week 6 reduction of 5.6 kg from 96.5 kg; not an independently supplied scale reading.
HOMA-IR retest	0.9 at Week 12	Exact specimen collection date not supplied
Follow-up weight	86.5 kg; Around 28 Sep 2026	Summary-reported weight; no body-composition measurement

Timing and provenance

The first HOMA-IR result belongs to Week 2, with retesting at Week 12. Programme weeks must not be substituted for exact specimen dates. The Week 12 date is approximate.

Treatment exposure

Prescription and delivery events appear in the treatment-exposure section. Supply records do not establish every administered dose, missed dose or complete titration schedule.

03 / BODY-WEIGHT OUTCOME

Change in body weight.

10 kg

10.4% of starting weight



Photograph is narrative context, not a measurement of body composition.

Reported starting weight

96.5 kg

Week 6 (calculated)

90.9 kg

Week 12 follow-up

86.5 kg

Figure 1. Separate checkpoints on a zero-based scale. An intermediate weight may be calculated from reported loss; no continuous trajectory is fitted.

Calculation

Absolute reduction = $96.5 - 86.5 = 10$ kg. Relative reduction = $(10 / 96.5) \times 100 = 10.4\%$, rounded to one decimal place.

Measurement boundary

Weighing conditions and body composition were not supplied. Using height 160 cm, calculated BMI is 37.7 to 33.8 kg/m². Fat-versus-lean loss cannot be described.

The available clinical data.

NR means not reported in the supplied material. It does not mean normal, absent or unchanged.

Measure / unit	First value	Follow-up	Status / interpretation
Body weight / kg	96.5	86.5	Week 12: Around 28 Sep 2026
HOMA-IR / index	3.3	0.9	Week 2 to Week 12; first result is not pretreatment
Fasting glucose / mmol/L	NR	NR	Required to audit HOMA calculation
Fasting insulin / $\mu\text{U/mL}$	NR	NR	Required to audit HOMA calculation
Height / cm	160	-	Recorded chart height
BMI / kg/m^2	37.7	33.8	Calculated using case-summary weights
Waist circumference / cm	NR	NR	Central adiposity not quantified
Blood pressure / mmHg	NR	NR	No haemodynamic outcome available
HbA1c / %	5.5	No pair	Normal range; date not supplied
Lipids / laboratory units	NR	NR	No lipid outcome available
Liver / renal markers	NR	NR	Clinical monitoring record incomplete
Adverse events / adherence	NR	NR	Not systematically documented

Table 1. Study outcome inventory. Reported findings are distinguished from domains not captured in the analytical dataset; NR does not mean normal or absent.

Understanding the HOMA-IR change.

Programme timepoint	Recorded index	Provenance
Week 2	3.3	Laboratory extract; confirmed programme week
Week 12	0.9	Retest; confirmed programme week

3.3 to 0.9

Absolute and relative change

The absolute difference is 2.4 index units. Relative reduction = $(2.4 / 3.3) \times 100 = 72.7\%$. That percentage describes the index, not a measured percentage recovery of insulin sensitivity or reduction in diabetes risk.

What the laboratory states

The supplied report lists HOMA-IR below 1.0 as insulin-sensitive. The follow-up index is read against that laboratory reference, without converting the reference category into a confirmed diagnosis. The laboratory labels values above 1.9 as early insulin resistance and above 2.9 as significant insulin resistance. Its Week 2 index exceeds the latter threshold; its follow-up index is below the stated insulin-sensitive threshold of 1.0.

Audit of the calculation

Original fasting glucose and insulin values are unavailable. The model version, collection preparation and assay information have not been independently verified. We cannot recalculate the reported index or determine which input drove the change.

Interpreting the direction

A lower index describes a change in the supplied values, not proof of a treatment-specific mechanism. Fasting and analytical comparability were not independently established.

HbA1c context

HbA1c was reported as 5.5%, normal range; the measurement date was not supplied. NIDDK describes HbA1c below 5.7% as the normal range. A single result does not show a change or establish remission. [10]

A surrogate, with important limits.



Scientific illustration, not microscopy or a representation of this participant's tissue.

The HOMA model

HOMA models the fasting, steady-state relationship between glucose and insulin. The original HOMA1 approximation is fasting insulin ($\mu\text{U/mL}$) multiplied by fasting glucose (mmol/L), divided by 22.5. HOMA2 uses a computer model and is not interchangeable with that simple equation. Assay variation and pulsatile insulin secretion affect estimates; model version and sample conditions belong in the methods record. [2]

Where the model began

HOMA estimates insulin resistance rather than measuring it directly. The original model was evaluated against clamp-based assessments. A longitudinal index is useful only when its inputs and collection conditions are understood; a change in the reported index alone cannot identify whether glucose, insulin, or both changed. [3]

Thresholds and population

Reference thresholds require context. In a national Iranian analysis of 3,071 adults aged 25–64, investigators derived cut-offs for identifying metabolic syndrome within that population. Such work illustrates why a laboratory reference is not a universal diagnostic boundary. Population, assay and the outcome being classified must be specified. [8]

Case-specific interpretation

Original fasting inputs, collection preparation and analytical comparability were not available for independent assessment. A reference-category change describes the reported index; it does not establish diabetes remission or a cardiovascular outcome.

Appetite, receptors and food intake.



GLP-1 receptor activation

A biological pathway.
Receptor activity was not measured in this study.

Conceptual receptor illustration. Neither receptor activity nor tissue-level signalling was measured in this case.

Evidence for appetite-related effects

In a randomized, double-blind crossover experiment involving 30 adults with obesity, 12 weeks of semaglutide titrated to 1.0 mg reduced total ad libitum energy intake across measured meals by 24% versus placebo. Appetite and eating-control measures also improved. The experiment supports an appetite-related mechanism; it does not establish any individual participant's dietary intake or treatment dose. [4]

Case-specific interpretation

Hunger, fullness, cravings and food intake were not quantified by validated questionnaires or food diaries in this study. Published mechanistic findings provide biological context. The observed weight change alone does not establish energy intake, receptor activity or a change in eating behaviour.

Trials, in clinical context.

Study	Population and duration	Published finding
STEP 1 [1]	1,961 adults without diabetes; BMI at least 30, or at least 27 with a weight-related condition. 68 weeks.	Mean weight change: -14.9% versus -2.4%. Estimated difference: -12.4 percentage points (95% CI -13.4 to -11.5; P<0.001).
SELECT [5]	17,604 adults aged at least 45 with established cardiovascular disease and overweight or obesity, without diabetes. Mean follow-up 39.8 months.	Primary cardiovascular events: 6.5% versus 8.0%; hazard ratio 0.80 (95% CI 0.72-0.90). Permanent discontinuation due to adverse events: 16.6% versus 8.2%.
STEP 1 extension [6]	327 participants; exploratory follow-up after withdrawal to week 120.	17.3% mean loss in the semaglutide subset by week 68; 11.6 percentage points regained by week 120; net loss 5.6%.

Trial design and estimand

Double-blind, 2:1 randomization; subcutaneous semaglutide 2.4 mg weekly versus placebo, both with lifestyle intervention. The mean and confidence interval describe a trial-level estimate; they are not uncertainty bounds for this participant. These are group averages at a different timepoint. An individual short-term observation cannot be ranked against the trial mean or treated as a controlled treatment effect.

Cardiovascular applicability

The endpoint combined cardiovascular death, nonfatal myocardial infarction and nonfatal stroke. The trial population and endpoint differ from a short-term weight and HOMA-IR case. A short-term HOMA-IR change cannot be substituted for an adjudicated cardiovascular endpoint.

Durability and follow-up

Short-term follow-up cannot establish maintenance. Later weight, treatment status and metabolic measurements are needed to describe durability. The extension withdrew both medicine and structured lifestyle intervention; follow-up was exploratory. Later observations are necessary to distinguish maintenance, further loss and regain.

What the literature adds

Controlled evidence provides biological and treatment context. It cannot supply missing doses, diagnoses, safety events or lifestyle information for a separate case. Comparing unlike timepoints as if they were treatment rankings would overstate what either record can show.

Ongoing care and monitoring.

Date	Record type	Recorded event
23 Jul 2026	prescription	Wegovy 1 mg prescribed
4 Sep 2026	review	Consultation completed and outcome recorded by Dr. Sami
4 Sep 2026	prescription	Wegovy 1.7 mg prescribed
5 Sep 2026	delivery	Wegovy 1.7 mg delivered
1 Oct 2026	prescription	Refill and prescription recorded by Dr. Sami; stay at current dose
5 Oct 2026	prescription	Wegovy 2.4 mg prescribed
6 Oct 2026	delivery	Wegovy 2.4 mg delivered

Supply versus administration

Prescriptions and deliveries document recorded medication supply, not each administered dose or complete titration. An administration log, adherence measure and interruption history were not included in the dataset.

Safety context

The June 2026 US label describes GLP-1 receptor activity, appetite-related reduction in calorie intake, glucose-dependent insulin secretion and glucagon suppression. It lists gastrointestinal reactions and warnings including pancreatitis, gallbladder disease, volume-depletion-related kidney injury and hypoglycaemia with insulin or secretagogues. Contraindications include personal or family history of medullary thyroid carcinoma, MEN 2 and serious hypersensitivity. This reference does not replace UAE prescribing information. [7]

Case-specific tolerability

An adverse-event record, active symptom assessment and event management were not supplied. Tolerability cannot be concluded from a reduction in body weight.

Interpretation of safety

The study dataset did not include systematic records of adverse-event severity, duration, attribution or management. Recorded prescription supply does not establish tolerability or a population adverse-event rate.

Clinical interpretation and conclusions.

Concordant direction, limited attribution

The supplied record contains a 10.4% reduction in reported body weight and a 72.7% reduction in the recorded HOMA-IR index. Their direction is concordant, but the measures have different sources and unresolved timing. Concordance is descriptive; it does not establish that one change caused the other.

Alternative explanations

Changes in nutrition, activity, other medicines, illness, fluid balance, measurement conditions and ordinary biological variability have not been characterized. Without a comparator or repeated baseline measures, their contribution cannot be separated from medication exposure.

No population-level inference

One selected participant cannot estimate average response, the likelihood of benefit or the frequency of harms. A patient selected for an illustrative story may differ from those with a smaller response, incomplete follow-up or treatment discontinuation. This case-selection context limits generalizability.

Missing information limits precision

The absence of glucose and insulin inputs prevents independent HOMA verification. Undocumented weighing conditions and dates prevent a standardized weight trajectory. A descriptive calculation can be exact for the supplied numbers while uncertainty remains about their clinical interpretation.

Outcomes not established

A single HbA1c result is reported; no HbA1c change or remission is established. No body-composition, cardiovascular, hepatic, renal or quality-of-life outcome can be attributed to this case. The photograph is personal context, not a validated endpoint. Nor does short-term follow-up establish persistence after treatment withdrawal.

Clinical conclusion

During the first 12 weeks of care, body weight decreased by 10 kg (10.4%). HOMA-IR decreased from 3.3 at Week 2 to 0.9 at Week 12. These findings describe one participant's clinical course. The study design and available measurements do not support causal attribution or population-level efficacy, safety or durability estimates.

11 / REFERENCES

The scientific sources.

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09 / CARE. Case report checklist: patient information, timeline, diagnostic assessment, intervention, follow-up, patient perspective and consent.

<https://www.care-statement.org/checklist>

10 / NIDDK. The A1C Test & Diabetes. HbA1c percentage and interpretation; normal range below 5.7%.

<https://www.niddk.nih.gov/health-information/diagnostic-tests/a1c-test>

Methods, provenance and study boundaries.

Data provenance

This clinical case study uses the confirmed case summary, available laboratory extracts and clinical activity records. Calculated weight, BMI and index changes are reproducible from the reported values. Identifiers, full laboratory reports and correspondence are excluded from this document.

Study design

Retrospective, descriptive clinical case study of one participant receiving routine care. The study had no randomized allocation, comparator or independent source-record audit. Missing measurements were retained as not reported rather than imputed.

Consent

DarDoc confirmed publication consent for the participant's first name, personal photographs, video and reported outcomes. Photographs provide personal context and were not standardized clinical measurements.

Clinical glossary

Surrogate marker: a measure reflecting a biological process rather than a directly observed clinical outcome.

Estimand: the treatment effect a study is designed to estimate.

Confidence interval: uncertainty around a study estimate, not a prediction interval for one participant.

Scientific context

Published clinical trials describe separate populations, methods and endpoints. Their findings provide context without becoming observations in this case. Cellular and receptor images are conceptual illustrations.

Reporting framework

The CARE checklist guided the structure of this report. Outcomes are interpreted within the recorded observation window and the limitations of a single-patient design. The study does not estimate the response an individual reader should expect.