Long-term type 2 diabetes mellitus remission in non-obese patients using a structured low carbohydrate diet programme: A case series of three Malaysian patients

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SUMMARY

Evidence for type 2 diabetes mellitus (T2DM) remission derives predominantly from overweight and obese populations using very low calorie diet (VLCD) approaches, leaving an evidence gap for non-obese patients with normal body mass index (BMI). Within an unselected real-world cohort of 45 patients on a single structured, medically supervised low carbohydrate diet (LCD) programme (carbohydrate 20–50 g/day) over five years, six had a normal BMI. Five of the six completed the six-month programme and all five achieved remission, spanning Chinese, Indian, and Malay ethnicity; one Chinese patient dropped out. Five-year durability was confirmed in three (two Chinese, one Indian; BMI 20.5–24.5 kg/m²), who form this report; the two Malay remitters were lost to contact after programme completion. The three patients with five-year data sustained remission (HbA1c 5.4%, 6.1%, 6.2%) with net weight loss of 3.7–7.6 kg; one had a transient HbA1c of 6.5% in year three (brief relapse) before returning to remission with renewed dietary adherence. No diabetes-related complications were reported. These observations suggest short-term remission may be readily attainable in non-obese South East Asian patients across ethnic groups, and that the principal barrier to confirming long-term durability may be retention rather than biological response. Larger prospective controlled trials with robust retention strategies are warranted.

INTRODUCTION

T2DM remission, defined by international consensus as an HbA1c below 6.5% (48 mmol/mol) sustained for at least three months without glucose-lowering pharmacotherapy, has become an achievable therapeutic target.[1] The evidence base relies predominantly on weight loss in overweight and obese individuals: the landmark DiRECT trial (BMI 27–45 kg/m²) achieved remission in 48% at one year,[2] though its five-year extension found only 10% of initial remitters remained in remission, with sustained weight loss the principal determinant.[3] Whether remission is achievable in non-obese patients is a critical emerging question. The ReTUNE study (2023) gave the first prospective evidence that approximately 70% of non-obese participants (mean BMI 24.8 kg/m²) could achieve remission through structured weight loss, the key mechanism being reduction of ectopic liver and pancreatic fat, consistent with the Personal Fat Threshold hypothesis.[4] However, ReTUNE used an 800 kcal/day total diet replacement formula; whether a less restrictive, more sustainable LCD achieves comparable remission in non-obese patients, particularly in South East Asians, remains unclear.

South East Asian populations, including Malaysians, develop T2DM at lower BMI thresholds than Western counterparts, driven by higher visceral-to-subcutaneous fat ratios and reduced beta-cell reserve.[5] Malaysia bears one of the region's highest T2DM burdens (18.3% of adults; NHMS 2019), with some subgroups exceeding 20%.[6] Identifying sustainable remission strategies for non-obese patients is therefore of clinical and public health importance. I report a case series drawn from an unselected cohort managed on a single structured LCD programme, focusing on the non-obese subgroup, three of whom have confirmed five-year outcomes. Baseline characteristics are summarised in Table I and clinical outcomes in Table II.

CASE PRESENTATION

Cohort and patient flow

Over the five-year period, 45 patients with T2DM across normal-weight, overweight, and obese categories were enrolled in a single remission programme using an identical protocol; recruitment was not by BMI. Six had a normal BMI (<25 kg/m²). Five of the six completed the programme and all five achieved remission — two Chinese, one Indian, and two Malay (one male, one female); the sixth (a Chinese female) dropped out. Of the five remitters, three (two Chinese males, one Indian male) completed five-year follow-up and are described below; the two Malay remitters could not be contacted after programme completion and were lost to the five-year assessment despite remission at six months.

Programme structure

All patients followed a six-month programme of a ketogenic-range LCD (carbohydrate 20–50 g/day, without protein or caloric restriction) and structured resistance exercise approximately three to four times weekly, individualised and predominantly home-based. Resistance training was chosen over aerobic exercise to preserve lean mass during weight loss and improve insulin sensitivity;[7] detailed session parameters were not prospectively recorded, a limitation. Structured glycaemic monitoring comprised daily SMBG (fasting and two-hour post-prandial) reported online, with weekly

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Table I: Baseline characteristics of the three patients prior to enrolment in the T2DM remission programme

Case	Patient Profile	Baseline Medications	Age (yr)	BMI (kg/m²)	HbA1c (%)	FBG (mmol/L)	BP (mmHg)	LDL-C[a] (mmol/L)	eGFR[a] (mL/min/1.73m²)
1	Malaysian Chinese male, aged 53; T2DM for 8 yr; no other comorbidities	Sitagliptin/metformin 50/1000 mg OD; gliclazide 60 mg OD	53	24.0	8.3	8.7	130/87	5.6	96
2	Malaysian Chinese male, aged 55; T2DM for 1 yr; hypertension	Empagliflozin 10 mg OD; metformin 850 mg BD; gliclazide MR 30 mg OD; perindopril 8 mg OD	55	24.5	7.2	6.5	128/70	4.8	94
3	Malaysian Indian male, aged 60; T2DM for 14 yr; no other comorbidities	Gliclazide 80 mg OD Gliclazide 80 mg OD	60	20.5	6.4	6.9	104/69	5.2	88

[a]DPP-4i: dipeptidyl peptidase-4 inhibitor; SU: sulfonylurea; SGLT2i: sodium-glucose cotransporter-2 inhibitor; FBG: fasting blood glucose; BP: blood pressure; BMI: body mass index; LDL-C: low-density lipoprotein cholesterol; eGFR: estimated glomerular filtration rate; OD: once daily; BD: twice daily; MR: modified release.

Table II: Clinical outcomes at Year 1 and Year 5 for all three patients

Case	Time Point	Age (yr)	BMI (kg/m²)	HbA1c (%)	FBG (mmol/L)	BP (mmHg)	Net Wt Loss (kg)	LDL-C[a]	Meds	Remission Status
1	Year 1 (2019)	53	22.6	4.5[1]	5.2	110/70	5.0	2.9	Nil	Remission
1	Year 5 (2025)	58	23.3	5.4	5.8	114/73	4.3	2.7	Nil	Sustained
2	Year 1 (2019)	55	22.2	6.0	5.2	135/82	7.0	3.1	Nil	Remission
2	Year 5 (2025)	60	22.0	6.1	5.8	130/84	7.6	2.8	Nil	Sustained
3	Year 1 (2019)	60	19.2	6.0	5.8	100/72	3.7	2.7	Nil	Remission
3	Year 5 (2025)	65	19.2	6.2	6.4	101/70	3.7	2.8	Nil	Re-remission[2]

[1]HbA1c 4.5% at programme end (6 months); no symptomatic hypoglycaemia documented; stabilised to 5.1% at the subsequent 3-month check and remained below 5.7% over 5 years. [2]Case 3 experienced a transient HbA1c of 6.5% in Year 3 (brief relapse); remission was re-achieved and maintained through Year 5. FBG: fasting blood glucose; BP: blood pressure; LDL-C: low-density lipoprotein cholesterol.

clinician consultations (in person or telemedicine) for medication titration. Sulfonylureas (and the SGLT2 inhibitor in Case 2) were tapered early given hypoglycaemia risk; patients reported any SMBG <4.0 mmol/L immediately, and no symptomatic hypoglycaemia occurred. HbA1c, FBG, renal profile (including eGFR) and fasting lipids (including LDL-C) were measured at baseline, three and six months, with annual HbA1c thereafter. Waist circumference, body fat percentage, apolipoprotein B, and sarcopenia or micronutrient screening were not performed (acknowledged as limitations). Written informed consent was obtained from all patients, whose identities are anonymised per the Declaration of Helsinki.

Case 1:
A 53-year-old Malaysian Chinese male with eight-year T2DM and no other illnesses was enrolled on sitagliptin/metformin 50/1000 mg and gliclazide 60 mg daily (Table I). Gliclazide was tapered first without hypoglycaemia, and he was medication-free by month four. At programme end his HbA1c was 4.5% — below the reference limit but without symptomatic hypoglycaemia (SMBG fasting 4.5–5.2 mmol/L) and, off all medication, most likely reflecting genuine normalisation rather than overtreatment. It rose to 5.1% three months later and has remained below 5.7% (5.4% in 2025). Net weight loss was 5.0 kg at year one and 4.3 kg at year five (BMI 23.3 kg/m^2). He remains medication-free with no complications.

Case 2:
A 55-year-old Malaysian Chinese male with one-year T2DM and hypertension was enrolled on empagliflozin 10 mg once daily, metformin 850 mg twice daily, gliclazide MR 30 mg once daily, and perindopril 8 mg once daily (Table I). Given additive hypoglycaemic risk, gliclazide and empagliflozin were tapered early and simultaneously; no hypoglycaemia occurred, and all oral hypoglycaemic agents were stopped by six months (HbA1c 5.7%). Perindopril was also discontinued, as home BP monitoring remained within an acceptable range off therapy; BP is kept under surveillance (130/84 mmHg at five years). HbA1c was 6.0% at three months post-programme (fulfilling remission criteria) and 6.1% at five years, with net weight loss of 7.6 kg (BMI 22.0 kg/m^2). He remains medication-free with no complications.

Case 3:
A 60-year-old Malaysian Indian male with 14-year T2DM was enrolled on gliclazide 80 mg once daily (Table I). Gliclazide was tapered from the first week without hypoglycaemia; by three months his HbA1c was 5.5% off medication, with a six-month value of 6.0% confirming remission. Net weight loss was 3.7 kg, maintained at five years (BMI 19.2 kg/m^2). A transient HbA1c of 6.5% in year three — the diagnostic threshold for diabetes — represented a brief relapse, attributed by the patient to higher carbohydrate intake and psychosocial stress; HbA1c returned below 6.5% with renewed dietary adherence and remained so through year five (meeting re-remission criteria). His five-year HbA1c was 6.2%. His maintained BMI of 19.2 kg/m^2, although within the normal range, lies at its lower end for an older adult; he was never clinically underweight, but formal sarcopenia, body-composition, and micronutrient monitoring were not performed and would be advisable in older patients undergoing such programmes. No complications were identified.

DISCUSSION

This series describes three non-obese Malaysian patients who achieved T2DM remission through a structured, medically supervised ketogenic-range LCD, with remission largely sustained over five years. As an uncontrolled observational series of selected patients, it cannot establish efficacy, but offers several hypothesis-generating observations for a phenotype under-represented in trials.

Mechanism and predictors. The prevailing framework — irrespective of starting BMI — is the Personal Fat Threshold hypothesis, whereby T2DM develops once liver and pancreatic fat exceed an individual's tolerance, impairing insulin sensitivity and beta-cell function even at normal BMI. ReTUNE showed that reducing ectopic fat in non-obese individuals produces metabolic changes identical to those in obese remitters.[4] Here, modest weight losses of 3.7–7.6 kg (6–10% of baseline) accompanied durable remission; carbohydrate restriction reduces hepatic glucose production and intrahepatic lipid independent of caloric intake.[8] Recognised predictors — shorter duration, lower baseline HbA1c, fewer medications[9] — are instructive: Case 2 (one-year duration) had the most favourable profile, Case 1 responded strikingly despite eight years of disease and high baseline HbA1c, and Case 3 (14 years) required the strictest adherence and relapsed transiently, consistent with remission rates falling from ~77% under one year to ~20% beyond 15 years.[10]

LCD versus VLCD, and the South East Asian phenotype.ReTUNE used an 800 kcal/day formula replacement — effective, but potentially less acceptable for non-obese patients with limited weight to lose; meta-analysis finds low and very low carbohydrate diets at least as effective as VLCDs for remission.[11] The LCD used here requires no formula, integrates with familiar South East Asian dietary patterns, and entails a gradual transition that may aid adherence. Malaysian patients of Chinese and Indian ethnicity show earlier-onset disease at lower BMI, greater visceral adiposity, and impaired insulin secretion;[5] even modest ectopic fat reduction may restore beta-cell function. With a national prevalence of 18.3%,[6] this supports evaluating structured remission programmes in primary care, with telemedicine and digital SMBG offering a scalable model.

Safety, and retention as the principal barrier. Hypoglycaemia during LCD-based tapering is the central safety concern, particularly with sulfonylurea and SGLT2i use; these were tapered early and no symptomatic hypoglycaemia occurred. The HbA1c of 4.5% in Case 1, without pharmacotherapy or symptomatic hypoglycaemia, most likely reflected physiological normalisation, though continuous glucose monitoring would be a valuable future adjunct. A notable observation arises from the cohort flow: all five non-obese patients who completed the programme achieved remission, spanning Chinese, Indian, and Malay ethnicity, suggesting

short-term remission is attainable across the major Malaysian ethnic groups. The constraint on confirming long-term durability was not biological non-response but loss of follow-up — two Malay remitters could not be traced at five years. This reframes the central question from "can non-obese patients achieve remission?" to "how can they be retained and monitored so that remission is sustained and documented?" Retention and recall infrastructure may be as important to real-world outcomes as the dietary intervention itself.

LIMITATIONS

This uncontrolled series cannot establish efficacy. Detailed five-year outcomes are confined to three patients; of six non-obese patients, one dropped out and two remitters were lost to contact, so confirmed long-term outcomes are limited to males of Chinese and Indian ethnicity and cannot be extrapolated to women, although short-term remission occurred in both sexes. Confirmed long-term outcomes for Malay patients — the highest-prevalence group — are absent owing to loss of follow-up and should be a research priority. Beta-cell reserve, waist circumference, body composition, apolipoprotein B, and sarcopenia and micronutrient screening were not measured, and the data derive from a single private clinic. Notwithstanding these limitations, the consistency of short-term remission across all three major Malaysian ethnic groups, with five-year durability in three patients, is a meaningful preliminary contribution.

CONCLUSION

This preliminary case series suggests that T2DM remission may be readily achievable in non-obese Malaysian patients through a medically supervised, ketogenic-range LCD with resistance training and structured glycaemic monitoring. Within the non-obese subgroup of an unselected cohort, all five completers achieved remission across Chinese, Indian, and Malay ethnicity, and five-year durability was confirmed in three. Notably, the principal barrier to confirming durability was loss of follow-up rather than loss of remission, suggesting retention infrastructure may be as decisive as the intervention itself. These hypothesis-generating findings, consistent with the Personal Fat Threshold hypothesis and ReTUNE, should be interpreted with caution given the uncontrolled design and small sample, and justify larger prospective controlled trials — with robust retention strategies, inclusive of women and all major Malaysian ethnic groups — to evaluate efficacy, safety, durability, and cost-effectiveness.

DECLARATION

Ethical approval and patient consent: Written informed consent for publication was obtained from all three patients. Patient identities have been fully anonymised.

CONFLICTS OF INTEREST

The author declares no conflicts of interest.

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