Brief Communication

Effect of a Low-Calorie Diet on Restoration of Normoglycemia in Obese subjects with Type 2 Diabetes

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Abstract

Introduction: Type 2 diabetes mellitus (T2DM) is considered to be an inevitably progressive disease. Complex therapies add to the financial and psychological burden. Very low-calorie diets (LCDs) are emerging as an option in the management of type 2 diabetes. **Methods:** We performed a clinical audit of patients with T2DM who received 12 weeks of LCD. **Results:** This case series documents that 6 out of 12 participants (median baseline HbA1c 9%) achieved HbA1c level in nondiabetes range with LCD despite stopping all antidiabetes medications. There was an improvement in serum triglycerides, HDL cholesterol, total cholesterol, C-Reactive protein, urine microalbumin, liver transaminases, liver fat and the indices of insulin resistance, beta cell secretory capacity, and insulin sensitivity. **Conclusion:** If long-term follow-up proves sustained benefits, such dietary restriction may be an alternative to more drastic options for reversal of type 2 diabetes. This may also help in changing the treatment perspective of a newly detected T2DM from an incurable and inevitably progressive disease to a potentially reversible disease.

Keywords: Nutrition, obesity, reversal, type 2 diabetes, very low-calorie diet

Introduction

Type 2 diabetes mellitus (T2DM) has long been considered as an inevitably progressive disease irrespective of the evolvement in pharmacological strategies.[1] Patients are expected to accept the diagnosis of a lifelong disease requiring sequentially increasing pharmacotherapy. Pharmacotherapy has not only added to the increasing cost of treatment but has also failed to permanently alter the pathophysiology of the disease. Contrary to this belief, recent clinical studies have shown that reversal of diabetes is possible with a calorie-restricted diet without using any pharmacological therapy.[2] It is also seen that there is a sustainable return to normalcy of beta cell function, hepatic glucose output, and visceral fat by acute restriction of dietary energy intake in individuals with T2DM.[3-6] Indeed, effect of acute food restriction has been compared with the glycemic improvement after bariatric surgery.[7]

We hereby present findings of a clinical audit on patients with T2DM who received 12-week low-calorie diet (LCD) as a part of their therapeutic plan. High rates of diabetes reversal in newly diagnosed Indian adults with T2DM are reported with intensive lifestyle therapy.[8] To our knowledge, there has been no experience of LCD (~1000 kcal/day) in type 2 diabetes from India.

Methods

A clinical audit was performed on the profile of 12 patients with T2DM who willingly enrolled for an LCD program for 12 weeks (The details of the program are described below). After a full discussion of the pros and cons of LCD, patients had been started on a specific dietary program consisting of 1000 kcal/day diet using meal replacement protein formula [Prototal Whey; containing 378.5 kcal, 48 g whey protein, 41 g of carbohydrate, and 2.5 g of fat along with micronutrients [Supplementary Table 1]. The total daily calories of 1000 kcal/

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day were achieved by giving three servings of 30 g formula with 150 ml of skimmed milk in addition to one regular meal and 2–3 small prespecified homemade snacks. The approximate nutritive values of sample dietary plan for a day are shown in Table 1. This meal plan constituted around 60% energy from carbohydrate, 30% energy from protein, and 10% energy from fat. The recipes of all homemade snacks with exact measurement of raw material were provided. Care was taken to provide at least 3 L of fluid/day.

All antidiabetes medications had been stopped in all but two participants who were both on basal bolus regimen of insulin in addition to three oral antidiabetes medications (metformin, sulfonylurea, and gliptin). These two patients continued to take metformin and basal insulin; meal-related insulin and other oral antidiabetes agents were stopped. Reduction in doses of metformin and basal insulin was done as necessary during LCD therapy. Statin therapy was continued in those who were taking it before the LCD therapy. Antihypertensive medications had been titrated as required during the due course. Home blood glucose monitoring with glucometer was done by testing fasting and 2 h post breakfast, lunch, and dinner on alternate days. Telephonic or E-mail follow-ups by nutritionists were done on a weekly basis to ensure compliance. Anthropometric parameters were collected monthly. Clinical and biochemical parameters were monitored monthly by a physician to detect any inadvertent occurrence of dyselectrolytemia, hyperuricemia, or gall stones. All participants were also advised moderate intensity aerobic and resistance exercise from the 2nd week after starting on LCD.

Results

We categorized participants as "Responders" (n = 6) and "Nonresponders" (n = 6) with respect to the improvement in HbA1c to nondiabetes range, i.e., an HbA1c below 6.5% without medications was classified as "responder". All participants (n = 12) had a significant fall in their HbA1c (median HbA1c dropped from 9% to 6.2%) after 12 weeks of LCD. Fifty percent (n = 6) of the participants had their HbA1c in nondiabetes range with all glucose control medication being stopped [Table 2]. In terms of median value, responders were younger with shorter duration of diabetes and needed less treatment for diabetes compared to nonresponders; two of the nonresponders were on basal bolus insulin at baseline. Responders also had marginally higher BMI and waist circumference but slightly lower HbA1c than nonresponders. There was a greater (30%) reduction in the median HbA1c of the responders compared to nonresponders (21%) with similar reduction in weight. Both responders as well as nonresponders demonstrated a fall in fasting and postmeal blood glucose levels. Nonresponders were also able to reduce the number of antidiabetes medicines including insulin doses. There was 50% reduction in insulin dose in one participant, whereas the other one was totally free of insulin at the end of 12 weeks. There was also a significant reduction in serum levels of liver transaminases in responders and improvement in liver fat measured by ultrasound (detailed result provided in supplement). Nonresponders had higher levels of insulin resistance (measured as HOMA-IR by HOMA2 model[9]), lesser beta cell secretory capacity (measured as HOMA-β by HOMA2 model[9]) as well as lesser insulin sensitivity (measured as QUICKI score[10]) at baseline [Figure 1]. Overall, there was an improvement in HOMA-IR, HOMA-β, and QUICKI score after 12 weeks of LCD in both responders as well as nonresponders. There was no unfavorable change in the nutritional parameters including hemogram, electrolytes, uric acid, serum proteins, and serum levels of Vitamin B12. There were no events of symptomatic hypoglycemia, and no participant experienced symptoms such as excessive hunger or lethargy showing that all participants tolerated the diet plan well. Detailed characteristics of individual participants are provided as Supplementary Tables 1 and 2.

Discussion

This case series documents that half of the participants with a significant baseline hyperglycemia (median HbA1c 9%) achieved HbA1c level in the nondiabetes range (<6.5%) at the end of 12 weeks of LCD therapy ($P < 0.001$) without need for antidiabetes medications. All except two patients in the nonresponder group showed improvement in the indices of insulin resistance, beta cell secretory capacity, and insulin sensitivity [Figure 1a-j]. The responders had a shorter duration of diabetes and needed lesser antidiabetes medications at the baseline suggesting that LCD works better if it is started earlier in the course of the disease. Although the diet was not carbohydrate restricted (energy from carbohydrate 60%), a balanced protein intake with the help of formula meal during LCD may be more effective in Indian participants

Table 1: Approximate nutritive values of sample dietary plan for a day during low-calorie diet therapy

Meal time	Food stuff	Calories (kcal)	Protein (g)	Carbohydrate (g)	Fat (g)
Early morning	200 ml skimmed milk	60	5	9.2	0.2
Breakfast	30 g formula + 150 ml skimmed milk	158	18.15	19.2	0.85
Mid-morning	1 apple/fruit equivalent	50	1	15	0
Lunch	30 g formula + 150 ml skimmed milk	158	18.15	19.2	0.85
Evening	200 ml mix vegetable soup + 150 g papaya/fruit equivalent	150	1	25	0
Dinner	Chapati-2 + vegeTable 150 g + salad-150 g + buttermilk-200 ml	275	6.23	36.82	7.71
Bed-time	30 g formula + 150 ml skimmed milk	158	18.15	19.2	0.85
Total		1059	67.68	143.62	10.46

Table 2: The comparison of all parameters studied in responders and nonresponders

	Responders ($n=6$)				*P* (before and after in responders)	Nonresponders ($n=6$)				*P* (before and after in nonresponders)
	Before (baseline)		After 12 Weeks LCD			Before (baseline)		After 12 Weeks LCD		
	Median	Minimum–maximum	Median	Minimum–maximum		Median	Minimum–maximum	Median	Minimum–maximum	
Age (years)	38.5	31-46	-	-	-	42.0	32-48	-	-	-
Males (*n*)	4	-	-	-	-	4	-	-	-	-
Duration of diabetes (years)	1.5	0.5-5.0	-	-	-	4.0	0.5-7.0	-	-	-
Family history of diabetes (*n*)	6	-	-	-	-	5	-	-	-	-
Number of OADs	1.5	0-3	0	0-0	0.066 (NS)	2.5	0-4	0.5	0-2	0.066 (NS)
Insulin dose	0	0-0	0	0-0	0.999 (NS)	0	0-31	0	0-16	0.157 (NS)
Weight (kg)	83.0	71-93	76.5	65.1-82.0	0.028*	84.5	69-100	78.5	70.1-93.0	0.046*
BMI (kg/m^2)	31.0	26.8-36.5	27.5	23.9-31.2	0.028*	29.9	27.4-34.6	28.5	25.2-32.2	0.046*
Waist circumference (cm)	105.5	97-110	96.5	92-105	0.023*	102.5	89-113	93.0	88-110	0.043*
HbA1c (%)	9.0	7.0-11.2	6.2	6-6.4	0.028*	9.1	7.5-10.7	7.1	7-8	0.043*
Fasting glucose (mg/dL)	164.5	106-244	111.5	102-119	0.046*	157.5	126-310	122.5	111-145	0.028*
Postprandial glucose (mg/dL)	266.0	117-399	110.0	91-164	0.028*	248.0	189-448	141.0	107-221	0.028*
Serum fasting insulin (mIU/ml)	16.9	9-27	9.5	6-18	0.043*	21.0	9-117	19.0	7-25	0.225 (NS)
Serum fasting C-peptide (ng/mL)	2.85	2.00-3.74	1.9	1.70-2.53	0.028*	2.81	1.63-5.00	2.38	1.20-3.50	0.066 (NS)
HOMA IR	2.51	1.68-3.26	1.49	1.30-1.93	0.028*	2.40	1.37-6.41	1.83	0.94-2.88	0.028*
Insulin sensitivity index (QUICKI)	0.285	0.27-0.34	0.33	0.3-0.36		0.275	0.24-0.3	0.29	0.29-0.34	
HOMA-beta	49.05	30.20-119.50	80.5	68.0-113.0	0.075 (NS)	37.85	20.90-99.80	75.85	51.7-92.3	0.249 (NS)
Serum triglycerides (mg/dL)	167.5	113-360	141.0	102-213	0.028*	134.0	70-403	113	59-204	0.075 (NS)
Serum LDL cholesterol (mg/dL)	151.5	106-194	152.0	127-199	0.600 (NS)	101.5	82-158	96.5	61-133	0.116 (NS)
Serum HDL cholesterol (mg/dL)	39.0	34-42	41.0	37-50	0.246 (NS)	39.0	35-50	41	35-58	0.686 (NS)
Serum total cholesterol (mg/dL)	186.0	171-281	192.0	174-249	0.345 (NS)	166.0	126-229	145.5	108-211	0.080 (NS)
Serum VLDL cholesterol (mg/dL)	28.7	18.1-57.7	23.7	16.4-100.0	0.345 (NS)	19.5	11.1-43.6	18.1	9.4-32.7	0.463 (NS)
C-reactive protein (mg/dL)	0.55	0.2-1.2	0.40	0.2-0.8	0.285 (NS)	0.40	0.1-0.6	0.20	0.1-0.5	0.102 (NS)
Microalbumin (mg/dL)	2.0	1.0-2.9	0.6	0.5-3.0	0.115 (NS)	0.8	0.5-1.4	0.6	0.5-1.9	0.854 (NS)
Serum alanine aminotransferase (IU/L)	34.0	16-76	18.5	15-30	0.046*	30.5	13-58	20.5	10-57	0.093 (NS)

The data are provided in median due to small sample size. *P* values by Wilcoxon's signed rank sum test, **P*<0.05, NS: Statistically nonsignificant, HOMA-IR: Homeostasis model assessment Insulin resistance, BMI: Body mass index, HbA1c: Glycosylated hemoglobin, QUICKI: Quantitative insulin sensitivity check index, LCD: Low calorie diet, LDL: Low-density lipoprotein, HDL: High density lipoprotein, VLDL: Very low-density lipoprotein, OADs: Oral antidiabetic drugs

who consume predominately high carbohydrate meals. The results are similar to "the Counterpoint study" (Counteracting Pancreatic Inhibition by Triglyceride) from the UK where dramatic effects on diabetes reversal were observed by giving very low-calorie diet (VLCD – 400–600 kcal/day) in the form of liquid meal formula with nonstarchy vegetables and salads for 8 weeks.[2] Our study suggests that such results may also be achieved with a more practical and acceptable LCD (1000 kcal/day). It is proposed that this state of acute energy crisis necessitates the utilization of the excess circulating glucose with corresponding improvement in insulin sensitivity as well as beta cell secretory capacity as measured by clamp studies.[2] It also results in the reduction of triglyceride stored in the liver as measured by magnetic resonance imaging.[3,11,12]

The follow up "Counterbalance study" (Counteracting BetA cell failure by Long-term Action to Normalize Calorie intakE)

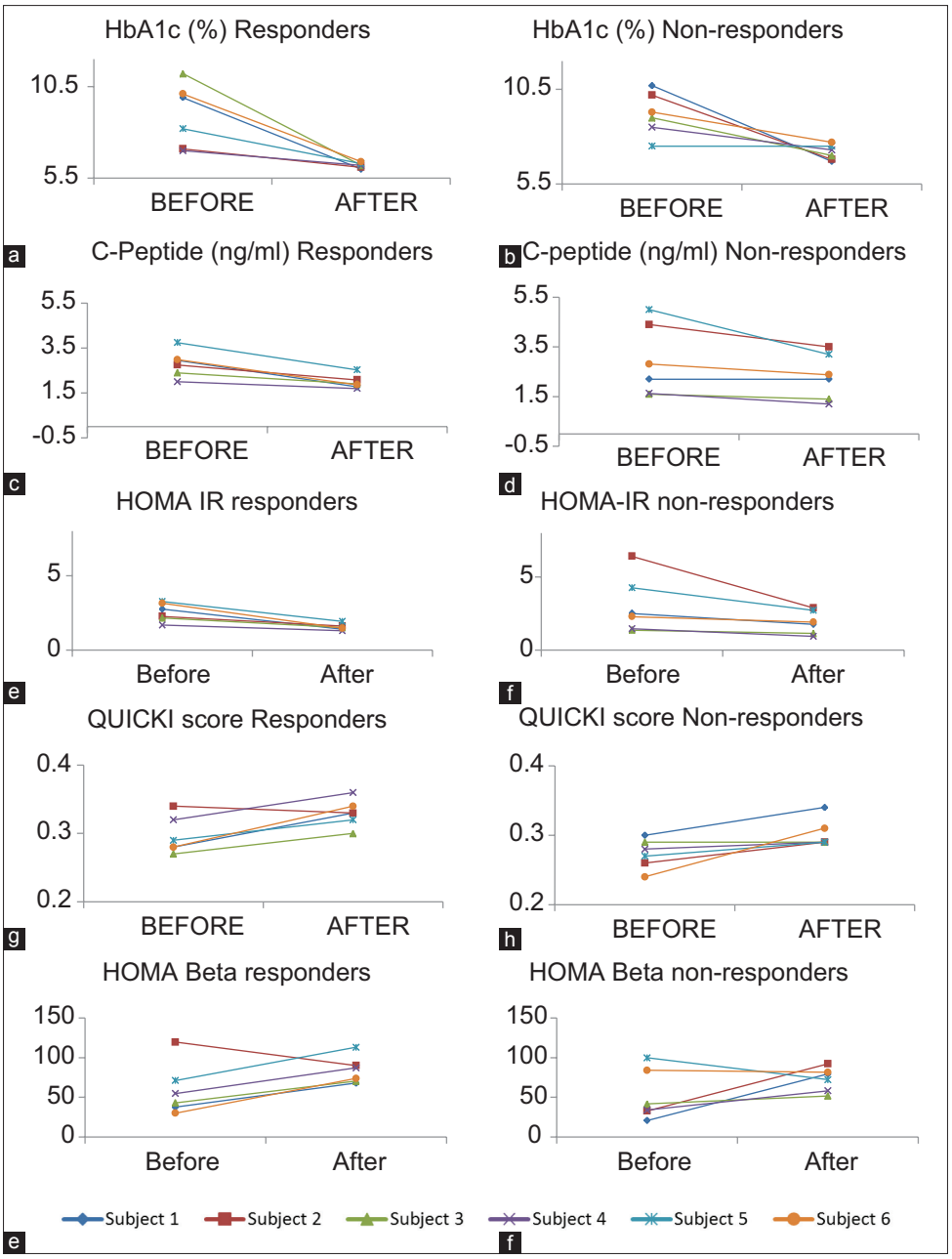

Figure 1: Changes in HbA1c and glycemic indices in responders and nonresponders

has shown that the result of VLCD is not only acute but also result in sustained (6 months after completion of 8-weeks therapy in this study) glycemic normalcy with pathophysiologic changes in metabolism of patients with diabetes even after reintroduction of isocaloric diet.[3] The reduction in hepatic fat was sustained with no redistribution of fat to the liver from the subcutaneous deposits even after 6 months of weight maintenance even though the participants did not attain weight normalcy. The second major change VLCD brings about is the reduction in pancreatic fat with recovery of first phase insulin secretion to nondiabetes levels.[3,13] It was also observed that the nonresponders were characterized by evidence of insulin deficiency at baseline and lack of ability to regenerate insulin secretion capacity. Our study could not observe this differentiation mainly because of a small sample size and HOMA2 indices done in our study being less accurate compared to clamp studies. Notably, all our participants were obese as per the Asia-specific BMI cutoff (>25 kg/m^2). In general, our study may reflect the benefit of LCD in obese type 2 diabetes. However, for obvious reasons, LCD may not benefit lean participants with recent weight loss and osmotic symptoms. Furthermore, participants with coexisting severe systemic illness would not be appropriate candidates for LCD therapy.

This study has many limitations. The most important is a small sample size, but the effect size of glycemic reduction is large, thus making the results significant. Second, advanced

metabolic studies such as clamp study were not performed, but other studies performing clamp studies have shown similar results.[2] A longer duration follow-up would be helpful in proving the sustainability of the results.

Conclusion

Once long-term follow-up studies prove sustained benefits, such dietary restriction may be an alternative to more drastic options such as medications, injections, or metabolic surgery for reversal of type 2 diabetes. These results have important implications in changing the perspective of a newly detected T2DM from an incurable and inevitably progressive disease to a potentially reversible disease, especially in those who are willing to do intense changes in their lifestyle.

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Conflicts of interest

There are no conflicts of interest.

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Supplementary Tables

Supplementary Table 1: Characteristics of individual patients in responder group

	Participant number					
	1	2	3	4	5	6
Age	31	42	34	42	46	35
Gender	Male	Female	Male	Male	Female	Male
Duration of diabetes (years)	0.5	2	1	5	0.5	5
Medication number of OADs						
Before	0	2	2	3	0	1
After	0	0	0	0	0	0
Insulin dose						
Before	0	0	0	0	0	0
After	0	0	0	0	0	0
Two generation family history of diabetes	Yes	Yes	Yes	Yes	Yes	Yes
Weight						
Before	71	92	93	81	79.9	85
After	65.1	80	82	73	70.4	80
BMI						
Before	26.8	36.5	32.2	30.5	31.6	29.5
After	23.9	31.2	28.4	27.1	27.9	27.2
Waist circumference						
Before	97	110	107	107	102	104
After	92	105	96	96	97	99
SBP						
Before	150	150	120	130	120	166
After	120	140	110	120	110	120
DBP						
Before	100	100	70	80	70	110
After	70	90	70	80	70	70
HbA1c						
Before	9.9	7.1	11.2	7	8.2	10.1
After	6	6.1	6.2	6.2	6.3	6.4
Blood sugar, fasting						
Before	205	106	172	139	157	244
After	118	108	119	102	103	115
Blood sugar, postprandial						
Before	399	155	327	205	117	367
After	94	111	164	109	91	164
Serum insulin fasting						
Before	18	9	27	10	17	16.8
After	10	9	18	6	14	8
Serum C-peptide fasting						
Before	2.95	2.75	2.4	2	3.74	2.98
After	1.77	2.08	1.9	1.7	2.53	1.86
HOMA-IR						
Before	2.75	2.27	2.16	1.68	3.26	3.15
After	1.41	1.62	1.52	1.3	1.93	1.47
Insulin sensitivity index (QUICKI)						
Before	0.28	0.34	0.27	0.32	0.29	0.28
After	0.33	0.33	0.3	0.36	0.32	0.34
HOMA-beta						
Before	37.7	119.5	43.2	54.9	71.3	30.2

Contd...

Supplementary Table 1: Contd...

	Participant number					
	1	2	3	4	5	6
After	68	90	70.4	87.1	113	73.9
Serum triglyceride						
Before	113	138	130	197	283	360
After	103	119	102	163	213	178
LDL cholesterol						
Before	150	106	109	153	164	194
After	143	175	136	127	161	199
Serum HDL cholesterol						
Before	36	34	39	40	39	42
After	41	45	41	37	50	39
Serum cholesterol						
Before	192	173	171	180	235	281
After	185	199	174	175	227	249
Serum VLDL cholesterol						
Before	18.1	22.1	26.06	31.4	45.3	57.7
After	16.6	19	16.4	100	34	28.4
C-reactive protein						
Before	0.2	1.2		0.3	0.8	
After	0.2	0.4	0.8	0.6	0.3	
Microalbumin						
Before	2.9	2.2	1.1	1	1.9	2.1
After	0.5	3	0.6	0.5	0.6	0.6
Hemoglobin						
Before	15.9	12.8	16.7	14.2	12.7	16.8
After	15.5	12.7	15.6	14.7	13	16.4
Hematocrit						
Before	46.7	39	46.76	42.7	38.6	49.1
After	44.5	39	46.1	43.9	38.6	48.1
Serum creatinine						
Before	0.9	0.7	0.6	0.8	0.9	0.8
After		0.8	0.8	0.7	0.9	0.8
eGFR						
Before	113	107	110	111	76	110
After		91	117	116	76	116
Serum uric acid						
Before	5.6	6.6	5.9	6.8	5.1	3.6
After	6.2	4.6	5.6	4.3	4.2	4.4
Serum calcium						
Before	10	9.4	9.02	8.5	9.8	9.9
After	10.2	9.8	9.7	9.3	9.4	
Serum aspartate aminotransferase						
Before	38	12	25	14	24	26
After	18	18	18	18	17	19
Serum alanine aminotransferase						
Before	76	16	52	21	29	39
After	19	18	21	17	15	30
Serum albumin						
Before	5.08	4.37	4.1	4.18	4.6	4.98
After	5.32	4.67	4.3	4.07	4.54	4.78
Serum globulin						
Before	2.2	2.2	2.82	2.7	2.7	2.5
After	2.3	2.9	2.5	2.7	3	2.3

Contd...

Supplementary Table 1: Contd...

	Participant number					
	1	2	3	4	5	6
Serum protein, total						
Before	7.34	6.61	6.92	6.89	7.3	7.51
After	7.55	7.56	6.8	6.8	7.46	7.1
Serum sodium						
Before	139	138	139	141	139	138
After	142	139	140	141	138	141
Serum potassium						
Before	5.1	3.9	4.8	4.5	4.6	4.5
After	4.7	5	5.6	4.3	4.8	5.2
Fatty liver on USG abdomen						
Before	Grade II–III	Grade II–III	Grade II–III	Grade II–III	Grade I	Not done
After	Normal	Grade I	Normal	Normal	Grade I	Not done

BMI: Body mass index, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, USG: Ultrasonography, eGFR: Epidermal growth factor receptor, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, VLDL: Very low-density lipoprotein, HOMA-IR: Homeostasis model assessment insulin resistance, BMI: Body mass index, HbA1c: Glycosylated hemoglobin, QUICKI: Quantitative insulin sensitivity check index, OADs: Oral antidiabetic drugs

Supplementary Table 2: Characteristics of individual patients in nonresponder group

	Participant number					
	1	2	3	4	5	6
Age	32	48	37	47	48	36
Gender	Male	Female	Male	Female	Male	Male
Duration of diabetes in years	1	0.5	5	7	3	6
Medication number of OADs						
Before	0	0	3	3	2	4
After	0	0	1	2	0	1
Insulin dose						
Before	0	0	0	31	0	15
After	0	0	0	16	0	0
Two generation family history of diabetes	Yes	No	Yes	Yes	Yes	Yes
Weight						
Before	94.5	69.2	84	77	100	85
After	86.9	70.1	79	73	93	78
BMI						
Before	31.2	28.4	29.8	30.1	34.6	27.4
After	28.7	28.6	28.1	28.5	32.2	25.2
Waist circumference						
Before	109	89	104	93	113	101
After	103	89	97	88	110	88
HbA1c						
Before	10.7	10.2	9	8.5	7.5	9.3
After	6.7	6.8	7	7.3	7.5	7.7
Blood sugar, fasting						
Before	263	310	148	167	147	126
After	118	131	125	111	145	120
Blood sugar, postprandial						
Before	394	448	254	238	189	242
After	166	129	149	107	133	221
Serum insulin fasting						
Before	9	19.4	20.4	21	39	117
After	7	19	22	25	20	13

Contd...

Supplementary Table 2: Contd...

	Participant number					
	1	2	3	4	5	6
Serum C-peptide fasting						
Before	2.2	4.4	1.6	1.63	5	2.81
After	2.2	3.5	1.4	1.2	3.2	2.38
Serum triglyceride						
Before	403	273	101	132	70	136
After	176	204	59	121	92	105
HOMA-IR						
Before	2.51	6.41	1.37	1.46	4.26	2.29
After	1.76	2.88	1.14	0.94	2.72	1.91
Insulin sensitivity index (QUICKI)						
Before	0.3	0.26	0.29	0.28	0.27	0.24
After	0.34	0.29	0.29	0.29	0.29	0.31
HOMA-beta						
Before	20.9	32.9	41.7	34	99.8	84.1
After	79.4	92.3	51.7	58.2	72.3	81.5
LDL cholesterol						
Before	103	138	82	99	158	100
After	105	61	74	104	133	89
Serum HDL cholesterol						
Before	40	35	36	38	50	44
After	36	35	39	43	43	58
Serum cholesterol						
Before	200	184	126	148	229	138
After	166	108	113	148	211	143
Serum VLDL cholesterol						
Before	18	43.6	16.2	21.1	11.1	21.8
After	28.1	32.7	9.4	19.4	14.8	16.8
C-reactive protein						
Before	0.6	0.4	0.5	0.6	0.1	0.1
After	0.5	0.2	0.1	0.4	0.1	0.1
Microalbumin						
Before	1.4	1.1	0.8	0.5	0.6	0.8
After	0.5	1.9	1	0.5	0.6	0.6
Hemoglobin						
Before	15.1	12.2	14.8	12	14.3	17
After	15.4	9.6	15.1	11.9	14.7	16.1
Serum creatinine						
Before	0.83	0.6	1	0.6	0.9	1
After	0.9	0.6	1	0.6	0.8	0.9
eGFR						
Before	110	108	84	110	101	95
After	106	108	96	110	106	108
Serum uric acid						
Before	4.8	3.3	6.7	4.1	6.5	6.2
After	6	3.9	7.2	5.4	7	6.8
Serum calcium						
Before	10.4	9.7	9.9	9	9.1	9.4
After	9.6	8.9	-	9.9	9.4	10
Serum aspartate aminotransferase						
Before	11	13	39	31	18	19
After	17	12	30	14	14	21
Serum alanine aminotransferase						

Contd...

Supplementary Table 2: Contd...

	Participant number					
	1	2	3	4	5	6
Before	17	13	58	38	26	35
After	20	10	57	19	21	29
Serum albumin						
Before	4.9	4.5	4.9	4.01	4.55	4.95
After	4.7	3.97	4.79	4.17	4.54	5.02
Serum globulin						
Before	2.8	2.9	2.4	2.8	2.4	2.3
After	3	2.4	2.1	2.5	2.2	2
Serum protein, total						
Before	7.6	7.3	7.26	6.78	7.04	7.34
After	7.7	6.37	6.94	6.72	6.7	7.02
Serum sodium						
Before	136	131	134	140	138	135
After	137	136	142	142	138	136
Serum potassium						
Before	5	4.2	4.7	4.1	4.6	4.1
After	4.6	4.3	4.7	4.1	5	4.2
Serum chloride						
Before	98	97	99	102	103	95
After	100	102	98	101	101	98
Fatty liver on USG abdomen						
Before	Normal	Grade I–II	Not done	Normal	Grade II–III	Grade II
After	Normal	Normal	Not done	Normal	Grade II	Normal

USG: Ultrasonography, eGFR: Epidermal growth factor receptor, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, VLDL: Very low-density lipoprotein, HOMA-IR: Homeostasis model assessment insulin resistance, BMI: Body mass index, HbA1c: Glycosylated hemoglobin, QUICKI: Quantitative insulin sensitivity check index, OADs: Oral antidiabetic drugs