Section: Biochemistry

Original Research Article

STUDY OF C-PEPTIDE AND GLYCOSYLATED HEMOGLOBIN IN OBESE INDIVIDUALS WITH AND WITHOUT TYPE 2 DIABETES MELLITUS IN RURAL TERTIARY CARE HOSPITAL IN KANCHEEPURAM DISTRICT

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ABSTRACT

Background: Obesity is associated with insulin resistance, dyslipidaemia and progression to type 2 diabetes mellitus. HbA1c reflects glycaemic exposure, while serum C-peptide indicates endogenous insulin secretion. The aim is to estimate serum C-peptide and HbA1c levels in obese individuals with and without type 2 diabetes and correlate them with fasting and post-prandial glucose and lipid profiles. **Materials and Methods:** This hospital-based observational study included 60 obese individuals aged 40–60 years (30 diabetics and 30 non-diabetics) from a rural tertiary care hospital. Fasting and post-prandial glucose, HbA1c, serum C-peptide, lipid profile and renal function were analysed using correlation and multiple linear regression analyses. **Result:** Diabetics were slightly older than non-diabetics (50.73 ± 6.75 vs 47.90 ± 6.01 years) with comparable BMI (33.03 ± 1.38 vs 32.81 ± 1.77 kg/m²). They had significantly higher FBS (162.23 ± 90.00 vs 86.77 ± 5.79 mg/dL), PPBS (242.27 ± 81.54 vs 131.63 ± 6.70 mg/dL) and HbA1c (9.50 ± 1.73% vs 5.27 ± 0.14%), along with higher total cholesterol, triglycerides, LDL and VLDL, and lower HDL. Serum C-peptide was slightly higher in diabetics (5.30 ± 3.02 vs 4.64 ± 1.92). BMI correlated positively with C-peptide in diabetics (r = 0.498) and non-diabetics (r = 0.600), while HbA1c correlated strongly with FBS and PPBS in diabetics. **Conclusion:** Obese diabetics exhibited higher glucose levels, HbA1c and a more atherogenic lipid profile than obese non-diabetics. HbA1c reliably assess glycaemic control, while serum C-peptide better indicates endogenous insulin secretion and β-cell function than insulin levels. HbA1c and C-peptide together distinguish metabolic differences between groups.

INTRODUCTION

Obesity, a major risk factor for type 2 diabetes mellitus (T2DM), is a metabolic condition characterised by increased adipose tissue and excessive fat accumulation resulting from an imbalance between energy intake and expenditure. Excess adiposity leads to complex metabolic disturbances that increase the risk of insulin resistance and T2DM.[1] In obese individuals, chronic overnutrition and an increase in adipose tissue lead to low-grade inflammation, lipotoxicity and hormonal imbalance, which impair insulin signalling. Constant insulin resistance increases the workload on pancreatic β-cells, which are initially compensated by increasing insulin secretion but will fail to fulfil demands later, resulting in progressive hyperglycaemia and overt diabetes. β-cell function has been assessed using serum insulin levels, but the insulin measurements have limitations due to peripheral insulin concentrations show post-hepatic clearance rather than true pancreatic secretion. C-peptide, released in equimolar amounts with endogenous insulin, is not affected by hepatic extraction and has a longer circulating half-life, making it a reliable indicator of endogenous insulin secretion and residual β-cell function than insulin itself.[2,3]

Glycosylated haemoglobin (HbA1c) is the gold-standard marker for assessing long-term glycaemic

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control, analysing the average blood glucose levels for the preceding two to three months. Elevated HbA1c levels are associated with the risk of both microvascular and macrovascular complications and are commonly used for the diagnosis and monitoring of diabetes.[4] In obese individuals, HbA1c levels often correlate with fasting and post-prandial glucose levels as well as lipid abnormalities. HbA1c levels help in understanding the broader metabolic dysfunction associated with insulin resistance and T2DM. Combining metabolic markers such as C-peptide and HbA1c might provide better evaluation of disease progression, phenotyping and risk stratification.[5]

Obesity-associated insulin resistance is closely linked to disturbances in lipid metabolism. Elevated triglycerides, reduced high-density lipoprotein cholesterol (HDL-C) and increased low-density lipoprotein cholesterol (LDL-C contribute to glucolipotoxicity, which further impairs β-cell function and worsens insulin resistance. Dyslipidaemia accelerates progression from obesity to T2DM and may also influence endogenous insulin secretion, affecting the circulating C-peptide levels.[6] Hence, evaluating lipid profiles alongside glycaemic indices and C-peptide offers a more comprehensive understanding of metabolic dysfunction in obese individuals.

The prevalence of obesity and T2DM is rising rapidly in India, including rural populations that were once considered low-risk populations. Lifestyle and dietary changes, reduced physical activity and delayed diagnosis contribute to poor metabolic control and increased complications.[7] However, data on C-peptide, HbA1c, and related metabolic markers from rural tertiary care hospitals are limited. Understanding these metabolic differences may help clinicians identify obese individuals at higher metabolic risk and guide early lifestyle or therapeutic interventions. Therefore, this study aims to estimate serum C-peptide and HbA1c levels and to evaluate their association with fasting and post-prandial blood glucose levels and lipid profile in obese individuals with and without type 2 diabetes mellitus attending a rural tertiary care hospital in Kancheepuram district.

MATERIALS AND METHODS

This hospital-based observational study was conducted in the Department of General Medicine at a rural tertiary care hospital in Kancheepuram district during the period from October 2018 to July 2020. The study was carried out after obtaining approval from the Institutional Ethics Committee, and written informed consent was obtained from all participants before to enrolment.

Inclusion Criteria

Obese individuals with a body mass index (BMI) of ≥25 kg/m² or more, belonging to either sex and aged between 40 and 60 years. The case group consisted of obese individuals diagnosed with T2DM. The control group included obese individuals with fasting blood glucose levels ≤126 mg/dL and post-prandial blood glucose levels ≤199 mg/dL, and who were not on oral hypoglycaemic drugs.

Exclusion Criteria

Patients with type 1 diabetes mellitus, acute infections, renal failure, polycystic ovarian syndrome, pancreatic disorders or pregnancy. Individuals with any other systemic illness that could affect glucose metabolism, as well as those unwilling to participate or not fulfilling the inclusion criteria.

Methods

60 eligible obese individuals were recruited from both outpatient and inpatient departments and divided into two groups comprising 30 obese individuals with T2DM (cases) and 30 obese individuals without T2DM (controls). The control group consisted of individuals attending routine health check-ups with no known medical illness that could influence the study outcomes. All patients were evaluated using a pre-designed proforma to record demographic details, including age and gender. Anthropometric measurements such as height and weight were obtained, and BMI was calculated, followed by a detailed clinical examination. After an overnight fast, venous blood samples were collected under aseptic conditions for estimation of fasting blood glucose, post-prandial blood glucose, glycosylated haemoglobin (HbA1c), serum C-peptide, lipid profile (total cholesterol, triglycerides, HDL, LDL and VLDL), and renal function tests (serum urea and creatinine). Serum C-peptide was measured using the chemiluminescence immunoassay method, fasting and post-prandial blood glucose were estimated by the glucose oxidase–peroxidase method, HbA1c by nephelometry, and serum urea and creatinine by urease and Jaffe's methods, respectively. The C-peptide insulin resistance index (CIR) was calculated using the formula CIR = 20 / (fasting C-peptide [ng/mL] × fasting glucose [mg/dL]), with lower CIR values indicating greater insulin resistance.

Sample size:

The sample size for the study was calculated using the formula:

$$n = \frac{4pq}{d^2},$$

Where p represents the estimated prevalence of the outcome of interest, q = (1 − p), and d denotes the allowable error. Based on this calculation, a minimum sample size of 60 participants was derived.

Statistical Analysis: The collected data were entered in Microsoft Excel and analysed using IBM SPSS version 25.0. Continuous variables were expressed as mean and standard deviation. The relationship between biochemical parameters was analysed using Karl Pearson's correlation coefficient. Multiple linear regression analysis was performed to evaluate the independent association of parameters. A p-value < 0.05 was considered statistically significant, and a

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95% confidence interval was applied wherever applicable.

RESULTS

Diabetic individuals were older than non-diabetics (50.73 ± 6.75 vs 47.90 ± 6.01 years), with comparable BMI (33.03 ± 1.38 vs 32.81 ± 1.77 kg/m²). FBS and PPBS were higher in diabetics (162.23 ± 90.00 and 242.27 ± 81.54 mg/dL vs. 86.77 ± 5.79 and 131.63 ± 6.70 mg/dL), along with higher HbA1c (9.50 ± 1.73% vs 5.27 ± 0.14%). Diabetics had higher total cholesterol, triglycerides, LDL and VLDL, with lower HDL (34.06 ± 7.21 vs 38.73 ± 13.08 mg/dL). C-peptide was higher (5.30 ± 3.02 vs 4.64 ± 1.92), while CIR was lower (0.04 ± 0.02 vs 0.06 ± 0.02). Haemoglobin, urea and creatinine were comparable between groups [Table 1].

Table 1: Baseline Demographic and Biochemical Characteristics

Parameter	Diabetic (Mean ± SD)	Non-Diabetic (Mean ± SD)
Age (years)	50.73 ± 6.75	47.90 ± 6.01
BMI (kg/m²)	33.03 ± 1.38	32.81 ± 1.77
FBS (mg/dL)	162.23 ± 90.00	86.77 ± 5.79
PPBS (mg/dL)	242.27 ± 81.54	131.63 ± 6.70
Total Cholesterol (mg/dL)	207.03 ± 35.67	196.90 ± 20.53
Triglycerides (mg/dL)	181.70 ± 58.89	135.43 ± 62.47
HDL (mg/dL)	34.06 ± 7.21	38.73 ± 13.08
LDL (mg/dL)	125.92 ± 31.74	121.78 ± 14.32
VLDL (mg/dL)	35.48 ± 12.17	27.08 ± 12.49
Haemoglobin (g/dL)	11.75 ± 1.34	11.70 ± 0.75
Urea (mg/dL)	24.80 ± 11.53	22.47 ± 8.90
Creatinine (mg/dL)	0.82 ± 0.31	0.81 ± 0.26
HbA1c (%)	9.50 ± 1.73	5.27 ± 0.14
C-Peptide	5.30 ± 3.02	4.64 ± 1.92
CIR	0.04 ± 0.02	0.06 ± 0.02

Sex distribution was similar in both groups (males 46.7%, females 53.3%). Most patients had a BMI of 25–29.9 kg/m² (93.3%), with 6.7% having a BMI ≥30. C-peptide levels were mostly 3–6 ng/mL (60% non-diabetics, 56.7% diabetics); 20% in both groups had levels 1.3–2.9 ng/mL [Table 2].

Table 2: Distribution of Sex, BMI Categories and C-Peptide Levels

Variable	Category	Non-Diabetic n (%)	Diabetic n (%)
Sex	Male	14 (46.7%)	14 (46.7%)
	Female	16 (53.3%)	16 (53.3%)
BMI Category (kg/m²)	25–29.9	28 (93.3%)	28 (93.3%)
	≥30	2 (6.7%)	2 (6.7%)
C-Peptide Level	1.3–2.9	6 (20%)	6 (20%)
	3–6	18 (60%)	17 (56.7%)
	6–12.7	6 (20%)	7 (23.3%)

In diabetics, HbA1c correlated strongly with fasting and post-prandial blood glucose (r = 0.665 and 0.832; $p < 0.01$), while these correlations were weak and non-significant in non-diabetics. HbA1c showed no significant association with lipid parameters in either group. C-peptide did not correlate significantly with fasting or post-prandial glucose but showed a significant positive correlation with BMI in both diabetics (r = 0.498, $p < 0.01$) and non-diabetics (r = 0.600, $p < 0.01$). CIR demonstrated significant negative correlations with fasting, post-prandial glucose and HbA1c in diabetics (r = −0.611, −0.637 and −0.642; $p < 0.01$), a negative correlation with BMI in non-diabetics (r = −0.593, $p < 0.01$), and strong inverse correlations with C-peptide in both groups (diabetics: r = −0.598; non-diabetics: r = −0.849; $p < 0.01$) [Table 3].

Table 3: Karl Pearson correlation of Biochemical parameters with c-peptides, HBA1C and CIR between groups

DIABETIC						
Parameters	**HbA1c**		**C-PEPTIDE**		**CIR**	
	R	**p-value**	**r**	**p-value**	**r**	**p-value**
FBS	0.665	0.000**	-0.05	0.794	-0.611	0.000**
PPBS	0.832	0.000**	0.141	0.459	-0.637	0.000**
Hb	-0.035	0.854	-0.169	0.372	0.01	0.959
Total Cholesterol	0.042	0.824	0.181	0.338	-0.216	0.252
TGL	0.244	0.195	-0.128	0.499	-0.122	0.522
HDL	0.099	0.603	-0.115	0.544	0.065	0.732
LDL	-0.023	0.906	0.173	0.361	-0.12	0.527
VLDL	0.246	0.19	-0.103	0.588	-0.212	0.262
UREA	0.098	0.06	-0.06	0.75	-0.103	0.588
CREATININE	-0.224	0.235	0.119	0.531	0.08	0.675
BMI	-0.246	0.189	0.498	0.005**	-0.121	0.525

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HbA1c	-	-	0.188	0.32	-0.642	0.000**
C-PEPTIDE	0.188	0.32	-	-	-0.598	0.000**
NON-DIABETIC						
Parameters	**HbA1C**		**C-PEPTIDE**		**CIR**	
	R	**p-value**	**r**	**p-value**	**r**	**p-value**
FBS	0.286	0.126	-0.006	0.977	-0.163	0.388
PPBS	-0.186	0.326	0.058	0.759	-0.04	0.834
Hb	-0.206	0.274	0.015	0.937	0.086	0.65
Total Cholesterol	0.039	0.839	0.168	0.374	-0.125	0.51
TGL	-0.068	0.723	0.35	0.058	-0.311	0.09
HDL	0.146	0.442	-0.095	0.617	0.194	0.305
LDL	-0.135	0.478	0.338	0.06	-0.263	0.161
VLDL	0.068	0.722	0.35	0.058	-0.311	0.095
UREA	0.112	0.557	-0.037	0.844	-0.043	0.822
CREATININE	0.186	0.326	0.416	0.022	-0.251	0.181
BMI	0.257	0.171	0.6	0.000**	-0.593	0.001**
HbA1c	-	-	0.105	0.582	-0.149	0.431
C-PEPTIDE	0.105	0.582	-	-	-0.849	0.000**

HbA1c showed a positive and significant correlation with FBS in the non-diabetic group. C-peptide showed a positive correlation with FBS, but this was not significant after adjusting for age. The multiple regression model including HbA1c, C-peptide, and age yielded an R^2 of 0.24, with $F(3,26) = 2.76$ and $p = 0.062$ [Figure 1 and 2).

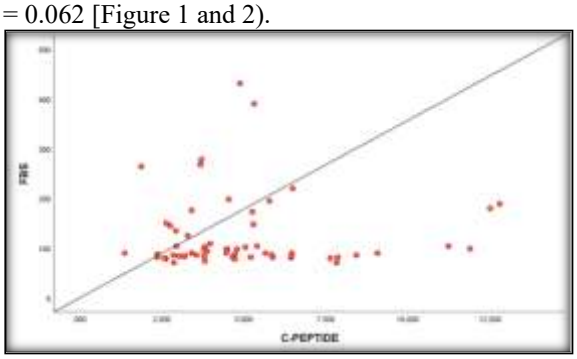

Figure 1: Scatter plot showing regression analysis of FBS with c-peptide.

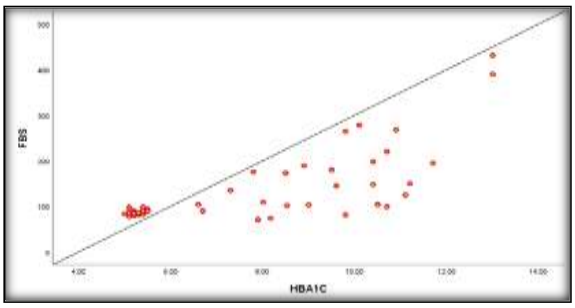

Figure 2: Scatter plot showing regression analysis of FBS with HbA1c.

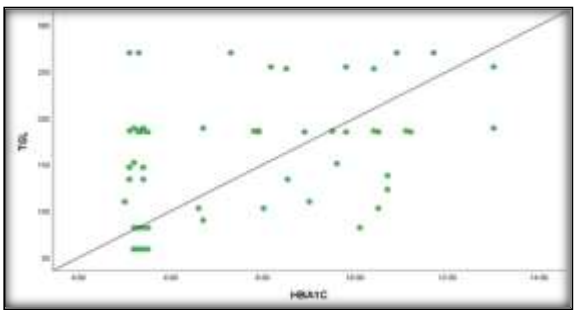

Figure 3: Scatter plot showing regression analysis of TGL with HbA1c.

C-peptide correlated positively with TGL. HbA1c was negatively correlated with TGL, but the association was not significant even after adjusting for age. The multiple regression model including HbA1c, C-peptide, and age produced an R^2 of 0.184, with $F(3,26) = 1.95$ and $p = 0.146$ [Figure 3 and 4].

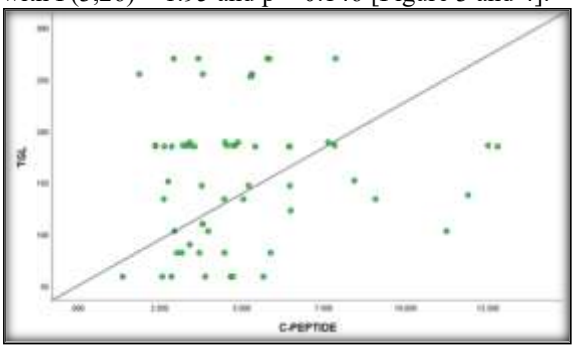

Figure 4: Scatter plot showing regression analysis of TGL with C-Peptide.

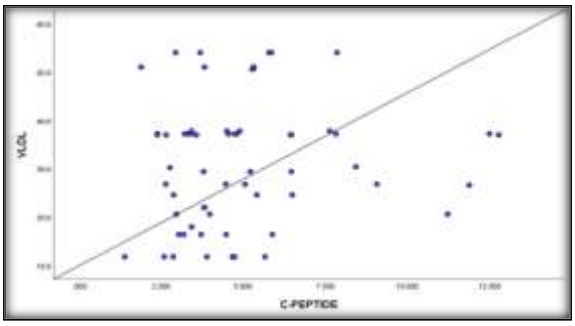

Figure 5: Scatter plot showing regression analysis of VLDL with C-Peptide.

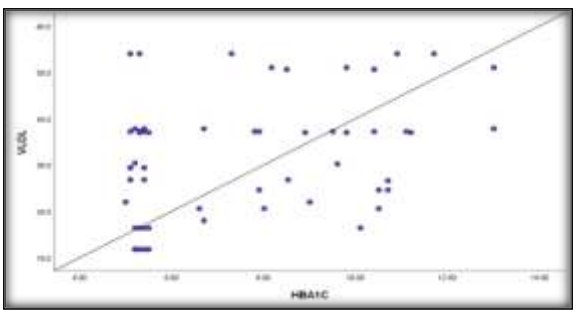

Figure 6: Scatter plot showing regression analysis of VLDL with HbA1c.

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C-peptide showed a positive correlation with VLDL. The multiple regression model including all three predictors produced an R^2 of 0.184 with F (3,26) = 1.95 and p = 0.146 [Figure 5 and 6].

DISCUSSION

Obesity is a major cause of insulin resistance and T2DM, leading to adverse glycaemic control, dyslipidaemia, and progressive β-cell dysfunction. This study evaluated serum C-peptide and HbA1c levels and their association with blood glucose parameters and lipid profile in obese individuals with and without T2DM attending a rural tertiary care hospital. Obese individuals with diabetes had poorer glycaemic control, more atherogenic lipid profiles, higher C-peptide levels with lower insulin resistance indices, and stronger correlations between HbA1c and glucose parameters.

In our study, obese individuals with type 2 diabetes had higher fasting and post-prandial blood glucose levels along with raised HbA1c. Lipid parameters were less favourable in the diabetic group, with higher total cholesterol, triglycerides, LDL and VLDL levels and lower HDL levels. Serum C-peptide levels were slightly higher in diabetics, while CIR values were lower. Mohan et al. reported that obese individuals with type 2 diabetes had significantly higher HbA1c values and a more atherogenic lipid profile, including increased triglycerides and LDL cholesterol and lower HDL cholesterol, when compared with obese non-diabetic subjects.8 Similarly, Deep HS et al. reported that patients with type 2 diabetes had significantly higher mean fasting blood glucose and HbA1c levels compared to non-diabetic individuals (mean FBS 213.21 mg/dL; mean HbA1c 10.17%), along with a high prevalence of dyslipidaemia present in over half of the study population. They also found mean serum C-peptide levels of 7.9 ng/mL and reported elevated endogenous insulin secretion in response to insulin resistance.[9] Similarly, Pathan et al. reported comparable findings among newly diagnosed type 2 diabetic subjects, wherein fasting plasma glucose (168.91 ± 53.72 mg/dL), HbA1c (9.12 ± 2.13%), total cholesterol and triglyceride levels were significantly higher, while HDL levels were significantly lower compared to non-diabetic controls. Serum C-peptide levels were elevated in diabetic patients (9.89 ± 4.28 ng/mL vs 1.24 ± 0.21 ng/mL in controls).[10] Thus, obese individuals with type 2 diabetes have poor glycaemic control and atherogenic dyslipidaemia even with preserved or elevated endogenous insulin secretion.

In our study, sex distribution and BMI categories were similar in both obese diabetic and non-diabetic groups. Most individuals in both groups had C-peptide levels between 3 and 6, with comparable lower and higher C-peptide levels. Similarly, Gadre et al. found that the majority of participants in both diabetic (59.25%) and non-diabetic (53.08%) groups had fasting C-peptide levels between 1 and 2.9 ng/mL. Higher C-peptide levels (3–6 ng/mL and >6 ng/mL) were observed in smaller proportions in both groups, and a significant positive association was noted between BMI and fasting C-peptide levels ($r = 0.403$, $p < 0.001$).[11] The similar distribution of C-peptide levels across obese diabetic and non-diabetic individuals indicates that endogenous insulin secretion is mostly preserved in obesity, irrespective of diabetic status. Thus, early lifestyle and weight-focused interventions should be aimed for improving insulin sensitivity, rather than targeting insulin secretion alone.

In our study, obese diabetic individuals showed higher mean values of total cholesterol, triglycerides, LDL and VLDL, along with lower HDL levels, when compared to obese non diabetic individuals. Similarly, Songa R M et al. reported that obese T2DM patients had significantly higher triglycerides (225.76 vs 139.9 mg/dL), LDL-C and VLDL-C levels, and significantly lower HDL-C compared to obese non-diabetic patients.[12] Similarly, Veeramalla et al. reported a significantly higher prevalence of hypertriglyceridemia (≈58% in males and 56% in females), elevated LDL and VLDL levels, and low HDL levels in diabetic patients compared to non-diabetic controls.[13] This suggests that dyslipidaemia in obesity and type 2 diabetes is mostly influenced by underlying insulin resistance rather than by the degree of chronic hyperglycaemia alone.

In our study, the diabetic individuals' HbA1c showed a strong positive correlation with fasting and post-prandial glucose, while these correlations were weak in non-diabetics. C-peptide showed a positive correlation with BMI in both obese diabetic and non-diabetic groups, but did not correlate with fasting or post-prandial glucose levels. CIR showed significant negative correlations with blood glucose in diabetics and with BMI in non-diabetics. Vittal et al. reported statistically significant positive correlations between HbA1c and fasting, post-prandial and random blood glucose levels in diabetic patients, with Pearson's correlation coefficients of $r = 0.6903$ for fasting blood glucose, $r = 0.6881$ for post-prandial blood glucose, and $r = 0.7005$ for random blood glucose. They reported that higher blood glucose levels correlate strongly with higher HbA1c values in type 2 diabetes.[14] Similarly, Anoop et al. observed that higher fasting C-peptide correlated significantly with surrogate measures of insulin resistance, such as HOMA-IR ($r = 0.42$, $p < 0.001$) and other insulin resistance indices in type 2 diabetic patients.[15] The strong inverse correlations observed between CIR and fasting glucose, post-prandial glucose and HbA1c in obese diabetic individuals suggest the worsening of glycaemic control even with elevated insulin secretion. Thus, CIR is a more sensitive marker of metabolic deterioration and insulin resistance than C-peptide alone.

These findings indicate that insulin resistance is the primary cause of poor glycaemic control and dyslipidaemia in obese type 2 diabetic individuals.

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The multiple regression analysis in our study shows that glycaemic status and dyslipidaemia in obese individuals are multifactorial and cannot be adequately predicted by a single biomarker. An integrated metabolic assessment, including glycaemic indices, lipid profile and insulin resistance markers such as CIR, may guide early intervention strategies targeting weight reduction and insulin sensitivity.

Limitations

The single-centre design may limit the generalisability of the results. Being a hospital-based observational study, causal relationships between C-peptide, HbA1c, insulin resistance, and dyslipidaemia cannot be established. The cross-sectional nature of the analysis also precludes assessment of temporal changes in metabolic parameters and progression from obesity to overt diabetes. Insulin resistance was assessed using the C-peptide insulin resistance index rather than gold-standard methods such as the hyperinsulinaemic–euglycaemic clamp, which may have influenced precision. Possible confounders such as dietary patterns, physical activity, duration of diabetes, and use of lipid-lowering medications were not fully assessed.

CONCLUSION

Obese individuals with type 2 diabetes in this rural tertiary care setting showed poorer glycaemic control, adverse lipid profiles and greater insulin resistance compared to obese non diabetic individuals. HbA1c reliably analyses glycaemic status, while serum C-peptide is a better index of endogenous insulin production and pancreatic beta cell function than insulin measurements. The combined assessment of HbA1c and C-peptide helps differentiate metabolic status in obese individuals and may aid early identification and better management of type 2 diabetes in rural populations. Interventional studies focusing on weight reduction and lifestyle modification are needed to evaluate their impact on insulin resistance, C-peptide dynamics, and glycaemic control, particularly in rural populations.

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