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Original Article

C-peptide Levels in Diagnosis of Diabetes Mellitus: A Case-control Study

Varun Shetty[1], H R Jain[2], G Singh[2], S Parekh[2], S Shetty[3]

[1]Associate Professor, Department of Medicine, Padmashree Dr. D. Y. Patil Medical College, Navi Mumbai, Maharashtra, India, [2]Post-graduate Student, Department of Medicine, Padmashree Dr. D. Y. Patil Medical College, Navi Mumbai, Maharashtra, India, [3]Professor, Department of Medicine, Padmashree Dr. D. Y. Patil Medical College, Navi Mumbai, Maharashtra, India

Abstract

Background: C-peptide has emerged as the most clinical and practically acceptable marker of β-cell function. Insulin and C-peptide are cosecreted into the portal circulation in equimolar concentration.

Materials and Methods: A total of 50 subjects of recently diagnosed diabetes mellitus (DM) within 1 month and 50 age and sex matched nondiabetic healthy controls were studied. These diabetics were further divided into three groups depending on the fasting serum C-peptide (FC) level, low FC group comprised subjects with FC level <0.54 ng/ml (*n* = 1). Intermediate FC group comprised subject with FC level >0.54-1.1 ng/ml (*n* = 2) high FC group comprised subjects with FC >1.11 ng/ml (*n* = 3).

Results: Predominant sufferers of diabetes were males 73% to 27% females. The cutoff range of C-peptide was taken as 0.8-3.58 ng/ml. In classifying, the sub types the low and intermediate group showing a low fasting and low postprandial C-peptide level indicate the benefit from early insulin therapy and the high FC group benefiting from oral hypoglycemic agents.

Conclusion: The classification of DM into its subtypes is the first step in approach to management. Data for various prospective studies have shown the relevance. The previously broader classification and the oversight of latent autoimmune diabetes is now becoming clearer and increasing in specificity from a treatment perspective. With a predominant Indian population suffering from a mixture of latent autoimmune diabetes in adults and Type II diabetes the measurements of fasting and postprandial C-peptide levels are now as important as diagnosing the illness itself.

Key words: β-cell, C-peptide, Diabetes mellitus, Insulin, Latent autoimmune diabetes in adults

INTRODUCTION

India is now ranked as a country with the most number of diabetics. This is being extrapolated to under 8 crore diabetics by the year 2030.[1] Diabetes is one among the most financially draining chronic noncommunicable disease. The costs for treatment rising and the burden it bears on family/individual finances and society makes it essential to approach this disease in a more collective entirety.[2]

An emerging pandemic with rising mortality and morbidity, diabetes mellitus (DM) is now being redefined with emphasis on a multipronged approach with equal stresses on all the factors. The prevention of Type II diabetes has been shown to be possible and requires action now. Trials have shown that sustained lifestyle changes in diet and physical activity can reduce the risk of developing Type II diabetes. For example, the finish diabetes prevention study showed that a better diet, increased physical activity and modest weight loss could substantially reduce the onset of Type II diabetes in middle-aged adults at high risk.

The scale of the problem requires population-wide measures.[3,4] Significant relevance has been placed on the etiology by the World Health Organization as does the American Diabetic Association (ADA)[5] emphasis is being laid to preempt and to manage diabetes through a combination of pharmacology, lifestyle diet, and exercise.

To effectively manage DM it is imperative to understand it. DM refers to a group of common metabolic disorders that share the phenotype of hyperglycemia. Several distinct

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Corresponding Author: Dr. Varun Shetty, Department of Medicine, Plot No. 247, 2nd Floor, Gokuldham, Sector 21, Nerul, Navi Mumbai, Maharashtra, India. Phone: +91-9833991811. E-mail: Shettyvarun81@gmail.com

types of DM exist and are caused by a complex interaction of genetics and environmental factors[6] and can also be characterized by an absolute or relative lack of insulin. Classified as Type I and Type II, respectively. Type IA diabetes (effects 18 years and younger) is a lack of insulin resulting from near total autoimmune destruction of the pancreatic β-cells and another subdivision Type IB also due to β-cells destruction. Its etiology is not ascertained yet.

Type II DM is a heterogeneous group of disorders characterized by variable degrees of insulin resistance, impaired insulin secretion, and increased glucose production. Distinct genetic and metabolic defects in insulin action and or secretion give rise to the common phenotype of hyperglycemia in Type II DM and have important potential therapeutic implications with newer pharmacologic agents that target specific metabolic derangements.[6]

The plethora of diabetes is its effect on a diabetic: The medical compilations it leads to second, the effect it has not only on the individuals, their care givers but also the socioeconomic repercussions that ensue. Compounding this growing problem is the rising incidence among the working population that is the young adult between 30 and 39 years.[3,4] Childhood obesity and an immoderate lifestyle is etiology for early onset and greater incidence.

Type I diabetes is a T-cell mediated autoimmune destruction of pancreatic β-cell begins in childhood, it can occur at any age. This manifest when near total pancreatic islet cell destruction occurs. Glutamic acid decarboxylase (GAD) autoantibodies and ICA512 and IA-2 autoantibodies[7] are now being used as a more specific cytoplasmic assay in the diagnosis of Type IA autoimmune mediated pancreatic β-cells destruction.

A third component of Type I diabetes is an antibody positive type that occurs in older individuals (older than 30 years of age). It has been found that approximately 40% of diabetes in India is of autoimmune variety that would benefit from insulin therapy. These thin built patients presenting in third or fourth decade having low C-peptide levels, positive GAD, or ICA antibodies, without an immediate need for insulin are labeled as type 1.5 diabetes or latent autoimmune diabetes in adults (LADA).

The expert committee on the diagnosis and classification of DM does not recognize the term LADA; rather, the expert committee includes LADA in the definition of Type I autoimmune diabetes ("Type I diabetes results from a cellular-mediated autoimmune destruction of the β-cells of the pancreas. In Type I diabetes, the rate of β-cell destruction is quite variable, being rapid in some individuals (mainly infants and children) and slow in others (mainly adults). The National Institutes of Health (NIDDK) defines LADA as "a condition in which Type I diabetes develops in adults." LADA is a genetically-linked, hereditary autoimmune disorder that results in the body mistaking the pancreas as foreign and responding by attacking and destroying the insulin-producing beta-islet cells of the pancreas. Simply stated, autoimmune disorders, including LADA, are an "allergy to self." Adults with LADA are frequently initially misdiagnosed as having Type II diabetes, based on age, not etiology.[8,9]

ADA recommends that antibodies positive patients to be grouped under Type I diabetes and antibodies negative patients to be grouped under Type II diabetes. The fasting and post lunch C-peptide levels are good indicators of insulin levels in the blood and pancreatic β-cell function. In contrast to this group, Type II diabetes have features resembling that of metabolic syndrome and increased plasma C-peptide levels with clustering of cardiovascular risk factors. Insulin and C-peptide are cosecreted into the portal circulation in equimolar concentration. However, due to hepatic extraction and peripheral metabolism of insulin, the peripheral levels of insulin do not necessarily reflect the level of secretion and it is this uncertainty that suggests peripheral C-peptide concentration may provide a more reliable picture of β-cell function than do peripheral insulin levels. A closer relationship existing between portal and peripheral C-peptide concentration, in association with clinical parameters it helps to accurately identify the type of diabetes. Patients having a low C-peptide response benefit by initiating early insulin therapy. This helps in preserving the β-cell function by decreasing the immune process of islet cell destruction. Insulin therapy may be discontinued confidently in patients having an adequate post mean C-peptide response.[10,11]

Hence with the above background and confusion regarding the type of diabetes in young patients (20-40 years of age) at presentation the relatively affordable C-peptide level is estimated to classify and effectively manage DM not only improving the outcome but more importantly reduce the complications caused by chronic hyperglycemia.[12,13]

MATERIALS AND METHODS

The present hospital based case-control study was conducted in the department of medicine. The study period was ½ year. 50 subjects of recently diagnosed DM within 1 month and 50 age and sex-matched nondiabetic healthy controls were studied. These diabetics were further divided into three groups depending on the fasting serum C-peptide (FC) level, low FC group comprised subjects

with FC level <0.54 ng/ml (n = 1). Intermediate FC group comprised subject with FC level >0.54-1.1 ng/ml (n = 2) high FC group comprised subjects with FC >1.11 ng/ml (n = 3). Various characteristics such as family history, age weight, height, body mass index (BMI), waist-hip ratio, blood pressure, fasting and post meal blood sugar and serum C-peptide, fasting and post meal, lipid profile, of each of these groups was studied. FC and post meal serum C-peptide was correlated with each of these characteristics. Ethical Committee approved the study.

Inclusion Criteria

- Freshly diagnosed case of DM, in the age group of 20-40 years, attending medicine outpatient department (OPD), diabetes OPD, and patients admitted to medicine ward were included in the study.
- Diagnosis of DM was made by ADA criteria for diagnosis of DM 2003.[3]
- Fasting plasma glucose 126 g/dl or 2 h post meal plasma glucose of 140 g/dl or symptoms of diabetes plus random blood glucose concentration >200 g/dl, or 2 h plasma glucose >200 mg/dl during oral glucose tolerance test.

Selection of Controls

Nondiabetic, age and sex match healthy control subjects selected randomly from:

- Patients attending OPD for minor ailments and seasonal infections or for physical checkup.
- Adult offspring's of diabetics visiting the admitted patient.
- Unrelated individuals into suffering from DM.
- Relatives of the diabetics above 18 years of age.

Exclusion Criteria

- Those subjects who had glucose toxicity are very high blood sugar level at presentation.
  And those presenting with diabetic ketoacidosis were initially excluded from the study.
  But later included once blood sugar become <200 g%-250 mg%.
- Subjects who did not give informed consent.
- Previously diagnosed diabetics even if noncompliant.

Past History

- Hypertension, coronary heart disease, cerebrovascular episode along with history of any major illness was noted.
- Smoking, tobacco chewing, and alcohol intake was noted, smoker was defined as a person who smokes at least l cigarette/pipe/cigar/bidi per day.
- Personal history regarding lifestyle noted.
- Family history of Type II DM in first second-degree relatives was noted.
- Menstrual history in females was noted.
- Treatment history in cases was taken in details.

Physical Examination Methods (167, 167, 169, 170)

Height

Standing height was measured with the subject in are foot, back square against the wall, and eyes looking straight ahead. A set square resting on the scale and the tape measurement from the wall was used to measure height to the nearest of 0.5 cm.

Weight

Weight was measured using a platform scale to the nearest of 200 gm. The scale was standardized to zero before each use.

BMI

BMI was calculated by the formula. Weight in kilogram divided by square of height in meter.

BMI = Weight in kilogram/(Height in meter^2).

BMI <25 was taken as normal

BMI >25 was taken as abnormal, i.e., increased.

Waist circumference

Waist circumference was measured to the nearest of 0.1 cm using a nonstretchable standard tape. Measurements were taken over the unclothed abdomen at the smallest diameter between the costal margin and the iliac crest. The tape measures were kept horizontal. Subject was made to relax with arms held loosely by sides. Two measurements were taken.

Hip circumference

It was measured to the nearest of 0.1 cm using a nonstretchable standard tape. Measurements were taken over light clothing at the level of greater trochanter (usually the widest diameter around the buttocks). This tape measure was kept horizontal. Subject was made to relax with arms held loosely by sides. Two measurements were recorded.

Waist-hip ratio (WHR)

WHR ≥0.90 for males and ≥0.85 for females was taken as a risk factor. Waist circumference:

- 102 cm in males and >88 cm in females was taken as a component of metabolic syndrome X according to ATP III Guidelines.

Blood Pressure

A mercury sphygmomanometer was used for measuring blood pressure. Systolic blood pressure was determined when the sounds appeared in the beginning when mercury column was lowered down (Korotkoffs phase l). Diastolic

blood pressured recorded at the level when the sounds just disappeared (Korotkoffs phase 5). The patient was rested at least for 5 min and was not allowed to smoke at least for 30 min before the measurement of blood pressure. Adequate cuff size was insured and cuff was made to encircle and cover 2/3rd of length of the arm with the bladder on anterior side of arm covering the brachial artery. Its lower border was kept one inch (about 2-3 cm) above the anticubital space. The bladder was deflated slowly. Two readings were taken at least 5 min apart and exact values were recorded.

Diagnostic of Hypertension

Hypertension was defined as presence of systolic blood pressure >140 mm if Hg and diastolic >90 mm of Hg 38 based on average of 2 readings taken on the two or more visits after an initial screening or one who was known case of hypertension with or without antihypertensive medication.

Blood pressure >130/>85 was taken as one of the components of metabolic syndrome X.

Fundus examination was performed in all cases and retinopathy.

Measurement of Blood Sugar

Folin-wu method was used for measurement of blood sugar (fasting and postprandial).

Measurement of Stream Total Cholesterol

CHOD-PAP method was used for measurement of total serum cholesterol.

Measurement of Serum High-density Lipoproteins (HDL) Cholesterol

Autozyme HDL cholesterol precipitation method 3 used for enzymatic determination of HDL cholesterol in serum.

Measurement of Serum Triglyceride

Serum lipid profile was estimated by calorimetric method 3 in the Biochemistry laboratory at Dr. D. Y. Patil Hospital.

Measurement of Serum C-peptide

The quantitative determination of circulating C-peptide concentration in human serum was carried out by C-peptide chemiluminescence which involves assay by microplate immunoenzymometric method. The C-peptide assay was carried out by Ranbaxy Laboratory, Bombay. Highly sophisticated and latest "Automated CLIA Analyzer" - ADVIA CENTAUR form Bayer, USA, was used for this assay.

C-peptide chemiluminescense immunoassay

Principle

The essential reagents required for an immunoenzymometric assay include high and specificity antibodies (enzyme conjugated and immobilized) with different and distinct epitope recognition, in excess, and native antigen. In this procedure, the immobilization takes place during the assay at the surface of a microplate well through the interaction sterptavidin coated on the well and exogenously added biotinylated monoclonal anti-insulin antibody. On mixing onoclonalbiotinylated antibody, the enzyme-labeled antibody and a serum containing the native antigen reaction occurs between the native antigen and the antibodies, without competition or steric hindrance, to form a soluble sandwich complex. The interaction is illustrated by the following equation.

enzAb(m)+Agc-pep+Ab(m)→

←

Simultaneously, the complex is deposited to the well through the high affinity reaction of streptavidin and biotinylated antibody. After equilibrium is attained, the antibody-bound fraction is separated from unbound antigen by decantation or aspiration. The enzyme activity in the antibody-bound fraction is directly proportional to the native antigen concentration. By utilizing several different serum references of known antigen values, a dose response curve can be generated from which the antigen concentration of an unknown can be ascertained.

Specimen Collection and Preparation

The blood should be collected in a plain red top venipuncture tube without additives. The blood is allowed to clot. Then the sample is centrifuged to separate serum from the cells. Samples may be refrigerated at 2-8°C for a maximum period of 2 days.

Sensitivity: The assay sensitivity is found to be up to 0.025 mg/ml.

Specificity: C-Peptide does not cross react with insulin, proinsulin, and glucagon.

OBSERVATION

The results are been depicted in tabular form in Tables 1-12.

DISCUSSION

The classification of DM into its subtypes is the first step in approach to management. Data for various prospective studies have shown the relevance. The previously broader classification and the oversight of latent autoimmune diabetes is now becoming clearer and increasing in specificity from a treatment perspective. With

Table 1: Age distribution in diabetic cases and control

Age group (years)	*n*=50 (%)		Total (*n*=100)
	Cases	Control	
20-24	4 (8.00)	6 (12.00)	10
25-29	5 (10.00)		12
30-34	8 (16.00)	10 (20.00)	18
35-40	33 (66.00)		60
Mean age±SD	34.72±5.55	32.08±6.01	

SD: Standard deviation

Table 2: Sex distribution cases and controls

Sex	*n*=50 (%)		Total (*n*=100)
	Case	Control	
Male	36 (72.00)	39 (78.00)	75
Female	14 (28.00)	11 (22.00)	25

Table 3: Presenting symptoms

Symptoms	Cases *n*=50 (%)
Paresthesia	1 (2.00)
Visual complaint	1 (2.00)
Nonspecific	4 (8.00)
Ketoacidosis	5 (10.00)
Skin infection	4 (8.00)
Urinary complaint	4 (8.00)
Weakness/fatigability	6 (12.00)
Weight loss	9 (18.00)
Polyphagia	8 (16.00)
Polydypsia	8 (16.00)
Polyuria	17 (34.00)

Table 4: BMI (kg/m^2)

BMI (kg/m^2)	*n*=50 (%)	
	Case	Control
≤	41 (82.00)	46 (92.00%)
25-29	9 (18.00)	4 (8.00%)
Mean±SD	21.18±4.07	20.78+4.38

SD: Standard deviation, BMI: Body mass index

Table 5: WHR in cases and controls

WHR	n=50 (%)	
	Cases	Control
Male		
≤0.90	18 (36.00)	20 (40.00)
>0.90	18 (36.00)	19 (38.00)
Female		
≤0.90	9 (18.00)	6 (12.00)
>0.90	5 (10.00)	5 (10.00)
Mean±SD	0.89±0.06	83±0.06

WHR: Waist-hip ratio, SD: Standard deviation

Table 6: Biochemical Parameters in cases and control subjects (*n*=50)

Blood sugar		
Fasting	147.70±18.44	87.04±14.05
Post meal	218.44±31.17	123.6±19.47
C-peptide		
Fasting	1.27±0.60	1.39±0.40
Post meal	2.96±2.24	5.04±1.24
Total cholesterol	161.76±34.01	154±14.35
LDL cholesterol	93.13±32.94	89.24±13.70
Serum triglyceride	131.66±38.13	111.44±16.62
HDL	41.89±7.9	43.5±6.7

LDL: Low-density lipoprotein, HDL: High-density lipoprotein

Table 7: Mean serum C-peptide in case and control

Mean serum C-peptide	(*n*=50)	
	Case	Control
C-peptide		
Fasting	1.27±0.60	1.39±0.4
Post meal	2.96±2.24	5.04±1.24

Table 8: Clinical profile of patients with metabolic syndrome versus nonmetabolic syndrome

Parameter	Metabolic syndrome *n*=18 (36.00%)	Nonmetabolic syndrome *n*=32 (64.00%)
Age	37.06±3.11	33.41±6.20
Systolic BP	131.69±15.69	123.06±9.58
Diastolic BP	84.83±9.38	83.41±8.14
BMI	23.29±3.40	20.0±3.98
Waist circumference	84.78±10.47	76.16±9.10
WHR	0.90±0.07	0.89±0.06
Blood sugar (F)	141.33±14.64	151.41±19.54
Blood sugar (PM)	25.22±26.11	225.88±32.51
C-peptide (F)	1.36±0.80	1.54±1.92
C-peptide (PM)	3.79±2.43	2.49±2.01
Triglyceride	145.4±35.95	123.91±37.64
LDL	101.4±35.41	88.98±31.07
HDL	37.61±4.88	44.22±+8.32
Cholesterol	166.0±34.05	159.38±34.29

LDL: Low-density lipoprotein, HDL: High-density lipoprotein, BP: Blood pressure, BMI: Body mass index

a predominant Indian population suffering from a mixture of LADA and Type II diabetes the measurements of fasting and postprandial C-peptide levels are now as important as diagnosing the illness itself.[14,15]

The investigation into incident discovery of impaired glucose tolerance will now be complete with the inclusion of C-peptide estimations in conjunction with HbA1C, fasting and post meal blood sugars and also the evaluation of urine sugars and micro albumins.[16-18]

With the confusion created by the BMI and the inclusion of metabolic syndrome with the Indian sufferer the C-peptide levels is the better tool in planning the line of long-term management.[19]

As DM is a precursor to various complications that raise the mortality and morbidity in any population by properly diagnosing the status of the β-cell function its degradation can be slowed thereby aiding in an effective glycemic control and most importantly preempting diabetes-induced retinopathy, nephropathy peripheral neuropathy and cardiovascular and cerebrovascular complications.[20]

Table 9: Clinical, biochemical, and serological characteristic of study group (low fasting serum C-peptide group (FC-0.54 ng/dl) nl (number of cases)

Variable	Low FC (<0.54 ng/ml)	Intermediate FC (0.54-1.11 ng/ml	High FC (>1.11 ng/ml)
Age	34.17±5.19	35.17±5.27	35.89±5.54
Systolic BP	20.68±3.73	19.19±3.47	22.22±4.38
Diastolic BP	3±9.38	83.41±8.14	35.17±5.27
BMI	23.29±3.40	20.0±3.98	35.17±5.27
Waist circumference	84.78±10.47	76.16±9.10	35.17±5.27
WHR	0.90±0.07	0.89±0.06	35.17±5.27
Blood sugar (F)	141.33±14.64	151.41±19.54	35.17±5.27
Blood sugar (PM)	25.22±26.11	225.88±32.51	35.17±5.27
C-peptide (F)	1.36±0.80	1.54±1.92	35.17±5.27
C-peptide (PM)	3.79±2.43	2.49±2.01	35.17±5.27
Triglyceride	145.4±35.95	123.91±37.64	35.17±5.27
LDL	101.4±35.41	88.98±31.07	35.17±5.27
HDL	37.61±4.88	44.22±8.32	35.17±5.27
Cholesterol	166.0±34.05	159.38±34.29	35.17±5.27

LDL: Low-density lipoprotein, HDL: High-density lipoprotein, BP: Blood pressure, BMI: Body mass index

Table 10: Metabolic syndrome in low FC Group (*n*=2)

Age	37.0±2.83
Systolic BP	125.00±7.07
Diastolic BP	75.00±7.07
BMI	22.74±4.48
Waist circumference	86.00±22.63
Blood sugar (F)	145.00±18.38
Triglyceride	165.00±28.99
Cholesterol	180.00±0.05
HDL	38.50±2.12
LDL	108.00±2.83

FC: Fasting serum C-peptide, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, BP: Blood pressure, BMI: Body mass index

Table 11: Metabolic syndrome in high FC Group (*n*=5)

Age	39.00±2.24
Systolic BP	122.00±20.20
Diastolic BP	77.2±11.37
BMI	23.79±3.47
Waist circumference	88.40±10.01
Blood sugar (F)	149.4±14.99
Blood sugar (PM)	214.00±31.50
Triglyceride	168.80±43.81
Cholesterol	171.2±28.54
HDL	38.00±5.48
LDL	107.6±39.52

LDL: Low-density lipoprotein, HDL: High-density lipoprotein, BP: Blood pressure, BMI: Body mass index

The BMI in all the cases was found to be either within the normal range or below the obesity mark, <23 kg/m^2. The CT abdomen showed that majority of the individuals suffering from LADA and Type II diabetes have a larger visceral fat deposit than peripheral fat deposit. Moreover, the second significant portion in the sample had large peripheral as well as visceral fat deposits. There a third percentage of individuals who have either peripheral fat or visceral fat deposit only.[21]

The difficulty of C-peptide estimations is that to evaluate and achieve an accurate result the individual should not have taken any form of antidiabetic medication either in the pill form or insulin in the pretending 3 months. While this is possible for new onset diabetics the trial and error method in misdiagnosed individuals can be abolished when the C-peptide estimation becomes part of the initial evaluation in suspected individuals who present with minor ailments hinting at DM.

There is definitely a practical advantage in including the C-peptide estimation in spite of its high cost. In doing so, the accurate line of management can be initiated keeping the costs of treatment to the minimum.[22]

CONCLUSION

From this study, the following conclusions were arrived at:

- Predominant sufferers of diabetes were males 73-27% females.
- The cutoff range of C-peptide was taken as 0.8-3.58 ng/ml
- The sample was divided into three groups low FC <54 ng/ml 18% intermediate FC 0.54-1.1 ng/ml 36% and high FC >1.1 ng/ml 48% and their features were compared.
- There was an increase in the low-density lipoprotein in all the 48% of high FC group along with triglycerides. Whereas, the other features such as blood pressure, serum cholesterol, age, and sex differentiation were almost similar ranging only marginally.
- 75% of the high FC group was diagnosed as metabolic syndrome there by suggesting Type II diabetes and LADA are inevitably going to develop metabolic syndrome and result in insulin resistance.
- Metabolic Syndrome is predominantly a feature associated of Type II DM and also latent autoimmune diabetes in the aged.
- In classifying the sub types the low and intermediate group showing a low fasting and low postprandial

Table 12: Treatment evaluation

Parameters	Sulfonylureas	Sulfonylureas+biguanides	Insulin
Age	37.71±2.36	37.61±13.5317	31.88±11.24
Systolic BP	123.14±6.62	126.56±14.52	126.58±13.11
Diastolic BP	84.29±7.87	81.67±8.52	85.04±8.83
Weight	52.00±10.32	61.44±11.62	49.38±9.48
BMI	21.11±3.84	23.33±3.41	19.52±4.04
Waist circumference	79.43±8.50	84.50±9.60	75.42±10.3
WHR	0.86±0.05	0.90±0.07	0.89±0.06
Blood sugar (F)	147.71±16.76	148.89±20.70	147.63±17.9
Blood sugar (PM)	218.00±38.22	212.00±32.15	223.51±30.50
C-peptide (F)	1.19±0.35	1.62±1.00	1.45±2.15
C-peptide (PM)	3.36±1.46	4.58±2.18	1.71±1.64
Triglyceride	114.71±38.11	144.33±34.19	126.85±40.16
Cholesterol	166.43±27.10	160.72±37.81	159.50±33.70
HDL	43.20±10.59	39.61±5.69	42.75±8.45
LDL	107.00±11.67	86.46±38.94	92.68±31.92

LDL: Low-density lipoprotein, HDL: High-density lipoprotein, BMI: Body mass index

C-peptide level indicate the benefit from early insulin therapy and the high FC group benefiting from oral hypoglycemic agents.

- By identifying the subtypes aggressive control by emptying the most appropriate treatment regimen for impaired plasma glucose levels but will also assist significantly in preventing complications such as retinopathy, nephropathy, neuropathy, and atherosclerotic changes in the macro and micro vasculature.

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