Derna University Journal of Medical Sciences (DUJMS)

UNIVERSITY OF DERNA

Volume 1 | Issue 1 | 2023 | Pages 121-130
ISSN: 2789-7389 (Online) | **DOI:** https://doi.org/10.58987/74p17n68

ORIGINAL RESEARCH

Serum C-Peptide Levels in Diabetes Mellitus Type II

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Received: 20 April 2023 **Accepted:** 23 May 2023 **Published:** 20 June 2023

ABSTRACT:

The role of serum C-peptide in estimating the exact amount of insulin secreted endogenously is getting substantial amount of recognition, just because C-peptide has no biologic activity on homologous or heterologous tissue and no ability to modify the action of insulin and/or proinsulin. The present study is to evaluate the serum C-peptide levels in type-2 diabetic patients and to compare their levels with those of matched healthy controls. Approximately, 56 cases of type- II diabetes mellitus (35 males, 21 females) were subjected to analysis of serum C-peptide, blood sugar (fasting, post meal), glycosylated hemoglobin, and lipid profile. The results were compared with age and sex matched healthy controls. It was seen that serum C-peptide level is significantly ($p<0.001$) decreased in long-standing type-2 diabetic patients as compared to sex and age matched apparently healthy controls. There is decrease in concentration of fasting serum C-peptide level both in male and female type-2 diabetic patients as compared to matched controls. As compared to matched control, the decrease is highly significant ($p<0.001$ and $p<0.05$). There was no significant difference in the concentration of serum C-peptide between males and females. The data suggests that insulin resistance is an avoidable consequence of abdominal fat accumulation with advancing

age. This work strongly confirms the earlier views of C-peptide being a valuable parameter in assessing progress of disease.

KEYWORDS: Serum C-Peptide , Insulin , Glycosylated Hemoglobin , Proinsulin , Type-II Diabetes Mellitus .

INTRODUCTION

Diabetes mellitus is a syndrome described as chronic hyperglycemia with disorder in carbohydrate, fat and protein metabolism. The disease comes from either an absolute or relative deficiency of insulin secretion and/or action. [(1)] Diabetes considers as an "iceberg" and a common disease in the world next to cardiovascular and ontological disorders. According to recent estimates, the prevalence of diabetes mellitus in adults was around 4% worldwide. This means that over 143 million are now affected. Also, it is projected that the disease occurrence will be 5.4% by the year 2025, with global diabetic population reaching 300 million. Of this, close to 77% of the global burden of disease is expected to occur in the developing countries. [(2)] The World Health Organization (WHO) has projected the rising trend of diabetes mellitus. The prevalence in India is expected to go up from 19.4 million in year 1995 to 57.2 million in years 2025.

The India populace has a raised susceptibility to diabetes mellitus. Because of the prevalence of disease in adults was 2.4% in rural and 4.0-11.6% in urban dwellers. [(3)] At least, there are two major identifiable pathological defects in patients with type-2 diabetes. [(4, 5, 6)] One occurs by decreased ability of insulin to act on the peripheral tissues. This is called insulin resistance and is thought to be the primary pathological process. Another is B-cells dysfunction, which is inability of the pancreas to produce sufficient insulin to compensate for the insulin resistance. Thus, there is a relative deficiency of insulin in the early stage of the disease and absolute insulin deficiency in the late stage of the disease. The debate over whether type-2 diabetes is primarily due to a defect in B-cell secretion, peripheral resistance to insulin or both has been raging for decades. However, there are data to support the concept that the insulin resistance is the primary defect, preceding the derangement in insulin secretion and clinical diabetes by as much as 20 years. [(5,6)]

Preproinsulin, a protein containing 100 amino acids (MW 12000), is formed by the ribosomes in the rough endoplasmic reticulum of the pancreatic B-cells. Preproinsulin is not detectable in the circulation under normal conditions because it is rapidly converted by cleaving enzymes to proinsulin (MW 9000), an 86-amino-acid polypeptide. [(7)] Proinsulin is cleaved to a 31-amino-acid connecting (C) peptide (MW 3600) and insulin. It connects the A and B chains of insulin in proinsulin molecule. Insulin is a heterodimeric polypeptide (MW 6000) consisting of two chains, A (30 A.A) and B (21 A.A) linked by two interchain disulfide bridges that connect A7 to 87 and A20 to B19. A third intrachain disulfide bridge connects residues 6 and 11 of the A chain. [(7)] Although insulin and C-peptide are secreted into the portal circulation in equimolar amounts, fasting levels of C-peptide are 5 to 10 fold higher than those of insulin owing to the longer half-life of C-peptide (about 35 min). The C-peptide has unknown biological activity with any homologous or heterologous tissue and no ability to modify the action of insulin and/or proinsulin. It is a distinct molecule from an antigenic standpoint.

The liver removes about 50% of insulin but very minor fraction of C-peptide is metabolized in liver. A review of studies concerned with the metabolism of human C-peptide showed that the kidney is a major organ in C-peptide removal. A fraction of unchanged C-peptide is excreted in the urine. [8, 9, 10, 11] Thus, C-peptide immunoassays can distinguish insulin secreted endogenously from insulin administered exogenously and can quantitate the former when anti-insulin antibodies preclude the direct measurement of insulin. The kidney is the major organ in C-peptide removal. A fraction of unchanged C-peptide is excreted in the urine. [8, 9, 10, 11] Thus, C-peptide immunoassays can distinguish insulin secreted endogenously from insulin administered exogenously and can quantitate the former when anti-insulin antibodies preclude the direct measurement of insulin. This work is aimed to assess the serum C-peptide levels in long standing type-2 diabetes mellitus patients. And, it is to assess the co-relation between serum C-peptide and pancreatic B-cell insufficiency.

MATERIALS AND METHODS

The present study was conducted on 56 patients of type-2 diabetes mellitus, taking treatment in diabetic clinic of the medicine department, India Gandhi Medical College and mayo hospital, Nagpur. Fasting and post meal blood sugar levels and glycosylated hemoglobin confirmed in all cases of type-2 diabetes mellitus. Out of 56 patients, 35 were males and 21 were females and their mean age was 57.93 years. Apparently, normal controls were taken out of which 22 were males and 10 were females having a mean age of 58.91 years. Following, biochemical parameters were evaluated in the serum sample of type-2 diabetic patients and controls in department of Biochemistry and Microbiology, India Gandhi Medical College and Mayo Hospital, Nagpur.

- Serum C-peptide levels.
- Glycosylated hemoglobin.
- Serum triglyceride level.
- Serum LDL cholesterol level*.
- Blood urea level.
- Blood glucose levels.
- Serum cholesterol levels.
- Serum HDL cholesterol level.
- Serum VLDL cholesterol level*.
- Serum Creatinine level.

*Serum LDL-C and VLDL-C is calculated from the Friedewald formula. [12]

Statistical Analysis

All the results were expressed as Mean ± SD. Student t test was used to assess statistical significance of the results between control and diabetic group. And the p value <0.001 was considered as highly significant, p value <0.01 was considered as significant and p value <0.1 was considered as insignificant.

RESULTS

Table 1. Kinetic Parameters of Insulin C-Peptide and Proinsulin in Man^17

Authors	Insulin	C-peptide	Proinsulin
Horwitz et.al. (31)	-	T½ = 11.1 min	-
Kuzuya & Matsuda (32)	T½= 9.8 ± 1.3 Min	T½ = 20.1 ± 1.6 min	-
Faber et al (33)	-	MCR = 4.4 ± 0.2 ml/ min/ Kg	-
Starr & Rubenstein (34)	T½ = 4.8 min	-	T½ = 17.2 min
Sonksen et al (35)	MCR=11.34 ml/ min/ Kg	-	MCR= 2.1 – 3.7 ml/ min/ Kg

T½ = half-life. MCR= Mean clearance rate.

Table 2: Distribution of Cases according to Duration of Diabetes.

Duration in (years)	Number of Diabetics	Percentage (%)
6-8	27	48.21
9-11	20	35.71
12-15	09	16.07

The table 2 shows 27 patients (48.21%) having 6-8 years of duration of diabetes, 20 patients (35.71%) having 9-11 years of duration of diabetes and 09 patients (16.07%) having 13-15 years of duration of diabetes.

Table 3: Biochemical Parameters in Diabetic Patients and Controls.

Parameters	Diabetic patients (n=56) Mean ± SD	Controls (n = 32) Mean ± SD
Fasting Plasma glucose (mg/dl)	140.43 ± 6.60	90.53 ± 7.78
Post meal plasma glucose (mg/dl)	170.30 ± 12.12	109.38 ± 16.83
Glycosylated Hemoglobin (%)	7.9 ± 0.30	7.2 ± 0.50
Blood urea (mg/dl)	28 ± 4.65	25.91 ± 5.21
Serum creatinine (mg/dl)	0.64 ± 0.13	0.65 ± 0.15

The table shows the mean values of the various biochemical parameters estimated of diabetic patients and controls. Also, Table 4 shows the mean of serum lipid levels in diabetic patients and controls.

Table 4: Serum Lipid Levels in Diabetic Patients and their Matched Controls.

Serum Lipids	Diabetic patients (n=56) Mean ± SD (mg/dl)	Controls (n = 32) Mean ± SD (mg/dl)
Total Cholesterol	230.3 ± 17.98	197.38 ± 20.86
Triglycerides	159.55 ± 48.34	110.03 ± 19.41
HDL Cholesterol	35.57 ± 8.94	42.84 ± 3.04
VLDL Cholesterol	31.77 ± 9.60	22.01 ± 3.88
LDL Cholesterol	162.68 ± 16.54	132.53 ± 18.66

Table 5: Fasting C-Peptide and Plasma Glucose Levels in Diabetic Patients and their Matched Controls.

Parameters	Diabetic patients (n=56) Mean ± SD	Controls (n = 32) Mean ± SD
Fasting plasma glucose (mg/dl)	140.43 ± 6.6	90.53 ± 7.78
Fasting C-peptide levels (pmol/ml)	0.76 ± 0.29	1.04 ± 0.36

Table 5 shows the mean of fasting plasma glucose and fasting C-peptide levels in diabetic patients and controls.

DISCUSSION

Type-2 diabetes mellitus is a disease usually seen after the 4th decade of life. Insulin resistance is an avoidable consequence of abdominal fat accumulation with advancing age. [13,14] In long standing cases of type-2

diabetes mellitus the worsening metabolic control due to secondary failure leads to various complications like retinopathy, nephropathy and neuropathy. Secondary failure, of worsening metabolic control despite sulphonylurea treatment, is a recognized event in type-2 diabetes. [15 , 16] Most studies have comprised patients referred to specialist clinics because of symptoms, unacceptable metabolic control or because of other diabetes related problems. [17] In the past, most attention has been focused on strict control of plasma glucose levels in order to prevent the long-term complications. Glycosylated hemoglobin, was used as an index of long –term glycemic control and derangement in the values indicated a tendency towards development of the inevitable complications. [18, 19, 20] Various international and Indian studies have shown an alternation in the plasma glucose levels both fasting as well as post meal in patients having long-term complications of type-2 diabetes mellitus. [15, 16, 18, 19] Various new markers that might better assess the deterioration of pancreatic B-cells function have been recently identified. These include serum insulin and serum C-peptide levels. [21]

In the present study, the availability of the immunoassay for measurement of serum C-peptide has been of great value in improving our understanding of the natural history and clinical characteristics of diabetes mellitus. However, the C-peptide assay has also been of utility in several conditions other than diabetes and indeed from a clinical standpoint has been most useful in these situations. [21, 22, 23] Despite the large body of literature pertaining to C-peptide that has been accumulated over the past three decades, the cross-reactivity between human, beef and pork insulin has been appreciated as well as the fact that the insulin immunoassay cannot be used to distinguish endogenous from exogenous insulin. The C-peptide assay however is species-specific. [22] In type-2 diabetes patients, the small amount of bovine or porcine proinsulin-like components that maybe present in exogenous insulin are not detected in the human C-peptide assay. Despite differences in their rate and mechanisms of catabolism, serum C-peptide levels correlate well with those of insulin in both the basal and stimulated state and in both the portal and peripheral circulation. For all these reasons C-peptide measurements are an excellent reflection of endogenous insulin secretion. [24]
In our study, we have studied 56 patients of type-2 diabetes mellitus (35 males, 21 females) and 32 matched healthy controls (22 males, 10 females). The diabetic patients were selected from diabetic clinic of Department of Medicine, India Gandhi Medical College and Hospital, Nagpur. The patients were selected strictly on the basis of specific criteria. Among fifty-six type-2 diabetic patients, it was seen that serum C-peptide level is significantly ($p<0.001$) decreased in long-standing type-2 diabetic patients as compared to sex and age matched apparently healthy controls.

Our findings are supported by earlier workers, namely Clauson P et al (25) (1994). Clauson P et al (25) (1994) showed that fasting levels of C-peptide decreased moderately but significantly with duration of diabetes ($p<0.05$). The fall in C-peptide occurred later than duration associated metabolic deterioration. Hence a 23% decrease (from 0.87 ± 0.05 to 0.69 ± 0.08 nmol/L) in C-peptide was observed between 0-5 years and 10-15 years of duration. No significant difference was observed in the values for male diabetic patients compared to female diabetic patients.

Table (1,2,3,4) show that there is decrease in concentration of fasting serum C-peptide level both in male and female type-2 diabetic patients as compared to matched controls. As compared to matched control, the decrease is highly significant ($p<0.001$ and $p<0.05$). There was no significant difference in the

concentration of serum C-peptide between males and females in our study.
Bergstrom R W et al (26) (1990) showed that elevated fasting C-peptide levels represents hyper-secretion of insulin and was interpreted to reflect a compensatory response to an underlying insulin-resistant state that precedes the development of NIDDM. Also, Faber O K et al (27) (1977) suggested that when the fasting C-peptide concentration alone is taken into account, the concomitant fasting blood glucose concentration should be considered, because the individual fasting C-peptide concentration varied with that of blood glucose.

Our study clearly documents that deterioration of metabolic control is associated with increasing duration of type-2 diabetes and is evident as early as after 5 years of known diabetes. The findings are in accordance with the findings of Clauson P et al (1994).

The decrease in fasting C-peptide levels with duration was even more notable when considered in relation to the increase in blood glucose that occurred with increased duration which should theoretically increase rather than decrease B-cell secretion. Bergstrom R W et al (26) (1990) showed that both fasting insulin and fasting C-peptide have been employed as a measure of insulin resistance. However C-peptide is not metabolized by the liver and therefore is a more reliable index of pre-hepatic insulin secretion than the fasting insulin.

Several studies have shown that diabetic patients, who have retained appreciable B-cell function, as indicated by serum C-peptide levels, generally have relatively "stable" or easily managed diabetes as quantitate by variability in blood and urine sugar levels or by number of episodes of ketoacidosis or hypoglycemia. [28, 29]

It seems likely that deterioration would have been less marked if treatment had been changed markedly with increasing duration from diet alone to diet sulphonylurea and later from sulphonylurea to insulin. Clauson P et al (1994) showed a high frequency of insulin treatment in patients with more than 15 years of duration. This frequency can probably, be equated to the occurrence of "secondary failure" during the preceding period of diabetes. Whether this deterioration is attributable to the aging processes or to the other factors such as increasing adiposity, abdominal obesity and/or physical inactivity remains equivocal. The decline in glucose tolerance from young to middle age could be explained by indexes of body composition and activity level. [30]

A decrease in B-cell function with duration in type-2 diabetic patients could be due to 'glucotoxicity' a long term negative influence of chronic hyperglycemia and/or other metabolic abnormalities of a diabetic state. Hence, it could be contemplated without ambiguity that the decrease in C-peptide levels is as a result of progressive insulin exhaustion. Collectively, our results and those of Broughton et al [13] and Coon et al [14] suggest that insulin resistance is an avoidable consequence of abdominal fat accumulation with advancing age.

Intra-abdominal fat accumulation maybe associated with the development of insulin resistance, although mechanisms have not been fully elucidated. [13, 14] Adipose tissue in the visceral region is highly sensitive to lipolytic stimuli, particularly in those regions drained by the portal circulation. As a consequence, as increase influx of free fatty acids to the liver maybe stimulating gluconeogenesis and inhibiting hepatic insulin clearance, leading to hyperinsulinemia and consequently insulin resistance.
This work strongly confirms the earlier views of C-peptide being a valuable parameter in assessing progress of disease. Further studies,

including a prospective one are however needed to establish a cause-defect relationship between long-term metabolic abnormalities of a diabetic state and a decrease in B-cell function.

CONCLUSION

The information about the rising prevalence of type-2 diabetes in a developing country like India, its declined prevalence in the developed countries by changes in life styles, and related risk factors, prompted us to corroborate the C-peptide status in type-2 diabetes.

The present study was undertaken on a total number of fifty six type-2 diabetic patients in India Gandhi Medical College and Hospital, Nagpur. Age and sex matched apparently healthy controls were also studied for comparison.

The results recorded indicate a highly significant decrease in the fasting serum C-peptide levels in both sexes compared to their respective controls. This decrease is attributed to duration-linked impairment of B-cell function. However, the impact of duration-associated deterioration of metabolic control has not been clarified.

Secondary failure of worsening metabolic control, despite oral hypoglycemic agents is a recognized event in type-2 diabetes mellitus. Thus the rate of "secondary failure" has ranged from essentially all patients after 10 years of duration to 100%.

Thus, we conclude that decreased serum C-peptide level is strongly associated with deterioration of B-cell function in long standing type-2 diabetic patients and the determination of C-peptide should be used as a tool to prevent the long-term diabetic complications.

ACKNOWLEDGEMENT

Author thanks Dr S. M. Chaudhari for his strong assistance for collecting the data. Also, we thank M. A. Lonje and N. S. Dange at Indira Gandhi Government Medical College and Mayo Hospital, India for their support.

REFERENCES

1. Powers A C. 2001. Diabetes Mellitus In : Braunwald E, Fauci A S, Kasper D L, Housa S L, Longo D L, Jameson J L. Harrison's Principal of Internal Medicine 15th ed. *McGraw-Hill, Inc*., **2** : 2110.
2. Park K. 2002. Diabetes Mellitus. Parks textbook of Preventive and Social Medicine.17th ed. *Banarsidas Bhanot Publishers Inc*., 294-298.
3. WHO .1998. Prevention and Control of Diabetes Mellitus, Report of an Inter country workshop, Dhaka, Bangladesh, 27-30 .
4. DeFronzo R A, Bonadonna, R C, Ferranninni E. 1992. Pathogenesis of NIDDM: a balanced overview. *Diabetes Care*; **15**:318-368.
5. Kahn C. R. 1994. Insulin action, diabetogenes, and the cause of type-II diabetes. Diabetes; **43**:1066-1084.
6. Sacks D B, McDonald J M. 1996. The pathogenesis of type II diabetes mellitus: a polygenic disease. *Am J Clin Pathol;* **105**: 149-156.
7. Sacks D B. 1999. Carbohydrates In: Burtis C.A, Ashwood ER et al ed. Tietz Textbook of Clinical Chemistry 3rd ed. W.B. *Saunders company, Inc*.; 767.

8. Duckworth W C, and Kitabchi A E. 1981. Insulin metabolism and degradation. *Endocrine Reviews*; **2**:210-233.
9. Lindstrom T, Arnquist H J, Ludvigsson J, Von Schenck H W. 1992. C-peptide profiles in patients with non-insulin dependent diabetes mellitus before and during treatment. *Acta Endocrinol Copenh*; **126** (6): 477 – 489.
10. Misbin R I. Merimee T J and Lowenstein J M. 1976. Insulin removal by isolated perfused rat liver. *Am J Physiol;* **203** : 171 – 77.
11. Stoll R W, Touber J L, Menahan L A and Williams R H. 1970. Clearance of porcine insulin, proinsulin, and connecting peptide by the isolated rat liver. *Proc Soc Exp Biol Med*; **133** : 894 – 96.
12. Friedewald W T, Levy R I, Fredrickson O S. **1972**. Estimation of the concentration of low density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. *Clin Chem*; **18** (6) : 499 – 502.
13. Broughton D L, James O W, Alberti K G, Taylor R. 1991. Peripheral and hepatic insulin insensitivity in healthy elderly human subjects. *Eur J Clin Invest*; **21**: 13-21.
14. Coon P J, Bleecker E R, Meyers D A et al. 1989. Effects of body composition and exercise capacity on glucose tolerance, insulin an lipoprotein lipids in healthy older men : a cross sectional and longitudinal study . *Metab*; **38**: 1201 – 209.
15. Grodsky G and Forsham P. 1960. An immunological assay of total extractable insulin in man. *J Clin Invest*; **39**: 1070-79.
16. Singer D, Horwitz D. 1967. Long term experience with sulphonylureas and placebo. *N Engl J Med*; **277**: 450- 469.
17. Kuzuya T, and Matsuda A. 1976. Disapperance rate of endogenous human C-peptide from blood. *Diabetologia*; **12**: 519 – 21.
18. Gabbay K H, Sosenko J M, Banuchi G A, Mininsohn M J, Fluckiger R. 1979. Glycosylated hemoglobins, increased glycosylation of hemoglobin A in diabetic patients. Diabetes; **28**: 337-40.
19. Little R R, England J D, Wiedmeyer H M, Mckenzie E M, Petlitt D J, Knowler W C, Goldstein D E. 1988. Relationship of glycosylated hemoglobin to oral glucose tolerance: Implications for diabetes screening. *Diabetes*; **37** : 60 – 64.
20. Santiago J V, Davis J E, Fisher F. 1978. Hemoglobin A1C levels in a diabetes detection program. *J Clin Endocrinal Metab*; **47** : 578 – 80.
21. Horwitz D L, Rubenstein A H, Mako M, Cruz A, Blix P M. 1978. C-peptide in conditions other than Diabetes mellitus. *Diabetes*; **27**, Suppl 1 : 267 – 71.
22. Heding L G, Turner R C, Harris E. 1975. C-peptide, proinsulin and insulin responses to fish-insulin induced hypoglycemia in the diagnosis of

insulinomas. *Diabetes*; **24** : suppl 2 : 412.

23. Sandler R, Horwitz D L, Rubenstein A H, Kuzuya H. 1975. Hypoglycemia and endogenous hyperinsulinism, complicating diabetes mellitus: application of the C-peptide assay to diagnosis & therapy. *Am J Med*; **59**: 730 – 736.

24. Horwitz O L, Starr J I, Mako M E. 1975. Proinsulin, insulin and C-peptide concentration in human portal and peripheral blood. *J Clin Invest*; **55**: 1278 – 83.

25. Clauson P, Linnarsson R, Gottsoter A, Sundkvist G, Grill V. 1994. Relationships between diabetes duration, metabolic control and B-cell function in a representative population of Type-2 diabetic patients in Sweden. *Diabetic Medicine*; **11** : 794 – 801.

26. Bergstrom R W, Newell-Morris L L, Leonetti D C, Shuman W P, Wahl P W, Fujimoto W Y. 1990. Association of elevated fasting C-peptide level and increased intrabdominal fat distribution with development of NIDDM in Japanese American men. *Diabetes.*; **39** :104 – 111.

27. Faber O K, Binder C. 1977. C-peptide response to glucagon: A test for the residual B-cell function in diabetes mellitus. *Diabetes*, **26**(7) : 605 – 10.

28. Horwitz D L, Reynolds C, Molnar G D et al. 1974. Stable and unstable diabetes: differences in endogenous insulin secretion as determined by C-peptide measurement. *Clin Res*; **22** : 471 A.

29. PiIdes R S, Grajwer L, Horwitz D L et al. 1975. C-peptide secretion in insulin-requiring juvenile diabetics. *Pediatr Res*; **9** : 354.

30. Shimokata M, Muller DC, Fleg J L et al. 1991. Age as independent determinant of glucose tolerance. *Diabetes* ; **40** : 44 -51.

31. Horwitz D L, Kuzuya H and Rubenstein A H et al. 1976. Circulating serum C-peptide: a brief review of diagnostic implications. *Engl J Med*; **295** : 207 – 09.

32. Kuzuya T, and Matsuda A. 1976. Disappearance rate of endogenous human C-peptide from blood. *Diabetologia*; **12** : 519 -21.

33. Faber O K, Kehlet H, Madsbad S, Binder C. 1978. Kinetics of Human C-peptide in Man. *Diabetes*, **27** Supp 1, 207-9.

34. Berson S A, Yalow R S, Bauman A, Rothschild M A, Newerly K. 1956. Insulin-I^131 metabolism in human subjects. Demonstration of insulin binding globulin in the circulation of insulin treated subjects. *J Clin Invest*; **35**: 170 -90.

35. Sonksen P H, Tompkins C V, Srivatava M C et al. 1973. A comparative study on the metabolism of human insulin and porcine proinsulin in man. *Cli Sci Molecul Med;* **45**: 633 – 54.

الملخص

يحظى دور الببتيد C في مصل الدم في تقدير كمية الأنسولين المُفرزة داخليًا باهتمام متزايد، نظرًا لعدم وجود نشاط بيولوجي لهذا الببتيد على الأنسجة المتجانسة أو غير المتجانسة، وعدم قدرته على تعديل عمل الأنسولين أو البروأنسولين. تهدف هذه الدراسة إلى تقييم مستويات الببتيد C في مصل الدم لدى مرضى السكري من النوع الثاني، ومقارنتها بمستويات مجموعة ضابطة من الأصحاء. خضع حوالي 56 مريضًا بالسكري من النوع الثاني (35 ذكرًا، 21 أنثى) لتحليل الببتيد C في مصل الدم، وسكر الدم (صائمًا، بعد الوجبات)، والهيموجلوبين السكري، ومستوى الدهون. قورنت النتائج مع مجموعة ضابطة من الأصحاء من نفس العمر والجنس. وقد لوحظ انخفاض ملحوظ ($p<0.001$) في مستوى الببتيد C في مصل الدم لدى مرضى السكري من النوع الثاني المزمنين، مقارنةً بمجموعة ضابطة من الأصحاء من نفس العمر والجنس. لوحظ انخفاض في تركيز الببتيد C في مصل الدم أثناء الصيام لدى مرضى السكري من النوع الثاني من كلا الجنسين، مقارنةً بمجموعة الضبط. وكان هذا الانخفاض ذا دلالة إحصائية عالية ($p<0.001$ و $p<0.05$ على التوالي). ولم يُلاحظ فرقٌ ذو دلالة إحصائية في تركيز الببتيد C في مصل الدم بين الذكور والإناث. تشير هذه البيانات إلى أن مقاومة الأنسولين هي نتيجةٌ يمكن تجنبها لتراكم الدهون في منطقة البطن مع التقدم في السن. يؤكد هذا العمل بقوة الآراء السابقة التي تُشير إلى أن الببتيد C يُعد مؤشرًا هامًا في تقييم تطور المرض.

الكلمات المفتاحية: الببتيد C في مصل الدم، الأنسولين، الهيموجلوبين السكري، البروأنسولين، داء السكري من النوع الثاني.