



### Study of Some Biochemical Parameters in Patients with Type II Diabetes Mellitus

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Article's Information	Abstract
Received: 21.02.2021 Accepted: 20.08.2022 Published: 30.09.2022	Type 2 diabetes (T2DM) is a growing global health concern that, usually occurs in adults which accounts for about 90% of population. The diabetic in type II diabetes mellitus begins with insulin resistance. This type of diabetes typically exists in citizens over the age of forty, however, it is now being diagnosed more frequently in young adults and children who are overweight. The aim of this study was to assess the 130 subjects,
<b>Keywords:</b> Diabetes mellitus Insulin resistance Glycated haemoglobin C-reactive protein	divided into 30 subjects as control and 100 subjects as diabetics group. For each analysis were done: blood sugar, glycated haemoglobin, blood urea, blood creatinine, lipids profile (T.Cholesetrol, triglyceride, LDL, HDL and VLDL) and CRP. The findings revealed significantly higher ( $p \le 0.01$ ) along with all parameter's concentration of diabetes in comparison with the control group.

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#### 1. Introduction

metabolic disease Α clustered characterized bv hyperglycemia absolute insulin secretion malfunction, insulin disturbances or both [3]. Diabetes mellitus II is the most special brand of diabetes, which accounts for about 90% of the population. The diabetic in type II diabetes mellitus begins with insulin resistance, that is a metabolic abnormality characterized by impairment in the ability of insulin to produce its normal biological, physiological, or clinical effects, that the body is capable of produce insulin but this is not adequate or the body is unable to respond to its effects causes decreased insulin-stimulated glucose transport and metabolism [1]. Insulin functions are enhanced glucose performance, lower utilization of glycogen in the liver, reduced glucose absorption in the organ, and increased lipolysis in adipose tissue to raise the levels of circulating free fatty acid (FFA) [4]. Several parameters will be a potential guide for diagnosis which could enhance patient survival. The aim of this study is to assess the levels of some biochemical including: blood sugar, HbA1c, B.urea, B.creatinine, lipids profile and Creactive protein were measured in the blood of diabetics and control group.

# 2. Subjects, Materials and Methods Study groups:

The present study comprised of 130 subjects aged from 30 to 60 years old, divided into 30 subjects as the control group and 100 subjects as diabetics group, included (50) male and (50) female, collected from local Hospital in Baghdad, Iraq.

#### Exclusion criteria:

- Type 2 diabetic subjects old (<60 years).
- Type 1 diabetic subjects.
- Any type 2 diabetic with acute illness.
- Gestational Diabetes.
- Obvious cognitive disorders.
- -Patients suffering from any other disease.
- Patients with blood diseases.

#### Blood sample collection:

A blood sample 5 ml was obtained from each patient. The samples were transferred directly into gel tubes and allowed to coagulate for 20 minutes at room temperature, the tubes were centrifuged at 3000 (rpm). Serum for each sample was stored in clean Eppendorf tubes and all tubes were kept at - 20C until the time of examination.

## Analysis of parameters level of diabetics and control groups:

Glucose, (HbA1c), Urea, Creatinine T.cholesterol, Triglyceride, HDL- and LDL were analyzed by Cobas, Germany kit. While CRP was analyzed by biosystems Company, Spain. All analyses were done according to the manufacturer's instruction.

#### Statistical analysis:

To detect the influence of differential variables on research parameters, the Statistical Analysis Method- SAS (2012) software was used. The LSD test (Analysis of Variation-ANOVA) was used for important contrast of means. In this ANJS, Vol.25 (3), September, 2022, pp. 16-19

analysis, the Chi-square test was used to greatly equate the ratio (0.05 to 0.01 probability).

#### 3. Results and Discussion

The present study revealed that the blood sugar (mean  $\pm$  SE) of diabetics and non-diabetics groups was (257.67  $\pm$ 12.72, 88.00  $\pm$ 1.72mg/dl) respectively. The result showed significant differences (P $\leq$ 0.01) as shown in Figure 1.

Our results were agreed with another study that confirmed a statistically significant increase in B.glucos level in diabetics [6]. This raise may be due to insulin resistance, which leads to increased glucose production and impaired glycogen metabolism, as well as activation of the glucose formation pathway in the liver, with reduced glucose uptake into the muscles [7].



Figure 1. Comparison of B.sugar between diabetics and non-diabetics groups.

HbA1c (mean  $\pm$  SE) of diabetics and non-diabetics groups was (9.09  $\pm$  0.20, 5.22  $\pm$  0.09 mg/dl) respectively. The result showed significant differences (p  $\leq$  0.01) as shown in Figure 2. Similar findings were reported with another study that proved there is a significantly raising of HbA1c in diabetics control [8]. This increase may be due to the high concentration of glucose in the blood [9].



non-diabetics groups.

In diabetics group, urea level is significantly higher than in non-diabetics group (p < 0.01) as shown in Figure 3.

In diabetics group, Creatinine level is significantly higher than in non-diabetics group (p < 0.01) as shown in Figure 4.

A similar finding was reported in other studies that agreed with our data showed that B.urea and creatinine significantly increase in diabetics compared to healthy [10]. The results demonstrated that elevation of B.urea and creatinine were may be due to The tiny blood vessels and tiny filters in the kidneys may be impaired by elevated blood glucose levels. leads to dysfunction of these vessels, causing abnormal amounts of creatinine and BUN would notice in the blood [11].



Figure 3. Comparison of urea between diabetics and nondiabetics groups.



Figure 4. Comparison of creatinine between diabetics and non-diabetics groups.

(Mean  $\pm$  SE) of serum TC level of non-diabetics group and diabetic groups were  $(138.53 \pm 5.03 \text{ vs.} 178.75 \pm 1.85)$ mg/dl), TG level (116.03  $\pm$  6.36 vs. 194.53  $\pm$  13.85 mg/dl), HDL-C (64.76 ± 5.08 vs. 42.43 ± 5.34 mg/dl), LDL-C (82.46 ±2.93 vs. 121.73 ±4.24 mg/dl), finally, VLDL-C  $(\text{mean} \pm \text{SE})$  was  $(23.20 \pm 1.27 \text{ vs.} 38.2 \pm 2.56 \text{ mg/dl})$ . These results were considerably higher (p < 0.01) than the control group (p<0.01). as shown in figures (5-9). The results in the present study agreed with another study that proved there is a significant increasing in lipid profile in diabetes compared to control [5]. This increase may be the response to dyslipidemia because Insulin tolerance and type II diabetes is characterized by such a triad of lipids: (1) elevated plasma triglyceride levels, (2) low HDL levels, and (3) thin, lowdensity lipoprotein appearance. As well as postprandial sdLDL), as well as, hyperlipemia. [12,13].

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Figure 5. Comparison among difference groups in cholesterol.



Figure 6. Comparison among difference groups in triglyceride.







Figure 8. Comparison among difference groups in LDL.



Figure 9. Comparison among difference groups in VLDL.

CRP concentration in diabetics group (mean  $\pm$  SE) was (46.83  $\pm$  1.69 mg/L) that significantly higher (p = 0.0001) than the non-diabetics group (5.93  $\pm$  0.41 ng/l) as shown in Figure 10. These results are in agreement with another study that proved significant increasing in CRP in diabetes as compared to control [14]. This increase may be due to The concentration of advanced glycation end products that have been shown to activate macrophages, increase upregulation of the synthesis of IL-1, IL-6 and TNF-a is elevated, resulting in the development of CRP by the liver [15].



Figure 10. Comparison of CRP between diabetics and non-diabetics groups.

#### 4. Conclusion

HbA1c and other parameters were increased in diabetic patients; the analyses revealed that there was a statistically significant increasing with ( $p \le 0.01$ ) between diabetics and control groups. HbA1c is a good indicator to flow up the diabetics.

#### References

- Piero M. N.; "Diabetes mellitus a devastating metabolic disorder," Asian J. Biomed. Pharm. Sci.; 4(40): 1–7, 2015, doi: 10.15272/ajbps.v4i40.645.
- [2] Goldenberg R. and Punthakee Z.; "Definition, Classification and Diagnosis of Diabetes, Prediabetes and Metabolic Syndrome Canadian Diabetes Association Clinical Practice Guidelines Expert Committee," Can. J. Diabetes, 37: S8–S11, 2013.
- [3] Sailakshmi B. N. S. and Devi K. S.; "Research and Reviews : Journal of Medical and Health Sciences A

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Short Review on Gastrointestinal Disorders," J. Med. Heal. Sci.; 5(3): 1–7, 2016.

- [4] Baynest H. W.; "Classification, Pathophysiology, Diagnosis and Management of Diabetes Mellitus," J. Diabetes Metab.; 06(05), 2015, doi: 10.4172/2155-6156.1000541.
- [5] Made Junior Rina Artha I.; et al.; "High level of individual lipid profile and lipid ratio as a predictive marker of poor glycemic control in type-2 diabetes mellitus," Vasc. Health Risk Manag.; 15: 149–157, 2019, doi: 10.2147/VHRM.S209830.
- [6] Bouaziz A.; Ben Nasr S.; Zinelabidine H. T. and Mnif W.; "Study of biochemical parameters in type 2 diabetes Tunisians mellitus patients," Microbiol. Hyg. Alim, 24(December 2012): 49–59, 2012.
- [7] Shouhip H. A.; "Diabetes mellitus [Diabetes mellitus]," Rev. Bras. Med.; 62(SPEC. ISS.): 60–71, 2005.
- [8] Florkowski C.; "HbA1c as a diagnostic test for diabetes mellitus - Reviewing the evidence," Clin. Biochem. Rev.; 34(2): 75–83, 2013.
- [9] Fonseca V.; Inzucchi S. E. and Ferrannini E. L. E.; "Redefining the diagnosis of diabetes using glycated hemoglobin," Diabetes Care, 32(7): 1344–1345, 2009, doi: 10.2337/dc09-9034.
- Pecoits-Filho R.; et al.; "Interactions between kidney disease and diabetes: Dangerous liaisons," Diabetol. Metab. Syndr.; 8(1): 1–21, 2016, doi: 10.1186/s13098-016-0159-z.
- [11] Narva A. S. and Bilous R. W.; "Laboratory assessment of diabetic kidney disease," Diabetes Spectr.; 28(3): 162–166, 2015, doi: 10.2337/diaspect.28.3.162.
- [12] Goldberg I. J.; "Clinical review 124: Diabetic dyslipidemia - Causes and consequences," J. Clin. Endocrinol. Metab.; 86(3): 965–971, 2001, doi: 10.1210/jc.86.3.965.
- [13] Ormazabal V.; Nair S.; Elfeky O.; Aguayo C.; Salomon C. and Zuñiga F. A.; "Association between insulin resistance and the development of cardiovascular disease," Cardiovasc. Diabetol.; 17(1): 1–14, 2018, doi: 10.1186/s12933-018-0762-4.
- [14] Kanmani S.; Kwon M.; Shin M. K. and Kim M. K.; "Association of C-Reactive Protein with Risk of Developing Type 2 Diabetes Mellitus, and Role of Obesity and Hypertension: A Large Population-Based Korean Cohort Study," Sci. Rep.; 9(1): 1–8, 2019, doi: 10.1038/s41598-019-40987-8.
- [15] Behl T.; et al.; "Role of C Reactive Protein in Diabetes Mellitus and Its Associated Complications," Indo Am. J. Pharm. Res.; 4(11): 5315–5320, 2014, [Online]. Available: http://www.scopemed.org/?mno=174659.