DocID: https://connectjournals.com/03896.2020.20.5695

Biochem. Cell. Arch. Vol. 20, No. 2, pp. 5695-5699, 2020 | www.connectjournals.com/bca | *ISSN 0972-5075*

PATHOLOGICAL CHANGES ASSOCIATED WITH TYPE 1 DIABETES IN THE CITY OF MOSUL

Amaal A. Alkhashab[1*], Banan R. Dabdoub[1] and Haitham L. Abdulhadi[2]

[1]Department of Biology, Education College for Pure Sciences, University of Mosul, Iraq.
[2]Department of Biology, Education College for Pure Sciences, University of Anbar, Iraq.
*e-mail : amal.biology@yahoo.com; amal1234biology@gmail.com

(Received 29 Macch 2020, Revised 5 June 2020, Accepted 15 June 2020)

ABSTRACT : The present study was designed to investigate some physiological effects of diabetic mellitus Patients in the city of Mosul, Iraq. 100 diabetic patients who were attended to the AL-Khansaa, Ibn- AL-Atheer Hospital and AL-Wafaa center in Mosul city, with 10 healthy males and Females as a control group (20 healthy individuals; 10 males and 10 females). The diabetic patients (type 1) were divided into two main groups; Group1: diabetes mellitus males (40 patients) and Group 2: diabetes mellitus Females (40 patients). Then each type were divided into three subgroups according to the period duration of the diabetes mellitus (SG1:1 -5 years, SG2: 6-10 years and SG3: more than 10 years). Blood samples were gathered and controlled from each patient.Blood samples were used for Blood Hemoglobin, Packed Cell Volume, White Blood Cell Count, Fasting Blood Glucose and Renal Function Tests. The results showed a significant decrease (pd" 0.05) in SG3 in hemoglobin level and PCV% in all subgroups of males and females when compared with the control group. Males showed significant increase in WBC count/SG3, while females showed the significantly in all groups. Also, there was a significant increase in two groups in concentration of Fasting Blood Glucose, blood urea and serum creatinine in all subgroups as compared with control group.

Key words : Diabetes mellitus, haemoglobin, leucocyte, B. Urea, S. creatinine.

INTRODUCTION

Type 1 diabetes mellitus (TIDM) is a glucose homoeostasis disease characterized by autoimmune destruction of insulin- producing pancreatic β- cell, which gradually contributes to insulin deficiency and resulting hyperglycemia. If associated untreated, insulin deficiency leads to progressive metabolic disturbance, with worsening hyperglycemia, ketoacidosis, hunger and death. In an attempt to restore and retain euglycemia, therapy tries to imitate the action of the indigenous β- cell by replacing insulin exogenously and involves regular monitoring of blood glucose concentrations (Justin *et al*, 2013; Al-Jobouri, 2020). Type 1 diabetes is liable for 5% to 10% of all kinds of diabetes. Its risk variables include autoimmune, genetic and environmental. There is no recognized technique to prevent type 1 diabetes so far (Deshpande *et al*, 2008). Diabetic nephropathy (DN) or diabetic renal illness relates to the decline in the function of the kidney seen in patients with chronic type 1 diabetes mellitus. The progression of the disease is known to happen in several phases and is associated with glycemic and blood pressure control (Sulaiman, 2019).

Patients with chronic kidney disease due to diabetes mellitus are at risk for diabetic retinopathy and need regular fundus examination and timely treatment to prevent blindness and visual impairment (Mian *et al*, 2019). Diabetes is a group of metabolic illnesses characterized by hyperglycemia caused by insulin secretion deficiencies, insulin action, or both (ADA, 2013). Chronic diabetes hyperglycemia is correlated with long-term harm, dysfunction and failure of various bodies, particularly of the eyes, kidneys, nerves, heart and blood vessels. A number of pathogenic processes are involved in the development of diabetes. These range from autoimmune destruction of pancreatic b-cells resulting in insulin deficiency to abnormalities resulting in insulin resistance. The deficiency of insulin action on target tissues is the basis of abnormalities in carbohydrate, fat and protein metabolism in diabetes. Deficient insulin action results from inadequate insulin secretion and/or decreased insulin response of tissues at one or more points in the complex pathways of hormone action. Insulin secretion impairment and insulin action defects often coexist in the same patient and it is often uncertain, which anomaly is the main cause of hyperglycemia, if either alone (American Diabetes Association, 2010).

Type 1 diabetes mellitus (T1DM) is a chronic lifelong

glucose homeostasis disease characterized by autoimmune destruction of the insulin-producing b-cell, leading to insulin deficiency and hyperglycemia. New-onset T1DM may present with classic results of polyuria, polydipsia, polyphagia and weight loss; as diabetic ketoacidosis (DKA) with vomiting, abdominal pain, and lethargy in relation to classic symptoms; or as an incidental finding found on urine or blood testing for other reasons. Treatment focuses on rehydration and insulin replacement. Because T1DM is a chronic disease, best management is achieved when patients and their families become acquainted with their condition as part of a continuous, empowering relationship with their diabetes care team. Optimal health and well-being are achieved when blood glucose is tightly controlled. Intensive control significantly reduces the likelihood of developing T1DM microvascular and macrovascular complications. (Gregory *et al*, 2013). Diabetes and its complications, fatalities and social expenses have an enormous and quickly increasing effect on the United States. Between 1990 and 2010, the amount of individuals living with diabetes has tripled and the amount of fresh instances (incidence) doubled annually. (Gregg and Wang, 2010). Adults with diabetes are at 50% higher risk of death from any cause than adults without diabetes, in addition to the risk of myriad complications. Due to the importance of this disease and its high incidence globally and locally (Centers for Disease Control and Prevention, 2015). The objective of this research is to investigate hematological markers (hemoglobin and white blood cells) in patients, to investigate biochemical markers (blood glucose, kidney function test) in patients.

MATERIALS AND PRACTICES

Blood collected from the patients' troughs in AL-Khansaa, Ibn-ALatheer Hospital and AL-Wafaa center in Mosul city, Iraq, with 100 patients with type 1 of diabetesdivided in to (40 males and 40 females) , with 20 healthy (males and females)as a control group. The diabetic patients (type 1) were divided into two main groups; Group 1: diabetes mellitus males (40 patients) and Group 2: diabetes mellitus Females (40 patients) was (1-16) years. Then each type were divided into three subgroups according to the period duration of the diabetes mellitus (SG1: 1-5 years, SG2: 6-10years, and SG3:more than 10 years). Blood samples were gathered and controlled from each patient.

Blood samples were used for blood hemoglobin, white blood cell count fasting blood glucose, renal function tests.

Analysis of statistics

All information was provided as the mean. Data was analyzed using Prism analysis program, Mean values were deemed statistically important at ($p \leq 0.05$).

RESULTS

Anemia rate

The findings of this research showed a substantial reduction in Hb levels and PCV% in all subgroups of males and females patients of type 1 ($P \leq 0.05$) with the control group (Table 1).

The results of WBC count in type 1 patients showed significant increase ($P \leq 0.05$) SG3 group in males and females.

Blood glucose

The present results of blood Glucose in males and females patients showed a significant increase ($P \leq 0.05$) in all subgroups in two groups compared with the control group (Figs. 1, 2).

Renal Function tests

Blood urea and serum creatinine

The mean level of blood urea and serum creatinine

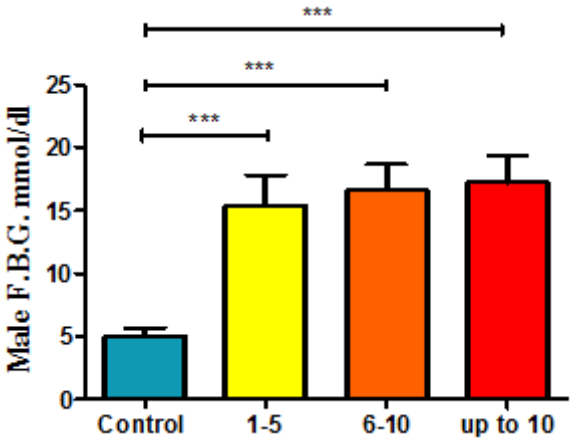

Fig. 1 : Level F.B.G in male. ***on each bar indicate significantly different groups.

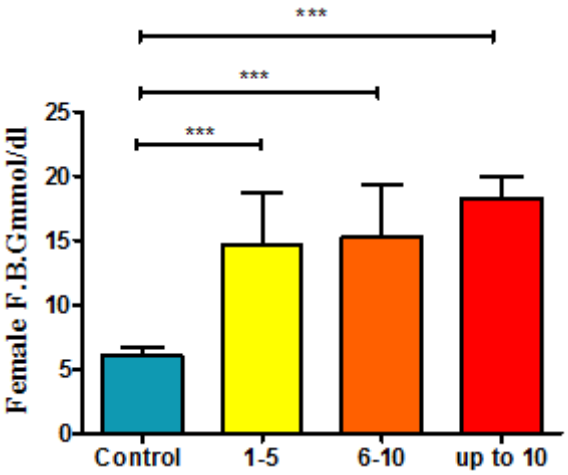

Fig. 2 : Level F.B.G in female. *** on each bar indicate significantly different groups.

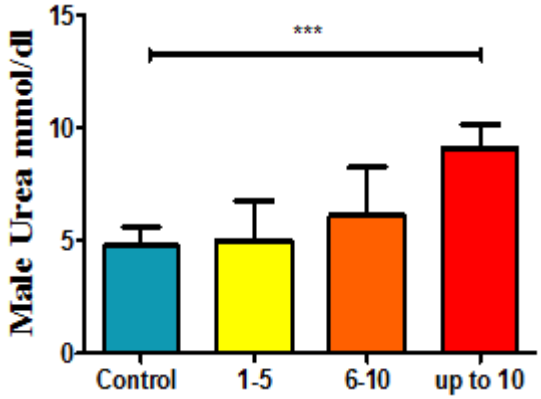

Fig. 3 : Blood urea level in male.

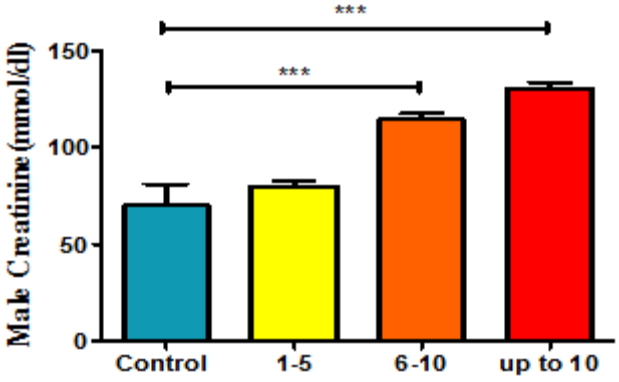

Fig. 5 : Creatinine level in male.

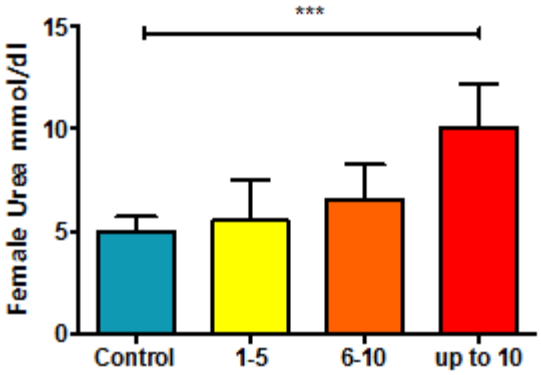

Fig. 4 : Blood urea level in female.

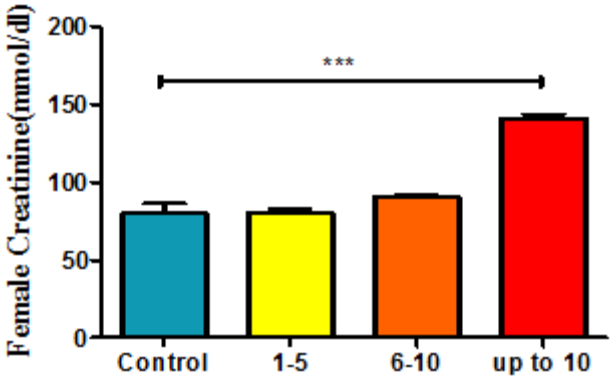

Fig. 6 : Creatinine level in female.

Table 1 : Level of blood haemoglobin (Hb) and PCV in males and females DM patients.

Patient Subgroups	Males Hb gm/dl Mean± SD	Males PCV% Mean± SD	Females Hb gm/dl Mean± SD	Females PCV% Mean± SD
Control	14.20 ± 0.1414	1.414± 0.6325	14.28± 0.8319	43.00 ± 2.121
SG_1	13.45 ± 0.8977	5.643± 0.9155	13.00± 0.9864	40.11 ± 2.883
SG_2	13.62 ± 0.7543	3.142± 0.5097	12.32 ± 1.435**	37.26 ± 2.668**
SG_3	12.11 ± 0.8682***	36.32± 3.322*	11.79 ± 1.298***	35.05 ± 3.770***

Table 2 : Level of WBCs count in males and females DM patients.

Patient Subgroups	Males WBCs Count Mean± SD Cells/µl of blood	Females WBCs Count Mean± SD Cells/µl of blood
Control	6.700 ± 0.5339	6.660± 0.5273
SG_1	7.658 ± 1.341	7.884 ± 1.851
SG_2	8.053 ± 1.754	8.947 ± 1.593
SG_3	9.605 ± 2.150**	9.737 ± 2.413**

in patients showed a significant increase ($p \leq 0.05$) in two groups with all subgroups compared with the control group (Figs. 3, 4, 5, 6 and Table 3).

DISCUSSION

The current research stated a decrease in hemoglobin and PCV percentage levels in patients in all groups' types relative to control group, the decrease in Hb% levels in diabetic patients due to vitamin deficiencies such as Folate and B_{12}, as well as iron and erythropoietin deficiencies (Bozkir, 2019).

Table 3: Level of the urea and creatinine males and females DM patients.

Patient groups	Males Blood Urea (mmol/dl) Mean± SD	Females Blood Urea (mmol/dl) Mean± SD	Males Creatinine (mmol/dl) Mean± SD	Females Creatinine (mmol/dl) Mean± SD
Control	4.750 ± 0.8292	5.000 ± 0.7071	70.00 ± 24.75	80.00 ± 14.14
SG I	5.000 ± 1.771	5.526 ± 1.955	80.00 ± 16.95	80.63 ± 13.61
SG2	6.105 ± 2.153	6.526 ± 1.720	114.5 ± 19.84***	90.53 ± 6.701
SG3	9.105 ± 1.034***	10.05 ± 2.117***	130.3 ± 20.48***	141.1 ± 17.01***

Anemia was revealed to be due to decreased manufacturing of erythropoietin owing to failure of the kidneys and enhanced on enzymatic glycosylation of RBC membrane proteins (Arun and Ramesh, 2002). Anemia is also associated with oxidative stress; erythrocytes are a significant antioxidant in the blood (Grune *et al*, 2000). Although, anemia is clearly associated with both micro- and macrovascular complications in patients with type 1 diabetes, it remains to be established what role anemia may have in the development or progression of these complications. Because there is a direct relationship between anemia and diabetic kidney disease, anemia such as albuminuria may be a marker of more potent microvascular disease rather than being directly pathogenic (Thomas *et al*, 2004).

Compared to the control group, the findings showed a substantial rise in blood glucose level in two groups.

Diabetes is a group of metabolic disorders characterized by a chronic type glycemic disorder caused by insulin secretion abnormalities, insulin action or both. Type 1 diabetes is the consequence of an autoimmune response to proteins in the pancreatic islet cells (Turkey, 2017).

The pathogenesis of selective β-cell destruction within the islet in type 1 diabetes mellitus is difficult to follow due to marked heterogeneity of the pancreatic lesions. At the onset of overt hyperglycemia, a mixture of pseudoatrophic islets with cells producing glycogen, somatostatin and pancreatic polypeptide. The autoimmune destruction of pancreatic β cells leads to a deficiency of insulin secretion that leads to the metabolic derangements associated with type 1 diabetes (Ozougwu *et al*, 2013).

Results obtained from the present study showed a significant increase of serum creatinine and blood urea in diabetic patients compared with control group. Levels of BUN and creatinine serum are used as a parameter of kidney function tests. Diabetes mellitus is the leading cause of chronic renal failure. As the period of diabetes progresses there is simultaneous increase of inflammatory markers (Acharjya *et al*, 2019). Patients of diabetes mellitus type 2 are known to have high levels of BUN and Serum creatinine levels as compared to non-diabetics. There is an association between the raised BUN, serum creatinine levels and poor glycemic controls, subsequently leading to complication like nephropathy in diabetic patients (Bhatia *et al*, 2019). The diabetic retinopathy is closely related to renal function in patients with type 1 diabetes. The patients with renal dysfunction have a high risk of diabetic retinopathy. Renal function may predict the occurrence and development of the diabetic retinopathy (Wan, 2019).

Hyperglycemia in diabetes mellitus can increase the risk of com plications in various organs, one of which is chronic renal failure. BUN is a normal waste product of normal metabolism that is excreted by the kidneys and is a residual product of protein breakdown. In cases of kidney function disorder, BUN is not excreted normally by the kidneys so it accumulates in the body and causes the levels of urea in the blood to become elevated (Puspitasari and Aliviameita, 2018).

CONCLUSION

The conclusion of this study that anemia, blood urea and creatinine are useful biomarkers for assessing kidney functions (nephropathy) in diabetic patients.

ACKNOWLEDGMENTS

We would like to thank all those who helped us in the University of Mosul and in Al Khansa Hospital and Al Wafa Specialized Center.

REFERENCES

Acharjya D, Banerjee U and Bhattacharyya S (2019) Serum Inflammatory Markers Of Diabetetic Kidney Disease And Their Correlation To Lipid Parameters Among Diabetetic Individuals of Eastern India. *Int. J. Scientific Res*. **8**, 1.

Al-Jobouri F R (2020) Assessment of immune response in patients with type 2 Diabetes mellitus. *Annals Trop. Med. Pub. Hlth* **23**(2), 51-57.

American Diabetes Association (2010) *Diabetes Care*. **33**(Suppl 1), S62–S69.

American Diabetes Association (2013) *Diabetes Care* **36**(Suppl 1), S67–S74.

Bhatia K, Misra, P, Singh A, Mukherjee B and Ambade V (n. d.) Studey of Blood Urea Nitrogen (BUN), Serum Creatinine In Diabetetic and Non-Diabetetic Patients in a Tertiary Care Hospital. *Int. J. Medical and Biomed. Stud.* **3**(4). https://doi.org/10.32553/ijmbs.v3i4.216.

Bozkir C (2019) Iron deficiency anemia and diabetes mellitus. *Crimson Publishers* **3**(2). DOI: 10.31031/IOD.2019.03.000557.

Centers for Disease Control and Prevention (2014) National diabetes statistics report: Estimates of diabetes and its burden in the United States. Available at: http://www.cdc.gov/diabetes/pubs/statsreport14/national-diabetes-report-web.pdf.

Deshpande A D, Harris-Hayes M and Schootman M (2008) Epidemiology of diabetes and diabetes-related complications. *J. Phys Ther*. **88**, 1254-1264.

Arun G and Ramesh K (2002) Improvement of insulin sensitivity by perindopril in spontaneously hypertensive and streptozotocin-diabetic rats. *Indian J. Pharmacol*. **34**, 156-164.

Gregg E W and Wang J (2014) Changes in diabetes-related complications in the United States, 1990–2010. *N Engl. J. Med.* **370**, 1514–1523.

Gregory J, Moore D and Simmons J (2013) Type 1 Diabetes Mellitus. *Pediatrics in Review* **34**(5), 203-215.

Grune T, Sommerburg O and Siems W G (2000) Oxidative stress in anemia. *Clic. Nephrol.* **53**(Suppl 1), S18–S22.

Justin M, Gregory D and Moore J (2013) Type 1 Diabetes Mellitus. *Pediatrics in Review* 34 / 5.

Mian L, Moin M and Afzal A (2019) DiabetesRetinopathy In Patients With Chronic Kidney Disease. *Pakistan Armed Forces Medical J.* **69**, 3.

Ozougwu J, Obimba K, Belonwu C and Unakalamba C (2013) The pathogenesis and pathophysiology of type 1 and type 2 diabetes mellitus. *J. Physiol. Pathophysiol.* **4**(4), 46-57. DOI:10.5897/Japap20130.001.

Puspitasari A (2018) Relationship Between Renal Function Test Serum and Lipid Profile in Patients with Diabetes Mellitus. *J. Physics: Conf. Series* **1114**, 012011 doi :10.1088/1742-6596/1114/1/012011.

Sulaiman M (2019) Diabetic nephropathy: recent advances in pathophysiology and challenges in dietary management. *Diabetology & Metabolic Syndromevolume* **11**, 7.

Thomas M, Richard J, MacIsaac C, Tsalamandris M, Goubina I, Fulcher G, Yue D and Jerums G (2004) Anemia in Patients with Type 1 Diabetes. *The J. Clin. Endocrinol. Metab.* **89**, 9, 1 4359–4363, https://doi.org/10.1210/jc.2004-0678.

Turkey endocrinology and metabolism association of diabetes mellitus and its complications diagnosis, treatment and monitoring guide (2017).

Wan B (2019) Relationship between the diabetic retinopathy and renal function in patients with type 1 diabetes. *Int. Eye Sci.* **19**(2), 272-274.