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ORIGINAL ARTICLE

Physiological and Biochemical Effects of Diabetic Ketoacidosis in Hypertensive Male and Female Individuals. A Cross Sectional Comparative Study

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ABSTRACT

The aims and objectives of this study were to observe the physiological and biochemical changes in hypertensive diabetic patients with diabetic ketoacidosis. Dehydration and hypertension in diabetic patients may cause severe diabetic ketoacidosis. A remarkable significant($p<0.05$) changes of random blood glucose, systolic blood pressure, diastolic blood pressure, urine ketone and dehydration levels in Group B and Group C were observed as compared to Group A.

Keywords: Diabetic ketoacidosis, Dehydration, Hypertension systolic blood pressure, diastolic blood pressure, Ketone bodies

INTRODUCTION

When the amount of insulin in human body produce less than the requirement for carbohydrate metabolism to get energy for cellular activities the conditions referred as diabetic ketoacidosis [5]. In diabetes type 2 the complications of diabetic ketoacidosis are less harmful than type 1. The problem of diabetic ketoacidosis started in case of high blood glucose levels and acidic substances increased in the form of ketones up to very dangerous levels in side human body [2]. In case if very low carbohydrate intake in daily food may cause ketosis [3]. Diabetic ketoacidosis is a process in which hepatic cells made fat into energy which releases ketones into the blood and high levels of ketone in the blood are very harmful for human body [4].

Diabetic ketoacidosis is a disorder in which multiple alterations in metabolic pathways are indicated [6]. It has seen in different studies that increased glycogenolysis, increased gluconeogenesis and decreased the utilization of glucose by muscle, fat, liver [10]. The production of ketone bodies, glucose from triglycerides and amino acids are released from the peripheral tissues [8, 7]. In a study it was concluded that high levels of catecholamine, cortisol and glucagon stimulated the production of phosphoenol pyruvate carboxykinase which is a gluconeogenic enzyme [9].

The breakdown of triglyceride into free fatty acids and glycerol occurred in adipose tissues when high levels of catecholamines coupled with insulinopenia and ultimately ketone bodies are formed [5]. Ketonemia indicated by the excess production of β-hydroxybutyrate and acetoacetate. The increased amount of ketoacids are neutralized by intracellular and extracellular buffers [11]. In case of increased levels of ketoacids than the neutralized levels during the process of hydrolysis to develop metabolic acidosis [12]. Different researchers described in their studies that increased levels of ketoacids in the biological system may cause lipid peroxidation and cytokines inflammation as well as C-reactive protein functions inhibition which leads to hyperglycemic crisis [14].

The findings of a study suggested that hypotension and vasodilation indications are associated with increased production of PG_{I2} and PGE_2, which leads to vasodilation and hypotension [16]. Prostaglandin E_2 (PGE_2) and Prostaglandin I2 (PG_{I2}) are local vasodilator hormones. It was also noted by different studies that stimulatory effect of adrenergic agonists is directly proportional to the production of PG_{I2} and PGE_2 in adipose tissue and this concept elaborate the co- rationing of PG_{I2} and PGE_2 with health and disease [15]. Another study concluded the production of $PG_{I2,}$ PGE_2 and diabetic ketoacidosis were increased in adipose tissue because of insulin deficiency.

MATERIALS AND METHODS

This is a cross- sectional comparative study in which 267 male and female individuals were selected and divided them into three groups. 30 female and 37 male normal individuals were in Group A, 70 male and 30 female diabetic non hypertensive individuals were in Group B while 69 male and 31 female diabetic hypertensive individuals were in Group C. The biomarkers such as random blood glucose, systolic blood pressure, diastolic blood pressure, Urine ketone and dehydration levels were measured by glucometer, sphygmomanometer, ketone test strips and on the basis of physical signs and symptoms (PSS) respectively. Raw data comparisons of different groups were processed statistically by mean standard deviation (mean± SD) and significant ($p<0.05$) regression of different parameters through SPSS 2021 comprehensive system for analyzing data.

RESULTS

Table 1: Control, 30 female, 37 male Age: (35-60 years)

Biomarkers	Units	mean± SD	P<0.05
Random blood glucose	mg/dL	120.10±9.11	0.00
Systolic blood pressure	mm Hg	120.01±0.01	0.00
Diastolic blood pressure	mm Hg	80.01±1.03	0.00
Urine ketone	mg/dL	20.10±3.12	0.00
Dehydration	PSS %	01.00±0.01	0.00

Table 2: Diabetic Non- Hypertensive individuals 30 female, 70 male, Age: (35-60 years)

Biomarkers	Units	mean± SD	P<0.05
Random blood glucose	mg/dL	270.10±11.13	0.00
Systolic blood pressure	mm Hg	130.01±0.01	0.00
Diastolic blood pressure	mm Hg	86.01±2.03	0.00
Urine ketone	mg/dL	37.20±11.12	0.00
Dehydration	PSS%	03.01±0.05	0.00

Table 3: Diabetic Hypertensive individuals 31 female, 69 male, Age: (35-60 years)

Biomarkers	Units	mean± SD	P<0.05
Random blood glucose	mg/dL	290.13±14.10	0.00
Systolic blood pressure	mm Hg	170.11±3.01	0.00
Diastolic blood pressure	mm Hg	100.11±12.03	0.00
Urine ketone	mg/dL	87.23±12.16	0.00
Dehydration	PSS%	9.31±2.15	0.00

Among 267 individuals 91 female and 176 male were of age 35-60 years. Diabetic ketoacidosis and hyperglycemia are correlated to dehydration, in this study the Diabetic Non-Hypertensive individuals of Group B showed a remarkable changes in random blood glucose, systolic blood pressure, diastolic blood pressure, Urine ketone and dehydration levels

(270.10±11.13, 130.01±0.01, 86.01±2.03, 37.20±11.12, 03.01±0.05) as compared to control Group A (120.10±9.11, 120.01±0.01, 80.01±1.03, 20.10±3.12, 01.00±0.01). Similarly a significant ($p<0.05$) changes were seen in Diabetic Hypertensive individuals of Group C (290.13±14.10, 170.11±3.01, 100.11±12.03, 87.23±12.16, 9.31±2.15) as compared to Group B and Group A regarding above parameters respectively. Graphically comparative changes in these groups represented in Fig-1

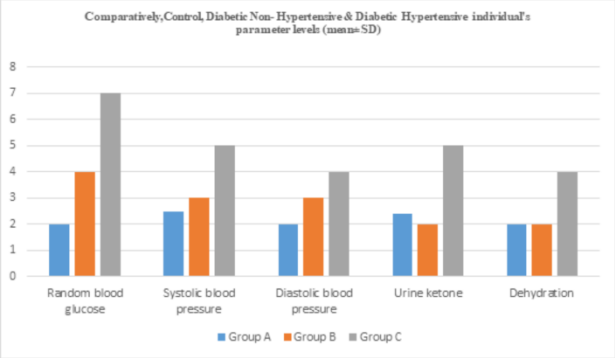

Fig 1:

DISCUSSION

Deeter KH et al., (2011) stated in their study that Starvation ketoacidosis, alcoholic ketoacidosis and diabetic ketoacidosis are metabolic disorders have close association pathologically with high concentrations of acetone, 3-hydroxybutyrate and acetoacetate in both serum and urine. Kuppermann N et al., (2018) suggested that diabetic ketoacidosis is caused by uncontrolled diabetes and it is life-threatening complication in those individuals how have uncontrolled diabetes. Oxidation of free fatty acids enhance the production of ketone bodies in the deficiency of insulin.

The concluded results of Habeeb Ba et al., (2017) study were very close to the present study they stated that the acute complication of diabetes mellitus is diabetic ketoacidosis in which dehydration because of high glucose levels in the body occurred. Ketonimia, acidemia or acidosis and hyperglycaemia all are biochemical metabolic disorders created different complications in the biological system. High levels of serum sodium caused hypertension and dehydration which is very harmful and an indication of cardiac complications.

The results of current study were very close to a study conducted by George JT et al., (2019) in which random blood glucose, systolic blood pressure, diastolic blood pressure, urine ketone and dehydration levels of hypertensive diabetic patients were compared with the non- hypertensive diabetic patients and a significant ($p<0.05$) changes were concluded in the patients of both groups as compared to the control i.e. normal group. It was also concluded from different studies that the chances of diabetic ketoacidosis become increased because of dehydration and hypertension in diabetic patients [19], therefore the findings of present study were similar.

CONCLUSION

Dehydration and hypertension in diabetic patients may cause severe diabetic ketoacidosis. A remarkable significant($p<0.05$) changes of random blood glucose, systolic blood pressure, diastolic blood pressure, urine ketone and dehydration levels in Group B and Group C were observed as compared to Group A.

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