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INFLAMMATORY MARKER IN DIABETES MELLITUS: A CASE CONTROL STUDY IN CENTRAL INDIA

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ABSTRACT

Aim and Objective: Type 2 diabetes mellitus (T2DM) is a heterogeneous group of metabolic disorders characterized by chronic hyperglycemia. In the pathogenesis of diabetes mellitus, it is recognized that chronic low-grade inflammation plays important role. The aim of present study was to evaluate level of inflammatory marker tumor necrosis factor-α (TNF-α) and their correlation with fasting glucose level in type -2 diabetic subjects.

Material and Methods: Total 100 study subjects were divided into two groups, group A comprising 50 apparently healthy age sex matched controls and group B comprising 50 patients of diagnosed type 2 diabetic mellitus**.** Subjects were enrolled after applying all inclusion and exclusion criteria and written informed consent were taken.Inflammatory marker,TNF-α was estimated by enzyme linked immunosorbent assay (ELISA) method.

Results: The mean TNF-α in the control group was 5.34± 1.98pg/mlwhile in the diabetes mellitus group it was 56.90±18.15pg/ml. The difference was found to be statistically significant ($p<0.05$), showing a higher TNF-αin the diabetes mellitus group in comparison to the control group. There is also positive correlation between TNF-α and fasting glucose ($r = 0.617$, $p<0.05$).

Conclusion: There is increased inflammation in diabetic condition as shown by greater elevation of inflammatory marker such as TNF-α. This pathogenic factor might be helpful to a consideration of new therapeutic approach in diabetes mellitus and further development of complications.

Keywords: Diabetes mellitus, tumor necrosis factor-α (TNF-α), marker

INTRODUCTION

The raising trends in the prevalence of diabetes mellitus documented over the last few decades in our country have been alarming. Type 2 diabetes mellitus (T2DM) is a heterogeneous group of metabolic disorders characterized by chronic hyperglycemia[1]. In the pathogenesis of diabetes mellitus, it is recognized that chronic low-grade inflammation and activation of the innate immune system are mainly involved[2-4]. Inflammation also plays important role in the development of insulin resistance[5,6]. Insulin therapy has a breakthrough role in control of chronic hyperglycaemia and also regulates inflammatory process central to diabetes due to its anti-inflammatory property[7].

Cytokines are a group of pharmacologically active, low molecular weight polypeptides[8]. In glucose metabolism, tumour necrosis factor-α (TNF-α) plays a direct pathogenic role[9]. By inducing serine phosphorylation of insulin receptor substrate-1 (IRS-1) and down-regulating the expression of IRS-1, TNF-α inhibits insulin signalling. TNF-α may also decrease the amount of glucose transporting protein 4 (GLUT4) and stimulates the production of interleukin-6 (IL-6)[10].

This study is mainly focusing to evaluate TNF-α level and their correlation with fasting glucose level in type -2 diabetic subjects.

MATERIALS AND METHOD

The study was conducted in M.Y. Hospital and M.G.M. Medical College, Indore includes 100 subjects of age group 45-65 years out of whom 50 were age and sex matched apparently healthy individuals (group I) and50 were diagnosed type 2 diabetic mellitus (group II) according to2006 ADA criteria. The written consent of patients was taken before starting the study and all ethical measures were taken.

The exclusion criteria included patients of diabetes mellitus on insulin therapy, previous history of any chronic disease including kidney or liver disease or any inflammatory disorders to minimize possible confounding of results.

Venous blood (5 ml) sample was withdrawn from the antecubital vein following overnight fasting. The blood sample was collected in clot activator tube and serum was collected. The

serum was analysed forbiochemical investigations on same day and remaining samples were preserved for further biochemical investigations at -20°C.

Fasting plasma glucose (glucose oxidase-peroxidase method) was measured using a biosystemautoanalyzer. Inflammatory marker, TNF-α was estimated by enzyme linkedimmunosorbent assay (ELISA) method in a commercially available kit (Thermofisher, ELISA reader and washer).

The data were expressed as mean ± standard deviation. SPSS version 20 software was used for statistical analysis. Unpaired Student t test was applied to compare TNF-α and fasting blood glucose level between two groups (control and cases). Pearson's coefficient of correlation was determined to assess correlation between TNF-α and fasting blood glucose level. P values less than 0.05 was considered significant.

RESULTS

The table 1 and figure 1 shows the distribution of patients according to sex in both the groups. In the control group, there were 22 females (44.0%) and 28 males (56.0%) whilein the diabetes mellitus group (case) there were 20 female (40.0%) and 30 males (60.0%).

Table 1. Distribution of patients according to sex in both the groups.

Sex	Control	Case
Male	28 (56%)	30 (60%)
Female	22 (44%)	20 (40%)

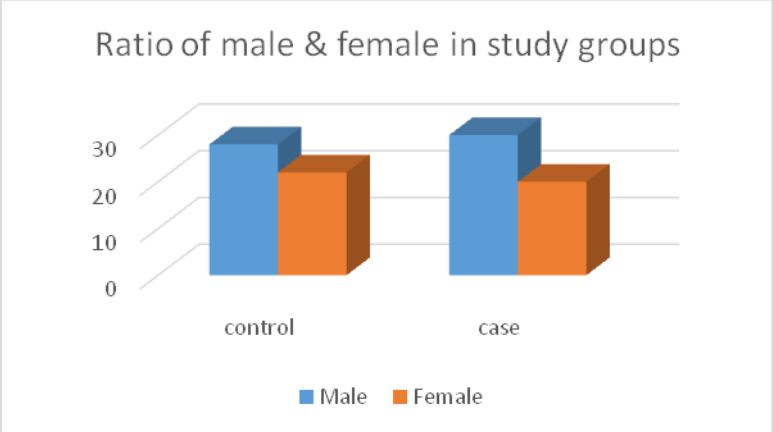

Figure 1. Distribution of patients according to sex in both the groups.

The biochemical profiles of the study subjects are shown in table 2 and figure 2, indicates that subjects with diabetes mellitus had significantly higher levels of fasting blood glucose and TNF-α compared to normal healthy subjects: fasting blood glucose (147.94± 17.88 vs 97.22±17.06) (p<0.05), TNF-α (56.90±18.12 vs 5.34± 1.98pg/ml) (p<0.05).

Table 2.Comparison of various biochemical parameters between case and control groups.

Parameter	Control (n=50)	Case (n=50)	p value
Age (years)	54.28 ± 6.06	54.32 ± 6.06	0.97
Sex (M:F)	28:22	30:20	
Fasting blood glucose (mg/dl)	97.22 ± 17.06	147.94 ± 17.88	<0.05*
TNF-α (pg/ml)	5.34 ± 1.98	56.90±18.15	<0.05*

**p< 0.05 was taken as statistically significant*

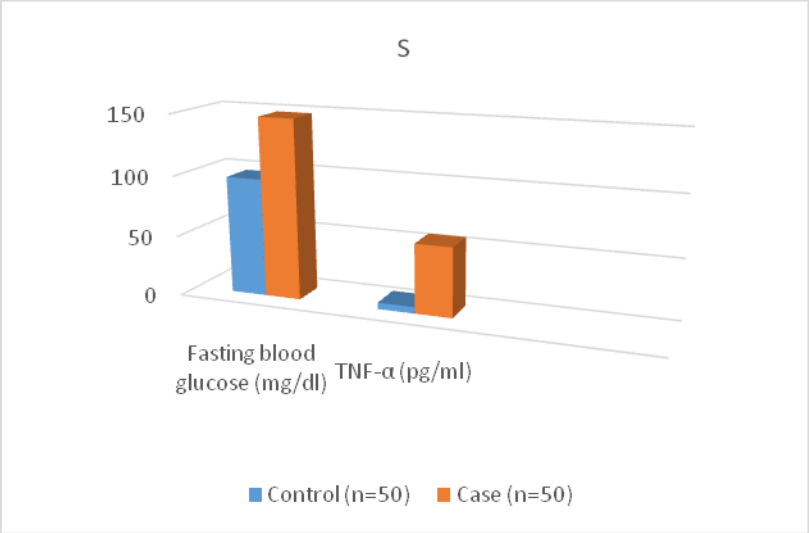

Figure 2. Comparison of parameters between control and case groups.

Table 3 and figure 3 shows Pearson correlation analysis of the inflammatory markers with fasting blood glucose in diabetes mellitus patients. In the total study subjects, TNF-α (r = 0.617, p<0.05), was significantly correlated with fasting blood glucose and showed a positive correlation.

Table 3.Correlation between fasting blood glucose and TNF-αlevels.

Parameter	Fasting blood glucose	
	r value	p value
TNF-α	0.617	< 0.05*

**p< 0.05 was taken as statistically significant*

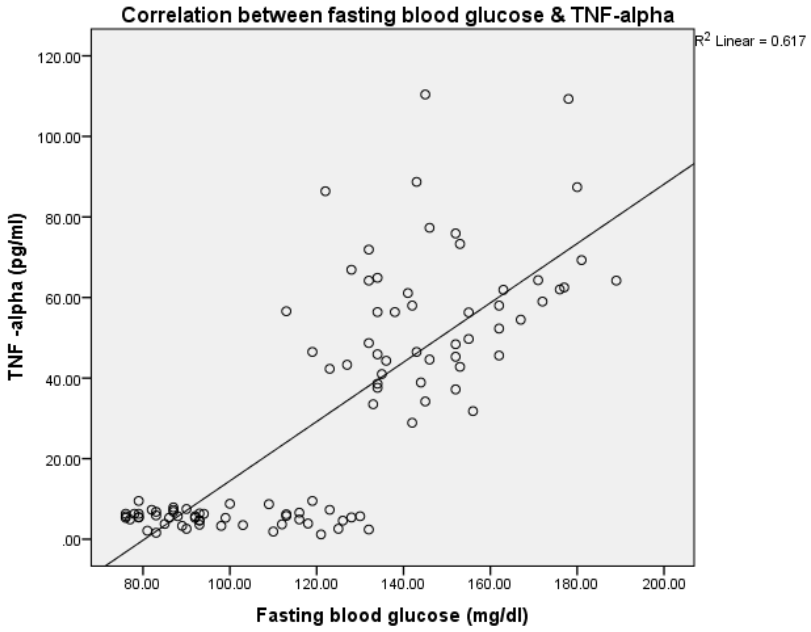

Figure 3. Correlation between fasting blood glucose and TNF-α.

DISCUSSION

The main findings of our study are as follows: (1) level of inflammatory marker, TNF-α is higher in subjects with diabetes mellitus (case) than those without diabetes mellitus (control); (2) there is a positive correlation between TNF-α and fasting blood glucose. Although inflammation has been demonstrated to play an important role in metabolic disorders, the relative importance of the different inflammatory markers in diabetes mellitus needs to be explored further.This study is mainly focusing on the possible role of inflammatory marker TNF-α along with fasting blood glucose in type -2 diabetic subjects.

TNF-α is inflammatory cytokine which was observed extensively in this study. TNF-α is produced by macrophages, lymphocytes and by variety of cell types. After observing the TNF-α calculation, we found a significant increase among case as compared to control with p value less than 0.05 as illustrated in table 2 and figure 2. Our findings are consistent with study of Gopal *et al.,* (2017)[9]. Itis thought to play a major role in the pathophysiology of insulin resistance through the phosphorylation of the insulin receptor substrate-1(IRS-1) protein on serine residues. The inhibition of signalling downstream of the insulin receptor is a primary mechanism through which inflammatory signalling leads to insulin resistance[9].TNF-α a paracrine/autocrine factor plays important role in the development of insulin resistance syndrome as well as β cell failure and type-2 diabetes mellitus[10].

CONCLUSION

Inflammatory pathways playing pivotal roles in the development and progression of diabetes and its complications. There is increased inflammation in diabetic condition as shown by greater elevation of inflammatory marker TNF-α levels.This pathogenic factor might be helpful to a consideration of new therapeutic approach in diabetes mellitus and further development of complications.

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