ORIGINAL ARTICLE

Effects of Diabetes on Heart and Liver in Population of Gujrat

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ABSTRACT

In this study we determine the effects of hyperglycemia on cardiovascular system as well as liver. Diabetes mellitus refers to a group of common metabolic disorders that share the phenotype of hyperglycemia, caused by a complex interaction of genetics and environmental factors. The metabolic dysregulation associated with Diabetes mellitus causes secondary pathophysiologic changes in multiple organ systems with an increasing incidence worldwide, Diabetes mellitus will be likely a leading cause of morbidity and mortality in the future. We have to check the blood glucose, CPK total, CK-MB, LDH and SGOT in diabetics and non-diabetics. Our main focus was on different values of liver enzymes, cardiac enzymes and these values are influenced by glucose levels in body. Then results showed that hyperglycemia has detrimental effects on liver as well as on cardiovascular system.
Keywords: Diabetes mellitus, hyperglycemia, metabolic dysregulation

INTRODUCTION

Several distinct types of Diabetes mellitus are caused by complex interaction of genetics, lifestyle and other related factors[1]. Depending on etiology of Diabetes mellitus, factors contributing to hyperglycemia include reduced insulin secretion, decrease glucose utilization and increase glucose production. Individuals with hyperglycemia are twice as likely to die of cardiovascular disease as those who don't and their risk of an acute myocardial infarction or stroke is three-fold higher[2,3]. Individuals with Diabetes mellitus may also have several forms of dyslipidemia. Because of additive cardiovascular risk of hyperglycemia and hyperlipidemia, lipid abnormalities should be assessed aggressively and treated as part of comprehensive diabetes care. The most common pattern of dyslipidemia is hypertriglyceridemia and reduced HDL cholesterol[4,5]. Diabetes mellitus itself does not increase levels of LDL. But small dense LDL particles found in type 2 Diabetes mellitus are more atherogenic because they are more easily glycated and susceptible to oxidation. Oxidation alterations and modification of lipoprotein are proposed by oxidative hypothesis and increase in atherogenicity occurred by alteration of receptor mediated uptake by cell in intima of blood vessels[6].

The aim of our study is to observe relationship between blood glucose, liver enzymes and cardiac enzymes tests so that we can check whether there are adverse effects of high blood glucose on liver and heart or not. We for this purpose, collected lab reports of patients who are diabetics as well as from those who are not between 30-75 years old females and males[7,8].

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METHOD

our team went to different medical labs where tests of diabetic patients are taken. We went to City Hospital, Gujrat, Doctors Hospital, Gujrat, Aziz Bhatti Shaheed hospital and Malik Haider Hospital lab, to get data of diabetics and non diabetics. Our main purpose is to compare and investigate different values of liver enzymes, cardiac enzymes and if these values were influenced by glucose level in body.

RESULTS AND DISCUSSION

About 300 patients data obtained and analyzed to check the effect of different variables on the diabetes and non-diabetes SPSSa statistical tool is used to analyze the link of diabetes with different cardiac enzymes and liver enzyme. Data has been collected for around 300 patients from city hospital, Malik Haider hospital, Aziz Bhatti Shaheed hospital and Doctors hospital Gujrat. According to the reference values set by WHO for normal fasting blood glucose level that is 90 to 110. 37 males were non diabetic and 134 males were diabetic and 29 females were non diabetic and 100 females were diabetic. According to the results analyzed by the SPSS as shown below in Table 1. It means that the frequency of diabetes in females is more than in males. With the increase of age the chances of type II diabetes also increases.

Table 1: Statistics

Diabetes status			Gender	DM
Male				
Non diabetic	N	Valid	37	37
		Missing	0	0
Diabetic	N	Valid	134	134
		Missing	0	0
Female				
Non diabetic	N	Valid	29	29
		Missing	0	0
Diabetic	N	Valid	100	100
		Missing	0	0

The average mean age of 300 individuals for non-diabetic patient is 47.77 and average mean age for diabetic is 52.1966. The p value of age is 0.017 which shows that with the increase in age the chances of diabetes also increase. The LFTs, blood pressure and the level of different heart enzymes of these diabetic and non-diabetic individuals were checked and compared. According to statistical analysis made from the SPSS the average mean of SGOT in non-diabetic is 3.55 and diabetic are 2.4.

The p value of SGOT is 0.00 that is less than from our alpha value that is 0.05 which shows that there is a strong relationship between diabetes and SGOT. This means that the increase in insulin level also affects SGOT which is a liver enzyme and cause liver abnormalities.

Table 2:

Diabetes Status	N	Mean	Std. Deviation	Std. Error Mean
Age				
Non Diabetic Diabetic	66 234	47.7727 52.1966	12.82881 13.29283	1.57912 .86898
FBG				
Non Diabetic Diabetic	66 234	89.3788 2.0652	24.15639 60.72235	2.97345 3.96954
Cpk total				
Non Diabetic Diabetic	66 234	5.9465E2 3.7891E2	663.79477 374.73027	81.70749 24.49688
CKMB				
Non Diabetic Diabetic	66 234	80.8333 65.7308	118.05143 76.83934	14.53113 5.02314
LDH				
Non Diabetic Diabetic	66 234	4.2864 4.2307	778.0451 187.077	21.015 12.229
SGOT				
Non Diabetic Diabetic	66 234	3.5555 2.4601	216.625 223.880	26.66 14.03

The p value for CpK total is less than 0.05 which shows that the diabetic patients have also increased level of this enzyme. The p values of different variables are shown in the table:

Independent Samples Test

		Levene Test For Equilty Of Variances		T test For Equilty			
		F	Sig.	T	Df	Sig (2 tailed	Mean Difference
Age	Equal Variances Assumed Equal Variances not Assumed	.342	.559	-2.406 -2.454	298 107.576	.017 .016	-4.42385 -4.42385
FBG	Equal Variances Assumed Equal Variances not Assumed	40.334	.000	-15.319 -23.619	298 266.767	.000 .000	-117.14258 -117.14258
CPKtotal	Equal Variances Assumed Equal Variances not Assumed	6.080	.014	3.411 2.529	298 77.037	.001 .013	215.73699 215.73699
CKMB	Equal Variances Assumed Equal Variances not Assumed	1.062	.304	1.238 .982	298 81.139	.217 .329	15.10256 15.10256
SGOT	Equal Variances Assumed Equal Variances not Assumed	.013	.910	.216 .222	298 108.840	.829 .825	5.56799 5.56799
LDH	Equal Variances Assumed Equal Variances not Assumed	.312	.577	3.535 3.601	298 107.345	.000 .000	109.532 109.532

CONCLUSION

Our study has shown that the prevalence of heart diseases and liver damage increases with hyperglycemia. The data collected from different hospital analyzed by SPSS showed the correlation of diabetes type II with different variables. The results have shown that diabetes type II is a disease which damage different organs. In type II diabetes there is an increase in risk of cardiovascular disease and liver damage as most heart and liver enzymes show abnormalities.

REFERENCES

1. Dungan, KM, Buse, JB, Largay, J, Kelly, MM, Button, EA, Kato, S (2006) 1,5-anhydroglucitol and postprandial hyperglycemia as measured by continuous glucose monitoring system in moderately

controlled patients with diabetes. Diabetes Care 29: pp. 1214-9
2. Standards of medical care in diabetes – 2010. Diabetes Care 33 Suppl 1: pp. S11-61
3. Vague, J, Vague, P, Tramoni, M, Vialettes, B, Mercier, P (1980) Obesity and Diabetes. Acta Diabetol Lat 17: pp. 87-99
4. Puigserver, P, Rhee, J, Donovan, J, Walkey, CJ, Yoon, JC, Oriente, F (2003) Insulin-regulated hepatic gluconeogenesis through FOXO1-PGC-1alpha interaction. Nature 423: pp. 550-5
5. Kendall, DM, Sobel, BE, Coulston, AM, Peters Harmel, AL, McLean, BK, Peragallo-Dittko, V (2003) The insulin resistance syndrome and coronary artery disease. Coron Artery Dis 14: pp. 335-48
6. Haffner, SM (2006) Relationship of metabolic risk factors and development of cardiovascular disease and Diabetes Obesity 14: pp. 121S-7
7. Fonseca, VA (2000) Risk factors for coronary heart disease in diabetes. Ann Intern Med 133: pp. 154-6
8. Suzuki, T, Katz, R, Jenny, NS, Zakai, NA, LeWinter, MM, Barzilay, JI (2008) Metabolic syndrome, inflammation, and incident heart failure in the elderly: the cardiovascular health study. Circ Heart Fail 1: pp. 242-8.