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Research Article

SIGNIFICANCE OF BLOOD SUGAR AND GLYCOSYLATED HAEMOGLOBIN LEVELS IN NON ALCOHOLIC FATTY LIVER DISEASE

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ABSTRACT

Non alcoholic fatty liver disease is being increasingly recognised as an important cause of morbidity and mortality particularly in the industrialised world. It is defined by significant lipid accumulation (5-10%) in hepatic tissue in the absence of significant chronic alcohol consumption. NAFLD is histologically similar to alcoholic liver disease, but by definition it occurs in the absence of excessive alcohol consumption and is not due to other identifiable causes of fatty liver such as hepatitis C and certain medications. NAFLD is a metabolic disorder and that Insulin resistance plays a key role in its genesis. Insulin resistance along with other potential biochemical abnormalities results in fatty liver and the generation of excessive free radicals in the liver, which produce liver injury. The present hospital-based, observational, analytical and comparative study was conducted on 80 subjects including 50 cases of ultrasonographically diagnosed NAFLD and 30 age and gender matched healthy subjects as controls. In this study, biochemical profile consisting of fasting blood sugar and glycosylated haemoglobin were evaluated in 50 USG diagnosed cases of NAFLD and 30 controls and the results were analysed statistically. The study found a statistically significant increase in Blood sugar and glycosylated haemoglobin levels in patients of Non alcoholic fatty liver disease indicating the insulin resistance. The present study concluded that increased blood sugar and HbA1C levels are independent risk factors for NAFLD.

INTRODUCTION

NAFLD is characterised by fatty infiltration of the liver, mostly in the form of triglycerides, which exceeds 5% of the liver weight. NAFLD is histologically similar to alcoholic liver disease, but by definition it occurs in the absence of excessive alcohol consumption and is not due to other identifiable causes of fatty liver such as hepatitis C and certain medications. The prevalence of NAFLD has increased in parallel with the epidemics of obesity and type-2 diabetes, which are risk factors for NAFLD.[1] It is now increasingly appreciated that NAFLD is one of the most common causes of liver function abnormalities and liver related morbidity in the western world. NAFLD is a metabolic disorder and Insulin resistance plays a key role in its genesis. Insulin resistance along with other potential biochemical abnormalities results in fatty liver and the generation of excessive free radicals in the liver, which produce liver injury.[2] Insulin resistance is often associated with chronic low-grade inflammation & numerous mediators released from immune cells & adipocytes may contribute liver damage & liver disease progression.[3] Most of the patients with NAFLD are asymptomatic, although some may experience fatigue, malaise or pain in the right hypochondriac region of abdomen.[4] The liver plays a major role in the regulation of carbohydrate metabolism, as it uses glucose as a fuel, it has the capability to store glucose as glycogen and also synthesize glucose from non-carbohydrate sources. This key function of liver makes it vulnerable to diseases in subjects with metabolic disorders, particularly diabetes.[5]

Recent studies indicated that impaired hepatic lipid and lipoprotein settling and increased oxidative stress in liver cells may increase liver fat accumulation and result in insulin resistance, thus increase the hepatic glucose production and export to the peripheral circulation, as a result, raise the level of serum glucose.[6,7,8] The level of HbA1c is influenced by lifespan and "glucose permeability" of the erythrocytes.[9] Based on this point, elevated HbA1c in NAFLD in this study can be stated as the increase of intracellular glucose in NAFLD patients. The other possible mechanism by which HbA1c linked with NAFLD is through oxidative stress. Oxidative stress is a key pathophysiological process responsible for NAFLD.[10] In response to oxidative stress, erythrocytes may undergo morphology change and decreased membrane fluidity, becoming easy to be captured by liver macrophages.[11]

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MATERIALS AND METHODS

The present study was conducted in Department of Biochemistry, Government Medical College, Patiala on 80 cases (including controls) reporting in Department of Medicine, Rajindra Hospital, Patiala from March, 2016 to November, 2016. The present hospital-based, observational, analytical and comparative study was subjected to statistically analysis. The study was approved by ethical committee of institute.

Sample Collection

5 ml of blood sample in EDTA vial was collected under all aseptic conditions by venipuncture. Further this sample was processed for blood sugar and glycosylated haemoglobin.

Investigations

1. Blood Glucose - Glucose Oxidase - Peroxidase Method.[12]

Reference values of Blood Glucose:
Fasting - 70-110 mg/dl
Post Prandial - 100-140 mg/dl.
Random- >140mg/dl

2. Glycosylated Hemoglobin - Glycohaemoglobin -Ion-Exchange Resin [13]

Reference values of Glycosylated Hemoglobin [13]

Interpretation	A.C	(HbA1c)	Mean plasma Glucose Level (mg%)
Normal (Non Diabetic)	4.3-6.22	6-8.3%	57-121
Diabetic ;good control	6.22-6.81	8.3-9%	121-141
Action Suggested	6.81-7.64	9-10%	141-169
Poor Control	>7.64	>10%	>169

RESULT

50 already diagnosed patients of NAFLD and 30 controls were taken and following observations were made from the study.

Table 1 Comparison of blood sugar levels in NAFLD patients and control group

Parameter	Study Group	Mean±S.D (mg%)	p value	Significance
Blood sugar	Case	144.92±21.91	0.00	HS
	Control	87.60±10.94		

Above table showed the comparison of blood sugar levels in NAFLD patients and control group. Mean±S.D. of case group was 144.92±21.91 mg%, while Mean±S.D. of control was 87.60±10.94 mg%. The p value was 0.00. So there was highly significant difference between the case and control groups on account of NAFLD.

Table 2 Comparison of HbA1C levels in NAFLD patients and control group

Parameter	Study Group	Mean±S.D (%)	p value	Significance
HbA1C	Case	6.81±0.51	0.00	HS
	Control	5.62±0.23		

The table above showed the comparison of HbA1C Levels in NAFLD patients and control group. Mean±S.D. of case group was 6.81±0.51%, while Mean±S.D. of control was 5.62±0.23 %. The p value was 0.00. So there was highly significant difference between the case and control groups on account of NAFLD.

DISCUSSION

Insulin resistance and visceral obesity have a major impact on regulatory processes of the (postprandial) lipoprotein and glucose metabolism. It has been demonstrated that a fatty liver is insulin resistant, resulting in an elevated glucose and very low-density lipoprotein production[14] . The level of HbA1c is influenced by lifespan and "glucose permeability" of the erythrocytes. As indicated by a study that transmembrane glucose gradient rather than the rate of glucose transport correlates with the HbA1c. Based on this point, elevated HbA1c in NAFLD in the study can be stated as the increase of intracellular glucose in NAFLD patients.[15]

The present study found higher levels of blood sugar and HbA1C in NAFLD patients. This study resembled Shivaram *et al*[16] 2014, Sharavnan *et al*[17] who found higher levels of blood sugar and Ma *et al*[15] 2014, Sharavnan *et al*[17] 2015 who found higher levels of HbA1C in NAFLD patients.

The link between HbA1c and NAFLD may provide a potential explanation for why NAFLD can be a risk for cardiovascular diseases. Independent association between NAFLD and cardiovascular disease was emphasized in several epidemiological studies. Strategies that aim at decreasing serum HbA1c level in order to reduce risk of cardiovascular disease in NAFLD patients may be taken into account.[15]

CONCLUSION

- Blood Sugar levels significantly increase in NAFLD patients compared to non NAFLD patients.
- HbA1C levels significantly increase in NAFLD patients compared to non NAFLD patients.
- As NAFLD is rapidly becoming an important problem. Undiagnosed, this condition may progress silently and result in cirrhosis, portal hypertension, and liver-related death in early adulthood. The assessment of biochemical profile for liver injury makes the development of non-invasive, readily available, and easy-to-perform markers like blood sugar, HbA1C a high priority for diagnostic purpose.

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