Non-high Density Lipoprotein Cholesterol Levels in Impaired Fasting Glucose


1Department of Biochemistry, SRM Medical College Hospital and Research Center, Tamilnadu, India.
2Department of Medicine, SRM Medical College Hospital and Research Center, Tamilnadu, India

ABSTRACT
Impaired Fasting Glucose (IFG) is defined as elevated fasting plasma glucose concentration ≥100 mg/dl and < 126 mg/dl. IFG has received increasing attention in recent years, because it is an intermediate stage in the development of diabetes and cardiovascular diseases. The assessment of non- High Density Lipoprotein Cholesterol (non-HDL-C) provides a measure of cholesterol contained in all atherogenic particles. We investigated the association between non-HDL-C and fasting plasma glucose levels in 50 patients with IFG in the age group of 20-35 years. The non-HDL-C levels were positively correlated with fasting plasma glucose levels (r=0.07). Measurement of non-HDL-C can be used to assess the cardiovascular risk which may not be accurately identified by estimating only Low Density Lipoprotein Cholesterol levels.

Keywords: Impaired Fasting Glucose, Non-High Density Lipoprotein Cholesterol.

INTRODUCTION
Impaired Fasting Glucose (IFG) and Impaired Glucose Tolerance (IGT) represents intermediate stages of abnormal glucose regulation that exists between normal glucose homeostasis and diabetes (1). IFG is defined as elevated fasting plasma glucose concentration ≥100 mg/dl and < 126 mg/dl (2). IFG has received increasing attention in recent years, because it is an intermediate stage in the development of diabetes and cardiovascular diseases (CVDs) (3,4 and 5). Current guidelines of the third National Cholesterol Education Program-Adult Treatment Panel (NCEP-ATP III) suggest that first line therapy should be directed towards LDL-C lowering (6).

In contrast to LDL-C, non-HDL cholesterol (non-HDL-C) quantifies all atherogenic apolipoprotein-B (apo-B) containing lipoproteins, including low density lipoprotein (LDL), very low density lipoprotein (VLDL), intermediate density lipoprotein (IDL), lipoprotein (a), chylomicrons and chylomicron remnants (7). Thus the assessment of non-HDL-C provides a measure of cholesterol contained in all atherogenic particles. Unlike LDL-C which can be incorrectly calculated in the presence of post prandial hypertriglyceridemia, non-HDL-C is reliable when measured in the non fasting state (8). Diabetic dyslipidemia most commonly manifests as elevated triglycerides and low levels of HDL cholesterol, with a predominance of small dense LDL particles amid relatively normal LDL cholesterol levels. In diabetic patients, non-HDL-C may be a stronger predictor of CVD than LDL-C or triglycerides because it correlates highly with atherogenic lipoproteins (9).

The Health Professionals Follow up study suggested that non-HDL-C may be more strongly associated with Coronary Heart Disease (CHD) risk than LDL-C (10). IFG is considered as a potential indicator of preventive importance for diabetes and CVDs. (11). This study was done to find out the correlation between the non-HDL-C levels and the fasting glucose levels in patients with IFG.

MATERIALS AND METHODS
Fasting blood samples (5ml) were obtained from 50 patients with IFG. All the participants were recruited from the Outpatient Department of our institute, SRM Medical College Hospital and Research Center, Kattankulathur, Kanchipuram district, India. The study was approved by the Institutional Ethical Committee and informed consent was obtained from all the participants. Patients with history of ischemic heart disease, clinical evidence of acute infection, renal and hepatic disease, hypothyroidism, recent surgery/major trauma and patients on lipid lowering and hypoglycemic drugs were excluded from the study.

The following investigations were performed:
1. Fasting plasma glucose – Glucose Oxidase- Peroxidase method, Fully automated analyser (Beckmann Coulter).
2. Total cholesterol and HDL-C - cholesterol oxidase method, Fully automated analyser (Beckmann Coulter).
3. Non-HDL-C was calculated by the formula, Total Cholesterol – High Density Lipoprotein Cholesterol = Non-HDL-C.

The association of non-HDL-C with Fasting plasma glucose was evaluated by using the Pearson’s correlation coefficient.

RESULTS
The age group of the participants ranged from 20-35 years. Table I shows that the non-HDL-C levels were positively correlated with fasting plasma glucose levels (r=0.07).
This association was not found to be statistically significant.

DISCUSSION

Lifestyle modifications have resulted in an increased prevalence of dysglycemia in young individuals. A graded relationship between plasma glucose and cardiovascular risk is observed in non-diabetic individuals with high glucose levels that are below the diabetic cut-offs (12,13). Hyperglycemia is often the consequence of insulin resistance and is commonly accompanied by an atherogenic dyslipidemic profile, altered fibrinolysis and inflammatory profile (14).

Further reduction in insulin secretion over time results in increasing glycemia and the development of diabetes which in turn is associated with the development of microvascular and macrovascular complications (1). IFG is thus considered as a potential indicator of preventive importance for diabetes and CVDs. (11). Assessment of non-HDL-C provides a measure of cholesterol contained in all atherogenic particles. In this study, a positive association was observed between non-HDL-C and fasting glucose levels. This association was not found to be statistically significant due to the smaller sample size. Liu et al (15) showed that CHD risk in those with diabetes did not increase with increasing LDL-C whereas it increased with increasing non-HDL-C. The Emerging Risk Factor Collaboration (ERFC) Group evaluated people without initial vascular disease and found that non-HDL-C and apo-B predicted CHD to a similar extent (16). Currently, NCEP-ATP III guidelines recommend non-HDL-C as a secondary target of therapy in persons with triglycerides &gt;200 mg/dl. The target for non-HDL-C is 30 mg/dl higher than the target for LDL-C (6). The major limitation of this study is the small sample size. Evaluation of non-HDL-C may help in better risk stratification of dysglycemic individuals and thereby reduce the long term increased risk of CHD.

REFERENCES


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