INTRODUCTION
Accumulation of excess lipids can produce liver injury and non-alcoholic fatty liver disease (NAFLD) which is a common disease. NAFLD have associated metabolic syndrome (MS)-a clustering of cardiovascular risk factors like obesity, diabetes, hypertension and dyslipidemia. It is estimated that about a quarter of the adult American population, currently has fatty liver and about 4% have fatty liver disease and about half of the NAFLD is associated with MS nevertheless the same is not known in Indian population. Recently the role of adipose tissue as an endocrine became more evident. Adipose tissue evolved is the largest and versatile endocrine organ in human body. Major adipokines (cytokines and hormones from adipocytes) include adiponectin, leptin, resistin, TNF-α and visfatin. All of them play an important role in lipid and carbohydrate metabolism. The genetic profile and lifestyle of an individual determine the initiation and progress of NAFLD. But we know little how these factors act to make an apparently healthy individual an NAFLD patient. Lipid and carbohydrate metabolism is deranged in NAFLD. Since adipokines play an important role in metabolism we studied the adipokine profile in NAFLD patients.

Adiponectin is the most abundant and perhaps the most important adipokine which has multiple roles from energy homeostasis to angiogenesis to immunity. So the mutations in the adiponectin gene were studied and correlated with the severity of NAFLD and progression. The protein coding and regulatory regions of the adiponectin gene were selected for the study of the mutations.

The current study is important in the understanding of the pathogenesis and possibly the management of NAFLD, a major global health concern.