Non alcoholic fatty liver disease (NAFLD) is a fatty liver disease occurring in patients without alcohol consumption. It includes a broad spectrum of liver diseases, from fatty infiltration, inflammation and fibrosis, to cirrhosis, usually having obesity, hyperlipidemia and diabetes mellitus as its etiology. Non alcoholic fatty liver disease (NAFLD) also describes a clinicopathological condition that is characterized by significant lipid deposition in the hepatocytes of the liver parenchyma in patients with no history of excessive alcohol consumption. The spectrum of this disease is broad, ranging from a simple steatosis to non alcoholic steatohepatitis, fibrosis and cirrhosis. Obesity and diabetes are well known risk factors for the development of a fatty liver. The unopposed lipolysis in the adipose tissue caused by insulin resistance in the adipocytes leads to accumulation of lipids within the liver and adipose tissue insulin resistance is positively correlated with liver fat content in type II diabetic obese patients.

Insulin resistance is thought to be the key factor in the development of hepatic steatosis due to the important action of insulin on skeletal muscle, adipocytes and the liver—all important organs in maintaining glucose and lipid homeostasis. Actions of insulin includes increased uptake of FFA, conversion to and storage as triglycerides and decreased lipolysis in adipose tissue, uptake of glucose by skeletal muscles, storage of glucose as glycogen in liver with inhibition of glycogenolysis and gluconeogenesis, increased de novo lipogenesis and reduced oxidation of fatty acids in the liver. The net result is to utilize glucose, reduce FFA
and promote storage of fats as triglycerides. Chronic hypertriglyceridemia is associated with the long term-consequences of diabetic obese that include damage and dysfunction of the cardiovascular system, retinopathy, nephropathy, neuropathy and polycystic ovary.

Therefore the present study was aimed to know the relationship between biochemical parameters with severity and early onset in non alcoholic fatty liver disease type II diabetic obese patients.

The study was conducted in 500 human subjects out of them 100 were normal healthy individuals (group I), 400 were from nonalcoholic fatty liver obese type II diabetic subjects which further sub divided into four groups according to BMI (body mass index): 74 were nonalcoholic fatty liver obese type II diabetic subjects (BMI 25 to 30- group II), 211 were nonalcoholic fatty liver obese type II diabetic subjects (BMI 30 to 35- group III), 75 were nonalcoholic fatty liver obese type II diabetic subjects (BMI 35 to 40- group IV), 40 were nonalcoholic fatty liver obese type II diabetic subjects (BMI more than 40- group V) subjects. The blood sample was analyzed for biochemical parameters in fasting state. Physiological parameters were also recorded.

The blood sample was analysed for the following parameters:-

1. Liver function tests
   
   Serum total Bilirubin
Summary

Alanine transaminase (ALT)

Alkaline phosphatase (ALP)

Gamma- Glutamine transpeptidase (GGT)

2. Fasting Blood Sugar

3. Lipid Profile

   Triglyceride (TG)

   Total Cholesterol (TC)

   High Density Lipoprotein (HDL-c)

   Low Density Lipoprotein (LDL-c)

   Very Low Density Lipoprotein Cholesterol (VLDL-c)

4. Fasting Insulin Level

5. High Sensitive C-Reactive Protein (hs-CRP)

SPSS 21, Chicago, Illinois, USA was used for all statistical analysis. All parameters were given as mean± standard deviation (S.D.). The comparisons between two groups were analyzed by Student's t-test. Correlations between variables were analyzed by Pearson’s correlation coefficient test $P < 0.05$ and $P < 0.01$ was considered significant in nonalcoholic fatty liver obese type II diabetic subjects.
The results of blood analysis showed that there were significantly higher levels of FBS, lipid profile, liver function enzymes, insulin resistance while HDL-c was significantly lower but no significant changes occurs in total bilirubin and hs-CRP in NAFLD subjects group II (BMI 25 to 30) as compared to group I healthy control subjects in both male and female subjects.

In our study, we found that in NAFLD subjects group III (BMI 30 to 35) all the biochemical parameters were significantly elevated except HDL-c but no significant changes occurs in total bilirubin as compared to healthy control subjects group I in both male and female subjects.

In NAFLD subjects group IV (BMI 35 to 40) all the biochemical parameters were significantly elevated except HDL-c as compared to healthy control subjects group I in both male and female subjects.

In this study we found NAFLD subjects group V (BMI more than 40) all the biochemical parameters were highly significantly (p<0.001) elevated except HDL-c as compared to healthy control subjects group I in both male and female subjects.

In group III, IV and V, fasting Insulin (HOMA-IR) and hs-CRP were found significantly elevated. It may be due to fatty infiltration, inflammation and fibrosis to cirrhosis, usually having metabolic syndrome-obesity, hyperlipidemia, hypertension and diabetes mellitus.
The results of correlative study in NAFLD subjects mainly showed that triglyceride was positively correlated with liver function enzymes, fasting insulin, HOMA-IR, hs-CRP and duration of diabetes. These results suggested that triglyceride may be an independent risk factor for the development of fatty liver in NAFLD type II diabetic subjects.

In this study we found that there was significantly positive correlation between BMI, fasting insulin (HOMA-IR), hs-CRP, duration of diabetes mellitus in non alcoholic fatty liver disease subjects.

From this study, it is observed that persistent hypertriglyceridemia is the main root cause of developing micro and macro vascular complications in nonalcoholic fatty liver disease type II diabetic obese patients in group II, III, IV, V subjects. Development of insulin resistance with increasing age could be one of the causative factors for non alcoholic fatty liver disease.

In this study we observed that females are more prone to non alcoholic fatty liver disease (NAFLD) as compared with male group due to the significant fat (hypertriglyceridemia) storage in subcutaneous tissues (adipose tissues).

From this study it is concluded that insulin resistance, persistent chronic hypertriglyceridemia, long standing of disease and advancing age in uncontrolled diabetic obesity causes simple steatosis to non alcoholic steatohepatitis, fibrosis and cirrhosis.
Insulin resistance leads to micro and macro-vascular complication in nonalcoholic fatty liver disease type II diabetic obese subjects. Therefore the higher values of HOMA-IR, attributed to the fact that these patients had more risk for complications.

Therefore non alcoholic fatty liver disease (NAFLD) patients should be counseled to start to lose weight at the "earliest possible time" and ameliorate their lipid profile particularly among obese females.

It is possible that in coming years the hope of new therapeutic strategies based on low triglyceride level and improves fasting insulin, hs-CRP and liver function enzymes properties with beneficial actions on nonalcoholic fatty liver disease type II diabetic obese complications can be translated into real clinical treatments.