A STUDY ON

"EFFECTIVENESS OF INDIAN DIABETES RISK SCORE AS A SCREENING TOOL FOR NON ALCOHOLIC FATTY LIVER DISEASE AMONG NON DIABETICS - AT TERTIARY CARE HOSPITAL IN SOUTH INDIA."

Dissertation submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMIL NADU

In partial fulfilment of the regulations for the award of the degree of

M.D.BRANCH -I (GENERAL MEDICINE)

REGISTRATION NUMBER : 201911059

DEPARTMENT OF GENERAL MEDICINE GOVERNMENT

STANLEY MEDICAL COLLEGE CHENNAI

MAY - 2022

CERTIFICATE

This is to certify that this dissertation entitled **"EFFECTIVENESS OF INDIAN DIABETES RISK SCORE AS A SCREENING TOOL FOR NON ALCOHOLIC FATTY LIVER DISEASE AMONG NON DIABETICS - AT TERTIARY CARE HOSPITAL IN SOUTH INDIA."** submitted by **Dr. S. HARSHINI** to the faculty of General Medicine, The Tamil Nadu Dr. M.G.R Medical University, Chennai, in partial fulfilment of the requirement for the award of M.D Degree Branch-I (General Medicine) is a bonafide research work carried out by her under direct supervision and guidance.

GUIDE

Prof. S. KALAICHELVI, M.D.,

Unit Chief,
Department of General Medicine,
Stanley Medical College & Hospital,
Chennai.

HOD

Prof. S. CHANDRASEKAR, M.D.,

Head of the Department,
Department of General Medicine,
Stanley Medical College & Hospital,
Chennai.

Prof. P. BALAJI M.S., FRCS., Ph.D., FCLS.,

Dean,

Government Stanley Medical College and Hospital,

Chennai.

DECLARATION

I solemnly declare that the dissertation titled **"EFFECTIVENESS OF INDIAN DIABETES RISK SCORE AS A SCREENING TOOL FOR NON ALCOHOLIC FATTY LIVER DISEASE AMONG NON DIABETICS - AT TERTIARY CARE HOSPITAL IN SOUTH INDIA."** is a bonafide work done by me at Government Stanley Hospital, Chennai between March 2021 and September 2021 under the guidance and supervision of **Prof. S. Kalaichelvi M.D.** I also declare that this bonafide work or a part of this work was not submitted by me or any other forward degree or diploma to any other university, board either in India or abroad. This dissertation is submitted to the Tamilnadu Dr. M.G.R Medical University, towards the partial fulfillment of requirement for the award of M.D. Degree (Branch – I) in General Medicine.

Place: Signature of the Candidate

Date:

(Dr.S.HARSHINI)

Reg.No:201911059

CERTIFICATE – II

This is to certify that this dissertation work titled **"EFFECTIVENESS OF INDIAN DIABETES RISK SCORE AS A SCREENING TOOL FOR NON ALCOHOLIC FATTY LIVER DISEASE AMONG NON DIABETICS - AT TERTIARY CARE HOSPITAL IN SOUTH INDIA."** of the candidate **Dr.S.Harshini** with Registration Number **201911059** for the award of **M.D. DEGREE** in the branch of **BRANCH-I (GENERAL MEDICINE).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains, from introduction to conclusion pages and result, shows **1%** percentage of plagiarism in the dissertation.

Guide & Supervisor Sign with Seal

SPECIAL ACKNOWLEDGEMENT

I gratefully acknowledge and thank **Prof. P. BALAJI M.S., FRCS., Ph.D., FCLS.,** Dean Government Stanley Medical College and Hospital, Chennai

For granting me permission to utilize the resources of this Institution for my study.

ACKNOWLEDGEMENT

"Gratitude is the fairest blossom which springs from the soul"-Henry Ward Beecher

I would like to express my humble gratitude to The Head of the Department **Prof.Chandrasekar M.D.,** sir for guiding and accepting the study.

My sincere thanks to my guide **Prof.A.Kalaichelvi M.D.,** Chief of Medicine for her vital guidance and support for the successful completion of my dissertation.

My special thanks to my beloved unit Assistant professors **Dr.K.Lakshmi M.D** and **Dr.R.Vijayanand M.D** and all the faculty of the Department of Medicine for their valuable support throughout the study.

My sincere gratitude to the patients and their attenders for the cooperation aiding in the successful conduct of my study.

Most importantly I am ever so grateful to God and my family for guiding me in all my endeavours and always being my greatest pillar of support. A special thanks to my friends for all their help.

ABBREVATIONS

NAFLD	Non-Alcoholic Fatty Liver Disease
NASH	Non-Alcoholic Steatohepatitis
IDRS	Indian Diabetes Risk Score
BMI	Body Mass Index
FBS	Fasting Blood Sugar
SHT	Systemic Hypertension
SBP	Systolic Blood Pressure
DBP	Diastolic Blood Pressure
TGL	Triglyceride
TC	Total Cholesterol
HDL	High Density Lipoprotein
LDL	Low density Lipoprotein
WHR	Waist-Hip Ratio
AST	Aspartate Amino Transferase
ALT	Alanine Amino Transferase
GGT	Gamma Glutamyl Transferase
ALP	Alkaline Phosphatase
USG	Ultrasonography
MRI	Magnetic Resonance Imaging
MRE	Magnetic Resonance Elastography
PCOS	Polycystic Ovarian Syndrome
CKD	Chronic Kidney Disease

TLR	Toll Like Receptors
FXR	Farnesoid X Receptor
HOMA	Homeostatic Model Assessment
IR	Insulin Resistance
THV	Terminal Hepatic Venule
RNA	Ribonucleic Acid
DNA	Deoxyribonucleic Acid
SIBO	Small Intestinal Bacterial Overgrowth
PDFF	Proton Density Fat Fraction
PPAR	Peroxisome Proliferator- Activated Receptors
OGTT	Oral Glucose Tolerance Test
PUFA	Polyunsaturated Fatty Acid
MUFA	Monounsaturated Fatty Acid
SNP	Single Nucleotide Polymorphism
PNPLA3	Patatin-like Phospholipase domain-containing Protein 3
ASBT	Apical Sodium Dependent Bile acid Transporter

TABLE OF CONTENTS

INTRODUCTION

NAFLD (non-alcoholic fatty liver disease) is a wide range of liver disorders ranging from steatosis, non-alcoholic steatohepatitis and cirrhosis. NAFLD which develops in absence of alcohol abuse has been considered as a major health burden. In western countries, there is a greater prevalence of NAFLD due to epidemics of obesity and related metabolic complications. NAFLD is replacing alcoholic and viral hepatitis as the most common aetiology of chronically elevated liver enzymes in developing countries. Type 2 diabetes mellitus, obesity, dyslipidemia, hypothyroidism, PCOS are considered as risk factors. Insulin resistance is also frequently reported in NAFLD. The IDRS (Indian diabetes risk score) is an established screening tool for diabetes using four simple parameters namely age, abdominal obesity, family history of diabetes mellitus and physical activity. It has been classified as low (<30), Medium (30-50) and high (>60) risk categories. This study is to ascertain whether IDRS can be used as a cost effective initial screening tool for NAFLD.

AIM AND OBJECTIVES OF THE STUDY

AIM OF THE STUDY:

Study of effectiveness of Indian diabetes risk score as a screening tool for non-alcoholic fatty liver disease among non-diabetics.

OBJECTIVES OF THE STUDY:

PRIMARY OBJECTIVES:

- To determine IDRS among non-diabetics.
- To assess the risk of NAFLD among non-diabetics using Indian diabetes risk score.
- To correlate Indian diabetes risk score with ultrasonographic evidence of fatty liver among non-diabetes.

SECONDARY OBJECTIVES:

- To correlate the BMI, waist circumference, blood pressure, FBS, HBA1C, lipid profile and liver function tests results with the IDRS score.
- To correlate the BMI, waist-hip circumference ratio, blood pressure, FBS, HBA1C, lipid profile and liver function tests results with the ultrasonographic findings.

REVIEW OF LITERATURE

Non-alcoholic fatty liver disease (NAFLD) is increasingly becoming as one of the most common etiology of chronic liver disease. It is one of the important causes of cirrhosis and hepatocellular carcinoma. NAFLD is a spectrum of liver diseases and is not a single entity. This spectrum of pathological liver disease include simple steatosis or non-alcoholic fatty liver, non-alcoholic steatohepatitis, cirrhosis and their complications, in the absence of excessive consumption of alcohol (threshold of less than 30 grams per day in men and less than 20 grams per day in women). As NAFLD patients have insulin resistance, visceral adiposity and /or hypertension, type 2 diabetes mellitus, hypertriglyceridemia and hypercholesterolemia, it is considered as hepatic manifestation of metabolic syndrome.(1–4)

EPIDEMIOLOGY

Prevalence estimates vary greatly based on the information in the population given and the diagnostic criteria used to demonstrate the diagnosis i.e. liver function biochemical parameters, ultrasonic findings and liver biopsy. The global prevalence of NAFLD is not completely defined because majority of patients are asymptomatic, however a global prevalence of 25 percent(2) was shown by the meta- analysis carried in 2016. Increased rates of NAFLD and NASH have been observed in obese patients, this had been confirmed

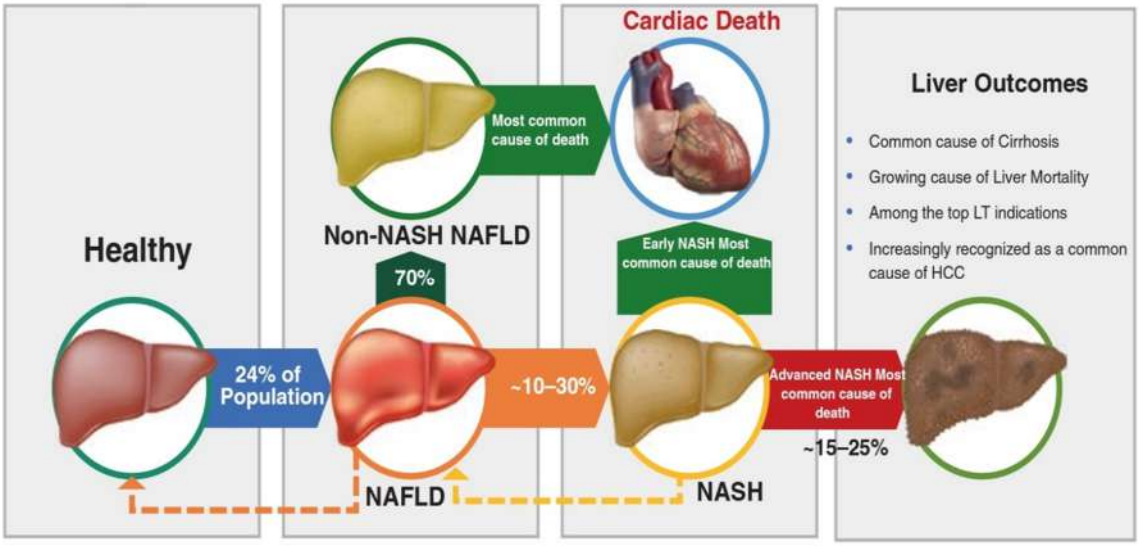
subsequently in studies with patients undergoing bariatric surgery where the frequency of NASH and NAFLD was said to be 37 percent and 91 percent respectively.(5)

Most cases have been diagnosed in the middle age that is in fourth to sixth decade; however there has been rising increase in frequency among the adolescents and children with overweight and obesity, the prevalence of which was found to be 30%.(6) NAFLD has been reported common in men more than women but with a peaking late in women than men which is probably suggesting relationship with menopause and sex hormones.(7,8)

NAFLD (especially NASH) has high association with type 2 diabetes mellitus with prevalence percentage of 60 to 76 for NAFLD and 22 percent for NASH.(9) The role for ethnicity comes from Dallas heart study which showed ethnicity has an association with highest prevalence among the Hispanics. Lifestyle also plays a role. Sedentary lifestyle and consumption of high sugar containing sodas was associated with increased rates of NAFLD. Genetic factors such as single nucleotide polymorphism (SNP) of certain specific genes were found to have association with risk of NAFLD. Patatin like phospholipase domain containing protein 3 (PNPLA3) gene was one of the first SNPs identified to have increased association with hepatic steatosis. This was common among Hispanics followed by Caucasians, which explains the increased prevalence among Hispanics. The interplay between host and genetic factors leads to development of steatosis and steatohepatitis.(10)

NATURAL HISTORY OF NAFLD

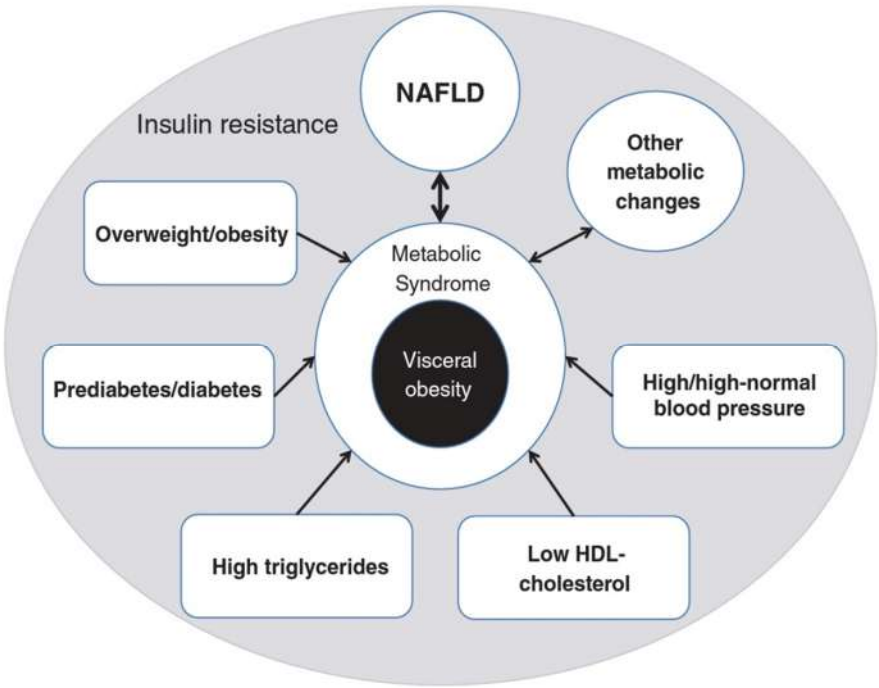
The natural course of NAFLD varies and fluctuates. Underlying risk factors influence the progression of the disease. Average rate of progression from one disease to another is about 7.7 years.(11) Some patients progress faster, particularly those with diabetes mellitus, older age, visceral obesity and Hispanic ethnicity.(12–14)



OBESITY AND NAFLD.

Obesity is the accumulation of body fat comes from positive energy balance, independent of the calorie intake and expenditure of energy by physical activity. Adipocytes protect tissues and organs during over nutrition and serve as reserve energy source during under nutrition. Apidocytes regulate food intake by secreting leptin which acts on hypothalamus. Adiponectin is a hormone that counteracts leptin and is found to be reduced in obesity. Although unhealthy

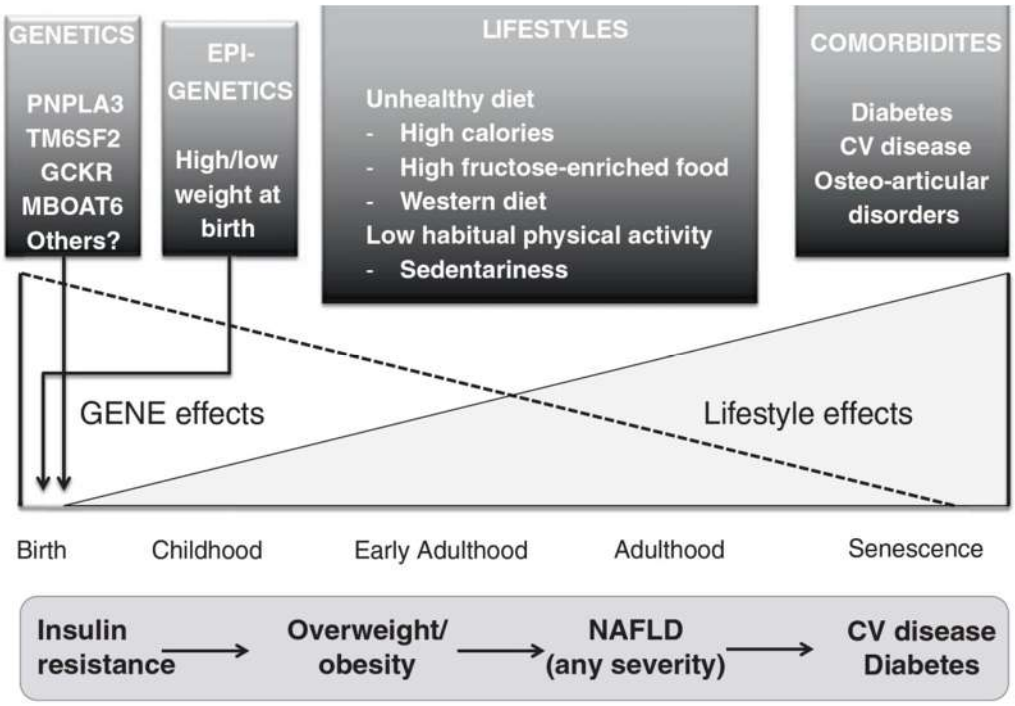
lifestyle regulates the total body fat, mutations in the genes for receptors for leptin and adiponectin can cause changes in their levels and increase in body fat accumulation. The anti-steatotic potential of the adipocyte is exceeded by the fatty aid recirculation and lipotoxic disease starts to develop which is marked by the fatty infiltration of non-adipose tissues. The adipose tissue secretes pro-oxidant substances and pro-inflammatory cytokines which cause insulin resistance on lipid and glucose metabolism. This leads to group of metabolic changes called metabolic syndrome.(15)



Enlargement of adipose tissue along with insulin resistance occurs following unhealthy diet and low physical activity , and there is a increased risk of hepatic lipid deposition due to both de novo lipogenesis and lipolysis. Diets rich in saturated fatty acids causes increase in liver fat whereas intake of PUFA and

MUFA tend to decrease liver fat. Fructose rich diet favors steatosis because it is metabolized mainly by the liver.

Genetic factors also play a role in body fat distribution. Gene polymorphism like PNPLA3 allele has been associated with hepatic inflammation and increase in hepatic fat levels. Other genes that are found to increase the risk are MBOAT7, TM6SF2, GCKR, and MERTK.(15)

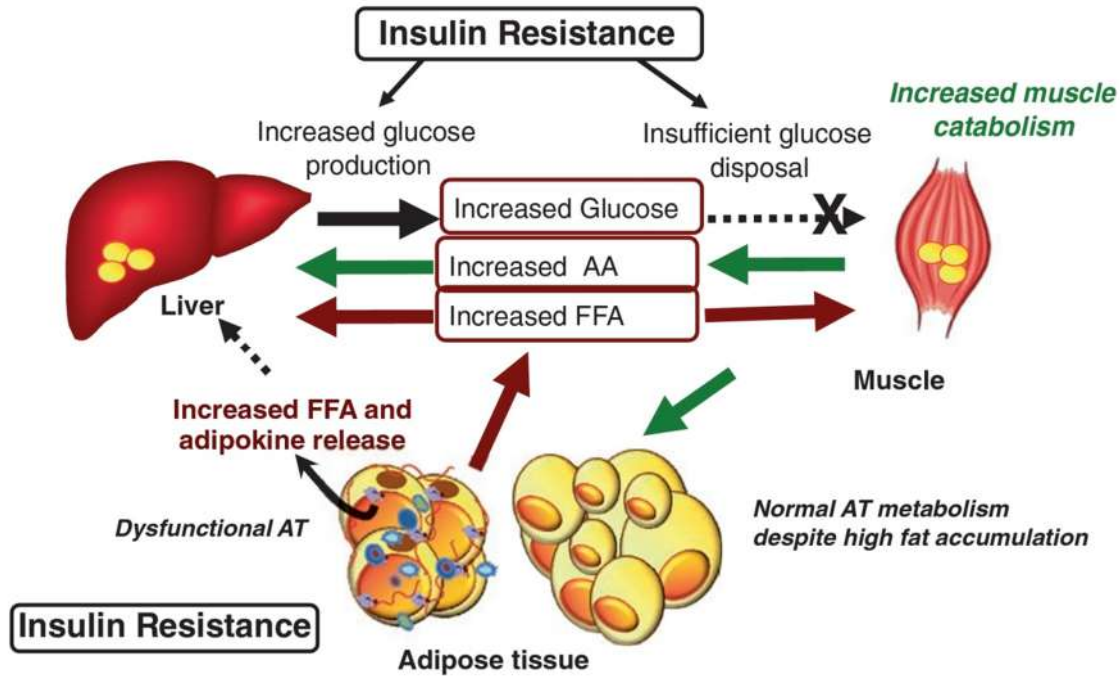


NAFLD AND INSULIN RESISTANCE.

Insulin resistance (IR) is the inability of known amount of insulin (endogenous or exogenous) to stimulate metabolism of glucose in various organs especially in adipose tissue, liver and muscle. Insulin effects on lipid and protein metabolism are:

1. Stimulates lipogenesis and inhibits lipolysis.
2. Stimulates protein synthesis and inhibits protein catabolism.

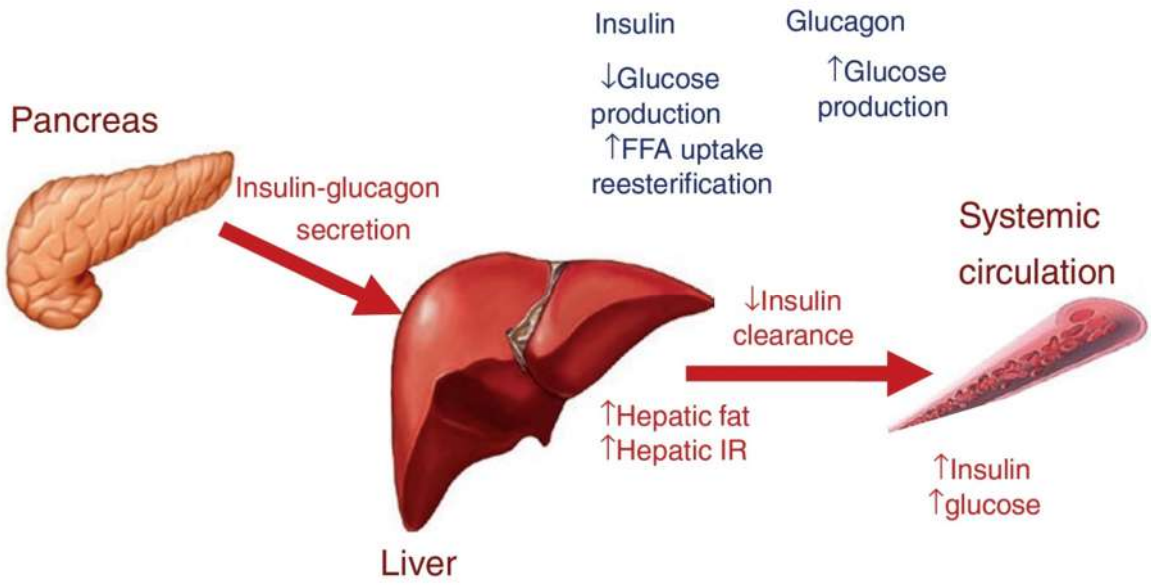
IR causes impairment of both its catabolic and anabolic effects on glucose, lipid and protein. With IR there is increased diversion of fatty acids to the liver which in turn raises the risk of NAFLD.



Maximum insulin secreted is degraded by the liver and partly by muscle and kidney. While liver does not degrade the c-peptide and it is excreted majorly by the kidney. Due to this it is being used to calculate prehepatic insulin secretion and insulin clearance. The main function of insulin is to have endogenous glucose production suppressed and promote glucose storage in liver, muscle and to promote synthesis of glycogen. The liver serves as a modulator of insulin concentration in peripheral tissue whereby it decreases the clearance of insulin

when there is a peripheral insulin resistance owing to the increased demand for insulin. Therefore hyperinsulinemia occurs when there is insulin resistance.

Glucagon on the other hand as effects opposite to that of insulin which is secreted by alpha cells of pancreas. In NAFLD, the concentrations of glucagon are increased which in turn contributes to increased endogenous glucose products and cause further hepatic insulin resistance.



IR can be assessed several ways in vivo. Though there are several indices, euglycemic hyperinsulinemic clamp is the gold standard method where infusion of insulin in pharmacological amounts along with infusion of glucose to maintain a constant plasma concentration of glucose. The indices can be either based on their fasting measurements or based on OGTT measurement. The fasting indexes are QUICKI and HOMA-IR; they are derived based on the product of fasting insulin and glucose concentration. As they are measured after an overnight fasting, it has been used commonly. The cutoff of HOMA-IR is 2.0

for NAFLD. However the more predictable indexes are the ones where the concentrations of insulin and glucose are measured during the OGTT. The most used indexes are OGIS index and Matsuda index.

In diabetic patients, infusion of tracer or hyperinsulinemic euglycemic clamp would give a reliable measurement of peripheral insulin resistance because in these patients glucose concentration are altered due to impaired insulin secretion rather than insulin resistance.

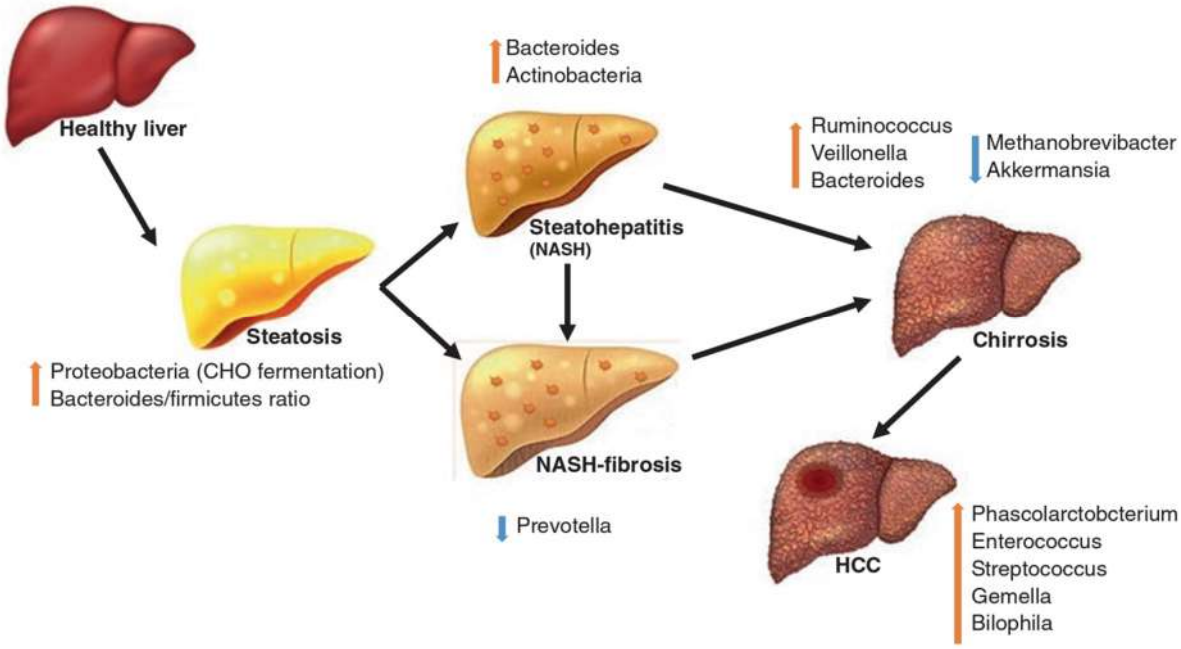
Hepatic insulin resistance is defined as the inability of insulin to suppress endogenous glucose production during fasting and/or during insulin infusion. Hepatic IR is associated strongly with the lipid accumulation in liver. There is proportional increase in fasting hepatic IR to the extent of hepatic steatosis. In NAFLD subjects, there is lipotoxicity which deranges metabolic signaling which leads to metabolic alteration of lipid and glucose and impaired secretion of insulin. There is production of lipotoxic products due to partial hydrolysis of triglyceride like diacylglycerol and ceramides which in turn leads to insulin resistance by activation of various signaling pathways. Therefore glucotoxicity, lipotoxicity and insulin resistance play a major role in hepatic IR.(16)

ETIOPATHOGENESIS OF NAFLD.

A. GUT DYSBIOSIS.

A critical factor in the NAFLD pathogenesis is intestinal microbiota. Gut dysbiosis is a modification of microbiome and promotion of microbial products and microorganism translocation into the portal circulation, where it can activate liver macrophages to produce pro-inflammatory cytokines causing liver damage. With the alteration in gut microbiota, there is increase in short -chain fatty acid due increased fermentation of carbohydrates and alongside stimulation of triglyceride synthesis de novo in the liver. Choline metabolism is also altered in gut dysbiosis in NAFLD patients, which is important for hepatic lipid export and synthesis of very low density lipoprotein.

Diets rich in animal protein, simple sugars and fat promote *Bacteroides* growth, while diets enriched in vegetable carbohydrates and fibers favor abundance increase in *Prevotella. Ruminococcus* genus was found to be strongly associated with liver fibrosis. *Ruminococcus* ability to produce alcohol by fermenting complex carbohydrates is also responsible for liver injury.



MICROBIOTA IN NAFLD AT DIFFERENT STAGES OF DISEASE.

B. **INTESTINAL PERMEABILITY**.

The intestinal permeability and barrier integrity is influenced by the intestinal microbiota. A causal relationship between NASH and small intestinal bacterial overgrowth (SIBO) has been established. SIBO causes alteration in gut permeability by damaging the tight junctions in the intestine which causes liver to get exposed to inflammatory cytokines like tumor necrosis factor -α, interleukin 1-α and pro- inflammatory cytokines like lipopolysaccharide, both leads to dysregulation of inflammatory activity and hepatic injury.

Together with intestinal dysbiosis and increased permeability of the intestine, there is translocation of microbial products and microorganism. The microbial associated molecular patterns get recognized by the receptors on stellate and kupffer cells through pattern recognition receptors and cause inflammation.

Various Toll like receptors (TLRs) get activated by the cell wall components like lipopolysaccharides and DNA material which subsequently causes liver damage by activation of inflammatory cascade.

C. BILE ACID METABOLISM.

In human the primary bile acids secreted are cholic acid and chenodeoxycholic acid. Before bile is excreted, these are conjugated to major extent with glycine and lesser with taurine and released after meal into the duodenum. In the distal ileum there is reabsorption of these conjugated bile acids by the apical sodium dependent bile acid transporter (ASBT) and recirculated through enterohepatic circulation via portal blood to the liver.

The nuclear farnesoid X receptor (FXR) regulates the synthesis of bile acid whereas microbiota influences bile acid excretion and production. Active reuptake of bile acid via ASBT in small intestine is prevented by deconjugation of bile acid by microbial bile salt hydrolases. These deconjugated bile acids are converted to secondary bile acids through a 7α-dehydroxylation by the microbiota. Desoxycholic acid and lithocolic acid are reabsorbed by passive diffusion as they are hydrophobic. This small proportion which enters the enterohepatic circulation acts as signaling molecules.

There is a shift of balance between the primary and secondary bile acids due to bacterial overgrowth, favoring the secondary bile acids. The two classes of bile acid has differences in affinity for FXR, the secondary bile acids causes an overall increase synthesis of bile acid by liver by modulating FXR. This

disturbance in balance causes host immune response and metabolic stress which leads to progression of liver disease.

D. DIET AND GUT MICROBIOTA.

Changes in dietary components of macronutrients have an effect on microbial metabolic activity. Fat and protein from animal based diet are associated with increase of microbial RNA and DNA encoding sulphite reductases and increase in bile concentration in feces. Hydrogen sulphite produces bowel inflammation. Impaired insulin sensitivity and white adipose tissue inflammation is caused by saturated fatty diet due to the alteration of microbiota. Protection against inflammation and adiposity is seen with microbiota, where the diet rich in unsaturated fats.

E. THERAPEUTIC PERSPECTIVE FROM GUT-LIVER AXIS.

Core of therapy is lifestyle modification. The strategies commonly used for manipulating gut microbiota are:

1. PREBIOTICS:

Acts by gut microbiota modulation by favoring the growth of certain species of bacteria by making nutrients available. It reduces the gut permeability by stimulation of glucagon peptide-2 thereby decreasing the bacterial translocation.

In NASH patients, there is a reduction of endotoxin, serum AST and hepatic steatosis observed in treatment with both prebiotics and probiotics.

2. PROBIOTICS:

Probiotics are the yeast or a live bacterium of human origin when consumed provides health benefits by modulation of microbiota of intestine. Similarly to prebiotics, probiotics minimizes endotoxin and normalize intestinal permeability. It also acts at the level of TLRs modulating the response.

3. ANTIBIOTICS:

Certain antibiotics like rifaximin which is non-absorbable used primarily in the treatment of hepatic encephalopathy, has shown to reduce the bacterial translocation. Rifaximin use was found to reduce the circulating endotoxin levels, AST and ferritin in NAFLD subjects. But due systemic effects, the timing of therapy should identified. The effect of neomycin and polymycin in combination with a fructose supplement has shown a decrease in hepatic fat accumulation. Cidomycin is another antibiotic molecule with positive effects on inflammatory factors and liver function indices like AST and ALT.

4. FECAL MICROBIOTA TRANSPLANTATION:

It is the process in which the feces from a healthy donor are collected and transplanted to a sick person. It transplants both dead and live microorganism, small and large intestine cells, small food particles and bacterial metabolic products. Benefits arise due to an increase in microbial diversity with favorable microbes and stimulation of mucosal immunity. It is an invasive procedure with adverse events, which are mainly infectious.(17)

HISTOPATHOLOGY OF NAFLD.

A. STEATOSIS:

Deposition of lipids in the hepatocyte cytoplasm mainly triglycerides is hepatic steatosis. Within the hepatocytes the lipids are stored in vesicles and push the nucleus to periphery when large. When the hepatocyte contains lipid droplets more than 5% it is considered as simple steatosis. It is usually describes as microvesicular, macrovesicular, or mixed.

1. MACROVESICULAR STEATOSIS:

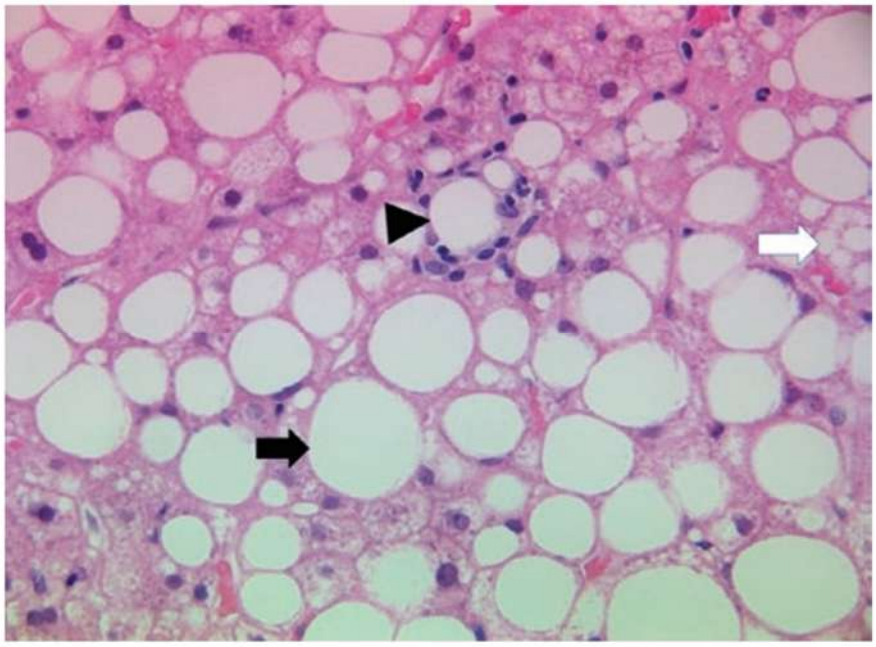
It is characterized by both a few small fat droplets and a nucleus that is centrally placed or by a large single fat droplet that displaces the nucleus to periphery and almost replaces the cytoplasm. The former is also called mediovesicular steatosis or small droplet macrovesicular steatosis.

2. MICROVESICULAR STEATOSIS:

Here the nucleus remains central but the cytoplasm of the hepatocyte has a foamy appearance because of the numerous tiny lipid droplets.

3. MIXED:

It is characterized by large areas of macrovesicular steatosis with few small non-zonal patches of microvesicular steatosis.



PICTURE SHOWING MACROVESICULAR STEATOSIS (BLACK AND WHITE ARROWS) AND LIPOGRANULOMA(ARROW HEAD).

The pattern of injury is centrilobular in early NAFLD where terminal hepatic venule (THV) is the first to show steatosis. But the entire acinus/lobule is affected as disease progresses. In NAFLD it is rare to have pure steatosis, there is usually chronic mononuclear cell infiltrate is present in liver parenchyma. Portal tracts can also show mild chronic inflammation.

B. STEATOHEPATITIS:

The diagnosis of steatohepatitis is made with minimal histological criteria like:

a. Steatosis.

b. Lobular inflammation.

c. Hepatocellular injury (in the form of balloning)

With centrilobular prediliction. Fibrosis is not a mandatory feature in the diagnosis of NASH.

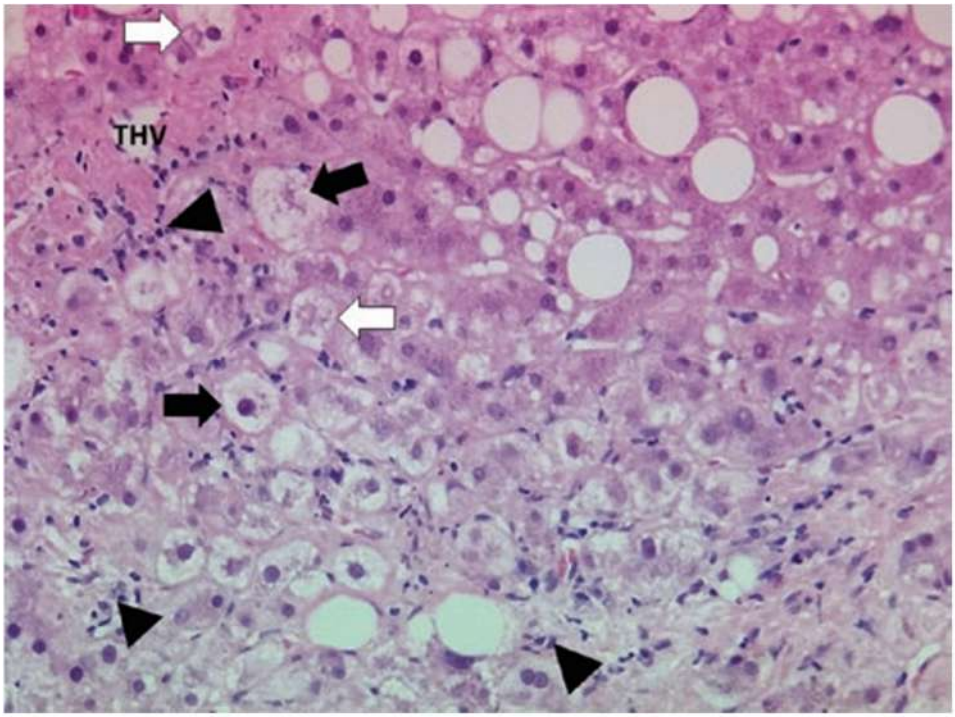
The key histological feature is hepatocyte ballooning and along with lobular inflammation it reflects the disease activity. Ballooning of hepatocyte occurs due to loss of keratin 8 and 18(filament cytoskeleton of hepatocyte), microtubular alterations secondary to oxidative stress, endoplasmic reticulum dilatation and modification of microvesicular intracytoplasmic fat. As a result they lose their polygonal shape and become rounded. It is described as classical and non classical forms.

a. ***Classical form***:

Here the ballooned hepatocytes are larger by 1.5 -2 times as compared to the normla non-steatotic hepatocytes.

b. Non-classical form:

These are norma sized ballooned hepatocyte retaining the characteristic cytoplasmic changes and the round shape.

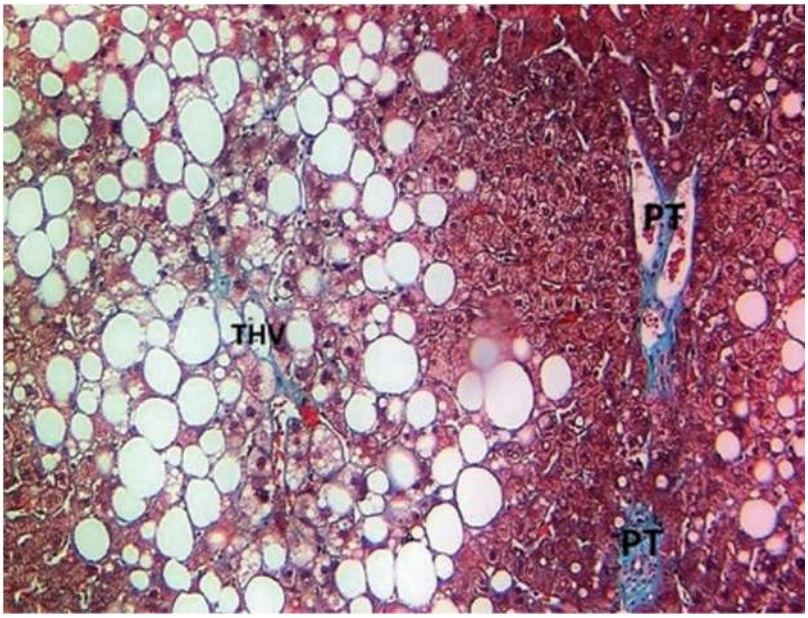


PICTURE SHOWING BALLOONED HEPATOCYTE (BLACK ARROWS), STEATOSIS WITH INFLAMMATORY FOCI (ARROW HEADS) AND MALLORY-DENK BODIES(WHITE ARROW) CLOSE TO THV.

Mallory-denk bodies are inclusions in cytoplasm of eosinophilic hyaline material comprising insoluble ubiquinated proteins, which occurs occasionally in the ballooned heaptocyte.

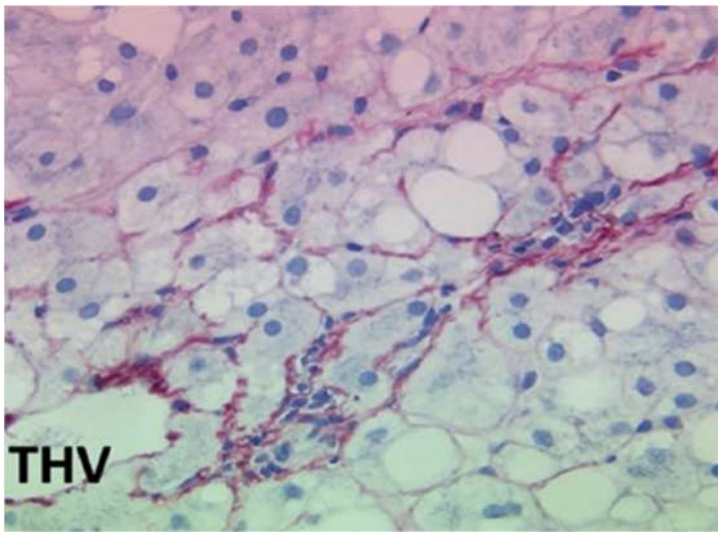
C. FIROSIS IN NASH.

Steatohepatitis is often followed by fibrosis which can progress to cirrhosis in few patients. The deposition of extracellular matrix ocurs initially around the sinusoids in the space of disse (sinusoidal or perisinusoidal fibrosis) and also around the heaptocytes (pericellular).

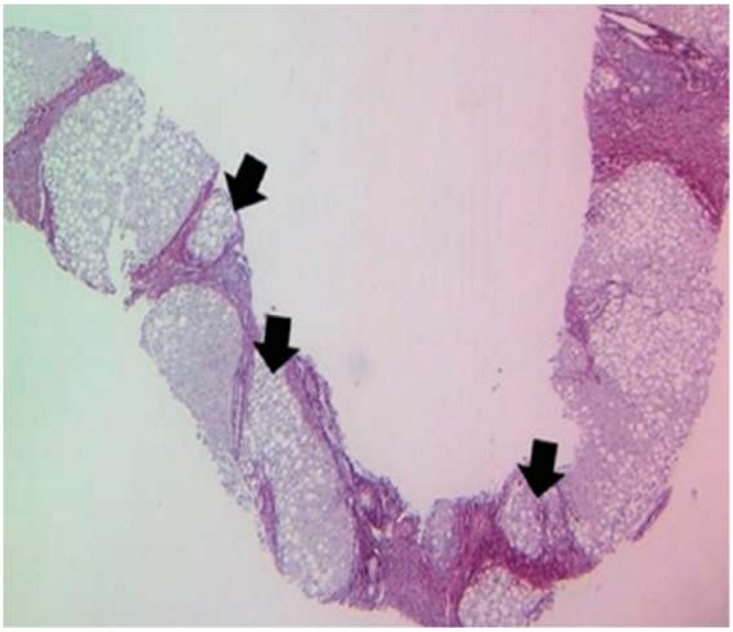


PICTURE SHOWS SINUSOIDAL FIBROSIS IN ZONE 3 AND PERIPORTAL FIBROSIS. MASSON TRICHROME STAINING COLLAGEN FIBRES BLUE.

The first affected are centrilobular areas and as disease progresses the portal and periportal areas get affected. As the disease progresses the fibrous septa originating from portal tracts and THV merge to form central-portal, portal-portal and central-central colagenous bridges.(bridging fibrosis). This leads to architectural remodeling resulting in regenerating hepatocyte as nodular areas surrounded by fibrous septa.



PICTURE SHOWS THIN SINUSOIDAL AND PERICELLULAR FIBROSIS NEAR ZONE 3.



PICTURE SHOWS BRIDGING FIBROUS SEPTA (THICK) WITH CIRUMSCRIBED STEATOTIC LIVER PARENCHYMAL NODULES.

The most important prognostic factor predicting mortality in NAFLD is the fibrosis stage. It also predicts the time to development of severe disease.

Other histological nonspecific features in NAFLD are:

I. Glycogenated nuclei in periportal hepatocytes.

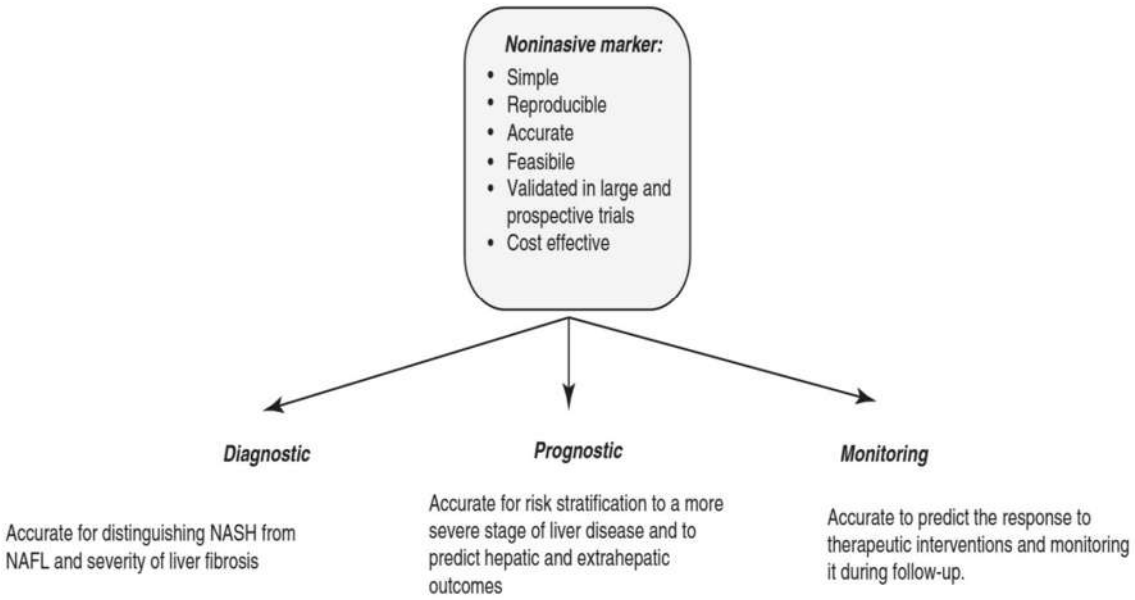
II. Megamitochondria.

III. Mild heaptic siderosis.(18)

NON-INVASIVE DIAGNOSTIC APPROACH TO NAFLD AND NASH

An ideal non-invasive biomarker should predict:

- Severity of the liver disease.
- Progression and occurrence of decompensation and complications.
- Response to pharmacological and lifestyle corrections.



A. BIOLOGICAL MARKERS.

1. MARKERS OF DISEASE SEVERITY.

In the pathogenesis of NASH apoptosis is one of the feature. One of the potential non-invasive marker is serum CK-18 fragments and total length CK-18 measurement is based on that. Other scoring invasive methods are: NASH test (panel of 13 parameters age, sex, height, weight, triglycerides, total cholesterol, α2-macroglobulin, apolipoproteina A1, haptoglobulin, GGT, ALT, AST and total bilirubin) ,NASH(ION) score (panel includes triglycerides, ALT, waist-hip ratio and HOMA) and NASH score which includes a genetic variable PNPLA3 genotype, fasting insulin and AST. Latest score developed with better accuracy is NASH ClinLipMet score.

2. PREDICTION OF FIBROSIS SEVERITY.

demographic/serum markers: these include APRI (AST: platelet ratio), BARD score (BMI, AST/ALT ratio, diabetes), FibroMeter (AST, glucose, ferritin, ALT, platelet, age, body weight), FIB-4 (age, ALT, AST, platelet), NFS (age, BMI, hyperglycemia, albumin, platelet, AST/ALT ratio) and HEPAMET (age, female gender, diabetes, HOMA, albulmin, AST and platelet). All these reflect biological processess and risk factors asscociated with fibrosis and not the mechanism.

Serum fibrosis panel: this includes ELF panel , FibroTest, HepaScore, ADAPT, NASH F2-F4 and MACK-3 test. These panels look at the collagen turnover within the liver. The mostly used ones are FibroTest , ELF and HepaScore. The fibrotest includes a combinations of apolipoprotein, haptoglobulin, GGT adgusted for gender and age, total bilirubin and serum α2-macroglobulin.

3. PROGNOSTIC MARKERS.

The markers with prognostic value are BMI, diabetes, waist circumference, arterial hypertension, HOMA-IR, NAFLD fibrosis score and fibrometer. Among them FibroMeterV2G had the best prediction of all cause mortality.

4. MARKERS OF TREATMENT RESPONSE.

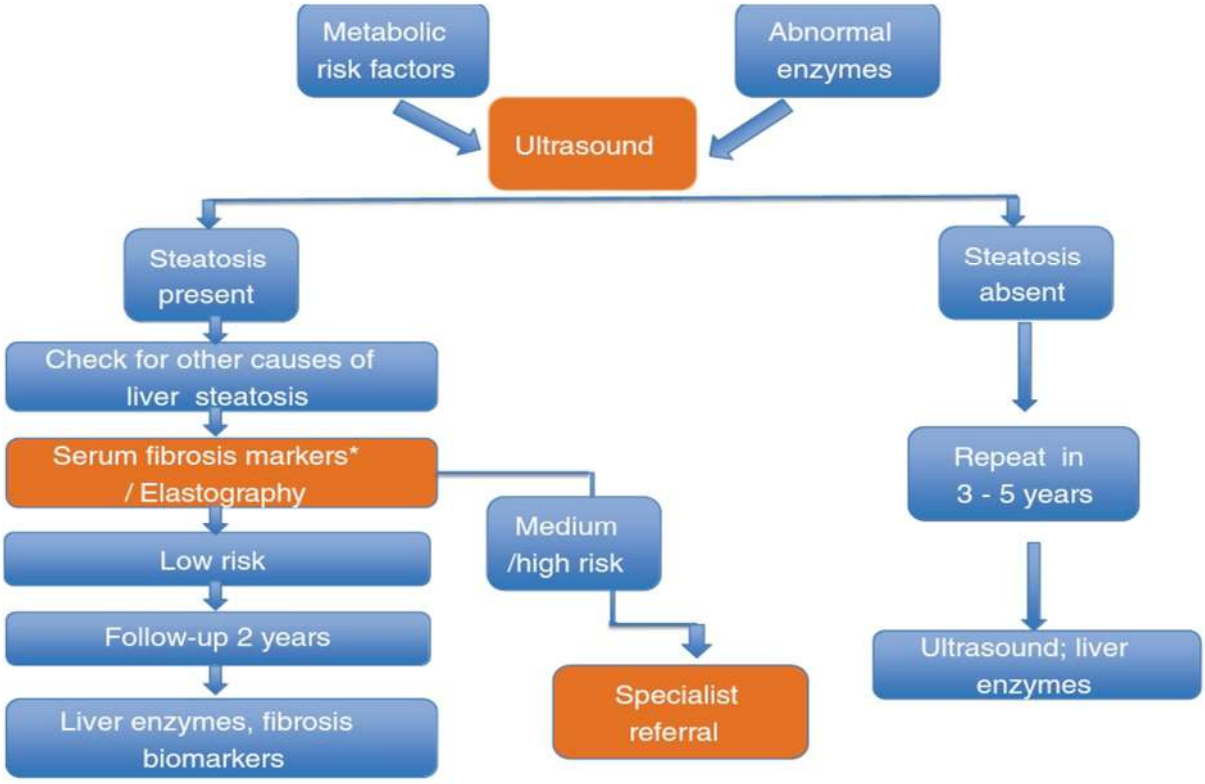
These are CK-18, NASHRES score, PNLPA3 G allele, HBA1C, ALT and NAFLD fibrosis score. Advantage is that it serves as preliminary tool to predict histological changes.(19)

B. RADIOLOGICAL DIAGNOSTICS.

Steatosis diagnosis*: ultrasound(USG)*- inferred qualitatively based on the brightness of liver sonographic images as compared to the adjacent structures and categorized as mild, moderate, severe degree. For screening purposes the approximate sensitivity and specificity are 80% and 86% respectively. *CT*- not used frequently due to high radiation exposure. It is more specific than USG. In unenhanced CT, where reduced attenuation correlated with the intrahepatic steatosis. *MRI*- there are two methods, MR spectroscopy (MRS) and MRI-based

proton density fat fraction (PDFF). In MRS the signal intensities correspond to specific frequency of which fat signal fraction is quantified. It had good sensitivity. MRI-PDFF assess the ratio between MR-visible triglyceride protons to sum of the water and triglyceride protons.

Fibrosis diagnosis: USG based methods include vibration controlled transient elastography, sheer wave elastography and acoustic radiation force impulse. Magnetic resonance elastography (MRE) is also used for quantitaive fibrosis assessment. Multiparametric MRI/MRE is a better methodology to assess the severity of NASH.(20)



DIAGNOSTIC ALGORITHM OF NAFLD AT PRIMARY CARE.

TREATMENT

A. DIETARY ADVICE:

Hypocaloric diet poor in carbohydrates and fats tend to reduce liver fat content. MUFA and PUFA are beneficial for NAFLD improvement.to reduce saturated fatty acids and fructose corn syrup consumption. Advised 5%-10% weight loss and calorie restriction of 500-750or less per day. Omega-3 fatty acid supplementation decreases steatosis.(21)

B. EXERCISE ADVICE:

Aerobic and/ or resistance training exercises help to improve the insulin resistance with a goal of expenditure of 400 calories per week.

- **Aerobic (e.g. jogging, cycling):**
  - **150–300 min/week of moderate-to-vigorous intensity** (50–70% VO_2peak) **≥3 days/week**
- **Resistance (strength training):**
  - **2–3 sets of 8–12 repetitions** (70–85% 1RM) **2–3 days/week**
- For weight maintenance: ↑ volume of exercise
- For improvement in cardiorespiratory fitness and glycaemic control: ↑ intensity of exercise

RECOMMENDED EXERCISE PRESCRIPTION IN NAFLD.(22)

C. PHARMACOTHERAPY:

i. ***Current pharmacotherapy:*** *thiazolidinediones* (glitazones) which are PPAR gamma agonist are used help by promoting adipocyte differentiation into insulin sensitive small adipocytes. It also increases

adiponectin. *Antioxidants* like *vitamin E* given as 800IU/day acts by preventing activation of stellate cell and improves liver fibrosis and necrosis. Other drugs used are *incretin mimetics, pentoxyphylline, statins* and *ezetimibe*.

ii. ***Future developing pharmacotherapy:*** *FXR agonists- obeticholic acid*, it is synthetic bile which on activation of FXR causes multiple metabolic effects which causes improvement of NASH histology. *PPAR alpha/delta agonist – elafibranor* also acts by inhibiting hepatic lipogenesis, inducing hepatic fatty acid beta oxidation and reduced hepatic inflammation. *Chemokines –cenicriviroc*, a selective inhibitor of CCR2 AND CCR5, is found to reduce chronic liver inflammation by decreasing monocyte infiltration. *Fatty acid- bile acid conjugate –aramchol* has antisteatotic action by decreasing the synthesis of MUFA and triglycerides. *Antifibrotics agents –simtuzumab, galectin-3 inhibitors.*(23)

D. BARIATRIC SURGERY.

Intragastric balloon, adjustable gastric banding, sleeve gastromy, roux-en-y gastric bypass and biliopancreatic division are some of the bariatric options for NASH.(24)

E. LIVER TRANSPLANTATION.

Comorbids limit eligibility and recurrent steatosis post transplantation reported.

MATERIALS AND METHODS:

- ***Type of study:*** Crossectional study.
- ***Study area:*** Government Stanley medical college and hospital**,** Chennai.
- ***Study population:*** Out Patients in the department of master health check-up, in government Stanley medical college and hospital.
- ***Inclusion criteria:***
    1. Patients in the age group 18 or more.
    2. Patients not previously diagnosed with diabetes mellitus**.**
- ***Exclusion criteria:***
    1. Patients age group less than 18.
    2. Patients previously diagnosed with diabetes mellitus.
    3. Any quantity of alcohol consumption based on history
    4. Patients with a known hepatic disease or cirrhosis.
    5. Pregnant woman.
- ***Sample size:***

  Based on the reference study done by Dr.Anbalagan et al, Mohan's diabetes foundation, Chennai

  Formula:

  n = Z2pq / d2

  Where Z = 1.96 (statistical significant constant for 95% CI)

  p =4.6 %(Proportion of Non diabetic adults belonging to Indian diabetes risk score less <30 (mild risk) from previous study.)

  q = 95.4% (100-p)

d = 4% absolute precision

On substituting, in the formula

n = 3.84 x 4.6 x 95.4 / 16

n = 105

Adding 10% non-response rate (i.e., 10% of 105=11)

n = 116 (minimum sample size)

Therefore Sample size n = 130 (1 group).

- ***Study duration:*** March 2021 to September 2021 (6 months).

- ***Methodology:***

  After obtaining informed written consent from the patients / attenders, relevant history was obtained and was subjected to physical examination. For each patient IDRS score was calculated and recorded.

Table 1 : The MDRF – Indian Diabetes Risk Score [IDRS][3]

Particulars	Score
Age:	
<35 years	0
35 – 49 years	20
≥ 50 years	30
Waist circumference:	
Waist < 80 cm [female], <90 cm [male]	0
Waist < 80 - 89 cm [female], < 90 – 99 cm [male]	10
Waist ≥ 90 cm [female], ≥ 100 cm [male]	20
Physical activity:	
Vigorous exercise [regular] or strenuous [manual] work at home / work	0
Moderate exercise [regular] or moderate physical activity at home / work	10
Mild exercise [regular] or mild physical activity at home / work	20
No exercise and sedentary activities at home / work	30
Family history of diabetes:	
No diabetes in parents	0
One parent is diabetic	10
Both parents are diabetic	20

If the score is….
≥ 60: Very **HIGH RISK** of having diabetes. Oral Glucose Tolerance Test (OGTT) is recommended to rule out diabetes. If this is not possible, at least a random blood sugar or a fasting blood sugar should be done
30 – 50 : The risk of having diabetes is **MODERATE**. It is still recommended to have the above check up.
<30 : Risk of having diabetes is probably **LOW**

Further various biochemical and radiological investigations like fasting blood sugar, Hba1c, liver function test, fasting lipid profile and ultrasonic scanning of abdomen were carried out.

The USG parameters included for diagnosis were increased echogenicity of liver, decreased penetration of sound, lack of visibility of vascular structures within the liver due to ill-defined portal walls and increase in the liver size in mid-clavicular line >15.5cm and the fatty liver was graded. The data collected was entered in,Microsoft Excel and analysed.

STATISTICAL ANALYSIS

The collected data were analysed with IBM SPSS Statistics for Windows, Version 23.0. (Armonk, NY: IBM Corp).To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables. To find the significant difference in the multivariate analysis the one way ANOVA with Tukey's Post-Hoc test was used. To find the significance in categorical data Chi-Square test was used. In all the above statistical tools the probability value .05 is considered as significant level.

Table 1: Age distribution

Age distribution		
	Frequency	Percent
21 - 30 yrs	33	25.4
31 - 40 yrs	58	44.6
41 - 50 yrs	27	20.8
Above 50 yrs	12	9.2
Total	130	100.0
Mean ± SD = 38 ± 9 yrs		

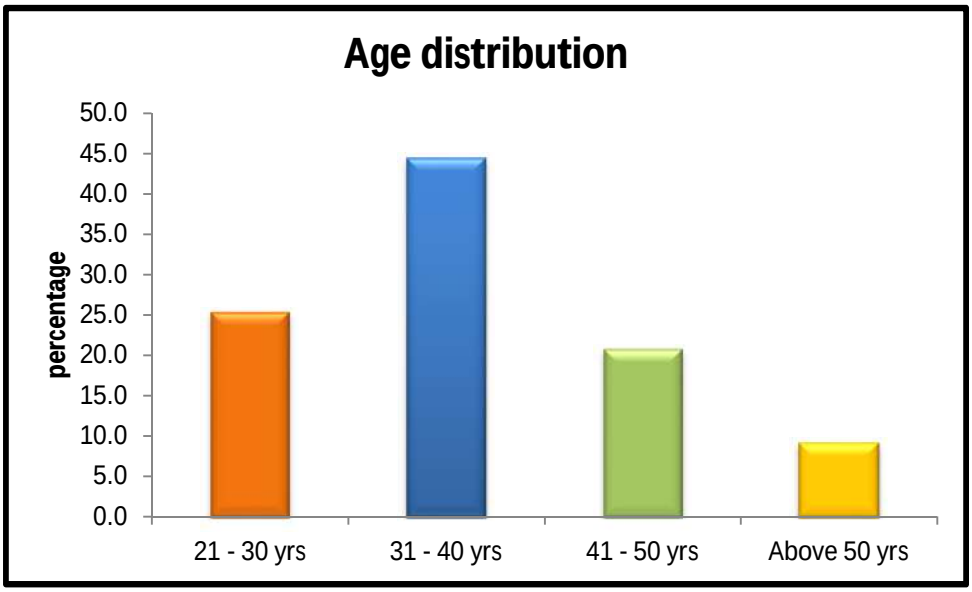


Figure 1

The above table shows Age distribution were <21-30 years is 25.4%, 31-40 years is 44.6%, 41-50 years is 20.8%, >50 years is 9.2%. The patients between 31- 40 years were the maximum number in the study population.

Table 2: Gender distribution.

Gender distribution		
	Frequency	Percent
Male	67	51.5
Female	63	48.5
Total	130	100.0

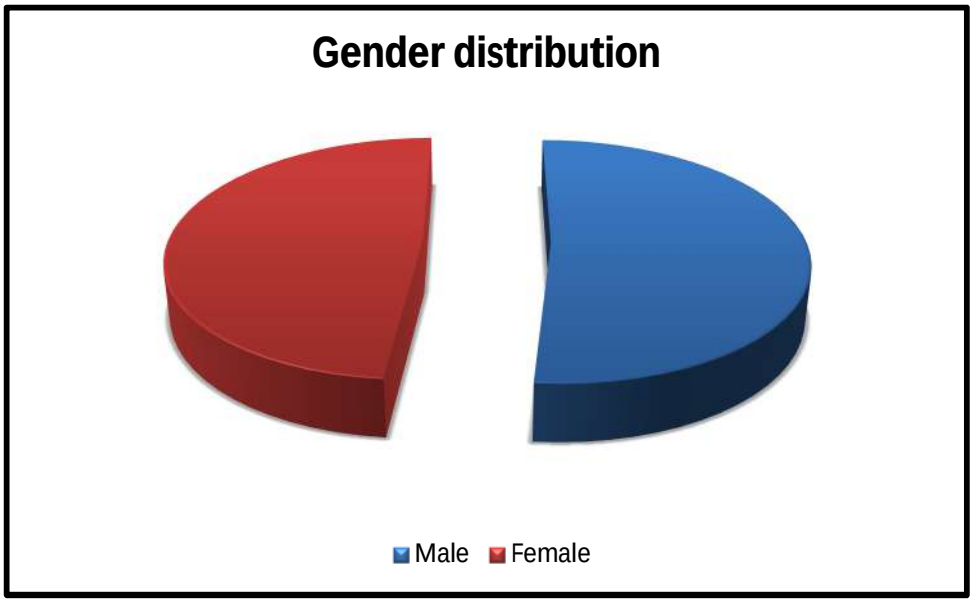


Figure 2

The above table depicts the Gender distribution, where females constitute 48.5% and males were 51.5% of the study population.

Table 3: Distribution of Comorbidities

Comorbidities		
	Frequency	Percent
HYPOTHYROID	11	8.5
PCOS	2	1.5
SHT	6	4.6
SHT,CKD	2	1.5
SHT,HYPOTHYROID	2	1.5
NO	107	82.3
Total	130	100.0

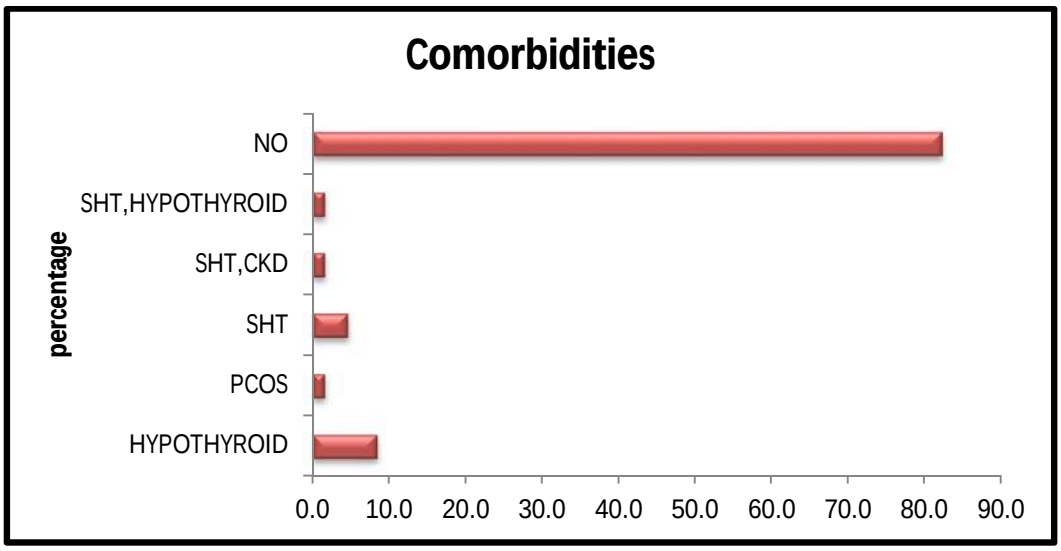


Figure 3

The above table shows Comorbidities distribution were hypothyroidism is 8.5%, PCOS is 1.5%, SHT is 4.6%, SHT with CKD is 1.5% and SHT together with hypothyroidism is 1.5%. Majority of the study population had no comorbidities which constitute 82.3%.

Table 4: Distribution of IDRS Score

IDRS Score		
	Frequency	Percent
< 30	12	9.2
30 - 50	70	53.8
>= 60	48	36.9
Total	130	100.0

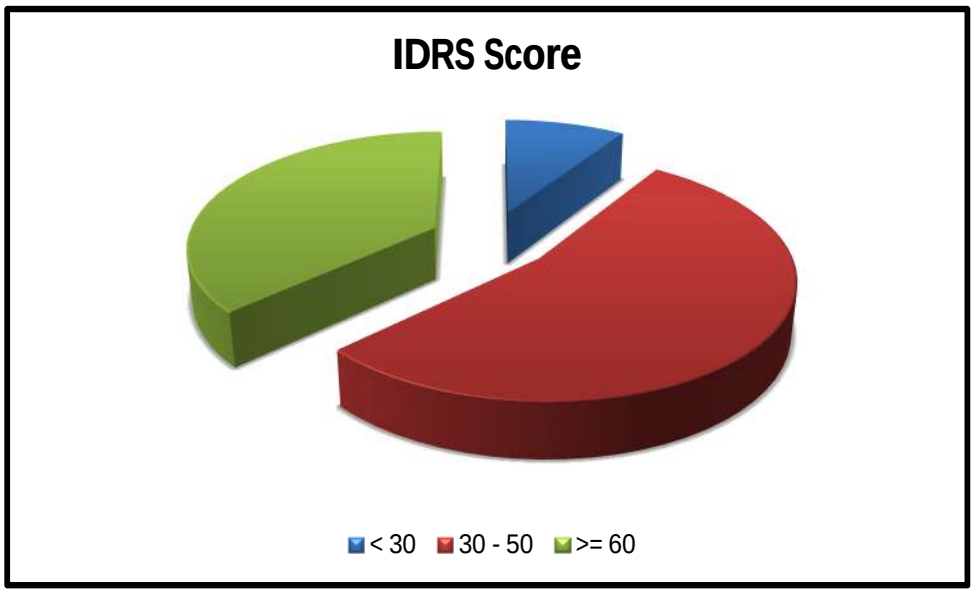


Figure 4

The above table depicts the distribution of IDRS score of the study population were 9.2% of them had score less than 30, maximum number of people in the study had score between 30-50 which is 53.8% and those with score >= 60 were 36.9%.

Table 5: Comparison of Gender with IDRS by Pearson's Chi-Square test

			IDRS			Total	$\chi 2$-value	p-value
			< 30	30 - 50	>= 60			
Gender	Male	Count	10	26	31	67	13.935	0.001 **
		%	83.3%	37.1%	64.6%	51.5%		
	Female	Count	2	44	17	63		
		%	16.7%	62.9%	35.4%	48.5%		
Total		Count	12	70	48	130		
		%	100.0%	100.0%	100.0%	100.0%		
** Highly Statistical Significant at p < 0.01 level								

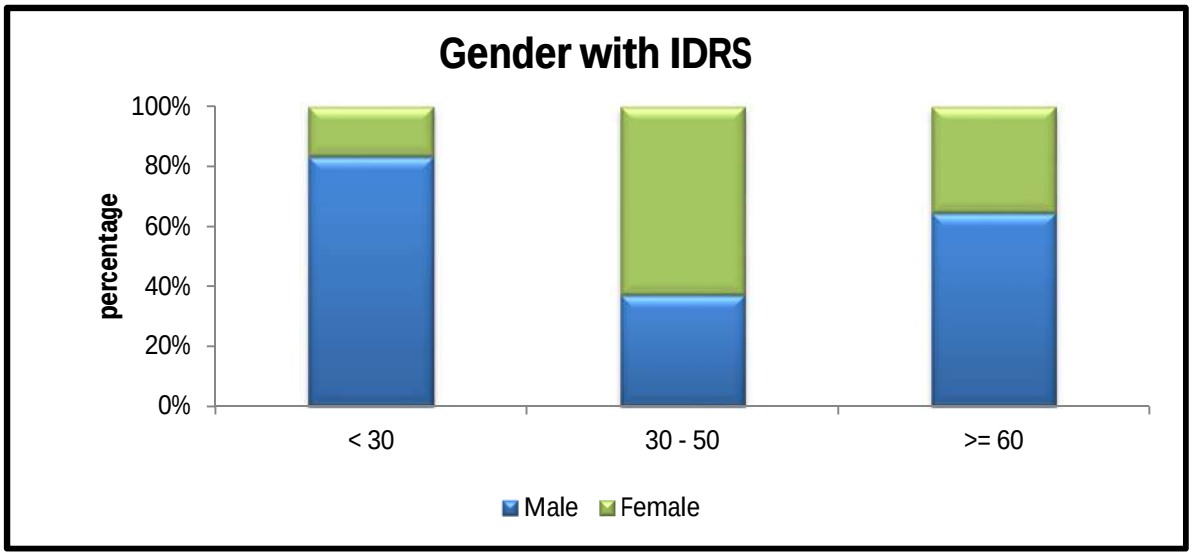


Figure 5

The above table shows comparison between Gender with IDRS by Pearson's Chi-square test were $\chi 2=13.935$, $p=0.001<0.01$ which shows highly statistical significant association between Gender and IDRS. The percentage of females were high for score ranging 30-50 whereas males were higher in the group with score greater than 60.

Table 6: Comparison of IDRS with USG by Pearson's Chi-Square test.

			USG				Total	χ^2-value	p-value
			Normal	I	II	III			
IDRS	< 30	Count	8	4	0	0	12	34.885	0.0005 **
		%	66.7%	33.3%	0.0%	0.0%	100.0%		
	30 - 50	Count	50	18	0	2	70		
		%	71.4%	25.7%	0.0%	2.9%	100.0%		
	>= 60	Count	21	8	17	2	48		
		%	43.8%	16.7%	35.4%	4.2%	100.0%		
Total		Count	79	30	17	4	130		
		%	60.8%	23.1%	13.1%	3.1%	100.0%		
** Highly Statistical Significant at p < 0.01 level									

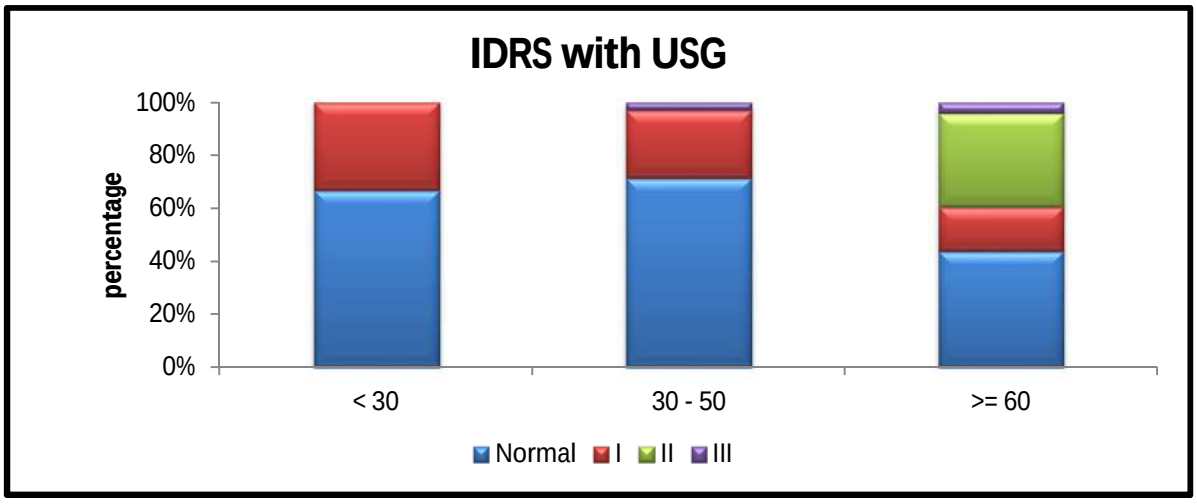


Figure 6

The above table shows comparison between IDRS with USG by Pearson's Chi-square test were $\chi 2=34.885$, $p=0.0005<0.01$ which shows highly statistical significant association between IDRS and USG.

Out of the 130 people in the study, 79 (60%) had normal ultrasonic finding and remaining 51(39.2%) had fatty liver in various grades. Of the 51 people who had fatty liver, 7.8% had low risk score, 39.2% had medium risk score and 52.9% with high IDRS score. Therefore maximum number of fatty liver subjects had scores greater than 60.

Table 7(a): Comparison of BMI with IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
BMI	< 30	12	22.4	0.5	34.989	0.0005 **
	30 - 50	70	23.4	1.4		
	>= 60	48	25.4	1.7		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
BMI	< 30	30 - 50	-.9352	.4627	0.111 #	-2.033	.162
		>= 60	-2.9833*	.4780	0.0005 **	-4.117	-1.850
	30 - 50	>= 60	-2.0481*	.2776	0.0005 **	-2.706	-1.390
** Highly Statistical Significant at p < 0.01 and # No Significant at p > 0.05							

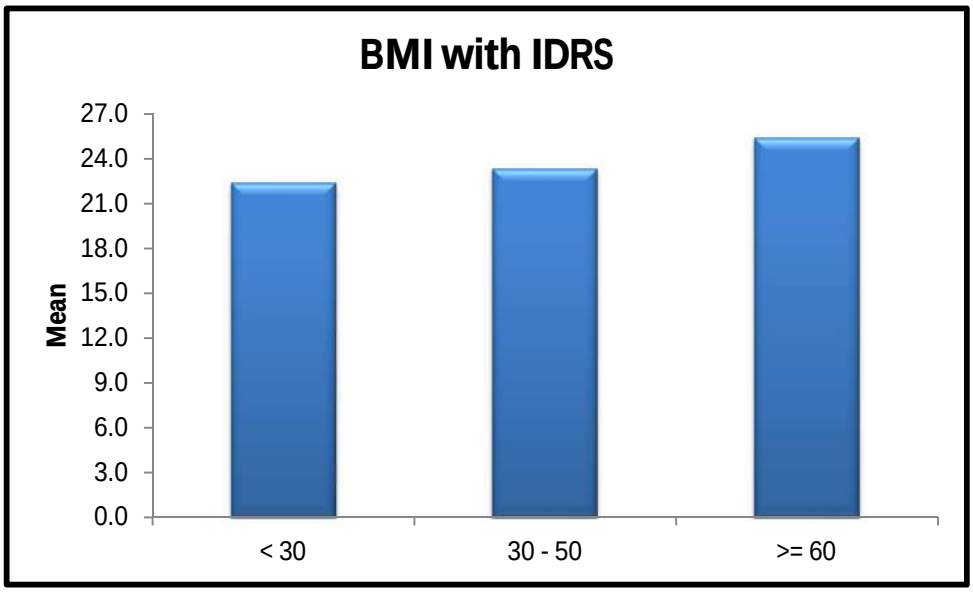


Figure 7(a)

The above table shows the comparison of BMI with IDRS by using Oneway ANOVA were F-value=34.989, p-value=0.0005<0.01, which shows highly statistical significant difference at p <0.01 level.

Table 7(b): Comparison of BMI with Fatty liver by Pearson's Chi-Square test.

			Fatty liver		Total	χ2-value	p-value
			Positive	Negative			
BMI	< 25	Count	21	71	92	35.528	0.0005 **
		%	41.2%	89.9%	70.8%		
	>= 25	Count	30	8	38		
		%	58.8%	10.1%	29.2%		
Total		Count	51	79	130		
		%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level							

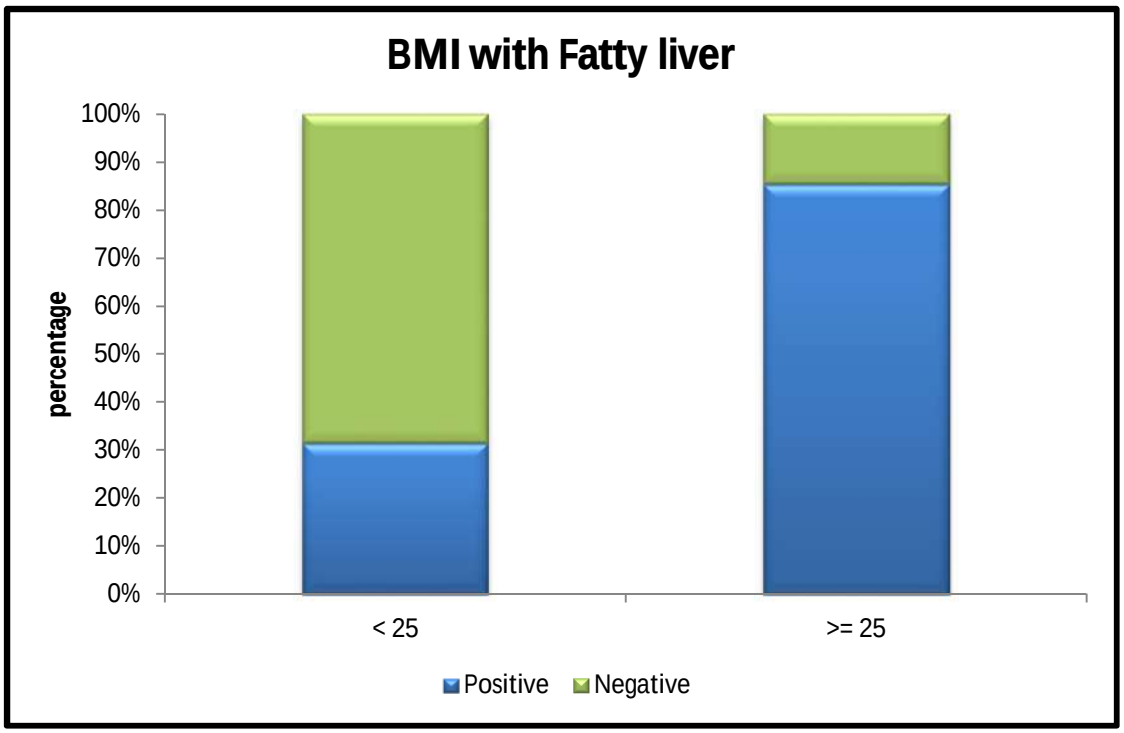


Figure 7(b)

The above table shows comparison between BMI with Fatty liver by Pearson's Chi-square test were $\chi 2=35.528$, $p=0.0005<0.01$ which shows highly statistical significant association between BMI and Fatty liver.

On comparing normal subjects with those having fatty liver, the proportion of people with BMI greater than or equal to 25 were higher among the fatty liver group. About 90% of the non-fatty liver group had BMI less than 25.

Table 8: Comparison of Waist Circumference with the IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
Waist Circumference	< 30	12	84.3	3.7	19.664	0.0005 **
	30 - 50	70	85.2	4.5		
	>= 60	48	90.0	4.2		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
Waist circumference	< 30	30 - 50	-.9095	1.3484	0.779 #	-4.107	2.288
		>= 60	-5.6667*	1.3929	0.0005 **	-8.970	-2.363
	30 - 50	>= 60	-4.7571*	.8088	0.0005 **	-6.675	-2.839
** Highly Statistical Significant at p < 0.01 and # No Significant at p > 0.05							

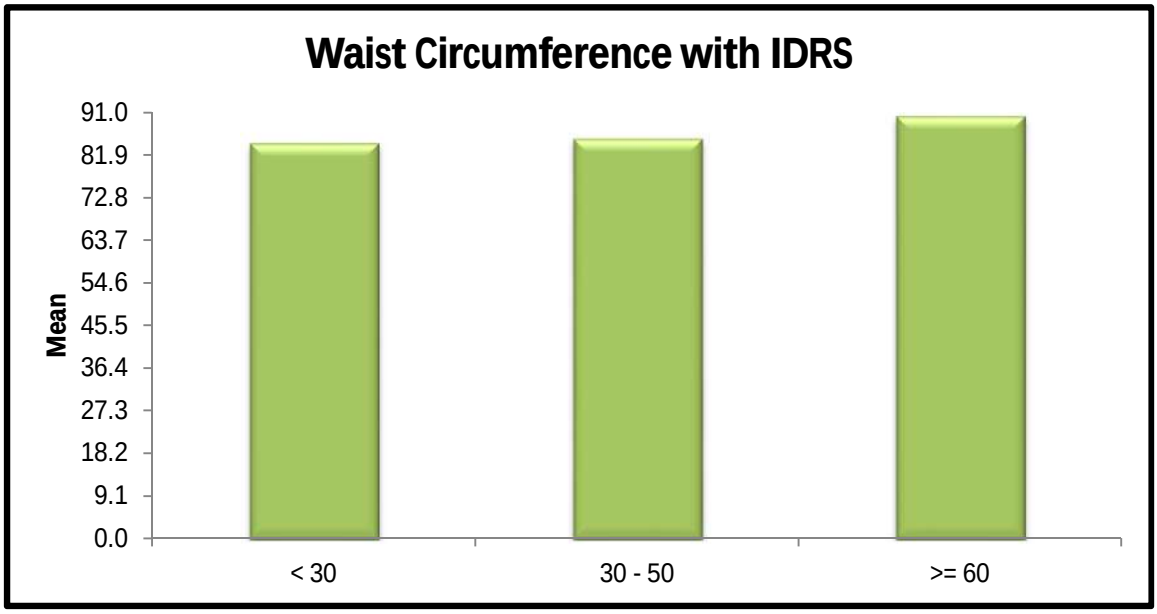


Figure 8

The above table shows the comparison of SBP with IDRS by using Oneway ANOVA were F-value=16.168, p-value=0.0005<0.01, which shows highly statistical significant difference at p <0.01 level.

Among the average waist circumference in three groups of IDRS, subjects with score greater or equal to 60 has the maximum of 90cm.

Table 9: Comparison of waist hip circumference with Fatty liver by Pearson's Chi-Square test.

Gender				Fatty liver		Total	χ^2-value	p-value
				Positive	Negative			
Male	WHR	< 0.9	Count	11	32	43	13.321	0.0005 **
			%	40.7%	84.2%	66.2%		
		>= 0.9	Count	16	6	22		
			%	59.3%	15.8%	33.8%		
	Total		Count	27	38	65		
			%	100.0%	100.0%	100.0%		
Female	WHR	< 0.85	Count	5	27	32	12.276	0.0005 **
			%	20.8%	65.9%	49.2%		
		>= 0.85	Count	19	14	33		
			%	79.2%	34.1%	50.8%		
	Total		Count	24	41	65		
			%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level								

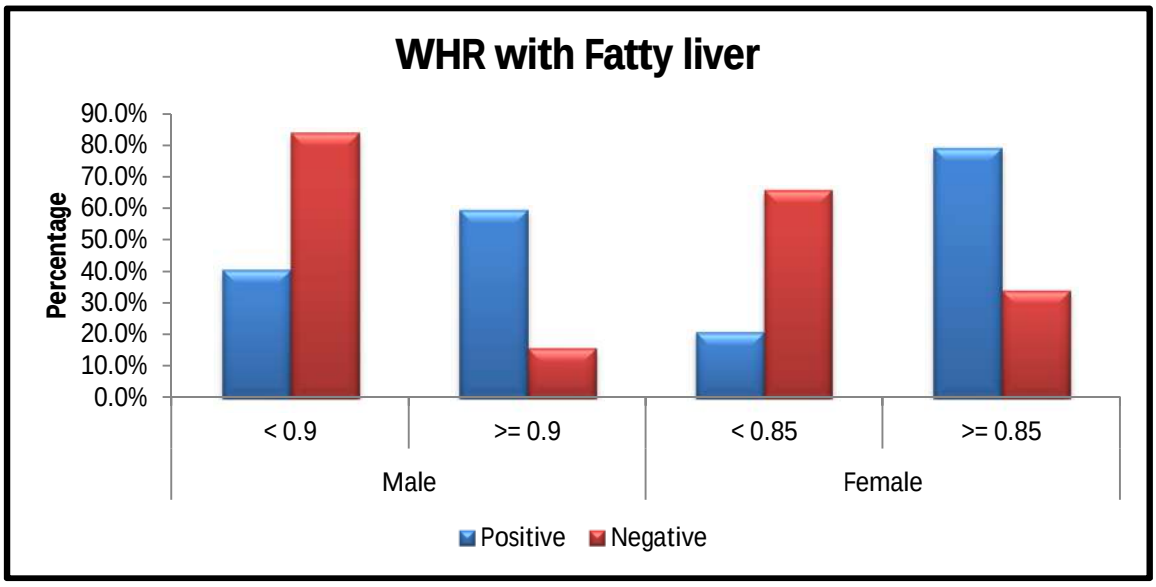


Figure 9

The above table shows comparison between WHR with Fatty liver by Pearson's Chi-square test were Male χ^2=13.321, p=0.0005<0.01, Female χ^2=12.276, p=0.0005<0.01 which shows highly statistical significant association between WHR and Fatty liver.

The above figure depicts the distribution of waist hip ratio gender wise between fatty liver positive and negative groups. Among males, the percentage of subjects with WHR >= 0.9 were greater among the fatty liver positive subjects whereas the proportion with WHR < 0.9 was higher with the non-fatty subjects.

Similarly among females, the proportion of subjects below 0.85 was higher with non-fatty liver people whereas for WHR greater than 0.85 the maximum proportion is from the fatty liver subjects.

Table 10: Comparison of SBP with the IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
SBP	< 30	12	115.3	6.3	16.168	0.0005 **
	30 - 50	70	118.5	6.3		
	>= 60	48	124.2	6.1		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
SBP	< 30	30 - 50	-3.1238	1.9436	0.246 #	-7.733	1.486
		>= 60	-8.8333*	2.0078	0.0005 **	-13.595	-4.072
	30 - 50	>= 60	-5.7095*	1.1658	0.0005 **	-8.474	-2.945
** Highly Statistical Significant at p < 0.01 and # No Significant at p > 0.05							

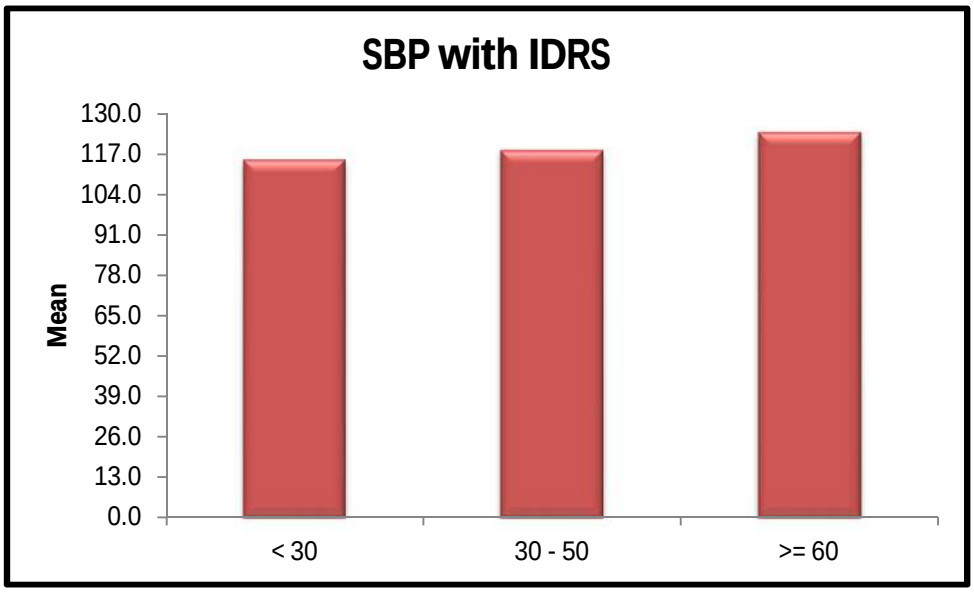


Figure 10

The above table shows the comparison of SBP with IDRS by using Oneway ANOVA were F-value=16.168, p-value=0.0005<0.01, which shows highly statistical significant difference at p <0.01 level.

Table 11: Comparison of DBP with the IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
DBP	< 30	12	72.7	6.8	9.541	0.0005 **
	30 - 50	70	73.8	6.4		
	>= 60	48	78.8	7.0		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
DBP	< 30	30 - 50	-1.1048	2.0713	0.855 #	-6.017	3.807
		>= 60	-6.1667*	2.1397	0.013 *	-11.241	-1.092
	30 - 50	>= 60	-5.0619*	1.2424	0.0005 **	-8.008	-2.116
** Highly Statistical Significance at p < 0.01 ,* Significant at p < 0.05 and # No Statistical Significance at p > 0.05							

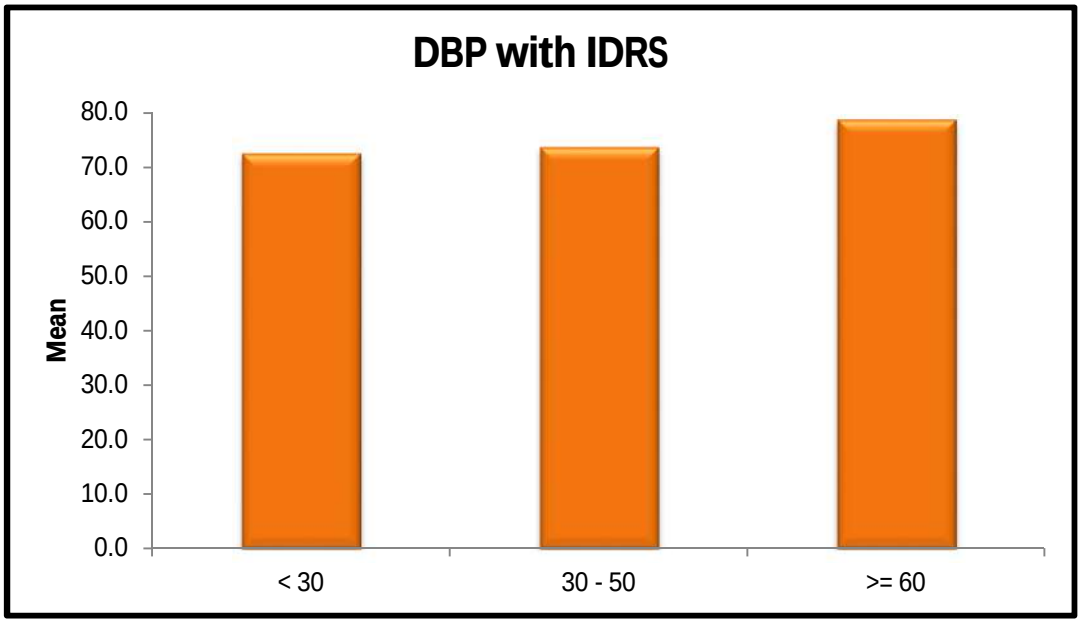


Figure 11

The above table shows the comparison of DBP with IDRS by using Oneway ANOVA were F-value=9.541, p-value=0.0005<0.01, which shows highly statistical significant difference at p <0.01 level.

Table 12a: Comparison of FBS with the IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
FBS	< 30	12	83.2	3.3	54.312	0.0005 **
	30 - 50	70	86.0	5.5		
	>= 60	48	96.6	6.8		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
FBS	< 30	30 - 50	-2.8333	1.8446	0.278 #	-7.208	1.541
		>= 60	-13.4792*	1.9055	0.0005 **	-17.998	-8.960
	30 - 50	>= 60	-10.6458*	1.1064	0.0005 **	-13.270	-8.022
** Highly Statistical Significant at p < 0.01 and # No Significant at p > 0.05							

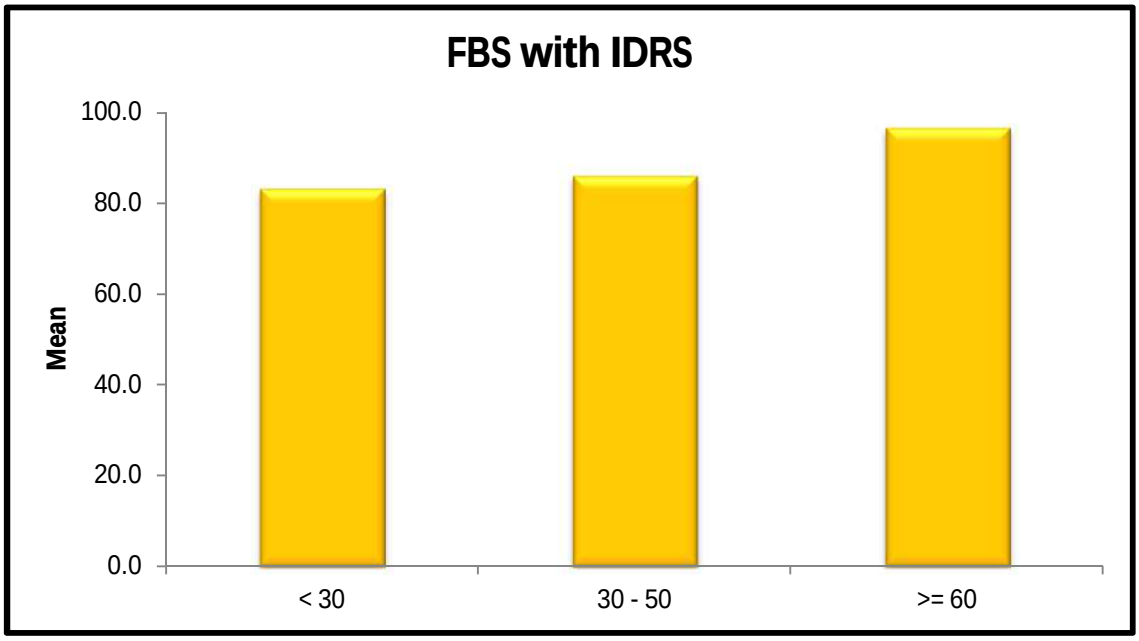


Figure 12a

The above table shows the comparison of FBS with IDRS by using Oneway ANOVA were F-value=54.312, p-value=0.0005<0.01, which shows highly statistical significant difference at p <0.01 level.

Table 12b: Comparison of FBS with Fatty liver by Pearson's Chi-Square test.

			Fatty liver		Total	χ2-value	p-value
			Positive	Negative			
FBS	< 100	Count	39	75	114	9.792	0.002**
		%	76.5%	94.9%	87.7%		
	100 - 125	Count	12	4	16		
		%	23.5%	5.1%	12.3%		
Total		Count	51	79	130		
		%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level							

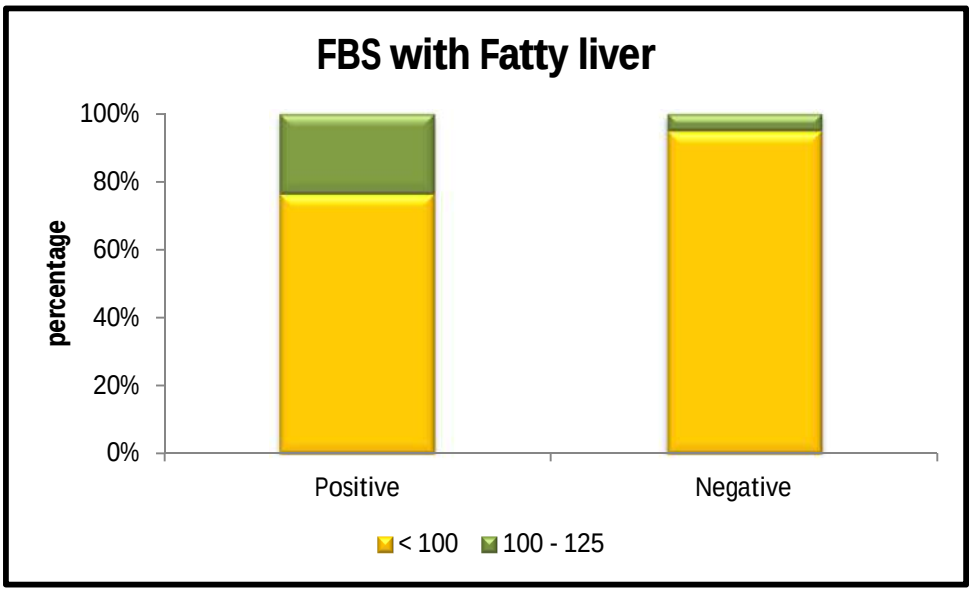


Figure 12b

The above table shows comparison between FBS with Fatty liver by Pearson's Chi-square test were χ^2=9.792, p=0.002<0.01, which shows highly statistical significant association between FBS and Fatty liver.

Among the 130 previously non-diabetic subjects, 23.5% among the fatty liver group and 5.1% among the non-fatty liver group had FBS in the pre-diabetic range. The highest FBS value observed in the study was 108 mg/dl.

Table 13(a): Comparison of HBA1C with the IDRS by Oneway ANOVA test

	IDRS	N	Mean	SD	F-value	p-value
HBA1C	< 30	12	5.5	0.1	46.393	0.0005 **
	30 - 50	70	5.5	0.2		
	>= 60	48	5.8	0.2		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
HBA1C	< 30	30 - 50	-.0957	.0544	0.187 #	-.225	.033
		>= 60	-.3813*	.0562	0.0005 **	-.514	-.248
	30 - 50	>= 60	-.2855*	.0326	0.0005 **	-.363	-.208
** Highly Statistical Significant at p < 0.01 and # No Significant at p > 0.05							

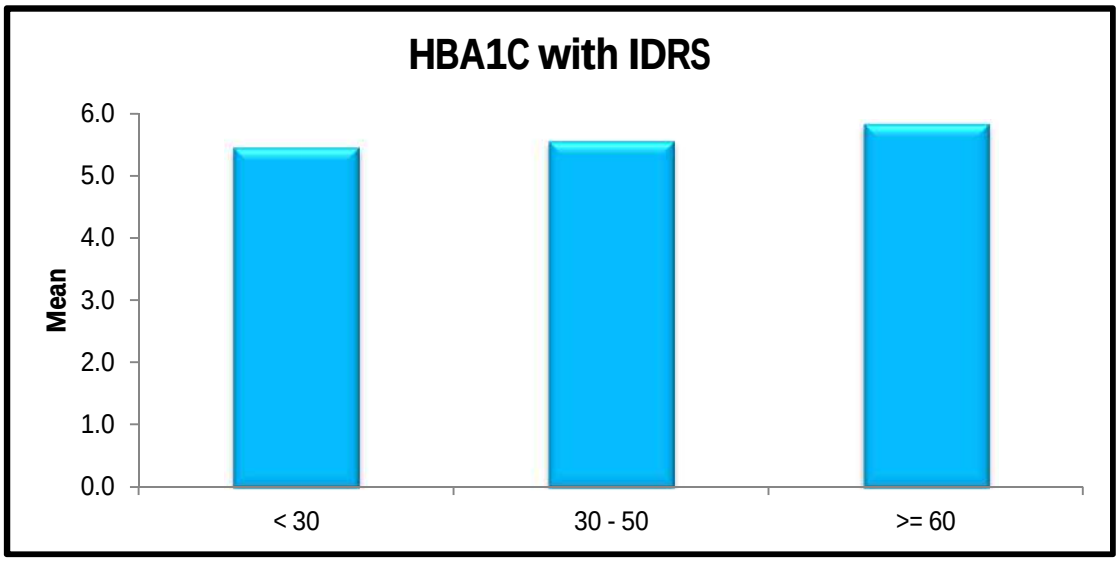


Figure 13(a)

The above table shows the comparison of HBA1C with IDRS by using Oneway ANOVA were F-value=46.393, p-value=0.0005<0.01, which shows highly statistical significant difference at p <0.01 level.

Table 13(b): Comparison of HBA1C with Fatty liver by Pearson's Chi-Square test

			Fatty liver		Total	χ2- value	p-value
			Positive	Negative			
HbA1C	< 5.7	Count	21	57	78	12.390	0.0005 **
		%	41.2%	72.2%	60.0%		
	5.7 - 6.4	Count	30	22	52		
		%	58.8%	27.8%	40.0%		
Total		Count	51	79	130		
		%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level							

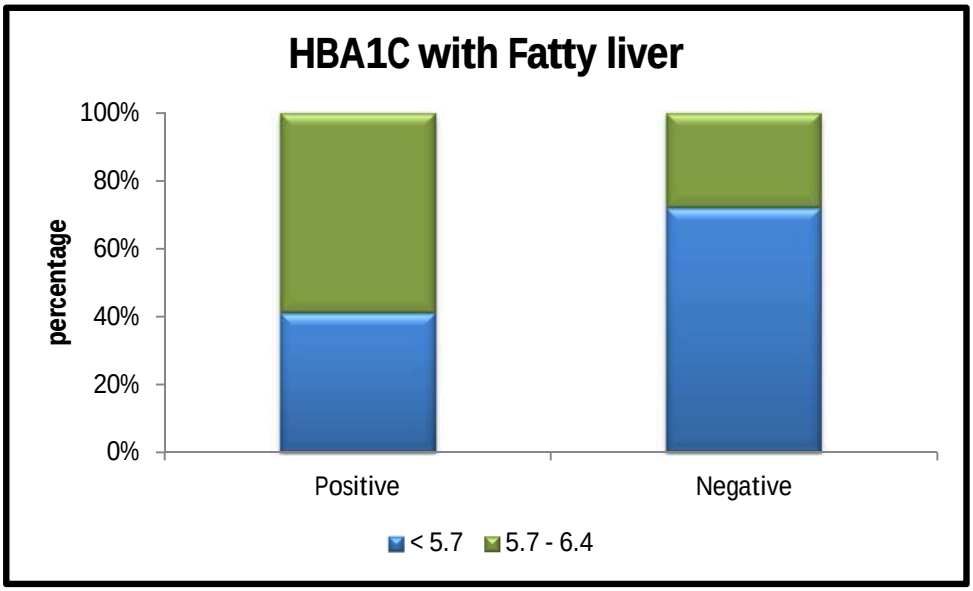


Figure 13(b)

The above table shows comparison between HBA1C with Fatty liver by Pearson's Chi-square test were $\chi 2=12.390$, $p=0.0005<0.01$ which shows highly statistical significant association between HBA1C and Fatty liver.

The percentage of subjects with HBA1C <5.7 are 41.2% and 72.2% among fatty liver positive and negative group respectively. On the other hand the percentage of fatty liver subjects were higher (58.8%) with HBA1C in the range 5.7-6.4, as compared to the non-fatty liver people (27.8%).

Table 14(a): Comparison of Total cholesterol with the IDRS by Oneway ANOVA test

	IDRS	N	Mean	SD	F-value	p-value
Total cholesterol	< 30	12	157.5	9.2	13.762	0.0005 **
	30 - 50	70	175.3	19.2		
	>= 60	48	190.3	26.0		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
Total cholesterol	< 30	30 - 50	-17.8143*	6.6884	0.024 *	-33.676	-1.953
		>= 60	-32.7917*	6.9090	0.0005 **	-49.176	-16.407
	30 - 50	>= 60	-14.9774*	4.0117	0.001 **	-24.491	-5.464
** Highly Statistical Significance at p < 0.01 and * Significant at p < 0.05							

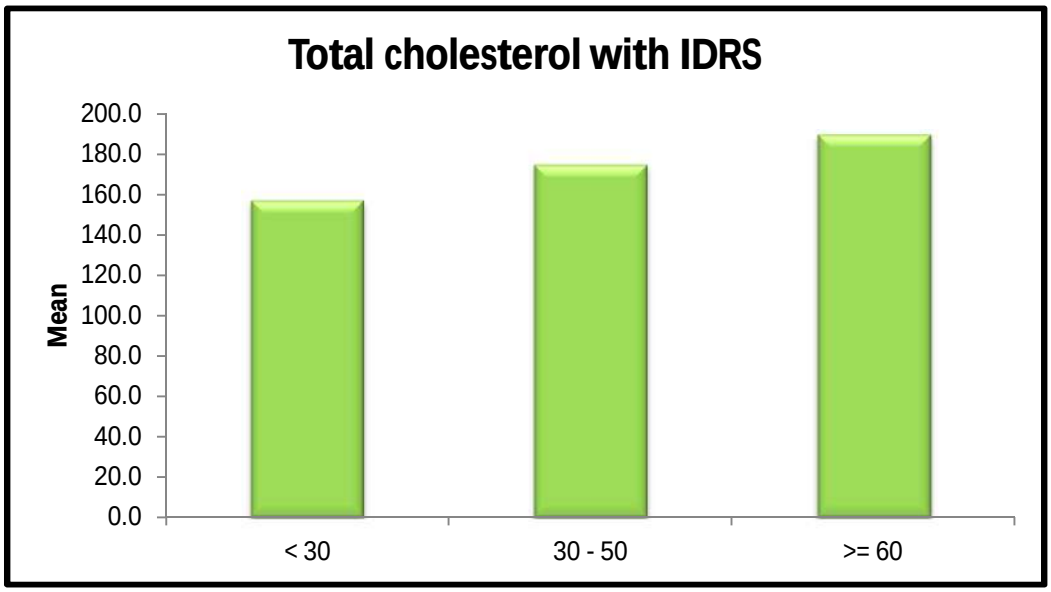


Figure 14(a)

The above table shows the comparison of Total cholesterol with IDRS by using Oneway ANOVA were F-value=13.762, p-value=0.0005<0.01, which shows highly statistical significant difference at p <0.01 level.

Table 14(b): Comparison of Total cholesterol with Fatty liver by Pearson's Chi-Square test

			Fatty liver		Total	χ2- value	p-value
			Positive	Negative			
TC	< 200	Count	28	71	99	20.872	0.0005 **
		%	54.9%	89.9%	76.2%		
	>= 200	Count	23	8	31		
		%	45.1%	10.1%	23.8%		
Total		Count	51	79	130		
		%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level							

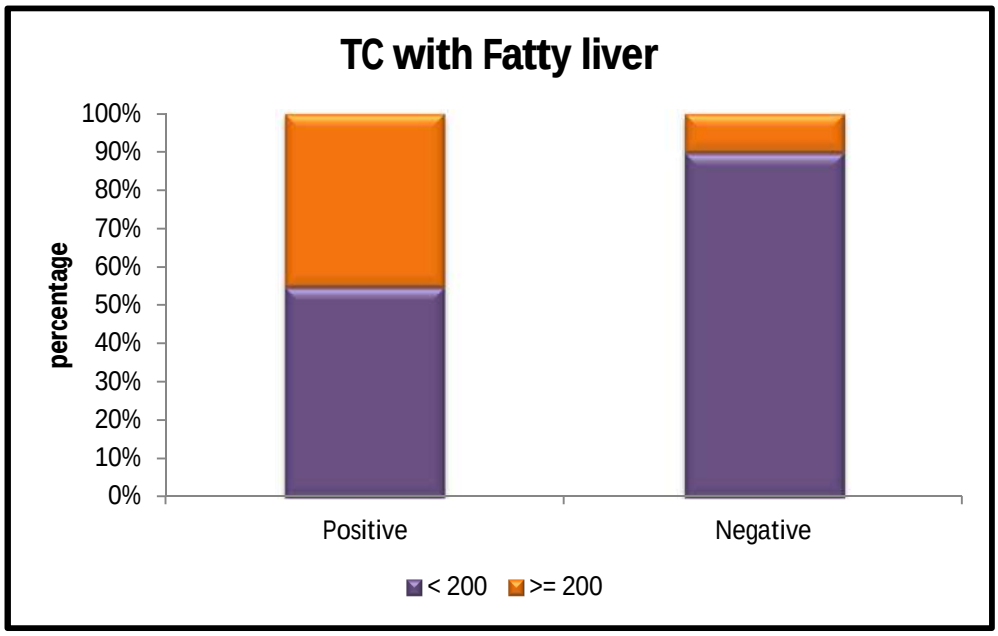


Figure 14(b)

The above table shows comparison between TC with Fatty liver by Pearson's Chi-square test were $\chi 2=20.872$, $p=0.0005<0.01$ which shows highly statistical significant association between TC and Fatty liver.

The figure depicts the percentage of subjects with TC lesser than 200 was maximum with non-fatty liver patients (89.9%) as compared to the fatty liver patients (54.9%). However fatty liver patients were found in greater proportion (45.1%) as compared to non-fatty liver subjects (10.1%) when the TC was greater than 200.

Table 15(a): Comparison of Triglyceride with the IDRS by Oneway ANOVA test

	IDRS	N	Mean	SD	F-value	p-value
Triglyceride	< 30	12	94.0	6.3	17.690	0.0005 **
	30 - 50	70	129.9	42.6		
	>= 60	48	170.4	56.4		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
Triglyceride	< 30	30 - 50	-35.8714*	14.5451	0.040 *	-70.365	-1.378
		>= 60	-76.4167*	15.0250	0.0005 **	-112.049	-40.785
	30 -50	>= 60	-40.5452*	8.7241	0.0005 **	-61.235	-19.856
** Highly Statistical Significance at p < 0.01 and * Significant at p < 0.05							

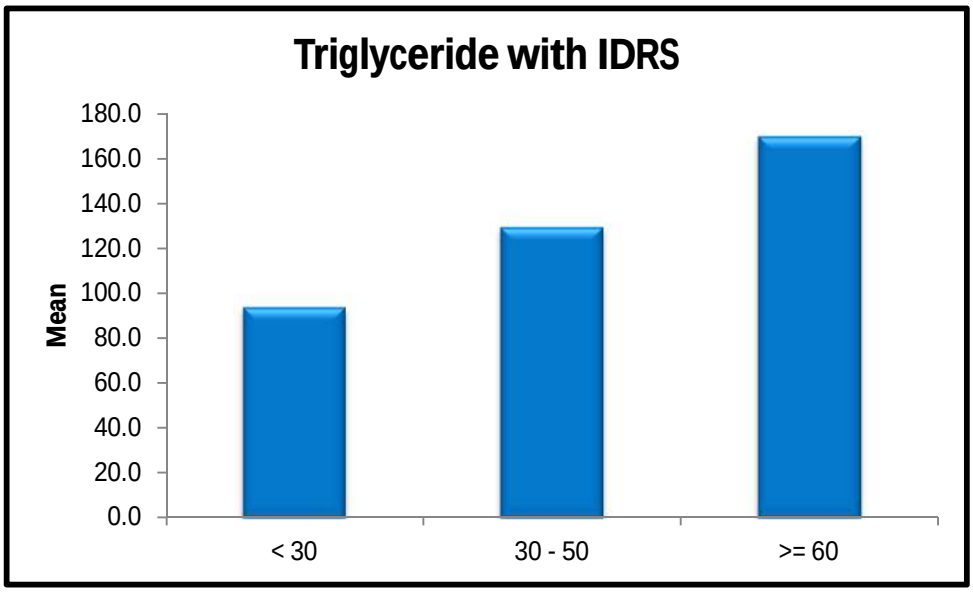


Figure 15(a)

The above table shows the comparison of Triglyceride with IDRS by using Oneway ANOVA were F-value=17.690, p-value=0.0005<0.01, which shows highly statistical significant difference at p <0.01 level.

Table 15(b): Comparison of Triglyceride with Fatty liver by Pearson's Chi-Square test

			Fatty liver		Total	χ2- value	p-value
			Positive	Negative			
TGL	< 150	Count	22	66	88	23.138	0.0005 **
		%	43.1%	83.5%	67.7%		
	>= 150	Count	29	13	42		
		%	56.9%	16.5%	32.3%		
Total		Count	51	79	130		
		%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level							

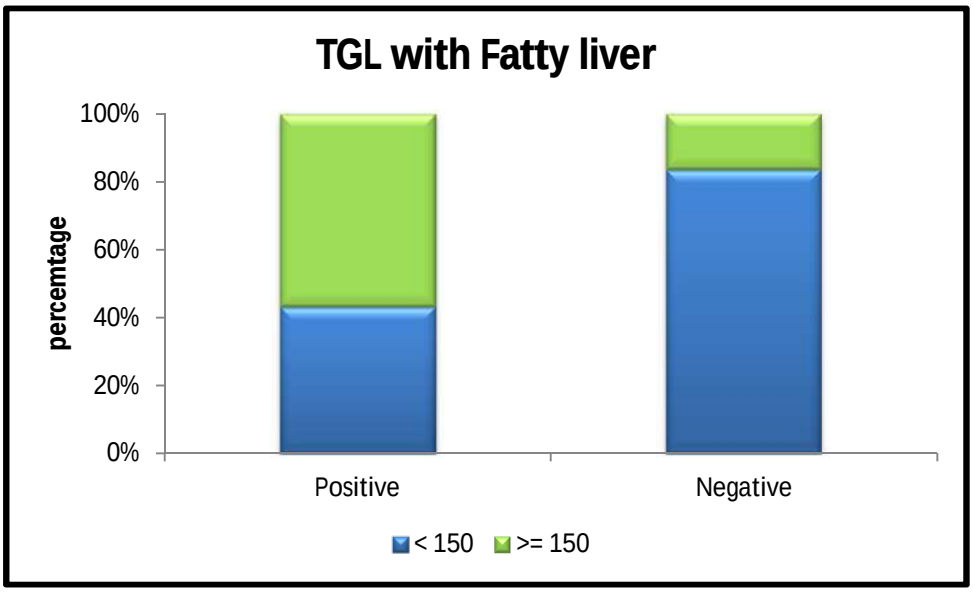


Figure 15(b)

The above table shows comparison between TGL with Fatty liver by Pearson's Chi-square test were $\chi 2=23.138$, $p=0.0005<0.01$ which shows highly statistical significant association between TGL and Fatty liver.

The figure that the shows frequency of TGL below 150 higher was more in the non-fatty liver subjects (83.5%) as compared to the fatty liver patients (43.1%). The subjects with TGL >= 150 were maximum with the fatty liver subjects (56.9%) as compared to the non-fatty liver subjects (16.5%).

Table 16(a): Comparison of HDL with the IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
HDL	< 30	12	41.7	4.0	6.166	0.003 **
	30 - 50	70	42.7	4.8		
	>= 60	48	39.8	3.5		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
HDL	< 30	30 - 50	-.9905	1.3415	0.741 #	-4.172	2.191
		>= 60	1.8333	1.3857	0.385 #	-1.453	5.120
	30 - 50	>= 60	2.8238*	.8046	0.002 **	.916	4.732
** Highly Statistical Significant at p < 0.01 and # No Significant at p > 0.05							

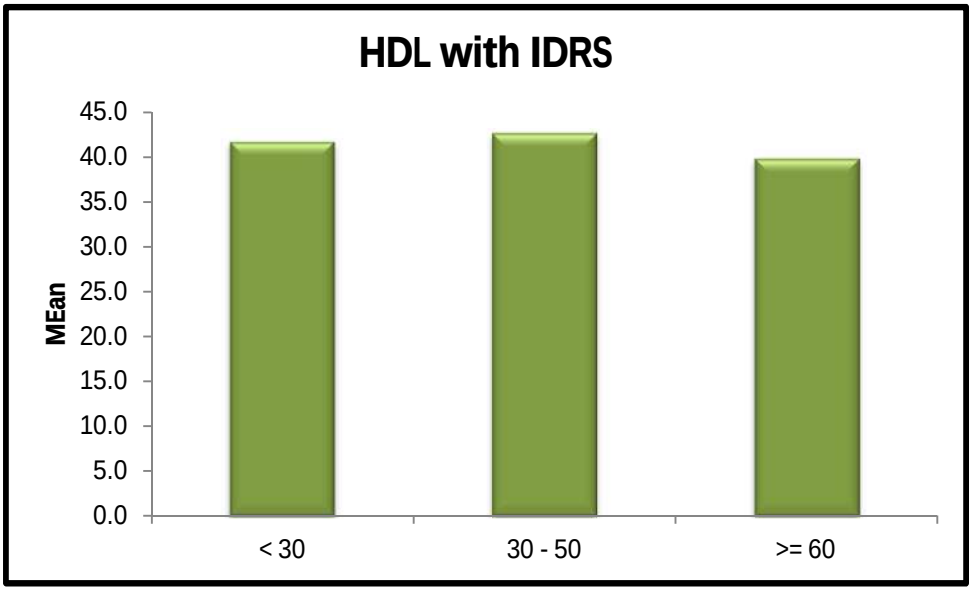


Figure 16(a)

The above table shows the comparison of HDL with IDRS by using Oneway ANOVA were F-value=6.166, p-value=0.003<0.01, which shows highly statistical significant difference at p <0.01 level.

Table 16(b): Comparison of HDL with Fatty liver by Pearson's Chi-Square test.

			Fatty liver		Total	χ2- value	p-value
			Positive	Negative			
HDL	< 40	Count	33	22	55	17.249	0.0005 **
		%	64.7%	27.8%	42.3%		
	>= 40	Count	18	57	75		
		%	35.3%	72.2%	57.7%		
Total		Count	51	79	130		
		%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level							

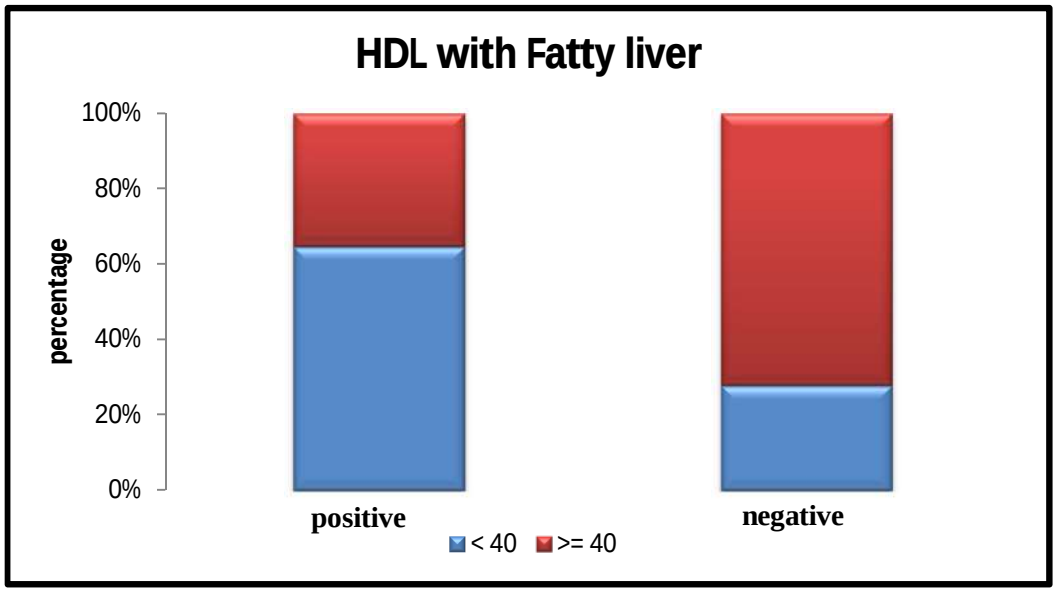


Figure 16(b)

The above table shows comparison between HDL with Fatty liver by Pearson's Chi-square test were $\chi 2=17.249$, $p=0.0005<0.01$ which shows highly statistical significant association between HDL and Fatty liver.

Among the fatty liver group majority of the subjects had HDL less than 40, whereas among the non-fatty liver patients majority of them had HDL greater than 40.

Table 17(a): Comparison of LDL with the IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
LDL	< 30	12	97.0	8.6	4.28	0.016 *
	30 - 50	70	108.1	18.0		
	>= 60	48	116.4	29.0		
* Statistical Significant at p < 0.05 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
LDL	< 30	30 - 50	-11.0381	6.9429	0.254 #	-27.503	5.427
		>= 60	-19.3417*	7.1720	0.022 *	-36.350	-2.333
	30 - 50	>= 60	-8.3036	4.1643	0.118 #	-18.179	1.572
* Significant at p < 0.05 and # No Statistical Significance at p > 0.05							

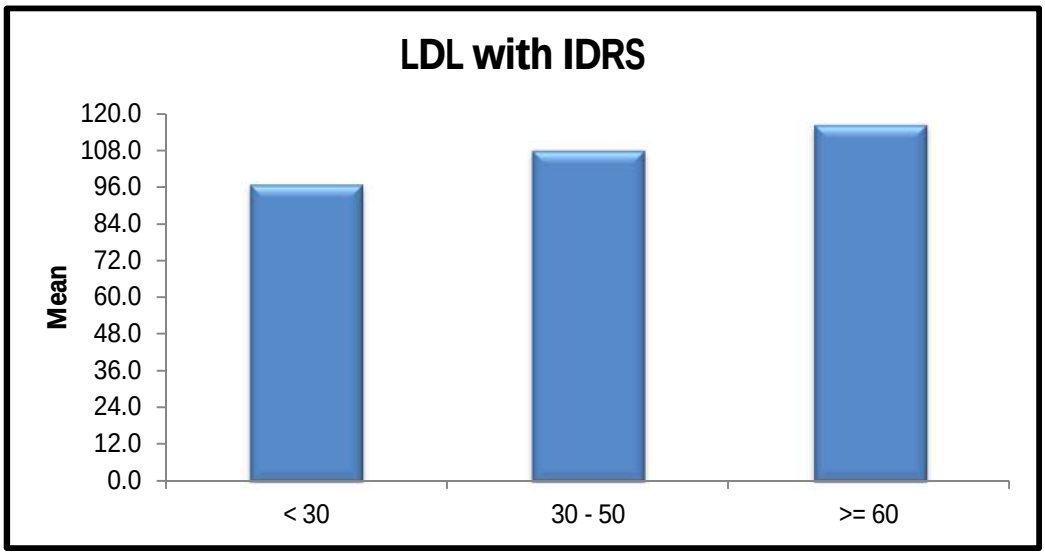


Figure 17(a)

The above table shows the comparison of LDL with IDRS by using Oneway ANOVA were F-value=4.280, p-value=0.016<0.05, which shows statistical significant difference at p <0.05 level.

Table 17(b): Comparison of LDL with Fatty liver by Pearson's Chi-Square test.

			Fatty liver		Total	χ2-value	p-value
			Positive	Negative			
LDL	< 100	Count	13	50	63	17.731	0.0005 **
		%	25.5%	63.3%	48.5%		
	>= 100	Count	38	29	67		
		%	74.5%	36.7%	51.5%		
Total		Count	51	79	130		
		%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level							

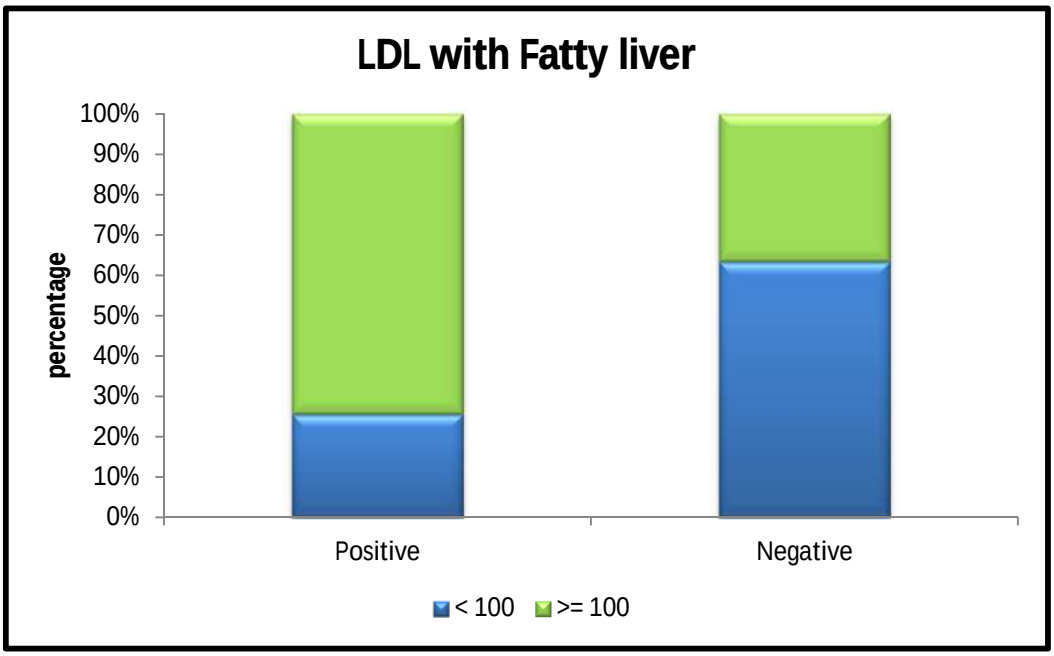


Figure 17(b)

The above table shows comparison between LDL with Fatty liver by Pearson's Chi-square test were $\chi 2=17.731$, $p=0.0005<0.01$ which shows highly statistical significant association between LDL and Fatty liver.

In this study, LDL values were derived from the measured values of total cholesterol, triglyceride and HDL. Among the fatty liver group majority of the subjects had LDL greater than 100 (74.5%), whereas among the non-fatty liver patients majority of them had LDL lesser than 100 (63.3%).

Table 18: Comparison of Total bilirubin with the IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
Total bilirubin	< 30	12	0.8	0.1	0.292	0.747 #
	30 - 50	70	0.8	0.1		
	>= 60	48	0.8	0.1		
# No Statistical Significance at p > 0.05 level						

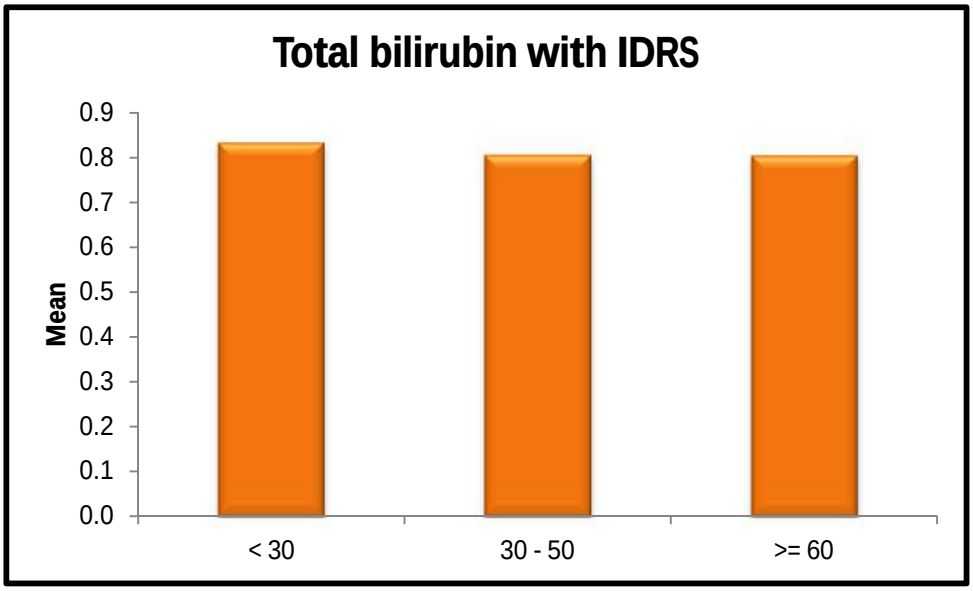


Figure 18

The above table shows the comparison of Total bilirubin with IDRS by using Oneway ANOVA were F-value=0.292, p-value=0.747>0.05, which shows no statistical significant difference at p >0.05 level.

Table 19: Comparison of Direct bilirubin between the IDRS by Oneway ANOVA test

	IDRS	N	Mean	SD	F-value	p-value
Direct bilirubin	< 30	12	0.3	0.0	1.038	0.357 #
	30 - 50	70	0.3	0.1		
	>= 60	48	0.3	0.1		
# No Statistical Significance at p > 0.05 level						

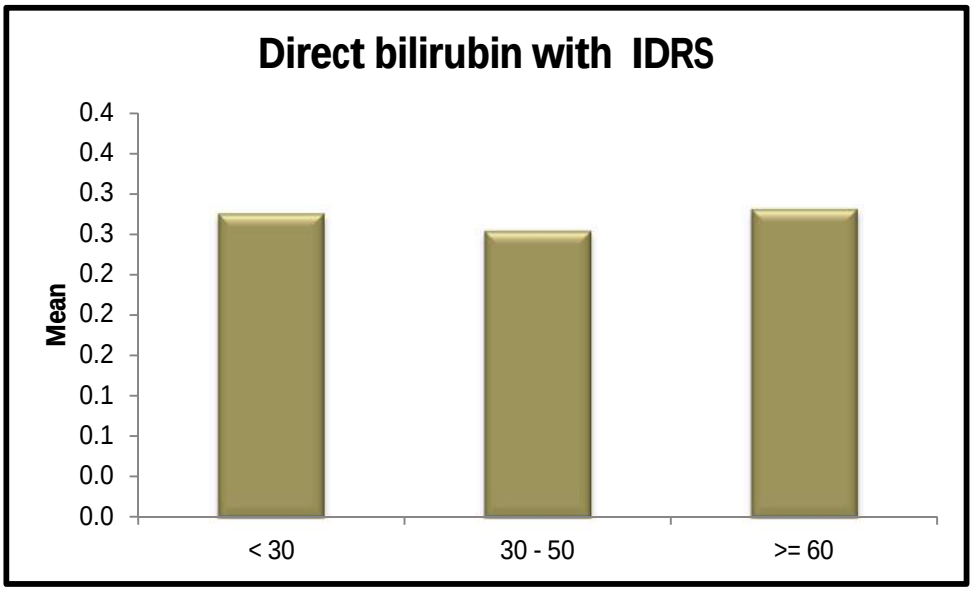


Figure 19

The above table shows the comparison of Direct bilirubin with IDRS by using Oneway ANOVA were F-value=1.038, p-value=0.357>0.05, which shows no statistical significant difference at p >0.05 level.

Table 20(a): Comparison of AST between the IDRS by Oneway ANOVA test

	IDRS	N	Mean	SD	F-value	p-value
AST	< 30	12	28.0	5.2	8.151	0.0005 **
	30 - 50	70	29.1	6.4		
	>= 60	48	33.3	5.6		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
AST	< 30	30 - 50	-1.1429	1.8769	0.816 #	-5.594	3.308
		>= 60	-5.3333*	1.9389	0.019 *	-9.931	-.735
	30 - 50	>= 60	-4.1905*	1.1258	0.001 **	-6.860	-1.521
** Highly Statistical Significance at p < 0.01 ,* Significant at p < 0.05 and # No Statistical Significance at p > 0.05							

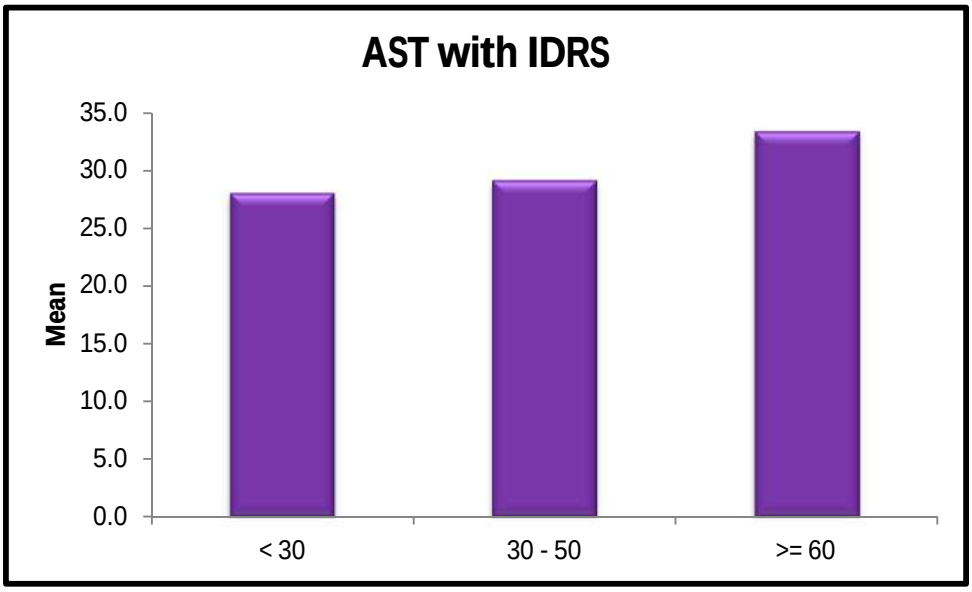


Figure 20(a)

The above table shows the comparison of AST with IDRS by using Oneway ANOVA were F-value=8.151, p-value=0.0005<0.01, which shows highly statistical significant difference at p <0.01 level.

Table 20(b): Comparison of AST with Fatty liver by Pearson's Chi-Square test

			Fatty liver		Total	χ2-value	p-value
			Positive	Negative			
AST	< 30	Count	15	51	66	15.316	0.0005 **
		%	29.4%	64.6%	50.8%		
	>= 30	Count	36	28	64		
		%	70.6%	35.4%	49.2%		
Total		Count	51	79	130		
		%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level							

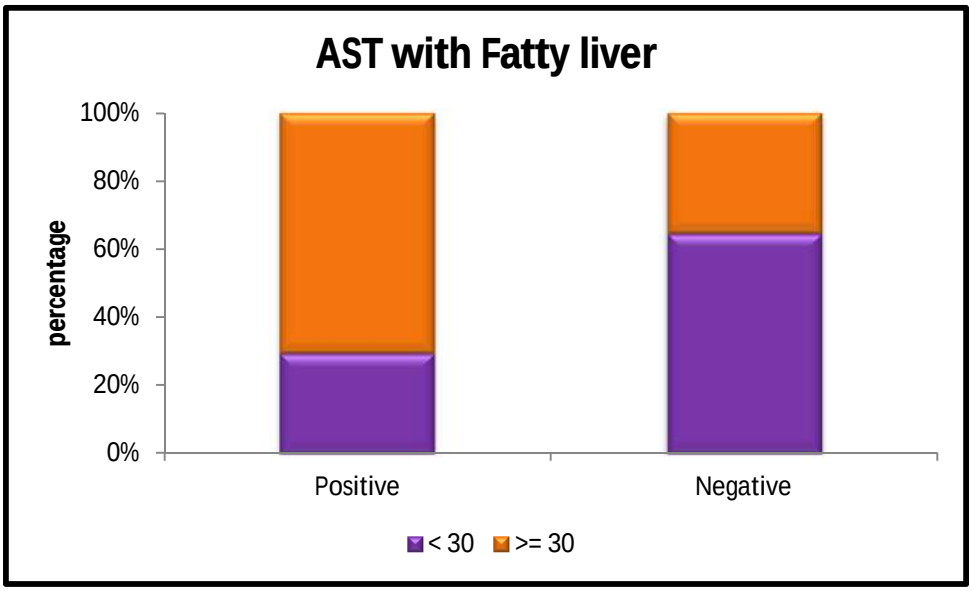


Figure 20(b)

The above table shows comparison between AST with Fatty liver by Pearson's Chi-square test were χ^2=15.316, p=0.0005<0.01 which shows highly statistical significant association between AST and Fatty liver.

Among the fatty liver group most of the subjects had AST greater than 30 (70.6%), whereas among the non-fatty liver patients majority of them had AST lesser than 30 (64.6%).

Table 21(a): Comparison of ALT with the IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
ALT	< 30	12	30.5	4.9	7.168	0.0005 **
	30 - 50	70	34.8	10.4		
	>= 60	48	40.0	8.2		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
ALT	< 30	30 - 50	-4.3000	2.8975	0.302 #	-11.171	2.571
		>= 60	-9.5417*	2.9931	0.005 **	-16.640	-2.444
	30 - 50	>= 60	-5.2417*	1.7379	0.009 **	-9.363	-1.120
** Highly Statistical Significant at p < 0.01 and # No Significant at p > 0.05							

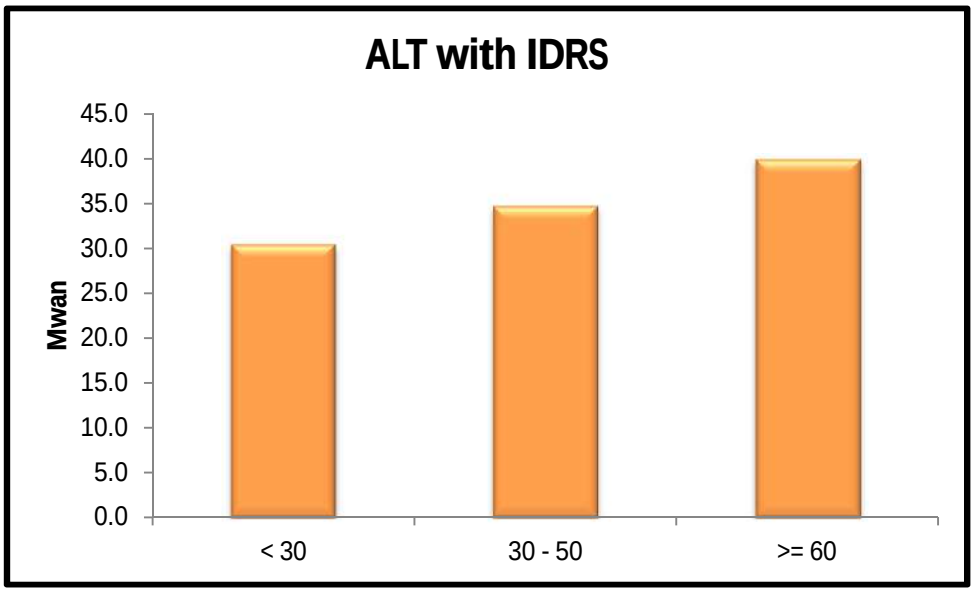


Figure 21(a)

The above table shows the comparison of ALT with IDRS by using Oneway ANOVA were F-value=7.168, p-value=0.0005<0.01, which shows highly statistical significant difference at p <0.01 level.

Table 21(b): Comparison of ALT with Fatty liver by Pearson's Chi-Square test

			Fatty liver		Total	χ2-value	p-value
			Positive	Negative			
ALT	< 40	Count	26	71	97	24.751	0.0005 **
		%	51.0%	89.9%	74.6%		
	>= 40	Count	25	8	33		
		%	49.0%	10.1%	25.4%		
Total		Count	51	79	130		
		%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level							

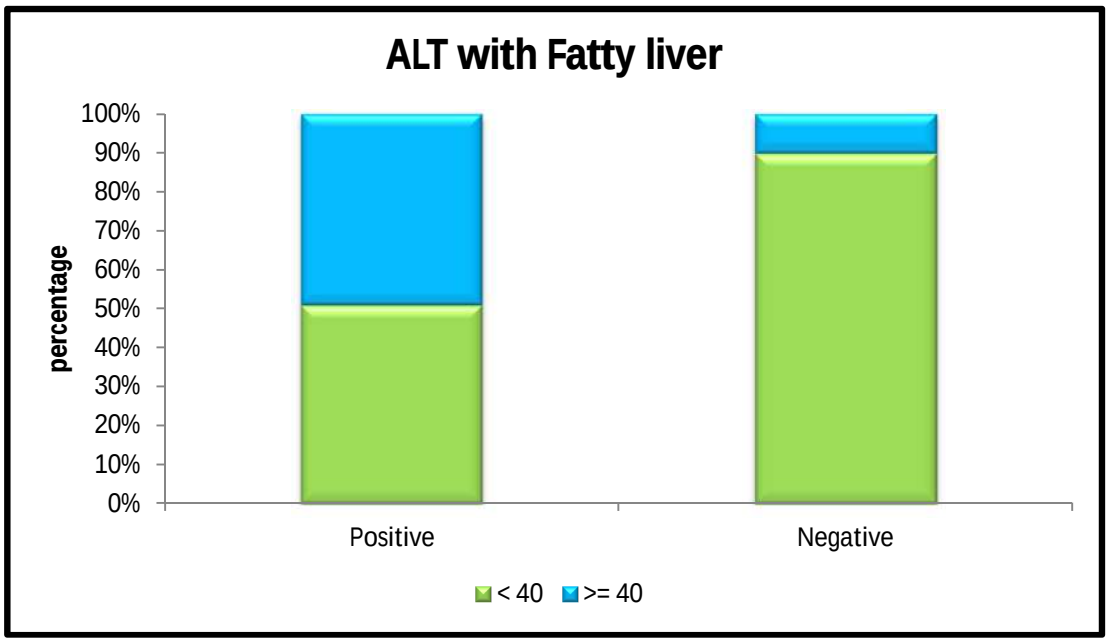


Figure 21(b)

The above table shows comparison between ALT with Fatty liver by Pearson's Chi-square test were $\chi 2=24.751$, $p=0.0005<0.01$ which shows highly statistical significant association between ALT and Fatty liver.

Among the non-fatty liver patients majority of them had ALT lesser than 40 (89.9%), whereas among the fatty liver group 51% had ALT less than 40 and 49% had ALT greater than 40.

Table 22: Comparison of AST/ALT between the IDRS by Oneway ANOVA test

	IDRS	N	Mean	SD	F-value	p-value
AST/ALT	< 30	12	0.9	0.0	1.363	0.260 #
	30 - 50	70	0.9	0.1		
	>= 60	48	0.8	0.1		
# No Statistical Significance at p > 0.05 level						

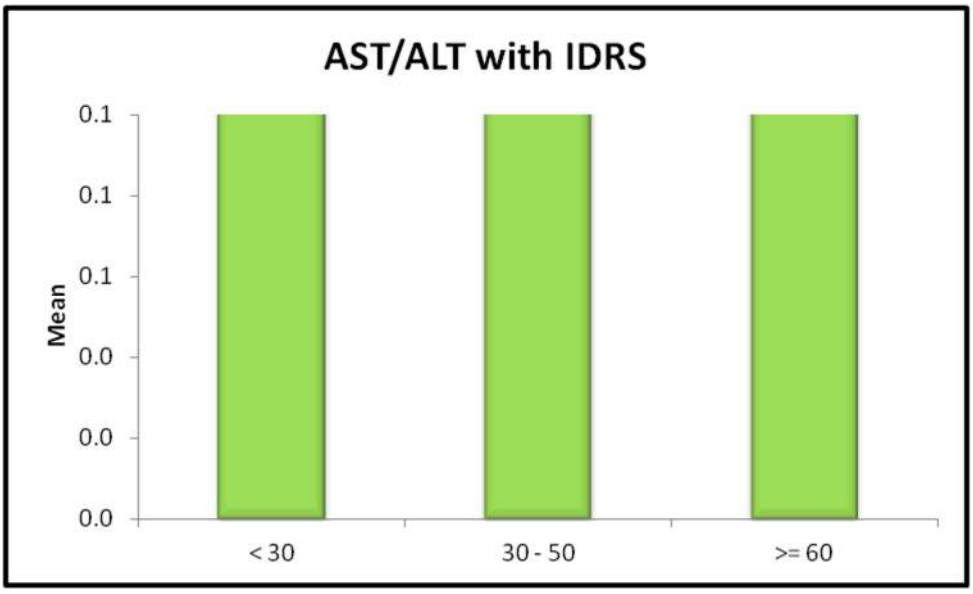


Figure 22

The above table shows the comparison of AST/ALT with IDRS by using Oneway ANOVA were F-value=1.363, p-value=0.260>0.05, which shows no statistical significant difference at p >0.05 level.

Table 23(a): Comparison of ALP with the IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
ALP	< 30	12	143.8	17.9	11.399	0.0005 **
	30 - 50	70	160.9	18.1		
	>= 60	48	174.0	26.0		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
ALP	< 30	30 - 50	-17.0810*	6.6675	0.031 *	-32.893	-1.269
		>= 60	-30.2083*	6.8874	0.0005 **	-46.542	-13.875
	30 - 50	>= 60	-13.1274*	3.9991	0.004 **	-22.611	-3.643
** Highly Statistical Significance at p < 0.01 and * Significant at p < 0.05							

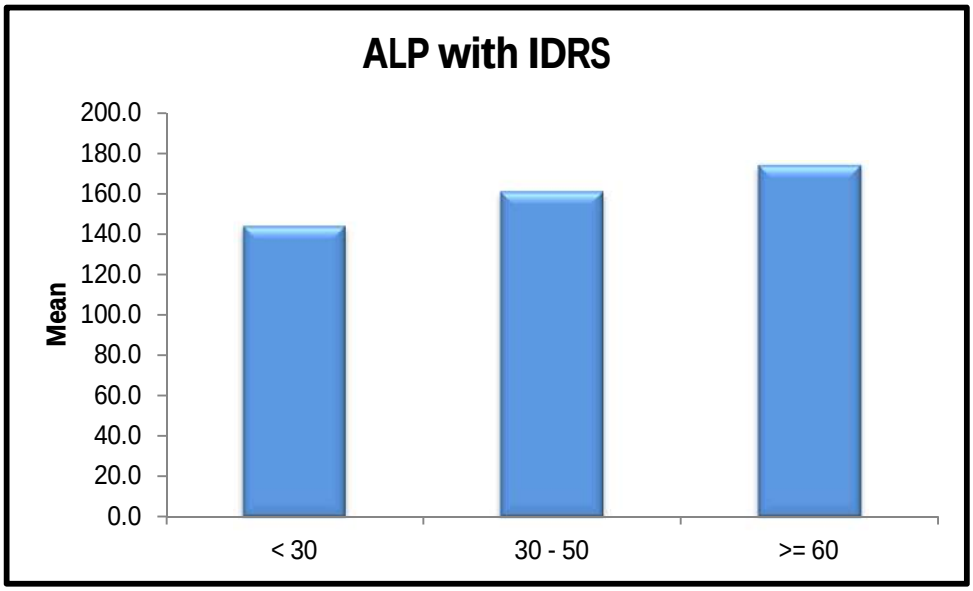


Figure 23(a)

The above table shows the comparison of ALP with IDRS by using Oneway ANOVA were F-value=11.399, p-value=0.0005<0.01, which shows highly statistical significant difference at p <0.01 level.

Table 23(b): Comparison of ALP with Fatty liver by Pearson's Chi-Square test

			Fatty liver		Total	χ2- value	p-value
			Positive	Negative			
ALP	< 150	Count	14	39	53	6.165	0.013 *
		%	27.5%	49.4%	40.8%		
	>= 150	Count	37	40	77		
		%	72.5%	50.6%	59.2%		
Total		Count	51	79	130		
		%	100.0%	100.0%	100.0%		
* Statistical Significance at p < 0.05 level							

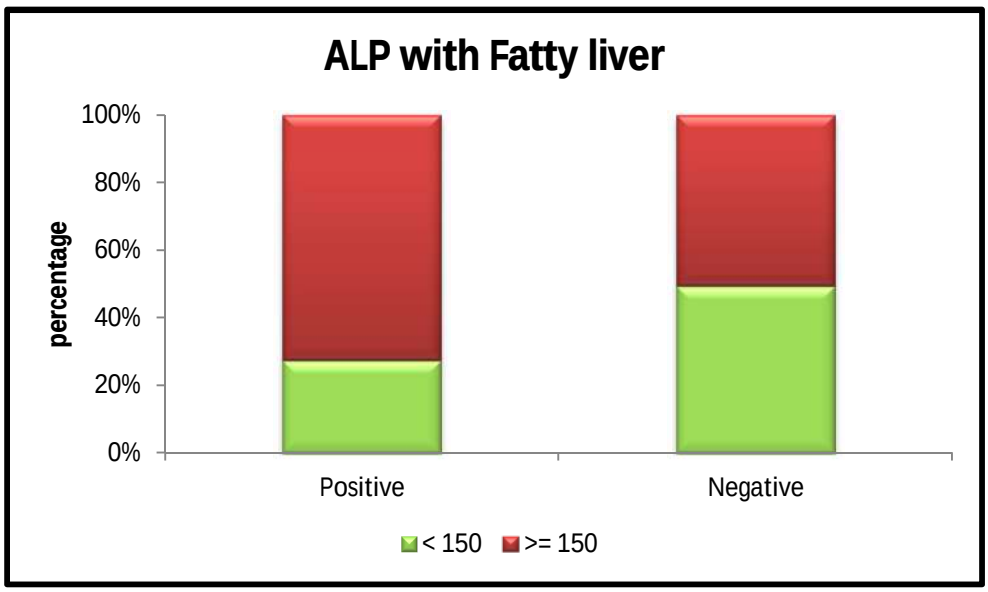


Figure 23(b)

The above table shows comparison between ALP with Fatty liver by Pearson's Chi-square test were $\chi 2=6.165$, $p=0.013<0.05$ which shows statistical significant association between ALP and Fatty liver.

Among the fatty liver patients most of them had ALP greater than or equal to 150 (72.5%), whereas among the non-fatty liver group 49.4% had ALP less than 150 and 50.6% had ALP greater than 150.

Table 24(a): Comparison of GGT with the IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
GGT	< 30	12	30.0	4.6	5.954	0.0005 **
	30 - 50	70	30.1	5.0		
	>= 60	48	33.1	4.8		
** Highly Statistical Significant at p < 0.01 level						

Dependent Variable			MD (I-J)	Std. Error	p-value	95% C.I	
						LB	UB
GGT	< 30	30 - 50	-.0714	1.5163	0.989 #	-3.667	3.524
		>= 60	-3.1042	1.5663	0.121 #	-6.819	.610
	30 - 50	>= 60	-3.0327*	.9095	0.003 **	-5.190	-.876
** Highly Statistical Significant at p < 0.01 and # No Significant at p > 0.05							

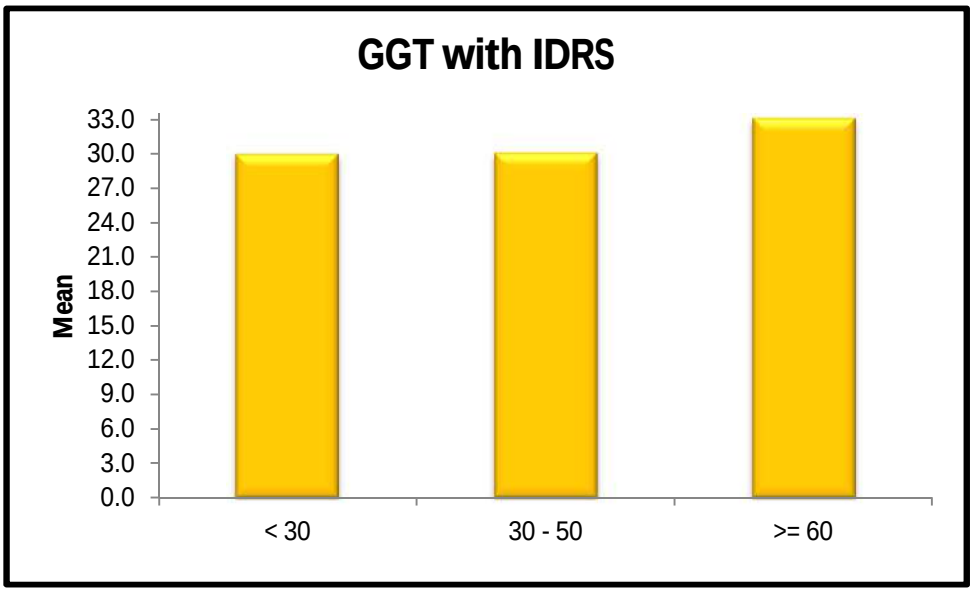


Figure 24(a)

The above table shows the comparison of GGT with IDRS by using Oneway ANOVA were F-value=5.954, p-value=0.0005<0.01, which shows highly statistical significant difference at p <0.01 level.

Table 24(b): Comparison of GGT with Fatty liver by Pearson's Chi-Square test.

			Fatty liver		Total	χ^2-value	p-value
			Positive	Negative			
GGT	< 30	Count	16	48	64	10.708	0.001 **
		%	31.4%	60.8%	49.2%		
	>= 30	Count	35	31	66		
		%	68.6%	39.2%	50.8%		
Total		Count	51	79	130		
		%	100.0%	100.0%	100.0%		
** Highly Statistical Significance at p < 0.01 level							

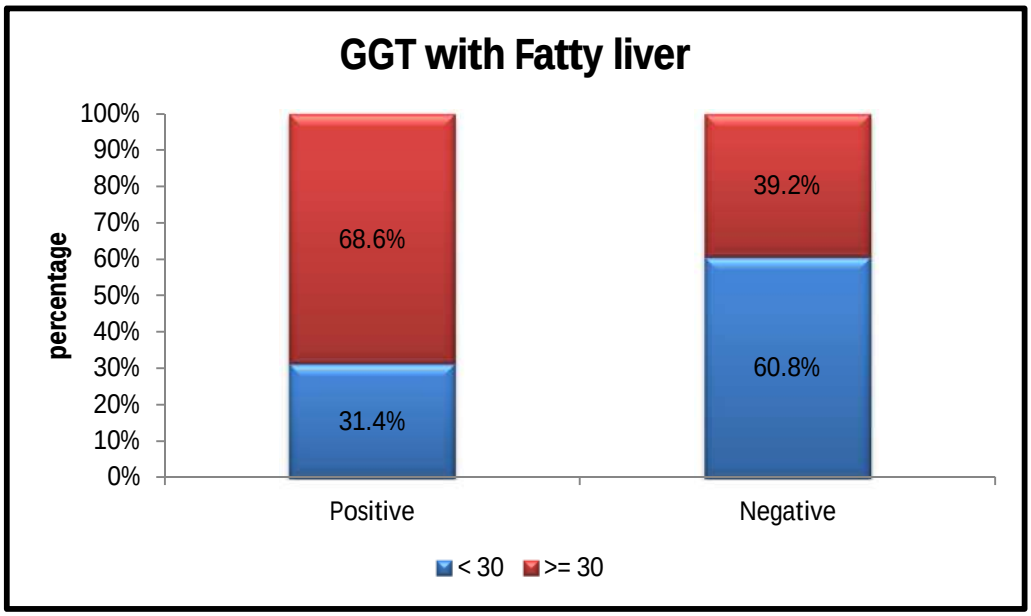


Figure 24(b)

The above table shows comparison between GGT with Fatty liver by Pearson's Chi-square test were $\chi 2=10.708$, $p=0.001<0.01$ which shows highly statistical significant association between GGT and Fatty liver.

Among the fatty liver group majority of the subjects had GGT greater than or equal to 30 (68.6%), whereas among the non-fatty liver patients more than half of them had GT lesser than 30 (60.8%).

Table 25: Comparison of Total protein with the IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
Total protein	< 30	12	7.8	0.2	0.056	0.945 #
	30 - 50	70	7.8	0.3		
	>= 60	48	7.8	0.3		
# No Statistical Significance at p > 0.05 level						

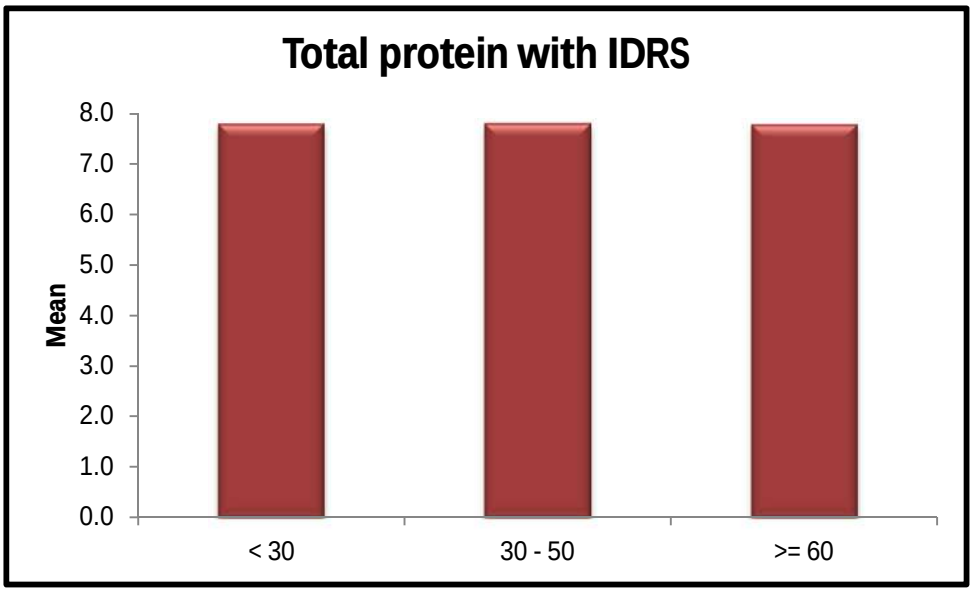


Figure 25

The above table shows the comparison of Total protein with IDRS by using Oneway ANOVA were F-value=0.056, p-value=0.945>0.05, which shows no statistical significant difference at p >0.05 level.

Table 26: Comparison of Albumin with the IDRS by Oneway ANOVA test.

	IDRS	N	Mean	SD	F-value	p-value
Albumin	< 30	12	4.0	0.3	0.53	0.590 #
	30 - 50	70	4.0	0.2		
	>= 60	48	3.9	0.2		
# No Statistical Significance at p > 0.05 level						

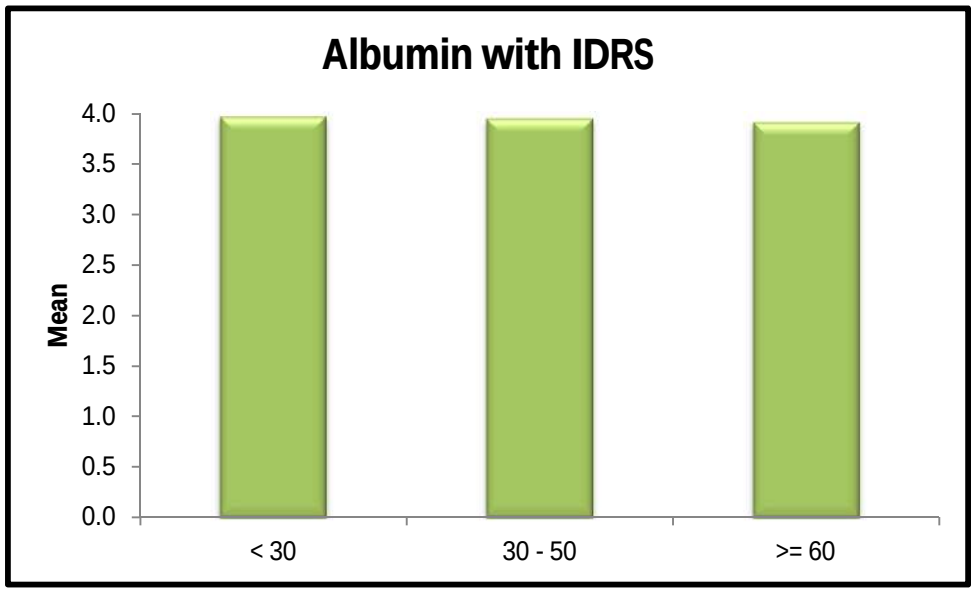


Figure 26

The above table shows the comparison of Albumin with IDRS by using Oneway ANOVA were F-value=0.530, p-value=0.590>0.05, which shows no statistical significant difference at p >0.05 level.

RESULTS

- The Age distributions were <21-30 years - 25.4%, 31-40 years - 44.6%, 41-50 years - 20.8% and >50 years - 9.2%.
- The Gender distribution were Female -48.5%, Male - 51.5%.
- The Comorbidities distributions were hypothyroidism - 8.5%, PCOS - 1.5%, SHT - 4.6%, SHT with CKD - 1.5% and SHT together with hypothyroidism - 1.5%. Majority of the study population had no comorbidities which constitute 82.3%.
- The IDRS Score distributions were < 30 is 9.2%, 30 – 50 is 53.8%, >= 60 are 36.9%.
- The comparison of Gender with the IDRS, which shows that there was highly statistical significant association between Gender and IDRS.
- The comparison of IDRS with the USG, which shows that there was highly statistical significant association between IDRS and USG.
- The comparison of BMI with the IDRS, which shows that there was highly statistical significant difference at p <0.01 level.
- The comparison of BMI with the Fatty liver, which shows that there was highly statistical significant association between BMI and Fatty liver.
- The comparison of Waist Circumference with the IDRS, which shows that there was highly statistical significant difference at p <0.01 level.
- The comparison of waist hip ratio with the Fatty liver, which shows that there was highly statistical significant association between waist hip ratio and Fatty liver.

- The comparison of SBP with the IDRS, which shows that there was highly statistical significant difference at p <0.01 level.
- The comparison of DBP with the IDRS, which shows that there was highly statistical significant difference at p <0.01 level.
- The comparison of FBS with the IDRS, which shows that there was highly statistical significant difference at p <0.01 level.
- The comparison of FBS with the Fatty liver, which shows that there was highly statistical significant association between FBS and Fatty liver.
- The comparison of HBA1C with the IDRS, which shows that there was highly statistical significant difference at p <0.01 level.
- The comparison of HBA1C with the Fatty liver, which shows that there was highly statistical significant association between HBA1C and Fatty liver.
- The comparison of Total cholesterol with the IDRS, which shows that there was highly statistical significant difference at p <0.01 level.
- The comparison of Total cholesterol with the Fatty liver, which shows that there was highly statistical significant association between Total cholesterol and Fatty liver.
- The comparison of Triglyceride with the IDRS, which shows that there was highly statistical significant difference at p <0.01 level.
- The comparison of TGL with the Fatty liver, which shows that there was highly statistical significant association between TGL and Fatty liver.

- The comparison of HDL with the IDRS, which shows that there was highly statistical significant difference at $p < 0.01$ level.
- The comparison of HDL with the Fatty liver, which shows that there was highly statistical significant association between HDL and Fatty liver.
- The comparison of LDL with the IDRS, which shows that there was statistical significant difference at $p < 0.05$ level.
- The comparison of LDL with the Fatty liver, which shows that there was highly statistical significant association between LDL and Fatty liver.
- The comparison of Total bilirubin with the IDRS, which shows that there was no statistical significant difference at $p > 0.05$ level.
- The comparison of Direct bilirubin with the IDRS, which shows that there was no statistical significant difference at $p > 0.05$ level.
- The comparison of AST with the IDRS, which shows that there was highly statistical significant difference at $p < 0.01$ level.
- The comparison of AST with the Fatty liver, which shows that there was highly statistical significant association between AST and Fatty liver.
- The comparison of ALT with the IDRS, which shows that there was highly statistical significant difference at $p < 0.01$ level.
- The comparison of ALT with the Fatty, which shows that there was highly statistical significant association between ALT and Fatty liver.
- The comparison of AST/ALT with the IDRS, which shows that there was no statistical significant difference at $p > 0.05$ level.

- The comparison of ALP with the IDRS, which shows that there was highly statistical significant difference at $p < 0.01$ level.
- The comparison of ALP with the Fatty liver, which shows that there was statistical significant association between ALP and Fatty liver.
- The comparison of GGT with the IDRS, which shows that there was highly statistical significant difference at $p < 0.01$ level.
- The comparison of GGT with the Fatty liver, which shows that there was highly statistical significant association between GGT and Fatty liver.
- The comparison of Total protein with the IDRS, which shows that there was no statistical significant difference at $p > 0.05$ level.
- The comparison of Albumin with the IDRS, which shows that there was no statistical significant difference at $p > 0.05$ level.

DISCUSSION

The overall prevalence of NAFLD among the 130 subjects was 39.2%. Indian study conducted by ***Anbalagan et al***(25) showed a prevalence of 24.7%.

The mean age in the study was found to be 38 ± 9 years. Similarly in the study conducted by ***Anbalagan et al***(25) showed mean age of 40 ± 11.9 years. In some IDRS scores, age factor has contributed to a high value which implies that the prevalence,of NAFLD increases with age.

The gender distribution of the study is 51.5% males and 48.5% females. Of the 51 individuals with fatty liver, 56.8% were males and 43.8% were females. Most relevant studies have reported NAFLD to be common,in men than women with a later peaking prevalence for advanced disease in postmenopausal women. Findings were similar to study conducted on the basis of ultrasound findings and biopsy, ***Singh SP et al., Amarapurkar et al., and Bahrami et al.,*** (26–28) showed male predominance in NAFLD,in their respective studies.

The mean body mass index of the study were 22.4 ±0.5 in low risk , 23.4 ±1.4 in medium risk and 25.4 ±1.2 kg/m^2 among the high risk group. Higher BMI was associated with higher score. Similar findings were found in the study conducted by ***Anbalagan et al.*** (25)

The main finding of the,study was higher prevalence of,NAFLD among individuals with,high risk IDRS group (52.9%) as compared with individuals with medium (39.2%) and low risk scores (7.8%). Findings were similar to the study conducted by ***Anbalagan et al***(25) which was published in the journal of

diabetes,science and technology. It shows that IDRS was independently associated with the ultrasonic finding of fatty liver.

Similar study carried out in the Indian population also validates the finding. ***Mori et al., published in journal of clinical and diagnostic research,***(29) has inferred that IDRS has statistical significant,association with the NAFLD and that it can be used as a screening tool for NAFLD.

CONCLUSION

There are many clinical scoring systems for NAFLD. However this study shows that a simple clinical tool which was validated originally to identify undiagnosed diabetes in population could be used as a screening tool for identify people with high risk for NAFLD.

Since NAFLD is associated with many risk factors which are primarily cardiometabolic like obesity, dyslipidemia, diabetes and hypertension, it also helps to identify cardiovascular disease and metabolic syndrome in the population.

Therefore IDRS scoring is a cost effective tool, which contains only four clinical parameters- age, family history, waist circumference and daily physical activity of the individual, to be used as screening tool to identify high risk individuals for NAFLD where resources are limited. Those individuals with high scores could then be subjected,to more definitive tests to confirm the presence of NAFLD.

LIMITATIONS

- Although ultrasound was sensitive in diagnosing fatty liver, its accuracy would be higher if more than 30 percent of liver has steatosis. As many studies have been conducted based on simple ultrasonogram, and so this is based on those lines.
- Liver biopsy which is gold standard for diagnosis, could not be done as it was not feasible in patients attending outpatient clinics.
- Certain investigations like fasting insulin could not be done in our setup to calculate HOMA-IR and demonstrate hyperinsulinemia.

BIBLIOGAPHY

1. Younossi Z, Anstee QM, Marietti M, Hardy T, Henry L, Eslam M, et al. Global burden of NAFLD and NASH: trends, predictions, risk factors and prevention. Nat Rev Gastroenterol Hepatol. 2018 Jan;15(1):11–20.

2. Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M. Global epidemiology of nonalcoholic fatty liver disease-Meta-analytic assessment of prevalence, incidence, and outcomes. Hepatol Baltim Md. 2016 Jul;64(1):73–84.

3. Younossi Z, Stepanova M, Ong JP, Jacobson IM, Bugianesi E, Duseja A, et al. Nonalcoholic Steatohepatitis Is the Fastest Growing Cause of Hepatocellular Carcinoma in Liver Transplant Candidates. Clin Gastroenterol Hepatol Off Clin Pract J Am Gastroenterol Assoc. 2019 Mar;17(4):748-755.e3.

4. Wong RJ, Aguilar M, Cheung R, Perumpail RB, Harrison SA, Younossi ZM, et al. Nonalcoholic steatohepatitis is the second leading etiology of liver disease among adults awaiting liver transplantation in the United States. Gastroenterology. 2015 Mar;148(3):547–55.

5. Machado M, Marques-Vidal P, Cortez-Pinto H. Hepatic histology in obese patients undergoing bariatric surgery. J Hepatol. 2006 Oct;45(4):600–6.

6. Ogden CL, Carroll MD, Curtin LR, Lamb MM, Flegal KM. Prevalence of high body mass index in US children and adolescents, 2007-2008. JAMA. 2010 Jan 20;303(3):242–9.

7. Torres DM, Williams CD, Harrison SA. Features, diagnosis, and treatment of nonalcoholic fatty liver disease. Clin Gastroenterol Hepatol Off Clin Pract J Am Gastroenterol Assoc. 2012 Aug;10(8):837–58.

8. Ruhl CE, Everhart JE. Epidemiology of nonalcoholic fatty liver. Clin Liver Dis. 2004 Aug;8(3):501–19, vii.

9. Targher G, Bertolini L, Padovani R, Rodella S, Tessari R, Zenari L, et al. Prevalence of nonalcoholic fatty liver disease and its association with cardiovascular disease among type 2 diabetic patients. Diabetes Care. 2007 May;30(5):1212–8.

10. Duseja A, Aggarwal R. APOC3 and PNPLA3 in non-alcoholic fatty liver disease: Need to clear the air: Editorials. J Gastroenterol Hepatol. 2012 May;27(5):848–51.

11. Singh S, Allen AM, Wang Z, Prokop LJ, Murad MH, Loomba R. Fibrosis progression in nonalcoholic fatty liver vs nonalcoholic steatohepatitis: a systematic review and meta-analysis of paired-biopsy studies. Clin Gastroenterol Hepatol Off Clin Pract J Am Gastroenterol Assoc. 2015 Apr;13(4):643-654.e1-9; quiz e39-40.

12. Pais R, Charlotte F, Fedchuk L, Bedossa P, Lebray P, Poynard T, et al. A systematic review of follow-up biopsies reveals disease progression in patients with non-alcoholic fatty liver. J Hepatol. 2013 Sep;59(3):550–6.

13. Fazel Y, Koenig AB, Sayiner M, Goodman ZD, Younossi ZM. Epidemiology and natural history of non-alcoholic fatty liver disease. Metabolism. 2016 Aug;65(8):1017–25.

14. McPherson S, Hardy T, Henderson E, Burt AD, Day CP, Anstee QM. Evidence of NAFLD progression from steatosis to fibrosing-steatohepatitis using paired biopsies: implications for prognosis and clinical management. J Hepatol. 2015 May;62(5):1148–55.

15. Brodosi L, Barbanti FA, Petroni ML, Marchignoli F, Marchesini G. Obesity and NAFLD: Same Problem? In: Bugianesi E, editor. Non-Alcoholic Fatty Liver Disease [Internet]. Cham: Springer International Publishing; 2020 [cited 2021 Dec 18]. p. 1–14. Available from: http://link.springer.com/10.1007/978-3-319-95828-6_1

16. Gastaldelli A. NAFLD and Insulin Resistance: A Multisystemic Disease. In: Bugianesi E, editor. Non-Alcoholic Fatty Liver Disease [Internet]. Cham: Springer International Publishing; 2020 [cited 2021 Dec 18]. p. 49–71. Available from: http://link.springer.com/10.1007/978-3-319-95828-6_4

17. Miele L, Biolato M, Conte C, Mangiola F, Liguori A, Gasbarrini A, et al. Etiopathogenesis of NAFLD: Diet, Gut, and NASH. In: Bugianesi E, editor. Non-Alcoholic Fatty Liver Disease [Internet]. Cham: Springer International Publishing; 2020 [cited 2021 Dec 18]. p. 73–95. Available from: http://link.springer.com/10.1007/978-3-319-95828-6_5

18. Tiniakos DG, Sakellariou S. Histopathology of Nonalcoholic Fatty Liver Disease. In: Bugianesi E, editor. Non-Alcoholic Fatty Liver Disease [Internet]. Cham: Springer International Publishing; 2020 [cited 2021 Dec 18]. p. 25–47. Available from: http://link.springer.com/10.1007/978-3-319-95828-6_3

19. Petta S, Giannetti A. Non-invasive Diagnostic Approach to NASH: Biological Markers. In: Bugianesi E, editor. Non-Alcoholic Fatty Liver Disease [Internet]. Cham: Springer International Publishing; 2020 [cited 2021 Dec 18]. p. 235–56. Available from: http://link.springer.com/10.1007/978-3-319-95828-6_13

20. Tincopa MA, Harrison SA. Noninvasive Diagnostic Approach to NASH: Radiological Diagnostics. In: Bugianesi E, editor. Non-Alcoholic Fatty Liver Disease [Internet]. Cham: Springer International

Publishing; 2020 [cited 2021 Dec 18]. p. 257–69. Available from: http://link.springer.com/10.1007/978-3-319-95828-6_14

21. Sánchez-Torrijos Y, Álvarez-Amor L, Aller R, García-Luna PP, Martín F, Romero-Gómez M. Dietary Approach to NAFLD. In: Bugianesi E, editor. Non-Alcoholic Fatty Liver Disease [Internet]. Cham: Springer International Publishing; 2020 [cited 2021 Dec 18]. p. 271–87. Available from: http://link.springer.com/10.1007/978-3-319-95828-6_15

22. Hallsworth K, Trenell M. Physical Activity in NAFLD: What and How Much? In: Bugianesi E, editor. Non-Alcoholic Fatty Liver Disease [Internet]. Cham: Springer International Publishing; 2020 [cited 2021 Dec 18]. p. 289–307. Available from: http://link.springer.com/10.1007/978-3-319-95828-6_16

23. Stern C, Ratziu V. Pharmacological Options for NASH. In: Bugianesi E, editor. Non-Alcoholic Fatty Liver Disease [Internet]. Cham: Springer International Publishing; 2020 [cited 2021 Dec 18]. p. 309–27. Available from: http://link.springer.com/10.1007/978-3-319-95828-6_17

24. Castagneto-Gissey L, Casella-Mariolo JR, Mingrone G. Bariatric Surgery and NASH: A Feasible Option. In: Bugianesi E, editor. Non-Alcoholic Fatty Liver Disease [Internet]. Cham: Springer International Publishing; 2020 [cited 2021 Dec 18]. p. 329–42. Available from: http://link.springer.com/10.1007/978-3-319-95828-6_18

25. Anbalagan VP, Venkataraman V, Vamsi M, Deepa M, Mohan V. A simple Indian diabetes risk score could help identify nondiabetic individuals at high risk of non-alcoholic fatty liver disease (CURES-117). J Diabetes Sci Technol. 2012 Nov 1;6(6):1429–35.

26. Singh SP, Nayak S, Swain M, Rout N, Mallik RN, Agrawal O, et al. Prevalence of nonalcoholic fatty liver disease in coastal eastern India: a

preliminary ultrasonographic survey. Trop Gastroenterol Off J Dig Dis Found. 2004 Jun;25(2):76–9.

27. Amarapurkar D, Kamani P, Patel N, Gupte P, Kumar P, Agal S, et al. Prevalence of non-alcoholic fatty liver disease: population based study. Ann Hepatol. 2007 Sep;6(3):161–3.

28. Bahrami H, Daryani NE, Mirmomen S, Kamangar F, Haghpanah B, Djalili M. Clinical and histological features of nonalcoholic steatohepatitis in Iranian patients. BMC Gastroenterol. 2003 Oct 16;3:27.

29. Mori KS, Patel AN, Amin AA, Phatak AG. Effectiveness of Indian Diabetes Risk Score as a Screening Tool for Non-alcoholic Fatty Liver Disease: A Study from Anand, Gujarat, India. J Clin Diagn Res [Internet]. 2019 [cited 2021 Dec 17]; Available from: https://jcdr.net/article_fulltext.asp?issn=0973-709x&year=2019&volume=13&issue=8&page=OC07&issn=0973-709x&id=13050

ANNEXURE - I : PROFORMA

NAME **:**

AGE **:**

SEX **:**

ADDRESS **:**

CONTACT NUMBER :

COMPLAINTS : (if any)

PAST H/O **:**

DIABETES: 1. Yes 2. No If yes specify_____

HYPERTENSION: 1. Yes 2. No If yes specify_____

THYROID DISORDER: 1. Yes 2. No If yes specify_____

LIVER DISEASE: 1. Yes 2. No If yes specify_____

CARDIAC ILLNESS: 1. Yes 2. No If yes specify_____

STROKE: 1. Yes 2. No If yes specify_____

AUTOIMMUNE DISORDER: 1. Yes 2. No If yes specify_____

PCOS: 1YES 2.NO

OTHERS _____

H/O CHRONIC DRUG INTAKE:

PERSONAL H/O:

SMOKING: 1. Yes 2. No

ALCOHOL INTAKE: 1. Yes 2. No

PHYSICAL ACTIVITY:

SEDENTARY	MILD	MODERATE	SEVERE

FAMILY HISTORY OF DIABETES:

NO DIABETES IN PARENTS	
ONE PARENT DIABETIC	
BOTH PARENTS DIABETIC	

GENERAL EXAMINATION:

ANTHROPOMETRY:

HEIGHT:

WEIGHT:

BMI:

WAIST CIRCUMFERENCE:

HIP CIRCUMFERENCE:

WAIST-HIP RATIO:

VITALS:

BP: PR: TEMP:

RR:

SYSTEMIC EXAMINATION:

CVS: RS: P/A:

CNS:

INVESTIGATIONS

FBS	
HBA1C	

LIPID PROFILE:

TOTAL CHOLESTEROL	
TRIGLYCERIDE	
LDL	
HDL	

LIVER FUNCTION TEST:

TOTAL BILIRUBIN	
DIRECT BILIRUBIN	
SGOT	
SGPT	
ALP	
GGT	
TOTAL PTOTEIN	
ALBUMIN	

USG ABDOMEN AND PELVIS:

NORMAL	
GRADE 1	
GRADE 2	
GRADE 3	

ANNEXURE - II

INSTITUTIONAL ETHICS COMMITTEE CERTIFICATE

GOVERNMENT STANLEY MEDICAL COLLEGE & HOSPITAL,CHENNAI -01
INSTITUTIONAL ETHICS COMMITTEE

TITLE OF THE WORK	:	"EFFECTIVENESS OF INDIAN DIABETES RISK SCORE AS A SCREENING TOOL FOR NON ALCOHOLIC FATTY LIVER DISEASE AMONG NON DIABETICS - AT TERTIARY CARE HOSPITAL IN SOUTH INDIA"
PRINCIPAL INVESTIGATOR	:	DR. HARSHINI. S,
DESIGNATION	:	PG IN GENERAL MEDICINE,
DEPARTMENT	:	DEPARTMENT OF GENERAL MEDICINE

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC meeting held on 17.02.2021 at the Council Hall, Stanley Medical College, Chennai-1 at 10am.

The members of the Committee, the secretary and the Chairman are pleased to approve the proposed work mentioned above, submitted by the principal investigator.

The Principal investigator and their team are directed to adhere to the guidelines given below:

1. You should inform the IEC in case of changes in study procedure, site investigator investigation or guide or any other changes.
2. You should not deviate from the area of the work for which you applied for ethical clearance.
3. You should inform the IEC immediately, in case of any adverse events or serious adverse reaction.
4. You should abide to the rules and regulation of the institution(s).
5. You should complete the work within the specified period and if any extension of time is required, you should apply for permission again and do the work.
6. You should submit the summary of the work to the ethical committee on completion of the work.

MEMBER SECRETARY,
IEC, SMC, CHENNAI.

ANNEXURE – III : PLAGIARISM CERTIFICATE

Curiginal

Document Information

Analyzed document	IDRS AND NAFLD .docx (D123227874)
Submitted	2021-12-20T14:37:00.0000000
Submitted by	HARSHINI S
Submitter email	harshini3020@gmail.com
Similarity	1%
Analysis address	harshini3020.mgrmu@analysis.urkund.com

ANNEXURE - IV

INFORMED CONSENT

EFFECTIVENESS OF INDIAN DIABETES RISK SCORE AS A SCREENING TOOL FOR NON ALCOHOLIC FATTY LIVER DISEASE AMONG NON DIABETICS - AT TERTIARY CARE HOSPITAL IN SOUTH INDIA.

Place of study: Govt. Stanley Hospital, Chennai- 600001

I ……. have been informed about the details of the study in my own language.

I have completely understood the details of the study.

I am aware of the possible risks and benefits, while taking part in the study.

I agree to collect samples of blood/saliva/urine/tissue if study needs.

I understand that I can withdraw from the study at any point of time and even then, I can receive the medical treatment as usual.

I understand that I will not get any money for taking part in the study.

I also understand that this study is done free of cost and I need not pay any money to the investigator.

I will not object if the results of this study are getting published in any medical journal, provided my personal identity is not revealed.

I know what I am supposed to do by taking part in this study and I assure that I would extend my full cooperation for this study.

Volunteer:	Witness:
Name and address:	Name and address
Signature/thumb impression:	Signature/thumb impression
Date:	Date:

Investigator Signature and date:

INFORMED CONSENT

EFFECTIVENESS OF INDIAN DIABETES RISK SCORE AS A SCREENING TOOL FOR NON ALCOHOLIC FATTY LIVER DISEASE AMONG NON DIABETICS - AT TERTIARY CARE HOSPITAL IN SOUTH INDIA.

Place of study: Govt. Stanley Hospital, Chennai- 600001

நான் ……,இந்த ஆராய்ச்சியில் விவரங்களை முற்றிலும் புரிந்து கொண்டேன்.

ஆய்வில் பங்கு எடுத்து போது, சாத்தியமான அபாயங்கள் மற்றும் பயன்களை பற்றிநான் அறிந்துள்ளேன்.

நான் எந்தவொருவேளையிலும் ஆய்வில் இருந்து திரும்பமுடியும், அதன்பின்னர், நான் வழக்கம் போல் மருத்துவசிகிச்சை பெறமுடியும் என்று புரிந்துகொள்கிறேன்

நான் ஆய்வில் பங்குஎடுத்து பணம் எதையும் பெறமுடியாது என்று அறிந்துள்ளேன்.

இந்த ஆய்வின் முடிவுகள் எந்த மெடிக்கல் ஜர்னலில் வெளியிடப்பட இருந்தால் நான் எதிர்க்கவில்லை,

என் தனிப்பட்ட அடையாளத்தை வெளிப்படுத்தப்பட்டு இருக்ககூடாது.

நான் இந்த ஆய்வில் பங்கெடுப்பதன் மூலம் நான் என்ன செய்யபோகிறேன் என்று தெரியும்

நான் இந்த ஆய்வில் என் முழு ஒத்துழைப்பையும் கொடுப்பேன் என்று உறுதியளிக்கிறேன்.

தன்னார்வளர்	சாட்சி
பெயர் மற்றும் முகவரி	பெயர் மற்றும் முகவரி
கையொப்பம் / விரல்ரேகை:	கையொப்பம் /விரல்ரேகை:
தேதி	தேதி

ஆராய்ச்சியாளராக கையொப்பம் மற்றும் தேதி

ANNEXURE – V : MASTER CHART

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O
1	S.NO	NAME	AGE	GENDER	COMORBIDS	SMOKIN	ALCOHOL	PHYSICAL ACT	FAMILY H/O [	HEIGHT (cm	WEIGHT(KG)	BMI	BMI (CATEGOR	WAIST CIRCU	HIP CIRCU
2	1	ANAND	35	1	NO	0	0	1	0	168	66	23.4	1	94	104
3	2	MOHAN	30	1	NO	1	0	2	0	172	68	23	1	88	102
4	3	SELVI	54	2	HYPOTHYROID	0	0	1	1	160	69	27	2	92	106
5	4	ISMAIL	56	1	SHT	0	0	2	1	172	82	27.7	2	92	98
6	5	PUSHPA	40	2	NO	0	0	2	2	160	72	28.1	2	84	93
7	6	MARY	32	2	NO	0	0	1	1	164	60	22.3	1	82	102
8	7	GAYATHRI	38	2	NO	0	0	2	1	161	62	23.9	1	84	96
9	8	JEEVA	23	1	NO	0	0	2	0	170	62	21.5	1	86	102
10	9	MUNUSAMY	48	1	NO	0	0	2	2	168	74	26.2	2	96	105
11	10	SATHIYA	41	2	HYPOTHYROID	0	0	1	1	159	62	24.5	1	87	96
12	11	HARSHA VARDHAN	27	1	NO	0	0	1	1	174	68	22.5	1	88	106
13	12	ELUMALAI	34	1	NO	0	0	2	0	175	70	22.9	1	86	105
14	13	MANI	38	1	NO	0	0	1	0	169	64	22.4	1	93	108
15	14	GEETHA	36	2	NO	0	0	1	0	167	60	21.5	1	78	94
16	15	SHIVANI	29	2	PCOS	0	0	1	2	162	68	25.9	2	89	101
17	16	RAMESH	35	1	NO	0	0	2	1	170	65	22.5	1	88	98
18	17	ANITHA	33	2	NO	0	0	2	1	156	55	22.6	1	77	90
19	18	VIJAYALAKSHMI	30	2	NO	0	0	2	1	163	68	25.6	2	85	103
20	19	SATHISH	42	1	SHT	1	0	2	1	174	75	24.8	1	86	103
21	20	BABU	44	1	NO	1	0	2	2	170	84	29.1	2	94	104
22	21	KAVITHA	39	2	NO	0	0	1	2	157	66	26.8	1	82	95
23	22	MOHANA PRIYA	34	2	NO	0	0	2	1	152	58	25.1	2	85	102
24	23	LAKSHMI	45	2	NO	0	0	1	1	160	59	23	1	86	102
25	24	GOWTHAM	27	1	NO	0	0	3	2	168	60	21.3	1	92	109
26	25	VIJAYA	41	2	NO	0	0	1	0	162	60	22.9	1	83	98
27	26	KARTHICK	34	1	NO	0	0	2	1	175	78	25.5	2	94	103
28	27	JOHNSON	50	1	SHT	1	0	1	1	167	70	25.1	2	96	102
29	28	ARUN	37	1	NO	1	0	2	0	168	67	23.7	1	87	105
30	29	KRITHIKA	32	2	NO	0	0	1	2	162	65	24.8	1	93	106
31	30	SALMA	30	2	NO	0	0	2	1	156	58	23.8	1	86	95
32	31	RAMESH	40	1	NO	0	0	2	0	172	68	23	1	87	99
33	32	MAHALINGAM	43	1	NO	1	0	1	1	170	65	22.5	1	88	96
34	33	NEELA	36	2	NO	0	0	2	1	165	62	22.8	1	79	94
35	34	RANI	26	2	NO	0	0	2	1	163	59	22.2	1	82	96
36	35	SANDHIYA	23	2	NO	0	0	2	1	160	53	20.7	1	83	101
37	36	MALARKODI	33	2	HYPOTHYROID	0	0	1	1	158	60	24	1	84	99
38	37	DILIBABU	42	1	NO	0	0	1	2	166	68	24.7	1	87	102
39	38	SRINIVASAN	45	1	NO	1	0	1	1	168	72	25.5	2	96	107
40	39	SRIKANTH	37	1	NO	0	0	1	1	160	59	23	1	87	95
41	40	GAYATHRI	47	2	NO	0	0	1	2	162	65	24.8	1	87	104
42	41	KAVITHA	36	2	NO	0	0	2	0	164	69	25.7	2	82	96
43	42	KARTHICK	26	1	NO	0	0	2	1	170	64	22.1	1	85	98
44	43	DURAI	58	1	NO	1	0	1	0	172	74	25	2	94	103
45	44	MARY	53	2	SHT,HYPOTHYR(	0	0	1	0	162	67	25.5	2	87	94
46	45	GOPAL	47	1	NO	0	0	1	1	167	72	25.8	2	91	102
47	46	UDHAY KUMAR	52	1	NO	0	0	2	1	169	68	23.8	1	95	106
48	47	LATHA	37	2	NO	0	0	2	0	165	62	22.8	1	82	97
49	48	LELAVATHI	34	2	NO	0	0	1	1	157	56	22.4	1	83	98
50	49	SHANTHI	38	2	NO	0	0	1	1	160	59	23	1	78	92
51	50	ARUMUGAM	39	1	NO	0	0	2	0	174	74	24.4	1	87	98
52	51	CHANDRASEKAR	46	1	SHT	1	0	1	1	172	79	26.7	2	91	104
53	52	INDRA KUMAR	29	1	NO	1	0	2	1	170	65	22.5	1	84	95
54	53	SRIRAM	31	1	NO	0	0	1	1	169	64	22.4	1	85	98
55	54	ANAJLI	26	2	HYPOTHYROID	0	0	2	1	160	58	22.7	1	86	101
56	55	UMA	37	2	NO	0	0	2	0	161	64	24.7	1	81	96
57	56	MOHAN	43	1	NO	1	0	2	2	165	65	23.9	1	94	104
58	57	VENKATESAN	46	1	NO	1	0	1	2	163	68	25.6	2	88	97
59	58	BALAMMAL	51	2	SHT,CKD	0	0	1	0	158	60	24	1	78	88
60	59	SANJAY	28	1	NO	0	0	2	2	172	68	23	1	94	108
61	60	PRIYA	21	2	HYPOTHYROID	0	0	1	0	168	58	20.5	1	86	101
62	61	DANIEL RAJ	35	1	NO	0	0	1	0	168	66	23.4	1	94	104
63	62	SHANTHA KUMAR	30	1	NO	1	0	2	0	172	68	23	1	88	102
64	63	RENUKA	54	2	HYPOTHYROID	0	0	1	1	160	69	27	2	92	106
65	64	IMRAN	56	1	SHT	0	0	2	1	172	82	27.7	2	92	98
66	65	SELVI	40	2	NO	0	0	2	2	160	72	28.1	2	84	93

fx 85

	P	Q	R	S	T	U	V	W	X	Y	Z	AA	AB	AC	AD	AE
1	WAIST-HIP RATIC	WHR (CATEC	IDRS SCORE	SBP	DBP	FBS	FBS (CATE	HBA1C	HBA1C (CA	TOTAL CHOLEST	CHOLESTER(	TRIGLYCERIDE	TGL (CATEG	HDL	HDL (CATE	LDL
2	0.9	2	2	120	70	94	1	5.6	1	196	1	180	2	35	1	125
3	0.86	1	1	110	74	87	1	5.6	1	152	1	98	1	38	1	94.4
4	0.86	B	3	132	80	108	2	6	2	210	2	256	2	38	1	120.8
5	0.93	2	3	128	86	102	2	5.8	2	220	2	138	1	35	1	157.4
6	0.9	B	3	118	68	98	1	5.9	2	226	2	230	2	34	1	146
7	0.8	A	2	116	72	94	1	5.5	1	202	2	138	1	44	2	130.4
8	0.87	B	2	120	72	89	1	5.6	1	201	2	252	2	52	2	98.6
9	0.84	1	1	108	74	84	1	5.6	1	157	1	86	1	43	2	96.8
10	0.91	2	3	122	80	102	2	5.8	2	202	2	176	2	38	1	128.8
11	0.9	B	3	124	92	92	1	5.9	2	160	1	134	1	38	1	95.2
12	0.83	1	2	118	80	88	1	5.8	2	159	1	117	1	47	2	88.6
13	0.81	1	1	126	84	79	1	5.4	1	156	1	97	1	48	2	88.6
14	0.86	1	2	120	78	82	1	5.6	1	174	1	145	1	37	1	108
15	0.82	A	2	118	72	76	1	5.5	1	201	2	135	1	47	2	127
16	0.88	B	2	116	70	92	1	5.5	1	163	1	168	2	43	2	86.4
17	0.89	1	2	120	76	94	1	5.6	1	158	1	110	1	39	1	97
18	0.85	B	1	114	62	86	1	5.2	1	148	1	86	1	42	2	88.8
19	0.82	A	2	132	86	98	1	5.8	2	156	1	126	1	34	1	96.8
20	0.83	1	2	120	60	82	1	5.5	1	189	1	132	1	35	1	132
21	0.9	2	3	118	70	89	1	5.9	2	234	2	146	1	38	1	166.8
22	0.86	B	3	120	76	103	2	6	2	181	1	116	1	42	2	115.8
23	0.83	A	2	114	68	76	1	5.7	2	202	2	235	2	36	1	119
24	0.84	A	3	126	82	99	1	5.8	2	170	1	162	2	39	1	98.6
25	0.84	1	2	120	70	89	1	5.4	1	164	1	96	1	45	2	99.8
26	0.84	A	2	110	72	84	1	5.6	1	162	1	136	1	46	2	136
27	0.91	2	2	118	80	86	1	5.4	1	179	1	220	2	37	1	98
28	0.94	2	3	138	90	106	2	6.1	2	175	1	192	2	38	1	98.6
29	0.82	1	2	120	80	86	1	5.3	1	153	1	76	1	46	2	91.8
30	0.87	B	3	124	82	98	1	5.5	1	183	1	197	2	47	2	96.6
31	0.9	B	2	120	80	84	1	5.6	1	213	2	121	1	44	2	144.8
32	0.87	1	2	118	72	86	1	5.4	1	157	1	106	1	46	2	89.8
33	0.91	2	3	120	76	94	1	5.6	1	201	2	178	2	38	1	127.4
34	0.84	A	2	110	72	79	1	5.5	1	204	2	140	1	42	2	134
35	0.85	B	2	114	70	87	1	5.6	1	183	1	141	1	42	2	112.8
36	0.82	A	2	106	72	83	1	5.6	1	174	1	106	1	51	2	101.8
37	0.84	A	2	120	90	86	1	5.2	1	169	1	121	1	46	2	98.8
38	0.85	1	3	116	74	92	1	5.8	2	184	1	294	2	42	2	83.2
39	0.89	1	3	128	92	104	2	6.1	2	168	1	186	2	42	2	88.8
40	0.91	2	3	126	82	99	1	5.8	2	204	2	172	2	39	1	130.6
41	0.83	A	3	126	76	94	1	6	2	156	1	86	1	39	1	99.8
42	0.85	B	2	110	74	87	1	5.6	1	223	2	106	1	41	2	160.8
43	0.86	1	1	114	72	79	1	5.4	1	156	1	95	1	36	1	101
44	0.91	2	3	130	80	88	1	5.8	2	260	2	136	1	40	2	192.8
45	0.92	B	3	130	70	94	1	6.2	2	169	1	268	2	42	2	73.4
46	0.89	1	3	128	78	92	1	5.8	2	195	1	112	1	38	1	134.6
47	0.89	1	3	124	84	87	1	5.7	2	183	1	169	2	39	1	110.2
48	0.84	A	2	116	76	84	1	6.1	2	152	1	98	1	41	2	91.4
49	0.84	A	2	122	72	81	1	5.5	1	169	1	86	1	52	2	99.8
50	0.84	A	2	114	64	79	1	5.5	1	164	1	93	1	42	2	103.4
51	0.88	1	2	116	70	89	1	5.3	1	182	1	120	1	46	2	112
52	0.87	1	3	118	68	87	1	5.6	1	170	1	183	2	38	1	95.4
53	0.88	1	1	120	70	84	1	5.5	1	176	1	102	1	43	2	112.6
54	0.86	1	2	128	80	78	1	5.7	2	187	1	97	1	47	2	117.6
55	0.85	B	2	118	64	80	1	5.6	1	162	1	83	1	38	1	107.4
56	0.84	A	2	120	80	86	1	5.3	1	158	1	118	1	39	1	95.4
57	0.9	2	3	110	70	106	2	5.8	2	162	1	103	1	49	2	92.4
58	0.9	2	3	128	78	98	1	5.6	1	168	1	96	1	40	2	108.8
59	0.88	B	2	142	80	93	1	5.4	1	157	1	85	1	42	2	98
60	0.87	1	2	122	78	90	1	5.7	2	171	1	186	2	45	2	88.8
61	0.85	B	2	114	68	87	1	5.6	1	159	1	92	1	39	1	101.6
62	0.9	2	2	120	70	94	1	5.6	1	196	1	180	2	35	1	125
63	0.86	1	1	110	74	87	1	5.6	1	152	1	98	1	38	1	94.4
64	0.86	B	3	132	80	108	2	6	2	210	2	256	2	38	1	120.8
65	0.93	2	3	128	86	102	2	5.8	2	220	2	138	1	35	1	157.4
66	0.9	B	3	118	68	98	1	5.9	2	226	2	230	2	34	1	146

	AF	AG	AH	AI	AJ	AK	AL	AM	AN	AO	AP	AQ	AR	AS	AT	AU
1	LDL (CATE	TOTAL BILIRUI	DIRECT BILIR	AST	AST (CATE	ALT	ALT (CATE	ALT/AST	AST/ALT	ALP	ALP (CATE	GGT	GGT(CATE	TOTAL PROTEIN	ALBUMIN	USG
2	2	0.8	0.4	26	1	28	1	1.07	0.92	176	2	30	2	7.4	4.2	1
3	1	0.9	0.3	24	1	27	1	1.12	0.88	138	1	26	1	7.8	4.2	0
4	2	0.8	0.3	32	2	54	2	1.68	0.59	216	2	38	2	7.5	4	2
5	2	1	0.5	30	2	48	2	1.6	0.62	184	2	33	2	7.3	4.1	2
6	2	0.7	0.3	28	1	32	1	1.14	0.87	208	2	35	2	7.4	4	2
7	2	0.8	0.3	25	1	29	1	1.16	0.86	165	2	27	1	7.8	4.1	0
8	1	0.7	0.3	28	1	34	1	0.82	0.82	185	2	33	2	8.2	4.2	0
9	1	0.8	0.3	24	1	27	1	1.12	0.88	148	1	26	1	7.8	3.8	0
10	2	1.1	0.4	27	1	34	1	1.25	0.79	146	1	28	1	8	4.3	1
11	1	0.7	0.3	28	1	27	1	0.96	1.03	146	1	27	1	7.7	3.9	0
12	1	0.8	0.3	22	1	28	1	1.27	0.78	173	2	27	1	8.1	4.3	0
13	1	0.9	0.3	25	1	29	1	1.16	0.86	147	1	29	1	8.2	4.4	0
14	2	0.8	0.2	27	1	28	1	1.03	0.96	194	2	42	2	7.8	3.7	0
15	2	1.1	0.5	22	1	36	1	1.63	0.61	135	1	26	1	7.4	3.6	0
16	1	0.7	0.2	26	1	29	1	1.11	0.89	136	1	27	1	7.8	4	1
17	1	0.7	0.3	30	2	29	1	0.96	1.03	129	1	28	1	8.2	4.2	0
18	1	0.9	0.3	25	1	26	1	1.04	0.96	110	1	27	1	7.9	3.8	0
19	1	0.7	0.2	30	2	36	1	1.2	0.83	187	2	29	1	7.6	3.7	1
20	2	0.8	0.3	20	1	28	1	1.4	0.71	143	1	28	1	7.8	3.6	0
21	2	0.9	0.2	34	2	42	2	1.23	0.8	230	2	38	2	7.4	3.7	3
22	2	0.8	0.4	34	2	36	1	1.05	0.94	184	2	34	2	8.2	4.1	0
23	2	1.2	0.5	26	1	33	1	1.26	0.78	144	1	27	1	7.4	3.6	1
24	1	0.9	0.4	28	1	36	1	1.28	0.77	174	2	37	2	7.4	3.8	0
25	1	0.7	0.2	26	1	27	1	1.03	0.96	148	1	28	1	7.8	4	0
26	2	0.9	0.3	34	2	39	1	1.14	0.87	146	1	32	2	7.9	3.8	0
27	1	0.8	0.3	19	1	27	1	1.42	0.7	147	1	25	1	7.8	3.7	0
28	1	0.8	0.2	38	2	43	2	1.13	0.88	197	2	42	2	7.4	3.7	2
29	1	0.7	0.2	27	1	28	1	1.03	0.96	172	2	24	1	8.2	4.3	0
30	1	0.9	0.4	26	1	29	1	1.11	0.89	165	2	27	1	7.8	3.6	0
31	2	0.8	0.3	28	2	27	1	0.96	1.03	182	2	36	2	7.7	3.7	1
32	1	0.9	0.3	38	2	37	1	0.97	1.02	164	2	24	1	7.4	4.2	0
33	2	0.7	0.3	40	2	45	2	1.12	0.88	147	1	30	2	7.8	4.2	2
34	2	0.8	0.3	27	1	34	1	1.25	0.79	174	2	28	1	7.5	4	0
35	2	0.8	0.2	26	1	39	1	1.5	0.66	139	1	42	2	7.3	4.1	0
36	2	0.6	0.2	31	2	38	1	1.22	0.81	141	1	32	2	7.4	4	0
37	1	0.7	0.3	26	1	30	1	1.15	0.86	183	2	29	1	7.8	4.1	0
38	1	0.8	0.2	38	2	47	2	1.2	0.8	143	1	38	2	8.2	4.2	0
39	1	0.8	0.2	37	2	46	2	0.8	0.8	201	2	42	2	7.8	3.9	1
40	2	0.9	0.4	28	1	36	1	1.28	0.77	174	2	35	2	8.2	4.2	2
41	1	0.7	0.2	35	2	32	1	0.91	1.09	169	2	32	2	7.8	3.8	0
42	2	0.8	0.2	54	2	83	2	1.53	0.65	183	2	42	2	8	4.3	3
43	2	0.7	0.3	36	2	38	1	1.05	0.94	156	2	35	2	7.7	4	1
44	2	0.8	0.4	38	2	41	2	1.07	0.92	162	2	31	2	8.1	4.2	2
45	1	0.8	0.3	48	2	56	2	1.16	0.85	148	1	27	1	8.2	4	2
46	2	0.6	0.2	32	2	28	1	0.87	1.14	189	2	36	2	7.9	3.6	0
47	2	0.9	0.2	37	2	40	2	1.08	0.92	152	2	31	2	7.4	3.7	1
48	1	1	0.3	32	2	34	1	1.06	0.94	167	2	26	1	7.8	4	0
49	1	0.9	0.3	27	1	28	1	1.03	0.96	148	1	36	1	8.2	4.2	0
50	2	0.8	0.3	40	2	52	2	1.3	0.76	138	1	25	1	7.9	3.8	0
51	2	1.1	0.4	29	1	34	1	1.17	0.85	181	2	37	2	7.7	3.7	0
52	1	0.7	0.3	42	2	53	2	1.26	0.79	206	2	30	2	7.9	3.6	0
53	2	0.8	0.3	34	2	36	1	1.05	0.94	164	2	37	2	7.4	3.7	1
54	2	0.8	0.3	32	2	31	1	0.96	1.03	162	2	30	2	8.1	4	0
55	2	0.7	0.2	27	1	32	1	1.18	0.84	168	2	28	1	7.6	3.7	1
56	1	0.9	0.3	33	2	39	1	1.18	0.84	162	2	27	1	7.8	3.7	0
57	1	0.6	0.2	29	1	37	1	1.27	0.78	145	1	32	2	7.5	3.8	0
58	2	0.7	0.3	32	2	38	1	1.18	0.84	143	1	24	1	8.2	3.6	0
59	1	0.7	0.3	28	1	29	1	1.03	0.96	172	2	29	1	7.8	4	0
60	1	0.8	0.2	36	2	44	2	1.22	0.81	172	2	31	2	8.2	4.2	1
61	2	0.7	0.2	37	2	46	2	1.24	0.8	148	1	29	1	8	3.9	1
62	2	0.8	0.4	26	1	28	1	1.07	0.92	176	2	30	2	7.4	4.2	1
63	1	0.9	0.3	24	1	27	1	1.12	0.88	138	1	26	1	7.8	4.2	0
64	2	0.8	0.3	32	2	54	2	1.68	0.59	216	2	38	2	7.5	4	2
65	2	1	0.5	30	2	48	2	1.6	0.62	184	2	33	2	7.3	4.1	2
66	2	0.7	0.3	28	1	32	1	1.14	0.87	208	2	35	2	7.4	4	2

	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O
66	65	SELVI	40	2	NO	0	0	2	2	160	72	28.1	2	84	93
67	66	VISHNU	32	2	NO	0	0	1	1	164	60	22.3	1	82	102
68	67	VARALAKSHMI	38	2	NO	0	0	2	1	161	62	23.9	1	84	96
69	68	SUGUMAR	23	1	NO	0	0	2	0	170	62	21.5	1	86	102
70	69	VIGNESH	48	1	NO	0	0	2	2	168	74	26.2	2	96	105
71	70	SABANA	41	2	HYPOTHYROID	0	0	1	1	159	62	24.5	1	87	96
72	71	RAHUL	27	1	NO	0	0	1	1	174	68	22.5	1	88	106
73	72	MADHESH	34	1	NO	0	0	2	0	175	70	22.9	1	86	105
74	73	PRABHAKAR	38	1	NO	0	0	1	0	169	64	22.4	1	93	108
75	74	FATHIMA	36	2	NO	0	0	1	0	167	60	21.5	1	78	94
76	75	GEETHA	29	2	PCOS	0	0	1	2	162	68	25.9	2	89	101
77	76	GOWTHAM	35	1	NO	0	0	2	1	170	65	22.5	1	88	98
78	77	LATHA	33	2	NO	0	0	2	1	156	55	22.6	1	77	90
79	78	MRIDULA	30	2	NO	0	0	2	1	163	68	25.6	2	85	103
80	79	SARAVANAN	42	1	SHT	1	0	2	1	174	75	24.8	1	86	103
81	80	DHARAN	44	1	NO	1	0	2	2	170	84	29.1	2	94	104
82	81	SANGEETHA	39	2	NO	0	0	1	2	157	66	26.8	1	82	95
83	82	ASHWANA	34	2	NO	0	0	2	1	152	58	25.1	2	85	102
84	83	RANI	45	2	NO	0	0	1	1	160	59	23	1	86	102
85	84	NARAYAN	27	1	NO	0	0	3	2	168	60	21.3	1	92	109
86	85	SARGUNAM	41	2	NO	0	0	1	0	162	60	22.9	1	83	98
87	86	KRISHNAMOORTH	34	1	NO	0	0	2	1	175	78	25.5	2	94	103
88	87	AJAY	50	1	SHT	1	0	1	1	167	70	25.1	2	96	102
89	88	MOHAN	37	1	NO	1	0	2	0	168	67	23.7	1	87	105
90	89	JAYASHRI	32	2	NO	0	0	1	2	162	65	24.8	1	93	106
91	90	VINITHA	30	2	NO	0	0	2	1	156	58	23.8	1	86	95
92	91	KUMAR	40	1	NO	0	0	2	0	172	68	23	1	87	99
93	92	SEKAR	45	1	NO	1	0	1	1	170	65	22.5	1	88	96
94	93	GOWRI	36	2	NO	0	0	2	1	165	62	22.8	1	79	94
95	94	BHARGAVI	26	2	NO	0	0	2	1	163	59	22.2	1	82	96
96	95	CHITHRA	23	2	NO	0	0	2	1	160	53	20.7	1	83	101
97	96	MALAR	33	2	HYPOTHYROID	0	0	1	1	158	60	24	1	84	99
98	97	KANNAN	42	1	NO	0	0	1	2	166	68	24.7	1	87	102
99	98	SATHISH KUMAR	45	1	NO	1	0	1	1	168	72	25.5	2	96	107
100	99	SIVARAMAN	37	1	NO	0	0	1	1	160	59	23	1	87	95
101	100	MEENATCHI	47	2	NO	0	0	1	2	162	65	24.8	1	87	104
102	101	SUPRIYA	36	2	NO	0	0	2	0	164	69	25.7	2	82	96
103	102	LOGESH	26	1	NO	0	0	2	1	170	64	22.1	1	85	98
104	103	ELANGOVAN	58	1	NO	1	0	1	0	172	74	25	2	94	103
105	104	ARPUDHAM	53	2	SHT,HYPOTHYRC	0	0	1	0	162	60	22.9	1	87	94
106	105	SIVA KUMAR	47	1	NO	0	0	1	1	167	72	25.8	2	91	102
107	106	TAMILARASAN	52	1	NO	0	0	2	1	169	68	23.8	1	95	106
108	107	NAGESHWARI	37	2	NO	0	0	2	0	165	62	22.8	1	82	97
109	108	NILA	34	2	NO	0	0	1	1	157	56	22.4	1	83	98
110	109	LAKSHMI	38	2	NO	0	0	1	1	160	59	23	1	78	92
111	110	MUNIVEL	39	1	NO	0	0	2	0	174	74	24.4	1	87	98
112	111	RAJESH	46	1	SHT	1	0	1	1	172	79	26.7	2	91	104
113	112	SANTHOSH	29	1	NO	1	0	2	1	170	65	22.5	1	84	95
114	113	RAM	31	1	NO	0	0	1	1	169	64	22.4	1	85	98
115	114	BAVANI	26	2	HYPOTHYROID	0	0	2	1	160	58	22.7	1	86	101
116	115	VASANTHI	37	2	NO	0	0	2	0	161	64	24.7	1	81	96
117	116	PALANISAMY	43	1	NO	1	0	2	2	165	65	23.9	1	94	104
118	117	NAASIR	46	1	NO	1	0	1	2	163	68	25.6	2	88	97
119	118	YESUDHA	51	2	SHT,CKD	0	0	1	0	158	60	24	1	78	88
120	119	PUNKAJ KUMAR	28	1	NO	0	0	2	2	172	68	23	1	94	108
121	120	NITHYA	21	2	HYPOTHYROID	0	0	1	0	168	58	20.5	1	86	101
122	121	SENTHIL	51	1	NO	0	0	2	1	172	82	27.7	2	92	105
123	122	THIRU	43	1	NO	0	0	1	2	166	68	24.7	1	87	102
124	123	SUMATHI	38	2	NO	0	0	1	2	157	66	26.8	2	82	97
125	124	REBECCA	32	2	NO	0	0	1	1	164	60	22.3	1	82	102
126	125	RADHA	38	2	NO	0	0	2	1	161	62	23.9	1	84	100
127	126	YOGALAKSHMI	28	2	HYPOTHYROID	0	0	2	1	160	66	25.8	2	86	98
128	127	ASHOK	37	1	NO	1	0	2	0	168	67	23.7	1	87	105
129	128	KRISHNA	35	1	NO	0	0	2	1	170	76	26.3	2	88	97
130	129	VALARMATHI	38	2	NO	0	0	1	1	160	59	23	1	78	93
131	130	SRIRAMAN	52	1	NO	0	0	1	1	169	72	25.2	2	85	94

	P	Q	R	S	T	U	V	W	X	Y	Z	AA	AB	AC	AD
66	0.9	B	3	118	68	98	1	5.9	2	226	2	230	2	34	1
67	0.8	A	2	116	72	94	1	5.5	1	202	2	138	1	44	2
68	0.87	B	2	120	72	89	1	5.6	1	201	2	252	2	52	2
69	0.84	1	1	108	74	84	1	5.6	1	157	1	86	1	43	2
70	0.91	2	3	122	80	102	2	5.8	2	202	2	176	2	38	1
71	0.9	B	3	124	92	92	1	5.9	2	160	1	134	1	38	1
72	0.83	1	2	118	80	88	1	5.8	2	159	1	117	1	47	2
73	0.81	1	1	126	84	79	1	5.4	1	156	1	97	1	48	2
74	0.86	1	2	120	78	82	1	5.6	1	174	1	145	1	37	1
75	0.82	A	2	118	72	76	1	5.5	1	201	2	135	1	47	2
76	0.88	B	2	116	70	92	1	5.5	1	163	1	168	2	43	2
77	0.89	1	2	120	76	94	1	5.6	1	158	1	110	1	39	1
78	0.85	B	1	114	62	86	1	5.2	1	148	1	86	1	42	2
79	0.82	A	2	132	86	98	1	5.8	2	156	1	126	1	34	1
80	0.83	1	2	120	60	82	1	5.5	1	189	1	132	1	35	1
81	0.9	2	3	118	70	89	1	5.9	2	234	2	146	1	38	1
82	0.86	B	3	120	76	103	2	6	2	181	1	116	1	42	2
83	0.83	A	2	114	68	76	1	5.7	2	202	2	235	2	36	1
84	0.84	A	3	126	82	99	1	5.8	2	170	1	162	2	39	1
85	0.84	1	2	120	70	89	1	5.4	1	164	1	96	1	45	2
86	0.84	A	2	110	72	84	1	5.6	1	162	1	136	1	46	2
87	0.91	2	2	118	80	86	1	5.4	1	179	1	220	2	37	1
88	0.94	2	3	138	90	106	2	6.1	2	175	1	192	2	38	1
89	0.82	1	2	120	80	86	1	5.3	1	153	1	76	1	46	2
90	0.87	B	3	124	82	98	1	5.5	1	183	1	197	2	47	2
91	0.9	B	2	120	80	84	1	5.6	1	213	2	121	1	44	2
92	0.87	1	2	118	72	86	1	5.4	1	157	1	106	1	46	2
93	0.91	2	3	120	76	94	1	5.6	1	201	2	178	2	38	1
94	0.84	A	2	110	72	79	1	5.5	1	204	2	140	1	42	2
95	0.85	B	2	114	70	87	1	5.6	1	183	1	141	1	42	2
96	0.82	A	2	106	72	83	1	5.6	1	174	1	106	1	51	2
97	0.84	A	2	120	90	86	1	5.2	1	169	1	121	1	46	2
98	0.85	1	3	116	74	92	1	5.8	2	184	1	294	2	42	2
99	0.89	1	3	128	92	104	2	6.1	2	168	1	186	2	42	2
100	0.91	2	3	126	82	99	1	5.8	2	204	2	172	2	39	1
101	0.83	A	3	126	76	94	1	6	2	156	1	86	1	39	1
102	0.85	B	2	110	74	87	1	5.6	1	223	2	106	1	41	2
103	0.86	1	1	114	72	79	1	5.4	1	156	1	95	1	36	1
104	0.91	2	3	130	80	88	1	5.8	2	260	2	136	1	40	2
105	0.92	B	3	130	70	94	1	6.2	2	169	1	268	2	42	2
106	0.89	1	3	128	78	92	1	5.8	2	195	1	112	1	38	1
107	0.89	1	3	124	84	87	1	5.7	2	183	1	169	2	39	1
108	0.84	A	2	116	76	84	1	6.1	2	152	1	98	1	41	2
109	0.84	A	2	122	72	81	1	5.5	1	169	1	86	1	52	2
110	0.84	A	2	114	64	79	1	5.5	1	164	1	93	1	42	2
111	0.88	1	2	116	70	89	1	5.3	1	182	1	120	1	46	2
112	0.87	1	3	118	68	87	1	5.6	1	170	1	183	2	38	1
113	0.88	1	1	120	70	84	1	5.5	1	176	1	102	1	43	2
114	0.86	1	2	128	80	78	1	5.7	2	187	1	97	1	47	2
115	0.85	B	2	118	64	80	1	5.6	1	162	1	83	1	38	1
116	0.84	A	2	120	80	86	1	5.3	1	158	1	118	1	39	1
117	0.9	2	3	110	70	106	2	5.8	2	162	1	103	1	49	2
118	0.9	2	3	128	78	98	1	5.6	1	168	1	96	1	40	2
119	0.88	B	2	142	80	93	1	5.4	1	157	1	85	1	42	2
120	0.87	1	2	122	78	90	1	5.7	2	171	1	186	2	45	2
121	0.85	B	2	114	68	87	1	5.6	1	159	1	92	1	39	1
122	0.87	1	3	128	86	102	2	5.7	2	220	2	138	1	35	1
123	0.85	1	3	116	74	92	1	5.8	2	184	1	294	2	42	2
124	0.84	A	3	120	76	103	2	6	2	181	1	116	1	42	2
125	0.8	A	2	116	72	94	1	5.7	2	169	1	138	1	44	2
126	0.84	A	2	120	72	89	1	5.3	1	172	1	135	1	48	2
127	0.87	B	2	118	64	80	1	5.6	1	170	1	160	2	35	1
128	0.82	1	2	120	80	86	1	5.3	1	153	1	76	1	46	2
129	0.9	2	2	120	76	94	1	5.6	1	158	1	159	2	39	1
130	0.83	A	2	114	64	79	1	5.5	1	164	1	93	1	42	2
131	0.9	2	3	128	80	78	1	5.4	1	187	1	172	2	47	2

	AE	AF	AG	AH	AI	AJ	AK	AL	AM	AN	AO	AP	AQ	AR	AS	AT	AU
67	130.4	2	0.8	0.3	25	1	29	1	1.16	0.86	165	2	27	1	7.8	4.1	0
68	98.6	1	0.7	0.3	28	1	34	1	0.82	0.82	185	2	33	2	8.2	4.2	0
69	96.8	1	0.8	0.3	24	1	27	1	1.12	0.88	148	1	26	1	7.8	3.8	0
70	128.8	2	1.1	0.4	27	1	34	1	1.25	0.79	146	1	28	1	8	4.3	1
71	95.2	1	0.7	0.3	28	1	27	1	0.96	1.03	146	1	27	1	7.7	3.9	0
72	88.6	1	0.8	0.3	22	1	28	1	1.27	0.78	173	2	27	1	8.1	4.3	0
73	88.6	1	0.9	0.3	25	1	29	1	1.16	0.86	147	1	29	1	8.2	4.4	0
74	108	2	0.8	0.2	27	1	28	1	1.03	0.96	194	2	42	2	7.8	3.7	0
75	127	2	1.1	0.5	22	1	36	1	1.63	0.61	135	1	26	1	7.4	3.6	0
76	86.4	1	0.7	0.2	26	1	29	1	1.11	0.89	136	1	27	1	7.8	4	1
77	97	1	0.7	0.3	30	2	29	1	0.96	1.03	129	1	28	1	8.2	4.2	0
78	88.8	1	0.9	0.3	25	1	26	1	1.04	0.96	110	1	27	1	7.9	3.8	0
79	96.8	1	0.7	0.2	30	2	36	1	1.2	0.83	187	2	29	1	7.6	3.7	1
80	132	2	0.8	0.3	20	1	28	1	1.4	0.71	143	1	28	1	7.8	3.6	0
81	166.8	2	0.9	0.2	34	2	42	2	1.23	0.8	230	2	38	2	7.4	3.7	3
82	115.8	2	0.8	0.4	34	2	36	1	1.05	0.94	184	2	34	2	8.2	4.1	0
83	119	2	1.2	0.5	26	1	33	1	1.26	0.78	144	1	27	1	7.4	3.6	1
84	98.6	1	0.9	0.4	28	1	36	1	1.28	0.77	174	2	37	2	7.4	3.8	0
85	99.8	1	0.7	0.2	26	1	27	1	1.03	0.96	148	1	28	1	7.8	4	0
86	136	2	0.9	0.3	34	2	39	1	1.14	0.87	146	1	32	2	7.9	3.8	0
87	98	1	0.8	0.3	19	1	27	1	1.42	0.7	147	1	25	1	7.8	3.7	0
88	98.6	1	0.8	0.2	38	2	43	2	1.13	0.88	197	2	42	2	7.4	3.7	2
89	91.8	1	0.7	0.2	27	1	28	1	1.03	0.96	172	2	24	1	8.2	4.3	0
90	96.6	1	0.9	0.4	26	1	29	1	1.11	0.89	165	2	27	1	7.8	3.6	0
91	144.8	2	0.8	0.3	28	2	27	1	0.96	1.03	182	2	36	2	7.7	3.7	1
92	89.8	1	0.9	0.3	38	2	37	1	0.97	1.02	164	2	24	1	7.4	4.2	0
93	127.4	2	0.7	0.3	40	2	45	2	1.12	0.88	147	1	30	2	7.8	4.2	2
94	134	2	0.8	0.3	27	1	34	1	1.25	0.79	174	2	28	1	7.5	4	0
95	112.8	2	0.8	0.2	26	1	39	1	1.5	0.66	139	1	42	2	7.3	4.1	0
96	101.8	2	0.6	0.2	31	2	38	1	1.22	0.81	141	1	32	2	7.4	4	0
97	98.8	1	0.7	0.3	26	1	30	1	1.15	0.86	183	2	29	1	7.8	4.1	0
98	83.2	1	0.8	0.2	38	2	47	2	1.2	0.8	143	1	38	2	8.2	4.2	0
99	88.8	1	0.8	0.2	37	2	46	2	0.8	0.8	201	2	42	2	7.8	3.9	1
100	130.6	2	0.9	0.4	28	1	36	1	1.28	0.77	174	2	35	2	8.2	4.2	2
101	99.8	1	0.7	0.2	35	2	32	1	0.91	1.09	169	2	32	2	7.8	3.8	0
102	160.8	2	0.8	0.2	54	2	83	2	1.53	0.65	183	2	42	2	8	4.3	3
103	101	2	0.7	0.3	36	2	38	1	1.05	0.94	156	2	35	2	7.7	4	1
104	192.8	2	0.8	0.4	38	2	41	2	1.07	0.92	162	2	31	2	8.1	4.2	2
105	73.4	1	0.8	0.3	48	2	56	2	1.16	0.85	148	1	27	1	8.2	4	2
106	134.6	2	0.6	0.2	32	2	28	1	0.87	1.14	189	2	36	2	7.9	3.6	0
107	110.2	2	0.9	0.2	37	2	40	2	1.08	0.92	152	2	31	2	7.4	3.7	1
108	91.4	1	1	0.3	32	2	34	1	1.06	0.94	167	2	26	1	7.8	4	0
109	99.8	1	0.9	0.3	27	1	28	1	1.03	0.96	148	1	36	1	8.2	4.2	0
110	103.4	2	0.8	0.3	40	2	52	2	1.3	0.76	138	1	25	1	7.9	3.8	0
111	112	2	1.1	0.4	29	1	34	1	1.17	0.85	181	2	37	2	7.7	3.7	0
112	95.4	1	0.7	0.3	42	2	53	2	1.26	0.79	206	2	30	2	7.9	3.6	0
113	112.6	2	0.8	0.3	34	2	36	1	1.05	0.94	164	2	37	2	7.4	3.7	1
114	117.6	2	0.8	0.3	32	2	31	1		1.03	162	2	30	2	8.1	4	0
115	107.4	2	0.7	0.2	27	1	32	1	1.18	0.84	168	2	28	1	7.6	3.7	1
116	95.4	1	0.9	0.3	33	2	39	1	1.18	0.84	162	2	27	1	7.8	3.7	0
117	92.4	1	0.6	0.2	29	1	37	1	1.27	0.78	145	1	32	2	7.5	3.8	0
118	108.8	2	0.7	0.3	32	2	38	1	1.18	0.84	143	1	24	1	8.2	3.6	0
119	98	1	0.7	0.3	28	1	29	1	1.03	0.96	172	2	29	1	7.8	4	0
120	88.8	1	0.8	0.2	36	2	44	2	1.22	0.81	172	2	31	2	8.2	4.2	1
121	101.6	2	0.7	0.2	37	2	46	2	1.24	0.8	148	1	29	1	8	3.9	1
122	157.4	2	1	0.5	30	2	48	2	1.6	0.62	194	2	33	2	7.3	4.1	2
123	83.2	1	0.8	0.2	22	1	47	2	2.13	0.46	173	2	38	2	8.2	4.2	0
124	115.8	2	0.8	0.4	34	2	36	1	1.05	0.94	184	2	34	2	8.2	4.1	1
125	97.4	1	0.8	0.3	25	1	29	1	1.16	0.86	145	1	27	1	7.8	4.1	0
126	97	1	0.7	0.3	28	1	34	1	1.21	0.82	185	2	33	2	8.2	4.2	0
127	103	2	0.7	0.2	27	1	32	1	1.18	0.84	147	1	28	1	7.6	3.7	1
128	91.8	1	0.7	0.2	27	1	28	1	1.03	0.96	148	1	24	1	8.2	4.3	0
129	87.2	1	0.7	0.3	30	2	29	1	0.96	1.03	169	2	28	1	8.2	4.2	1
130	103.4	2	0.8	0.3	25	1	52	2	2.08	0.48	142	1	37	2	7.9	3.8	0
131	105.6	2	0.8	0.3	32	2	31	1	0.96	1.03	145	1	30	2	8.1	4	1

ENTRY KEYS

	A	B	C	D	E	F	G	H	I	J
1	S.NO.	NUMBERS			HEIGHT	NUMBER			TOTAL BILIRUBIN	NUMBER
2									DIRECT BILIRUBIN	NUMBER
3	AGE	NUMBERS			WEIGHT	NUMBER				
4									AST	NUMBER
5	GENDER	FOR MALES- MARK AS 1			BMI	NUMBER				<30 -1
6		FOR FEMALES -MARK AS 2				<25 -1				> =30 -2
7						>=25-2				
8									ALT	NUMBER
9					WAIST CIRCUMFERENCE	NUMBER				<40 -1
10	SMOKING	YES -1								>=40 -2
11		NO-0			HIP CIRCUMFERENCE	NUMBER				
12									AST/ALT	NUMBER
13	ALCOHOL INTAKE	YES-1			WAIST -HIP CIRCUMFERENCE	NUMBER				
14						MALE <0.9 -1 ,>=0.9 -2				
15						FEMALE <0.85 -A, > =0.85 -B				
16		NO-0							ALP	NUMBER
17					IDRS SCORE	<30 -1				<150 -1
18	PHYSICAL ACTIVITY	SEDANTRY-0				30-50-2				>=150 -2
19		MILD EXERCISE -1				>= 60-3				
20		MODERATE EXERCISE-2							GGT	NUMBER
21		SEVERE EXERCISE-3			SBP	NUMBER				<30 - 1
22					DBP	NUMBER				>=30 -2
23	FAMILY H/O DIABETES	NO DIABETES IN PARENTS-0								
24		ONE PARENT DIABETIC -1			FBS	NUMBER			TOTAL PROTEIN	NUMBER
25		BOTH PARENT DIABETIC-2				<100-1			ALBUMIN	NUMBER
26						100-125-2				
27						>125- 3				
28										
29					HBA1C	NUMBER			USG	NORMAL-0
30						<5.7 -1				GRADE1 FATTY LIVER-1
31						5.7-6.4 -2				GRADE-2 FATTY LIVER-2
32						>=6.5 -3				GRADE -3 FATTY LIVER`3
33	COMORBIDS	TEXT								
34					TOTAL CHOLESTEROL	NUMBER				
35						<200 -1				
36						>= 200 -2				
37										
38					TRIGLYCERIDE	NUMBER				
39						<150 -1				
40						>=150 -2				
41										
42					HDL	NUMBER				
43						<40 -1				
44						>= 40 -2				
45										
46					LDL	NUMBER				
47						<100 -1				
48						>= 100 -2				
49										
50										