ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

Study of Serum Levels of CRP & its Relation to Asymptomatic and Incidentally Detected Fatty Liver Disease

Dr. Evuri Pramod Reddy[1]; Dr. Chanumolu Sri Varshini[2]; Dr. Pedireddy Mounika[3]
Katuri Medical College and Hospital

Abstract:-

➢ ***Background***

Fatty liver disease is a condition that can be detected incidentally during routine checkups or imaging tests. The rise in obesity and metabolic syndrome has also contributed to an increase in fatty liver diagnoses. This condition can be caused by alcohol consumption (alcoholic fatty liver disease) or by other factors (nonalcoholic fatty liver disease).

➢ ***Aims & Objectives***

This study aims to investigate the clinical characteristics and inflammatory markers in individuals with asymptomatic fatty liver disease identified through routine imaging tests like ultrasounds (USG) and CT scans of the abdomen. Specifically, the researchers will measure serum levels of high-sensitivity C-reactive protein (hs-CRP) to assess potential inflammation and its association with coronary artery disease (CAD) risk in this patient population.

➢ ***Methods***

The study appears to be investigating asymptomatic fatty liver disease detected through ultrasounds in adults (age > 14 years) who underwent health checkups at a medical college between October 2022 and September 2023.

I. INTRODUCTION

The diagnosis of Fatty liver is frequently unexpected. Often, the diagnosis of Fatty liver is incidental, occurring during radiological tests such as abdominal ultrasound or CT scan, which were originally conducted for unrelated reasons. Medical professionals are observing an increasing number of cases when fatty liver disease is diagnosed using radiological imaging, sometimes inadvertently. The rise in utilization of radiographic diagnostic techniques, health examinations, and screening initiatives, together with the prevalence of obesity and metabolic syndrome, are factors that contribute to this occurrence.

Fatty liver is a clinical and histopathological condition. Put simply, it refers to the buildup of fat (triglyceride vacuoles) in liver cells. Fatty liver disease encompasses a spectrum of conditions, ranging from steatosis to steatohepatitis. This disorder may arise due to alcohol consumption (alcohol-related fatty liver) or in the absence of significant alcohol intake (nonalcoholic fatty liver disease, often known as NAFLD).

This study aims to further investigate radiologically identified fatty liver disease in patients who are completely asymptomatic and otherwise in good condition. A diagnosis of Fatty liver holds considerable importance, especially in cases where the patient is asymptomatic and appears completely healthy. This could potentially be the initial indication of a hazardous metabolic anomaly, the toxic consequences of alcohol misuse, drug-related issues, or some uncommon liver conditions. This discovery necessitates a comprehensive clinical and laboratory assessment as well as specific treatment approaches.

This study aims to examine the characteristics of people with incidentally identified fatty liver who do not show any symptoms. An analysis is conducted on the ratio of fatty liver disease cases that are related to alcohol consumption vs those that are not. An Endeavor is undertaken to examine the diverse clinical and biochemical abnormalities, their prevalence, and their correlation with metabolic syndrome. This study enables the development of a potential algorithm that can be used to address this particular scenario, specifically within the study community of Northern Chennai. Subsequently, the levels of high sensitive C reactive protein are being assessed. Being a pro-inflammatory marker and an in dependant cardiovascular risk factor, hsCRP may provide a clue regarding the cardiovascular risk status of such instances and also the potential link between all the metabolic abnormalities.

➢ *Aim of the Study*

- The objective is to evaluate the clinical characteristics of individuals who have been identified with fatty liver using standard radiological screening modalities such as ultrasound (USG) and computed tomography (CT) of the abdomen, although not showing any symptoms.
- The objective is to examine the laboratory pattern linked to both alcoholic and non-alcoholic causes of fatty liver disease.
- So that we may calculate the prevalence of alcoholic and non-alcoholic fatty liver diseases relative to one another.
- To get insight into the prevailing and closely linked metabolic abnormality associated with fatty liver disease.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

- To investigate the frequency of Metabolic Syndrome within the group.
- The objective of this research is to assess the inflammatory marker high sensitivity C-reactive protein (CRP) levels in the blood of fatty liver disease patients. C-reactive protein is a known risk factor for coronary artery disease.
- Assess the relationship between hsCRP levels and other clinical and biochemical problems, and determine the correlation.

II. MATERIAL AND METHODS

➢ *Inclusion Criteria :*

Patients of both genders, aged 14 years and older, who are otherwise asymptomatic, have been diagnosed with fatty liver using ultrasonography.

➢ *Exclusion Criteria :*

- Anyone experiencing any kind of symptom, whether temporary or chronic, throughout any organ system is considered a symptomatic patient.
- Patients with preexisting chronic or concomitant illnesses.
- Patients who have consumed any type of medication
- Individuals have diagnosed cases of Acute or Chronic Liver disease, regardless of the underlying cause.
- Patients who have experienced hunger or prolonged fasting within the past four weeks.
- Medical background of Gastrointestinal Surgery or surgery performed within the last three months.
- Patients with a high-sensitivity C-reactive protein (hsCRP) level of 5mg/L or higher

The study population consisted of individuals who attended the Master Health Checkup at KATURI Medical College, CHINAKONDURPADU, between OCTOBER 2022 to SEPTEMBER 2023, including both patients and members of the general public.

The presence of fatty liver was confirmed using an Ultrasonogram, performed by a Radiologist. Another radiologist thoroughly examined all the images to reduce the likelihood of observer mistake. The degree of fatty liver was classified on a spectrum ranging from mild to severe. It was either present or absent for all practical purposes. The ultrasound equipment utilized for the procedure was a Lasen & Toubro model, equipped with a 3.5 MHz probe.

After getting the patient's written informed permission in their local language, we made sure they didn't have any serious medical history or current symptoms by doing a full physical examination. Medical records pertaining to previous procedures and medications were reviewed. Additionally, we inquired as to whether or not the women in our sample used hormonal therapies or oral contraceptives. There was also documentation of the duration, amount, and history statistics of alcohol use.The verification of alcohol consumption was subsequently corroborated by a relative of the patient, acting independently. In certain instances, assistance was sought from psychiatric counselors.

After gathering relevant background information, the selected group underwent a thorough physical examination. Vital signs such as pulse and blood pressure were recorded. We measured the subjects' anthropometric traits, such as their weight, height, and waist size. In order to measure a patient's waist circumference, we ask them to remove all of their clothes or, if that is not feasible, to wear very thin clothing. Sufficient measures were implemented to minimize any potential faults. The BMI is determined by dividing the weight in kilograms by the square of the height in meters. Patients were categorized into four groups based on their calculated BMI.

- Underweight: Body Mass Index (BMI) less than 18.5 kg/m2
- Optimal weight range: Body Mass Index (BMI) between 18.5 and 24.9 kg/m2
- Overweight refers to having a body mass index (BMI) between 25 and 29.9 kg/m2.
- Obese individuals are classified as having a Body Mass Index (BMI) that exceeds 30 kg/m2.

Laboratory analysis required the collection of a blood sample. After an 8-hour fast the night before, the patient will have a battery of tests to assess their liver function, including total bilirubin, as well as AST, ALT, and alkaline phosphatase. Their lipid profile will be completed, which includes total cholesterol, triglycerides, HDL, and LDL.The nephelometric approach, in conjunction with the turbox-sf analyzer, was used to quantify the plasma high-sensitivity C-reactive protein (hs-CRP) level.

➢ *Reference Ranges for Laboratory Values:*

- A range of 5 to 35 IU/L is considered typical for AST.
- An ALT level between 5 to 35 IU/L is considered normal.
- Levels of 44–147 IU/L of serum alkaline phosphatase are considered normal.
- Less than 1 mg/dl is considered normal for serum total bilirubin.

➢ *Research Design*

Radiological screening for fatty liver has led to this cross-sectional research, which intends to examine the clinical and laboratory features of individuals diagnosed with this condition. Serum hsCRP levels are also intended to be measured.

III. STATISTICAL ANALYSIS

In order to compare the means of the two groups of participants, the statistical analysis of the study's data was carried out using the 'z' test or the 'normal' test. We used Fisher's exact test where it was appropriate. A 5% significance threshold ($P = 0.05$) was used for the calculations. To find out how hsCRP related to other

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

variables, we used the Pearson product-moment correlation coefficient. Excel® and the statistical program SSPS were used to perform the study's predictive analyses.

➢ *Observations and Results*

As a result of 67 patients meeting the inclusion and exclusion criteria, this inquiry was conducted. The majority of patients who went through the screening process at the Master Health Checkup Clinic had fatty liver detected using ultrasonography.

- There were 41 female patients (61% of the total) and 26 male patients (39% of the total).
- Participants' average ages were 48 years with a standard deviation of 12.5 years.
- Forty patients, or over half of the total, were between the ages of 31 and 50.

➢ *Human Body Size and Measurements*

Body mass index, height, and waist size are all part of the anthropometric profile. Please see the average values in the table below. A Student's t-test was used to get the p-values.

Table 1 Anthropometric Traits of Men and Women

	Male	**Female**	**Pvalue**
Weight (kg)	72.23±12.36	67.4±13.25	0.14
Waist Circumference (cm)	85.73±11.58	83.7±12.61	0.51
BMI (kg/m2)	26.79±4.54	26.7±5.02	0.95

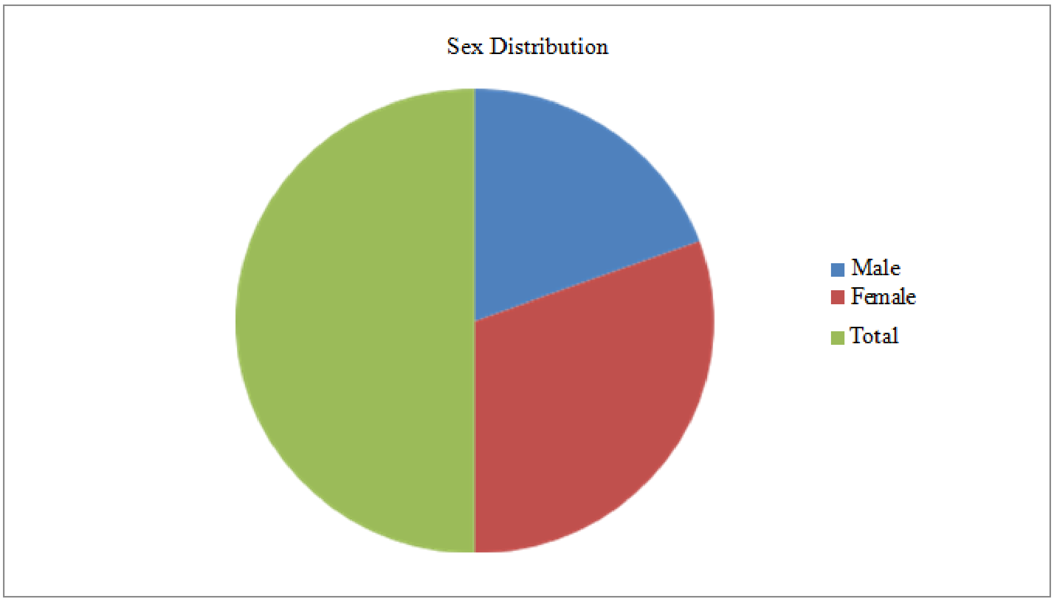

Fig 1 Sex Distribution Pie Chart

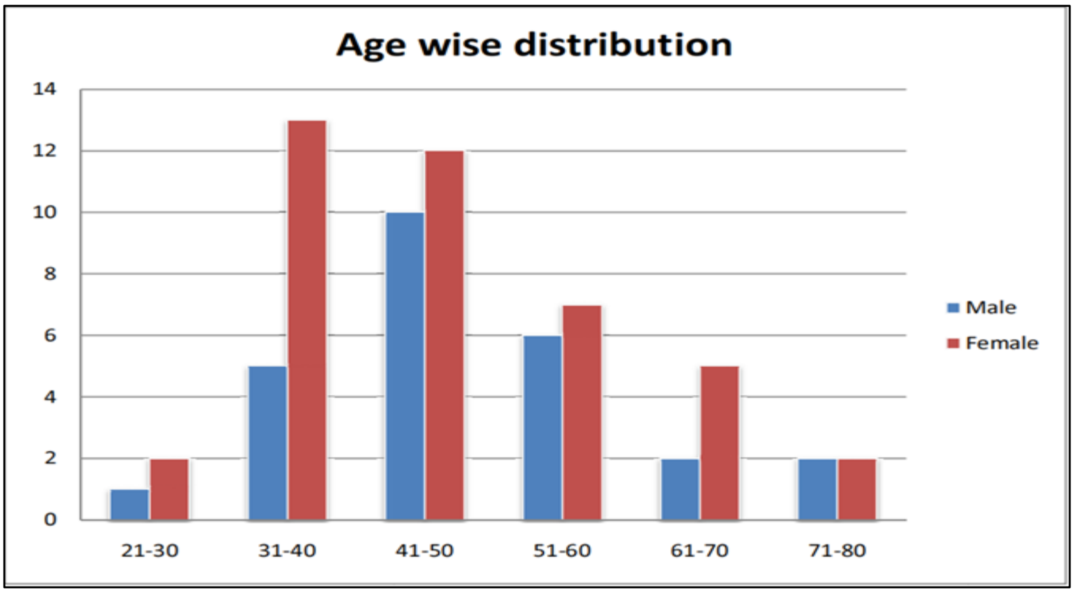

Fig 2 Age Distribution Bar Diagram

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

Males averaged 72.2±12.4 kg, while females averaged 67.4±13.3 kg. Males had a waist circumference of 85.73±11.58 cm, while females had an average of 83.7±12.61 cm. There were no significant differences in any of the measured factors between men and women.

➢ *Waist Circumference*

The information below applies to waist circumference. According to the ATP III criterion, 17 men and 19 females had an increased waist circumference.

Table 2 Waist Circumference in Men and Women

Waist Circumference (Males)	**Number**	**Percentage (%)**
<90 cm	17	65.4
>=90 cm	9	34.6

Waist Circumference (Females)	**Number**	**Percentage (%)**
<80 cm	19	46.3
>=80 cm	22	53.7

➢ *BMI*

The Body Mass Index (BMI) is determined by using the weight and height according to a certain formula. The Body Mass Index (BMI) is calculated by dividing the weight in kilograms by the square of the height in meters. The population was thereafter categorized into normal, overweight, obese class 1, class 2, and class 3 based on their BMI. The BMI figure for males was 26.79 with a standard deviation of 4.54, while for females it was 26.7 with a standard deviation of 5.02. The subsequent table presents the acquired results.

Table 3 BMI Classes of the Study Population

BMI (kg/m^2)	**Male**	**Female**	**Total**
Underweight with a BMI of 18.5	1	2	3
The range of 18.5-25 is considered normal.	10	16	26
from 25 to 29.9 Overweight persons	9	12	21
30–34.9 Class 1 Obesity	5	9	14
Class 2 Obesity, 35–39.9	0	1	1
Class 3 Obesity - >40	1	1	2

Among the total of 67 individuals, only 2 were diagnosed with morbid obesity. There were 26 patients that exhibited normal BMI features. Out of the total of 21 individuals, 14 of them were classified as having Class 1 obesity, while the remaining individuals were overweight. One patient exhibited class 2 obesity. There were three patients, one male and two females, who were classified as underweight according to their BMI.

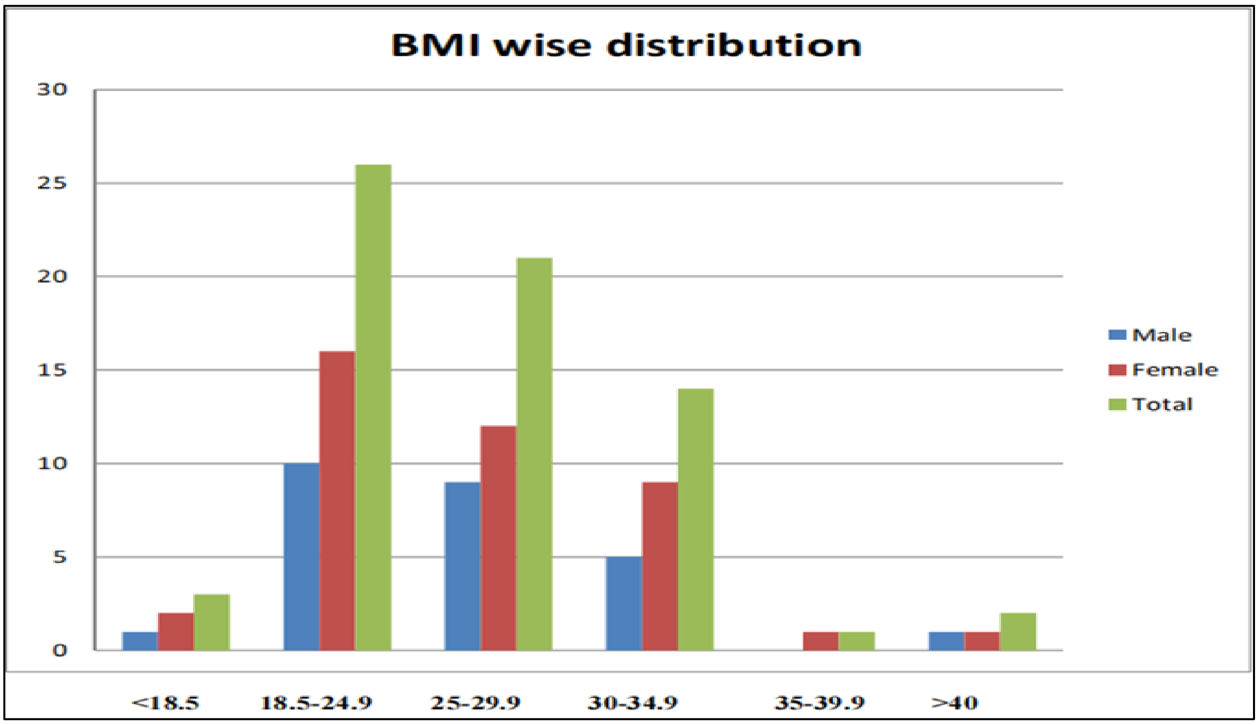

Fig 3 BMI Wise Distribution

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

➢ *Clinical Characteristics*

There were six patients who had a medically significant level of alcohol consumption. This accounts for 8.9% of the total population. Liver enlargement was observed in 6 cases, accounting for 8.9% of the total. Viral serology testing for hepatitis revealed negative results for both Hepatitis B and C in all 67 cases.

Table 4 Individuals' Medical Histories

Heavy Alcohol Consumption	**6(8.9%)**
Enlarged liver	6(8.9%)
HbsAg +ve , anti-HCV +ve	Nil

➢ *Blood Pressure*

Average blood pressure readings are 129.7 mm Hg during the systolic phase and 85.3 mm Hg during the diastolic phase. The gender gap in blood pressure readings is not statistically significant.

Table 5 Blood Pressure Differences between Males and Females

(mmHg)	**Male**	**Female**	
BP systolic	128.3±16.1	130.6±15.	0.57
BP diastolic	85.4±12.5	85.1±11.5	0.94

➢ *Laboratory Values*

The table below gives the comprehensive mean data of the laboratory values analyzed in the study.

Table 6 Mean Laboratory Values – Comprehensive Table

Fasting Blood Sugar (mg/dl)	**102 ± 38.49**
Total Cholesterol (mg/dl)	213.76±45.66
Triglycerides (mg/dl)	128.01±51.37
LDL (mg/dl)	140.0±42.91
HDL (mg/dl)	48.41±10.14
Total Bilurubin (mg/dl)	1±1.02
AST (IU/L)	32.71±15.34
ALT (IU/L)	32.74±22.42
ALP (IU/L)	108.6±53.2

➢ *Fasting Blood Sugar*

The mean blood sugar level was 102±38.49mg/dl. Among the 67 patients assessed, 19 (28%) were identified with either newly detected impaired fasting glucose levels or diabetes mellitus.

Table 7 Fasting Blood Sugar Values

Fasting Blood Sugar Values (mg/dl)	**n**	**Percentage (%)**
Fasting blood sugar level is less than 100.	48	71.6
FBS 100-126 (106)	10	14.9
FBS greater than 126	9	13.4

➢ *Lipid Profile*

With triglycerides at 128.01±51.37 mg/dl, HDL at 48.41±10.14 mg/dl, total cholesterol at 213.76±45.66 mg/dl, and LDL at 140.0±42.91 mg/dl. Table 5.8 shows the distribution of occurrences across various lipid level groups as per the ATP III recommendations.

Table 8 Adult Treatment Panel III Categorization of Total, LDL, and HDL Cholesterol Lipid Profiles

LDL Cholesterol (mg/dl)	**No. of Values**	**Percentage (%)**
LDL<100 - Optimal	12	17.9
100 to 129 - Near normal or above optimal	17	25.4
130 to 159 - Borderline high	20	29.9
160 to 189 - High	10	14.9
190 - Very High	8	11.9

Total cholesterol (mg/dl)	**No. of values**	**Percentage (%)**
<200 - Desirable	29	43.3
200-239 - Borderline High	20	29.9
240 - High	18	26.8

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

HDL cholesterol (Males) (mg/dl)	**No. of values**	**Percentage (%)**
<40 - Low	6	23.1
40 - High	20	76.9
HDL cholesterol (Females) mg/dl	No. of values	Percentage (%)
<50 - Low	21	51.2
50 - High	20	48.8

➤ *Liver Function Tests*

The studies group's liver function results are shown in the tables below.

Table 9 Liver Function Tests

	Number	**Percentage**
AST > 35 IU/L	20	29.8
ALT > 35 IU/L	19	28.3
ALP > 147 IU/L	10	14.9

Table 10 Liver Function Tests Compared in Alcoholics and Non Alcoholics

	Alcohol Related Fatty liver	**NAFLD**	**P value**
AST	44.66+33.0	31.54+12.25	0.38
ALT	57.5+63.5	30.5+12.19	0.34
ALP	112.5+27.5	108.16+55.21	0.74

➤ *Metabolic Syndrome*

The ATP III criteria were used to assess the prevalence of metabolic syndrome in the subject of the research population. Metabolic syndrome was identified in 19 out of 67 participants. You can see the numbers on the graph.

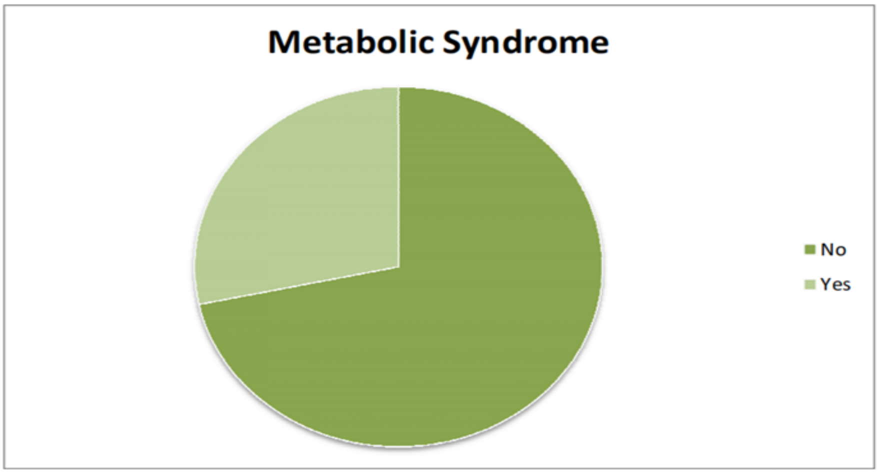

Fig 4 Shows the Proportion of Fatty Liver Individuals who have Metabolic Syndrome According to the NCEP – ATP III Recommendations for the Asian Population

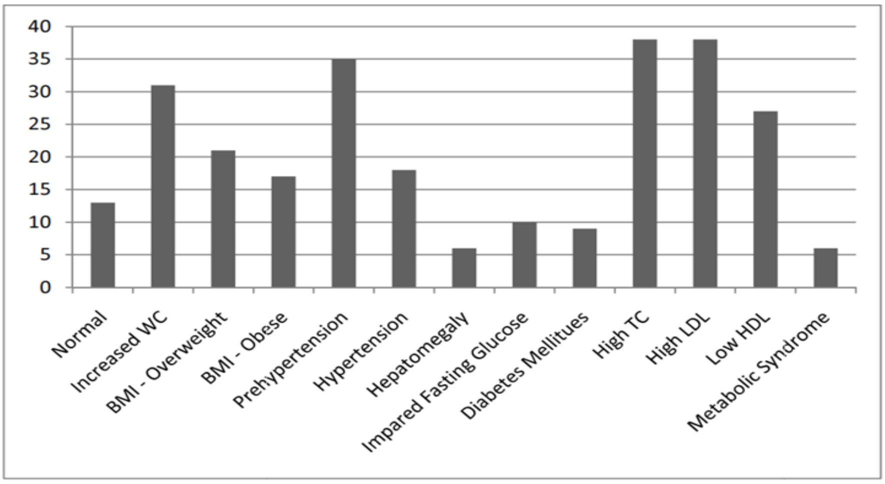

Fig 5 Displays a Bar Graph Illustrating Clinical and Laboratory Abnormalities

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

➢ *High Sensitive C – Reactive Protein Levels.*

High Sensitivity C-Reactive Protein (hsCRP) levels were measured in all 67 participants. Only cases with hsCRP levels less than 5mg/l were included in the research, as previously stated. The mean hsCRP level was 2.16±1.11 mg/l. The values ranged from 0.48 to 4.82.

- *The Table below Displays the Statistical Characteristics of the Blood Levels of hsCRP.*

Table 11 Displays the Statistical Properties of hsCRP Values.

Mean value	**2.1607**
Highest value	4.8200
Lowest value	0.4800
95% CI for the mean	1.8905 to 2.4310
Median	2.0800
95% CI for the median	1.7306 to 2.3298
Variance	1.2277
Standard deviation	1.1080
Relative standard deviation	0.5128 (51.28%)
Standard error of the mean	0.1354
Coefficient of Skewness	0.5931 (P=0.0455)
Coefficient of Kurtosis	-0.5229 (P=0.3087)
D'Agostino-Pearson test for Normal distribution	accept Normality (P=0.0807)

- *Below are the Mean hsCRP Levels for Men and Females. The Difference is not Statistically Significant.*

Table 12 Presents a Comparison of hsCRP Values between Men and Females

	Male (n=26)	**Female (n=41)**	**p-value**
hsCRP	2.21±1.143	2.18±1.117	0.41

The average hsCRP levels in individuals with metabolic syndrome and those lacking metabolic syndrome are respectively. The p-values are less than 0.001, showing a very strong statistical significance.

Table 13 Shows the Comparison of hsCRP Levels in Individuals with Fatty Liver who have Metabolic Syndrome and those who do not

Metabolic Syndrome (n=19)	**No Metabolic Syndrome (n=48)**	**p-value**
3.12±0.998	1.796±0.928	<0.001

There was a substantial variation in high sensitivity CRP levels among individuals with metabolic syndrome.When comparing the hsCRP levels of individuals with alcoholic and non-alcoholic fatty liver, the following results were observed.

Table 14 Comparison of hsCRP Values between Alcoholic and Nonalcoholic Individuals

	Related to alcohol Hepatic steatosis	**Fatal liver illness that is not caused by alcohol**	**p- value**
Mean high-sensitivity C-reactive protein	1.963±0.584	2.182±0.924	0.64

The mean hsCRP level among alcoholics was 1.963±0.584, and for the NAFLD group it was 2.182±0.924. There was no statistically significant disparity between the two groups.

Group, considering the study's limited sample size of individuals with alcoholic fatty liver.

The Centers for Disease Control and Prevention and the American Heart Association proposed a cardiovascular risk categorization based on hs-CRP levels in adults: low risk (<1.0 mg/L), moderate risk (1.0 to 3.0 mg/L), and high risk (>3.0 mg/L).

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

The research group is being categorized according to the information shown in table 14.

➢ *Correlation between Multiple Variables and hsCRP.*

The Pearson product-moment correlation coefficient was used to assess the association between fatty liver, hsCRP, and other clinical and laboratory findings. Table 15 shows the acquired findings.

The variables of the circumference of the waist and body mass index showed a modest degree of correlation. The correlation value of 0.61 indicates that waist circumference has the strongest link.

The blood pressure exhibited a 0.3 modest positive connection. Within the lipid profile, there was a weak positive connection observed for total cholesterol, triglycerides, and LDL, and a weak negative correlation observed for HDL.

The liver function test correlation coefficients were not highly significant.

The following scatter diagrams represent representative correlation variables with hsCRP levels on the X axis.

Table 15 The Study Population was Categorized into Different Degrees of Cardiovascular Risk based on their hsCRP Values

hsCRP levels	Number	Percentage (%)
<1	7	10.4
1-3	46	68.7
>3	14	20.9

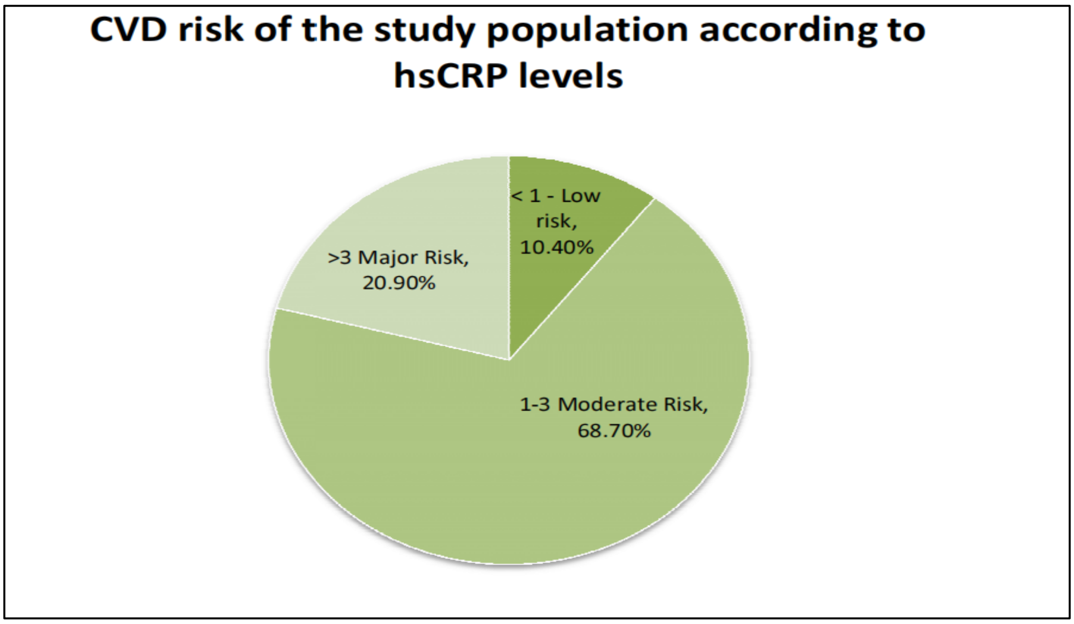

Fig 6 A Pie Chart Illustrating the Categorization of the Research Population's Cardiovascular Risk based on their hsCRP Values

Table 16 The Pearson Correlation Coefficient (r) Measures the Strength and Direction of the Linear Relationship between hsCRP and other Factors

Factor	Correlation	p-value
Age	-0.04	0.74
Waist	0.61	<0.001
Weight	0.51	<0.001
BMI	0.48	<0.001
BP(Systolic)	0.32	0.008
BP(Diastolic)	0.31	0.010
FBS	0.06	0.620
TC	0.31	0.010
TG	0.22	0.073
HDL	-0.26	0.033
LDL	0.3	0.013
T.Br	-0.06	0.629
AST	-0.01	0.936
ALT	0.1	0.420
ALP	-0.01	0.936

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

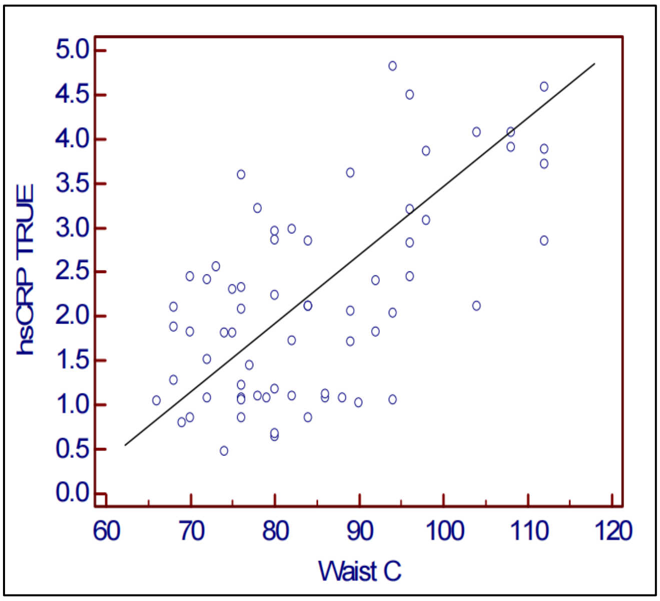

Fig 7 Displays a Scatter Plot Illustrating the Association between hsCRP (high-Sensitivity C-Reactive Protein) and Waist Circumference

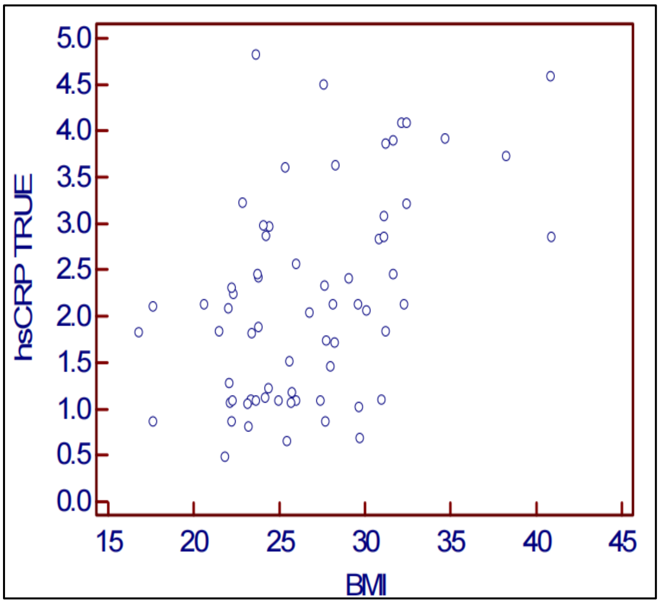

Fig 8 Displays a Scatter Plot Illustrating the Association between hsCRP (high-Sensitivity C-Reactive Protein) and BMI (Body Mass Index)

IV. DISCUSSION

➢ *Demographic Profile*

Following the application of the exclusion criteria, 67 cases in total were included in the study. Many cases with fatty liver diagnoses were eliminated from the study because patients with any concomitant symptom, disease, or chronic condition were not allowed to participate. Twelve individuals met the eligibility criteria despite having high hsCRP levels (greater than 5 mg/l). A number more than 5 mg/dl is thought to indicate a traumatic etiology, infection, inflammation, or surgical procedure rather than cardiovascular risk. [61] Thus, these situations were not included in the research.

Sixty-one percent of the participants in this study were female. The participants' average age was 48 ± 12.5 years. 40 patients, or the majority, were in the 31–50 age range. Nearly every study conducted in India states that the percentage of men is higher, if not equal. A male to female ratio of 2:1 was reported by Amarapurkar et al. in 2000, whereas a 3:1 ratio was recorded by Bhat et al. in 2006. [62,63] Naturally, this is dependent on the demographics of the people that visit the Master health check-up facility. However, this female predominance may be noteworthy given that six male patients with alcoholic fatty liver disease are also included.

➢ *Anthropometric Characteristics*

The mean BMI of the anthropometric variables that were measured was 26.7±4.9. Out of the total number of patients (n = 26; 39%), the largest proportion belonged to the Normal BMI category, followed by the Overweight group (n = 21; 31%). Several Indian research studies have shown similar conclusions. A research conducted in coastal eastern India found that only around 46% of patients with non-alcoholic fatty liver disease (NAFLD) were classified as overweight or obese. Interestingly, more than 50% of these patients had a normal body mass index (BMI) according to the standards set by the World Health Organization (WHO). In contrast, Uchil et al. and Duseja et al. found that only about 22% of patients had a normal BMI in their examinations.

In contrast, 40%–100% of NAFLD patients in the West have been found to have abnormal BMIs. [14, 15, 18], Therefore, it is clear that, in comparison to western data, a smaller proportion of individuals meet the criteria for obesity based on BMI values.

Race and population factors are not taken into account while assigning BMI groups. Given that Indians, particularly those from South India, are known to have higher levels of visceral fat than other populations, BMI measurements may be underestimating the hazards associated with obesity.

In this context, waist circumference appears to be more trustworthy. 46% of girls and 65% of males, respectively, had waist circumferences that were higher than the NCEP-ATP recommendations' cutoff points. According to Duseja et al.,72% of men and 93% of females surpassed the specified threshold for waist circumference, which is more than 90 cm for males and greater than 80 cm for females. However, other Indian research, such as Amarkpur et al.'s, which found that 57% of the patients had high WC, demonstrate a lower prevalence. [62, 63] All patients of fatty liver disease, however, seem to have waist circumferences that are higher than average.

➢ *Clinical Features and Laboratory Measurements.*

Throughout the trial, 35 pre-hypertensive and 18 new hypertensive individuals (or 27% of the total) were identified. In Chennai, South India, age-adjusted hypertension was found to be 21.8% in a sample of 532 men and 421 women over the age of 40, according to research by Ramachandran et al. The whole population was included in

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

this study, not people with fatty livers. Insulin resistance, the development of fatty liver, and the metabolic syndrome are all associated with essential hypertension. In the Journal of Human Hypertension, M. J. Brookes and B. T. Cooper emphasize the need of assessing liver function tests during diagnosis, considering the presence of fatty liver disease in patients with hypertension, and being vigilant about even minor increases in serum aminotransferases.

Given that these people had no symptoms and that their blood pressure would not have been discovered if not for the health screening, it is highly advised that patients with fatty livers be screened for hypertension.

The results of the study indicated that 9 individuals (13.4%) had diabetes mellitus and 10 out of the 67 (15%) asymptomatic patients presenting with fatty liver had impaired fasting blood sugars. This must be taken into account while keeping in mind that a study by Mohan et al. found that the general population in Chennai had a reported 13.5% diabetes prevalence. [64]

Additional studies conducted in India have shown a prevalence rate of 12-25% for diabetes mellitus. A recent research revealed that the occurrence of non-alcoholic fatty liver disease (NAFLD) among individuals with diabetes in Italy was determined to be 69.5%. In a research conducted on healthy Japanese persons with normal fasting glucose, the occurrence of non-alcoholic fatty liver disease (NAFLD) increased from 27% to 43% in those with impaired fasting glucose, and further jumped to 62% in those newly diagnosed with diabetes. Additional investigation conducted in the Asia-Pacific area found that 54.5% of patients with diabetes had NAFLD. The prevalence of this number is much higher than that of those with pre-diabetes (IGT or IFG) (33%), who in turn had a larger prevalence than those with normal glucose tolerance (22.5%). [65, 66]

Just six of the 67 patients exhibited heaptomegaly that was clinically significant. According to various research, the prevalence of hepatomegaly ranges from (Saudi series (2003) - (88%) Lal et al. (1991) - 40%; Virginia series (1996) - 22%. However, it was just 9% in our investigation. [67]

The mean transaminase values for ALT and AST across the liver function tests were 32.7±22.42 and 32.7±15.3, respectively. The liver function test results from the current investigation did not show a clinically meaningful correlation. In our investigation, almost 30% of participants exhibited elevated transaminases; we used 35 IU/L as the cutoff point for AST and ALT. According to Mofrad et al. (2003), there is little association between histology, ultrasonography, and biochemistry, and people with normal transaminase levels can exhibit the whole range of NAFLD histology. [68] According to certain studies, the liver enzyme levels of NAFLD patients vary, with up to 78% of individuals having normal values at any given moment. When levels are raised, they are often limited to either or both of the aminotransferases aspartate aminotransferase (AST) and alanine aminotransferase (ALT). In the absence of cirrhosis, the AST:ALT ratio may invert, but it is typically less than 1. [69] In the study group, the AST:ALT ratio was 0.9. Since this is a widely used technique to identify non-alcoholic fatty liver disease (NAFLD) in the general population, it is not unexpected that numerous studies have found that the level of alanine aminotransferase exhibited the strongest correlation with fatty liver.[70] However, our investigation was unable to uncover such a relationship. In clinical practice, aminotransferase levels are frequently used to estimate liver inflammation. However, because more than two-thirds of NASH patients have aminotransferase levels that fluctuate from normal levels at any given time, the use of ALT levels as a reliable indicator of the severity of NAFLD is controversial. [68, 69,70] . It was discovered that in individuals with fatty livers The similarity between those with normal and abnormal ALT levels at ultrasonography suggests that ALT is not a useful tool for distinguishing between various medical illnesses or disease stages. Alkaline phosphatase values were 108.6±53.2 on average.

The study population included a large number of lipid abnormalities. Two-thirds had a total cholesterol level above 240 mg/dl. Twelve percent, or 8 out of 67, had an LDL level higher than 190 mg/dl. Out of the 67, 38 had LDL readings higher than 130 mg/dl. 27 had low, abnormal HDL levels. Patients with fatty livers had lower levels of adiponectin and high-density lipoprotein (HDL) and greater levels of triglycerides, according to the Framingham Heart study. Globally conducted follow-up studies verified it.[71]

- *Alcohol Related Fatty Liver*

When asked about their past, six patients revealed that they had consumed a large amount of alcohol. Each of the six was a healthy male. With the exception of one patient who had slightly raised transaminase and bilurubin levels, there were no clinical signs of chronic liver disorders or significant abnormalities of liver function. Patients with alcohol-related fatty liver disease typically have normal body mass indexes and weights, with few additional abnormalities. Nevertheless, there were not enough patients (n=6) with alcohol-related conditions to draw any statistically meaningful findings.

- *Metabolic Syndrome*

Nineteen individuals, or 28% of the patients with fatty livers, had metabolic syndrome. If NAFLD patients alone are included, the percentage drops to 31%. 33 More people are realizing the connection between metabolic syndrome and non-alcoholic fatty liver disease (NAFLD). A study conducted by Lizardi-Cervera J et al. in Spain found that 22% of adult NAFLD patients have metabolic syndrome. Numerous investigations by Sameul et al., Hellen Kang et al., and others have determined that 18–40% of people have metabolic syndrome. Ninety percent of NAFLD patients may have at least one metabolic syndrome component. However, since less than one in three of the patients fit the criteria for the metabolic syndrome, it is still uncertain whether NAFLD can be referred to as the hepatic component of the syndrome.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

➢ *Hscrp Levels*

The mean hsCRP readings in this study were 2.61±1.11 mg/L. There is little data available about the average hsCRP levels in the general population.

Research has shown that when the amount of C-reactive protein (CRP) in the body exceeds 1 mg/L, it indicates a considerable likelihood of coronary artery disease (CAD). Consequently, the whole study group is susceptible to coronary artery disease, with an average of 2.61±1.11 mg/L. There is a correlation between the presence of common cardiovascular risk factors and increased levels of C-reactive protein (CRP) in the blood. This suggests that these risk factors may contribute to inflammation in the blood vessels. Hence, it is difficult to determine definitively whether the presence of fatty liver is a distinct factor that directly affects the levels of CRP in this study. Based on data from 11 early population-based studies, a meta-analysis conducted in 2000 found that there was a two-fold increase in relative risk (95% CI 1.6-2.5) for major coronary events between those with high and low levels of hs-CRP assessment or lipid profile, regardless of their clinical risk. Given that hsCRP has been identified as a separate risk factor for coronary artery disease (CAD), fatty liver and CAD may have a common cause. Ndumele C et al. [74] discovered that there is a separate and combined correlation between elevated systemic inflammation and hepatic steatosis, obesity, and the metabolic syndrome.

The AHA designated <1, 1 to 3, and >3 mg/L as low, intermediate, and high risk values. 46 patients (68.7%) and 14 patients (20.9%) in this investigation had hsCRP values greater than 1 mg/L and more than 3 mg/L, respectively.

Out of the study group, only 7 had CRP readings below 1 mg/L. The statistically significant high levels of hsCRP observed in patients with metabolic syndrome are another noteworthy observation. This is consistent with earlier research that indicates patients with metabolic syndrome have elevated levels of hsCRP. According to a study conducted in Spain by Rodríguez-Leal et al., hsCRP levels are high, but its low sensitivity and specificity in differentiating between different levels of hepatic inflammation limit its use in identifying the hepatic inflammatory response in patients with metabolic syndrome and non-alcoholic fatty liver disease.[72]

The relationship between the waist circumference and the blood levels of hsCRP is moderately to strongly positive (r = 0.61). Waist circumference demonstrated a stronger positive association than BMI and body weight. circumference of the waist appears to be the most appropriate anthropometric measure for classifying obesity risk in our sample. The fact that it is simple to measure and all that is required is a measuring tape. On the other hand, a height scale and weighing device are required for determining BMI. Additionally, the data calculation (weight/height 2) must be completed.

The results of this investigation are consistent with those of earlier investigations on fatty liver and hsCRP. CRP levels and related results were observed in the ARIC trial, which evaluated the relationship between incident CHD and 19 new risk indicators, including serum CRP, in almost 16,000 persons followed for up to 15 years. In contrast to simple non-progressive steatosis, a recent study found that patients with histologically proven NASH had higher serum levels of hs-CRP. Zhou et al.'s study from China WD demonstrates a strong relationship between insulin resistance and hsCRP. Patients with steatohepatitis have higher hsCRP values, according to a study done on the hipsanic population in Chile. [73,74] James P. Luyendyk and Grace L. Guo's editorial suggests that addressing nonalcoholic fatty liver disease might be a strategy to reduce systemic inflammation and decrease the likelihood of negative cardiovascular outcomes in individuals with metabolic syndrome. The reason for this is the presence of a robust association between fatty liver disease and hs-CRP levels. Seventy-five Based on our study, it is suggested that hs-CRP might be included as one of the non-invasive indicators for diagnosing NAFLD. Further investigation is needed to confirm this, but if proven, it would provide hepatologists and primary care physicians with an additional diagnostic tool for NAFLD.

➢ *Study Constraints*

- The research population consists of adults who often participate in health checks. They should not be considered representative of the wider population.
- Some research, such as serum iron levels, insulin levels, transferrin saturation, ferritin levels, and ceruloplasmin levels, were not conducted because to logistical and technical limitations. The liver biopsy could not confirm the presence of fatty liver as established by radiological imaging due to technical challenges and the patient's unwillingness to undertake an invasive procedure.
- Due to the limited number of alcohol-induced fatty liver cases, there was insufficient statistically meaningful data available from that group.

V. CONCLUSIONS

- A significant proportion of clinical and metabolic issues are seen in individuals who have been diagnosed with fatty liver but do not exhibit any symptoms.
- Obesity, pre-diabetes, dyslipidemias, hypertension, diabetes, and a large percentage of individuals were found to have these previously undetected conditions.
- While hepatomegaly, right hypochondrial pain, and generalized malaise are commonly associated with nonalcoholic fatty liver disease (NAFLD), a significant number of patients with fatty liver disease may remain undiagnosed in society despite having specific cardiovascular risk factors.
- Asymptomatic patients need comprehensive evaluation and treatments since they have a high prevalence of heart disease and atherogenic risk variables.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

- Out of the group with fatty livers, Out of the group with fatty livers, only 8.9% were alcoholics. The current count of non-alcoholic fatty liver disease patients is 61, accounting for 91.1% of the total.
- Waist circumference seems to be the most effective metric for measuring obesity and metabolic risk.
- No significant relationships were found between the AST and ALT levels, often used for diagnosing and categorizing NAF and NASH, and other factors in this study. This supports the notion that measuring AST and ALT levels is not a reliable or accurate method for diagnosing non-alcoholic fatty liver disease (NAFLD), since these values might vary even as the illness advances. Imaging or a biopsy must be included as additional components.
- Only a portion of NAFLD patients satisfied the criteria for metabolic syndrome as defined by the NCEP ATP III. Out of the patients who were qualified, nineteen had been diagnosed with metabolic syndrome.
- According to the AHA classification CRP for coronary artery risk, 93% of the research groups are susceptible to developing atherosclerotic coronary artery disease.
- Within the research group, a notable correlation was seen between CRP levels and weight, BMI, and waist circumference, indicating that CRP values tend to increase in individuals with obesity.
- Patients suffering from fatty liver disease due to metabolic syndrome had significantly higher levels of C-reactive protein (CRP).
- C-reactive protein (CRP) may aid in the categorization of the risk of fatty liver disease. In order to verify this, it is necessary to conduct a comprehensive research over an extended period of time.
- Further examination of CRP and other pro-inflammatory markers in fatty liver disease might provide valuable understanding of the cause of the ailment and perhaps lead to innovative targeted treatment methods.

REFERENCES

[1]. Joy D, Thava VR, Scott BB. Diagnosis of fatty liver diseases: is biopsy necessary? Eur J Gastroenterol Hepatol 2003;15;539-43.

[2]. Tarantino G. Is NAFLD an incidentaloma? Gastroenterology 2006;130:1014

[3]. Mark Feldman MD , Bruce F. Scharschmidt MD , Marvin H. Sleisenger MD, Sleisenger and Fordtran's Gastrointestinal and Liver Disease: Pathophysiology,Diagnosis,Management.

[4]. Flack-Ytter Y, Younossi ZM, Marchesini G, MC Cullough AJ. Clinical features and natural history of nonalcoholic steatosis syndromes. Semin Liver DIs 2001;21:17-26

[5]. Brunt EM, Tiniakos DG. Alcoholic and nonalcoholic fatty liver disease. IN: Odze RD, Goldblum JR, eds. Surgical Pathology of the GI Tract, Liver, Biliary Tract and Pancreas 2nd ed. Philadelphia : Elsevier;2009:1007-14 73

[6]. Farrell GC. Non alcoholic steatohepatitis: What is it, and why is it important in the Asia Pacific Region? J Gastroenterol Hepatol 2003:18;124-38

[7]. Ludwig J, Viggiano T, McGill D, Ott B: Nonalcoholic steatohepatitis. Mayo clinic experiences with a hitherto unnamed disease. Mayo Clin Proc 1980; 55:434-8

[8]. Kotronen A, Peltonen M, Hakkarainen A, et al. Prediction of non-alcoholic fatty liver disease and liver fat using metabolic and genetic factors. *Gastroenterology*. Sep 2009;137(3):865-72

[9]. Bacon BR, Farahvash MJ, Janney CG, Neuschwander Teteri BA. Nonalcoholic steatohepatitis: an expanded clinical entity. Gasteroenterology 1994;107:1103-9

[10]. Amarapurkar D, Kamani P, Patel N, et al: Prevalence of non-alcoholic fatty liver disease: Population based study. Ann Hepatol 2007; 6:161-3

[11]. Park S, Jeon W, Kim S, et al: Prevalence and risk factors of non-alcoholic fatty liver disease among Korean adults. J Gastroenterol Hepatol 2006; 21:138-43.

[12]. Flores Y, Yee HJ, Leng M, et al: Risk factors for chronic liver disease in blacks, Mexican Americans, and whites in the United States: Results from NHANES IV, 1999-2004. Am J Gastroenterol 2008; 103:2231-8.

[13]. Matteooni CA, Younossi ZM, Gramlich T, Boparai N, Liu YC, McCollough AJ. Nonalcoholic fatty liver diseases: a spectrum of clinical and pathological severity. Gastroenterology 1999;116:1413-9

[14]. Duseja A, Das A, Das R, et al. Clinicopathological proile of Indian patients with nonalcoholic fatty liver disease is different from the west. Dig Dis Sci 2007;52:2368-74.

[15]. Farrell GC, Larter CZ: Nonalcoholic fatty liver disease: From steatosis to cirrhosis. Hepatology 2006; 43:S99-S112.

[16]. Hsiao PJ, Kuo KK, Shin SJ, et al: Significant correlations between severe fatty liver and risk factors for metabolic syndrome. J Gastroenterol Hepatol 2007; 22:2118-23.

[17]. Ong JP, Younossi ZM. Epidemiology and natural history of NAFLD and NASH. Clin Liver Dis 2007;11:1-16

[18]. Duseja A, Das A, Dhiman RK, et al. Indian patients with NAFLD presenting with raised transaminases are different at presentation. World J Gastroenterol 2007;13:649-50.

[19]. Pathology of fatty liver disease. AU - Brunt EM , Mod Pathol. 2007 Feb;20 Suppl 1:S40-8

[20]. Nonalcoholic steatohepatitis: definition and pathology. Brunt EM, Semin Liver Dis. 2001;21(1):3

[21]. Lefkowitch JH, Haythe J, Regent N. Kupffer cell aggregation and perivenular distribution in steatohepatitis. Mod Pathol 2002;15:699-704

[22]. Feldstein AE, Werneburg NW, Canbay A, et al: Free fatty acids promote hepatic lipotoxicity by stimulating TNF expression via a lysosomal pathway. Hepatology 2004; 40:185-94.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

[23]. Gramlich T, Kleiner D, McCullough A, et al: Pathologic features associated with fibrosis in nonalcoholic fatty liver disease. Hum Pathol 2004; 35:196-
[24]. Flack-Ytter Y, Younossi ZM, Marchesini G, MC Cullough AJ. Clinical features and natural history of nonalcoholic steatosis syndromes. Semin Liver DIs 2001;21:17-26
[25]. Ong JP, Younossi ZM. Epidemiology and natural history of NAFLD and NASH. Clin Liver Dis 2007;11:1-16
[26]. Adams LA, Lymp JF, St. Sauver J, et al. The natural history of non-alcoholic fatty liver diseases - a population-based cohort study. Gastroenterology 2005;129:113-21
[27]. Ekstedt M, Franzen LE, Mathiensen UI, et al. Long-term follow-up of patients with NAFLD and elevated liver enzymes. Hepatology 2006;44:865-
[28]. Caldwell SH, Oliveira PMS, de Lima VMR, Park SH. NASH, cryptogenic cirrhosis and hepatocellular carcinoma, In: Bataller R, ed. International Update on Hepatology, Permanyer Publication; 2007.
[29]. Third report of the National Cholesterol Education Program (NCEP) Expert Panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). Circulation 2002; 106:3143
[30]. Ogden C, Yanovski S, Carroll M, Flegal K: The epidemiologyof obesity. Gastroenterology 2007; 132:2087-102.
[31]. Carter-Kent C, Zein NN, Feldstein AE: Cytokines in the pathogenesis of fatty liver and disease progression to steatohepatitis: Implications for treatment. Am J Gastroenterol 2008; 103:1036-42.
[32]. Zafrani E: Non-alcoholic fatty liver disease: An emerging pathological spectrum. Virchows Arch 2004; 444:3-12.
[33]. Kleiner DE, Brunt EM, Van Natta M, et al. Design and validation of a hisological scocring system for nonalcoholic fatty liver disease Hepatology 2005;41:1313-12
[34]. Kleiner DE, Brunt EM, Van Natta M, et al. Design and validation of a hisological scocring system for nonalcoholic fatty liver disease Hepatology 2005;41:1313-12
[35]. Gramlich T, Kleiner D, McCullough A, et al: Pathologic features associated with fibrosis in nonalcoholic fatty liver disease. Hum Pathol 2004; 35:196
[36]. Ramanchandran A, Snehalatha C, Satyavani K, Sivasankari S, Vijay V. Metabolic syndrome in urban Asian Indian adults – a population study using modified ATP-II criteria. Diabetes Res Clin Pract 2003;60:199-204.
[37]. Deepa R, Shanthirani CS, Premalatha G, Sastry NG, Moahan V. Prevalence of insulin resistance syndrome in a selected south Indian population- the Chennai urban population study – 7 [CUPS-7]. Indian J Med Res 2002;115:118-27.
[38]. Ratziu V, Giral P, Charlotte F, et al: Liver fibrosis in overweight patients. Gastroenterology 2000; 118:1117-23.
[39]. Mark Feldman MD , Bruce F. Scharschmidt MD , Marvin H. Sleisenger MD ,Sleisenger and Fordtran's Gastrointestinal and Liver Disease: Pathophysiology,Diagnosis,Management.
[40]. Stranges S, Dorn J, Muti P, et al: Body fat distribution, relative weight, and liver enzyme levels: A population-based study. Hepatology 2004; 39:754-63
[41]. David Sutton , Textbook of Radiology and Imaging, 7ed International 2003
[42]. Ronald G. Grainger MB ChB(Hons) MD FRCP DMRD FRCR FACR(Hon) FRACR(Hon) (Editor), David J. Allison BSc MD MRCS LRCP MB BS DMRD FRCR FRCP (Editor), Adrian K. Dixon MD FRCR FRCP FRCS FMedSci FFRRCSI(Hon) FRANZCR(Hon) (Editor)Grainger & Allison's Diagnostic Radiology: A Textbook of Medical Imaging
[43]. Noninvasive assessment of hepatic fibrosis . Michael P Curry, MD ,Nezam H Afdhal, MD, FRCPI, Section Editor Bruce A Runyon, MD , Deputy Editor - Peter A L Bonis, MD. Uptodate clinical reference 19.1
[44]. Neuschwander-Tetri B, Caldwell S: Nonalcoholic steatohepatitis: Summary of an AASLD single topic conference. Hepatology 2003; 37:1202-19.
[45]. Biochemical markers of liver fibrosis in patients with hepatitis C virus infection: a prospective study. Imbert-Bismut F; Ratziu V; Pieroni L; Charlotte F; Benhamou Y; Poynard T Lancet 2001 Apr 7;357(9262):1069-75.
[46]. Stranges S, Dorn J, Muti P, et al: Body fat distribution, relative weight, and liver enzyme levels: A population-based study. Hepatology 2004; 39:754-
[47]. Mofrad P, Contos MJ, Haque M, et al: Clinical and histologic spectrum of nonalcoholic fatty liver disease associated with normal ALT values. Hepatology 2003; 37:1286-92
[48]. Siegelman E, Rosen MA: Imaging of hepatic steatosis. Semin Liver Dis 2001; 21:71-80
[49]. Saadeh S, Younossi Z, Remer E, et al: The utility of radiological imaging in nonalcoholic fatty liver disease. Gastroenterology 2002; 123:745-50.
[50]. Mishra A, Mishra R. Asian Indians and insulin resistance syndrome: Global perspective. Med Syndr and Rel Disord 2003;1:277-8
[51]. Andersen T, Gludd C, Franzmann ME, et al. Hepatic effects of dietary weight loss in morbidly obese subjects. J Hepatol 1991-;12:224-9
[52]. Houmard JA, Tanner CJ, Slentz CA, et al. Effect of the volume and intensity of exercise training on insulin sensitivity. J Appl Physiol 2004;96;101-6
[53]. Angelico F, Burattin M, Alessandri C, et al. Drugs improving insulin resistance for non-alcoholic fatty liver disease and/or non-alcoholic fatty liver disease and/or non-alcoholic steatohepatitis. Chochrane Databse Syst Rev 2007:CD005166.

ISSN No:-2456-2165 https://doi.org/10.38124/ijisrt/IJISRT24MAR1124

[54]. Microvesicular steatosis of the liver. Hautekeete ML; Degott C; Benhamou JP ,Acta Clin Belg 1990;45(5):311-26
[55]. Clinical syndromes of alcoholic liver disease. Adachi M; Brenner DA Dig Dis. 2005;23(3-4):255-63.
[56]. Evaluation of nine automated high-sensitivity C-reactive protein methods: implications for clinical and epidemiological applications. Part 2. Roberts WL; Moulton L; Law TC; Farrow G; Cooper-Anderson M; Savory J; Rifai N Clin Chem 2001 Mar;47(3):418-25.1
[57]. Kushner, I. The phenomenon of the acute phase response. Ann N Y Acad Sci 1982; 389:39.1
[58]. Pearson, TA, Mensah, GA, Alexander, RW, et al. Markers of inflammation and cardiovascular disease: application to clinical and public health practice: A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. Circulation 2003; 107:499.
[59]. Pearson, TA, Mensah, GA, Alexander, RW, et al. Markers of inflammation and cardiovascular disease: application to clinical and public health practice: A statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. Circulation 2003; 107:499
[60]. High-sensitivity C-reactive protein: potential adjunct for global risk assessment in the primary prevention of cardiovascular disease. Ridker PM Circulation 2001 Apr 3;103(13):1813-8.
[61]. Amarapukar DN, Amaapurkar A. Non alcoholic statohepatitis clinicopathological profile. JAPI 2000;48;311-3
[62]. Bhat G, Pnadey, Baba CS et al. Metabolic syndrome in NAFLD patients an Indian perspective.
[63]. High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey Ramachandran, C. Snehalatha, A. Kapur, V. Vijay, V. Mohan, A. K. Das, P. V. Rao, C. S. Yajnik, K. M. Prasanna Kumar and Jyotsna D. Nair, et al.
[64]. Non-alcoholic fatty liver disease in the Asia-Pacific region: Definitions and overview of proposed guidelines.Chitturi S; Farrell GC; Hashimoto E; Saibara T; Lau GK; Sollano JDJ Gastroenterol Hepatol. 2007 Jun;22(6):778
[65]. The natural history of nonalcoholic Fatty liver disease: a population-based cohort study. Adams LA; Lymp JF; St Sauver J; Sanderson SO; Lindor KD; Feldstein A; Angulo P Gastroenterology 2005 Jul;129(1):113-21
[66]. Fan JG, Zhu G, Li XJ, et al. Prevalence of and risk factors for fatty liver in a general population of Shanghai China. J Hepatol 2—5;43:508-13.
[67]. Clinical and histologic spectrum of nonalcoholic fatty liver disease associated with normal ALT valuesPouneh Mofrad, Melissa J. Contos,Mahmadul Haque, Carol Sargeant,Robert A. isher, Velimir A. Luketic1 ,Richard K. Sterling, Mitchell L. Shiffman,Richard T. Stravitz, Professor of Medicine Arun J. Sanyal M.D.
[68]. The relationship between serum transaminase activities and fatty liver in children with simple obesity.Noguchi H, Tazawa Y, Nishinomiya F.
[69]. Mildly Elevated Liver Transaminase Levels in the Asymptomatic Patient Paul t Gibony., Keck School of Medicine, University of Southern California, Los Angeles, California.
[70]. Ramachandran A *et al.* Clustering of cardiovascular risk factors in urban Asian Indians. *Diabet Care* 1998; 21: 967–971.
[71]. Fatty liver is associated with dyslipidemia and dysglycemia independent of visceral fat: The Framingham heart studyElizabeth K. Speliotes Joseph M. Massaro Udo HoffmannRamachandran S. VasanJames B. Meig Dushyant V. SahaniJoel N. HirschhornChristopher J. O'Donnell3Caroline S. Fox
[72]. Assessment of high sensitivity C-reactive protein (HS-CRP) as a marker of liver inflammation in patients with metabolic syndrome.- Rodríguez-LealGA, Morán S, Gallardo I, Milke P, Guevara-González L.Source - Clínica de Gastroenterología, Hospital Medica Sur, México, DF.
[73]. Non-alcoholic fatty liver, high sensitivity C reactive protein and insulin resistance. Zhou WD, Yang YL, Che ZH.Department of Endocrinology of Second Xiangya Hospital, Institute of Metabolism and Endocrinology, Diabetes Center, Metabolic Syndrome Research Center, Central South University, Changsha 410011, China
[74]. Ndumele CE, Nasir K, Conceicao RD, Carvalho JA, Blumenthal RS, Santos RD Hepatic steatosis, obesity, and the metabolic syndrome are independently and additively associated with increased systemic inflammation. Arterioscler Thromb Vasc Biol. 2011;31:1927–1932.
[75]. EditorialsSteatosis DeLIVERs High-Sensitivity C-Reactive Protein - James P. Luyendyk, Grace L. Guo Atherosis, thrombosis and vascular biology.