Journal of Cardiovascular Disease Research
ISSN: 0975-3583, 0976-2833 VOL15, ISSUE 05, 2024

ASSOCIATION OF NAFLD IN CLINICAL AND SUBCLINICAL HYPOTHYROIDISM

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Abstract

Introduction: Liver is the largest organ in the body which is the main organ of metabolism and energy production. Any disruption in anatomical, physiological and functional aspects of liver leads to disease condition. Acute and chronic liver diseases can interfere with the functions of the liver and thereby cause symptoms. Liver disease rates are steadily increasing over the years. Chronic liver disease occurs throughout the world irrespective of age, sex, region or race. The most common liver diseases are of various types which include acute hepatitis, chronic hepatitis, fatty liver disease, cirrhosis and cancer.

Materials and Methods: A Hospital Based Cross Sectional Study conducted at Sri Manakula Vinayagar Medical College& Hospital, Kalitheerthalkuppam. Patients aged more than 18 years with hypothyroidism (subclinical on medication or recently diagnosed) were included in the study. Patients consuming significant alcohol, chronic viral hepatitis patients, Diabetes mellitus, Liver cirrhosis, acute liver failure, Hepatocellular carcinoma, Drug induced hepatitis, Metabolic syndrome were excluded from the study. Patients with clinical features of hypothyroidism (on medication), sub-clinical hypothyroidism will be undergoing following study. All patients included in the study will be examined with complete history and physical examination including anthropometry (weight, height, body mass index). The diagnosis will be confirmed by ultra sonography with ultrasonographic scoring system developed by Mahaling DU et al included hepatorenal echo contrast, liver brightness, deep attenuation, and vascular blurring. Score > or = 2 corresponded to NAFLD with a high, 92% sensitivity and 100% specificity, and a high level of intraobserver reliability.

Results: Mean age of subjects was 40.28 ± 13.17 years. Majority of subjects were in the age group 31 to 40 years (33.8%), 25% were in the age group <30 years, 23.8% were in the age group 41 to 50 years, 6.2% were in the age group 51 to 60 years and 61 to 70 years respectively and 5% were in the age group >70 years. In the study 48.8% had Grade 1 Fatty liver, 31.2% had Grade 2 Fatty liver, 6.2% had Grade 3 fatty liver and 13.8% had no fatty liver.

Conclusion: This study found that non-alcoholic fatty liver disease is more prevalent in untreated hypothyroid subjects. The severity of non-alcoholic fatty liver disease is more in patients with overt hypothyroidism. Therefore, ultrasonography of hypothyroid patients can be helpful in the early detection of fatty liver. Early treatment with thyroxine can prevent the progression of the fatty liver and grave consequences like non-alcoholic steatohepatitis and cirrhosis.
Key words: Chronic liver disease, NAFLD, Liver cirrhosis, acute liver failure, Hepatocellular carcinoma.

INTRODUCTION

Liver is the largest organ in the body which is the main organ of metabolism and energy production. Any disruption in anatomical, physiological and functional aspects of liver leads to disease condition. Acute and chronic liver diseases can interfere with the functions of the liver and thereby cause symptoms. Liver disease rates are steadily increasing over the years. Chronic liver disease occurs throughout the world irrespective of age, sex, region or race. The most common liver diseases are of various types which include acute hepatitis, chronic hepatitis, fatty liver disease, cirrhosis and cancer.

Liver diseases are recognized as the second leading cause of mortality amongst all digestive diseases in the US. According to World Health Organization Global Health Observatory (GHO) data among the causes of death cirrhosis of liver was the 4th leading cause of death among 50-59 years of age, 6rd leading cause of death among 30-49 yrs of age & 60-69yrs of age.

Nonalcoholic fatty liver disease (NAFLD) is defined based on the requirement that (a) there is evidence of hepatic steatosis, either by imaging or by histology and (b) there are no causes for secondary hepatic fat accumulation such as significant alcohol consumption, use of steatogenic medication or hereditary disorders. NAFLD is a rapidly growing diagnosis, and it is the most common cause of abnormal liver function tests worldwide.[2] The growing pattern of NAFLD prevalence is generally attributed to a global increase in the prevalence of obesity and other metabolic risk factors.[3]

NAFLD is histologically further categorized into nonalcoholic fatty liver (NAFL) and nonalcoholic steatohepatitis (NASH), which is a more progressive type of liver disease. . NAFL is defined as the presence of hepatic steatosis with no evidence of hepatocellular injury in the form of ballooning of the hepatocytes. NASH is defined histologically by presence of HS with evidence for hepatocyte damage.[1] Although some of the pathological features can be associated with hepatic fibrosis (HF), the most important histological feature associated with mortality in NASH is presence of significant fibrosis.[2,3] NASH has been recognized as one of the leading

causes of cirrhosis in adults in the United States.[4,5] and NASHrelated cirrhosis is currently the second indication indication for liver transplants in the United States.[6,7]

Clinically, NAFLD patients tend to be obese, with insulin resistance and/or type 2 diabetes, dyslipidemia, hypertriglyceridemia, and hypertension, which are all risk factors for cardiovascular diseases (CVDs).[9] The overall obesity prevalence estimates among NAFLD patients and among NASH patients were 51.34% and 81.83% respectively. The overall diabetes prevalence estimates among NAFLD and among NASH patients were 22.51% and 43.63%. The overall hyperlipidemia/dyslipidemia prevalence estimates among NAFLD and among NASH patients were 69.16% and 72.13%. Overall hypertension prevalence estimates among NAFLD and among NASH patients were 39.34% and 67.97%.[9]

Prevalence of NAFLD in patients with components of Metabolic syndrome is high.[10] The overall MetS prevalence estimates among NAFLD and among NASH patients were 42.54% and 70.65%. Over 90% of severely obese patients undergoing bariatric surgery have NAFLD. Given the common risk factors between NAFLD and CVDs, cardiac-related death is one of the leading causes of death for NAFLD patients.

Hypothyroidism is associated with decreased thermogenesis, decreased metabolic rate, and has also been shown to correlate with a higher body mass index (BMI) and a higher prevalence of obesity. There is clinical evidence suggesting that even mild thyroid dysfunction in the form of subclinical hypothyroidism is linked to significant changes in body weight and represents a risk factor for overweight and obesity. TSH levels are at the upper limit of the normal range or slightly increased in obese children, adolescents, and adults and are positively correlated with BMI.

OBJECTIVES

1. To identify the presence of NAFLD in hypothyroid patients, both clinical and subclinical and grade its severity.

2. Correlate the grade of NAFLD with the thyroid hormone levels FT3, FT4, lipid profile, liver enzymes, insulin resistance and body weight.

MATERIALS AND METHODS

Study Design: Hospital Based Cross Sectional Study

Study Area: Sri Manakula Vinayagar Medical College& Hospital, Kalitheerthalkuppam.

Study Population: The Patient's who attend the outpatient and admitted as inpatient in the department of General Medicine.

Inclusion Criteria:

Patients aged more than 18 years with hypothyroidism (subclinical on medication or recently diagnosed)

Exclusion Criteria:

1) Patients consuming significant alcohol.

2) Chronic viral hepatitis patients.

3) Diabetes mellitus.

4) Liver cirrhosis.

5) Acute liver failure.

6) Hepatocellular carcinoma.

7) Drug induced hepatitis.

8) Metabolic syndrome.

Sample Size:

S.S Formula= $4pq / l^2$where p value is 27.4%, q value is p-q, so q=72.6% and l is constant.

Sample Size was calculated by using above formula & the Sample size was calculated to be 79.56, rounded off to 80.

So sample size is 80.

Study Duration: 18 months

Methods:

1. Patients with clinical features of hypothyroidism (on medication), sub-clinical hypothyroidism will be undergoing following study

2. All patients included in the study will be examined with complete history and physical examination including anthropometry (weight, height, body mass index)

3. The diagnosis will be confirmed by ultra sonography with ultrasonographic scoring system developed by Mahaling DU et al[51]included hepatorenal echo contrast, liver brightness, deep attenuation, and vascular blurring. Score > or = 2 corresponded to NAFLD with a high, 92% sensitivity and 100% specificity, and a high level of intraobserver reliability.

Fatty liver will be graded[51] according to:

Grade I:

Minimal diffuse increase in the fine echoes. Liver appears bright compared to the cortex of the kidney. Normal visualization of diaphragm and intahepatic vessel borders.

Grade II:

Moderate diffuse increase in the fine echoes. Slightly impaired visualization of the intrahepatic vessels and diaphragm

Grade III:

Marked increase in the fine echoes. Poor/no visualization of intrahepatic vessels and diaphragm and poor penetration of the posterior, segment of the right lobe of the liver.

4. A valid informed consent in patient's native language will be obtained after explaining to the patient about the nature of study and any queries from the patients will be cleared.

5. The study was conducted after obtaining ethical committee clearance.

Instrumentation:

1.Blood Glucose levels both fasting and post-prandial are calculated by Glucose oxidase and glucose peroxidase method. (cut off values being FBS-106, PPBS-140)

2. Fasting Lipid Profile (Total cholesterol-220, triglycerides-160, HDL-60, LDL-165,VLDL-35), where total cholesterol and triglycerides are calculated by using cholesterol oxidase and cholesterol peroxidase, HDL by direct method, and LDL and VLDL are calculated.

3. Blood urea (40mg/dl) and serum creatinine (1.2mg/dl), where blood urea is calculated by UV kinetic method and serum creatinine by picric acid method

4. Thyroid profile (FT3, FT4, TSH), calculated by chemiluminiscence immunoassay method (CLIA) and cut off values being for FT3-4.2pg/ml,FT4-1.76ng/dl,TSH-5.5mIU/ml

5. Ultra sonography abdomen; to study the presence of fatty liver and its severity.

6. Liver enzymes (AST-50, ALT-37), calculated by UV kinetic method.

7. TPO antibody, calculated by CLIA (chemiluminiscence immunoassay method).

8. HOMA-IR (HOMEOSTASIS ASSESSMENT INDEX)

HOMA-IR will be calculated by using the following formula:

$$\frac{\text{fasting plasma glucose levels X fasting insulin levels}}{405}$$

More than 2 considered as insulin resistance, where fasting insulin levels are calculated by CLIA (chemiluminiscence immunoassay method).

List Of Variables		Measurement Plan
Age		Date of Birth
Gender		Observation
Occupation		Where and what do you work?
Symptoms		Patient history and medical records
Family History		Any similar family history?
Co morbid conditions	Diabetes mellitus	Patient history and medical records
	Hypertension	
Blood pressure	Systolic	
	Diastolic	
Thyroid profile	FT3	
	FT4	
	TSH	
Lipid profile	Total cholesterol	
	Triglycerides	
	LDL	
	VLDL	
	HDL	
Liver Enzymes	ALT	
	AST	
Anthropometry	Height(in metres)	
	Weight(in kgs)	
	BMI	
Blood glucose	Fasting	
	Post-prandial	
Renal function test	Blood urea	
	Serum creatinine	

TPO antibody		
HOMA-IR		
Ultrasoonography abdomen	Grade-0	
	Grade-1	
	Grade-2	
	Grade-3	

BIAS:

Anticipated Biases in The Study	Plan to Address The Anticipated Biases
Interviewer Bias	Use of pre-designed, pretested semi-constructed questionnaire for patient history.
Observational Bias	Observer will be trained in interpretation of clinical investigations conducted and of USG, which will minimize observational bias.
Misclassification Bias	The experienced faculty from General medicine will diagnose the hypothyroidism and experienced faculty from Radiodiagnosis will perform the USG.
Measurement Bias	Using a validated instrument used successfully for other studies.

Statistical Analysis:

Data obtained from the study groups will be entered in Microsoft Excel and analyzed with the use of computer software SPSS version. Study variables will be described in terms of frequency, mean, standard deviation (S.D) and then people with fatty liver and hypothyroidism will be compared using spearmen's correlation. A p value <0.05 is considered to be significant.

Implication of the study:

As we come across many non-alcoholic patients, especially females presenting with subclinical hypothyroidism and patients with unexplained fatty liver for whom routinely checking thyroid function will be worthwhile if this study is undertaken.

RESULTS

Table 1: Age distribution of subjects

		Count	%
Age	<30 years	20	25.0%
	31 to 40 years	27	33.8%
	41 to 50 years	19	23.8%
	51 to 60 years	5	6.2%
	61 to 70 years	5	6.2%
	>70 years	4	5.0%
	Total	80	100.0%

Mean age of subjects was 40.28 ± 13.17 years. Majority of subjects were in the age group 31 to 40 years (33.8%), 25% were in the age group <30 years, 23.8% were in the age group 41 to 50 years, 6.2% were in the age group 51 to 60 years and 61 to 70 years respectively and 5% were in the age group >70 years.

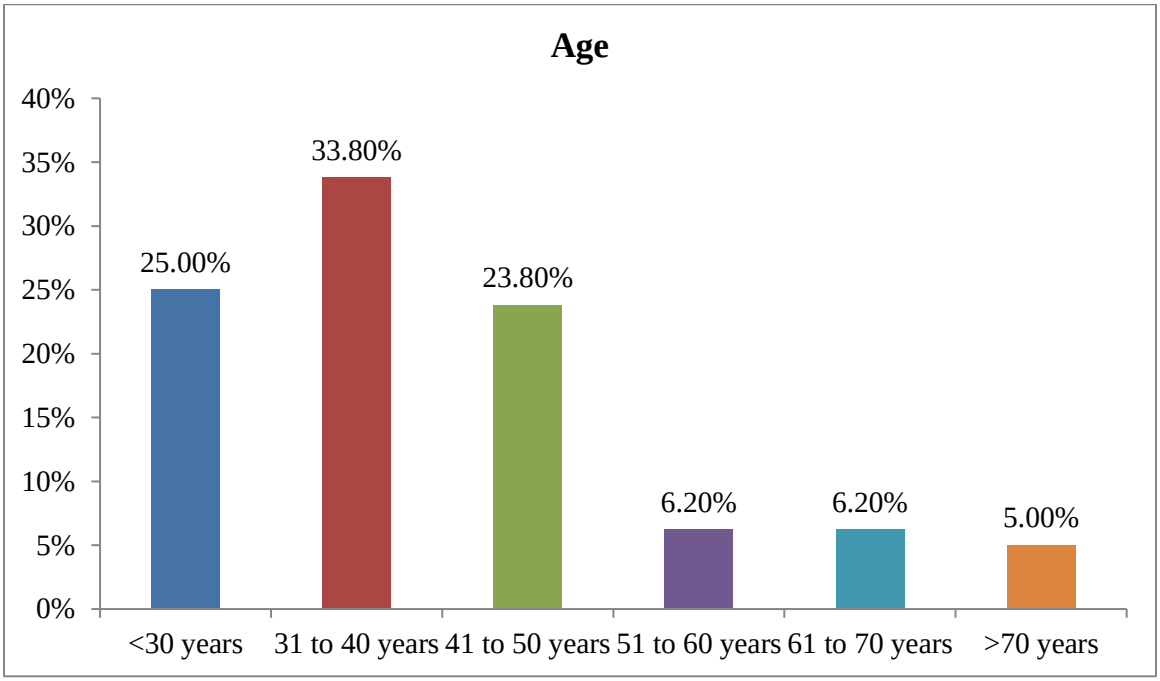

Figure 1: Bar diagram showing Age distribution of subjects

Table 2: Sex distribution of subjects

		Count	%
Sex	Female	65	81.2%
	Male	15	18.8%

In the study 81.2% were females and 18.8% were males.

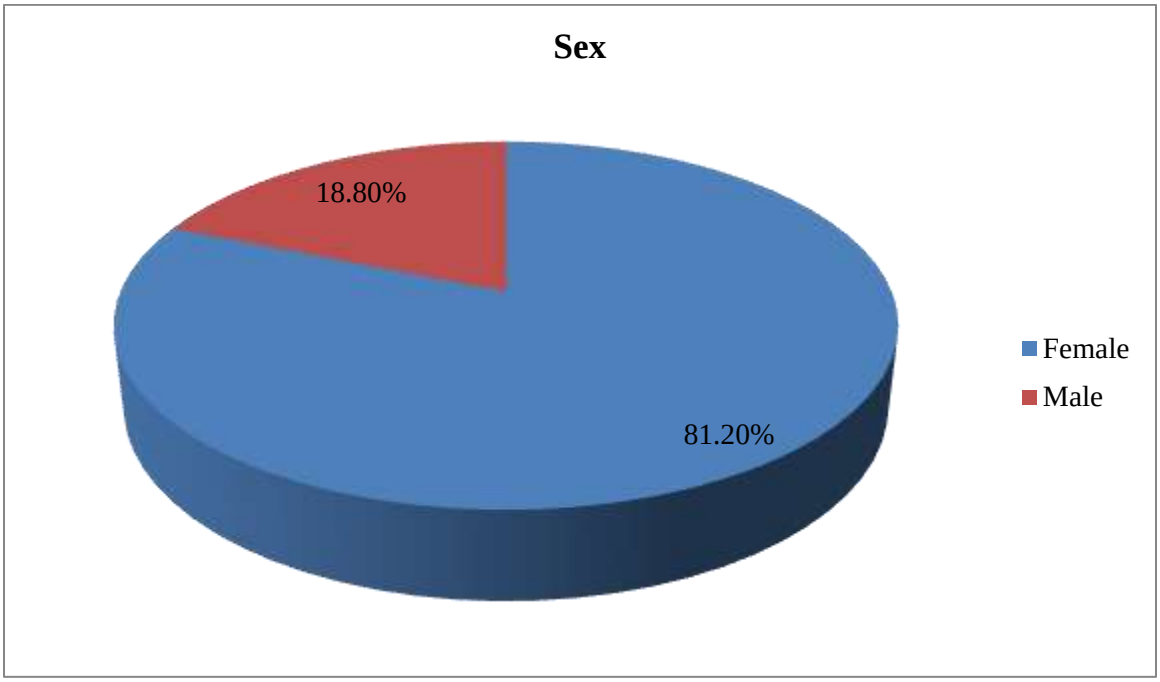

Figure 2: Pie diagram showing Sex distribution of subjects

Table 3: Grade of Fatty Liver distribution among subjects

		Count	%
Grade of Fatty Liver	Grade-1	39	48.8%
	Grade-2	25	31.2%
	Grade-3	5	6.2%
	NAD	11	13.8%

In the study 48.8% had Grade 1 Fatty liver, 31.2% had Grade 2 Fatty liver, 6.2% had Grade 3 fatty liver and 13.8% had no fatty liver.

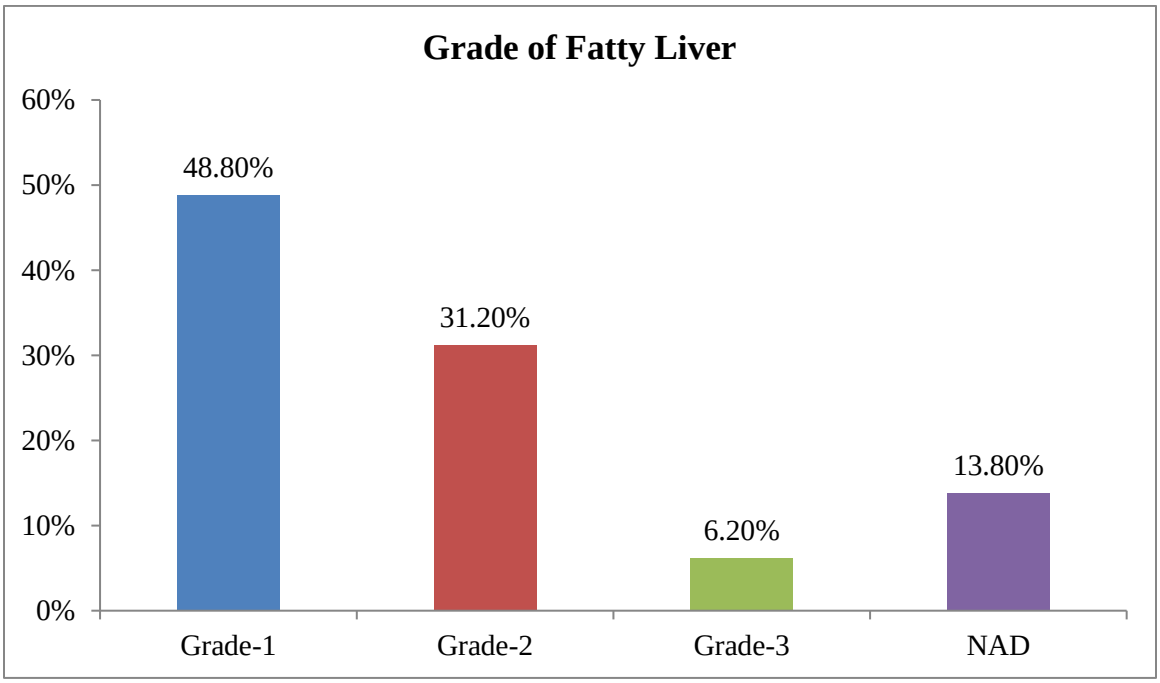

Figure 3: Bar diagram showing Grade of Fatty Liver distribution among subjects

Table 4: Comparison of Thyroid Profile mean values with respect to Grade of Fatty Liver

	Grade						P value
	Grade 1		Grade 2		Grade 3		
	Mean	SD	Mean	SD	Mean	SD	
FT3 (pg/ml)	2.38	1.03	1.18	0.57	0.64	0.36	<0.001*
FT4 (ng/dl)	1.01	0.77	0.42	0.23	0.22	0.08	<0.001*
TSH (mIU/ml)	46.44	47.45	131.48	22.35	141.60	18.78	<0.001*
Anti-TPO (IU/ml)	100.68	306.85	27.23	38.97	325.60	712.42	0.116

In the study mean FT3 among those with Grade 1 Fatty live was 2.38 ± 1.03 pg/ml, among those with Grade 2 fatty liver was 1.18 ± 0.57 pg/ml and among those with Grade 3 fatty liver was 0.64 ± 0.36 pg/ml. There was significant difference in mean FT3 between different grades of fatty was statistically significant.

Mean FT4 among those with Grade 1 Fatty live was 1.01 ± 0.77 ng/ml, among those with Grade 2 fatty liver was 0.42 ± 0.23 ng/ml and among those with Grade 3 fatty liver was 0.22 ± 0.08 ng/ml. There was significant difference in mean FT4 between different grades of fatty was statistically significant.

Mean TSH among those with Grade 1 Fatty live was 46.44 ± 47.45 mIU/ml, among those with Grade 2 fatty liver was 131.48 ± 22.35 mIU/ml and among those with Grade 3 fatty liver was

141.60 ± 18.78 mIU/ml. There was significant difference in mean TSH between different grades of fatty was statistically significant.

There was no significant difference in mean Anti TPO between different grades of fatty liver.

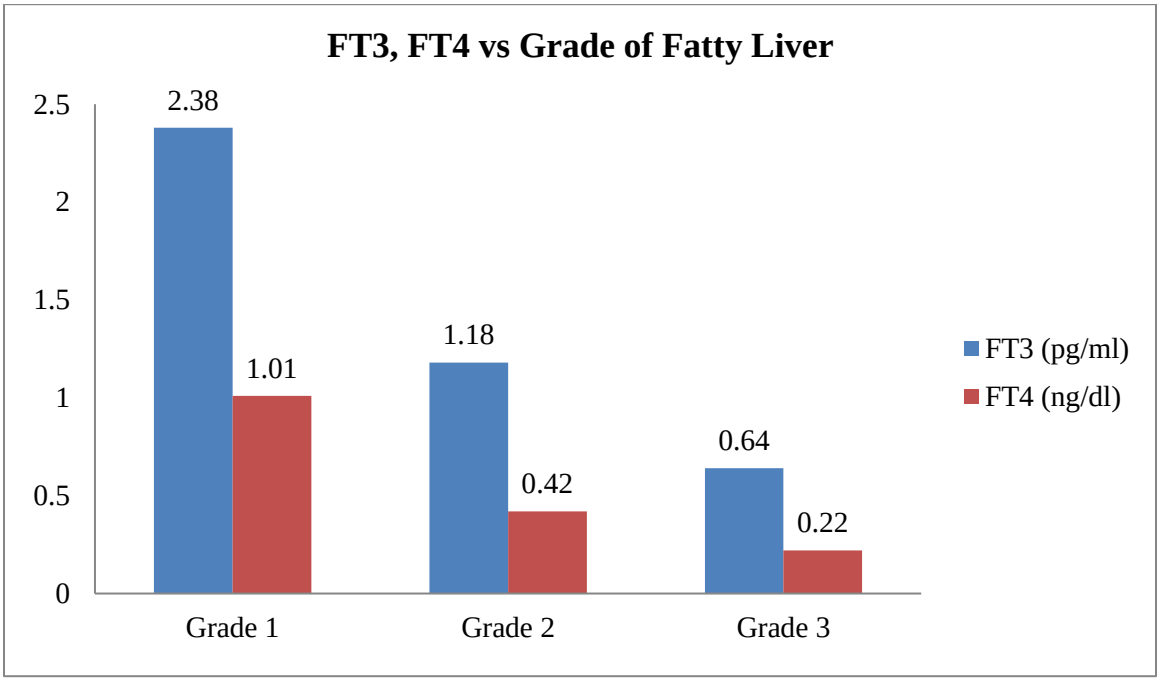

Figure 4: Bar diagram showing Comparison of Thyroid Profile mean values with respect to Grade of Fatty Liver

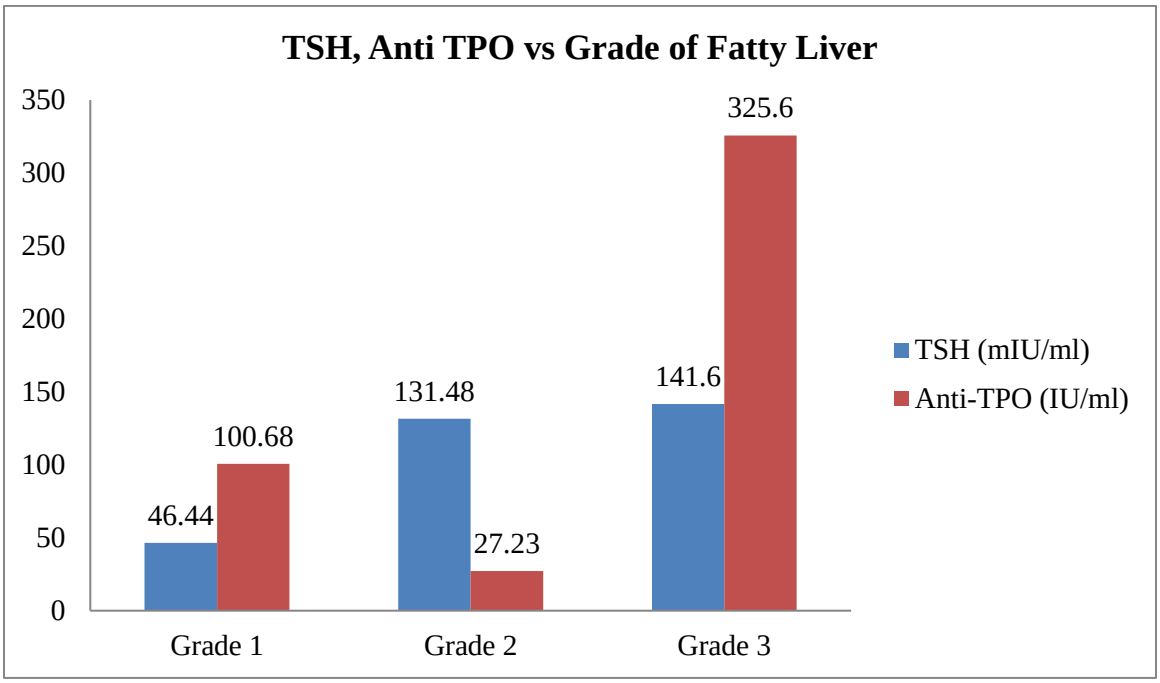

Figure 5: Bar diagram showing Comparison of TSH and Anti TPO mean values with respect to Grade of Fatty Liver

Table 5: Comparison of HOMA IR values with respect to Grade of Fatty Liver

	Grade						P value
	Grade 1		Grade 2		Grade 3		
	Mean	SD	Mean	SD	Mean	SD	
HOMA-IR (uIU/ml)	32.26	23.02	35.30	24.70	30.80	26.41	0.859

In the study mean HOMA-IR among those with Grade 1 Fatty live was 32.26 ± 23.02 uIU/ml, among those with Grade 2 fatty liver was 35.30 ± 24.70 uIU/ml and among those with Grade 3 fatty liver was 30.80 ± 26.41 uIU/ml. There was no significant difference in mean HOMA-IR between different grades of fatty was statistically significant.

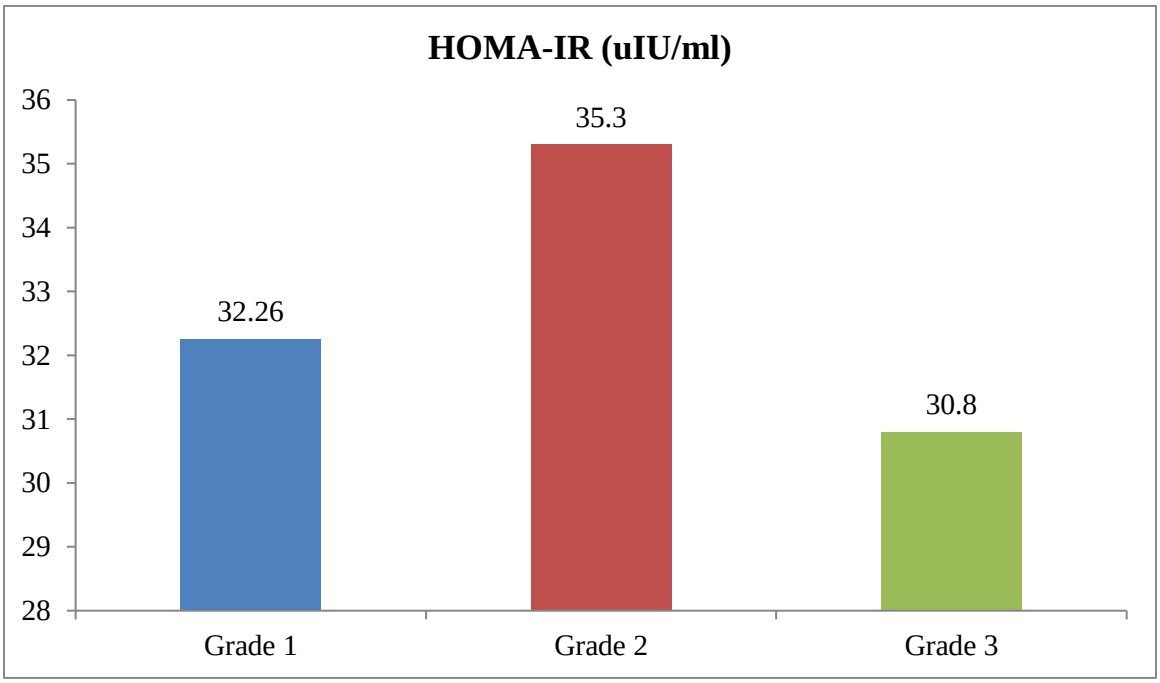

Figure 6: Bar diagram showing Comparison of HOMA IR values with respect to Grade of Fatty Liver.

Table 6: Comparison of BMI values with respect to Grade of Fatty Liver

	Grade						P value
	Grade 1		Grade 2		Grade 3		
	Mean	SD	Mean	SD	Mean	SD	
BMI (kg/m2)	20.73	1.51	21.30	1.99	22.32	2.25	0.115

In the study mean BMI among those with Grade 1 Fatty live was 20.73 ± 1.51 kg/m2, among those with Grade 2 fatty liver was 21.30 ± 1.99 kg/m2 and among those with Grade 3 fatty liver was 22.32 ± 2.25 kg/m2. There was no significant difference in mean BMI between different grades of fatty was statistically significant.

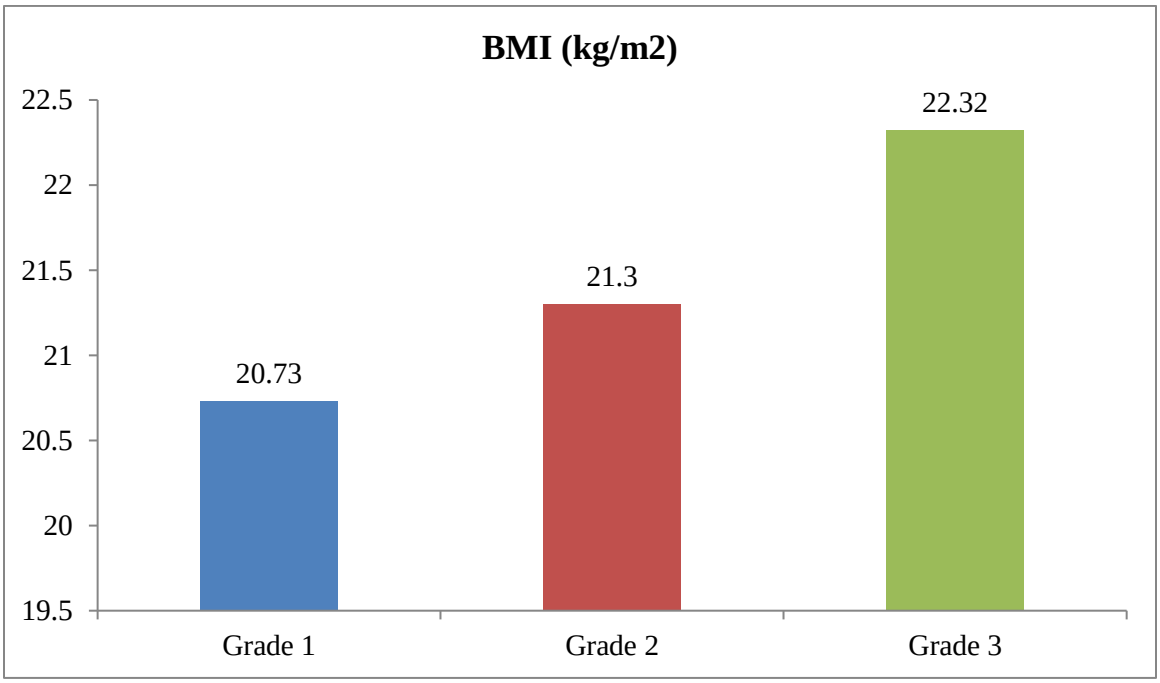

Figure 7: Bar diagram showing Comparison of BMI values with respect to Grade of Fatty Liver

able 7: Correlation between Grade of Fatty Liver with FT3, FT4, TSH, Anti TPO, HOMA IR and BMI

Correlations									
			Grade	FT3 (pg/ml)	FT4 (ng/dl)	TSH (mIU/ml)	ANTI-TPO (IU/ml)	HOMA-IR (uIU/ml)	BMI (kg/m2)
Spearman's rho	Grade of Fatty liver	Correlation Coefficient	1.000	-0.713**	-0.659**	0.684**	-0.043	0.019	0.236
		P value	.	<0.001*	<0.001*	<0.001*	0.727	0.878	0.051
		N	69	69	69	69	69	69	69
**. Correlation is significant at the 0.01 level (2-tailed).									

In the study there was significant negative correlation between Grade of fatty liver with FT3, FT4. I.e. with increase in Grade of Fatty liver there was decrease in FT3 and FT4 and vice versa.

There was significant positive correlation between Grade of fatty liver with TSH. I.e. with increase in Grade of Fatty liver there was increase in TSH and vice versa.

There was no significant correlation between Grade of Fatty liver with Anti TPO, HOMA IR and BMI.

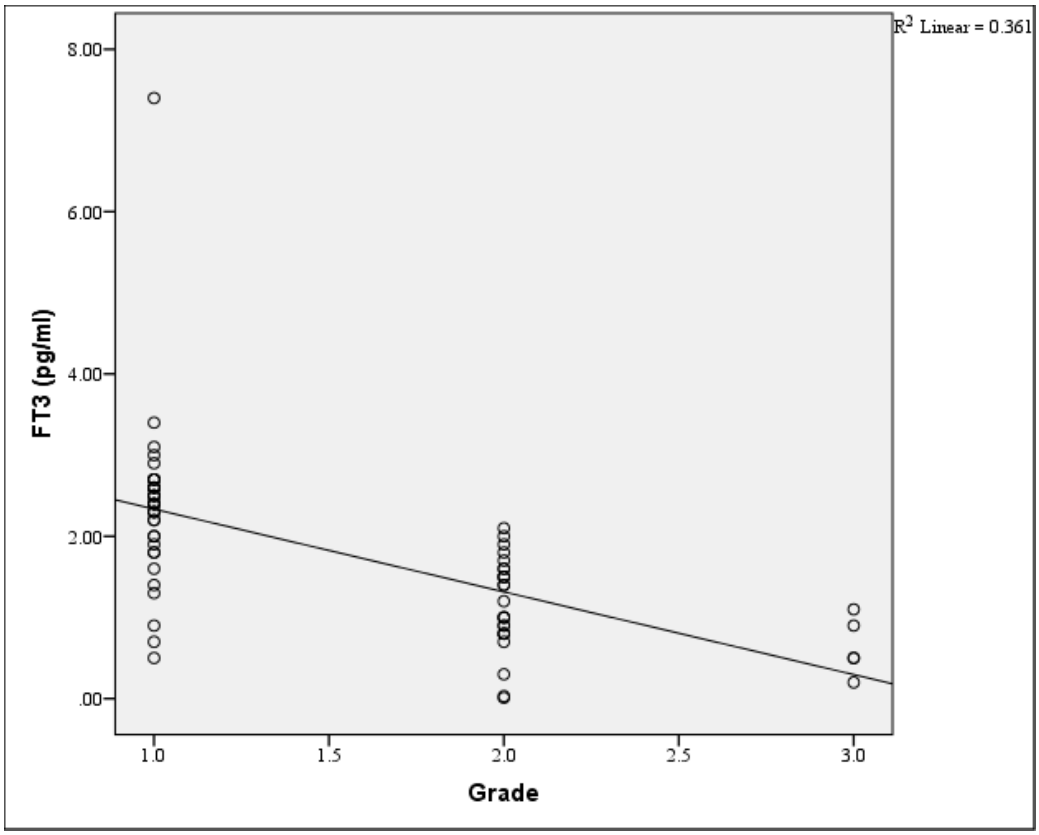

Figure 8: Scatter Plot showing Negative Correlation between Grade of Fatty Liver with FT3

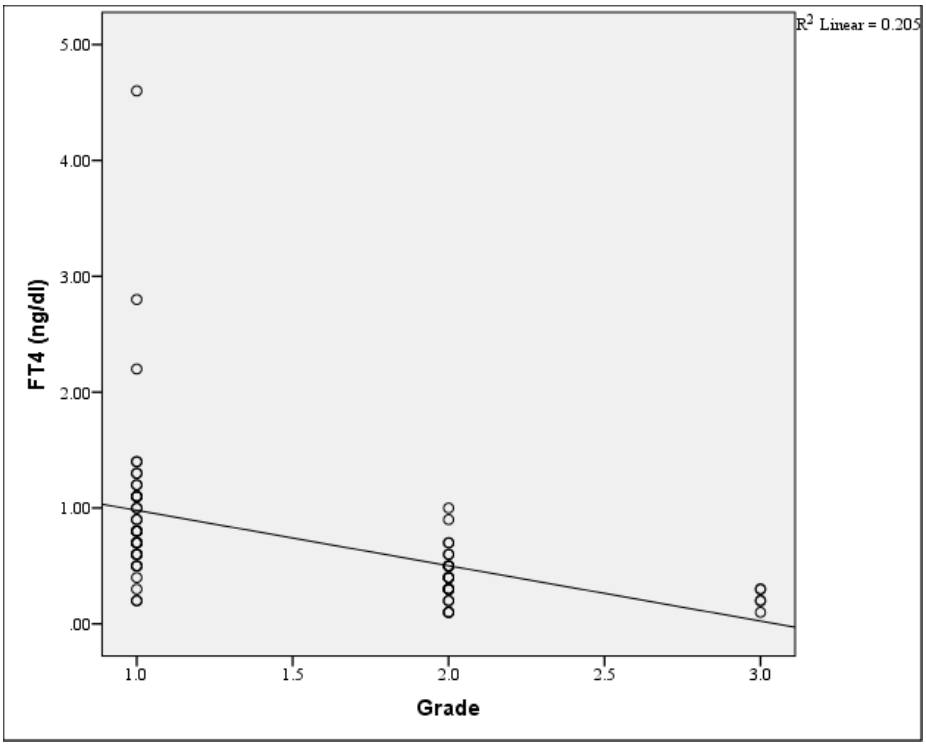

Figure 9: Scatter Plot showing Negative Correlation between Grade of Fatty Liver with FT4

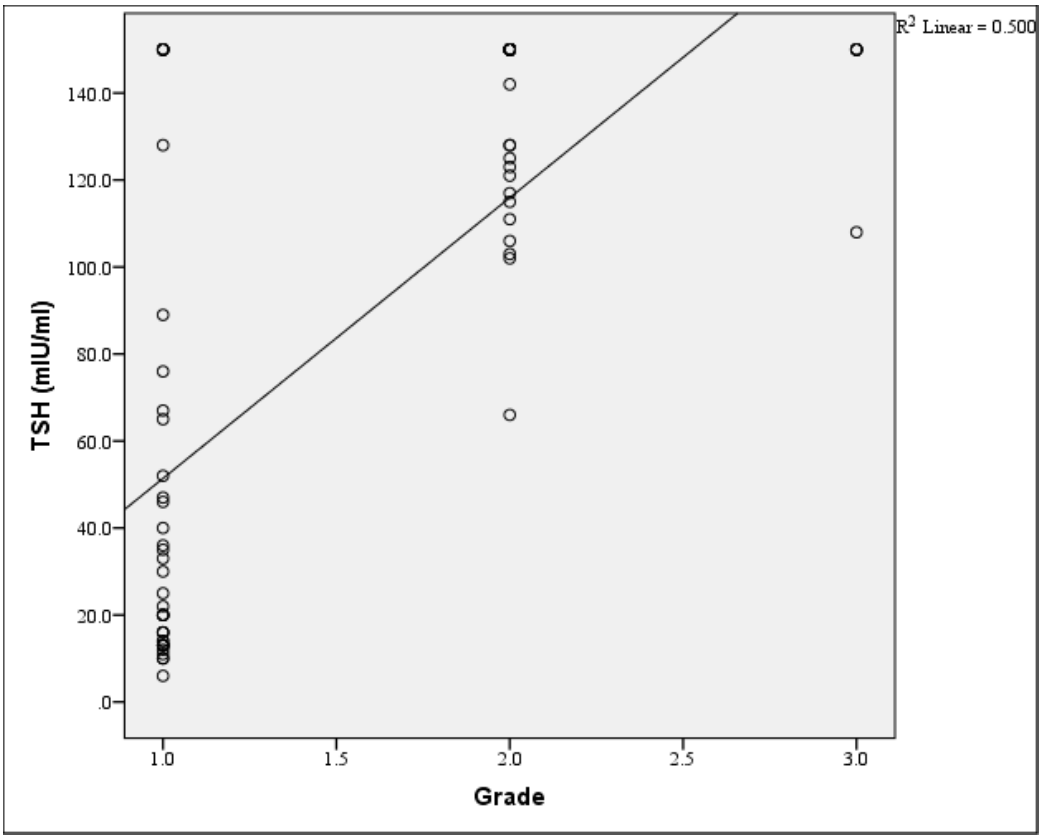

Figure 10: Figure 9: Scatter Plot showing Positive Correlation between Grade of Fatty Liver with TSH

DISCUSSION

In our study the most common age group was between 31-40 years (33.8%) with mean age being 40.28 years(S.D: ± 13.1) similar to the studies done by Parikh P et al and Kolesnikova E were the mean age was 44.3 years 47.2 years(S.D:±2.6). In our study 81.2% were females and 18.8% were males which highlight the female predominance as shown in studies done by Kassem et al with females (66.7%) and 20 males (33.3%), Kolesnikova E et al 60.8% females & 39.2% males, Bano et al 56.5% were females, pagadala et al 56.2% females. The female predominance could be explained by the fact that hypothyroidism is more common among women than in men which is documented in a study done by National Health and Nutrition Examination Survey (NHANES III) in U.S were the percentage of subjects with a high-serum TSH concentration was higher for women than for men in each decade of age, and ranged from 4 to 21% in women and 3 to 16% in men.

In our study 48.8% had Grade 1 Fatty liver, 31.2% had Grade 2 Fatty liver, 6.2% had Grade 3 fatty liver and 13.8% had no fatty liver similar to study done by Rahman F et al where majority of the subjects 52.9% patients had grade 1, 32.3% had grade 2 and 14.8% had grade 3 fatty liver. There was no statistically significant ($p>0.05$) difference with the grading of fatty liver with age. In women, the prevalence of NAFLD peaks after age 50 years. Estrogens may be partially

protective against steatosis. Due to geographical variations, racial, ethnic differences, genetic causes, different lifestyle and increased life expectancy may have significant impacts to developed fatty liver.

In the study mean BMI among those with Grade 1 Fatty live was 20.73 ± 1.51 kg/m2, among those with Grade 2 fatty liver was 21.30 ± 1.99 kg/m2 and among those with Grade 3 fatty liver was 22.32 ± 2.25 kg/m2. There was no significant difference in mean BMI between different grades of fatty was statistically significant. In the study done by Kassem et al it was observed that the mean BMI was significantly ($p<0.05$) higher in grade 3 (30.0±4.1) followed by grade 2 (29.2±4.5) and grade 1(26.9±3.6). The normal BMI values in our study may be due to the fact that most of the study subjects were women.

There are various pathways via which the beneficial effects of thyroid hormone on NAFLD risk can be mediated. Thyroid dysfunction is related to several cardiovascular risk factors that are in turn associated with an increased NAFLD risk (eg, higher BMI and dyslipidemia).Studies in rodents have demonstrated a regression of hepatic steatosis after treatment with liver-targeted thyroid hormone receptor agonists. Thyroid hormone induces intrahepatic lipolysis through lipophagy that involves the sequestration and degradation of lipid droplets within hepatic lysosomes. Thyroid hormone receptor-mediated lipophagy enhances fatty acid oxidation, which may accelerate the clearance of liver lipids and reduce hepatosteatosis. Decreased activity of hepatic lipases that occurs under hypothyroid conditions can promote NAFLD via decreased triglyceride clearance and hepatic triglyceride accumulation. In addition, the insulin resistance state associated with hypothyroidism can contribute to NAFLD by concomitantly inducing “de novo” lipogenesis and generating a flux of free fatty acids from adipose tissue to the liver. Furthermore, decreased thyroid hormones might affect circulating levels of adipocytokines, such as TNF, leptin, and adiponectin. zhang Altered adipocytokines may then contribute to hepatic inflammation and fibrosis, by exerting direct hepatotoxic effects or promoting oxygen radicals.

There was no significant difference in mean Anti TPO between different grades of fatty liver. A putative role of thyroid autoimmunity has also been suggested in NAFLD pathogenesis, because various autoantibodies such as antinuclear antibodies and antismooth muscle antibodies, have been reported in patients with NAFLD but Bano et al[64] findings do not support the hypothesis, as there was no association between TPOAb and NAFLD. The findings consistently demonstrated that low thyroid function is associated with an increased risk of developing NAFLD, as well as higher risk of having NAFLD with fibrosis. Therefore, it can be hypothesized that a hypothyroid state might accelerate the progression of liver steatosis to fibrosis. Alternatively, low thyroid function might contribute on the development of liver fibrosis, independently of steatosis.

There was no significant difference in mean HOMA-IR between different grades of fatty liver. Study by Kassem et al showed IR increased as the degree of steatosis increased parallel to

increased TSH levels and there was a relationship between TSH and IR in correlation analysis. The results of the study correlated and stated that IR, leading to impaired hepatic glucose production and glucose uptake in muscle is a component of the MS. Recent studies have revealed that subclinical hypothyroidism worsens IR. It has been reported that increasing levels of TSH and decreasing levels of FT4 are associated with increased IR.

In our study the scatter plot showed negative correlation between FT3 & fatty liver grades, FT4 and fatty liver grades, Positive correlation with TSH and fatty liver grades. Similar results were found in study done by Rahman et al where correlation between FT4 and grades of fatty liver showed significant negative correlation between FT4 and grades of fatty liver & significant positive correlation was found between TSH and grades of fatty liver. Chung et al also reported that FT4 were inversely associated with an increased prevalence of NAFLD after adjustment for risk factors. In this study it was observed that serum FT4 levels were significantly decreased with the grading of fatty liver. A cross sectional study by Zhanget al reported that NAFLD had higher serum TSH levels and lower FT4 levels, and the severity of NAFLD was negatively correlated with serum FT4 levels but positively correlated with TSH levels which is similar to this study. However FT3 was not statistically significant. A significant correlation between the serum TSH, FT4 level and metabolic syndrome parameters indirectly supported the relationship between thyroid function and NAFLD.Bano et al also showed a negative linear association between FT4 levels and incident NAFLD, even among euthyroid subjects, as well as a positive linear association for TSH levels. Moreover, the risk of NAFLD progressively decreased from a hypothyroid to a hyperthyroid state. Hypothyroidism was associated with a higher NAFLD risk compared with euthyroidism. Lower thyroid function was also associated with an increased risk of having NAFLD with fibrosis.

Study done by Kassem et al showed there was a significant positive association between hepatic steatosis (defined by the presence of a hyperechogenic ultrasound pattern of the liver and increased ALT concentrations) with TSH concentration, but inverse associations with FT4 and FT3 concentrations, thus both overt (5%) and subclinical hypothyroidism (SH) (16.7%) in the study was associated with hepatic steatosis. In another study by Pagadala MR et al a significant inverse association between the FT4 concentration of NAFLD was demonstrated but no significant association could be identified for TT3 or TSH which underscores the importance of the TT4 or FT4 concentration as a marker for hepatic steatosis in the general population. By contrast, the TT3 concentration, in studies done by Ittermann et al and Xu et al had no identified value as a marker. This finding can be related to an inhibition of the conversion of TT4 to TT3, possibly explaining the subordinate diagnostic role of the TT3 or FT3. Even the study of Chung et al which presented clear evidence of the association between hypothyroidism and NAFLD, did not ascribe any diagnostic value to the TT3 concentration.

CONCLUSION

This study found that non-alcoholic fatty liver disease is more prevalent in untreated hypothyroid subjects. The severity of non-alcoholic fatty liver disease is more in patients with overt hypothyroidism. Therefore, ultrasonography of hypothyroid patients can be helpful in the early detection of fatty liver. Early treatment with thyroxine can prevent the progression of the fatty liver and grave consequences like non-alcoholic steatohepatitis and cirrhosis.

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