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RESEARCH ARTICLE

STUDY OF METABOLIC PROFILE OF LEAN AND OBESE SUBJECTS WITH NON ALCOHOLIC FATTY LIVER DISEASE AT A TERTIARY HOSPITAL

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Abstract

Background: Non-Alcoholic Fatty Liver Disease (NAFLD) is often understood to be the hepatic expression of metabolic syndrome. In both lean & obese individuals, one common theme of NAFLD metabolism is the prevalence of Insulin Resistance (IR).

Aims & Objectives: To assess & compare the Metabolic profile of obese & lean subjects with NAFLD.

Materials & Methods: Hospital based Prospective study done in 60 Patients selected by random sampling procedure matching the inclusion criteria. A pretested pro forma was used Results: 26.7% in the obese group and 13.3% in the lean Group had Positive Family history of Diabetes and Hypertension. Mean waist circumference in obese group was 91.23 ± 7.84 and in lean group was 88.73 ±9.60. BMI in the obese group was 33.14± 2.10 and in lean group was 22.66 ±3.32. SBP in the obese group it was 132.93 ±15.06 and in lean group was 120.66 ±7.03.Triglyceride levels in the obese group it was 173.76 ±29.97 and in lean group was 147.46 ±36.35.FBS levels in the obese group it was 110.23 ±11.36 and in lean group was 85.43 ± 9.00

Conclusion: The Present Study suggesting obese and lean individuals who had comorbidities , have more metabolic dysregulation than obese and lean individuals without having comorbidities.

Introduction:-

Currently, NAFLD affects about 25% of the worldwide population with estimates ranging from 13% to 32%. NAFLD can be described as the presence of excess adipose tissue accumulation in the liver, also known as hepatic steatosis, that is not caused by excessive alcohol intake. More specifically, non-alcoholic fatty liver disease is defined as at least 5% hepatic steatosis without hepatocellular injury NAFLD represents a spectrum of diseases ranging from simple Steatosis to NASH to Cirrhosis and Hepatocellular carcinoma

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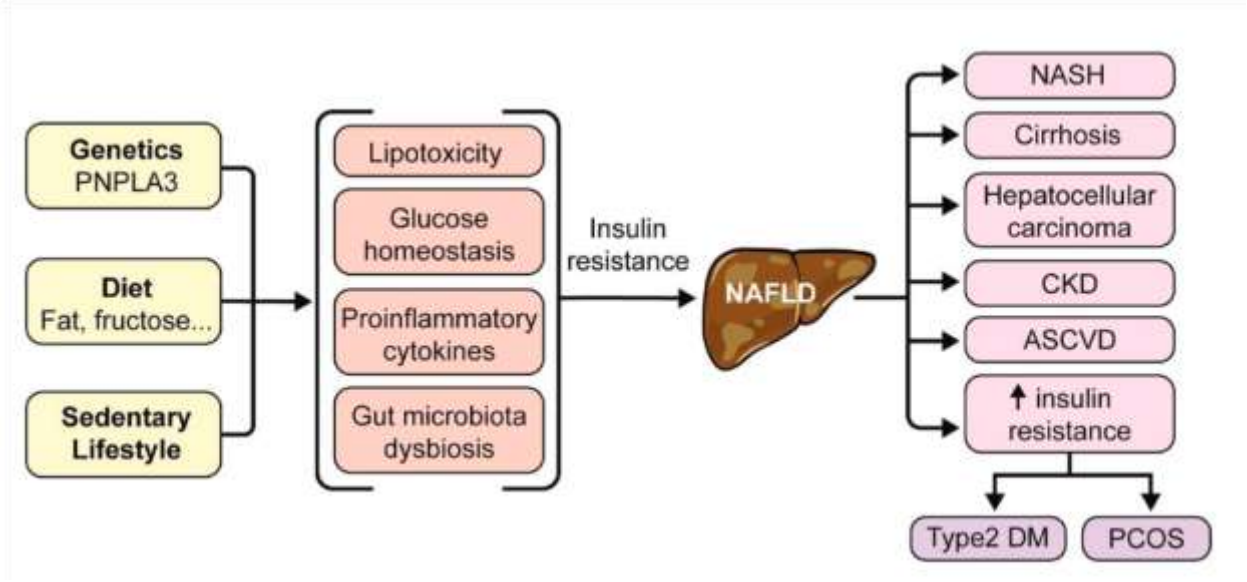
However, the traditional ”two-hit” hypothesis of NAFLD pathogenesis has been replaced by the ”multiple-hit” hypothesis in order to explain the several molecular and metabolic changes of NAFLD. The metabolic syndrome is a constellation of cardio-metabolic risk factors including increased visceral adiposity and an increase in IR causing impaired glucose tolerance and T2DM, dyslipidemia, and hypertension

Despite being closely linked with Obesity, NAFLD can also manifest itself in non-obese individuals. In fact, about 10–20% of non-obese Americans may present with NAFLD.9 ”Lean” NAFLD is most commonly seen in Asian individuals in whom the majority of ”lean” NAFLD studies have been carried out. Between 7 and 18% of the non-obese population in Asia (including China, Korea, and Japan) may have NAFLD.

The metabolic development of ”lean” NAFLD can be most concisely explained by the idea that increased lipolysis overwhelms the body's ability to store lipids subcutaneously and this leads to free fatty acid accumulation in visceral areas of the body, including in the liver. This errant lipid metabolism drives IR and inflammation leading to NAFLD progression and is similar to NAFLD progression in more classic obesity-driven and T2DM-driven pathogenesis.



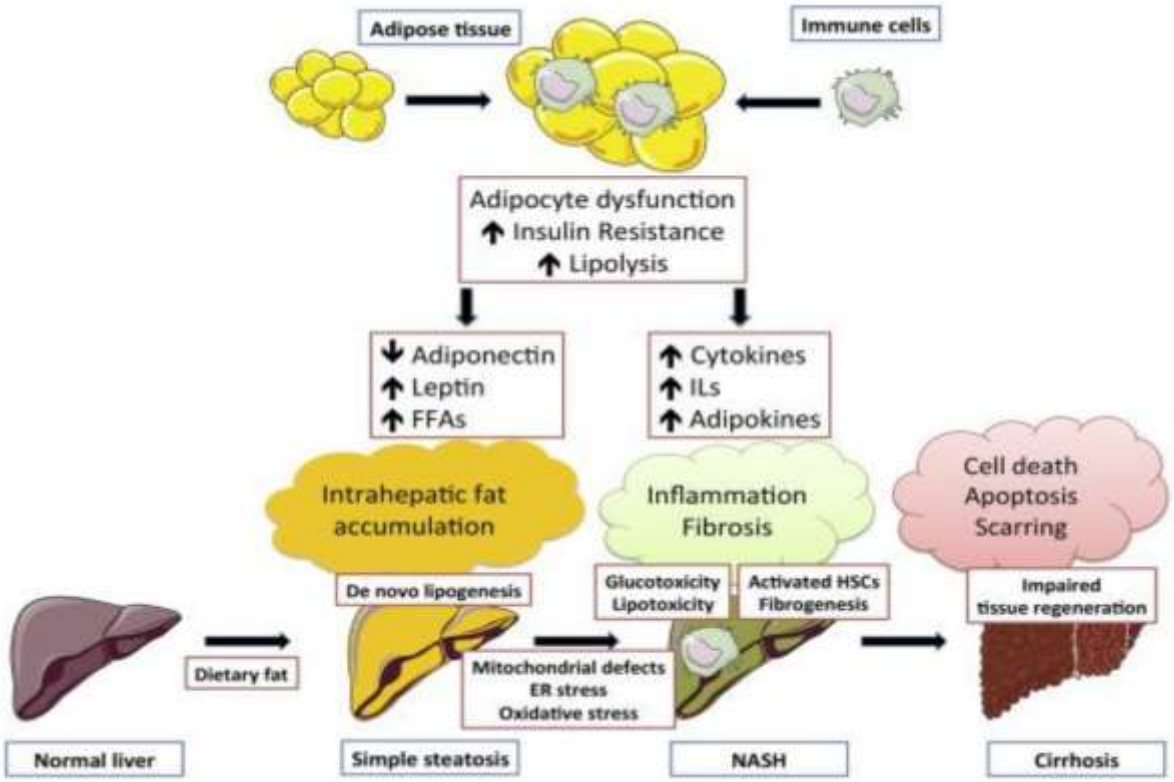
The consequences of NAFLD are mainly driven by the severity of IR as NAFLD progresses toward NASH and HCC. The worsening hepatic pathology drives further increases in IR, creating a vicious cycle of NAFLD progression.

The abnormal glucose and lipid metabolism in patients with NASH further exacerbates NAFLD progression and thus targeting glucose metabolism and IR through the use of GLP-1 receptor agonists has been shown to reduce NASH severity.GLP-1 receptor agonists can rescue this impaired metabolic phenotype by improving lipid transport and increasing the liver's ability to metabolise fatty acids. Most importantly, GLP-1 receptor agonists reduce IR. In fact, One study showed that 39% of Patients with NASH who were treated with GLP-1 receptor agonists exhibited reversal in NASH hepatic morphology.

Causes of NAFLD in lean subjects

General/nutritional	Metabolic	Toxicity of drugs
Acute systemic illness	Cystic fibrosis	
Starvation	Wilsons -disease	Steroids
Hepatitis C	α1 antitrypsin deficiency	Ethyl alcohol
complete parenteral nutrition	Galacto -semia	l -asparaginase,
		Methorexate
Ulcerative colitis & crohns disease	Fructosemia	Vitamin A
Celiac disease	Wolman- disease	Valproate
Maur-iac syndrome	Glycogen storage disease	Tamoxifen
	Mitochondrial defects of fatty acid oxidation	Anti - retroviral theraphy
	Lipodystrophies	
	Abeta -lipoprotein -aemia	
	WeberChristian disease	

Pathogenesis of NAFLD in Obese



Aims and Objectives:-

To compare the Metabolic profile of lean & obese subjects with non alcoholic fatty liver disease. 1.To assess the Metabolic profile of lean & obese subjects with non alcoholic fatty liver disease.
2.To compare the Metabolic profile of lean & obese subjects with non alcoholic fatty liver disease.
3.To study subclinical end organ dysfunction.

Methods & Materials:-

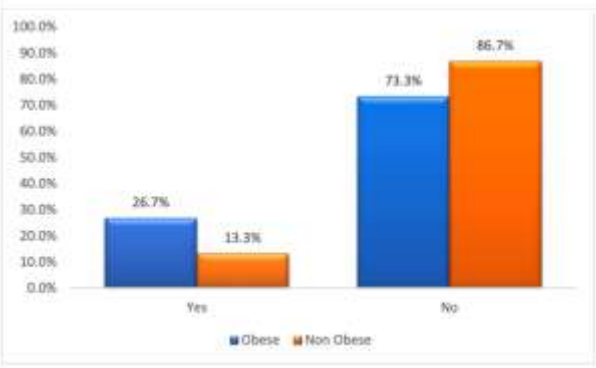
Prospective study done in 60 patients in period of 18 months attending OP/ Admitted in Dr. PSIMS & RF Inclusion Criteria:
Patients with NAFLD are included According to WHO: 1.Subjects with Body Mass Index [BMI] >= 25 Kg/m2 will be taken as obese. 2. Subjects with Body Mass Index [BMI] < 18.5 Kg/m2 will be taken as lean.

Exclusion Criteria:
1. Patients with alcohol intake are excluded. 2.Patients with Chronic liver disease due to other aetiologies are excluded. 3 .Pregnant women are excluded.

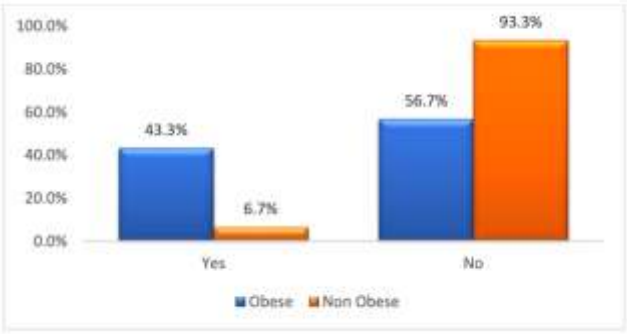
Data entry was done using Microsoft excel 2013 and analysis done using SPSS V 16. Qualitative Data was expressed in frequencies and percentages and Quantitative data in mean and standard deviation. Parametric tests include t - test for Intergroup Comparison was used. . Bar diagrams and pie chart were used to represent the data. p value of <0.05 was considered statistically significant.

Results:-

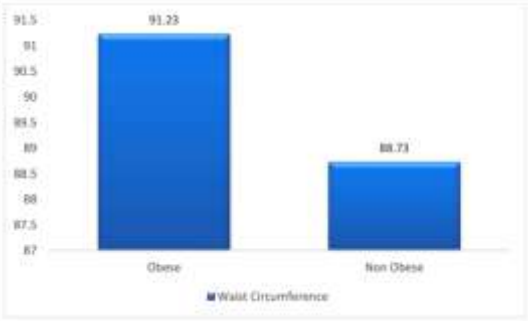
- The mean Age of the obese group in years was 38.06 ±8.66 & in the was 37.93 ±8.91 In the obese group, 20% were Male & 80% were female. In the lean group, 23.3% were Male & 76.7% were female.

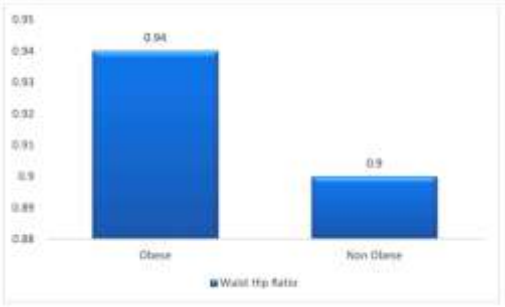


- 26.7% in the obese group and 13.3% in the lean Group had Positive Family history of Diabetes and Hypertension .

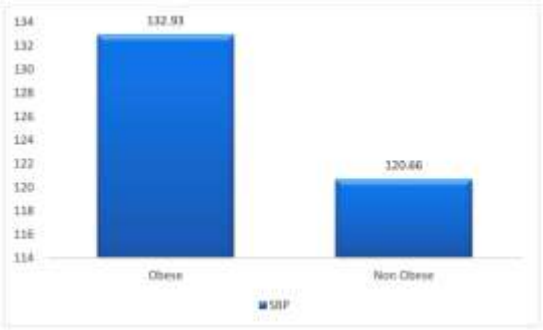


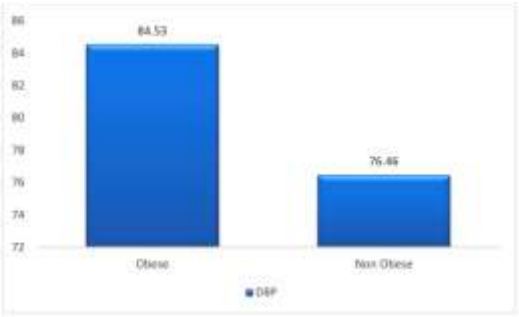
- 43.3% in the obese group and 6.7% in the lean Group had Positive Family history of obesity
- Mean waist circumference in obese group was 91.23 ±7.84 ad in lean group was 88.73 ±9.60. Mean waist hip ratio in the obese group was 0.94 ±0.004 and in lean group was 0.90 ±0.06.BMI in the obese group was 33.14 ±2.10 and in lean group was 22.66 ±3.32.



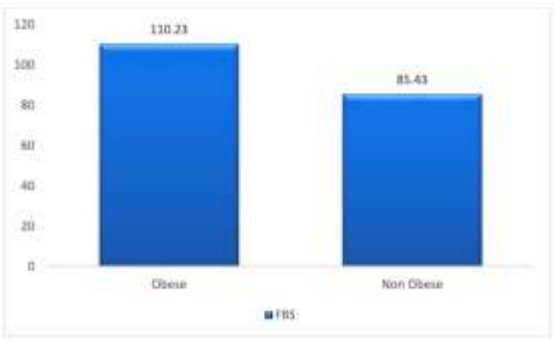


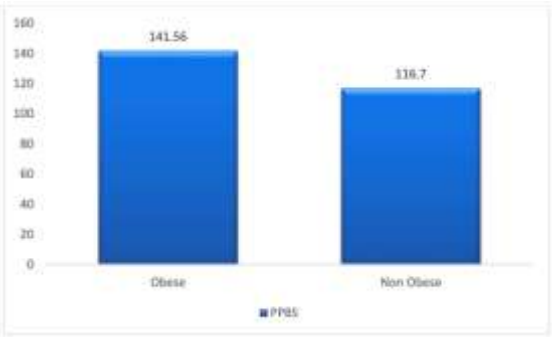
- SBP in the obese group it was 132.93 ±15.06 and in lean group was 120.66 ±7.03.DBP in the obese group it was 84.53 ±8.62 and in non lean group was 76.46 ±4.53





- Triglyceride levels in the obese group it was 173.76 ±29.97 and in lean group was 147.46 ±36.35, HDL levels in the obese group it was 37.70 ±4.10 and in lean group was 43.43 ±4.58. VLDL levels in the obese group it was 34.75 ±5.99 and in lean group was 33.44 ±5.53. LDL levels in the obese group it was 126.06 ±34.89 and in lean group was 98.30 ±36.12. Total cholesterol levels in the obese group it was 196.01 ±37.31 and in lean group was 168.01 ±38.36.
- FBS levels in the obese group it was 110.23 ±11.36 and in lean group was 85.43 ±9.00.PPBS in the obese group it was 141.56 ±11.95 and in lean group was 116.70 ±14.01.





Discussion:-

The pathogenesis of non alcoholic steato-hepatitis (NASH) is complex, involves numerous components, and is not currently fully understood.Some of the processes that lead to this syndrome include dietary factors, glucose intolerance, genetic variations, dyslipidaemia, and a disturbed gut flora.

The diagnostic criteria for Non-alcoholic fatty liver disease is the presence of at least 5 % steato-hepatitis without signs of hepatocellular damage, such as hepatocyte ballooningNASH, which is composed mainly of 5percent hepatosteatosis and inflammation with hepatocellular damage, with or without fibrosis, develops in some people. Some people advance to NASH, whereas the majority of individuals seem to stay in the benign NAFLD stage. Fibrosis is a defining feature of this stage.

The present study was conducted to Evaluate the risk factors and Metabolic profile of lean & obese individuals with NAFLD, that would help in prevention and treatment of effected individuals. 20% in the present study had +ve family history Diabetes and Hypertension. 26.7% in the obese group and 13.3% in the lean group had +ve family history of Diabetes and Hypertension . 25% in the present study has +ve history of obesity. 43.3% in the obese group and 6.7% in the lean group had +ve Family history of obesity. There were no statistically discernible differences found with relation to family history of obesity between the groups as p- value calculated to be >0.05. Anderson et al in their Systematic review of 74 observational studies evaluating rate of Non- alcoholic fatty liver disease (NAFLD) in children.

Diagnostic tests for NAFLD included ultrasound scan (51 studies), increased ALT levels (27 studies), and/or MRI (9 studies) .

Mean prevalence of Non- alcoholic fatty liver disease (NAFLD) In general population studies: Overall 7.6% (95% CI 5.5%-10.3%) in analysis of 20 studies.

Chang et al in their cohort study on 4,246 nondiabetic men followed for 16,830 person-years. Weight gain ≥ 2.3 kg (5.07 lbs) Associated with increased risk for ultrasound detected NAFLD (adjusted hazard ratio 1.26, 95% CI 1.01-1.58).Based on systematic review46 of 22 randomized trials evaluating weight-loss interventions and reporting on biomarkers of liver disease in 2,588 patients with NAFLD. 16 trials enrolled patients with any stage of NAFLD and 6 trials enrolled only Patients with nonalcoholic steatohepatitis (NASH) mean patient age 45 years, 66% men, and mean Body Mass Index 33.7 kg/m2.median trial duration 6 months (range 1-24 months)
In the Present Study, distribution based on SBP where in the obese group it was 132.93 ±15.06 and in lean group was 120.66 ±7.03. This observation is Statistically Significant (p<0.0001*) Distribution based on DBP where in the obese group it was 84.53 ±8.62 and in lean group was 76.46 ±4.53. This observation is Statistically Significant (p<0.0001*)

Distribution based on Triglyceride levels, HDL & LDL in the obese group & lean group was Statistically Significant ($p<0.0001$*). Distribution based on FBS & PPBS in the obese group & lean group was Statistically Significant ($p<0.0001$*)

Conclusion:-

The growing number of young people affected with obesity , lean are leading to an increased occurrence of hepatic steatosis and its correlated comorbidities with an alarming worldwide prevalence. It consists of a disease with a multifactorial etiopathogenesis. In fact, although environmental factors are widely proven to be associated with hepatic steatosis, it is getting stronger the evidence of a genetic background influencing the Onset and Progression of disease.

The Present Study suggesting obese and lean individuals who had comorbidities , have more metabolic dysregulation than obese and lean individuals without having comorbidities . Among the obese a significant derangement in the lipid profile observed in the study compared to lean group.

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