

# North Asian International Research Journal Consortium

*North Asian International Research Journal*

*Of*

*Multidisciplinary*

Chief Editor

Dr. Nisar Hussain Malik



Publisher

Dr. Bilal Ahmad Malik

Associate Editor

Dr. Nagendra Mani Trapathi

Honorary

Dr. Ashak Hussain Malik

NAIRJC JOURNAL PUBLICATION

North Asian  
International  
Research Journal Consortium



## Welcome to NAIRJC

ISSN NO: 2454 - 2326

North Asian International Research Journal is a multidisciplinary research journal, published monthly in English, Hindi, Urdu all research papers submitted to the journal will be double-blind peer reviewed referred by members of the editorial board. Readers will include investigator in Universities, Research Institutes Government and Industry with research interest in the general subjects

### Editorial Board

J.Anil Kumar Head Geography University of Thirvanathpuram	Sanjuket Das Head Economics Samplpur University	Adgaonkar Ganesh Dept. of Commerce, B.S.A.U Aruganbad
Kiran Mishra Dept. of English,Ranchi University, Jharkhand	Somanath Reddy Dept. of Social Work, Gulbarga University.	Rajpal Choudhary Dept. Govt. Engg. College Bikaner Rajasthan
R.D. Sharma Head Commerce & Management Jammu University	R.P. Pandday Head Education Dr. C.V.Raman University	Moinuddin Khan Dept. of Botany SinghaniyaUniversity Rajasthan.
Manish Mishra Dept. of Engg, United College Ald.UPTU Lucknow	K.M Bhandarkar Praful Patel College of Education, Gondia	Ravi Kumar Pandey Director, H.I.M.T, Allahabad
Tihar Pandit Dept. of Environmental Science, University of Kashmir.	Simnani Dept. of Political Science, Govt. Degree College Pulwama, University of Kashmir.	Ashok D. Wagh Head PG. Dept. of Accountancy, B.N.N.College, Bhiwandi, Thane, Maharashtra.
Neelam Yaday Head Exam. Mat.K..M .Patel College Thakurli (E), Thane, Maharashtra	Nisar Hussain Dept. of Medicine A.I. Medical College (U.P) Kanpur University	M.C.P. Singh Head Information Technology Dr C.V. Rama University
Ashak Hussain Head Pol-Science G.B, PG College Ald. Kanpur University	Khagendra Nath Sethi Head Dept. of History Sambalpur University.	Rama Singh Dept. of Political Science A.K.D College, Ald.University of Allahabad

**Address: - Ashak Hussain Malik House No. 221 Gangoo, Pulwama, Jammu and Kashmir, India - 192301, Cell: 09086405302, 09906662570, Ph. No: 01933-212815,**

**Email: [nairjc5@gmail.com](mailto:nairjc5@gmail.com), [info@nairjc.com](mailto:info@nairjc.com) Website: [www.nairjc.com](http://www.nairjc.com)**

## FATTY LIVER AFFECTED BY TWO DIFFERENT FOOD HABIT VEG. AND MIXED IN NON ALCOHOLIC TYPE 2 DIABETIC PATIENTS



Mr. Saini Krishan<sup>1</sup>, Mr. Sharma Pravesh Kumar<sup>2</sup>, Dr. Kumawat Radhey Shyam<sup>3</sup>

1. M-pharma (Pharmacology), Student of Maharishi Arvind Institute of Pharmacy, Mansarovar, Jaipur – 302020
2. Assistant Professor of Maharishi Arvind Institute of Pharmacy, Mansarovar, Jaipur – 302020
3. Head of Department of Maharishi Arvind Institute of Pharmacy, Mansarovar, Jaipur – 302020

### ABSTRACT

**Object** – Fatty liver affected by two different food habit veg. And mixed In non alcoholic type 2 diabetic patients.

**METHODS-** The study included 100 patients of type 2 diabetes mellitus. Patients who presented to the GLAXY SPECILITY CENTER, Jaipur form November 2014 to June 2015 were enrolled into the study. Clinical data such as age, sex, height, weight, body-mass index (BMI) and blood pressure (BP) were recorded. Serum total cholesterol, triglycerides, high-density lipoprotein (HDL) cholesterol, uric acid and liver enzyme levels (SGOT OR SGPT) were determined by enzymatic methods with a chemistry analyzer. Hba1c level measured by chromatography based HPLC assay. Gradding of NAFLD was measured by ultrasonography. Differences between mean values in the NAFLD were analyzed by unpairedt-test. Multiple logistic regression analysis using NAFLD as a dependent variable was conducted to determine the relative contributions made by each variable to the outcome variable. Significant independent variables were chosen using the stepwise variable selection method. A value of P,0.05 was

used to indicate statistical significance. Data were analyzed by one sample t-test, Shapiro- Wilk normality test by PRISM Version 5.01.

**Results-** The study included 100 patients of type 2 diabetes mellitus. Eighty (80%) patients were veg. and twenty (20%) were mixed (veg/nonveg). Among 80 patient observed Veg. patients 35% were found with Grade -0, 55% with Grade-1, 8.75% with Grade-2 and 1.25% with Grade 3 NAFL. Similarly among 20 patient observed with NAFL, 35% were found with Grade -0, 45% with grade -1, 15% with grade -2, 5% with grade-3 NAFL. Group-1 (NAFLD+VEG., n=80) were compared with a Group-2 (NAFLD+ MIXED (VEG/NON VEG., n= 20). Pre established null hypothesis for diastolic blood pressure, HbA1c, TC, TG, LDL, VLDL, TC and TG could be rejected which showed there was no significance difference while the alternative hypothesis could be accepted at 0.05% significant level by Statistical Analysis.

**Conclusions-** we concluded that progression of fatty liver grading and presence of higher graded fatty liver is more prone in patients with non vegetarian food habit in compare to patients with vegetarian food habit. Incidence of Liver abnormalities with increased Levels Of Liver Enzymes in blood were found higher in non vegiterian patients in compare to vegiterian patents. Deviated HbA1c level from its standard reference value was more comman in nonvegiterian patients.

**Keywords:** NAFLD, NASH, Metabolic disorders, Ultrasound Sonography, TC, HDL, LDL, VLDL, TG, SGOT, SGPT, BMI, HbA1c.,S. Billirubin Total, S. Billirubin Indirect, S. Billirubin Direct

## INTRODUCTION

Non-alcoholic fatty liver disease (NAFLD) is the most common liver disease and the third leading indication for liver transplantation [1]. The prevalence of NAFLD has been reported to be 15-30% in the general population [1-2] and in type-2 diabetes mellitus population, the prevalence is 70-75% [3]. NAFLD has been proposed as one of the components of metabolic syndrome [4]. It has been found to be a composite of confirmed cases with central obesity, type -2 diabetes mellitus.[5,6] Non-alcoholic fatty liver disease (NAFLD) is the build up of extra fat in liver cells that is not caused by alcohol.[7] It is normal for the liver to contain some fat.[6,7] However, if more than 5% - 10% percent of the liver's weight is fat, then it is called a fatty liver (steatosis).[7,8,9] NAFLD tends to develop in people who are overweight or obese or have diabetes, high cholesterol or high

triglycerides.[8,10] Rapid weight loss and poor eating habits also may lead to NAFLD.[9] The common Symptoms are fatigue, weakness, weight loss, loss of appetite, nausea, abdominal pain, spider-like blood vessels, yellowing of

the skin and eyes (jaundice), itching, fluid buildup and swelling of the legs (Edema) and abdomen (ascites), and mental confusion.[11,12,13,14,15]

The Diagnosis Of NAFLD Is Initially Suspected If Blood Tests Show High Levels Of Liver Enzymes.[12,14,16] An Ultrasound Is Used To Confirm The NAFLD Diagnosis.[16,17] Prevention Of Nafld Include Maintain A Healthy Weight,[14] Eat A Healthy Diet, Exercise Regularly, Limit Alcohol Intake, [18]Only take medicines that you need and follow dosing recommendations.[19,20] NAFLD is closely associated with obesity.[19,21] Obesity results from greater energy intake than consumption, with excessive energy accumulated as fat.[22]

## **METHODS**

This was a prospective study of histologic severity in diabetic patients with NAFLD. The study included 100 patients of type 2 diabetes mellitus. The patient age was between 30 years to 70 years. Patients who presented to the GLAXY SPECILITY CENTER, Jaipur from November 2014 to June 2015 were enrolled into the study.

## **STUDY DESIGN**

Clinical data such as age, sex, height, weight, body-mass index (BMI) and blood pressure (BP) were recorded. The systolic and diastolic pressures were measured on the arm of the seated subject, who had rested in a sitting position for 5 min before the measurement.[23,24] Waist and hip ratio (cm/cm) was determined by measurement of the circumference of the waist and hips in a standing position. [25]Waist circumference was measured to the nearest 0.1 cm at the level of the iliac crest by a tape while the subject was at minimal respiration. Hip circumference was measured at the level of the anterior superior iliac spine. [26, 27]

Serum total cholesterol, triglycerides, high-density lipoprotein (HDL) cholesterol, uric acid and liver enzyme levels (SGOT OR SGPT) were determined by enzymatic methods with a chemistry analyzer. [28, 29, 24] Hba1c level measured by chromatography based HPLC assay. Gradding of NAFLD was measured by ultra sonography.[26,30] A single experienced radiologist blinded to the laboratory data performed ultrasonography liver examinations. Fatty liver was defined as a bright liver on ultrasonography. [17, 21, 26]

## **STATISTICAL ANALYSIS**

Differences between mean values in the NAFLD were analyzed by unpairedt-test. Multiple logistic regression analysis using NAFLD as a dependent variable was conducted to determine the relative contributions made by each variable to the outcome variable. Age, weight, Height, BMI, waist/hip ratio, Systolic, Diastolic Blood presser,

Hba1c, SGOT, SGPT, S. Billirubin total, S. Billirubin Direct, S. Billirubin Indirect, TC, HDL, TG etc. were employed as independent variables.[7,15,25] Significant independent variables were chosen using the stepwise variable selection method.[23] A value of P, 0.05 was used to indicate statistical significance. Data were analyzed by one sample t-test, Shapiro- Wilk normality test by PRISM Version 5.01.

**RESULTS**

**I. Observational study:** The study included 100 patients of type 2 diabetes mellitus. Eighty (80%) patients were veg. and twenty (20%) were mixed (veg. /non-veg.). Among 80 patients observed Veg. patients 35% were found with Grade -0, 55% with Grade-1, 8.75% with Grade-2 and 1.25% with Grade 3 NAFL. Similarly among 20 patient observed with NAFL, 35% were found with Grade -0, 45% with grade -1, 15% with grade -2, 5% with grade-3 NAFL. Group-1 (NAFLD+VEG., n=80) were compared with a Group-2 (NAFLD+ MIXED (VEG/NON VEG., n= 20). Pre established null hypothesis for diastolic blood pressure, HbA1c, TC, TG, LDL, VLDL, TC and TG could be rejected which showed there was no significance difference while the alternative hypothesis could be accepted at 0.05% significant level by Statistical Analysis.

**(1) Percentage of Veg. And Mixed (Veg. / Non Veg.) Patient Involved In Study.**

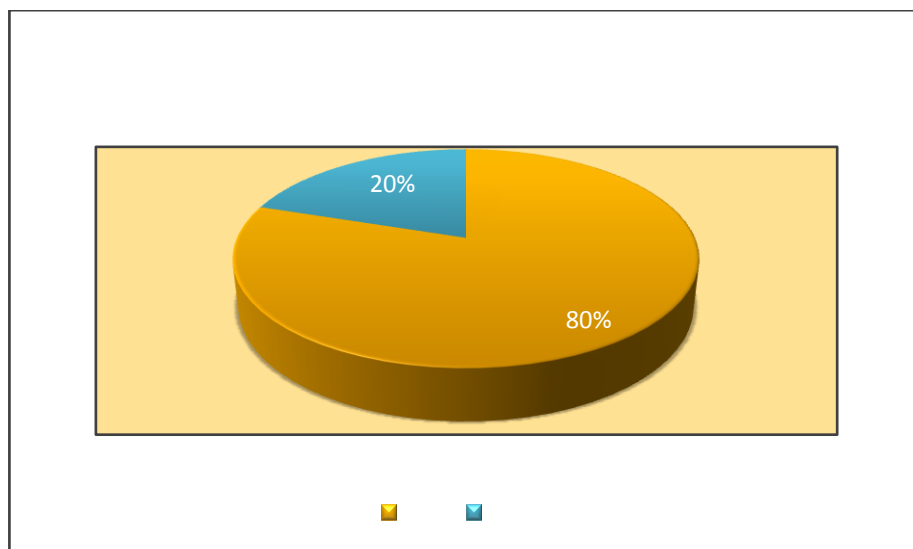


Fig1 - Distribution of veg. and non veg. patient.

**(2) Frequency and percentage of different type of fatty liver grading observed in veg. patient.**

Among 80 patient observed **Veg. patients** 35% were found with **Grade -0**, 55% with **Grade-1**, 8.75% with **Grade-2** and 1.25% with **Grade 3 NAFL**.

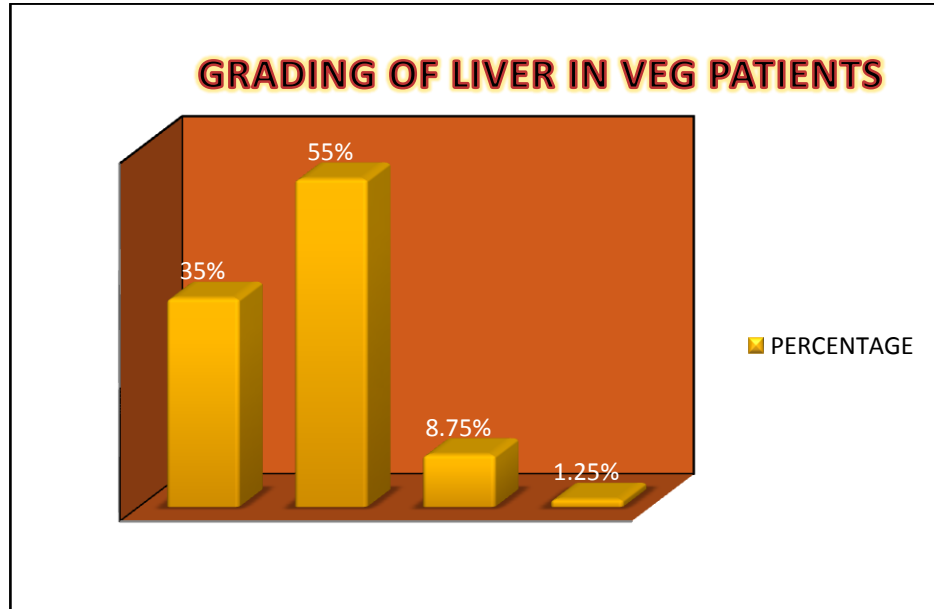


Fig 2 - Percentage wise distribution of fatty liver grading present in veg. Patient.

**(3) Frequency and percentage of different type of fatty liver grading observed in mixed(veg./non veg.) patient.**

Similarly among 20 patient observed with NAFL, 35% were found with **Grade -0**, 45% with **grade -1**, 15% with **grade -2**, 5% with **grade-3 NAFL**.

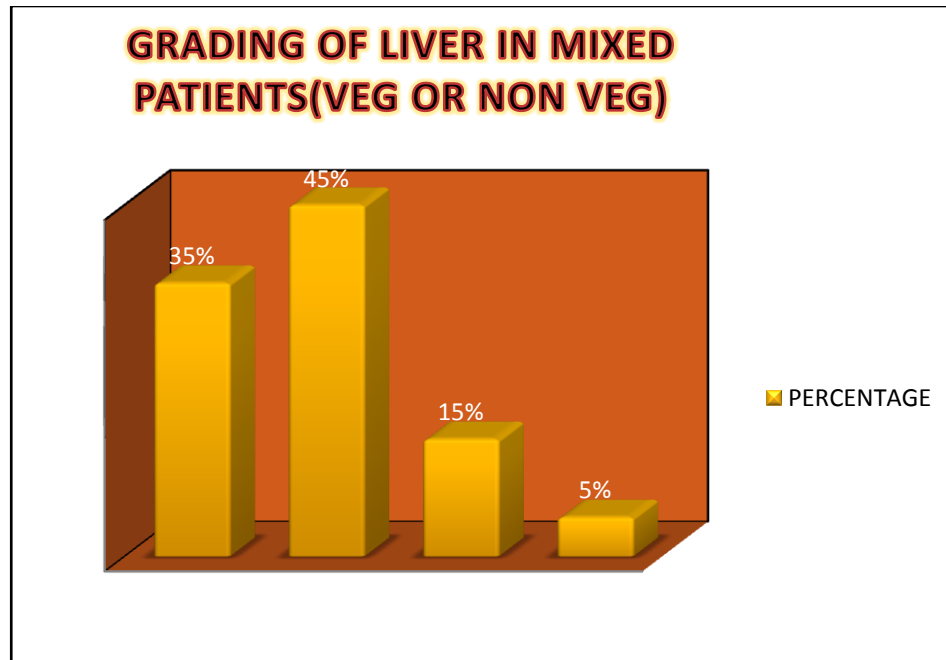


Fig 3 - Percentage wise distribution of fatty liver grading present in mixed (veg. / non.veg.) patient.

## II. STATISTICAL ANALYSIS

Group-1 (NAFLD+VEG., n=80) were compared with a Group-2 (NAFLD+ MIXED (VEG/NON VEG., n= 20). The mean vale of BMI, S.BILIRUBIN DIRECT, DIASTOLIC B.P. and PULSE of group-1 is slightly lower than group- 2 while the mean value of WEIGHT, HEIGHT, SYTOLIC B.P., DURATION OF DIABETES, Hb, HbA1c, SGOT, SGPT, S. BILLIRUBIN TOTAL, S. BILLIRUBIN INDIRECT, TOTAL CHOLESTEROL, LDL, VLDL, TG and HDL found slightly higher in group 2.

According to analysed data of calculated p-value-

- 1) Pre established null hypothesis for diastolic blood pressure, HbA1c, TC, TG, LDL, VLDL, TC and TG could be rejected which showed there was no significance difference while the alternative hypothesis could be accepted at 0.05% significant level.
- 2) Pre established null hypothesis for SYSTOLIC BLOOD PRESSURE, PULSE RATE, S. BILLIRUBIN DIRECT, S. BILLIRUBIN INDIRECT, S. BILLIRUBIN TOTAL Hb, SGPT and SGOT could be rejected which showed there was no significance difference while the alternative hypothesis could be accepted at 0.05% significant level i.e. true signify.



VARIABLES	GROUP-1(N=80)	GROUP-2(N=20)	P VALUE
Age	55.73+1.16	51.78+2.10	0.7953ns
Weight	72.59+2.03	77.31+4.11	0.4774ns
Height	162.8+2.04	165.2+1.08	0.0781ns
BMI	20.20+0.05	26.73+0.80	0.0133ns
Systolic	129.8+1.73	133.6+1.64	0.0233**
Diastolic	80.10+0.90	83+1.87	< 0.0001***
Pulse	81.77+0.60	80.60+0.45	0.0033**
Duration of diabetes	3.26+0.85	5.80+1.55	0.0351*
Hb	11.67+0.87	13.69+0.14	0.0451*
Hba1c	7.38+0.40	9.49+0.24	<0.0001***
SGOT	20.54+0.86	25.60+1.92	0.0368*
SGPT	21.33+1.20	33.60+2.25	0.0303*
S. Billirubin total	0.63+0.04	0.75+0.02	0.0148*
S. Billirubin Direct	0.35+0.23	0.38+0.41	0.0205*
S. Billirubin Indirect	0.31+0.72	0.42+0.19	0.0028**
TC	159.1+10.42	178+6.38	0.0102*
HDL	38.73+1.41	44.78+1.32	0.3612ns
LDL	42.20+0.63	53.34+0.93	<0.0001**
VLDL	32.41+1.03	37.20+1.23	<0.0001**
TG	115.52+10.57	157.4+9.09	0.0043**

**Table 1: Demographic, Hemodynamic and Biochemical details of the study groups.**

- 3) Pre established null hypothesis for AGE, WEIGHT, HEIGHT, BMI, and HDL could not be rejected which showed there was no significance difference while the alternative hypothesis could not be accepted at 0.05% significant level i.e. false significance.

## DISCUSSIONS

NAFLD is more commonly seen in type 2 diabetic patients. The important finding of this study was that NAFLD, as diagnosed by liver ultrasound, which is the most widely used imaging test for detecting hepatic steatosis. [2,17] The average age of the patients was 53.84 years (Ranging from 30 to 70). In our study 65% Patients was found with fatty liver and 35% was without fatty liver in which common factor was presence of diabetes. Patients with fatty liver further categorized in group according to their lipid profile. Grading of NAFL was observed in VEG. and MIX.(VEG./NONVEG.) participants- Among 80 patient observed Veg. patients 35% were found with Grade -0, 55% with Grade-1, 8.75% with Grade-2 and 1.25% with Grade 3 NAFL. Similarly among 20 patient observed with NAFL, 35% were found with Grade -0, 45% with grade -1, 15% with grade -2, 5% with grade-3 NAFL.

This study compares the dietary intake, physical activity, and liver histology in patients with and metabolic syndrome. [24] We found that NAFLD patients with metabolic syndrome consumed more carbohydrates, less fat, and equal amounts of protein and total calories, compared. Physical activity did not differ between the two groups. When comparing histologic severity of NAFLD, patients with metabolic syndrome had higher scores for steatosis grade, NASH activity, and global NASH severity. Current recommendations are to lose weight through diet and exercise with no robust evidence to support this recommendation. Individuals who are physically active and consume a low carbohydrate diet have a lower risk of having metabolic syndrome.[25,28] In addition, obese patients have improved IR, abdominal obesity, and lower odds for hepatic inflammation with a lower carbohydrate diet.[22,29] Although a low-fat diet has generally been recommended in patients with NAFLD, recent studies show that a greater short-term weight loss and improvement in markers of the metabolic syndrome is associated with a low-carbohydrate diet without significant adverse effects. The role of dietary composition with a decrease in carbohydrate relative to the total calories consumed, as well as increased physical activity, in NAFLD histology, however, needs to be further studied.[21,14,30] Patients with a higher global NASH score were more likely to be obese. Patients who have greater splanchnic (visceral) fat distribution may be particularly prone to the effects of IR and possibly to a more aggressive form of NASH.[27]

Pre Established Null Hypothesis For Diastolic Blood Pressure, Hba1c, Tc, Tg, Ldl, Vldl, Tc And Tg Could Be Rejected Which Showed There Was No Significance Difference While The Alternative Hypothesis Could Be Accepted At 0.05% Significant Level. Pre Established Null Hypothesis For Systolic Blood Pressure, Pulse Rate, S. Billirubin Direct, S. Billirubin Indirect, S. Billirubin Total Hb, SGPT And SGOT Could Be Rejected Which Showed There Was No Significance Difference While The Alternative Hypothesis Could Be Accepted At 0.05% Significant Level I.E. True Significance. Pre Established Null Hypothesis For Age, Weight, Height, BMI, And HDL Could Not Be Rejected Which Showed There Was No Significance Difference While The Alternative Hypothesis Could Not Be Accepted At 0.05% Significant Level I.E. False Significance.

## CONCLUSIONS

We concluded that progression of fatty liver grading and presence of higher graded fatty liver is more prone in patients with non vegetarian food habit in compare to patients with vegetarian food habit. When comparing histologic severity of NAFLD, patients with metabolic syndrome with non veg. food habit had higher scores for steatosis grade, NASH activity, and global NASH severity. Incidence of Liver abnormalities with increased Levels of Liver Enzymes in blood was found higher in non vegetarian patients in compare to vegetarian patents. Deviated HbA1c level from its standard reference value was more common in non-vegetarian patients.

## REFERENCES

1. Clark JM, Brancati FL, Diehl AM. The prevalence and an etiology of elevated aminotransferase levels in the United States. *Am J Gastroenterol* 2003; 98:960-7.
2. Bedogni G, Miglioli L, Masutti F, Tiribelli C, Marchesini G, Bellentani S. Prevalence of and risk factors for nonalcoholic fatty liver disease: the Dionysos Nutrition and Liver Study. *Hepatology* 2005; 42:44-52.
3. Medina J, Fernández-Salazar LI, García-Buey L, Moreno-Otero R. Approach to the pathogenesis and treatment of nonalcoholic steatohepatitis. *Diabetes Care* 2004; 27:2057-66.
4. Kotronen A, Yki-Järvinen H. Fatty liver: a novel component of the metabolic syndrome. *Arterioscler Thromb Vasc Biol* 2008; 28:27-38.
5. Angulo P., "Nonalcoholic fatty liver disease", *N Engl J Med* 2002, 346, 1221–31.
6. Clark JM, Brancati FL and Diehl AM., "Nonalcoholic fatty liver disease". *Gastroenterology* 2002, 122, 1649–57.
7. Dowman JK, Tomlinson JW and Newsome PN., "Systematic review: the diagnosis and staging of non-alcoholic steatohepatitis." *Aliment Pharmacol Ther*, 2011, 33, 525–40.
8. Fabbrini E, Sullivan S and Klein S. "Obesity and nonalcoholic fatty liver disease: biochemical, metabolic, and clinical implications." *Hepatology*, 2010, 51, 679–89.
9. Rafiq N and Younossi ZM., "Nonalcoholic fatty liver disease: a practical approach to evaluation and management." *Clin Liver Dis*, 2009, 13, 249–66.

10. Ratziu V et, al., “A position statement on NAFLD/NASH based on the EASL 2009 special conference.” *J Hepatol*, 2010, 53, 372–84.
11. Torres DM and Harrison SA., “Diagnosis and therapy of nonalcoholic steatohepatitis. *Gastroenterology*”, 2008, 134, 1682–98.
12. Vernon G., Baranova A .and Younossi ZM., “Systematic review: the epidemiology and natural history of non-alcoholic fatty liver disease and non-alcoholic steatohepatitis in adults”, *Aliment Pharmacol Ther* ,2011, 24, 274–85.
13. Vuppalachi R and Chalasani N.” Nonalcoholic fatty liver disease and nonalcoholic steatohepatitis: selected practical issues in their evaluation and management.” *Hepatology*, 2009, 49, 306–17.
14. Younossi ZM, et. al, “Changes in the prevalence of the most common causes of chronic liver diseases in the United States from 1988 to 2008.” *Clin Gastroenterol Hepatol* .,2011, 9,524–530.
15. Alisi A, et, al., “Association between type two diabetes and non-alcoholic fatty liver disease in youth”, *Ann Hepatol*. 2009. 44–50.
16. Yatsuji S. et. al, “Clinical features and outcomes of cirrhosis due to non-alcoholic steatohepatitis compared with cirrhosis caused by chronic hepatitis C.” , *J Gastroenterol Hepatol*., 2009, 24, 248–254.
17. Bugianesi E et. al, “Non-alcoholic fatty liver disease/non-alcoholic steatohepatitis (NAFLD/NASH): treatment.” , *Best Pract Res Clin Gastroenterol*, 2004, 18, 1105–1116.
18. Greene GW and Rossi SR. “Stages of change for reducing dietary fat intake over 18 months.” *J Am Diet Assoc.*, 1998, 98, 529–534.
19. 46. Bowman SA, et al., “Effects of fast-food consumption on energy intake and diet quality among children in a national household survey.” *Pediatrics*. 2004, 113, 112–118.
20. Feskens EJ, Loeber JG and Kromhout D. “Diet and physical activity as determinants of hyperinsulinemia: the Zutphen Elderly Study.” *Am J Epidemiol*, 1994, 140, 350–360.
21. Fierbințeanu-Braticevici C, et al., “The risk factors of fibrosis in nonalcoholic steatohepatitis.” *Rom J Intern Med.*, 2002, 40, 81–88.
22. Paik HD and Park JS, “Park E. Effects of *Bacillus polyfermenticus* SCD on lipid and antioxidant metabolisms in rats fed a high-fat and high-cholesterol diet.” *Biol Pharm Bull*, 2005, 28, 1270–1274.
23. Ashutosh M. Somalwar and Arshish D. Raut, “Study of association of non alcoholic fatty liver disease (NAFLD) with micro and macrovascular complications of type 2 diabetes mellitus (T2DM)”, *International Journal of Research in Medical Sciences*, 2014 May;2(2):493-497.

24. Shobha Luxmi, Rukhsana Abdul Sattar and Jamal Ara , “Association of Non Alcoholic Fatty Liver with type 2 Diabetes Mellitus” , JLUMHS September - December 2008, 188-193.
25. Ratziu V and Giral P, et. al., “Liver fibrosis in overweight patients.” *Gastroenterology*, 2000; 118: 1117–23.
26. Dixon JB, Bhathal PS and O’Brien PE., “Non-alcoholic fatty liver disease: predictors of non-alcoholic steatohepatitis and liver fibrosis in the severely obese”, *Gastroenterology*, 2001; 121: 91–100.
27. Pagano G and Pacini G, et. al., “Non-alcoholic steatohepatitis, insulin resistance, and metabolic syndrome: further evidence for an etiologic association”, *Hepatology*, 2002; 35: 367–72.
28. Wanless IR and Lentz JS., “Fatty liver hepatitis (steatohepatitis) and obesity: an autopsy study with analysis of risk factors”, *Hepatology*1990; 12: 1106–10.
29. Brunt EM. “Non-alcoholic steatohepatitis: definition and pathology”, *Semin Liver Dis*, 2001; 21: 3–16.
30. Matteoni CA, Younossi ZM and Gramlich T et al., “Nonalcoholic fatty liver disease: a spectrum of clinical and pathological severity”, *Gastroenterology*1999; 116: 1413–9.

## Publish Research Article

Dear Sir/Mam,

We invite unpublished Research Paper, Summary of Research Project, Theses, Books and Book Review for publication.

**Address:-Ashak Hussain Malik House No-221, Gangoo Pulwama - 192301  
Jammu & Kashmir, India**

**Cell: 09086405302, 09906662570,**

**Ph No: 01933212815**

**Email: [nairjc5@gmail.com](mailto:nairjc5@gmail.com), [info@nairjc.com](mailto:info@nairjc.com)**

**Website: [www.nairjc.com](http://www.nairjc.com)**

