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# **Original Research Article**

# The prevalence of NAFLD in CAD and it's correlation with their severity

Chhabi Satpathy<sup>1</sup>, Nirmal Kumar Mohanty<sup>1</sup>, Satya Narayan Routray<sup>1</sup>, Bijay Kumar Dash<sup>1</sup>, Ashirbad Parhi<sup>1,\*</sup>

<sup>1</sup>Dept. of Cardiology, SCB Medical College, Cuttack, Odisha, India



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#### ABSTRACT

Aim: To study the prevalence of Non-Alcoholic Fatty Liver Disease in Coronary Artery Disease patient and the correlation of Coronary Artery Disease severity in patients of Non-Alcoholic Fatty Liver Disease **Materials and Methods:** Study included 124 patients of Coronary Artery Disease who underwent detailed clinical and anthropometric examination and routine blood investigations. Ultrasonography of abdomen was done and Non-Alcoholic Fatty Liver Disease grading was done. Coronary angiography was done and angiographic severity was calculated by Gensini score.

**Result:** Non-Alcoholic Fatty Liver Disease was found in 71% of patient. There is a significant difference of Gensini score between patients with and without Non-Alcoholic Fatty Liver Disease. All the stage of Non-Alcoholic Fatty Liver Disease had a significant difference of Gensini score among them with but there was no difference of Gensini score between stage 1 and 2.

**Conclusion:** The presence of Non-Alcoholic Fatty Liver Disease Is associated with a higher Gensini score, however a higher grade of Non-Alcoholic Fatty Liver Disease doesn't mandate a higher Gensini score.

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# 1. Introduction

Cardiovascular diseases (CVDs) cause a third of world mortality, of which the most common cause is ischemic heart disease (IHD), causing approximately 7 million deaths per year.<sup>1</sup> A surge of established determinants of CVD such as non-alcoholic fatty liver disease (NAFLD), dyslipidemia, and obesity is seen recently.<sup>2</sup>

Non-alcoholic fatty liver disease (NAFLD) is defined by triglyceride deposition in the liver exceeding 5% of the total Hepatic weight in the absence of a history of heavy alcohol intake and other causes of hepatic pathology.<sup>3</sup> NAFLD is associated with metabolic syndrome and insulin resistance.<sup>4</sup> NAFLD is a complex pathology, causing structural and functional modification of the liver, increasing morbidity and mortality from progression to end stage, liver disease, and hepatocellular carcinoma.<sup>5</sup> Studies have shown that NAFLD has extra hepatic influence, also encompassing cardiovascular system (CVS) consequences.<sup>6</sup> Surprisingly maximum mortality among NAFLD patients is cardiovascular, mostly ischemic heart disease.<sup>7</sup> Evidences indicate that NAFLD causes endothelial dysfunction, inflammation and subclinical atherosclerosis of carotid artery.<sup>8</sup> The probability of NAFLD as a marker and cause of atherosclerosis has been lately evaluated.<sup>9</sup> Some studies however, reported that NAFLD may not be a causative agent of increased cardiovascular (CV) risk.<sup>10</sup>

The Gensini score is used to assess the severity of the Coronary Artery Disease (CAD).<sup>11</sup> It considers the morphology, anatomy, location and the number of obstructions and the existence of the collaterals.

\* Corresponding author. E-mail address: parhiashirbad@gmail.com (A. Parhi).

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# 2. Material and Methods

The present study was designed and conducted to determine the prevalence of NAFLD in patients with CAD. This prospective study was carried out from Jan 2020 to Dec 2020 at a tertiary care center in the Department of Cardiology, SCB Medical College and Hospital. All patients with diagnosis of CAD admitted to the department of Cardiology, SCB Medical College during the period January 2020 to December 2020 who had undergone coronary angiogram were included in the study.

# 2.1. Inclusion criteria

Patients who underwent coronary artery angiography in our Department of Cardiology SCB MCH, CUTTACK for acute coronary syndrome or chest pain with or without any degree of stenosis in the coronary arteries or their branches in coronary angiography were included in the study.

# 2.2. Exclusion criteria

Patients with age of less than 18 years, history of coronary artery bypass graft (CABG), history of alcohol consumption, any hepatic disorder, cor pulmonale, chronic renal disease, cancer, acute or chronic infections, positive serology for hepatitis B, C, human immunodeficiency virus (HIV) or syphilis were excluded from the study

Coronary angiography was performed using the various diagnostic catheters, by the femoral or radial artery approach in the SIEMENS Artis Zee image intensifier machine. At least 3 orthogonal projections in all the patients were used to evaluate the severity of coronary atherosclerotic lesions. The Gensini score was used to assess the severity of CAD.

The Gensini score was derived for each segment by multiplying the multiplication coefficient allotted based on the importance of the myocardial area supplied by that segment: (the proximal segment of the right coronary artery, 1; the mid segment of the right coronary artery, 1; the distal segment of the right coronary artery, 1; and the posterior descending artery, 1;the proximal segment of the circumflex artery, 2.5; the distal segment of the circumflex artery, 1; the obtuse marginal branch, 1; the posterolateral branch, 0.5; the left main coronary artery, 5; the proximal segment of the left anterior descending coronary artery, 2.5; the mid segment of the left anterior descending coronary artery, 1.5; the distal segment of the left anterior descending coronary artery, 1; the first diagonal branch, 1; the second diagonal branch, 0.5;) by the severity score, assigned to each range of coronary stenosis according to the degree of narrowing of the lumen (narrowing up to 25%, 26-50%, 51-75%, 76-90%, 91-99%, and complete occlusion were given Gensini scores of 1, 2, 4, 8, 16, and 32, respectively). The scores for each segment were summed and total Gensini scores were derived for each patient.

Diagnosis of NAFLD was made according to the ultrasound findings. All ultrasound examinations were performed after 12 hours of fasting by one radiologist using the device GELIGIQ F8; Samsung HS 70 A. Echogenicity of liver was compared to the echogenicity of the left kidney and using the following grading system: grade 0, no fatty liver; grade 1, mild disease; grade 2, moderate disease; and grade 3, severe disease. The method described by Saverymuttu et al was used to assess hepatic steatosis.<sup>12</sup> The method works based on the abnormally intense, high level echoes from the hepatic parenchyma, liver-kidney difference in echo amplitude and echo penetration into the deep portion of the liver and clarity of vascular pattern of the liver.<sup>13</sup>

Body mass index (BMI) was calculated as body weight in kilograms and divided by height in meter square. Obesity was defined as having a BMI  $\geq$ 30 kg/m2.<sup>14</sup> Hypertension was defined as systolic blood pressure  $\geq$ 130 mmHg, diastolic blood pressure  $\geq$ 80 mmHg as per 2019 American College of Cardiology hypertension guidelines, or requirement for antihypertensive medication.<sup>15</sup> Diabetes mellitus was defined according to ADA guidelines.<sup>16</sup>

# 2.3. Statistical analysis

Statistical analysis was done with IBM SPSS, 24.0 software. Continuous variables are expressed as Means  $\pm$  SD and Median. Categorical variables are expressed as absolute numbers and percentages. Comparisons of continuous variables were performed using the unpaired Kruskal Wallis for analyses containing more than two groups and Mann Whittney test was carried out for patients having two groups. Categorical variables in the form of NAFLD staging and Gensini category based on severity score i.e., <20 vs  $\geq$ 20 was analysed with the Chi-square test. Spearman rho correlation analysis was used for analysis of Correlation of Gensini with continuous variables. Significant level was set at <0.05.

## 2.4. Ethics

All the research participant of the study population had signed on informed consent permission from institute ethic committee was also taken

# 3. Results

Of the 124 participants, 112 (90.3%) patients had ST Elevated Myocardial infarction, 6 (4.8%) patients had Non-ST elevation myocardial infarction and 6 (4.8%) patients had chronic stable angina.

Gender distribution of the study showed 104 (83.9%) men and 20 (16.1%) women. A total of 36 (29.03%) patients were hypertensive, 60 (48.38%) patients were diabetic, 30 (24.19%) patients were hyperlipidemic, and 88 (71%) patients were having fatty liver on ultrasonography.

**Table 1:** Mean and median value of the base line characters of the study group.

Variable	Mean ± S.D.	Median
Age (years)	54.6±10.34	57
Hb (gm %)	$10.76 \pm 1.33$	10.6
Gensini Score	$34.47 \pm 27.68$	32
TLC	9024.19±2227.08	8450
FBS (mg/dL)	138.81±67.01	114
PPBS (mg/dL)	$186.66 \pm 78.45$	153
Urea (mg/dL)	26±7.79	24
Creatinine (mg/dL)	$1.17 \pm 1.28$	1
EF (%)	47.98±11.06	50.5
Cholesterol (mg/dL)	$182.53 \pm 50.73$	171.5
TG (mg/dL)	151.76±74.67	135
LDL (mg/dL)	110.1±43.61	104
HDL (mg/dL)	43.23±8.94	42
VLDL (mg/dL)	28.42±11.67	28
BMI (kg/m <sup>2</sup> )	23.22±1.89	23.4

Table 1 shows Mean and Median value of the base line characters of the study group shows a mean and median age of  $54.6\pm10.34$  years and 57 years. The mean and median Gensini was  $34.47\pm27.68$  and 32. Mean and median values for PPBS were  $186.66\pm78.45$  and 153 respectively. level of Triglyceride with a mean of  $151.76\pm74.67$  and median of 135 and cholesterol with a mean of  $182.53\pm50.73$  and median 171.5 and LDL with a mean of  $110.1\pm43.61$  and median of 104. Mean and Median value of Ejection fraction were  $47.98\pm11.06$  and 50.5 respectively.

Table 2. SEA distribution	Table	2:	Sex	distribution
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Sex	Frequency	Percent
Female	20	16.1
Male	104	83.9
Total	124	100.0

Table 2 shows a predominant male population of patients constituting 83.9% (n=104) of population was found in the study.

Table 3: NAFLD distribution

NAFLD grade	Frequency	Percent
absent	36	29.0
present	88	71.0
Total	124	100.0
NAFLD grade	Frequency	Percent
0	36	29.0
1	24	19.4
2	64	51.6
Total	124	100.0

Table 3 shows 71% of patients had NAFLD. The highest proportion formed by the NAFLD group 2 i.e., 51.6% (n=64) followed by NAFLD 1 constituting 19.4 %. (n=24). None of the patient in our study group had stage 3 NAFLD.

**Table 4:** Frequency distribution of patients with severe and non-severe gensini category

Gensini category	Frequency	Percent
<20 (Lower)	42	33.9
≥20 (Higher)	82	66.1
Total	124	100.0

Table 4 shows Patients having aGensisi score of more than equal to 20 i.e., higher Gensini category constituted 66.1% of the total study population (n=82).

Table 5 shows Spearman's Rho analysis of meanGensini with different continuous variables (Hb, TLC, FBS, PPBS, Urea, Creatine, EF, Cholesterol, TG, LDL, HDL, VLDL, BMI) showed that only creatinine is associated significantly with Gensini score (p = 0.04), negatively (Spearman's Rho = -0.25).

Table 6 shows Chi square test of higher and lower Gensini categories with NAFLD Grade 0,1 and 2 showed a significant association of NAFLD stages and Gensini severity category with chi square value of 15.26 which is significant with a p value of <0.001.

Table 7 shows Kruskal Wallis test of median value of Gensini showing a significant difference between the three groups formed by the NAFLD 0, 1 and 2 with H value of 10.05 which attained a significance level of p of 0.007. The highest Mean and Median were in the NAFLD stage 1 with a mean value of  $44\pm12.38$  and Median value of 43.5 followed by stage 2 with a mean value of  $39.18\pm31.21$  and median of 32.5

Table 8 shows Mann Whitney comparison between NAFLD 0 and 1.

Table 9 shows Mann Whitney comparison between NAFLD 0 and 2.

Table 10 shows Mann Whitney comparison between NAFLD 1 and 2 showed a non-significant difference between groups with a p value of 0.2.

#### 4. Discussion

In our study the mean and median age was  $54.6\pm10.34$  and 57 yrs respectively. This was similar to findings by, Sharma et al ( $54.71\pm19.9$  years) (17) Risk of CAD increases with age.<sup>17</sup>

In our study, 71% of patients have NAFLD, among which the highest proportion is formed by the NAFLD group 2 constituting 51.6% (n=64) followed by NAFLD 1 constituting 19.4%. (n=24). No NAFLD was found in 29% of patients. None of the patient in our study group had stage 3 NAFLD. This is corroborated in study by Majumder et al where 36/150 (24%) patients constituted no NAFLD.<sup>18</sup>

Our study shows a predominant male population of patients constituting 83.9% (n=104) of population. Risk of CAD is more in male patients. This was also seen in study by Sharma et al where males constituted 79.5 % of the total

Variable		Spearman's Rho	р
	Hb(gm%) 10.76±1.33	-0.24	0.06
	TLC9024.19±2227.08	-0.16	0.2
	FBS(mg/dL)138.81±67.01	-0.1	0.4
	PPBS(mg/dL)186.66±78.45	-0.14	0.27
	Urea(mg/dL) 26±7.79	-0.11	0.39
	Creatinine(mg/dL1.17±1.28	-0.25	0.04
Gensini Score 34.47±27.68	EF(%)47.98±11.06	0.02	0.8
	Cholesterol(mg/dL) 182.53±50.73	0.12	0.3
	TG(mg/dL) 151.76±74.67	-0.03	0.8
	LDL(mg/dL) 0110.1±43.61	0.06	0.6
	HDL(mg/dL) 43.23±8.94	0.05	0.7
	VLDL(mg/dL) 28.42±11.67	0.009	0.9
	BMI (kg/m2) 23.22±1.89	0.09	0.48

## Table 5: Spearman's Rho correlation analysis of mean Gensini with different continuous variables

Table 6: Chi square test of Gensini less than 20 and more than equal to 20 with NAFLD Grade 0,1 and 2

Cross Tabulation			Gensini Categor	у	
Cross Tabula		<20 (Lower)	$\geq$ 20 (Higher)	Chi Square	р
	0	24(57.1%)	12(14.6%)		
NAFLD	1	-	24(29.3%)	15.26	< 0.001
	2	18(42.9%)	46(56.1%)		

**Table 7:** Kruskal Wallis test of median value of Gensini showing a significant difference between the three groups formed by the NAFLD 0, 1 and 2

	Gensini Score Mean ± SD	Median	IQR	Mean Rank	Kruskal-Wallis H	р
NAFLD Score 0	$19.72 \pm 23.18$	10.75	2.25-40	21.03		
NAFLD Score 1	44±12.38	43.5	32.25-55.25	41.21	10.05	0.007
NAFLD Score 2	39.18±31.21	32.5	13.25-57.75	33.75		

## Table 8: Mann Whitney comparison between NAFLD 0 and 1

	Gensini Score Mean ± SD	Median	IQR	Mean Rank	Mann-Whitney U	р
NAFLD score 0 NAFLD score 1	19.72±23.18 44±12.38	10.75 43.5	2.25-40 32.25-55.25	11.64 21.29	38.5	0.003
NAFLD score I	$44\pm12.38$	43.5	32.25-55.25	21.29		

# Table 9: Mann Whitney comparison between NAFLD 0 and 2

	Gensini Score Mean ± SD	Median	IQR	Mean Rank	Mann-Whitney U	р
NAFLD score 0	$19.72 \pm 23.18$	10.75	2.25-40	18.89	169	0.01
NAFLD score 2	39.18±31.21	32.5	13.25-57.75	29.22	109	0.01

# Table 10: Mann Whitney comparison between NAFLD 1 and 2

	Gensini Score Mean ± SD	Median	IQR	Mean Rank	Mann-Whitney U	р
NAFLD score 1	44±12.38	43.5	32.25-55.25	26.42	145	0.2
NAFLD score 2	39.18±31.21	32.5	13.25-57.75	21.03	143	0.2

patients.<sup>19</sup> The difference in sex noted was due to effects of oestrogen in women.<sup>20,21</sup>

Spearman's Rho analysis of mean Gensini with different continuous variables shows only significant negative association with creatinine with a coefficient of -0.25 which was found to be significant with a p of 0.04. This is in contrast to studies by Cerne et al.<sup>22</sup> The difference may be due to exclusion of patient with abnormal creatinine from the study to avoid contrast induced nephropathy

Chi square test of Gensini less than 20 and more than equal to 20 with NAFLD Grade 0,1 and 2 shows a significant association of chi square value of 15.26 which is significant with a p value of <0.001. Kruskalwallis test of median value of Gensini showing a significant difference between the three groups formed by the NAFLD 0, 1 and 2 with H value of 10.05 which attained a significance of p value of 0.007.

Mann Whitney comparison between NAFLD 0 and 1 shows a significant difference which attained a p value of 0.003. Comparison between NAFLD 0 and 2 shows a significant difference with a p value of 0.01. However, comparison between NAFLD 1 and 2 shows a non-significant difference between groups with a p value of 0.2.

Patients with NAFLD patients had significantly higher Gensini value (90.2 $\pm$ 40.0 vs. 36.4 $\pm$ 28.9) than participants without NAFLD (p<0.001) as reported by Alper et al. In their study, the degree of NAFLD were significantly correlated with the severity of CAD.<sup>23</sup>

Arslan et al showed that the relative risk of the presence of CAD was 6.73 times higher in patients with NAFLD than in patients without it (p=0.035). The presence of NAFLD was an independent factor for the presence and severity of CAD.<sup>24</sup>

Studies by Wong et al showed NAFLD prevalence of 58.2% while significant CAD was observed in 76.0% of patients. It was observed in their study that NAFLD is associated independently with CAD.<sup>25</sup>

Most accepted hypothesis establishing relationship between CAD and NAFLD suggest inflammatory conditions accompanied with increased reactive oxygen species, adipocytokines as the most important factors leading to atherosclerosis and hepatic steatosis.<sup>26,27</sup> Our study indicates NAFLD as a risk factor for CAD proven angiographically. Further large-scale studies are needed to explain the mechanism of this relationship, and to identify the measures which can be taken in order to prevent future occurrence of CAD.

# 5. Conclusion

Presence of NAFLD is associated with a more severe form of coronary artery disease compared to the participants without NAFLD. But the presence of a higher grade of NAFLD does not mandate a higher Gensini score. USG guided diagnosis of NAFLD presence is a more practical, easily available and non-invasive tool for predicting higher Gensini category ( $\geq 20$ ) in Patients of CAD undergoing coronary angiogram. Though CT Scan abdomen / fibroscan / Liver Histology, which are more accurate modalities for diagnosis of NAFLD were not done in my patient subgroup which was a limitation in our study.

#### 6. Source of Funding

No financial support was received for the work within this manuscript.

## 7. Conflict of Interest

The authors declare they have no conflict of interest.

#### References

- Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study. *Lancet*. 2010;380(9859):2095–128. doi:10.1016/S0140-6736(12)61728-0.
- Moore JX, Chaudhary N, T A. Metabolic syndrome prevalence by race/ethnicity and sex in the United States, national health and nutrition examination survey. *Prev Chronic Dis.* 2017;14:1988–2012. doi:10.5888/pcd14.160287.
- Chen CH, Nien CK, Yang CC, Yeh YH. Association between nonalcoholic fatty liver disease and coronary artery calcification. *Dig Dis Sci.* 2010;55(6):1752–60. doi:10.1007/s10620-009-0935-9.
- Tuyama AC, Chang CY. Non-alcoholic fatty liver disease. J Diabetes. 2012;4(3):266–80.
- Mantovani A, Scorletti E, Mosca A, Alisi A, Byrne CD, Targher G, et al. Complications, morbidity and mortality of nonalcoholic fatty liver disease. *Metabolism.* 2020;111S:154170. doi:10.1016/j.metabol.2020.154170.
- Ismaiel A, Srouji NA. Subclinical left ventricular systolic dysfunction assessed using myocardial strain measured by speckle tracking in nonalcoholic fatty liver disease - systematic review. *Glob J Med Berap.* 2020;2(2):1–8. doi:10.46982/gjmt.2020.104.
- Targher G, Day CP, Bonora E. Risk of cardiovascular disease in patients with nonalcoholic fatty liver disease. *New Engl J Med.* 2010;363(14):1341–50. doi:10.1056/NEJMra0912063.
- Gastaldelli A, Kozakova M, Hojlund K, Flyvbjerg A, Favuzzi A, Mitrakou A, et al. Fatty liver is associated with insulin resistance, risk of coronary heart disease, and early atherosclerosis in a large European population. *Hepatology*. 2009;49(5):1537–44.
- Targher G, Arcaro G. Non-alcoholic fatty liver disease and increased risk of cardiovascular disease. *Atherosclerosis*. 2007;191(2):235–40. doi:10.1016/j.atherosclerosis.2006.08.021.
- Lauridsen BK, Stender S, Kristensen TS. Liver fat content, nonalcoholic fatty liver disease, and ischaemic heart disease: mendelian randomization and meta-analysis of 279 013 individuals. *Eur Heart J*. 2018;39(5):385–93. doi:10.1093/eurheartj/ehx662.
- Gensini GG. A more meaningful scoring system for determining the severity of coronary heart disease. *Am J Cardiol.* 1983;51(3):606. doi:10.1016/s0002-9149(83)80105-2.
- Saverymuttu SH, Joseph AE, Maxwell JD. Ultrasound scanning in the detection of hepatic fibrosis and steatosis. *Br Med J (Clin Res Ed)*. 1986;292(6512):13–5.
- Yajima Y, Ohta K, Narui T, Abe R, Suzuki H, Ohtsuki M, et al. Ultrasonographical diagnosis of fatty liver: significance of the liverkidney contrast. *Tohoku J Exp Med.* 1983;139(1):43–50.
- WHO, Physical Status: The Use and Interpretation of Anthropometry

   Report of a WHO Expert Committee ; 1995.

- Whelton PK, Carey RM, Aronow WS, Jr CD, Collins KJ, Himmelfarb CD, et al. PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: Executive Summary: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Hypertension*. 2017;71(6):1269–324. doi:10.1161/HYP.00000000000066.
- American Diabetic Association. Diagnosis and Classification of Diabetes Mellitus. *Diabetes care*. 2013;36(1):67–74. doi:10.2337/dc13-S067.
- Aydın AM, Kayalı A, Poyraz AK. The relationship between coronary artery disease and pericoronary epicardial adipose tissue thickness. J Int Med Res. 2015;43(1):17–25.
- Majumder B, Tandel V, Ghosh S, Chatterjee S. Association of nonalcoholic fatty liver disease and coronary artery disease. *Int J Res Med Sci.* 2016;4(10):4359–64.
- Singh RB, Sharma JP, Rastogi V, Raghuvanshi RS, Moshiri M, Verma SP, et al. Prevalence of coronary artery disease and coronary risk factors in rural and urban populations of north India. *Eur Heart J.* 1997;18(11):1728–35. doi:10.1093/oxfordjournals.eurheartj.a015167.
- Rossouw J. Estrogens for prevention of coronary heart disease. Circulation. 1996;94(11):2982–5. doi:10.1161/01.cir.94.11.2982.
- Lennep HW, Westerveld HT, Zwinderman A, van Lennep JR, Slot HB, Erkelens DW, et al. Differential effect of female gender on coronary artery disease and peripheral artery disease. *Neth Heart J.* 2002;10(12):500–5.
- Cerne D, Kaplan-Pavlovcic S, Kranjec I, Jurgens G. Mildly elevated serum creatinine concentration correlates with the extent of coronary atherosclerosis. *Ren Fail*. 2000;22(6):799–808. doi:10.1081/jdi-100101965.
- 23. Alper AT, Hasdemir H, Sahin S, Onturk E, Akyol A, Nurkalem Z, et al. The relationship between nonalcoholic fatty liver disease and the severity of coronary artery disease in patients with metabolic syndrome. *Turk Kardiyol Dern Ars*. 2008;36(6):376–81.

- Arslan U, Türkoğlu S, Karakan T, Çengel A. Association between nonalcoholic fatty liver disease and coronary artery disease. *Coron Artery Dis.* 2007;18(6):433–6. doi:10.1097/MCA.0b013e3282583c0d.
- Wong VW, Wong GL, Yip GW, Lo AO, Limquiaco J, Chu WC, et al. Coronary artery disease and cardiovascular outcomes in patients with nonalcoholic fatty liver disease. *Gut.* 2011;60(12):1721–7. doi:10.1136/gut.2011.242016.
- Targher G, Bertolini L, Padovani R, Rodella S, Zoppini G, Zenari L. Relations between carotid artery wall thickness and liver histology in subjects with nonalcoholic fatty liver disease. *Diabetes Care*. 2006;29(6):1325–30. doi:10.2337/dc06-0135.
- Torres DM, Harrison SA. Diagnosis and therapy of non alcoholic steatohepatitis. *Gastroenterology*. 2008;134(6):1682–98. doi:10.1053/j.gastro.2008.02.077.

#### Author biography

Chhabi Satpathy, Associate Professor

Nirmal Kumar Mohanty, Associate Professor

Satya Narayan Routray, Professor

Bijay Kumar Dash, Assistant Professor

Ashirbad Parhi, Senior Resident

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