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# Nonalcoholic fatty liver disease and outcomes in patients with acute coronary syndrome

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## Abstract

### Background

According to the results of longitudinal cohort study, patients with nonalcoholic fatty liver disease (NAFLD) were found to have a higher mortality owing to coronary heart disease than the liver cirrhosis. Patients with NAFLD may also have a higher prevalence of subclinical atherosclerosis independent of established cardiovascular risk factor.

### Patients and methods

This study was performed at Shebin El-Kom Teaching Hospital from January 2018 to March 2018. All consecutive patients with nonfatal acute coronary syndrome (ACS) admitted were included in this study. Patients with excessive alcohol consumption, patients with established cirrhosis, and patients with identified risk factors for liver disease were excluded from study. All patients underwent ultrasound scan of liver.

### Results

There were 118 participants with ACS. The number of patients with NAFLD was 55 (46.6%) and patients without NAFLD was 63 (53.4%). The number of male patients with NAFLD was 31 (56.4%) versus 40 (63.5%) in patients without NAFLD. There was a significant increase in total Global Registry of Acute Coronary Events score in patients with NAFLD versus patients without NAFLD and also patients with NAFLD showed significant increased high and intermediate risk of death during the hospital stay versus patients without NAFLD.

### Conclusion

Patients with NAFLD and ACS require aggressive treatment of CAD and higher predicted mortality.

**Keywords:** Acute coronary, fatty liver, nonalcoholic

## INTRODUCTION

Nonalcoholic fatty liver disease (NAFLD) affects 30% of adults in general population [1]. NAFLD comprises a spectrum of disorders ranging from simple steatosis (fatty infiltration of liver) to inflammatory steatohepatitis to possible long-term injury (fibrosis and cirrhosis) [2]. Its underlying predisposing cardiometabolic traits are those of insulin resistance, abdominal obesity, and dyslipidemia. NAFLD occurs in 70–90% of those with recognized type 2 diabetes mellitus [3]. Some studies suggested that NAFLD was manifestation of the metabolic syndrome. Pathological feature was abnormal deposition of lipid in liver cells [4]. According to the result of longitudinal cohort study, patients with NAFLD were found to have a higher mortality owing to coronary heart disease than the

liver cirrhosis [5]. Patients with NAFLD may also have a higher prevalence of subclinical atherosclerosis independent of the established cardiovascular risk factors [6]. To assess subclinical atherosclerosis, potent noninvasive procedures are available, such as carotid intima-media thickness measurement, brachial artery flow mediated dilatation, and arterial stiffness [7]. Using coronary imaging, such as multislice computed tomography (CT), studies have also shown that NAFLD was significantly related to lipid core and calcified plaques [8]. Apart from this elevation, a

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common marker of liver injury [ $\gamma$ -glutamyltransferase, alanine aminotransferase (ALT), aspartate aminotransferase, alkaline phosphatase, and bilirubin] are associated with the risk of cardiovascular disease. However, it is currently unknown if liver enzyme concentrations are associated with the severity of a stenosis in acute myocardial infarction [9].

## PATIENTS AND METHODS

Ethical committee approval was taken. This study was performed at Shebin El-Kom Teaching Hospital from January 2018 to March 2018. Patients with acute coronary syndrome (ACS) admitted during the study period were included. Inclusion criteria were (i) age between 18 and 80 years at admission to the ICU with ACS, within 24 h of admission, (ii) ECG changes with ACS, and (iii) positive cardiac troponin. Exclusion criteria were (i) alcoholic hepatitis, (ii) liver cirrhosis, (iii) any known risk factor for liver disease, (iv) patients who died before reevaluation of NAFLD, (v) use of drugs that produce fatty liver such as steroid, amiodarone, and chemotherapeutic agents within the previous 6 months.

Anthropometric measures such as weight, height, BMI, and waist circumference were measured. Obesity was defined as BMI of at least 27.5 kg/m<sup>2</sup>. High waist circumference was taken as more than 90 cm in males and 80 cm in females. Diabetes was defined as 2-h postprandial blood glucose more than 200 mg/dl, fasting blood glucose more than 126 mg/dl, and glycated hemoglobin A1c more than 6.5. Serum triglyceride, high-density lipoprotein, low-density lipoprotein, and total cholesterol were measured. Abdominal ultrasound was performed by radiologist to diagnose fatty liver as increase in echogenicity of liver in comparison with right renal cortex, poor visualization of intrahepatic structures, and attenuation of the ultrasound beam.

Global Registry of Acute Coronary Events (GRACE) score was calculated in all patients on admission to assess severity of ACS. Using the GRACE risk score, eight factors independently predict risk of heart attack and/or death, age, heart rate, systolic blood pressure, renal function, congestive heart failure, ST-segment deviation, cardiac arrest, and elevation biomarkers. This categorization was used in our study to predict mortality during hospital stay [10].

### Statistical analysis

Statistical analysis was performed using SPSS 17.0 version SPSS, IBM Corporate headquarters 1 New Orchard Road Armonk, New York 10504-1722 United States US: 914-499-1900. The Student's *t*-test was used to test statistical difference in the means between groups, and the  $\chi^2$ -test was used to test difference in frequency of population and clinical variables between groups. *P* value of less than 0.05 was considered significant. Value were expressed as mean  $\pm$  SD.

## RESULTS

There were 118 participants with ACS. The number of patients with NAFLD was 55 (46.6%), and the number of patients without NAFLD was 63 (53.4%). The number of male patients with

NAFLD was 31 (56.4%) versus 40 (63.5%) in patients without NAFLD, those with diabetes was 37 (67.3%) versus 31 (39.2%), those with hypertension was 43 (78.1%) versus 41 (65.1%), those with dyslipidemia was 30 (54.5%) versus 22 (34.9%), those with smoking was 25 (45.5%) versus 30 (47.6%), and those with obesity was 20 (36.4%) versus eight (12.3%) (Table 1).

Metabolic risk factor profile in patients with NAFLD and without NAFLD shows significant increase of total cholesterol (214.3  $\pm$  39.6 vs. 188.7  $\pm$  30.5), triglyceride (160.3  $\pm$  45.8 vs. 120.4  $\pm$  45.3), low-density lipoprotein (150.4  $\pm$  37.3 vs. 121.3  $\pm$  22.6) in patients with NAFLD versus patients without NAFLD. There was a nonsignificant increase in fasting blood sugar (157  $\pm$  70.4 vs. 139  $\pm$  71.5) and high-density lipoprotein (34.3  $\pm$  22.5 vs. 35.5  $\pm$  25.8) in patients with NAFLD versus patients without NAFLD (Table 2).

Risk categories in patients with NAFLD and without NAFLD according to GRACE score showed significant increase in total GRACE score in patients with NAFLD versus patients without NAFLD and also patients with NAFLD showed significant increase in high and intermediate risk of death during the hospital stay versus patients without NAFLD (Table 3).

## DISCUSSION

This study evaluated the effect of NAFLD on predicted mortality from ACS. Previous studies have compared

**Table 1: Demographic variable in patients with nonalcoholic fatty liver disease and without nonalcoholic fatty liver disease**

Variables	NAFLD [n=55 (46.6%)]	Non-NAFLD [n=63 (53.4%)]	P
Age (years)	60.7 $\pm$ 11.6	61.3 $\pm$ 11.3	>0.05
Male	31 (56.4)	40 (63.5)	>0.05
Female	24 (43.6)	23 (36.5)	>0.05
Diabetes	37 (67.3)	31 (39.2)	<0.05
Hypertension	43 (78.1)	41 (65.1)	>0.05
Dyslipidemia	30 (54.5)	22 (34.9)	<0.05
Smoking	25 (45.5)	30 (47.6)	>0.05
Obesity (BMI >27.5 kg/m <sup>2</sup> )	20 (36.4)	8 (12.7)	<0.05

NAFLD, nonalcoholic fatty liver disease.

**Table 2: Metabolic risk factor profile in patients with nonalcoholic fatty liver disease and without nonalcoholic fatty liver disease**

Variables	NAFLD	Non-NAFLD	P
FBS	157 $\pm$ 70.4	139 $\pm$ 71.5	>0.05
Total cholesterol	214.3 $\pm$ 39.6	188.7 $\pm$ 30.5	<0.001
Triglyceride	160.3 $\pm$ 45.8	120.4 $\pm$ 45.3	<0.001
LDL	150.4 $\pm$ 37.3	121.3 $\pm$ 22.6	<0.001
HDL	34.3 $\pm$ 22.5	35.5 $\pm$ 25.6	>0.05
ALT	60.2 $\pm$ 44.2	29.6 $\pm$ 12.5	<0.001

ALT, alanine aminotransferase; FBS, fasting blood sugar; HDL, high-density lipoprotein; LDL, low-density lipoprotein; NAFLD, nonalcoholic fatty liver disease.

**Table 3: Risk categories in patients with nonalcoholic fatty liver disease and without nonalcoholic fatty liver disease according to Global Registry of Acute Coronary Events score**

ACS severity	NAFLD (n=55) [n (%)]	Non-NAFLD (n=63) [n (%)]	P
GRACE score	121.4±25.3	91.4±23.1	<0.05
Predicted in hospital mortality			
Low risk	24 (43.6)	49 (77.8)	<0.001
Intermediate risk	18 (32.7)	11 (17.5)	
High risk	13 (23.7)	3 (4.7)	

ACS, acute coronary syndrome; GRACE, Global Registry of Acute Coronary Events; NAFLD, nonalcoholic fatty liver disease.

angiographic or multislice CT data as a measure of coronary artery disease (CAD) burden in patients with NAFLD. Functional significance of NAFLD-related coronary lesions is not proved, and studies in patients with ACS are lacking [11].

The prevalence of NAFLD in our study was 46.6 versus 46.7 in other study [11]. It is higher than the previously reported community prevalence of NAFLD in western countries [12]. This indicates a strong association between NAFLD and CAD. Our study population represented a group with increase metabolic risk factors compared with healthy adults. In our study, NAFLD prevalence is comparable to data from China reporting a CT-detected NAFLD prevalence of 45.8% [13]. ALT had a significant increase in patients with NAFLD; although serum ALT is a sensitive marker for NAFLD, it lacks specificity especially in the acute care setting [14]. Higher concentration may have been a product of severe heart failure or medication use. Hypertension, diabetes, and dyslipidemia were common in the NAFLD group in our study. Insulin resistance was a major driving force for atherogenic dyslipidemia and contributes to pathogenesis of NAFLD [14]. Hepatic and systemic inflammation in NAFLD may lead to the release of proatherogenic factors from the liver mediated by nuclear factor- $\kappa$ B [15]. Adipokine originating from adipose tissue may further amplify this inflammatory cascade by stimulating hepatic release of C-reactive protein, an established risk factor for cardiovascular disease [16]. Our study found that significantly higher GRACE score in patients with NAFLD compared with patients without NAFLD, similar to a previous study [11]. More patients with NAFLD were seen in the high- and intermediate-risk groups. Mortality predicted by the GRACE score during the hospital stay was high among patients with NAFLD, similar to the previous study [11].

## CONCLUSION

Patients with NAFLD and ACS require aggressive treatment of CAD and higher predicted mortality.

## Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have

given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Nil.

## Conflicts of interest

There are no conflicts of interest.

## REFERENCES

- Chalalani N, Younossi Z, Lavine JE, Diehl AM, Brunt EM, Cusi K, *et al.* The diagnosis and management of non-alcoholic fatty liver disease: Practice Guideline by the American Association for the Study of Liver Diseases, American College of Gastroenterology, and the American Gastroenterological Association 2012;55:2005-23.
- Ong JP, Pitts A, Younossi ZM. Increased overall mortality and liver related mortality in non-alcoholic fatty liver disease. *J Hepatol* 2008; 49:608-612.
- Cali AM, De Oliveira AM, Kim H, Chen S, Reyes-Mugica M, Escalera S, *et al.* Glucose dysregulation and hepatic steatosis in obese adolescents: is there a link?. *Hepatology* 2009;49:1896-903.
- Targher G, Marra F, Marchesini G. Increased risk of cardiovascular disease in non-alcoholic fatty liver disease: causal effect or epiphenomenon? *Diabetologia* 2008; 51:1947-1953.
- Ekstedt M. Long-term follow-up of patients with NAFLD and elevated liver enzymes. *Hepatology* 2006; 44:865-873.
- Blackett PR, Sanghera DK. Genetic determinants of cardiometabolic risk: a proposed model for phenotype association and interaction. *J Clin Lipidol* 2013; 7:65-81.
- Ozturk K, Altun B, Kurt O, Demirci H. Neutrophil-to-lymphocyte ratio for predicting fibrosis in nonalcoholic fatty liver disease. *European Journal of Gastroenterology and Hepatology* 2015;27:1479.
- Akabame S, Hamaguchi M, Tomiyasu KI, Tanaka M, Kobayashi-Takenaka Y, Nakano K, *et al.* Evaluation of vulnerable coronary plaques and non-alcoholic fatty liver disease (NAFLD) by 64-detector multislice computed tomography (MSCT). *Circulation Journal* 2007;72:618-25.
- Park JB, Kang DY, Yang HM, Cho HJ, Park KW, Lee HY, *et al.* Serum alkaline phosphatase is a predictor of mortality, myocardial infarction, or stent thrombosis after implantation of coronary drug-eluting stent. *European Heart Journal* 2012;34:920-31.
- The GRACE Investigators. Rationale and design of the GRACE (Global Registry of Acute Coronary Events) Project: a multinational registry of patients hospitalized with acute coronary syndromes. *Am Heart J* 2001; 141:190-199.
- Perera N, Indrakumar J, Abeysinghe WV, Fernando V, Samaraweera WM, Lawrence JS. Non alcoholic fatty liver disease increases the mortality from acute coronary syndrome: An observational study from Sri Lanka. *BMC Cardiovascular Disorders* 2016;16:37.
- Loria P, Adinolfi LE, Bellentani S, Bugianesi E, Grieco AL, Fargion S, *et al.* Practice guidelines for the diagnosis and management of nonalcoholic fatty liver disease: A decalogue from the Italian Association for the Study of the Liver (AISF) Expert Committee. *Digestive and Liver Disease* 2010;42:272-82.
- Ling S, Shu-Zheng LU. Association between non-alcoholic fatty liver disease coronary artery disease severity. *Chin Med J* 2011; 124:867-872.
- Yun KE, Shin CY, Yoon YS, Park HS. Elevated alanine aminotransferase levels predict mortality from cardiovascular disease and diabetes in Koreans. *Atherosclerosis* 2009;205:533-7.
- Ioannou GN, Weiss NS, Boyko EJ, Mozaffarian D, Lee SP. Elevated serum alanine aminotransferase activity and calculated risk of coronary heart disease in the United States. *Hepatology* 2006;43:1145-51.
- Ravichandran L, Goodman SG, Yan AT, Mendelsohn A, Ray JG. Non-alcoholic fatty liver disease and outcomes in persons with acute coronary syndromes: insights from the GRACE-ALT analysis. *Heart Asia* 2012;4:137-40.