Association between Nonalcoholic Fatty Liver Disease and Coronary Artery Disease in Patients with Angina Pectoris

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Aims and objectives

Nonalcoholic fatty liver disease (NAFLD) is one of the most common hepatic disorders (1 on page ). Prevalence of this disorder is 10-30% in general population and its prevalence is increasing in both developing and developed countries (2-5 on page ). NAFLD has been strongly associated with the insulin resistance and metabolic syndrome; most of the patients are overweight or obese, and they have glucose metabolism deficiencies, dyslipidemia, and hypertension which all are the risk factors of NAFLD (6-8 on page ).

Coronary artery disease (CAD) is the first cause of death in rich countries and the second cause of death in the medium and low income countries (9 on page ). Risk factors for CAD include age, gender (10-14 on page ), diabetes mellitus(15-18 on page ), low physical activity, smoking, hyperlipidemia, metabolic syndrome, and diet (9 on page ). Study that was conducted in Iranian population shows that 24% of adults in Isfahan city have these risk factors (19 on page ).

NAFLD has been thought to be a new risk factor for CAD. Other new risk factors are inflammatory markers, fibrinolytic and hemostatic function. Recent studies revealed that NAFLD patients have an accelerated risk of increased carotid intima and media thickness (20 on page , 21 on page ), reduced endothelial function (22 on page ), increased coronary artery calcification (23 on page , 24 on page ), and increased arterial stiffness (20 on page ). According to some studies, NAFLD and CAD have the similar risk factors including dyslipidemia, hypertension, physical inactivity, insulin resistance and inflammation, therefore, a possible relationship between NAFLD and CAD can be assumed and evaluated (8 on page , 25 on page ,26 on page ).

This study aims to investigate the possible association between NAFLD and CAD in patients with angina pectoris.

Methods and materials

Participants This is a case-control study conducted on the patients with angina pectoris and the candidates for coronary angiography. Patients with the history of recent myocardial infarction or other general disorders, pregnant women and alcoholics were excluded. Coronary angiography was performed and the severity of CAD was assessed in those with the positive coronary angiography results. 82 patients with the confirmed CAD in coronary angiography and 82 individuals with matched age, sex, sugar and lipid
profile, and weight and height as the control group with the normal coronary angiography results were selected with simple randomization.

**Conventional X-Ray Coronary Angiography**

All participants were undergone standard clinical diagnostic x-ray angiography. Conventional x-ray coronary angiography was performed with an integrated digital cardiac catheter imaging system. Then, the x-ray angiograms were investigated by the three cardiologists, and, the percentage of stenosis diameter, as the reference diameter, was determined, according to the SI Unit.

**Hepatic ultrasound scanning**

Hepatic ultrasound scanning was performed in all the subjects by one trained radiologist, who was unaware of all the clinical and laboratory characteristics of the patients, using a Toshiba Nemio 30 scanner (just vision model) (Toshiba CO. Ltd, Tokyo, Japan) with a 3.5 MHz linear transducer. The presence or absence and the grading of fatty infiltration of the liver were recorded. Absence or grade 0 of fatty infiltration was considered to be the normal liver echogenicity. Grade 1 (mild) fatty infiltration was considered when the echogenicity slightly increased, with normal visualization of the diaphragm and the intrahepatic vessel borders. Grade 2 (moderate) of fatty infiltration was defined when echogenicity moderately increased, with slightly impaired visualization of the diaphragm or intrahepatic vessels. Grade 3 (severe) of fatty infiltration was considered when echogenicity markedly increased with the poor visualization of diaphragm, the intrahepatic vessels and the posterior portion of the right lobe.

**Statistical analysis**

All data were analyzed by SPSS (version 16) software. Data are presented as the mean ± standard deviation (SD) or frequencies. Statistical analyses included the independent T-test (for continues variables) and the Chi-square test (for categorical variables). P<0.05 was considered statistically significant.

**Results**

This study was conducted on 82 CAD patients and 82 normal participants. Case and control groups were matched by age, sex, blood sugar and lipid profile, weight and height. There was 24 females (29.26%) and 58 males (70.73%) in each group (CAD patients and control group).
Our results showed that patients with CAD had significantly higher NAFLD compared to the control group (p=0.001). In addition, the proportions of subjects with NAFLD in CAD patients group were significantly higher than control group in the two genders. 50 males and 20 females in CAD group had NAFLD (Table 1).

The mean severity of NAFLD in CAD group was significantly higher than the control group (1.24 vs 0.59). Frequencies of various grades of NAFLD in CAD patients and control group were shown in figure 1.

There was no significant difference between the CAD group and control group, about the liver size (4.9% vs 3.7%). The mean size of spleen in control group was higher than CAD group. However this difference was not statistically significant. The mean size of portal vein in CAD group was significantly greater than the control group (Table 1).

**Conclusion**

The present study shows that the prevalence of NAFLD in CAD patients with angina pectoris was more than the control group in the whole population and the two genders. Grade of fatty liver and the portal vein size in CAD patients were more than the control group. Therefore, NAFLD can be a risk factor and the cause of CAD in patients with angina pectoris. Previous studies shows that NAFLD independently enhances the risk of CAD and peripheral vascular diseases (23 on page , 27 on page , 28 on page ). Gastaldelli et al reported that fatty liver is significantly associated with the increased CAD risk and reduced insulin sensitivity in non-diabetic subjects (29 on page ). According to the findings, cardiovascular mortality rate is increasing in NAFLD patients (30 on page ). Targer et al (31 on page )reported that NAFLD is a predictor of CVD in type II diabetes. Hamaguchi et al (32 on page )showed this relationship between NAFLD and CVD in healthy patients. Several mechanisms were suggested for the relationship between NAFLD and CAD. Oxidative stress increases in NAFLD and finally leads to modification of low-density lipoproteins (LDL) and produces oxidized LDL that is a risk factor for atherosclerosis and CAD (33 on page ). Studies showed that NAFLD is strongly associated with insulin resistance (IR) as well as reduced insulin sensitivity (34 on page ). IR leads to the endothelial dysfunction and finally atherosclerosis (35 on page ). Studies show that the levels of IL-6 and CRP is increased in NAFLD ,and NAFLD was considered as a chronic inflammatory condition. Inflammation mediates all the stages of atherosclerosis (9 on page ). In addition , patients with NAFLD have reduced levels of adiponectin (36 on page ), and lower plasma adiponectin concentration is associated with early CAD onset and multiple atherosclerotic lesions in coronary arteries.

This study that was conducted among the patients with angina pectoris shows that the NAFLD is more prevalent in CAD patients. Thus, NAFLD is a risk factor for the presence
of CAD in patients with angina pectoris and clinicians must pay attention to this risk factor in health care.

**Personal information**

This study was performed in Isfahan University Of Medical Sciences, Radiology department and cardiovascular research center.

**Images for this section:**

![Ultrasonographic fatty liver disease](image)

**Fig. 1:** ultrasonographic fatty liver disease
Fig. 2: ultrasonographic fatty liver disease
Fig. 3: coronary angiography
References:


