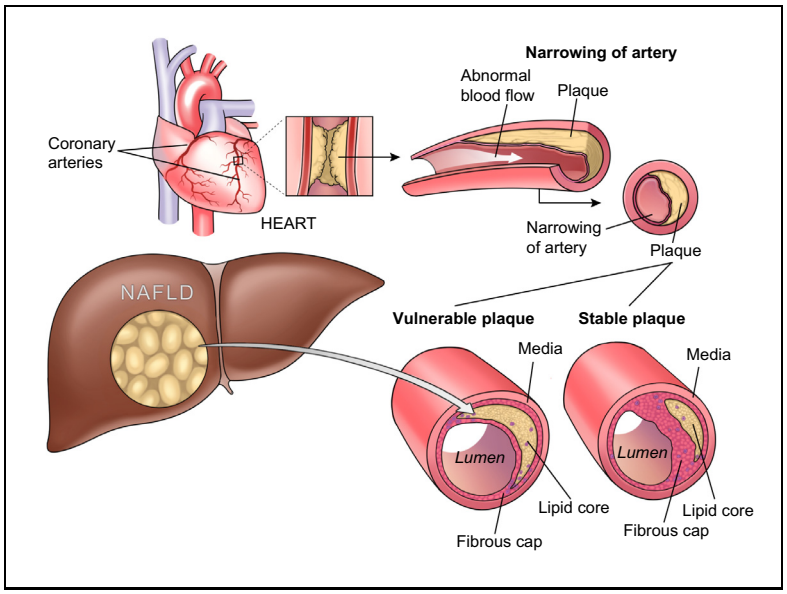
Research Article
Genetic and Metabolic Diseases

JOURNAL
OF HEPATOLOGY

Association between non-alcoholic fatty liver disease and subclinical coronary atherosclerosis: An observational cohort study

Graphical abstract



Highlights

- NAFLD was associated with an increased risk of cardiovascular events.
- Non-calcified plaque is a vulnerable plaque with potential cardiac risks.
- NAFLD was an independent risk factor for non-calcified plaque.
- Appropriate therapy for NAFLD was required to reduce the future cardiac events.

Authors

Seung Bum Lee, Gyung-Min Park, Jong-Young Lee, ..., Hong-Kyu Kim, Jaewon Choe, Han Chu Lee

Correspondence

min8684@hanmail.net
(G.-M. Park)
jyleeheart@naver.com
(J.-Y. Lee)

Lay summary

In asymptomatic individuals, non-alcoholic fatty liver disease (NAFLD) was an independent risk factor for non-calcified plaque, which has been known as a vulnerable plaque associated with sudden and unexpected cardiac events. Therefore, appropriate medical therapy for NAFLD was required to reduce future cardiac events.

http://dx.doi.org/10.1016/j.jhep.2017.12.012

Research Article
Genetic and Metabolic Diseases

JOURNAL OF HEPATOLOGY

Association between non-alcoholic fatty liver disease and subclinical coronary atherosclerosis: An observational cohort study

Seung Bum Lee[1], Gyung-Min Park[1,*], Jong-Young Lee[2,*], Byung Uk Lee[1], Jae Ho Park[1], Byung Gyu Kim[1], Seok Won Jung[1], In Du Jeong[1], Sung-Jo Bang[1], Jung Woo Shin[1], Neung Hwa Park[1], Dong Hyun Yang[3], Joon-Won Kang[3], Tae-Hwan Lim[3], Hong-Kyu Kim[4], Jaewon Choe[4], Han Chu Lee[5]

[1]*Department of Internal Medicine, Ulsan University Hospital, University of Ulsan College of Medicine, Ulsan, South Korea;* [2]*Department of Internal Medicine, Kangbuk Samsung Hospital, School of Medicine, Sungkyunkwan University, Seoul, South Korea;* [3]*Department of Radiology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, South Korea;* [4]*Health Screening and Promotion Center, Asan Medical Center, University of Ulsan College of Medicine, Seoul, South Korea;* [5]*Department of Internal Medicine, Asan Medical Center, University of Ulsan College of Medicine, Seoul, South Korea*

See Editorial, pages 890–892

Background & Aims: There are limited data on the association between non-alcoholic fatty liver disease (NAFLD) and subclinical coronary atherosclerosis. This study investigated the influence of NAFLD on subclinical coronary atherosclerosis as detected by coronary computed tomography angiography (CCTA) in an asymptomatic population.

Methods: A total of 5,121 consecutive asymptomatic individuals with no prior history of coronary artery disease or significant alcohol intake voluntarily underwent abdominal ultrasonography and CCTA as part of a general health examination. Fatty liver was assessed by ultrasonography examination. The fatty liver index and NAFLD fibrosis score were also calculated. Coronary atherosclerotic plaques on CCTA were evaluated. The association between NAFLD and subclinical coronary atherosclerosis was determined by logistic regression analysis.

Results: Of the study participants, 1,979 (38.6%) had ultrasonography-diagnosed NAFLD. After adjustment for cardiovascular risk factors, there were no statistically significant differences in the adjusted odds ratios of NAFLD for calcified plaque (1.03; 95% CI 0.89–1.20; $p = 0.673$) and mixed plaque (1.15; 95% CI 0.93–1.42; $p = 0.214$). However, adjusted odds ratios for any atherosclerotic plaque (1.18; 95% CI 1.03–1.35; $p = 0.016$) and non-calcified plaque (1.27; 95% CI 1.08–1.48; $p = 0.003$) were significantly higher in NAFLD. In addition, there was a significant association of fatty liver index ≥30 with non-calcified plaque (1.37; 95% CI 1.14–1.65; $p = 0.001$) and NAFLD fibrosis score ≥−1.455 with non-calcified plaque (1.20; 95% CI 1.08–1.42; $p = 0.030$).

Conclusions: In this large cross-sectional study of asymptomatic individuals undergoing CCTA, NAFLD was consistently associated with non-calcified plaque, suggesting an increased cardiovascular risk.

Lay summary: In asymptomatic individuals, non-alcoholic fatty liver disease (NAFLD) was an independent risk factor for non-calcified plaque, which has been known as a vulnerable plaque associated with sudden and unexpected cardiac events. Therefore, appropriate medical therapy for NAFLD was required to reduce future cardiac events.

Introduction

Non-alcoholic fatty liver disease (NAFLD) is a prevalent liver disease in the general population, affecting up to 30% of the adult population in the United States and Europe.[1,2] NAFLD is also considered as a hepatic manifestation of metabolic syndrome, with insulin resistance a common pathophysiology.[3,4] The presence of NAFLD has been known to be associated with an increased prevalence and incidence of cardiovascular disease, independently of other well-known cardiovascular risk factors.[5–9] However, there are limited data regarding the association between NAFLD and subclinical coronary atherosclerosis in asymptomatic individuals. Recently, with the advent of multidetector row computed tomography, coronary computed tomography angiography (CCTA) has proven to be effective in providing a comprehensive evaluation of coronary atherosclerosis, including lesion location, severity, and plaque characteristics.[10] A recent meta-analysis with CCTA showed that the specific characteristics of atherosclerotic plaques determined the different risk of future cardiovascular events.[11] Therefore, this study sought to evaluate the relationship between NAFLD and characteristics of atherosclerotic plaques through a large cohort of asymptomatic Korean individuals who voluntarily underwent CCTA for early detection of coronary artery disease (CAD).

Patients and methods

Study population

From January 2007 to December 2011, 9,249 consecutive South Korean individuals aged ≥20 years who had undergone

Keywords: Non-alcoholic fatty liver disease; Coronary artery disease; Atherosclerosis; Coronary computed tomography angiography.
Received 30 May 2017; received in revised form 19 September 2017; accepted 10 December 2017; available online 20 December 2017

* Corresponding authors. Address: Department of Internal Medicine, Ulsan University Hospital, University of Ulsan College of Medicine, 877, Bangeojinsunhwando-ro, Dong-gu, Ulsan 44033, South Korea. Tel.: +82 52 250 8993; fax: +82 52 250 7048 (G.-M. Park), or Department of Internal Medicine, Kangbuk Samsung Hospital, School of Medicine, Sungkyunkwan University, 29, Saemunan-ro, Jongno-gu, Seoul 03181, South Korea. Tel.: +82 2 2001 1407; fax: +82 2 2001 2400 (J.-Y. Lee).
E-mail addresses: min8684@hanmail.net (G.-M. Park), jyleeheart@naver.com (J.-Y. Lee).