Original Article

Impact of Smoking Status on Lipoprotein Subfractions: Data from an Untreated Chinese Cohort*

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Abstract

Objective Cigarette smoking is one of the established risk factors of atherosclerotic cardiovascular disease, however, its impact on lipids is not completely understood, especially in the Chinese population. Therefore, this study evaluated the impact of smoking status (non, former, and current smoking) on the distribution of lipoprotein subfractions in untreated patients with angina-like chest pain.

Methods A total of 877 patients were consecutively enrolled and divided into nonsmoking (n = 518), former smoking (n = 103), and current smoking (n = 256) groups. Both low- and high-density lipoprotein cholesterol (LDL-C and HDL-C) subfractions were measured using the Quantimetrix Lipoprint System. The distributions of lipoprotein subfractions were evaluated among the groups.

Results Compared with nonsmoking subjects, the current smoking group had significantly lower large/medium HDL-C (both $P < 0.001$) concentration and large HDL subfraction percentage but higher small HDL-C and medium LDL-C concentrations as well as medium LDL subfraction percentage. Importantly, former smoking subjects showed elevated levels of large HDL-C concentration, large HDL particle percentage, and mean LDL particle size and attenuation in small HDL/LDL percentages and small LDL-C concentration, but these levels did not reach the optimal status compared with those of the non-smoking group (data not shown).

Conclusion Smoking has an adverse impact on the lipoprotein subfractions, presented as lower large HDL particles besides higher small HDL and medium LDL particles, whereas smoking cessation could reverse these change to a certain degree.

Key words: Smoking; Smoking cessation; Lipoprotein subfractions; Chinese

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INTRODUCTION

Epidemiological studies have demonstrated that cigarette smoking (CS) is an independent risk factor for the development of atherosclerotic cardiovascular disease (ASCVD) and significantly contributes to the morbidity and mortality[1-3]. The effects of CS on cardiovascular system were reported to be doserelated[4], but fortunately, it has also been

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reported that smoking cessation can diminish the risk and improve the outcomes of ASCVD[5-6]. Importantly, the deleterious impact of CS on the cardiovascular system has been intensively examined, and its potential role has been suggested through multiple interrelated mechanisms, such as inflammation, vasomotor dysfunction, and lipid modifications[7-8]. For example, with regard to lipid metabolism, evidence shows that smokers always have an atherogenic lipid profile, with elevated levels of total cholesterol (TC), low-density lipoprotein (LDL) cholesterol (LDL-C) and triglyceride (TG) and attenuated levels of high-density lipoprotein (HDL) cholesterol (HDL-C)[9-12], suggesting a key role for smoking in lipid metabolism and ASCVD.

Recently, it has been proposed that lipoprotein particles can more accurately capture the atherogenic properties compared with cholesterol concentrations contained in the lipoproteins[13-16]. Previous studies have already indicated a negative impact of smoking on lipoprotein subfraction profiles, especially on HDL subfractions[17-19], even if there was no significant change in the serum levels of cholesterols[19]. The reversal impact of smoking cessation on lipid profile has also been documented in a meta-analysis[20]. However, there is not much information regarding the impact of smoking on lipoprotein subfractions, especially in individuals with smoking cessation. In addition, the mortality and morbidity of coronary artery disease (CAD) have rapidly increased in the young, which is paralleled with the elevated rate of current smoking in Chinese population[21]. This is also a reason due to which we performed the present study focusing on the impact of smoking status on the lipoprotein subfractions.

Apparently, intensive studies on the relationship between smoking and lipoprotein subfractions deepen the current understanding of the link between smoking and the risk of ASCVD[17]. Therefore, in the present study, we consecutively enrolled a relatively large Chinese cohort who were not under a lipid-lowering drug treatment and investigated the impact of current smoking on the distribution of lipid profile and lipoprotein subfractions.

METHODS

Study Design and Population

The present study fully complied with the Declaration of Helsinki and was approved by the Ethics Committee of FuWai Hospital and Cardiovascular Institute, Beijing, China. All included patients provided prior written consent.

This study recruited consecutive 1017 patients with angina-like chest pain. The exclusion criteria: 1) aged < 18 years; 2) with a treatment history of statins and/or other lipid-lowering drugs prior to entering the study; 3) severe end-stage diseases, such as renal and/or liver dysfunction, heart failure, and malignant carcinoma; 4) systematic inflammatory disease or severe infection; 5) thyroid disorder; and 6) pregnancy. The flow chart of patient recruitment is shown in Figure 1.

Patients with complete data were assessed for medical history, baseline clinical features, and traditional cardiovascular risk factors, and the final 877 eligible patients were subjected to analysis. Smoking status was confirmed using a questionnaire at the lipid clinics and/or during hospitalization[22]. Current smoking was defined as people who smoked at least 100 cigarettes in lifetime and were continuing smoking at the time of interview. Former smoking was defined as those who smoked at least 100 cigarettes in lifetime but had quit smoking at the time of interview. Never smoking was defined as people who never smoked or who have smoked less than 100 cigarettes in lifetime[23]. Type 2 diabetes was diagnosed if the repeated fasting blood glucose (FPG) level was ≥ 7.0 mmol/L and/or non-FPG level was ≥ 11.1 mmol/L or if the subject was currently taking oral hypoglycemic agents or receiving insulin therapy. CAD was diagnosed by elective coronary angiography as least one major epicardial coronary artery (≥ 2 mm) having a diameter of stenosis ≥ 50%. Hypertension was defined as blood pressure measurement of ≥ 140/90 mmHg in multiple determinations under different environments or patients taking antihypertensive drugs although the blood pressure was normal. Patients were divided into nonsmoking (n = 518), former smoking (n = 103), and current smoking (n = 256) groups.

Laboratory Analysis

Fasting venous blood was obtained and collected into ethylenediaminetetraacetic acid (EDTA)-containing tubes and subsequently stored at -80 °C for the measurement of lipoprotein subfractions. Concentrations of traditional lipid parameters (TG, TC, LDL-C, and HDL-C) and glucose levels were measured by an automatic biochemistry analyzer (Hitachi 7150, Tokyo, Japan). Using the