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ORIGINAL ARTICLES OR RESEARCH ARTICLES

Association between cigarette smoking and dyslipidemia

Association entre le tabagisme et la dyslipidémie

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KEYWORDS
Cigarettes smoking;
Lipid profile

Summary
Objective. — The purpose of this study was to examine the effect of cigarettes smoking on lipid profile.
Methods. — The initial study was conducted with 300 healthy subjects, among them, 138 non-smokers aged 38.47 ± 21.91 years and 162 smokers aged 35.55 ± 16.03 years. TG, TC, cHDL, cLDL were determined by enzymatic colorimetric method; ApoA1, ApoB, Lp(a) were determined by immunoturbidimetry on Konélab 30^{TM}.
Results. — We noted a significant increase in smokers compared to non-smokers, in TG (1.79 ± 1.03 vs. 1.40 ± 1.24 mmol/L; $P \leq 10^{-3}$), TC (4.13 ± 1.18 vs. 3.70 ± 1.04 mmol/L; $P \leq 10^{-3}$), c-LDL (1.35 ± 0.56 vs. 1.16 ± 0.61 mmol/L; $P \leq 10^{-3}$), Lp (a) (230 ± 226 vs. 179 ± 190 UI/L; $P = 10^{-3}$) and ApoB/ApoA1 (0.83 ± 0.52 vs. 0.52 ± 0.15; $P = 0.03$) and significant decrease in c-HDL (0.94 ± 0.25 vs. 1.07 ± 0.27 mmol/L; $P \leq 10^{-3}$).
TG values were higher in heavy than mild smokers (2.30 ± 0.96 vs. 1.63 ± 1.11 mmol/L; $P \leq 10^{-3}$) and c-HDL levels decreased particularly in heavy smokers. We found a strong correlation between TC, TG and c-LDL levels and consumption duration ($r = 0.957$, $r = 0.991$, $r = 0.954$; respectively).
Conclusion. — Cigarette smoking is associated with perturbations in lipid profile, especially low levels of c-HDL, increase of TG which can explain the atherosclerosis risk.

MOTS CLÉS
Tabagisme ;
Profil lipidique

Résumé
Objectif. — Le but de notre travail était d'examiner l'effet du tabagisme sur le profil lipidique.
Méthodes. — Notre travail a été mené auprès de 300 sujets volontaires en bonne santé, dont 138 non-fumeurs âgés de 38,47 ± 21,91 ans et 162 fumeurs âgés de 35,55 ± 16,03 ans. Les

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triglycérides (TG), le cholestérol total (CT), le HDL-cholestérol (HDL-c) et le LDL-cholestérol (LDL-c) ont été déterminés par une méthode colorimétrique enzymatique ; les ApoA1, ApoB, et la lipoprotéine(a) (Lp(a)) ont été déterminées par immunoturbidimétrie sur Konelab 30™.
Résultats. — Nous avons noté chez les fumeurs, par rapport aux non-fumeurs, une augmentation significative des TG (1,79 ± 1,03 vs 1,40 ± 1,24 mmol/L, $p < 10\text{-}3$), du CT (4,13 ± 1,18 vs 3,70 ± 1,04 mmol/L, $p < 10\text{-}3$), du LDL-c (1,35 ± 0,56 vs 1,16 ± 0,61 mmol/L, $p < 10\text{-}3$), de la Lp(a) (230 ± 226 vs 179 ± 190 UI/L, $p = 10\text{-}3$) et du rapport ApoB/ApoA1 ($p = 0{,}03 \pm 0{,}52$ vs 0,83 0,52 ± 0,15) ; et une diminution significative du HDL-c (0,94 ± 0,25 vs 1,07 ± 0,27 mmol/L, $p < 10\text{-}3$). Les valeurs moyennes de TG étaient plus élevées chez les grands fumeurs par rapport aux autres groupes (2,30 ± 0,96 vs 1,63 ± 1,11 mmol/L, $p < 10\text{-}3$) et le HDL-c était significativement plus bas chez les gros fumeurs. Nous avons également noté une corrélation entre les taux de CT, TG et LDL-c et l'ancienneté de l'exposition ($r = 0{,}957$, $r = 0{,}991$, $r = 0{,}954$;respectivement).
Conclusion. — Le tabagisme est associé à des perturbations du profil lipidique, en particulier une diminution du HDL-c et une augmentation des TG, ce qui peut expliquer le risque d'athérosclérose.

Introduction

Dyslipidemia is an abnormal level of lipids in the blood, referred to increases in the levels of total cholesterol, low-density lipoprotein (LDL) cholesterol, and triglyceride, and a decrease in high-density lipoprotein (HDL) cholesterol. Many previous studies reported that dyslipidemia increases the risk of cardiovascular disease [1]. Cigarette smoking is also an established risk factor for atherosclerotic cardiovascular disease, such as coronary artery disease, stroke and peripheral arterial disease. The effects of smoking on atherosclerotic progression result from multiple pathophysiologic mechanisms, including vascular injury, thrombosis, vascular dysfunction and lipid peroxidation. Smoking also has been reported to have atherogenic effect through abnormal lipid metabolism [2]. It is known that smoking causes an alteration in the serum lipoprotein profiles characterized by an increase in total cholesterol and triglycerides and a reduction in HDL cholesterol [1]. Smoking is the principal preventable cause of disease, disability, and premature death in industrialized countries. Inhalation of cigarette smoke is a leading causer of cancer, chronic obstructive lung disease, and cardiovascular disease. Premature coronary heart disease (CHD) is the most important medical consequence of cigarette smoking. Several mechanisms have been described to explain the deleterious effects of tobacco on the cardiovascular system. Among them, modifications of the plasma lipid profile have been well documented.

The purpose of this study was to investigate the effects of cigarette smoking on lipid profile perturbation.

Materials and methods

Study design

Population

This study was approved by the local ethical committee, and all subjects were of Tunisian origin. Our sample included 300 voluntary subjects: 138 non-smokers (62 men and 76 women) aged 38.47 ± 21.91 years and 162 current smokers (145 men and 17 women) aged 35.55 ± 16.03 years. Subjects with peripheral vascular disease, diabetes mellitus, renal disease, hepatic disease and hyperlipidaemia or hypertension or receiving any medication were also excluded. Subjects on a weight-control diet, morbidly obese (body mass index $\geq 35\ kg/m^2$), or using hypolipidemic drugs (i.e. statins, niacin, fish oil, etc) were also excluded. Written informed consent was obtained from all voluntary adult participants and from the parents of minors.

Samples

After a 12-hour overnight fasting, venous blood for each subject was drawn in tubes containing lithium heparinate, immediately centrifuged. The plasma samples were stored at −20 °C until the biochemical analysis. Urine samples were obtained from the smokers and non-smokers. These samples were either used the same day or frozen at −20 °C until required for analysis. All the samples were analysed for urine cotinine.

Methods

Smoking questionnaire

Subject information and cigarette smoking outcome data were collected in a structured interview. The available data were limited to the classification of smoking to two categories: never and current smokers. All subjects were questioned about their age, gender, and cigarette and alcohol consumption habits. Differences between patients and controls in terms of gender, body mass index (BMI), and alcoholic beverage consumption were noted. Therefore, these variables were considered as potential confounder factors for this analysis. The majority of subjects were able to provide information on the number of cigarettes they smoked and the duration of smoking. All subjects were questioned about their sociodemographic characteristics including age, gender, education and employment.

Biochemical assays

Lipid profile assay

Total cholesterol (TC), HDL cholesterol (cHDL) and triglycerides (TG) were determined by enzymatic methods, and