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Low arterial blood oxygenation is associated with calcification of the coronary arteries in patients with chronic obstructive pulmonary disease

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ABSTRACT

Background: Cigarette smoking is a well-known major cause of both chronic obstructive pulmonary disease (COPD) and atherosclerosis. However, few studies have investigated the correlation between COPD and coronary atherosclerosis.

Methods: We recruited 54 patients with stable COPD (51 men, 3 women) but without angina symptoms. Arterial blood gas analyses were performed, pulmonary function was assessed, and calcification of the coronary arteries was evaluated by computed tomography (CT).

Results: Calcification of the coronary arteries was noted in 25 patients. There were no significant differences in age, body mass index, respiratory function, and levels of low-density lipoprotein cholesterol, hemoglobin A1c, glucose, or C-reactive protein between patients with or without calcification of the coronary arteries. Arterial blood oxygenation was significantly lower in patients with calcification of the coronary arteries. On both univariate and multivariate analyses, low arterial blood oxygenation was an independent risk factor for calcification of the coronary arteries.

Conclusions: In patients with COPD, low arterial blood oxygenation was strongly associated with calcification of the coronary arteries and may be a significant predictor of cardiovascular disease.

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1. Introduction

Cigarette smoking causes a variety of respiratory disorders, including chronic obstructive pulmonary disease (COPD) [1]. COPD, a common disease characterized by varying airflow limitation levels, is globally the leading cause of death and a major medical problem in both Japan and worldwide [2]. A previous study showed that the prevalence of airflow limitation in Japanese people aged ≥ 40 years was ≥ 10% [3,4].

COPD was recently shown to be both a respiratory and systemic disease. Higher serum levels of pro-inflammatory cytokines such as interleukin (IL)-6, IL-8, and tumor necrosis factor (TNF)-α have been observed in patients with COPD [5–7]. Comorbidities such as lower body mass index (BMI), cardiovascular disease, osteoporosis, diabetes mellitus, and renal dysfunction are recognizes to occur more frequently in patients with COPD [2,8–10]. As we previously demonstrated, differences in genetic backgrounds among patients with COPD are involved in this exaggerated inflammatory response [11–13].

COPD and atherosclerosis are both strongly associated with cigarette smoking [14], and large numbers of patients with COPD die of cardiovascular disease [2]. Smokers with airflow limitations have increased subclinical atherosclerosis. Iwamoto et al. measured carotid intima-media thickness and focal atheromatous plaques as indicators of subclinical atherosclerosis [14]. It is important to evaluate atherosclerosis in patients with COPD to estimate the prevalence of cardiovascular disease. However, detecting ischemic heart disease in patients with COPD is difficult because their ventilation limitations during exercise constrain the cardiac demands [15]. Little is known about the burden of subclinical coronary artery disease in patients with COPD, and there have been few studies investigating the correlation between COPD and coronary atherosclerosis.

Despite being an invasive procedure, coronary angiography is commonly used to evaluate atherosclerosis of the coronary arteries. Multi-detector computed tomography (MDCT) is a non-invasive and reliable examination method that is useful for evaluating coronary artery stenosis or calcification [16]. Coronary artery calcification is associated with atherosclerosis [17] and predictive of cardiovascular events [18]. In this study, we evaluated coronary artery calcification in patients with COPD using 64-slice MDCT and analyzed the correlations between coronary artery calcification and clinical parameters in patients with stable COPD. The aim of this study was to identify predictive clinical factors for coronary artery calcification in patients with stable COPD who had neither history nor symptoms of angina pectoris such as chest pain.

2. Materials and methods

2.1. Subjects

We recruited 54 patients with stable COPD (51 men, 3 women) who had no angina symptoms and who had not had a disease exacerbation within the 3 months before the study. The study was approved by the Institutional Ethics Committee of Yamagata University School of Medicine (Approval date: October 21, 2009; Approved #: 21), and all participants provided written informed consent. COPD was diagnosed by using spirometry when the post-bronchodilator ratio of forced expiratory volume in 1 s (FEV_1)/forced vital capacity (FVC) was < 0.7 [19]. The Japanese Respiratory Society reference values were used [20]. Smoking habits were self-reported. The patients' profiles are summarized in Table 1. Six subjects were receiving long-term oxygen therapy (LTOT).

2.2. Laboratory data

Clinical information was obtained from the patients' medical records. Peripheral blood counts, liver and renal functions, and levels of glucose, glycated hemoglobin A1c (HbA1c), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides (TG) were measured using routine laboratory techniques. Hypertension was diagnosed as a systolic blood pressure ≥ 140 mmHg and/or diastolic pressure ≥ 90 mmHg or the current use of anti-hypertensive drugs. Diabetes mellitus was diagnosed as a fasting plasma glucose concentration ≥ 126 mg/dL, HbA1c level ≥ 6.1% (Japan Diabetes Society), or current use of insulin or hypoglycemic agents. Dyslipidemia was diagnosed as concentrations of LDL-C ≥ 140 mg/dL, TG ≥ 150 mg/dL or HDL-C < 40 mg/dL, or current use of anti-hyperlipidemic drugs. Blood samples were obtained from the femoral artery of the subjects without LTOT on room air and from subjects with LTOT inhaling their usual flow of oxygen. Blood gas levels were measured after rest in the supine position for over 10 min.

2.3. Evaluation of coronary artery calcification

Cardiac multi-detector computed tomography (MDCT) was performed using a 64-slice MDCT scanner (Aquilion 64; Toshiba, Tokyo, Japan). A total of 51–100 mL of contrast media (Iopamidol; Bayer Co. Ltd., Leverkusen, Germany) was injected at a flow rate of 3.0–4.6 mL/s depending on the patient's body weight. The region of interest (ROI) was placed within the ascending or descending aorta, and scanning was commenced when the CT density reached 250 Hounsfield units (HU) at the ascending aorta or 180 HU at the descending aorta. Scans were performed between the diaphragm and the tracheal bifurcation (collimation width, 0.5 mm; rotation speed, 0.4 s/rotation; tube voltage, 120 kV; and effective tube current, 400–450 mA). Cardiac images were evaluated at the most motionless phase of the cardiac cycle, which was most frequently the mid-diastolic phase, with retrospective cardiac gating at 75% of the R-R interval. Calcification of the coronary arteries was evaluated as lesions composed exclusively of structures with a CT density greater than that of the enhanced coronary lesions [16,21].

2.4. Statistical analyses

All data are expressed as means ± SD except for C-reactive protein (CRP) level (expressed as median [95% confidence interval]). The Mann–Whitney *U*-test or chi-square test was

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