Acute beneficial effects of smoking cessation on coronary flow reserve: a pilot study


Departments of Chest Disease, *Cardiology and **Sports Medicine, Faculty of Medicine, Istanbul University; Istanbul-Turkey
1Clinic of Cardiology, American Hospital; Istanbul-Turkey
2Department of Chest Disease, Faculty of Medicine, Acıbadem University; Istanbul-Turkey

Smoking is one of the most common causes of cardiovascular (CV) mortality (1). Besides its atherogenic effects, smoking may also lead to acute fatal CV events such as ventricular fibrilation and sudden death as a result of acute sympathetic and hemodynamic responses (2).

The oxygen demand of the myocardium may lead to increase in the coronary blood flow up to 4-6 folds via vasodilatation. Coronary flow reserve (CFR) is a parameter, which shows the status of epicardial coronary arteries and the microvascular coronary circulation by reflecting the coronary vasodilator capacity. Moreover it is an important marker for cardiac morbidity and mortality (3). CFR is defined as the ratio between hyperemic peak and basal peak diastolic coronary flow velocities (CFV). A CFR value ≤2.0 is generally considered abnormal (4). Transthoracic Doppler echocardiography (TTDE) of mid-distal left anterior descending artery (LAD) during vasodilator pharmacologic stress test is one of the recommended techniques (5).

Smoking immediately decreases CFR and increases coronary resistance even in healthy subjects. Thus the risk of acute myocardial ischemia increases especially in the patients with either high-risk or previously known coronary disease (6). On the other hand, the cessation of smoking is associated with a rapid improvement in general physiology, with the fastest improvement occurring in the CV system (7, 8). However, no accurate data demonstrating the time of onset of this improvement and the impact of smoking cessation on CFR variability is available. Therefore we primarily aimed to assess the acute effects of smoking cessation on CFR.

Twenty apparently healthy male smokers between 20 and 50 years of age, admitted to the Nicotine Dependence Centre willing to quit smoking and 10 non-smoking healthy males as a control group were included in our prospective controlled pilot study. The Local Ethics Committee approved the study protocol and the subjects all gave signed informed consent before participation in the trial. The exclusion criteria were the presence of poor quality of echogenicity, established obstructive airway disease, diabetes, migraine, ischemic heart disease, hypertension (≥140/90 mm Hg), and arrhythmia, impaired functional capacity for any reason (class II or more according to New York Heart Association), recent use of any vasoactive medication, and obesity (BMI >30 kg/m²). Following a complete routine echocardiographic study (GE vingmed, Ge-Vivid 7 Pro, General Electric, Florida, USA), CFV was measured at mid-distal LAD in all subjects by TTDE at baseline and during intravenous dipyridamole infusion (0.56 mg/kg in 4 minutes) thereby CFR was calculated. For a period of 14 days, close follow-up was conducted via phone calls and face-to-face interviews to detect the recurrence of smoking in the study group. No medical treatment for smoking cessation was used due to its potential impact on the study results. On the 14th day, the levels of exhaled carboxyhemoglobin (CO) were measured using a carboxymeter (pico Smokerlyzer Breath CO Monitor Bedpoint Scientific, USA) and the subjects whose values were less than 10 ppm were considered “not to have smoked”, and the procedure was repeated in those cases.

Only fourteen subjects (70%) could successfully complete the 14-day smoking-free period (mean exhaled CO level was 3.61±2.15 ppm). The demographic findings of the participants have been summarized at Table 1. All baseline echocardiographic studies were normal in both groups with normal ejection fraction (>55%). Furthermore no segmental left ventricular wall motion abnormality was observed after dipyridamole infusion, although one patient in the control group had experienced spontaneously regressing angina. The mean CFR values at baseline and 14th day of not-smoking period were 2.03±0.44 and 2.26±0.59 in the study group respectively, and 2.49±0.71 in the control...
The data regarding the impact of smoking on CFR is conflicting. The different results can be attributed to differences in age and gender of the trial populations, differences in the methods for measuring CFR, or the use of different stress agents (adenosine or dipyridamole) (6, 9). However, in a recent twin study, researchers found a significant decrease in the CFR values of the smokers comparing to non-smokers probably because of the chronic effects of smoking (10). Our result regarding the lower CFR values in the smoker group is consistent with the previous literature, although the difference was not statistically significant (p=0.382) (Table 2).

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As a conclusion, we assessed, for the first time, the acute effect of smoking cessation on CFR, which is a good indicator for the CV events. The small number of subjects is the primary limitation of this study. In addition, the relatively young study population who has not any CV risk factors other than smoking might not fully reflect the overall smoker population. However the CFR values mildly increased after two-week smoking-free period in the study group. Although this increase could not reach statistically significance, it is a promising result, which provides further evidence of the acute, favourable effects of smoking cessation on the CV system. We believe that this benefit should be considered as a motivating factor for clinicians and patients in the fight against smoking. But large-scale prospective studies are still needed for a definite conclusion.

Conflict of Interest: None declared.

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References