To the Editor:

Drs Shimada et al recently conducted a compact study on the effect of smoking cessation on blood viscosity using various parameters relating to cardiovascular risk. They also presented the relationship between scores on a self-rating depression scale (SDS) and blood passage time (BPT) using microchannel array flow analyzer (MC-FAN). They presented the level of association between blood viscosity and physical or mental parameters, including smoking information. I wish to support the significance of their report from 2 aspects (handling of psychological factors and the smoking cessation program), and also make suggestions on improving their outcome.

Concerning affective disorders, Clark and Watson developed the tripartite model of anxiety and depression, which identifies negative affect (NA), anhedonia and low positive affect (PA), and anxious arousal (AA). Using this model, Ameringer and Leventhal recently summarized the association between tripartite affective dimensions (NA, PA, and AA) and smoking variables using smoking status, heaviness, chronicity, dependence and cessation. They concluded that each dimension has a differential effect on smoking. Rozanski et al also made a precise review on the association between psychological factors, including depression, and the pathogenesis of cardiovascular disease.

Lifestyle modifications, including smoking cessation, are effective in controlling cardiovascular risk, and a shift toward healthy behavior could lead to a reduction in the incidence of metabolic components such as diabetes mellitus. Schipf et al reported that current smoking prevalence in subjects with type 2 diabetes was lower than those without type 2 diabetes in Germany, but I obtained different information. As I recognize that a smoking cessation program should be strictly applied to patients with diabetes, precise data are presented as follows.

A cross-sectional study was conducted twice with a more than 3-year interval. Phase 1 was a prevalence study of diabetes and smoking in male and female subjects from 2004 to 2005, and the study was repeated from 2008 to 2010 (Phase 2). A total of 2,523 subjects (1,932 males, 591 females) in Phase 1 and 4,188 subjects (3,269 males, 919 females) in Phase 2 were enrolled. Mean age (standard deviation) in Phases 1 and 2 was 51.4 (9.3) and 52.6 (9.9) years, respectively. Diabetes was diagnosed according to the standard criteria in Japan in combination with oral glucose tolerance test and HbA1c level.

Current smoking was defined as regular cigarette smoking at the time of the interview. The project was approved by the ethical committee of the Institutional Review Board of Ota General Hospital, Gunma Prefecture, Japan (July 17, 2010). Informed consent was given by each participant.

The percentage of current smoking in male diabetic and non-diabetic subjects in Phase 1 (Phase 2) was 45.6 (35.8) % and 38.8 (33.3) %, respectively. The percentage of current smoking in respective female subjects was 11.1 (7.9) % and 7.6 (5.9) %. Diabetic subjects showed a higher percentage of smoking, although there was no significant difference in the percentage of smoking between diabetic and non-diabetic subjects.

Although the reason for the discrepancy between the gap in the prevalence of current smoking in diabetic and non-diabetic subjects in Germany and in Japan cannot be explained clearly, urgent intensive health guidance to stop smoking by diabetic patients is needed to prevent cardiac complications in Japan.

Considering the previously mentioned points relating to cardiovascular risk, I want to make 3 comments on the outcome found by Shimada et al.

1. They rounded BPT values over 120s to 120 for statistical analysis. But as presented in their Figure 1D, for example, the correlation between BPT and SDS score was apparently negative when the rounded values were excluded. The same trends were also observed for daily consumption of tobacco (Figure 1A) and Brinkman’s Index (Figure 1C). The handling of outliers can create different results, and the distribution of parameters including BPT should be checked.

2. The explanation rates by (stepwise) multiple regression analysis, which was presented by R square value, are required to understand the level of prediction in Table 1. There is a possibility that the lower R value means a lack of associated factors on blood viscosity such as blood hematocrit etc.

3. SDS was not developed to judge the severity of depression but to know the level of depressive state. As the authors also mentioned, the pathophysiology of depressive state is complicated and many factors should be considered simultaneously. Simple linkage between BPT and depressive state runs the risk of misunderstanding.

Disclosure

I declare that I do not have any conflict of interest regarding this study.

References


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