Treatment of coronary spasm in patients with the -786C allele

Strict follow-up is necessary in coronary spasm patients with the -786C allele to monitor for reattack and/or acute myocardial infarction. Additional medications like long-acting CCB and/or nitrite and/or other antiangina agents, should be prescribed for coronary-spasm patients with the -786C allele; however, there was no significant difference with the readmission rates between those with long-acting CCB and those with short-acting CCB in this study.

It was revealed that the incidence of readmission due to a reattack of coronary spasm was significantly higher in patients who were administered two CCB compounds than in patients who were administered one CCB compound; moreover, all the readmission patients with two CCB compounds carried the -786C allele. We usually medicate a patient who has severe and/or medicine-resistant coronary spasm, with a combination of two CCB compounds. These results indicate that a combination of two CCBs is effective in patients without the -786T/C polymorphism, but is not effective in the severe coronary-spasm patients with the -786T/C polymorphism. Additional medications such as HMG-CoA reductase inhibitor, ACE-I, or angiotensin II type 1 receptor blocker are possibly needed in the patients with severe coronary spasm with the -786T/C polymorphism.

We recently reported that fluvastatin, an HMG-CoA reductase inhibitor, increases the transcriptional activity of the eNOS gene in the endothelial cells, especially in those with the -786C allele [16]. We therefore suggested that fluvastatin possibly prevents reattacks of coronary spasm, especially in patients with the -786C allele. It was reported that ACE-I or angiotensin II type 1 receptor blocker induces eNOS bioactivity; therefore, those drugs possibly are effective in the patients with coronary spasm [17,18]. A further clinical study is, however, necessary to verify this.

Study limitation

It has been previously suggested that the pathogenesis of coronary-artery spasm is closely related to the process of atherosclerosis [19,20]; however, in this study, there were relatively few patients who were readmitted owing to a progression of coronary stenosis. A longer follow-up period might be necessary to elucidate whether the incidence of patients with coronary stenosis will increase. Also, there may be racial, and/or environmental, and/or lipid-profile differences in the pathogenesis of atherosclerosis. A study on a larger follow-up population will be beneficial to further elucidate this topic.

The -786T/C polymorphism has only a modulatory role in the development and the recurrence of coronary spasms, which possibly also occur in some patients with angiographically detectable stenoses; therefore, further study will be necessary to elucidate other predictive factors for coronary spasm.

Conclusion

Considering the strong association of the C allele of the eNOS gene -786T/C polymorphism with the prognosis for coronary spastic angina patients, we conclude that the -786T/C polymorphism is an independent predictor for readmission in patients with coronary spasm. The -786T/C polymorphism of the eNOS gene is an important factor to consider in determining the clinical course of coronary spastic angina. A strict follow-up is necessary in coronary-spasm patients with the -786C allele. There is no simple test to measure for the -786T/C polymorphism at present; however, if a test is developed in the near future, it will be valuable for treating patients with coronary spasm, especially those with -786T/C polymorphism.

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