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poor correlation of leptin with CAD. This has been also reported in a recent meta-analysis and systematic review by Yang *et al.* [10] which found no significant correlation between leptin and risk of CAD and stroke.

Considering CAD as a chronic multifactorial disease, the relationship between serum leptin levels and classical CAD risk factors can also be helpful in further understanding the correlation between leptin and CAD. The study conducted by Bickel *et al.* [11], which was performed on patients who were referred for angiography with at least 30% stenosis in one major coronary artery, also showed significant influence of clinical variables of age, body weight and renal function on leptin concentration. Elevated leptin level has been shown to be related to insulin resistance, type 2 diabetes mellitus and subsequent increased cardiovascular risk [24,25]. Hypertension has also been widely investigated and revealed a positive association with serum leptin level [26]. However, our study showed no significant differences in mean serum leptin level between the diabetic and nondiabetic, and also hypertensive and nonhypertensive patients. Other cardiac risk factors in this study also showed no statistically significant correlation with leptin. These results of subgroup analysis, which could be partly explained by limited sample size, are in agreement with our main finding. Therefore, it can be indicated that the correlation of leptin and evidence of CAD might be associated with the rate and prevalence of other risk factors in the population.

Finally, it should be stated that perfusion abnormalities on the MPI SPECT mostly imply the functional and hemodynamic significance of anatomical coronary artery stenosis. Thus, an abnormal scan result could not be totally considered to be the same as angiographically proven CAD [27]. Consequently, our results should not be directly compared with those studies that used angiography as the diagnostic test. According to the purpose of our study, however, our result suggested that leptin is not beneficial enough to further stratify suspected patients noninvasively and is not correlated with the MPI SPECT result as an established method.

This study has also some limitations. Although the total sample size was acceptable, the subgroups of different cardiac risk factors in the two sexes were relatively small for analysis. In addition, MPI SPECT is not a gold standard in the diagnosis of CAD, and the possibility of interfering image artifact and subsequent misinterpretation is inevitable. Further research studies with larger samples and other modalities are required to evaluate the predictive role of leptin in suspected CAD patients.

Conclusion

According to our results, it seems that serum leptin level is not correlated with perfusion abnormalities on MPI SPECT and consequently CAD in suspected CAD

patients. Although more dedicated prospective studies are still needed, this might be somewhat related to the prevalence of other cardiac risk factors that are influenced by leptin in their pathogenesis.

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Conflicts of interest

There are no conflicts of interest.

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