Lower FXII activity and thrombosis: a comment

Düşük FXII aktivitesi ve tromboz: bir yorum

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To the Editor,

I read the recent publication by Rasighaemi et al. with great interest. Rasighaemi et al. noted that "Lower FXII activity is not a risk factor; rather, it simply represents a risk marker for thrombosis [1]." I have some concern regarding this work. First, there is the question of whether or not this is a good case-control study. The authors stated that a matched control group was used; however, the large discrepancy between the number of cases and controls makes it difficult to believe that good matching was actually performed. Second, a polymorphism is a population–scale phenomenon; hence, it is questionable whether the rather low number of subjects in this work can be statistically acceptable to answer the research question. Third, there is no evidence to conclude that lower FXII activity represents a risk marker for thrombosis. To conclude this, the complete set of diagnostic test evaluations (sensitivity, specificity, accuracy and predictive value) must be performed.

References

1. Rasighaemi P, Kazemi A, Ala F, Jazebi M, Razmkhah F. Association of FXII 5'UTR 46C>T polymorphism with FXII activity and risk of thrombotic disease. Turk J Hematol 2010;27:15-9.

Author Reply

Although in case-control studies, it is better to have the same number of controls and cases, or more controls than cases, it is not an obligation. If we review the literature, there have been some studies in which control numbers were less than case numbers, such as in the study of Altieri et al. [1], which included 122 patients and 45 controls. In the present study, the controls and cases were not matched for numbers, but they were matched for other important factors that may influence a study's results and judgements, such as gender and age; furthermore, none of them had cirrhosis, nephritic syndrome or cancer. The issue of the number of cases and controls was also consulted upon with a statistical specialist, and based on our population and study, we selected the number of cases and controls that would give us reliable results to judge. Nevertheless, we accept that in a polymorphism study, a higher number of samples would result in more reliable and accurate findings.

Nearly all of the recent studies have agreed upon the great importance of the C46T genotype on FXII level. For example, in our study, not only controls but also patients had lower FXII activity in the TT genotype compared with the CT and CC genotypes. Given that there was no association between TT genotype and thrombosis risk, and the importance of the C46T polymorphism on FXII activity, we speculated that the reduced FXII activity in patients with venous thromboembolism is caused by consumption of FXII in both coagulation and fibrinolysis upon thrombosis formation, or by other agents produced in thrombotic patients that can affect FXII activity. Thus, one of the possible explanations for this finding could be consideration of FXII as a risk marker, which is a consequence of thrombosis.

Sincerely,
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References

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doi:10.5152/tjh.2010.13