Journal of Geriatric Cardiology (2016) 13: 471–472

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Platelet reactivity in patients with atrial fibrillation

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J Geriatr Cardiol 2016; 13: 471–472. doi:10.11909/j.issn.1671-5411.2016.05.009

Keywords: Atrial fibrillation; Heart rate; Platelet reactivity; Thromboembolism

We read the article entitled 'Gender and tachycardia: independent modulation of platelet reactivity in patients with atrial fibrillation' with great interest. In this article Procter, *et al*.[1] reported that gender and heart rate are independent determinants of platelet function in patients with acute atrial fibrillation (AF). The authors pointed that female sex correlated with impaired nitric oxide (NO) responses independent of platelet aggregability and admission heart rate. However, we have some suggestions about this study: there was no control group and all patients included were > 45 years old; also, mean platelet volume (MPV) is not investigated in study group.

It is known MPV is simple and inexpensive to obtain, easy to interpret, and routinely measured by automated cell counters. As compared with other markers of platelet activity, which require specialized equipment, MPV is a practical and prognostically important biomarker of cardiovascular disease.[2]

Furthermore, it has been shown that MPV has a predictive value for the presence of thrombotic event in patients with AF.[3]

In conclusion, it would be better to be investigated MPV level by authors. But we celebrate the authors due to their nice study.

References

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Authors' reply

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We thank the researchers concerned for their interest in our work on the relationship between gender and tachycardia as modulators of platelet NO response in patients with AF.

As regards the issues which they raise: (1) Lack of control group. The study related to intra-group variability in potential thrombotic diathesis in a complete cohort of AF patients.[1] It is hard to imagine why a control group would be desirable for this exercise, and indeed what would constitute an appropriate control group.

(2) Patients were > 45 years old. We do not understand this criticism. As AF affects almost entirely aging individuals, we would not expect to find many patients younger than 45 years of age. Indeed, if we included mainly young patients, the study would have been non-representative.

(3) MPV, an index of thrombotic risk in AF, was not measured.[2] We thank the researchers for their interesting suggestion. Unfortunately, we did not measure MPV.

Reading the report of Xu, *et al*.,[2] we note that the study was a comparison of 57 control AF patients with 57 post-thrombotic AF patients. In other words, this was a *post hoc* study. It is open to two potential interpretations: either increased MPV predisposed to thrombosis, or thrombosis predisposed to increased MPV. Furthermore, given that thrombosis only occurs in a minority of AF patients, it seems inappropriate to claim predictive value using equal numbers of patients/controls in a retrospective cohort study. Finally, large platelet size is a marker of recent platelet release from the marrow.[3] We have shown that impairment of NO signaling occurs most frequently in acute, rather than chronic, AF.[4] Potentially, the bases for acute platelet release and for impaired NO signaling may exhibit commonalities.

References

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