Effects of obstructive sleep apnea and atrial fibrillation on blood pressure variability

To the Editor,

We have read with great interest the article published by Taher et al. (1), which was about the effects of blood pressure variability (BPV) on cardiovascular outcomes of patients with hypertension. It is impressed in the article that increased BPV is associated with increased future cardiovascular events (1).

Obstructive sleep apnea (OSA) is defined as the occurrence of the complete or partial obstruction of airways during sleep. OSA is common in overweight and obese people, and it is associated with increased rates of cardiovascular events, including coronary artery disease, heart failure, pulmonary hypertension, stroke, and atrial fibrillation (2). During night time, blood pressure usually decreases to nearly 10%–20% of the daytime values due to increased vagal tonus, and this situation is described as "dipping". In patients with OSA, blood pressure may not decrease at night and may even remain similar to that at day time. Therefore, OSA leads to increased BPV (3). In the present study, some of the participants were obese, and OSA might be present in a part of the study population.

Atrial fibrillation is the most common chronic arrhythmia in the general population. Cardiac output differs by beat-to-beat in patients with atrial fibrillation, and this condition causes beat-to-beat BPV (4). The hypothesis that BPV is related to increased risk for new onset atrial fibrillation is conflicting. Although it is reported in a meta-analysis that BPV is not associated with increased risk of new onset atrial fibrillation, there are some studies demonstrating the positive correlation between BPV and atrial fibrillation development (5).

To conclude, OSA and atrial fibrillation are frequent diseases and they are related to both BPV and future cardiovascular events. Therefore, we think that it could be better if the presence and effects of these comorbidities were also evaluated in the study.

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Author`s Reply

To the Editor,

We would like to thank authors for their interest in our paper (1) and their valuable comment.

The main aim of our study (1) was to know which method of blood pressure variability assessment is better in predicting the complications; thus, our inclusion and exclusion criteria as well as the study design were selected to answer this main question. We did not collect data regarding obstructive sleep apnea (OSA) or atrial fibrillation. However, we could figure out the prevalence of obesity in our data as it is relatively related to OSA. The prevalence of obesity in our data was 16.4%. Interestingly, after applying Mann–Whitney U test, we found that obese patients are more likely to present with high systolic BPV in their visit-to-visit measurements with a significant p-value of 0.03.

We think such comments open a new area of research to find other possible causes of high BPV as few studies had tackled this issue.

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How to improve the management of a patient with heparin-induced thrombocytopenia?

To the Editor,

Heparin-induced thrombocytopenia (HIT) is an adverse drug reaction caused by immunoglobulin G platelet-activating antibodies against platelet factor 4 (PF4)/heparin complexes, leading to venous and arterial thromboembolism (1). I read with keen interest the case report describing a fatal case of probable HIT in a young man who experienced pulmonary embolism (PE) and concomitant deep vein thrombosis (2). The case of this patient with intracardiac thrombus formation and severe ischemic stroke highlights the high risk of thromboembolic events in patients with HIT despite anticoagulant treatment with fondaparinux (5 mg/d), which was ineffective in case of the patient even when the platelet count increased to 150.000/uL. However, without any description of the patient's weight, it remains unclear whether the dosage of fondaparinux was appropriate. The use of vitamin K antagonist (VKA) after normalization of the platelet count, along with the administration of low-molecular-weight heparins or non-VKA oral anticoagulants immediately after the diagnosis of PE in a hemodynamically stable patient could lower the risk of HIT development and significantly improve the prognosis (1, 3). The rationale for choosing unfractionated heparin (UFH), the most common cause of HIT, in the patient was not presented.

In 2018, we had reported our experience with the diagnosis and management of patients suspected of having HIT (4). We have also observed a male patient with PE who was heterozygous for factor V Leiden, as in the present case; was receiving UFH; and was found to have intracardiac thrombi at diagnosis; however, fondaparinux (7.5 mg/d) was effective in that patient (Undas unpublished data). A major limitation of this report is the lack of laboratory confirmation of HIT. On the basis of our experience, we consider that the most commonly used anti-PF4/heparin antibody enzyme immunoassays can frequently detect clinically irrelevant antibodies, with a risk of overdiagnosis. However, a high OD value above 2 well correlates with the positive results of specific assays, e.g., a platelet serotonin-release assay, and such assays can be used in low-income countries such as Poland and Turkey (1, 4). Considering other strong prothrombotic factors, authors did not present conclusive evidence for the absence of occult cancer, which might contribute to the resistance to the anticoagulant used (5). Autopsy could clarify such uncertainties. This interesting report supports not using UFH as a first-line therapy in most PE patients and treating PE vigourously to prevent life-threatening thrombotc events, including HIT, especially in young patients without serious comorbidities.

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Author`s Reply

To the Editor,

We would like to thank the authors for their interest in our article titled "Mitral valve and right ventricular thrombi possibly caused by heparin-induced thrombocytopenia" and for taking