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Letter to the Editor

Histology of human right atrial tissue in patients with high-risk obstructive sleep apnea: More work needed☆

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Dear Editor,

We read with interest the letter by Keymal et al. [1] who commented on our recent publication [2] wherein we investigated right atrial tissue in patients undergoing coronary bypass grafting for histological changes potentially associated with obstructive sleep apnea (OSA). We summarized that there were no differences observed between patients at high or at low risk for OSA. We thank these authors for their interest in our work and for their meaningful feedback. We would like to briefly address some of their concerns.

There was concern for the utilization of the Berlin Questionnaire (BQ) for evaluation of the presence of OSA. Several studies have validated the BQ in identifying patients at high risk for OSA [3–5]. A systematic review assessing various screening tools of OSA found that the BQ had a pooled sensitivity of 77% and a pooled specificity of 74% [6]. While polysomnography remains the gold standard, given that it is expensive and not widely available the BQ can reasonably categorize patients as high- or low-risk for OSA. We have thus used the BQ as a diagnostic correlate for OSA, openly recognizing the limitations in this approach, including the limitation that OSA severity (via apnea–hypopnea index) could not be evaluated.

We agree with the authors that OSA is associated with an increased incidence of atrial fibrillation (AF) and in fact our group has acknowledged and investigated this association thoroughly in the past [7–10]. AF induces atrial remodeling at a structural, microstructural, biomolecular, electrical, and ionic level [7]. In order to determine that histological changes were indeed induced primarily due to OSA, and not to pre-existent AF, we chose to exclude patients with AF in our study. We appreciate that this could exclude patients with the greatest microscopic morphological tissue changes, given the macroscopic manifestation of an atrial arrhythmia, however we did not want to confound any positive findings in this small population with multiple etiologies that might explain any observed histological changes.

In conclusion, we appreciate the limitations of our small pilot study, and despite the lack of any significant findings, we agree with the authors' sentiment that in a larger, more robust study, histological changes in human atrial myocytes induced by OSA may yet be observed. We look forward to further studies in this field.

Conflict of interest

The authors have no disclosures or conflicts of interest to declare.

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