Author's Reply

Obstructive Sleep Apnea Hypopnea Syndrome: An Incognito Player Contributing to Repeated Pulmonary Embolism?

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Thanks for your reading and priceless comments to our recent publication.[1] The major concern is that whether OSAHS is an independent risk factor of PE, or just a companion of other diseases which may lead to thrombotic lesions, such as diabetes, hypertension, smoking, obesity, etc. Actually the independent relationship between OSAHS and PE has already been revealed,[2-4] although the pathophysiological mechanism is far from being determined. As to the repeated PE, yet no literature is available pertaining to its association with OSAHS. Independent prognostic value of OSAHS cannot be concluded from current study because of the limited cases of recurrent PE ($n = 10$) and thus the statistical irrationality of adjusting for the traditional risk factors mentioned in the above letter as confounding variables in Cox proportional hazards model. Given the high prevalence of OSAHS and the high morbidity and mortality associated with PE, large-scale studies are warranted.

The international normalized ratio and warfarin dose is influenced by many factors, such as medication, which is a challenge for both researchers and clinicians. Exclusion of all potential influential factors is not logistically feasible during the follow-up, although randomized controlled study recruiting patients with minimum influencing factors in the baseline is an ideal research state. It is judicious to keep a stable diet rather than change the kinds and amounts of vegetables one typically consumes. Not only for the purpose of study, but also for clinic safety, the pamphlet of warfarin self-management strategy and food instructions were routinely given to patients, while the coagulation condition has been monitored closely by the investigator.

Whether CPAP, the golden strategy to treat OSA, will contribute to the prophylaxis of thrombotic issues is a super interesting topic, which is currently under investigation. To the best of our knowledge, CPAP has been proved to possess the potential to reduce hyper-coagulation state in patients with OSAHS.[5-7] However, because warfarin dosage depends largely on genetic background, it is not sure that whether CPAP users, whose hyper-coagulant state may be reversed to some extent, need reduced dose of warfarin, or whether the risk of bleeding might be increased when patients initiate CPAP and thereby changing the previous stable coagulant state. More clues will be revealed in future study.

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