showed that children with congenital heart disease are susceptible to some serious infections, particularly respiratory tract infections, endocarditis, and brain abscess. Moreover, these children do not have the ability to form effective antibodies to withstand these infections. In 1968, Dr. DiGeorge initially reported about the T cell dysfunction among children with congenital heart disease, and the immunological characteristics of these children are cell-mediated (thymic-dependent) immune deficiency with reduced numbers and function of T cells, antibody deficiency, and even neutrophil dysfunction (4). In a recent report, a generally reduced lymphocyte count was observed among NCP patients, as the mean lymphocyte count [0.88 (0.6–1.2), $\times 10^9$/L] is below the reference ranges (1.0–3.3, $\times 10^9$/L), which seems to be an inadequate immune response of those infected with SARS-CoV-2 (5). Although there are no evidences supporting that children are susceptible or there is also no reported high proportion of cases among children, children with congenital malformations must be given enough attention. These children with congenital immune deficiency and congenital heart disease might be challenges in the process of herd immunity (community immunity) and vaccination.

In hospitalized NCP patients, emerging cardiovascular damages can be diagnosed accurately using clinical judgment, ECG, X-ray, Doppler ultrasound, cardiac magnetic resonance imaging detections, etc. However, for some early mild lesions including vascular endothelial damage and cardiac valve lesions (mild regurgitation of blood via the cardiac valve, changes of valve softness and elasticity) cannot be assessed using traditional measures. These occult lesions will add to the risk of coronary atherosclerosis, hypertension, and heart valve disease in the future. Lessons from the viral myocarditis and cardiac rheumatic/degenerative valve diseases revealed that these diseases with the feature of gradual progression have no significant cardiac dysfunction and clinical symptoms in the early stage, but with the passage of time, the heart and fibrous rings of the heart valve will expand progressively, which can lead to structural cardiac diseases. Therefore, close follow-up is necessary for discharged patients with NCP.

Therefore, this short study discussed several additional issues regarding cardiovascular injury in patients with NCP, which have never been mentioned before. This study aimed to extend the perspective how to control NCP-related cardiovascular damages. Thus, further investigation regarding these issues is necessary.

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The association of hypertension with obstructive sleep apnea and polysomnographic features

To the Editor,

I read with great interest the article entitled, "Clinical and polysomnographic features of hypertension in obstructive sleep apnea: A single-center cross-sectional study" by Gürün Kaya et al. (1) published in Anatol J Cardiol 2020; 23: 334-41. They found that age, Epworth sleepiness scale, oxygenation parameters, and apnea duration are related to hypertension (HT) in patients with obstructive sleep apnea (OSA). This study strengthens earlier research that OSA is associated with HT and cardiovascular diseases (2, 3). The authors declared that the more OSA causing sleep disorders associate with the greater hypertensive response. However, the study has some methodological issues, ignoring the fact that prehypertensive or normotensive patients with OSA may have increased arterial stiffness, endothelial dysfunction, and excessive sympathetic response, irrespective of their age, sex, and other comorbidities. The percentage of patients with OSA with prehypertension or masked HT is not low in the population with OSA (4, 5). The body mass index of the normotensive group was lower than that of the hypertensive group. Variables including confounding factors, such as diabetes mellitus, smoking, hyperlipidemia, or drug use were not considered. Therefore, the study's findings were suspected to provide an additive prediction power of OSA causing polysomnographic sleep disorders to identify the possibility of hypertension in patients with OSA. The study results could have been more validated if

they had included prehypertensive and masked groups, as well as confounding factors for hypertension.

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Author`s Reply

To the Editor,

We would like to thank the authors of this letter for their comments on our study (1). We agree that the normotensive patient group of our study population may include participants with prehypertensive or masked hypertension; these patients may tend to have excessive sympathetic response. As we mentioned in the method section of our study, patients with hypertension (HT) were defined as those with an established diagnosis of HT and ongoing antihypertensive treatment for at least 3 months based on patient self-reports confirmed using the electronic national medical record system. Patients' blood pressure was measured before and after polysomnography (1). To diagnose HT and monitor blood pressure, measurements should be obtained two or more times for at least two separate visits or monitoring with ambulatory or home blood pressure monitoring (2, 3). However, we did not have the appropriate equipment to continuously monitor blood pressure in this way. Thus, we could not define patients with prehypertension or masked HT.

HT and obstructive sleep apnea (OSA) do not only have common risk factors, such as obesity, dyslipidemia, diabetes, and smoking, but also common pathophysiological features, including endothelial dysfunction, systemic inflammation, and sympathetic activation. These findings are thought to be result of intermittent hypoxia and reactive oxygen species production in OSA (4, 5). Besides that, arterial stiffness may result from aging and HT (6). Although endothelial dysfunction, arterial stiffness, and sympathetic activation are highly associated with HT, those can also be detected in patients with OSA without HT, and those may be consequences of OSA and intermittent hypoxia-related sleep disorders (4, 6, 7). We concur with the authors of the letter that defining patients with those factors in the normotensive group would be beneficial to validate the results more for risk for HT. However, considering that the normotensive group includes patients with masked HT and prehypertension with endothelial dysfunction or arterial stiffness, the differences with the hypertensive group become more significant.

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