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OPINION

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Obstructive sleep apnea and left ventricular strain: Useful tool or fancy gadget?

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Obstructive sleep apnea (OSA) syndrome is the most common sleep-breathing disorder, which is characterized with snoring, interruptions in breading during sleeping, or daytime sleepiness, and choking or gasping during sleep. Polysomnography defined OSA as 5 or more episodes of obstructive apneas/hypopneas per hour during sleep.[1,2] There are many risk factors for OSA, but the most important is obesity, and therefore, many studies are devoted to the assessment of the influence of weight reduction in OSA.[3] It is also known that OSA increases risk of resistant arterial hypertension, coronary artery disease, heart failure, pulmonary hypertension, and various cardiovascular conditions.[4] Moreover, investigations demonstrated the association between OSA and cardiac remodeling. However, these studies were usually concentrated on left ventricular (LV) structure and function, which mainly included basic echocardiographic parameters such as LV hypertrophy, LV ejection fraction, and several basic parameters of LV diastolic (dys)function.[5] Investigations regarding LV mechanics in patients with OSA are scarce.

Altekin et al[6] included 58 OSA patients with different severity of OSA. Patients were classified as mild OSA when the apnea-hypopnea index (AHI) was between 5 and 15, moderate OSA was diagnosed in patients with AHI between 15 and 30 and severe OSA was defined as AHI higher than 30.[6] The authors reported that main parameters of LV diastolic function (E/A, E/e′, mitral deceleration time, left atrial volume index) deteriorated gradually from mild to severe OSA.[6] More interestingly, the authors found gradual reduction in LV longitudinal strain from mild, across moderate, to severe OSA. The reduction in LV circumferential and radial strain was revealed in moderate and severe OSA patients, but not in those with mild OSA. However, strain rates referring to LV mechanics during early and late diastole also gradually deteriorated from mild to severe OSA in all three directions—longitudinal, circumferential, and radial.[6] The correlation between AHI and LV longitudinal strain and strain rates was significantly stronger than between AHI and parameters of LV diastolic function.[6] On the other hand, there was no correlation between AHI and LV ejection fraction and only weak correlation with LV mass index.[6] One should underline that there was no significant difference in body mass index (BMI) between three observed groups. However, the authors did not perform multivariate regression analysis including BMI in the model in order to exclude potential effect of obesity on the final results. Varghese et al[7] also reported significantly lower LV longitudinal strain in patients with severe OSA, but without significant difference in LV circumferential strain. LV longitudinal strain, but not circumferential strain, was found to correlate significantly with AHI.[7]

Cho et al[8] included 25 patients with OSA and compared LV remodeling in these patients with healthy controls and obese patients, as well as the impact of bariatric surgery in patients with OSA. The authors did not find significant difference in LV structure (LV wall thickness and LV mass index) between obese and OSA patients, but they found significantly worse LV diastolic function in patients with OSA than in the obese and control groups. LV global longitudinal strain was also significantly more reduced in OSA patients than in obese patients.[8] LV diastolic function and longitudinal strain significantly improved after bariatric surgery in OSA patients, even though statistical significance was only borderline. However, these findings are most probably the result of limited number of patients.

The percentage of OSA in patients with resistant arterial hypertension is remarkable.[4] The RESIST-POL study included patients with resistant hypertension and divided them into four groups using OSA and metabolic syndrome as criteria and found that patients with OSA and metabolic syndrome had the lowest LV longitudinal strain, even though blood pressure level, LV mass index, and prevalence

of LV hypertrophy was similar between four groups.[9] Interestingly, the authors did not find significant difference between patients with isolated OSA and isolated metabolic syndrome. However, the limited number of patients again represents an important limitation of this study that made difficult reaching of statistical significance.

Left ventricular wall contains three layers—endocardial, mid-myocardial, and epicardial. This complex structure is necessary in order to provide efficient LV contraction. Additionally, there are several LV motions that enable this efficient LV contraction. Besides longitudinal, circumferential, and radial direction, LV twist and torsion are very important to support LV power. Zhou et al[10] demonstrated that OSA impacted all three myocardial layers in longitudinal and circumferential strain. The findings showed that longitudinal strain was reduced in all myocardial layers in all cardiac segments (basal, mid, and apical) in patients with severe OSA compared with controls or mild OSA.[10] Circumferential strain in all three layers was lower in patients with OSA than in controls, but there was no difference between mild, moderate, and severe OSA patients. There was a strong correlation between AHI and multilayer strain (endo-, mid-, and epicardial).

Wang et al[11] investigated 3D LV multidirectional strain in patients with OSA and revealed that 3D LV longitudinal and area strain were significantly lower in the patients with moderate and severe OSA than in the control group and patients with mild OSA,[11] whereas 3D circumferential and radial strain were significantly reduced only in patients with severe OSA in comparison with controls. It should be emphasized that severe OSA patients had significantly lower all types of 3D strain in comparison to controls or mild and moderate OSA patients. In other words, 3D multidirectional strain gradually and significantly reduced from mild, across moderate, to severe OSA patients.[11] Additionally, 3D longitudinal, circumferential, radial, and area strain correlated well with AHI. However, LV mass index was increasing and LV ejection fraction, as well as LV diastolic parameters, was deteriorating in the same direction—from mild to severe OSA.

Vitarelli et al[12] investigated LV mechanics in patients with OSA using LV twist and torsion, besides LV strains, and showed that apical rotation and basal rotation, as well as LV torsion, were significantly higher in patients with severe OSA than in control group or in patients with mild OSA. Interestingly, there was no difference in rotation and twist between controls and mild OSA. LV longitudinal strain was also lower in patients with severe OSA, whereas LV circumferential and radial strains were not different between controls and OSA patients.[12] Vural et al[13] reported gradual deterioration of LV longitudinal strain from mild to severe OSA group, but circumferential and radial strains were lower only in moderate and severe OSA patients. Apical rotation and torsion were lower only in severe OSA comparing with controls, but there was no difference between controls and mild or moderate OSA.[13]

The effect of therapy which includes continuous positive airway pressure (CPAP) on LV remodeling in patients with OSA is still matter of debate. Kim et al[14] compared the effect of CPAP and sham treatment on LV remodeling in patients with OSA and reported that CPAP treatment significantly improved LV global longitudinal strain compared with the sham treatment after 3 months of therapy, even though the differences in LV dimension or ejection fraction was not noticed. D'Andrea et al[15] found that LV morphology was comparable before and during non-invasive ventilation, whereas LV ejection fraction and LV regional peak myocardial strain were significantly improved in patients with OSA during non-invasive ventilation. Hammerstingl et al[16] included 82 OSA patients with indication for CPAP therapy and found that LV ejection fraction, LV performance index, and stroke volume improved significantly after CPAP therapy. However, the authors investigated only right ventricular strain, which also improved, but not LV strain. Vitarelli et al[12] found that LV longitudinal strain significantly improved after CPAP therapy, which was not the case with circumferential and radial strains. However, circumferential and radial strains were not reduced in patients with OSA even before therapy and therefore it was not expected to improve. Vural et al[13] showed significant improvement of LV longitudinal strain, basal and apical rotation after CPAP therapy. However, there was no improvement in circumferential and radial strain and torsion in patients with OSA after CPAP therapy.

There are several mechanisms that could explain the relationship between OSA and LV mechanics, despite arterial hypertension, obesity, and metabolic syndrome that are commonly seen in patients with OSA. Repetitive hypoxia due to OSA could affect the interaction between myocardial oxygen demand and supply, which results with the development of relative myocardial ischemia and subclinical LV systolic dysfunction. Hyperactivation of sympathetic nervous system in OSA could also be one of the reasons for LV remodeling. Increased inflammation is additional mechanism that could explain cardiac changes in patients with OSA. Interventricular interaction represents an important mechanism that is partly responsible for LV dysfunction because patients with OSA usually have increased pulmonary pressure and right ventricular changes that could influence the LV.

In conclusion, LV strain represents an important imaging tool that could detect subclinical LV changes in patients with OSA even when conventional echocardiographic parameters of LV structure and function are in the normal range. However, larger follow-up studies or at least meta-analysis with summarizing of current data are necessary to further investigate relationship between LV strain and OSA and to determine the predictive value of LV strain on outcome in these patients.

CONFLICT OF INTEREST

None.

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