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Editorial

Echocardiographic Evaluation of Right-sided Heart in Pediatric Patients with Adenotonsillar Hypertrophy

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▸ See the article "Right Atrial Deformation Mechanics in Children with Adenotonsillar Hypertrophy" in volume 26 on page 201.

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Conflict of Interest
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Adenotonsillar hypertrophy (ATH) is a common cause of upper airway infection, upper airway obstruction, and obstructive sleep apnea (OSA) in children.[1] Intermittent hypoxia related to this obstruction can result in pulmonary vasoconstriction, pulmonary hypertension (PH), and right ventricular (RV) dysfunction.[2] Also, RV dysfunction may enlarge atria and enlarged atria may influence the conduction time of the electrical impulse originating in the sinus node and its spread. Consequently, children with moderate to severe ATH may show prolongation in P-wave duration, P-wave dispersion (PWD), and inter- and intra-atrial electromechanical delays upon 12-lead electrocardiogram (ECG).[3]

Many studies have attempted to show evidence for RV dysfunction in this disease group using several echocardiographic parameters (e.g., conventional and pulsed-wave tissue Doppler echocardiography [TDE]).[2][4-8] The RV myocardial performance index by pulsed wave TDE in patients with ATH was significantly higher than in the control group and showed a strong correlation with mean pulmonary artery pressure.[2][4][7] Especially, the patients with moderate to severe OSA showed more prominent changes than the patients with mild OSA and such changes were reversible after surgical treatment.[5][7][8] Tricuspid annular peak early diastolic velocity (E′) by pulsed wave TDE is a sensitive marker of TDE for evaluating diastolic function and has the strongest effect on cardiac outcomes among the TDE parameters significantly increased after adenotonsillectomy.[6] Tricuspid annular plane systole excursion, tricuspid isovolumic acceleration, and pulmonary acceleration time were significantly lower in the preoperative group compared with the control group.[7]

Right atrial (RA) analysis for evaluation of RV dysfunction, morbidity, and mortality has been tried in studies of other disease groups including tetralogy of Fallot and Eisenmenger syndrome. Recently, one study showed damaged RA deformation in patients with PH.[9] In this study, patients with PH showed significantly lower peak longitudinal RA strain (PLRAS) than controls and this was gradually reduced with the development of cardiac insufficiency. There was a significant positive correlation between global PLRAS and 6-minute walk distance. Global PLRAS showed the highest diagnostic accuracy (AUC of 0.979) with a sensitivity of 86.8% and a specificity of 84%. This result suggests that PLRAS is possibly valuable for predicting functional status and exercise capacity in patients with PH.

Since one of the mechanisms of RV dysfunction in patients with significant ATH associated with sleep-related breathing disorders (ATH-SRBD) is elevated pulmonary pressure, RA analysis may predict RV dysfunction. In this issue of the journal, Kang and Kwon[10] investigated the usefulness of PLRAS for evaluation of RV dysfunction in patients with ATH-SRBD. They evaluated PLRAS, tricuspid annulus E/E′ using TDE, maximum P-wave duration (Pmax), and PWD in 12-lead ECG. The authors showed subtle RV dysfunction by evaluating PLRAS during systole in patients with ATH-SRBD. Preoperatively, PLRAS was significantly decreased in children with ATH-SRBD compared with controls even when RV longitudinal peak systolic strain and strain rate were similar. Also, increased tricuspid annulus E/E′ were found. In addition, they showed increased prolonged Pmax and PWD which indicates RA distortion and atrial remodeling caused by negative intrathoracic pressure change.[11]

The authors suggested the possible usefulness of PLRAS as a parameter for RV dysfunction by providing a ROC curve analysis which showed that PLRAS was not inferior to tricuspid annulus E/E′, Pmax, and PWD for differentiating children with ATH-SRBD from controls. But, unfortunately, they couldn't provide the discriminative ability of PLRAS. Also, there was no difference in preoperative mean pulmonary artery pressure between patients and controls and no change in all echocardiographic parameters before and after the operation, which is different from other previous studies.[5][8] However, since only limited data have been available until recently, the present study provides new diagnostic challenges that show the possibility of PLRAS as a potentially useful parameter for evaluating subtle RV dysfunction in this disease group. The RA mechanics in patients with ATH-SRBD needs further evaluation.

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