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Combined CMR and catheterization data in determining right ventricular-arterial coupling in children and adolescents with pulmonary arterial hypertension

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Background

Pulmonary arterial hypertension (PAH) remains a disease with high morbidity/mortality in pediatrics. Understanding ventricular-arterial coupling, a measure of how well matched the ventricular and vascular function is, may elucidate the pathway leading to right heart failure.

Methods

This retrospective study included subjects with PAH who a cardiac magnetic resonance (CMR) study within 14 days of cardiac catheterization between January 2009-August 2013. The effective elastance (Ea, index of arterial load) and right ventricular maximal end-systolic elastance (Emax, index of contractility) were determined by a combination of CMR and hemodynamic data. Ea is defined as (mean pulmonary arterial pressure minus pulmonary capillary wedge pressure)/stroke volume. Emax is defined as mean pulmonary arterial pressure/end systolic volume. Ea/Emax ratio was derived. Additionally, a measure of non-invasive ventricular arterial coupling (assuming PWCP is insignificant, making Ea/Emax = end systolic volume/stroke volume) was derived from only CMR. Pulmonary vascular resistance indexed (PVRi) and pulmonary vascular reactivity, as defined by Barst criteria (decrease in mean pulmonary artery pressure of > 20%, unchanged/increased cardiac index, and decreased/unchanged pulmonary to systemic vascular resistance ratio), were also determined. Pearson correlation coefficients were calculated between PVRi and Ea, Emax, and Ea/Emax. Receiving operating characteristic (ROC) curve analysis determined the diagnostic value of Ea/Emax in predicting vascular reactivity.

Results

Sixteen subjects were identified for inclusion with equal gender distributions. Age ranged from 3 months to 23 years (mean 11.3+7.4 years). Ea and Ea/Emax increased with increasing severity defined by PVRi, with $p < 0.001$ for both. Ea/Emax (range 0.43-2.82) was highly correlated with PVRi ($r = 0.92$, 95% CI 0.79-0.97, $p < 0.0001$). Non-invasively derived ventricular arterial coupling was found

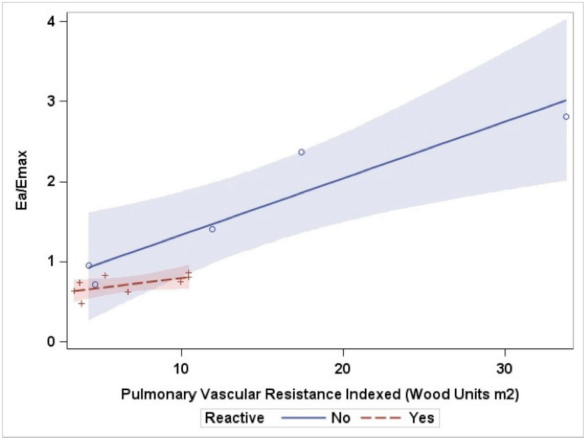

Figure 1 Regression of ventricular arterial coupling ratio (Ea/Emax) and pulmonary vascular resistance indexed by reactivity. The shaded areas represent the 95% confidence interval for each regression line. The lines depict different trajectories based on reactivity, which approached, but not reach statistical significance ($p > 0.05$).

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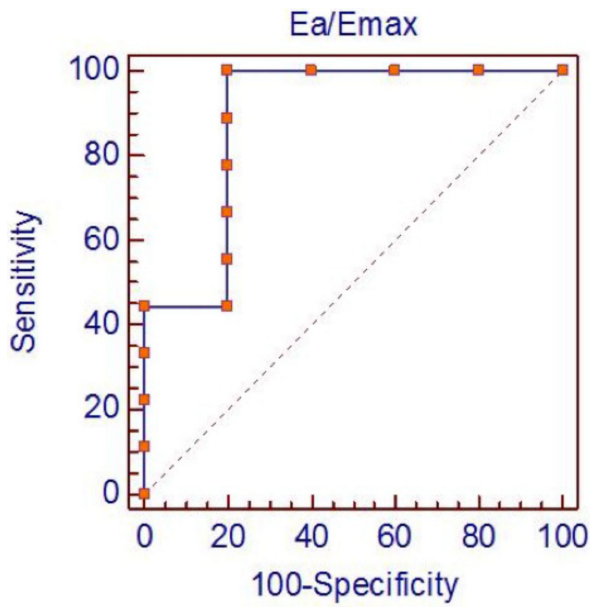

Figure 2 Receiver operating characteristic curve demonstrating an optimal threshold Ea/Emax ratio of 0.85. Using this criterion, is associated with a sensitivity of 100% and a specificity of 80%.

to be significantly correlated with PVRi (r = 0.85, 95% CI 0.62-0.95, p < 0.0001), but with a lower correlation coefficient than with Ea/Emax derived from combined hemodynamic and CMR data. Regression of Ea/Emax and PVRi demonstrated differing lines when separated by reactivity, however, the lines were not significantly different (Figure 1). ROC curve analysis (Figure 2) revealed high accuracy of the Ea/Emax ratio in determining vascular reactivity. Ea/Emax of 0.85 had a sensitivity of 100% and a specificity of 80%. The area under the curve is 0.89 (p = 0.008), suggesting good discrimination between those who were and were not reactive.

Conclusions

Measurement of ventricular arterial coupling, Ea/Emax, in pediatrics is feasible. Pulmonary vascular non-reactivity may be due to ventricular-arterial decoupling in which ventricular contractility fails to parallel increasing afterload in severe PAH. Use of Ea/Emax may have significant prognostic implication.

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