Right Ventricular Plasticity

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A 37-year-old woman with primary pulmonary hypertension since the age of 30, presented with worsening dyspnea on exertion and hypotension. Physical examination revealed jugular venous distention, right ventricular heave, increased

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intensity of P2, 3/6 holosystolic murmur, abdominal flank dullness to percussion and minimal peripheral edema. A chest film revealed severe cardiomegaly (A). Electrocardiogram (ECG) showed complete right bundle branch block with right atrial and ventricular hypertrophy. Apical 4-chamber view of a transthoracic echocardiogram (C) showed massive right atrial (RA) and right ventricular (RV) enlargement with severe compression of the left ventricle in systole (arrow) and a normal left atrium (LA) with right ventricular systolic pressure of 112 mmHg. Torrential tricuspid regurgitation (TR) was evident by color-flow Doppler in systole (D). Magnetic resonance imaging (B) revealed a prominent pulmonary artery (PA) with severe RA and RV enlargement

and hypertrophy with RV ejection fraction of 23% (normal 53-73%) and RV end-diastolic volume indexed by BSA of 306 cc/m² (normal 50-118 cc/m²). The patient is currently receiving inotropes and diuretics while awaiting heart-lung transplantation.

Severe septal shift alters left ventricular geometry (C) and is responsible in part for the presenting hypotension (1). The myocardium has a dynamic growth rate of at least 100% in response to stress (2). Decompensation is related to apoptosis, fibrosis, calcium homeostasis perturbations, and chamber dilatation, and it may be irreversible (2), such as in our patient.

References

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