A Case of Lead-Induced Severe Tricuspid Regurgitation and Right-Sided Heart Failure

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Abstract Body:

Background:

Tricuspid regurgitation (TR) is a complication that can occur during defibrillator implantation procedures, particularly when the leads are placed in the right ventricle. TR is caused when the tricuspid valve does not close properly, causing blood to backflow to the right atrium during systole resulting in heart failure. The exact causes of TR after lead placement are not completely understood, but it is thought to be related to entrapment, impingement, perforation and adhesion of pacemaker leads. TR that develops from lead implantation has been associated with increased hospitalizations and decreased long-term survival. Our patient's TR was multifactorial and managed accordingly with transcatheter aortic valve implantation, guideline-directed medical treatment for heart failure with reduced ejection fraction, and finally tricuspid valve replacement.

Case:

A 78-year-old male with history of 4-vessel coronary artery bypass grafting, heart failure with reduced left ventricular ejection fraction (HFrEF) 35-40%, paroxysmal atrial fibrillation, and inducible ventricular tachycardia with defibrillator placement presented with worsening lower extremity pitting edema, dyspnea, abdominal bloating and a systolic murmur along the left sternal border. Transthoracic echocardiogram (TTE) and follow-up transesophageal echocardiogram (TEE) showed severe aortic stenosis, right ventricular systolic pressure (RVSP) estimated at 34.6 mmHg, severe biatrial enlargement, and severe TR in the region of his device lead.

Decision Making:

Tricuspid valve replacement was initially deferred due to his prior bypass surgery with patent grafts. Transcatheter aortic valve implantation (TAVI) was performed to reduce pulmonary artery pressures. Concomitant guideline directed medical therapy (GDMT) for HFrEF was also optimized to reduce TR from right ventricle pressure overload resulting from left sided heart disease and RVSP. Post-TAVI, the patient had only marginal improvement in symptoms. TTE showed RVSP 42 mmHg and moderate to severe TR. Due to his persistent symptoms, he was sent for minimally invasive tricuspid valve replacement. Follow-up TTE demonstrated RVSP estimated at 50 mmHg, and mild to moderate TR.

Conclusion:

Medical therapies for severe TR are limited. Management is focused upon the underlying etiology of the TR. Our patient's TR was multifactorial and managed accordingly with TAVI, GDMT for HFrEF, and finally tricuspid valve replacement.