Diffuse Ventricular Hematoma after Heart Transplant and Repeated Myocardial Instrumentation

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Abbreviations: CAD: Coronary Artery Disease; EMB: Endomyocardial Biopsy; LV: Left Ventricle; OHT: Orthotopic Heart Transplant; RV: Right Ventricle; TTE: Trans-Thoracic Echocardiography

Description of Case

A 69-year-old male Jehovah’s Witness with a past medical history of non-ischemic cardiomyopathy, coronary artery disease (CAD), obesity, gout, and nephrolithiasis presented to a large academic medical center for evaluation for advanced options. He had a myocardial infarction in 2004, requiring a single stent to the left anterior descending artery. He was found to have an ejection fraction of 10% that was thought to be out-of-proportion to his CAD. His cardiac function did not improve with guideline-directed medical therapy, including a biventricular implantable cardioverter-defibrillator, and on presentation he was no longer able to tolerate significant neurohormonal blockade. Of note, family history was notable for a mother with a “large heart,” but the rest of his history was non-contributory. The patient was deemed a good candidate for advanced options, and he subsequently underwent bloodless orthotopic heart transplant (OHT) in June 2016.

Initial post-operative transthoracic echocardiogram (TTE) showed a moderate pericardial effusion without hemodynamic compromise. Three weeks post-OHT, the patient developed various atrial arrhythmias, including blocked premature atrial contractions resulting in bradycardia and concerning for rejection. He underwent endomyocardial biopsy (EMB) which showed grade 0 rejection. The next day, a permanent pacemaker was implanted with the single right ventricular (RV) lead placed in the apical, septal region with active fixation. The following day, the temporary epicardial pacing leads were removed at bedside; removal of the second RV lead was complicated by resistance and induction of premature ventricular contractions. The next day, TTE was notable for hematoma in the myocardium of the RV and the apicolateral segment of the left ventricle (LV). Because of this finding, future monitoring for rejection was performed with TTE, and biopsy deferred for 4 weeks. On repeat TTE ten days later, the hematoma had resolved, and the patient had good graft function. Repeat EMB four weeks after the initial diagnosis of hematoma remained Grade 0.

The patient has continued to have good cardiac function, without evidence of complication from the intraventricular hematoma, and he has not had any significant rejection in the first eleven months following OHT.

Discussion

We present a case of ventricular hematoma in a patient one month post-OHT following three consecutive days of endocardial and epicardial manipulation. Ventricular hematoma was diagnosed by visualization of a bright, echo-dense region within the myocardium of the LV, RV, and interventricular septum (Figure 1). Given the appearance of this finding following multiple cardiac procedures, it most certainly represents blood consistent with a ventricular hematoma.

Each of the three procedures in the days leading up to diagnosis carries a low risk of ventricular perforation. The initial procedure, completed three days prior to diagnosis, was EMB, a routine procedure indicated after OHT to monitor for rejection. Cardiac perforation is a known, rare complication, with a reported incidence of 0-0.3% [1-5], but only one actual case of ventricular hematoma has been described, and this was a unique case of septal hematoma [6]. A single-lead pacemaker was placed in the RV two days prior to diagnosis. Myocardial perforation has been reported as a rare complication during cardiac device lead placement, occurring <1% of the time [7-9], but there has been no reported association with ventricular hematoma. On the day prior to diagnosis of the ventricular hematoma, the patient's temporary epicardial pacing wires were removed at the bedside, with significant resistance met when pulling one of the two RV leads. The incidence of cardiac tamponade associated with this procedure is 1% [10], though, again, incidence of ventricular perforation or hematoma is not specifically described.
Figure 1: Ventricular hematoma was diagnosed by visualization of a bright, echo-dense region within the myocardium of the LV, RV, and interventricular septum.

**Post-transplant, prior to procedures**
1. Apical 4-chamber
2. Parasternal long

**Following procedures**
3. Apical 4-chamber. Arrow indicates biventricular hematoma.

**Figure 1**: Ventricular hematoma was diagnosed by visualization of a bright, echo-dense region within the myocardium of the LV, RV, and interventricular septum.
Our case describes the first reported instance of diffuse biventricular hematoma following repeated cardiac manipulation identified by TTE. It remains unknown which of the three specific cardiac manipulations caused this hematoma, as there was no imaging done between procedures. The precise mechanism of hematoma formation remains unknown as well. However, we speculate it is due to a small ventricular perforation, either via the endocardial, in the case of the EMB or pacemaker lead placement, or epicardial, as with the epicardial lead removal, surface. Of note, the patient did not have a coagulopathy at the time of the procedures.

This case represents a unique finding that may have been caused by any of the three procedures performed, and it is a distinctive complication of them all. This serves to illustrate the cautionary approach with which invasive cardiac procedures should be considered.

References