Constrictive Pericarditis Presenting as a Late Complication of Epicardial Ventricular Tachycardia Ablation

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Radiofrequency epicardial ablation of ventricular tachycardia (VT) is a safe, effective modality for treatment of VT. Epicardial VT circuits were originally described in Chagas disease, but the importance of nonendocardial substrates for VT increasingly are recognized in other cardiomyopathies. In arrhythmogenic right ventricular cardiomyopathy (ARVC), failure of endocardial VT ablation often is related to epicardial VT origination, with high rates of success seen with epicardial VT ablation. While hemopericardium or pericardial inflammation are rare complications of epicardial VT ablation, to our knowledge no case of pericardial constriction and heart failure following epicardial VT ablation has been reported. Here we describe a case of pericardial constriction and severe heart failure requiring stripping and “bridge to recovery” right-ventricular assist-device (RVAD) support, which occurred subsequent to recurrent epicardial VT ablations.

Case Presentation

A 46-year-old male who presented with palpitations and episodic syncope underwent cardiac magnetic resonance imaging, which was consistent with ARVC, followed by implantable cardioverter-defibrillator implantation (1999). Five years later, he developed exercise-induced VT and underwent endocardial ablation. An echocardiogram revealed a normal left ventricle but a mildly dilated right ventricle with normal function. After receiving multiple shocks for VT, he underwent repeat ablation in May 2008, followed by epicardial VT ablation in January 2009. Six months later, he underwent repeat epicardial ablation, after which he reported pleuritic chest pain, which may have represented pericarditis. A fourth endocardial and epicardial attempt at VT ablation was made in September 2010.

In the ensuing months he developed right upper quadrant pain and ongoing intermittent chest pain. His laboratory studies were unremarkable, but right upper quadrant ultrasound revealed significant hepatomegaly and a markedly thickened gallbladder without stones. In June 2011 he underwent laparoscopic cholecystectomy. Two days later he presented with right upper quadrant pain, a 15-pound weight gain, and abdominal swelling. CT scan of the abdomen was consistent with passive congestion of the liver and no evidence of bile leak or abscess. The Heart Failure service was consulted and intravenous furosemide was recommended for worsening right heart failure. Echocardiography revealed normal left ventricular size with preserved function, a mildly dilated right ventricle with moderately to severely decreased function, a thickened pericardium, a septal bounce, a small to medium-sized circumferential pericardial effusion, and septal E’ velocity greater than lateral suggesting tethering of the lateral wall, consistent with pericardial constriction. As his clinical status deteriorated, right heart catheterization revealed a right atrial pressure of 28 mm Hg, an elevated right ventricular end-diastolic pressure of 25 mm Hg without a classic dip-and-plateau sign, a pulmonary capillary wedge pressure of 24 mm Hg, and cardiac index of 1.2 L/min/m². He was initiated on milrinone, but had worsening renal and liver failure. He was taken to the operating room for a pericardial window, and 400 mL of bloody fluid were drained, with initial improvement. Unfortunately, within 24 hours he again had severe right heart failure and was listed urgently for cardiac transplantation. He was taken back to the operating room, where he underwent a radical pericardectomy. Operating room findings included a thick, orange colored epicardial peel (Figure) that was circumferential around all 4 cardiac chambers. After pericardial stripping, right ventricular function improved, but 2 attempts to separate him from bypass failed and a Thoratec paracorporeal RVAD was placed. Over the course of the next month, he continued to improve and was discharged home. After extensive provocative exercise testing with invasive hemodynamic monitoring revealed improved right ventricular function with RVAD turndown, his RVAD was explanted. A biopsy of his right ventricle was performed and showed fibrofatty infiltration, consistent with ARVC.

Discussion

The immediate antecedent cause of his severe heart failure requiring RVAD support and transplant evaluation was, therefore, pericardial constriction and tethering of the right
ventricular and left ventricular lateral walls. One possibility is that acute pericarditis caused by epicardial VT ablation led to formation of adhesions, resulting in pericardial constriction. Because of his underlying diagnosis of ARVC, pericardial constriction was not recognized immediately as a cause of worsening right heart failure. Notably, it is precisely this type of patient, with a nonischemic cardiomyopathy, who often requires epicardial access for a VT ablation. The symptoms of pericardial constriction easily could be missed, as they may be confused for worsening underlying myopathy, which often presents with restrictive physiology. This report highlights a late, severe complication that may be seen after epicardial VT ablation; with the increasing use of epicardial access, particularly in patients with nonischemic cardiomyopathies, the potential consequences of pericardial constriction should be entertained.

Disclosures

None.

References


Key Words: epicardial ablation ■ ventricular tachycardia ■ heart failure ■ pericardial constriction ■ arrhythmogenic right ventricular cardiomyopathy
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