

Late papillary muscle rupture and tricuspid regurgitation related to transvenous endocardial lead extraction



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Introduction

Cardiac implantable electronic devices are being increasingly utilized for a growing number of indications, with an estimated 400,000 devices implanted in the United States each year.¹ This increase in device implantations has been linked to a surge in device extractions in recent years driven primarily by lead infections occurring in an estimated 13,000 patients annually.²

Despite an estimated 1-year mortality of up to 8%, primarily driven by complications related to systemic infection, transvenous lead extraction is associated with an extremely low intraprocedural mortality when performed in experienced centers.³ Although rare, serious complications of this procedure, such as cardiac perforation, have been well classified. Damage to the tricuspid valve is an increasingly recognized complication related to lead extraction and may present with variable timing following this procedure. Complex removal of leads with significant fibrotic encapsulation can result in tearing of the tricuspid leaflets, damage to chordae, and traumatic papillary muscle rupture. This disruption of adhesions between the leads and portions of the tricuspid valve apparatus ultimately results in tricuspid regurgitation (TR). Intraprocedural transesophageal echocardiography (TEE) during lead extraction may identify traumatic tricuspid regurgitation (TTR) in up to 12% of patients, although rarely severe, and may be identified in either an acute or a subacute setting.^{4,5} While not always clinically significant, in rare

cases TTR may require surgical correction, based primarily on patients' symptoms and significant hemodynamic impact on the right ventricle.

We present a case in which a patient with an implantable cardioverter-defibrillator (ICD) required extraction for lead fracture. This case was complicated by TTR as evidenced by new tricuspid regurgitation on echocardiography in the setting of patients' worsening symptoms 2–3 months after the procedure, ultimately requiring surgical tricuspid valve replacement and placement of an epicardial system.

Case report

A 72-year-old man with a history of myocardial infarction and coronary artery bypass grafting with subsequent ischemic cardiomyopathy and a secondary prevention single-lead ICD placed 12 years ago for monomorphic ventricular tachycardia was noted to have electrical noise on his ICD lead (St Jude Medical, Model 7121), suggesting a lead conductor coil fracture. The patient underwent laser lead extraction using a 16F excimer laser sheath (GlideLight and CVC-300 Laser system, Phillips Medical Systems, Wayne, PA). Bindings in the subclavian and brachiocephalic veins and at the tricuspid valve were lysed with the laser. The lead was extracted in its entirety without difficulty. Access to the right heart was maintained by a guide wire and a new right ventricular (RV) lead was connected to a single-chamber ICD. The patient had a known left ventricular ejection fraction of 25% with normal RV size and function. Periprocedural TEE and postprocedural transthoracic echocardiography (TTE) showed no significant tricuspid valve disruption or regurgitation immediately after extraction (Figure 1, left). The procedure was uncomplicated, and the patient was discharged home the following day. Over the next 3 months the patient experienced an appropriate ICD shock for rapid ventricular tachycardia and reported progressive fatigue and dyspnea with minimal exertion. Worsening symptoms prompted repeat cardiac evaluation. Coronary

KEYWORDS Cardiovascular implantable electronic devices; Lead extraction; Papillary muscle rupture; Tricuspid regurgitation; Tricuspid valve replacement

(Heart Rhythm Case Reports 2021;7:577–580)

Conflicts of Interests for all authors: None. Funding Sources: The authors have no funding sources to disclose. **Address reprint requests and correspondence:** Dr Nicholas J. Beccarino, North Shore University Hospital, 300 Community Dr, Manhasset, NY 11030. E-mail address: nbeccarino@northwell.edu.

KEY TEACHING POINTS

- Late papillary muscle rupture may occur after endocardial lead extraction, leading to clinical deterioration and requiring surgical correction.
- Lead fibrosis and the use of laser sheaths during extraction increases the risk of traumatic tricuspid regurgitation.
- Options for avoiding interaction of right ventricle endocardial leads with native or prosthetic tricuspid valves include the use of epicardial pacing leads and defibrillation patches or the use of coronary sinus left ventricle branches.

angiography showed all grafts to be patent with no new lesions. TTE revealed a left ventricular ejection fraction of 20% with new torrential TR and a large mobile mass of 1.75 cm × 0.7 cm in the right atrium found to be flail chordae and papillary muscle on subsequent TEE (Figure 1, right, and Supplemental Video 1). In preparation for a tricuspid valve replacement the patient underwent removal of his endocardial ICD system, following which the patient underwent an uncomplicated bioprosthetic tricuspid valve replacement using a 33 mm Magna Ease valve. At surgery the flail segment of the tricuspid apparatus consisted of the ruptured head of the septal papillary muscle attached to the chordae. In order to prevent further hardware traversing the tricuspid valve prosthesis, epicardial defibrillating patch and ventricular pacing leads were implanted. Postoperatively, a single-chamber ICD was implanted. Since discharge, the patient has reported a marked improvement in symptoms with a normally functioning tricuspid valve and minimal regurgitation on recent echocardiography.

Discussion

We report a case of TTR related to lead extraction of an endocardial RV lead for lead malfunction. In this case septal

papillary muscle rupture occurred late after extraction of a defibrillating lead with progressive TR. Subsequent tricuspid valve replacement and epicardial lead placement resulted in marked clinical improvement.

Endocardial pacing or defibrillating leads interact with the tricuspid valve, leading to severe tricuspid regurgitation by several mechanisms. An autopsy study demonstrated interference of endocardial leads with the TV apparatus in 42% of cases.⁶ This and other surgical reports have revealed interactions that include entanglement of the lead in the subvalvular chords, perforation of the leaflets, and impingement of or lead adherence to the leaflets, with occasional valve immobilization.⁷ When severe, these interactions are often managed surgically; however, lead extraction has been used successfully as well. Recovery of tricuspid valve and right heart functioning may occur later after percutaneous lead removal, occurring up to 1 year postextraction in some cases.⁸

Lead removal carries risk of tricuspid valve damage as well, as seen in our case. Extraction is typically performed using a subclavian vein approach, as it allows for the procedure to be completed using a single incision. Femoral and internal jugular vein approaches may have to be utilized in particularly high-risk cases where significant fibrosis is present or when retained lead remnants have to be retrieved. While simple traction is successful in nearly one-third of cases, extraction becomes more complex as leads and surrounding cardiac structures develop increasing fibrotic encapsulation and adhesion over time. In these cases, coaxial sheaths that rely on laser or rotational forces to break the adhesions are employed. Although effective, these more invasive approaches have higher complication risks, which can include vascular rupture, cardiac perforation with pericardial effusion or tamponade, thromboembolism, hemothorax, and death.⁹

TTR is an increasingly recognized delayed complication of RV lead extraction. Complex removal of leads with significant fibrotic encapsulation, particularly using laser sheaths, has been found to be associated with a higher risk of TTR.¹⁰ Whether this effect is mediated by properties specific to this method or is simply owing to the fact that laser is generally implemented in more complex and high-risk procedures with high fibrotic burden has been difficult to assess. The risk is particularly high when utilized after failure of

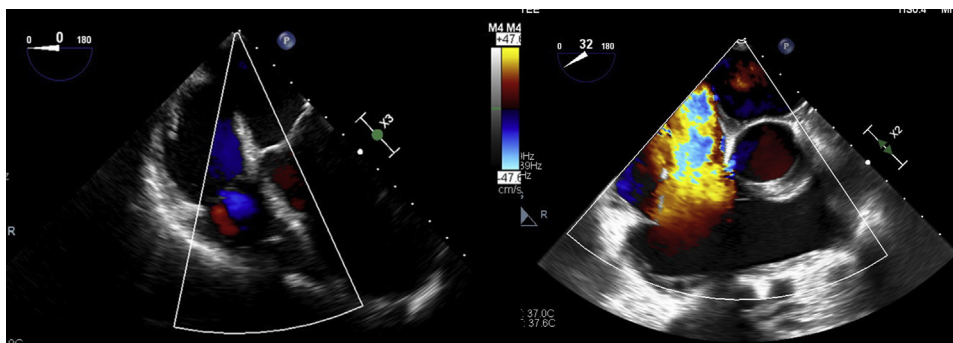


Figure 1 Echocardiography immediately following lead extraction showing intact valve without significant regurgitation (left) as well as follow-up transesophageal echocardiography showing severe tricuspid regurgitation and flail tricuspid leaflet (right).

simple traction.⁴ Implant duration is one of the major factors that predict worsening TR after lead extraction and supports fibrotic burden as a predominant risk factor. Other risk factors include younger age at implant, the presence of multiple ventricular leads, and endocarditis involving the valve.^{4,11} Smaller lead diameters have been shown to be a predictor of perioperative complications, and indeed, the extraction of pacemaker leads has been associated with higher risk of TTR as compared to ICDs.^{9,12}

While TTR is often apparent immediately after extraction, the initial insult of TTR may lead to progressive valve dysfunction as well, as shown in this case. Late rupture of the papillary muscle has not been previously reported. The exact timing of the rupture is unclear, as no echocardiogram was performed until several months after the procedure when the patient presented with symptoms. In the absence of further interim instrumentation, it has to be assumed that the trauma of lead extraction caused partial papillary muscle rupture at the time of extraction, with subsequent total rupture and severe TR necessitating surgical intervention. Such variability in presentation can make routine postprocedural screening and determination of the exact mechanism of TTR difficult to assess. The incidence of worsening TR after lead extraction was observed to be 11.5% in 1 study.¹² However, the majority of patients do not require surgical intervention for valve repair. Cases of severe TTR requiring surgical repair are limited to isolated case reports.¹³ Intraoperative TEE should be routinely performed, particularly during high-risk lead extraction, in order to identify early tricuspid valve damage; however, this approach may not capture a significant portion of TTR patients. While serial echocardiography postprocedure may not be necessary in all patients postextraction, those at high risk for TTR or with the development of new signs or symptoms of right heart failure should undergo TTE. The most significant factors that may increase the utility of serial echocardiography postextraction include the presence of significant fibrotic encapsulation, complex extraction especially utilizing laser sheaths, and increased time since implant. In these patients, follow-up echocardiography at 1 and 3 months may be a reasonable surveillance strategy for the development of TTR.

While cause-specific data regarding outcomes in patients experiencing significant postextraction TTR are sparse, there is strong evidence suggesting that TR is associated with poor outcomes. Isolated severe TR has been shown to be associated with adverse cardiac events as well as all-cause mortality independent of RV function and pulmonary pressures.⁷ This effect appears to be especially pronounced in patients with an effective regurgitant area of greater than 40 mm², which can be expected in cases of severe TTR.¹⁴

Indications for surgical intervention for TTR can be extrapolated from guidelines regarding primary TR. Severe TR in patients undergoing left-sided valve surgery and symptomatic severe TR unresponsive to medical therapy are both relatively strong surgical indications.¹⁵ Surgery can also be considered in patients with asymptomatic or minimally symptomatic severe primary TR with at least moderate RV

dilation or failure.¹⁵ These criteria were met in the setting of symptomatic severe TR.

Options for avoiding interaction of RV endocardial leads with native or prosthetic tricuspid valves include the use of epicardial pacing leads and defibrillation patches or the use of coronary sinus left ventricle branches. The recognition of the deleterious effects of endovascular lead systems has led to the advent of extravascular or leadless systems. However, at present leadless pacemakers only provide RV pacing and hence, they are not options for patients who need atrial pacing.¹⁶ Subcutaneous defibrillators are an option for patients without pacing requirement including antitachycardia pacing. Newer extravascular defibrillating systems capable of delivering pacing therapies are under investigation.¹⁷

Conclusion

TTR is an increasingly recognized complication of RV lead extraction as this procedure becomes increasingly common, driven by a growing number of device implantations as well as infections. Although regurgitation may be trivial immediately following the extraction, a subset of patients may develop severe TTR requiring surgical intervention for symptoms of RV failure. Data are sparse on the long-term impact of TTR, but recent trials on primary severe TR suggest an increase in cardiac events and mortality.¹⁴ Fibrosis and adhesion requiring more complex extraction appears to be a significant risk factor for development of this complication. Routine postprocedural echocardiography may not capture a significant proportion of patients who develop TTR given its variable timing of presentation. Serial echocardiography in high-risk patients may be a more effective approach for identification of this condition. Further data is needed to better classify patient and procedural risk factors, as well as long-term prognosis and effective treatment modalities for TTR.

Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at <https://doi.org/10.1016/j.hrcr.2021.05.013>.

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