INTRODUCTION

Inversion of the left atrial appendage (LAA) is an uncommon occurrence after heart surgery. It can easily be misdiagnosed as more commonly encountered pathology such as a vegetation, atrial myxoma, or thrombus in the left atrium. Accurate diagnosis is important as this is often found in the postsurgical setting where a delay in diagnosis or application of the wrong intervention based on misdiagnosis can have dire consequences. We present a case of an inverted LAA presenting with acute right ventricular (RV) failure after left ventricular assist device (LVAD) implantation.

CASE PRESENTATION

A 57-year-old male with a medical history of nonischemic cardiomyopathy due to Chagas disease with left ventricular (LV) ejection fraction of 10%-15%, presented to the hospital with decompensated heart failure. Right heart catheterization confirmed decompensated hemodynamics with markedly reduced cardiac index. He was well-known to the advanced heart failure service and was deemed as an appropriate candidate for durable mechanical circulatory support as a bridge to transplant. A Heart Mate III LVAD was placed without complication via midline sternotomy (Figure 1). There was slow flow in the left atrium as evidenced by echocardiography and color flow imaging. Preoperative transesophageal echocardiogram (TEE) showed a dilated left ventricle and left atrium with no masses present (Figure 1). There was slow flow in the left atrium as evidenced by echocardiographic and a well visualized LAA without clots present. Intraoperative TEE showed LVAD cannula in the left ventricle postimplant with no masses visualized in the left atrium (Figure 2). Color flow imaging shows mitral regurgitation without obstruction of the mitral inflow (Video 1). On postoperative day 3, his pulmonary artery pressures increased with an associated increase in right-sided filling pressures and concern for RV failure. His hemodynamic decline required an increase in inotropic support and LVAD speed in the setting of elevated pulmonary artery pressures of 50/24 mm Hg and central venous pressure of 16 mm Hg (Figure 3). In addition, a new echo-density at the level of left atrium was identified as concerning for a thrombus (Figure 4A). The decision was made to obtain computed tomography angiography (CTA) for further characterization of the new echogenicity in the left atrium, which showed compression of the left atrium from what appeared to be an extracardiac mass concerning for hematoma (Figure 4B). However, the CTA was read as not showing a mass “inside” the left atrium. Due to the discrepancy between the CTA and TEE, progressively worsening hemodynamics as evidenced by persistently elevated pulmonary artery pressures from indwelling Swan-Ganz catheter and increasing pressor requirements, the decision was made to obtain a TEE in the operating room to determine the need for RV support. On postoperative day 5, he was taken to the operating room where TEE demonstrated near total obstruction of the mitral valve inflow and pulmonary veins due to inversion of the LAA with functional mitral stenosis necessitating mediastinal exploration (Figures 5 and 6; Videos 2 and 3). At this time, the LVAD speed was 5,600 rpm with flow of 4.7 L/minute. The LVAD speed was reduced, and the patient was started on intravenous hydration while being prepped for mediastinal exploration. However, the LAA inversion resolved spontaneously during surgical prep. Given the high likelihood of recurrence, appendage ligation was performed. The base of the appendage was sized, and after confirming that there was no visible thrombus in the appendage on TEE, an AtriClip device was deployed along the base of the appendage to prevent repeat inversion (Figure 7). He had an unremarkable postoperative course with eventual discharge from the hospital.

DISCUSSION

Inversion of the LAA is rare, with fewer than 30 cases reported in the medical literature to date. The initial reports of LAA inversion were in pediatric patients usually in the postoperative setting, but spontaneous inversion has also been reported. Allen et al. first described this phenomenon by reporting two cases of LAA inversion in a 5-month-old undergoing tetralogy of Fallot repair and a 54-year-old female undergoing mitral valve repair. Since then, there have been several other case reports on intraoperative discovery of LAA mass following aortic valve replacement, mitral valve repair, and, more recently, LVAD implantation. The current prevailing theory is that the most likely cause of left atrial inversion is the intracardiac negative pressure environment created by an LV vent or as part of deairing maneuvers following cardiopulmonary bypass (CPB). The appendage usually spontaneously reverts to its anatomical position as the increasing left atrial pressure in atrial diastole counteracts this negative pressure. However, in some instances, the LAA does not revert and thus can present as a new density in the left atrium. It has been postulated that the size of the LAA orifice may play a
role, with shorter appendages with a wider base being more likely to invert in a negative pressure environment. 

The imaging modality of choice to confirm this diagnosis is TEE, due to its optimal visualization of the left atrium and LAA, which is in the near field of the TEE probe. A TTE demonstrating a homogenous, hyperechogenic mass arising from the anterolateral wall of the left atrium between the pulmonary vein and mitral valve should raise suspicion of an inverted LAA in the appropriate clinical setting. The inverted appendage is usually directed at the mitral valve orifice and has echo image intensity similar to the atrial wall. Doppler color flow interrogation does not demonstrate evidence of flow within the structure. Routinely performed intraoperative TEE helps in differentiating inverted LAA from thrombus, vegetation, or myxoma as these groups usually do not present as a new echogenicity in the immediate postoperative setting. Left atrial thrombus might be present but is less likely in our case, since patients are often anticoagulated and the intensity of a thrombus is usually different from the left atrial wall. Of note, our patient had Chagas cardiomyopathy, which is due to infection by Trypanosoma cruzi and is associated with LV enlargement with segmental or global systolic dysfunction. Right ventricular dysfunction and mural thrombus formation at the LV apex is also commonly seen, which would significantly complicate the implantation of the LVAD cannula as this is cored into the LV apex. Given the etiology of the patient’s cardiomyopathy, the diagnosis of thrombus, although not classically seen in the left atrium, was important to rule out. Computed tomography angiography can also be utilized but is limited in its ability for more specific intra-atrial resolution.

The LAA inversion associated with CPB is usually diagnosed early by intraoperative TEE during or immediately after weaning off CPB. In our case, initial intraoperative TEE after LVAD placement demonstrated no mass. Moreover, acute RV failure was seen 3 days later, suggesting that LAA inversion was unrelated to perioperative maneuvers and most likely due to negative pressure induced by the presence of LVAD. Our patient had mitral inflow obstruction and functional mitral stenosis due to LAA inversion. This resulted in pulmonary hypertension and elevated right-sided filling pressures with associated acute RV failure. This presents a clinical dilemma as the initial approach to elevated right-sided filling pressures and pulmonary capillary wedge pressure is diuresis. In this case, this would exacerbate hemodynamic compromise if acute RV failure secondary to LAA inversion is not recognized in a timely fashion. In our case, the LAA inversion resolved spontaneously after intravenous hydration and reduction in LVAD speed. It is not possible to clearly delineate the contribution of LVAD speed reduction or intravenous hydration to the resolution of LAA inversion. However, we hypothesize that LVAD speed directly contributes to the negative suction force and as such reduction in speed most likely resulted in resolution of LAA inversion in this setting.

Khachatryan et al. reported a similar case in a 17-year-old with nonischemic cardiomyopathy status post-LVAD. The patient experienced hypoxic cardiopulmonary arrest 13 days after LVAD implantation requiring extracorporeal membrane oxygenation. A TEE discovered large mobile echo-density in the left atrium, and the patient was taken back to the operating room where an inverted LAA was discovered. The appendage was manually expressed, and AtriClip was applied to prevent future thrombus formation. This finding is of immense significance given the increased utilization of LVADs in the advanced heart failure patient population. An important clinical consideration in these patients who often have some element of RV dysfunction at baseline is the development of RV failure following LVAD implantation. Patients requiring an RV assist device in this setting typically have a higher morbidity and mortality necessitating heart transplant as bailout if possible.

In addition, the new HeartMate III has a continuous centrifugal flow design with intermittent speed alteration that creates artificial pulsatility. This theoretically creates an intracardiac environment with variable negative suction force more amenable for spontaneous LAA inversion. Additionally, these patients have long-standing heart failure with a thin-walled atrium due to chronically elevated pressures. As such, the transplant team has to be attuned to the possibility of LAA inversion as an etiology of RV failure in the right setting as timely intervention is critical.

The possible complications of LAA inversion includes necrosis of the appendage from compromised perfusion, thrombus formation with associated risk of embolism and mitral valve inflow obstruction leading to functional mitral stenosis, pulmonary hypertension, and RV failure, as seen in our case. Meticulous assessment of RV function quantitatively with TAPSE, RV S velocity, fractional area change, and invasive hemodynamics is critical in the immediate post-LVAD implantation setting.

Given the low incidence of LAA inversion, there is currently no standardized treatment approach, and patients are evaluated for conservative versus surgical management on a case-by-case basis. The likelihood of spontaneous eversion, reinversion, and complications

Figure 1 TEE midesophageal four-chamber view prior to LVAD implantation showing dilated left ventricle and left atrium with no masses present.

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Figure 2  (A) TEE midesophageal two-chamber view obtained intraoperatively just prior to LVAD implantation showing dilated LAA with slow flow and echogenic smoke. The LAA is also well visualized without evidence of clot. Papillary muscle is also visualized in the left ventricle.  (B) TEE midesophageal four-chamber view just after LVAD implantation showing cannula in the LV apex.

Figure 3  TTE showing markedly reduced RV function as evidenced by TAPSE of ~0.8 cm and RV S’ velocity of 3.48 cm/sec.

Figure 4  (A) TTE apical four-chamber view showing new left atrial mass.  (B) Computed tomography of the chest with contrast showing mass compressing left atrium.
based on LAA appendage orifice size and presence of triggering factors should be taken into consideration to determine the need for surgical intervention.\(^1\)\(^-\)\(^9\) Digital manipulation, forceps eversion, and percutaneous and surgical LAA ligation are the current available modalities. In our case, the LAA reverted back to its anatomic position spontaneously during preparation for mediastinal exploration due to the reduction of LVAD speed and volume loading. However, due to the high likelihood of recurrence, atrial appendage ligation was performed.

The size of the atrial orifice to appendage height dimension might play a role in LAA inversion.\(^1\) These parameters should be employed as a standard measurement in LVAD patients with consideration for empiric ligation of the atrial appendage. This intervention would add minimal risk with a possibility to mitigate a complication with the potential for significant morbidity and mortality. Further research is needed to better understand its etiology given the increasing prevalence of LVADs in the population.

**CONCLUSION**

This is the first reported case that describes LAA inversion causing RV failure from obstruction of the mitral valve inflow and functional mitral stenosis in association with LVAD implantation. Given the increased utilization of LVADs in the heart failure population, the transplant team must be cognizant of the possibility of LAA inversion as an etiology of RV failure. The importance of LVAD speed assessment cannot be overstated as this was a major contributor in our case. If the transplant team is more familiar with this possible complication, then initial attempts at volume loading and LVAD speed reduction might noninvasively address this issue. However, the need for more definitive LAA ligation should also be a consideration given the risk for catastrophic complications. Perhaps inversion of LAA might emerge as a more common complication of LVADs.

**SUPPLEMENTARY DATA**

Supplementary data related to this article can be found at https://doi.org/10.1016/j.case.2020.03.004.

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