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Percutaneous Transcatheter Therapies for the Management of Left Ventricular Assist Device Complications

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Abstract

Heart failure is a growing epidemic in the United States and throughout the world. The utilization of continuous-flow left ventricular assist devices (LVADs) has greatly increased over the last decade. In addition, a limited supply of organ donors has led to a rise in the use, and duration, of LVADs for destination therapy. The increased use of LVAD therapy has led to the observation of mechanical complications such as device thrombosis, *de novo* aortic insufficiency, and outflow graft stenosis, all of which are associated with prolonged LVAD support. Surgical repair for these complications remains the therapy of choice; however, surgery may be associated with high operative risk in some patients. The purpose of this article is to discuss mechanical complications associated with LVAD therapy and interventional transcatheter therapies that have been used to solve these increasingly complex problems.

Keywords

left ventricular assist device; LVAD; LVAD thrombosis; aortic insufficiency; outflow graft stenosis; HeartMate II; HeartWare

Heart failure has become a major public health epidemic, with approximately 5.1 million patients diagnosed with this condition in the United States alone; this is expected to increase by 25% by 2030.[1] Left ventricular assist devices (LVADs) have become vital in the treatment of end-stage heart failure, with more than 12,000 implants between April 2008 and December 2014, and almost 2500 implants in 2014 alone.[2] Given the relatively unchanged number of heart transplants over the last two decades, the number of patients receiving LVADs for treatment of end-stage heart failure is expected to continue to rise.[3] Currently, there are two Food and Drug Administration (FDA)-approved continuous-flow LVADs (CF-LVADs) in the United States: HeartMate II (HMII; Thoratec), approved for bridge to transplant (BTT) and destination therapy (DT); and HVAD (HeartWare), approved for BTT only. [4] Since the FDA approval of HMII for DT in 2010, the proportion of patients receiving CF-LVADs for DT has risen significantly, peaking at 46% of all LVAD implantations in

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2014. Importantly, the survival rates with CF-LVAD support remain excellent, with 1-year and 2-year survival rates at 80% and 70%, respectively, for both DT and BTT patients.[2]

The increasing use of CF-LVADs for DT, along with long transplant wait times,[3,5] has led to a significant number of patients who are supported by CF-LVADs for an extended duration of time. The increased treatment time with CF-LVADs has resulted in a rise in complications associated with CF-LVAD support.[2] The common complications associated with CF-LVAD therapy include gastrointestinal bleeding (event rates ranging from 0.27-0.67 gastrointestinal bleeds per patient-year of support,[6-8] thromboembolic events (up to 0.17 events per patient-year of support despite anticoagulation, with up to 12% experiencing at least one event, [7,9] and infection (up to 12% over the life of the device).[10] Mechanical complications, such as device thrombosis, *de novo* aortic insufficiency, and outflow graft stenosis, have also been encountered at increasing frequency. Surgery remains the therapy of choice for these mechanical complications; however, a subset of patients may have high operative risk, precluding them from undergoing surgical management. In this article, we will discuss the common mechanical complications associated with CF-LVAD support and interventional transcatheter therapies that have been used to solve these increasingly complex problems.

Aortic Insufficiency

Incidence

The development of aortic insufficiency (AI) has been recognized as an important complication of prolonged CF-LVAD therapy.[11-13] AI while on CF-LVAD support reduces the efficiency of the LVAD as a portion of the forward flow from the LVAD outflow cannula enters back into the left ventricle via the regurgitant aortic valve, creating a circulatory loop with diminishing forward flow and, subsequently, decreasing end-organ perfusion.

The rates of mild or worse AI have been shown to be as high as 52% with CF-LVAD support, with numerous studies demonstrating that duration of CF-LVAD support and closed aortic valve are the strongest risk factors associated with development and progression of de novo AI.[12-19] Pak et al showed that freedom from AI in patients with CF-LVADs was 75% at 12 months of support, with AI being more common in patients with aortic valves that failed to open.[12] Similarly, in the study by Jorde et al, freedom from at least mild AI was 77% after 1 year of CF-LVAD support, with moderate AI developing in 38% of patients after 3 years of support.[13] A persistently closed AV was again strongly associated with development of AI.[13]

Development of AI in patients with CF-LVADs often leads to clinical symptoms of heart failure and has been shown to reduce survival.[13,17,18] Toda et al noted a statistically significant reduction in survival in patients with any degree of AI (2-year survival 93% vs 82%; P=.02).[18] In their cohort, among patients who died with *de novo* AI after at least 2 years of LVAD support, 6 of the 7 deaths were secondary to development of heart failure.[18] In the study by Jorde et al, 7 out of 21 patients that developed at least moderate AI had symptomatic heart failure and required surgical intervention.[13] Similarly, in another study, patients with mild or greater AI had increased rates of cardiac events, defined as heart failure or arrhythmia requiring admission (33.3% vs 3.5%; P=.03).[20] As is evident from these

results, AI is a common and persistent issue with LVAD support, often with significant clinical implications.

Pathophysiology and diagnosis

The mechanical unloading of the left ventricle during LVAD support results in decreased aortic valve opening when compared with normal physiology. This leads to increased closure time, exposing the valve leaflets to extensive collagen deposition and subsequent commissural fusion.[21,22] Mudd et al described a series of 9 patients with HMII, of which 8 patients had evidence of commissural fusion of the native aortic valve leaflets at the time of explant during heart transplantation.[21] Additionally, increased shear forces from the flow through the outflow cannula (as well as retrograde flow from the outflow cannula) predispose the aortic root to dilation, further increasing risk of AI.[12,22,23] Furthermore, increased transvalvular gradients during high LVAD support likely contribute to development of AI.[24]

Diagnosis of symptomatic AI should be suspected in a patient with LVAD and symptoms of heart failure, such as lower-extremity edema, dyspnea, fatigue. A transthoracic echocardiogram (TTE) should be obtained to quantify the degree of AI. Measuring AI in patients with CF-LVADs is challenging, as it tends to be present throughout the cardiac cycle compared to only in diastole during normal physiology. A recent study by Grinstein et al showed that AI using traditional TTE grading criteria may be underestimated and novel TTE parameters (peak systolic to diastolic velocity ratio and the diastolic acceleration of the LVAD outflow cannula) may more accurately measure AI in patients with continuous flow LVADs.[25,26] LVAD parameters are often not helpful in diagnosis of AI, as the flows are preserved despite a low cardiac output and no significant change in the power requirement is observed.[27] Therefore, right heart catheterization should be performed to assess true cardiac output (Figure 1).

Management

Initial management of symptomatic heart failure in the setting of moderate or severe AI should involve diuresis and afterload reduction. If symptoms fail to improve, ramp study for LVAD speed optimization should be considered.[13] If symptoms persist despite speed optimization, definitive therapy with surgical or percutaneous means may be required.

The International Heart and Lung Transplantation guidelines recommend surgical intervention with either aortic valve replacement or repair during LVAD implantation in patients who have greater than mild AI at baseline.[28-30] In patients who develop AI after LVAD, so called *de novo* AI (Table 1), surgical treatment includes replacement with bioprosthetic valve[31] (mechanical valves should be avoided due to high risk of thrombosis given low-flow state,[24,32] valve repair using a coaptation stitch [“Park’s stitch”],[33,34] or closure). While surgical management may be appropriate for many patients, re-do surgery is complex and high risk. Therefore, in select patients, less invasive, non-surgical transcatheter options must be explored. Transcatheter options for treatment of *de novo* AI include application of a septal occluder device to close the aortic valve or percutaneous transcatheter aortic valve replacement (TAVR).[35-39] Grohmann et al first reported the use of an Amplatzer

post-infarct muscular ventricular septal defect (VSD) occluder (St. Jude Medical) in a patient presenting with severe AI and symptoms consistent with congestive heart failure.[35] The aortic valve was percutaneously closed via left subclavian approach under transesophageal echocardiographic (TEE) guidance using a 24 mm VSD occluder, sized using a preoperative computed tomography (CT) scan. While there was an improvement in the hemodynamics, the patient experienced significant transfusion-requiring hemolysis before ultimately being successfully discharged home.[37] Since then, numerous studies have been published using the Amplatzer Cribriform device (St. Jude Medical). Russo et al described a patient who underwent surgical aortic valve repair 8 months post LVAD implantation and subsequently presented in cardiogenic shock 3 months later due to recurrent severe AI.[36] Using transfemoral approach with a 7 Fr sheath, the left ventricular side of the Amplatzer occluder device was first exposed followed by the deployment of the aortic side under TEE guidance. The device was successfully deployed with no further AI and significant hemodynamic improvement. Subsequently, the group has published further results of their experience with the Cribriform device (Figure 2).[35] In 5 patients with AI and excessively high surgical risk, percutaneous closure successfully improved AI (from severe to mild or absent; P=.04) with concurrent improvement in the hemodynamics. There were no changes in the LVAD parameters after the AV closure. In 1 of these patients, the device embolized to the aortic arch on day 4 and was successfully retrieved percutaneously. In this sick cohort, 2 patients were alive at 30 days with well-positioned devices and no residual AI. In a larger retrospective cohort of 10 LVAD patients with high surgical risk (STS mortality risk, 22%) and severe AI who underwent Cribriform septal occluder implantation, the 6-month survival was 30%.[39] In this cohort, device-to-annulus ratio of less than 0.9 and lack of significant preoperative right heart dysfunction was associated with improved survival. Smaller devices may apply less pressure on the interventricular septum and hence may not affect the RV filling and outflow, leading to improved survival. It is important to note that with the Cribriform device deployed, the patient is fully reliant on the LVAD and a device malfunction can quickly become fatal.

Another strategy to percutaneously treat AI involves TAVR (Figure 3). A case report by Khan et al described a patient with history of aortic valve replacement and homograft conduit who underwent LVAD implantation 10 years later, and subsequently developed medically refractory severe symptomatic AI.[40] The patient was deemed to not be a surgical candidate and he successfully underwent a Melody transcatheter pulmonary valve placement in the aortic position via femoral approach. There was trace AI after valve deployment, with significant improvement in hemodynamics. The patient was discharged on day 6 and survived 10 additional months. Another case by D'Ancona used an oversized 29 mm Edwards Sapien valve (Edwards Lifesciences) via left anterior thoracotomy under femo-femoral bypass.[41] The valve was successfully deployed without immediate complications, but no information regarding longer-term follow-up is available. In another case report by Santini et al, a patient with refractory symptomatic AI and no surgical options underwent 29 mm CoreValve (Medtronic) implantation via femoral approach.[42] A second valve was implanted immediately due to periprosthetic regurgitation with minimal residual leak after deployment of the second valve. The patient recovered and was successfully discharged. While these case reports are encouraging, TAVR in AI without valvular calcification should

be approached with extreme caution due to concerns regarding anchoring of the valve and risk of embolization. Additionally, there is high likelihood of fusion of the leaflets of the newly implanted bioprosthetic valve.[43]

In summary, percutaneous treatment of LVAD-acquired and clinically significant moderate-to-severe AI, not amenable to medical management (use of optimal LVAD speed and lower mean arterial pressure goal), has been documented. Percutaneous interventional therapies are important options for patients who are not deemed to be surgical candidates or have high surgical risk. These therapies should be approached with caution since associated outcomes are not ideal and long-term data are lacking.

LVAD Thrombosis

Incidence

Pump thrombosis, defined as a clot located either in the inflow cannula, the central rotary component, or in the outflow graft, is the leading cause of LVAD failure.[44] The most common location of thrombosis is typically within the central rotary component and will be the focus of this section. In patients implanted with HMII, rates of pump thrombosis increased dramatically in 2011 and were reported between 8%-12%, with median time to thrombosis decreasing from 18.6 months prior to 2011 to just 3 months since then.[45-48] Recently, a reduction of device thrombosis rate was reported in the PREVENT study as a result of improved implantation techniques and focus on heparin bridging and appropriate long-term anticoagulation.[49] Similar to HMII, the rate of thrombosis with HVAD has been high, up to 8% at 1 year, with median time to thrombosis event of 245 days.[50] The presence of pump thrombosis is associated with significant morbidity and mortality, with data from the Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) suggesting a drop in 12-month survival from 82% for the overall cohort to 70% for patients who have first pump exchange due to thrombosis ($P<.001$).[48] Najjar et al also noted decreased 1-year survival in those with pump thrombosis compared to those without evidence of pump thrombosis (69.4% vs 85.5%; $P=.21$).[50] Additionally, many of these patients require pump exchange, which can be associated with significant morbidity as well as economic burden.[51]

Pathophysiology and diagnosis

Several mechanisms responsible for LVAD thrombosis have been proposed, but the pathophysiology remains unclear. The underlying etiology is likely multifactorial, with activation of the coagulation cascade[52] along with shear-stress induced platelet dysfunc tion[53] playing a major role. Several pump-related (ie, intrinsic heat generated by the pump, malposition of the inflow cannula, regions of flow stasis within the circuit), patient-related (ie, atrial fibrillation, non-compliance, intrinsic hyper coagulable state, etc), and management-related risk factors (inappropriate antithrombotic and antiplatelet agents) may increase risk of pump thrombosis. In the study by Najjar et al, mean arterial pressure greater than 90 mm Hg, aspirin dose <81 mg, and international normalized ratio (INR) <2 were associated with increased risk of pump thrombosis.[50]

Pump thrombosis should be suspected in patients with symptoms of left heart failure and/or isolated elevation of lactate dehydrogenase (LDH) levels (2.5× greater than the upper limit of normal), evidence of hemolysis (elevated LDH, plasma free hemoglobin, dark urine), sustained power elevation (>24 hours or power increase of 2W above base-line), or decreasing LVAD flows.[54-59] If any of these findings are present, a TTE is the preferred next step. Echocardiographic parameters such as rightward deviation of septum, increased left ventricular end-diastolic diameter, or aortic valve opening with each cardiac cycle are suggestive of pump thrombosis and further evaluation with a “ramp” test may be warranted. Uriel et al have demonstrated that failure of the left ventricular end-diastolic dimension (LVEDD) to change with increasing LVAD speeds is a strong predictor of device thrombosis in HMII patients.[57] Ramp test should not be used to diagnose device thrombosis in HVAD patients as they are less informative.[58] In patients with HVADs, analysis of the log files may allow for earlier detection of LVAD thrombosis.[60] Importantly, CT of the chest should be ordered in patients with suspected pump thrombosis to rule out mechanical issues such as malpositioned inflow cannula or to identify thrombus within the inflow or outflow cannula.[61-63] An algorithm for diagnosis and management of LVAD thrombosis is shown in Figure 4.

Management

Pump thrombosis is treated by either medical therapy alone or in combination with surgical management via either pump exchange or transplantation. Regardless of the route chosen, heparin and antiplatelet agents are requisite therapy for suspected or confirmed pump thrombosis and more aggressive medical therapy may involve the addition of intra-venous tissue plasminogen activator (tPA). ISHLT provides a consensus document with an algorithm for the diagnosis and treatment of device thrombosis in a setting of HMII.[55]

Thrombus in an HVAD (which tends to be laminar fibrin deposition) is generally felt to be more amenable to thrombolytic therapy than thrombus in an HMII (which tends to be globular clot and therefore less amenable to thrombolytics at the time of detection).[54,60,64] Stulak et al reported results in a multi-center cohort of 175 patients who underwent HVAD implantation.[65] In this retrospective study, there were 36 pump thromboses, of which 29 were initially treated medically (majority with tPA) and 7 were initially treated surgically (device exchange). Significant morbidity and mortality were associated with medical management (hemorrhagic stroke in 21%, need for urgent device exchange or transplant in 21%, and death in 10%) compared with no early complications in those initially treated surgically. Similarly, Levin et al reported significant improvement in overall survival in patients treated with early device exchange compared with medical management in patients with suspected pump thrombosis.[66]

While surgical management is the treatment of choice for pump thrombosis,[45,53,65,66] some patients may not be candidates due to significant morbidity and mortality, especially in the setting of an acutely unstable patient.[50,67,68] As such, thrombolysis may be necessary in a select few patients. While there have been case reports of successful use of glycoprotein IIb/IIIa inhibitors for pump thrombosis,[69,70] in larger studies it has been associated with poor therapeutic response and significant morbidity.[71] Given these results, the risks of eptifibatide

outweigh the benefits in treatment of pump thrombosis and they were not included in the algorithm proposed by Goldstein et al.[55]

Compared with glycoprotein IIb/IIIa inhibitors, thrombolytics have demonstrated higher success and lower complication rates. In the trial of HVAD patients by Najjar et al, success rate with thrombolytics was 63% (12/19), with similar success rates when thrombolytics were administered peripherally (6/8) or centrally, within the LV cavity (4/7).[50] The route of administration was unknown in 4 patients. The complications included 5 bleeding events, with 2 hemorrhagic cerebrovascular accidents, 2 gastrointestinal bleeds, and 1 pocket implantable cardioverter defibrillator hematoma, and the study did not indicate if there were differences in the incidences of adverse events between peripheral and central tPA. In a case series of 8 patients who failed therapy with heparin and eptifibatide for LVAD throm bosis, intraventricular alteplase was administered via pigtail catheter in the left ventricle at 1 mg/min over 30-50 minutes with concomitant heparin administration.[72] Of the 8 patients, 3 patients were successfully treated, 3 patients died, and 1 patient underwent emergent LVAD exchange while another under went heart transplantation. Similarly, in a small case series of 2 patients by Thenappan et al, patients received intraventricular alteplase via a pigtail catheter in the left ventricle (Figure 5).[73] Unlike the previously described case series, these patients received lower bolus dose of alteplase but with longer duration (alteplase 25 mg bolus and 1 mg/hour for 30 hours in 1 patient and 25 mg bolus followed by 1 mg/hour for 96 hours in 1 patient). In each case, the markers of thrombosis improved significantly over the course of thrombolytic therapy. There have been many other case reports of successful treatment of LVAD thrombosis with intraventricular thrombolytics as well.[74,75]

As is evidenced from the results above, thrombolysis with tPA may successfully treat pump thrombosis in some HVAD patients, albeit with significant risk of bleeding, specifically hemorrhagic cerebrovascular accident. Jorde et al have shown that while overall success rate of tPA therapy for HVAD pump thrombosis was only 57%, when using log file parameters, the success of medical therapy increases to 77%.[60] Using this algorithm, those with lower measures of expected power and gradual power increase are more likely to benefit from thrombolysis compared with those who suffer sudden power increase.

In summary, treatment for pump thrombosis varies on the type of device, with HMII patients experiencing significantly higher morbidity and mortality with thrombolysis than HVAD patients. Therefore, thrombolysis should be avoided in HMII patients and pump exchange should be the treatment of choice. In those with HVADs, recent data suggest that initial surgical management is preferred, but medical management may be selected in those with appropriate log file parameters. In these patients, the data for peripheral vs central administration of tPA are lacking. Intraventricular thrombolytics to maximize the dose administered directly to the LVAD rotor may be an effective technique to treat thrombosis in patients with HVADs. Using either radial or femoral approach, a pigtail catheter in the left ventricle can be used to deliver tPA as a bolus followed by either short or long course of tPA while carefully monitoring LVAD and laboratory parameters for signs of improvement.

Outflow Graft Stenosis

Incidence

Outflow graft stenosis is a very rare complication of LVAD therapy (Table 2).[2] The rates of this complication are significantly lower than the more common structural complications such as LVAD thrombosis or *de novo* AI described in this paper. Clinical implications of outflow graft stenosis or thrombosis are similar to the LVAD thrombosis described above, with decreased LVAD flows, increased power requirements, and clinical symptoms of heart failure.

Pathophysiology and diagnosis

The outflow graft is prone to various mechanical as well as non-mechanical issues. Mechanical obstruction can be caused by kinking of the out-flow graft or due to bend relief disconnect, which is the most common cause for outflow graft stenosis.[76] On the other hand, non-mechanical causes of outflow graft obstruction include aortic atherosclerosis resulting in suboptimal surgical landing site, fibrotic changes of the aorta, or thrombosis of the outflow graft.[62,77,78] The mechanisms of these complications are not fully understood. If thrombosis is diagnosed, low flow state within the outflow graft may be the culprit.[79]

Patients with outflow graft stenosis present in the same manner as those with LVAD thrombosis, with LVAD parameters showing decreasing flows and increasing power requirements, laboratory evidence of hemolysis, and clinical symptoms of heart failure. As part of the algorithm, Goldstein et al recommend contrast CT scan of the chest to assess the inflow cannula position and the outflow graft.[55]

Treatment

Management of outflow graft is often surgical, especially if a mechanical issue such as kinking of the outflow graft is diagnosed. Acharya et al describe a case where significant outflow graft stenosis near the aortic anastomosis was identified with a CT scan and subsequently treated with surgical graft revision.[63]

Percutaneous treatment of outflow graft stenosis or thrombosis has been described in multiple case reports in the last few years. In a case report by Retzer et al, a patient with HVAD was diagnosed with outflow graft stenosis (90%) resulting in a substantial gradient of 120 mm Hg across the lesion.[78] Serial balloon dilations followed by placement of 10 × 38 mm, stainless-steel, balloon-expandable Atrium iCAST stent (Maquet Gettinge Group) resulted in immediate improvement in LVAD parameters (flow increased from 2.5 L/min to 9.0 L/min at 2900 rpm, necessitating drop of the speed to 2350 rpm, with flow of 4 L/min), with reduction of the gradient to 30 mm Hg (Figure 6). Similarly, in a case series of 3 patients with HVADs presenting with heart failure symptoms and diagnosed with graft outflow stenosis, all patients were treated with percutaneous transcatheter dilation of the outflow graft and implantation of the Smart Control 12 × 40 mm stent.[80] Another case report by Ganapathi et al showed percutaneous treatment of outflow graft pseudoaneurysm in a patient with prohibitive surgical risk for open repair.[79] A Gore Excluder iliac limb stent graft (W.L. Gore) was successfully deployed with interval follow-up imaging showing well-

positioned stent-graft without evidence of endoleak and complete resolution of the pseudoaneurysm. Another case described the use of a 20 × 55 mm AneuRx iliac limb stent-graft (Medtronic) to treat an arterio-bronchial fistula of the outflow graft.[81]

In a case report by Abraham et al, an outflow graft thrombosis of HMII was treated via subclavian approach and retrograde access to the outflow graft.[82] A 9 × 59 mm Atrium stent was deployed in the outflow graft followed by deployment of an additional stent, resulting in satisfactory improvement in angiographic and clinical findings. The patient was discharged 4 days later on anticoagulation. Similarly, an HVAD patient with history of two prior LVAD exchanges was diagnosed with LVAD outflow graft thrombosis.[83] This was successfully treated via brachial arterial approach along with embolic protection in the carotid artery via femoral approach. A 12.0 × 61 mm covered Atrium stent was deployed and postdilated with a balloon with significant improvement in the degree of stenosis. The patient did well and was discharged on postoperative day 8. The patient did well until 8 months later, at which time the patient had signs of hemolysis and ultimately underwent heart transplantation. It should be noted that when stenting the outflow graft, risk of embolism is present, especially when thrombosis of the outflow graft is present, and it is reasonable to protect supra-aortic branches with dilated balloons or carotid filters.[82-84]

Percutaneous therapies for outflow graft stenosis, a rare complication of LVAD therapy, have been described in the literature. While long-term follow-up data are not available, percutaneous outflow graft stenting is a viable option for many patients with LVAD outflow graft stenosis and its clinical application is expected to increase due to the rising number of LVAD implantations and longer duration of LVAD therapy. However, more long-term follow-up data are needed regarding the durability of outflow graft stenting compared with surgical replacement. It is evident that outflow graft stenting is a viable alternative and should be explored prior to proceeding with surgical replacement.

LVAD Decommissioning

A small subset of patients with LVADs may require decommissioning of the LVAD either due to myocardial recovery or due to pump failure. During cases of acute pump failure, valveless CF-LVADs allow retrograde flow from the aorta resulting in acute elevation of left-sided filling pressures akin to acute AI.[85] In addition, stasis of flow in the outflow graft leads to thrombus formation, which can result in distal embolization with potentially devastating complications. While the outflow graft is expected to thrombose over time due to the low-flow state, the risk of embolization persists until it is completely thrombosed. Surgical explanation of CF-LVADs often necessitates sternotomy with cardiopulmonary bypass, which carries an inherent risk in this patient population. Minimally invasive surgical techniques that reduce morbidity associated with explantation have been recently described.[86-88]

Percutaneous LVAD decommissioning has the ability to quickly stabilize patients who experience sudden pump failure resulting in clinical deterioration. Acute tamponade of the outflow graft using 14-16 mm peripheral balloons has been described by Chrysant et al.[89] In this case series, both patients presented with acute decompensation in the setting of pump

failure and each underwent balloon tamponade of the outflow graft with eventual surgical pump exchange after clinical stability. A catheter-based outflow graft occlusion using 22 mm Amplatzer Vascular Plug II (St. Jude Medical) has been described in a patient with HMII who did not wish to undergo repeat sternotomy for device explantation after myocardial recovery.[90] Recently, percutaneous decommissioning of HVAD has been described with a 14 mm Amplatzer septal occluder successfully deployed in the outflow graft in a patient with pump thrombosis who was not a candidate for device exchange (Figure 7).[91]

Percutaneous LVAD decommissioning of the LVAD outflow graft is an intriguing option for a select group of patients as it allows for rapid correction of retrograde flow within the outflow graft. It can also exclude the outflow graft in patients with myocardial recovery in whom device explantation is not an option.

Conclusion

LVADs have become critical in the treatment of end-stage heart failure with significant rise in device implantations and duration of LVAD therapy. The increasing use of LVADs has led to the observation of increasing complications. In some patients, surgical therapy may be associated with prohibitively high risk, thereby leading to the development of minimally invasive transcatheter interventional therapies to treat LVAD-associated AI, LVAD thrombosis, outflow graft stenosis, and pump failure.

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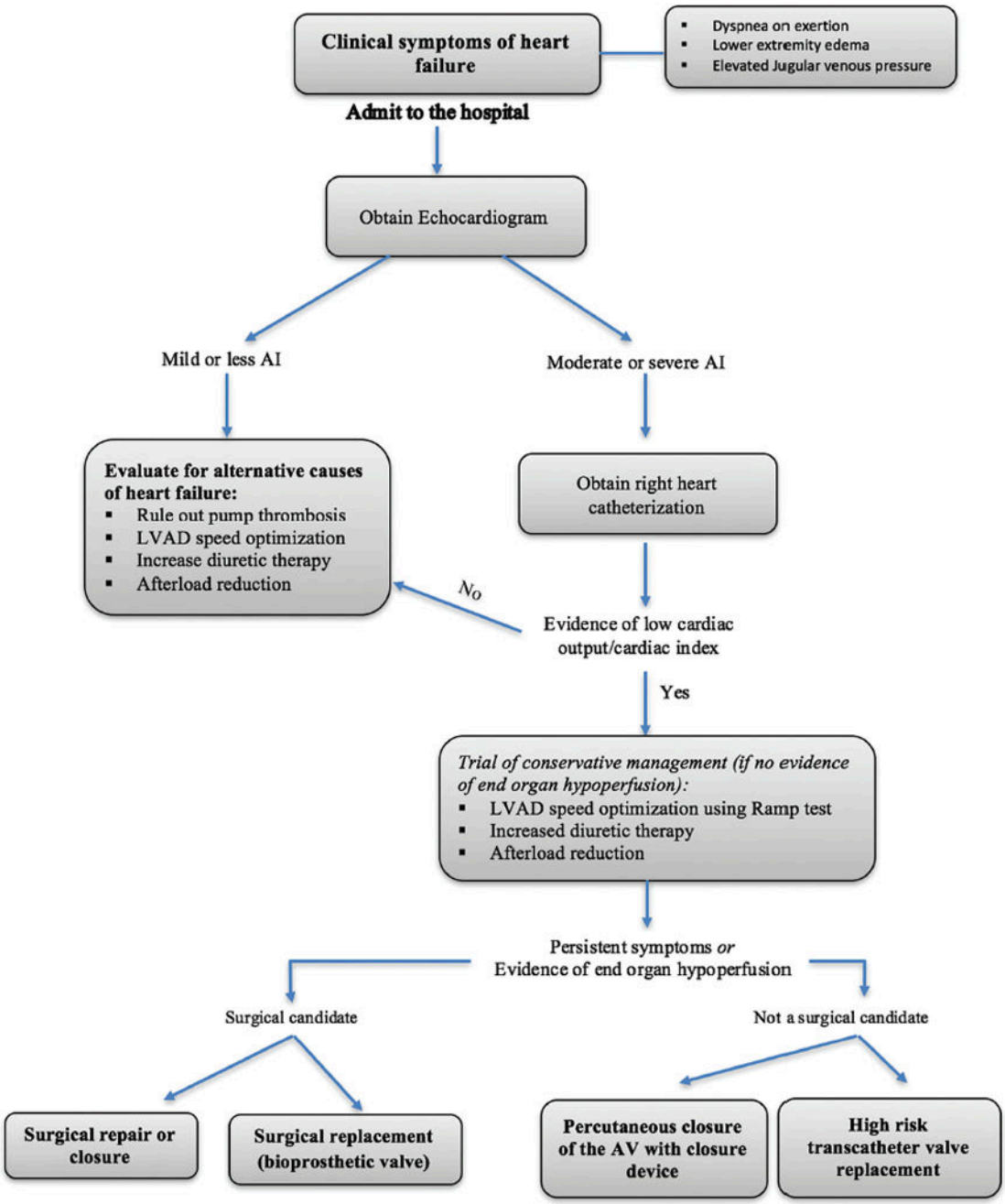

FIGURE 1.
Diagnosis and management algorithm for *de novo* aortic insufficiency.

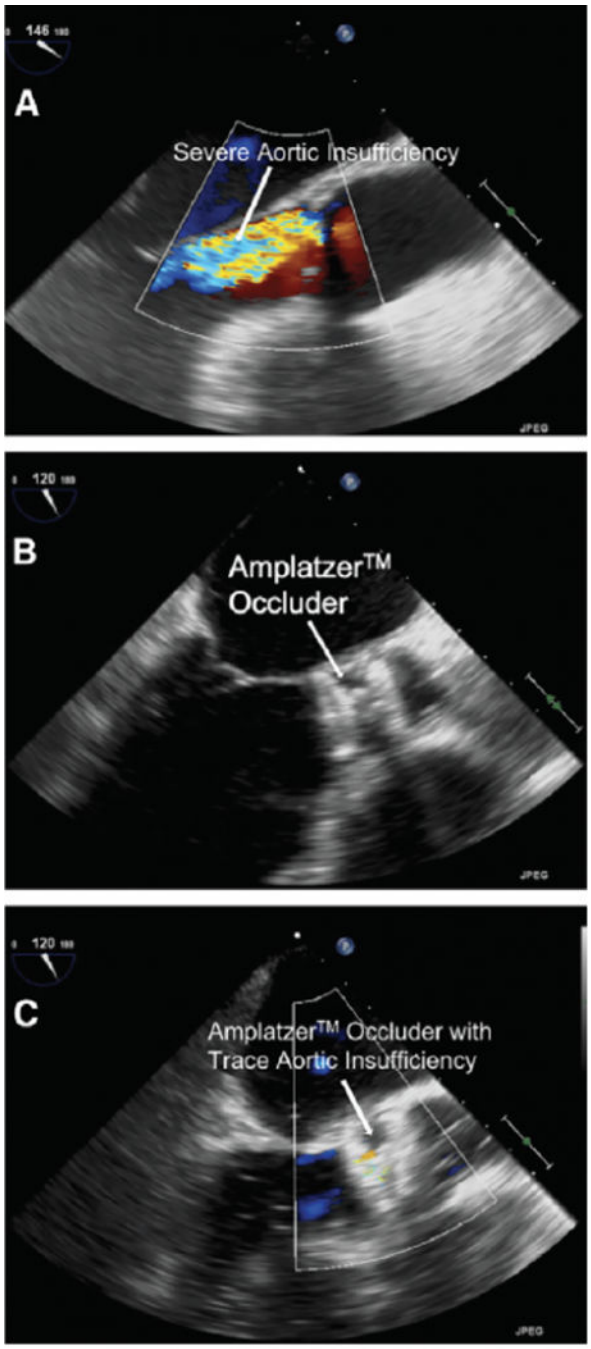

FIGURE 2.

Treatment of aortic insufficiency using the Amplatzer occluder device. (A) Transesophageal echocardiogram showing severe aortic insufficiency. (B) Deployment of the occluder. (C) Transesophageal echocardiogram demonstrating trace residual aortic insufficiency after device deployment. Reprinted with permission from Parikh et al.[35]

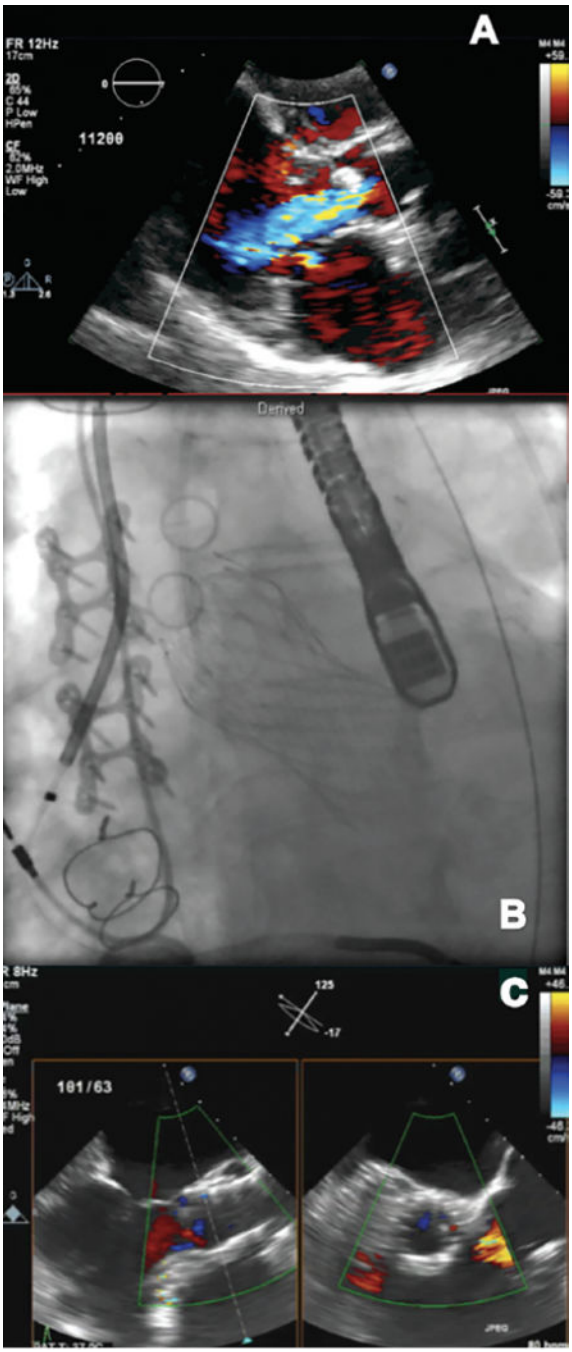

FIGURE 3.

Transcatheter aortic valve replacement in a patient with left ventricular assist device (LVAD) and severe symptomatic aortic insufficiency (AI). (A) Parasternal long-axis view on the transthoracic echocardiogram demonstrating severe AI. (B) Fluoroscopic image demonstrating a CoreValve deployed in the aortic position. (C) Transesophageal echocardiogram demonstrating significant improvement in AI after CoreValve deployment.

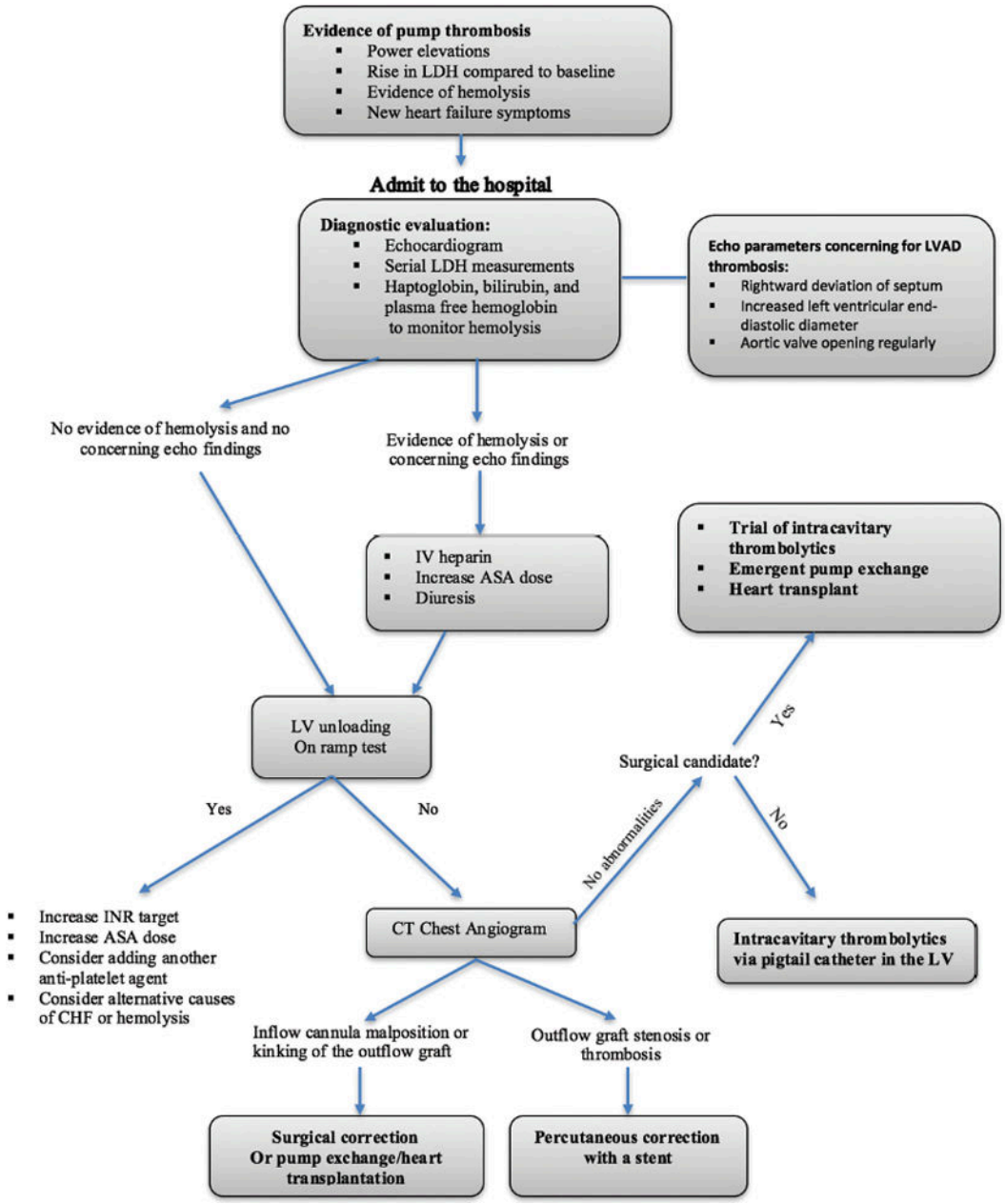

FIGURE 4.
Algorithm for diagnosis and management of pump thrombosis.

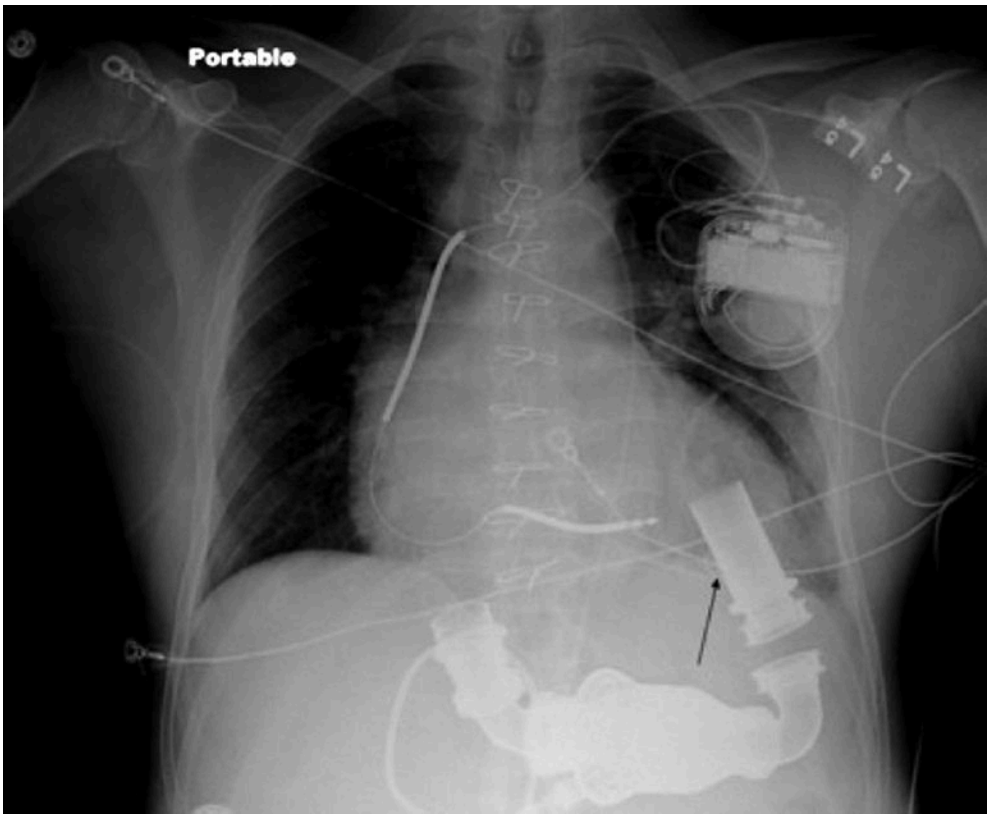

FIGURE 5.
Chest x-ray demonstrating the location of pigtail catheter (arrow) in the left ventricle. Reprinted with permission from Thenappan et al.[73]

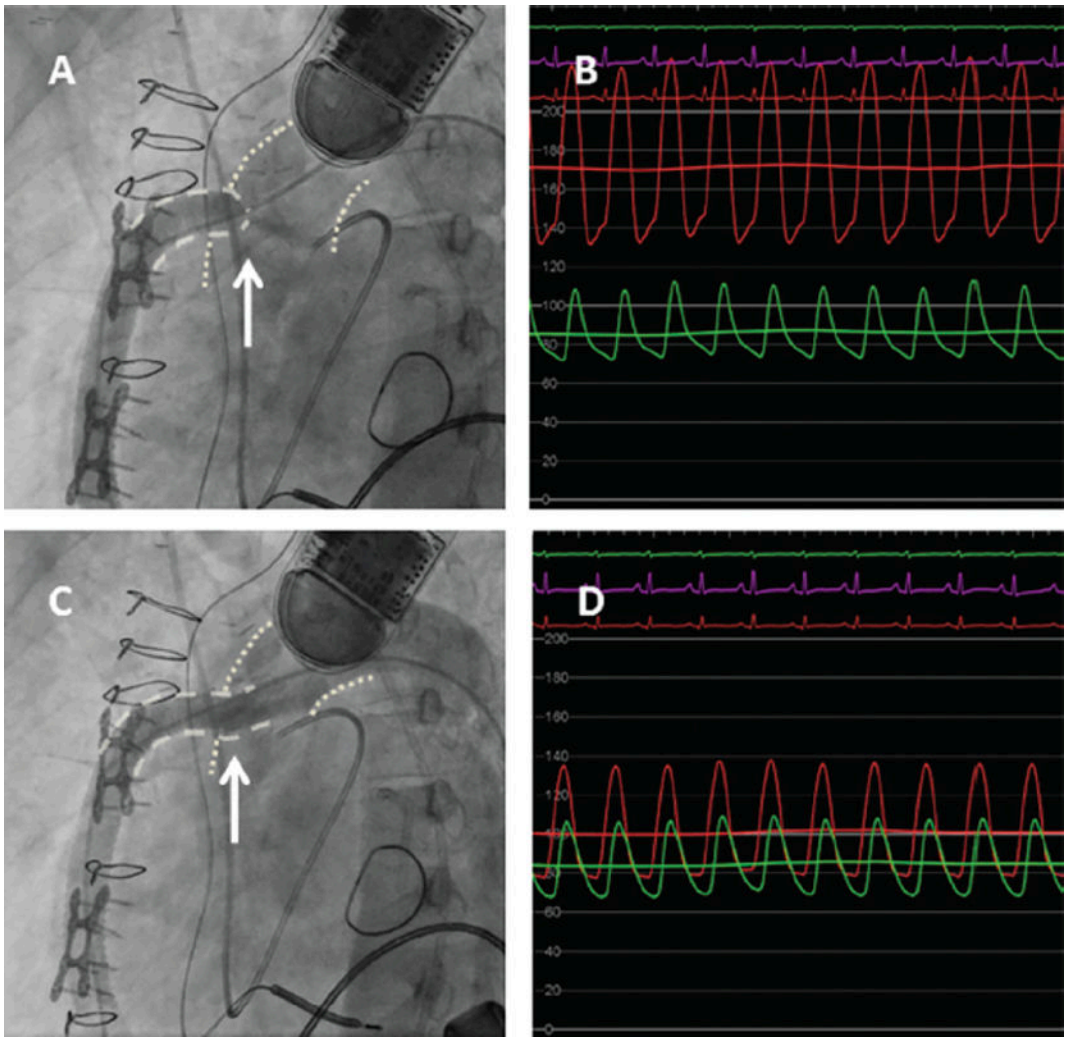

FIGURE 6.

Outflow graft stenosis in a patient with left ventricular assist device, treated with an Atrium 10 × 38 mm covered stent. (A) Significant stenosis at the aortic-outflow graft anastomosis seen on the angiogram with the arrow pointing to the tightest area of stenosis with narrow contrast jet extravasation into the aorta. (B) Hemodynamic assessment demonstrating a significant gradient across the lesion with green aortic waveform and the red waveform measured directly in the outflow graft. (C) Angiography post deployment of the Atrium 10 × 38 mm covered stent. (D) Significant improvement of the hemodynamics, from 120 mm Hg gradient to 30 mm Hg gradient. Reprinted with permission from Retzer et al.[78]

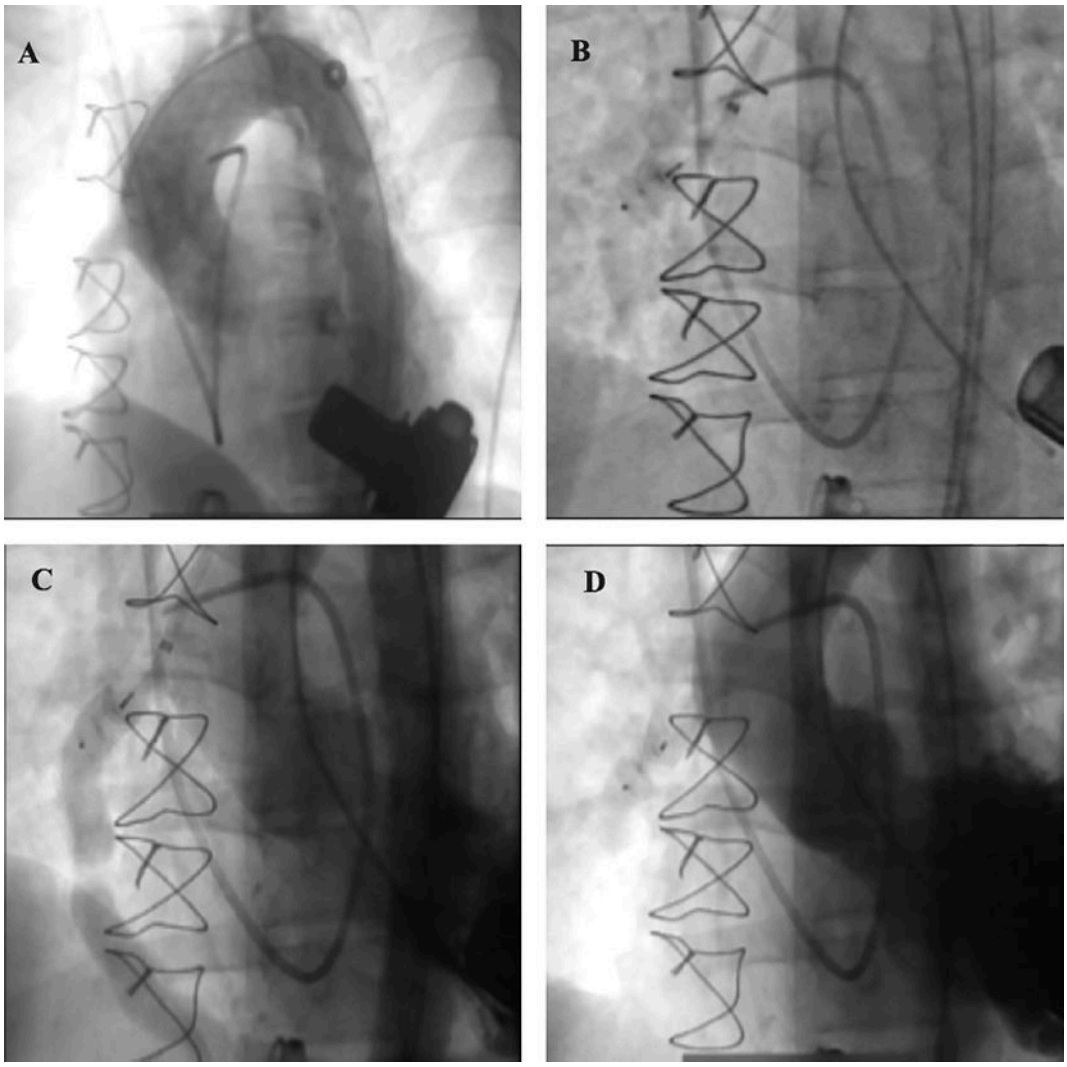

FIGURE 7.

Percutaneous left ventricular assist device (HVAD; HeartWare) decommissioning with a 12 mm Amplatzer Vascular Plug II (St. Jude Medical) deployed in the left ventricular assist device outflow cannula via femoral approach in a patient whose cardiac function recovered. (A) HVAD and outflow graft are visible with anterograde flow from the graft. (B, C) Amplatzer Vascular Plug II positioned in the outflow graft. (D) Fully deployed Vascular Plug II with completely occluded outflow graft and no anterograde or retrograde flow.

Table 1

Percutaneous therapies for *de novo* aortic insufficiency (AI) with continuous flow left ventricular assist devices.

Author	Year	Case Series	Indication/Diagnosis	Intervention	Outcome	Long-Term Outcome
Russo et al[36]	2012	1 patient with HMII	Cardiogenic shock due to severe AI 2 months after surgical repair of the AI	Amplatzer Cribriform septal occluder	- Successfully discharged on day 6 without AI	- At 2 months, patient clinically improved with no further AI
Parikh et al[35]	2013	5 patients: 4 with HMII, 1 with HVAD	Patients with symptomatic AI, deemed to have high surgical risk	Amplatzer Cribriform septal occluder	- Improvement of AI from severe to less than mild in all - Device embolized in the aorta (successfully retrieved) in 1	- Only 2 were alive at 30 days - 1 patient died from sepsis on day 18 and another died from right heart failure on day 12
Retzer et al[39]	2015	10 patients, 6 HMII, 4 HVAD	Patient with severe AI and high surgical risk (STS mortality 22%)	Amplatzer Cribriform septal occluder	- 30% survived to discharge - Those that died had worse RV function	- All 3 patients who were discharged were alive at 6 months
D'Ancona et al[41]	2012	1 patient with LVAD	Severe AI	Edwards SAPIEN 29 mm oversized valve via left anterior thoracotomy under fem-fem bypass	- Successful deployment of the valve without immediate complications	- No long-term follow-up
Santini et al[42]	2012	1 patient with HMII	Refractory symptomatic AI and no surgical options	29 mm CoreValve implantation via femoral approach (required 2 valves due to paravalvular leak after first CoreValve)	- Minimal residual AI - Successfully discharged - On heart transplant list	- No long-term follow-up
Khan et al[40]	2013	1 patient with HMII	Medically refractory symptomatic AI	Melody transcatheter pulmonary valve in aortic position (femoral approach)	- Trace AI after procedure - Successfully discharged on post-procedure day 6	- Survived 10 months

Table 2

Percutaneous therapies for outflow graft stenosis.

Author	Year	Case Series	Indication/Diagnosis	Intervention	Outcome	Long-Term Outcome
Ganapathi et al[79]	2013	1 patient with HMII	- Presented with worsening heart failure symptoms - CT scan showed 13cm pseudoaneurysm of outflow graft	- Gore Excluder (Gore Medical) iliac limb stent-graft (16 mm proximal diameter, 18 mm distal diameter × 9.5 cm length) deployed in the HMII outflow graft excluding the pseudoaneurysm via left axillary approach	- Patient discharged with improvement in heart failure symptoms - Follow-up imaging at 6 mo. showed well-positioned stent-graft without endoleak and with complete resolution of pseudoaneurysm	- Patient doing well at 10 months post procedure
Retzer et al[78]	2015	1 patient with HVAD	- Symptoms of persistent heart failure - Slow decrease in flows with reduced pulsatility - Outflow graft stenosis (90%) with 120 mm Hg gradient across the lesion For on left heart cath and direct outflow graft pressure measurements	- Series of balloon dilations followed by placement of 10 × 38 mm Atrium iCAST stent (Maquet Gettinge Group) - Balloon-expandable stainless-steel stent	- Immediate improvement in LVAD flows and reduction of the gradient to 30 mm Hg with subsequent improvement in renal function and right ventricular function - Discharged to rehab	- Not reported
Abraham et al[82]	2015	1 patient with HMII	- Presented with low-flow alarms and dyspnea - TTE showed aortic valve opening with each beat - CT with contrast of the Personal chest showed narrowing of the outflow graft with extensive thrombus	- Left brachial artery cutdown - Kumpe catheter (AngioDynamics) placed retrograde into outflow graft - A 9 × 59 mm Atrium stent deployed in the cannula while LVAD speed reduced to 6000 rpm	- Flows improved after stent was deployed - Patient discharged 4 days later on coumadin	- 1-month follow-up CT scan showed intact stents and only mild residual thrombus
Hanke et al [83]	2015	1 patient with HVAD (history of 2 exchanges previously)	- Development of dyspnea on exertion - Increased HVAD power levels - CT chest showed severe outflow graft thrombosis	- Atrium 12 × 61 mm aortic stent deployed in the outflow graft and dilated by a balloon to 8 atm. - 12 mm balloon into the right brachiocephalic trunk and embolic protection filter in left carotid artery	- Extubated in the operating room, improvement in hemodynamic parameters - CT scan 2 days later showed no restenosis - Discharged on postoperative day 8	- Developed hemolysis 6 months after the procedure – LVAD turned off - Heart transplant 9 mo. post procedure

Author	Year	Case Series	Indication/Diagnosis	Intervention	Outcome	Long-Term Outcome
Hubbert et al 84	2016	1 patient with HMII	- Acute low flow pump alarm - TTE showed low flow velocity in the outflow graft (0.4 m/s) - CT angiography showed thrombosis in the outflow graft	- 26 Fr destination (90 cm) introducer used to place 7 mm SpiderFX carotid filter Only (Covidien) in distal common carotid arteries bilaterally - Initial dilation with 4 × 40 mm PTA balloon, then deployment of Excluder endoprosthesis (Gore Medical) and post dilation with 14 × 40 mm PTA balloon	- Immediate improvement in LVAD flows - TTE confirmed rise in velocity in the outflow graft - Patient suffered subarachnoid hemorrhage 1 week after procedure. Recovered without neurologic sequelae	- No long-term follow-up
Wiedermann et al[80]	2016	3 patients with HVAD	- Symptoms of heart failure - Decrease in power consumption in HVAD log files - CT angiography chest showed stenosis of the distal outflow graft	- Consecutive percutaneous transcatheter angioplasty followed by implantation of Smart Control 12 × 40 stent	- HVAD log files showed increased flow and power consumption - Symptomatic improvement	- Not reported

TTE = transthoracic echocardiogram; HVAD = HeartWare ventricular assist device; HMII = HeartMate II; CT = computed tomography; PTA = percutaneous transluminal angioplasty; mo. = months.