



Peer-Reviewed Case Report

Successful Percutaneous Management of LVAD Outflow Graft Stenosis: Role of Invasive Hemodynamics in Decision Making

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Abstract

Left ventricular assist device (LVAD) outflow graft stenosis (OGS) is a rare but lethal complication. We present a case of a 79-year-old male with pertinent past medical history of an LVAD implanted as destination therapy, stage III chronic kidney disease, and hypertension. He was admitted for low-flow alarms, and the echocardiogram showed stable right ventricle function and no pericardial effusion. Invasive hemodynamic assessments demonstrated a peak-to-peak gradient of 90 mm Hg in the outflow graft between the mid and distal ends of the graft on pullback. Contrast angiography confirmed OGS. The OGS was successfully treated with a VBX-covered stent (Gore). OGS should be considered when low flow alarms are found in patients with LVADs.



Background

Left ventricular assist device (LVAD) outflow graft stenosis (OGS) is a rare but lethal complication with an incidence of around 5%.¹ The probability of developing OGS increases with time from LVAD implant.¹ A recent systematic review included 26 patients with OGS; 37.5% of patients had a HeartMate II LVAD (Abbott Laboratories) and 58.3% had Heartware VAD (Medtronic).² The median time from LVAD implant to OGS is reported to be around 2-3 years.^{2,3} We present a case of OGS that was successfully treated with a VBX-covered stent to relieve low-flow alarms.

Case Report

A 79-year-old male was admitted secondary to LVAD low-flow alarms. The patient received a HeartMate II implant and underwent a Park stitch for aortic regurgitation 42 months prior to the hospital admission. The international normalized ratios (INRs) were in the therapeutic range two weeks prior to the admission; however, he was advised to cease taking warfarin and bridge with enoxaparin four days before a planned colonoscopy. Two days after the warfarin hold, LVAD low-flow alarms began, and he was admitted to the intensive care unit. He was asymptomatic and denies any tea-colored urine, stroke, or syncope. His doppler mean arterial pressure was 120 mm Hg. On examination, he had no jugular venous distension, no lower extremity or abdominal swelling, and no driveline site tenderness.

The patient's medical history was significant for non-ischemic cardiomyopathy, stage IIIB chronic kidney disease (CKD), a chronic driveline infection treated with cefadroxil, hypertension, hyperlipidemia, and monoclonal gammopathy of undetermined significance.

The differential diagnosis of low flow alarms includes uncontrolled hypertension, hypovolemia, right ventricular failure, cardiac tamponade, arrhythmia, malpositioning of the inflow cannula, inflow pump thrombosis, and outflow graft stenosis (OGS).

Laboratory investigations are detailed in Table 1. All liver function tests were normal, and the LVAD parameters at the time of admission were as follows: speed of 9200 rpm; pulsatility index of 4.1; a flow of 2.4 to 2.5 L/min; and power was 4.1 W. Before admission, the flow was 3.4 to 5.7 L/min, and power was 4.6 – 5.8 W.

Immediate bedside echocardiogram showed an LV ejection fraction of 15%, no aortic regurgitation with the valve mostly closed throughout the cycle, normal right ventricle (RV) size, moderately reduced RV function and no pericardial effusion. Compared to a previous echocardiogram, the LV and RV functions remained the same. A non-contrast computed tomography (CT) of the chest was chosen because of the CKD; it showed normal LVAD position and no external compression of the outflow graft.

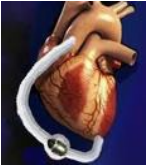


Table 1. Laboratory values upon admission and initial right heart catheterization results

Laboratory Test (Units)	Value on Admission	Baseline/Normal
Hemoglobin (gm/dL)	7.6	Baseline was 7.8-8.1
Total Plasma Hemoglobin (mg/dL)	< 30	Normal is 0-40
International Normalized Ratio	1.6	
Creatinine(mg/dL)	2.4	Baseline was 2-2.2
Lactate Dehydrogenase (U/L)	181	Normal is 100-210
Right Heart Catheterization		Value
Right Atrial (mm Hg)		7
Right Ventricle (mm Hg)		39/10
Pulmonary Artery (mm Hg)		42/40 (23)
Pulmonary Capillary Wedge Pressure (mm Hg)		17
Cardiac Output (L/min)		5.47
Cardiac Index (L/min/m ²)		2.85
PVR (Woods unit)		3.3
Pulmonary Artery Saturation (%)		63

Upon admission, the patient was started on a heparin drip for subtherapeutic INR, and a nicardipine drip was used for hypertension management. Despite controlling the blood pressure, the low-flow alarm persisted. He was given IV fluids and one unit of packed red blood cells for volume resuscitation; dobutamine was initiated for RV support. The next day, the low-flow alarm persisted even after the LVAD controller was exchanged. The device log did not show any electrical faults. The echocardiogram did not show much change from the previous one; hence, RV failure was thought to be a less likely etiology, and dobutamine was stopped. With normal hemolysis labs, no teal color urine, and no spike in power, pump thrombosis was low on the list of differential diagnoses. Given the patient's stage IIIB CKD, we then investigated the outflow graft for stenosis. First, invasive hemodynamics were used as a renal-protective strategy. The patient underwent invasive hemodynamic assessment via right heart catheterization (RHC) (Table 1). The LVAD outflow graft showed a peak-to-peak gradient of 90 mm Hg between the middle and distal portions of the outflow graft on pullback (Figure 1). Thus, lumenography was done with contrast and showed focal 95% eccentric stenosis of the outflow graft (Figure 2A and [Video 1](#)).

After a multidisciplinary team discussion, it was decided to treat the lesion percutaneously. The outflow graft was cannulated using a JB1 catheter (Cook), and an Amplatz wire (Boston Scientific) was advanced through the JB1 catheter. A 11 mm x 79 mm VBX stent (Gore) was advanced over the Amplatz wire and deployed at the stenosis with post-dilation using a 14 mm x 4 cm balloon ([Video 2a](#)). A second 11 mm x 59 mm VBX stent was placed in an overlapping fashion



and post-dilated with a 14 mm angioplasty balloon to extend to the anastomosis site ([Video 2b](#)). Follow-up angiography

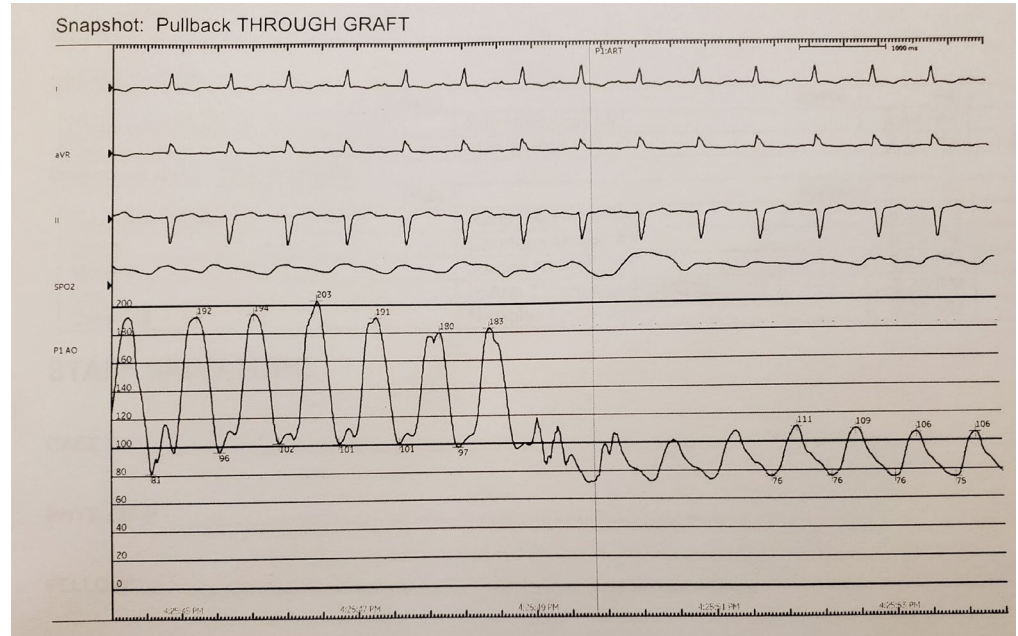


Figure 1. Hemodynamic tracing at time of pullback showing peak-to-peak gradient of 90 mm Hg between mid to distal outflow graft.

showed brisk flow through the outflow graft and no residual stenosis (Figure 2B and [Video 3](#)). The low-flow alarms subsided immediately, and log files showed immediate improvement in the LVAD flow (Figure 3). The patient was discharged the next day on warfarin (INR goal 2-2.5) and aspirin 325 mg daily.

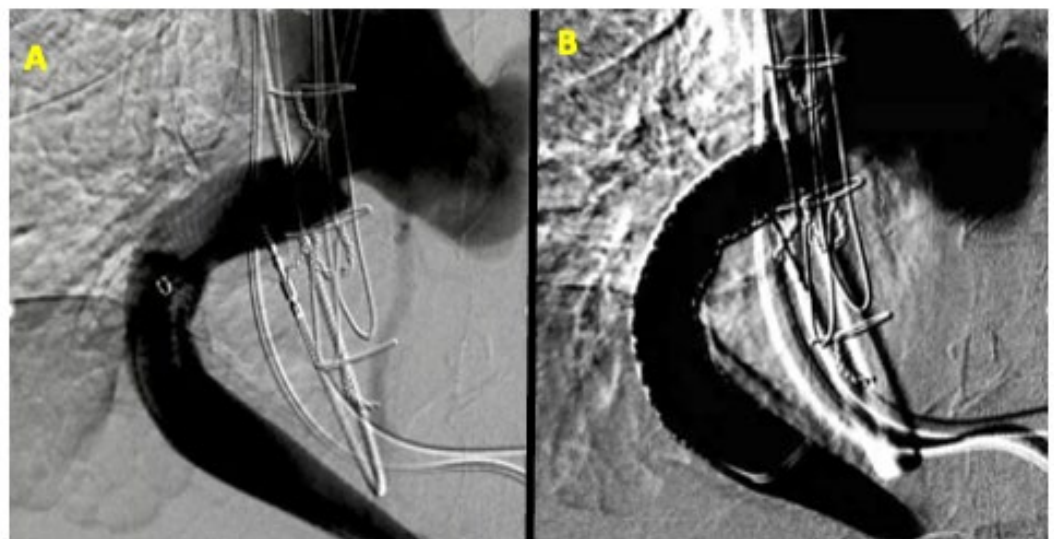


Figure 2. A: Lumenography of the left ventricular assist device (LVAD) outflow graft shows 95% stenosis. B: Lumenography of the LVAD outflow graft after stent intervention is displayed.

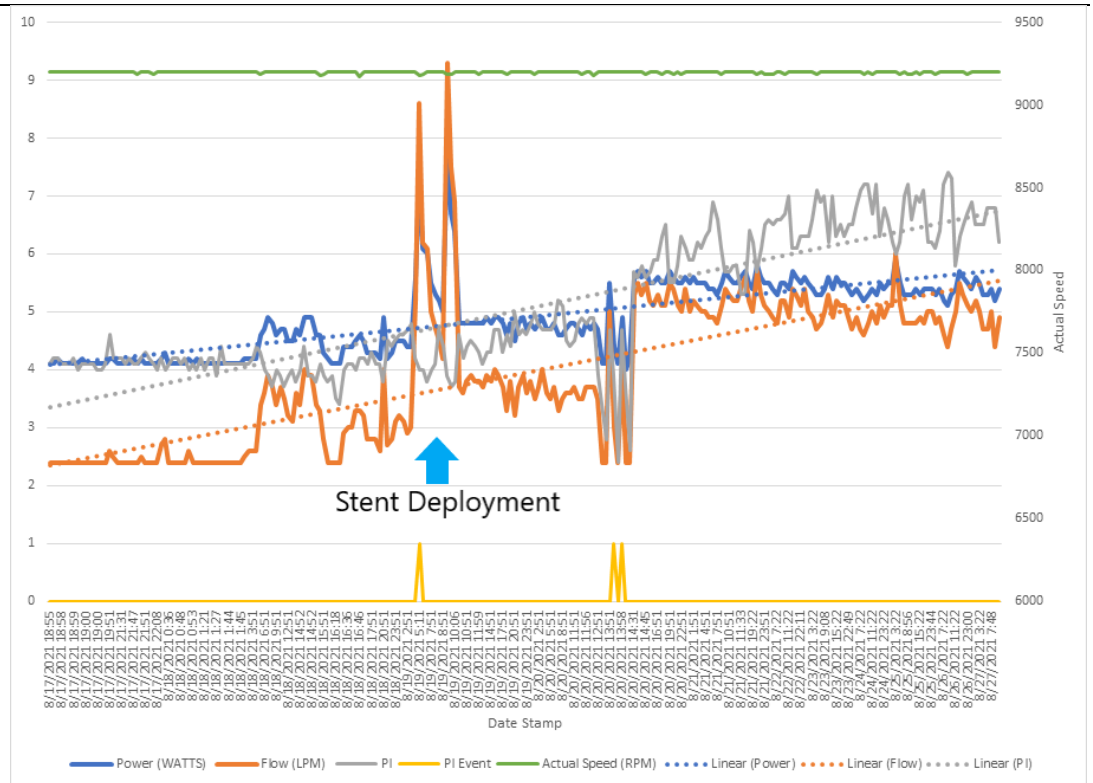


Figure 3: Log files show immediate improvement in flow after stent deployment.

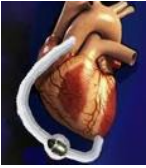
He was recently seen in the clinic nine months post discharge, and no recurrent low-flow alarms or admission for heart failure has occurred since discharge.

Discussion

Our patient had a HeartMate II LVAD implanted 3.5 years before the admission described in this case report. Almost every patient with OGS will present with low-flow alarms, and the majority of patients present with heart failure symptoms; however, others can present with stroke and cardiogenic shock symptoms.³ The flow chart demonstrating our institutional work-up of low flow alarms in patients with LVADs is shown (Figure 4).

The common reasons for OGS include thrombosis, kinking, pseudoaneurysm, external compression, and stenosis of the outflow graft. Obstruction of the external outflow graft by periprosthetic biodebris between the graft and the protective covering is increasingly being reported.^{1,4} Our patient's outflow graft was likely compressed by periprosthetic biodebris. Also, chronic driveline infections can predispose patients to increased risk of thrombosis⁵; hence, the INRs and anti-Xa levels of patients like ours should be closely monitored to avoid thrombotic events.

The diagnosis of OGS is traditionally made by CT angiography, conduit angiography, or intravascular ultrasound.^{2,6} Invasive hemodynamics are rarely



utilized for OGS diagnoses, as was done in our case.⁷ Prior studies have demonstrated high mortality and poor prognosis in patients with LVADs who require hemodialysis.^{8,9} The risk of renal failure and hemodialysis led us to utilize invasive hemodynamics across the aorto-outflow graft as the primary investigative strategy.

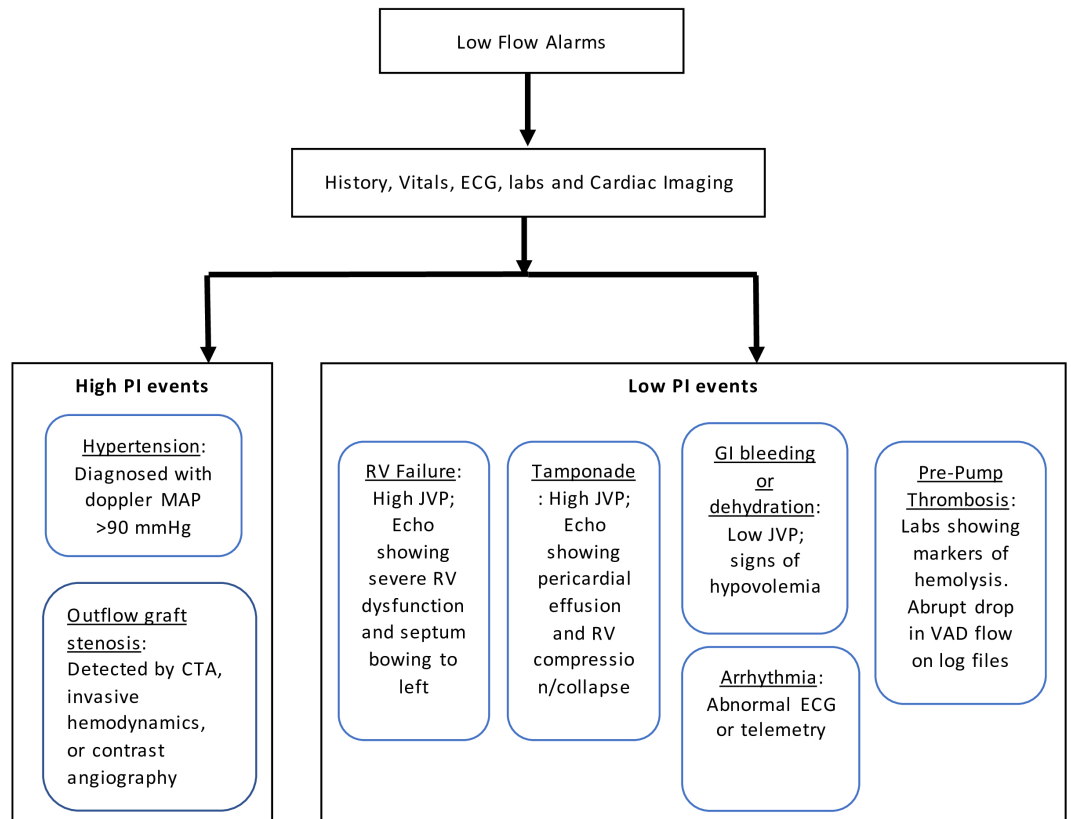


Figure 4. Flow Chart showing etiologies of low flow alarm and work-up

Due to potential life-threatening nature of OGS, prompt diagnosis and treatment should be pursued.¹ The treatment strategies include surgical pump exchange, decompression, and/or systemic thrombolysis.¹⁰ Several case series and reports have shown that OGS can be successfully treated with percutaneous intervention.^{10,11} Moreover, stenting of the OGS is preferred, followed by surgical correction if unsuccessful.¹² Although data is limited, long-term outcomes after stent deployment have been favorable, and recurrent stenosis is rare after stent deployment.^{1,11}

Conclusion

LVAD OGS can be diagnosed using invasive hemodynamics and could be useful in elderly patients who are at risk of contrast-related renal failure. The OGS can be



successfully treated by percutaneous stent intervention with a favorable long-term outcome.

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