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CASE REPORT

ADVANCED

CLINICAL CASE

Intermittent Papillary Muscle Suction

A Rare Cause of Left Ventricular Assist Device Low Flow

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ABSTRACT

Low flow alarms represent a management challenge in patients with left ventricular assist devices because they are often a consequence of complex patient-device interactions. We present a case of intermittent suction of the postero-medial papillary muscle into the left ventricular assist device inflow cannula during diastole, causing low flows. This case highlights the importance of a systematic approach and use of multiple investigation modalities in making an accurate diagnosis. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2021;3:1680–1684)

HISTORY OF PRESENTATION

A 54-year-old man with a left ventricular assist device (LVAD) (HVAD, Medtronic) insertion 4 months prior presented to the hospital with a 3-day history of fatigue. On the day of admission, he noticed dark stools and a sudden decrease in flow in his LVAD to 2-2.5 L/min, from a baseline of 3.5-4 L/min. On examination his mean arterial pressure was 82 mm Hg, heart rate was 84 beats/min, and there were no physical signs of heart failure. Auscultation of the chest revealed a rough, low-pitched added sound to the usual LVAD hum (Figure 1). The LVAD parameters were flow of 2.5 L/min, 2,400 revolutions/min, and power of 2.7 W. Flow trace on the device monitor showed high amplitude trace with low diastolic troughs (Figure 2).

LEARNING OBJECTIVES

- To understand basic LVAD physiology and the determinants of the calculated LVAD flow.
- To be able to work through differential diagnoses and diagnostic modalities for LVAD low flow alarms, a very common but challenging and potentially life-threatening problem.

MEDICAL HISTORY

The patient had a history of coronary artery disease with a previous left anterior descending coronary artery stenting complicated by late stent thrombosis requiring emergency coronary artery bypass grafting. Unfortunately, the patient was left with severe heart failure requiring an LVAD insertion as bridge to cardiac transplantation. Pre-LVAD implantation transthoracic echocardiogram (TTE) revealed a nondilated left ventricle (left ventricular end diastolic dimension of 56 mm) with severe segmental systolic impairment (left ventricular ejection fraction of 30%), a normal right ventricle, and no valvular pathologies. The LVAD implantation was performed via median sternotomy. The site for device implantation was confirmed via

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digital indenting of the apex, with transesophageal echocardiogram (TEE) confirming both apical positioning and no inflow obstruction. There was no evidence of mitral valve or papillary muscle pathology. The LVAD sewing ring was fixed to the apex of the left ventricle using continuous 3/0 polypropylene suture. A hole was cored in the left ventricle, and the HVAD was inserted in the ring and secured. The position of the inflow cannula was confirmed on TEE, with the inflow cannula facing toward the mitral valve and well clear of the subvalvular apparatus. As per our institution protocol, the Lavare cycle was disabled.

Other relevant history included hypertension, asthma, heparin-induced thrombocytopenia, and previous pulmonary embolism.

DIFFERENTIAL DIAGNOSES

LVAD pump flow (Q) is dependent on the pressure differential between left ventricle (LV) and the ascending aorta, called pump head pressure (H), and can be estimated according to the pump function (H-Q) curve (1). This pressure differential and the flow produced by the LVAD is preload dependent and afterload sensitive (2).

Low flow alarms are a consequence of low preload, high afterload or inaccurate hematocrit settings on the pump (pseudo low flow) (3).

Common causes of low flow secondary to low preload include dehydration, arrhythmias, gastrointestinal (GI) bleeding, right-sided heart dysfunction, inflow pump thrombus, and tamponade. On the other hand, high preload states causing low flow include high blood pressure and thrombotic or nonthrombotic conduit outflow obstruction.

ABBREVIATIONS AND ACRONYMS

GI = gastrointestinal

LV = left ventricle

LVAD = left ventricular assist device

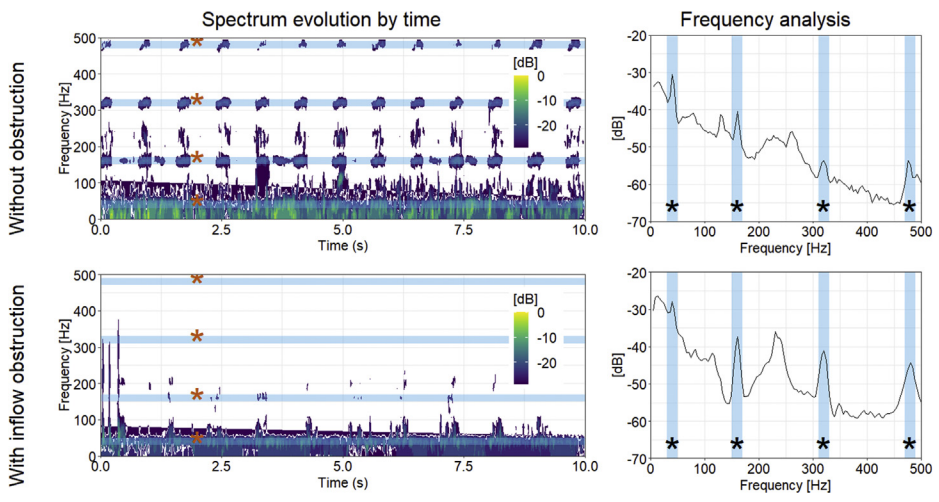
TEE = transesophageal echocardiogram

TTE = transthoracic echocardiogram

INVESTIGATIONS

The electrocardiogram revealed sinus rhythm with anterior Q waves and T-wave inversion in leads V_4-V_6, which was unchanged compared with the previous one. TTE was emergently performed and identified a normal sized LV cavity with a left ventricular end diastolic dimension of 50 mm and a left ventricular ejection fraction of 20%. The right ventricle was normal in size and function. It was not possible to assess the mitral and aortic valve using color doppler due to aliasing throughout the whole ventricular cavity. TEE subsequently demonstrated intermittent suction of the postero-medial papillary muscle in diastole into the LVAD inflow cannula. This appeared to be causing cyclical inflow obstruction and consequent low LVAD flow (Video 1). Analysis of the LVAD logfiles demonstrated a slow and progressive decrease in flow over the 24 hours preceding the admission (**Figure 3**). Blood tests showed a hemoglobin of 92 g/L (for a baseline of 133 g/L), creatinine of 85 μmol/L, troponin of 8 ng/L,

FIGURE 1 Sound Waveform Analysis



The **top 2 graphs** show the sound waveform of the left ventricular assist device (LVAD) prior to the event. The harmonics are clearly identifiable: harmonic 1 at 40 Hz, harmonic 4 at 160 Hz, harmonic 8 at 320 Hz, and harmonic 12 at 480 Hz. The **bottom 2 graphs** show the waveforms at the time of presentation, revealing a more chaotic pattern with added white noise.

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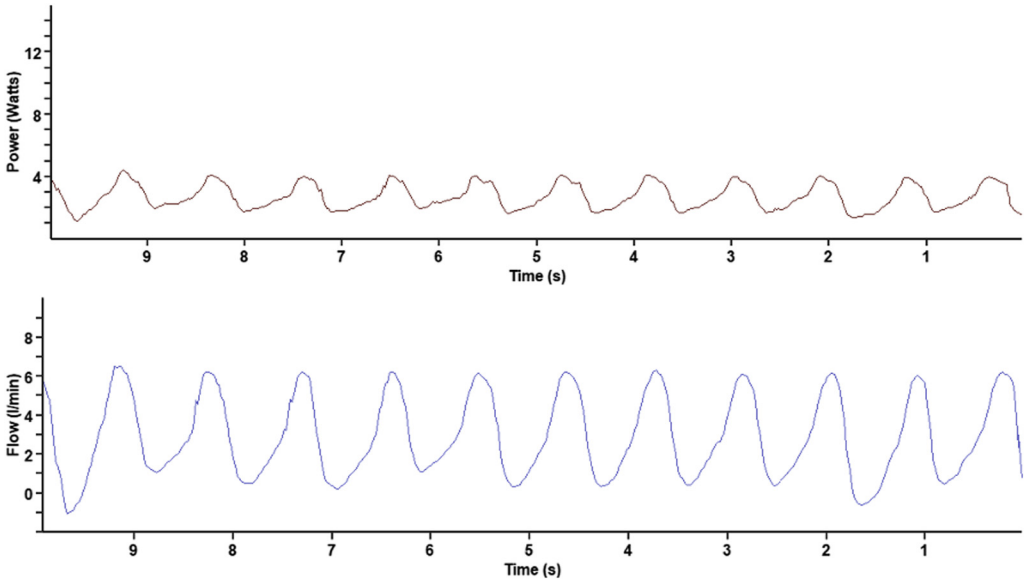
FIGURE 2 Power and Flow Waveforms



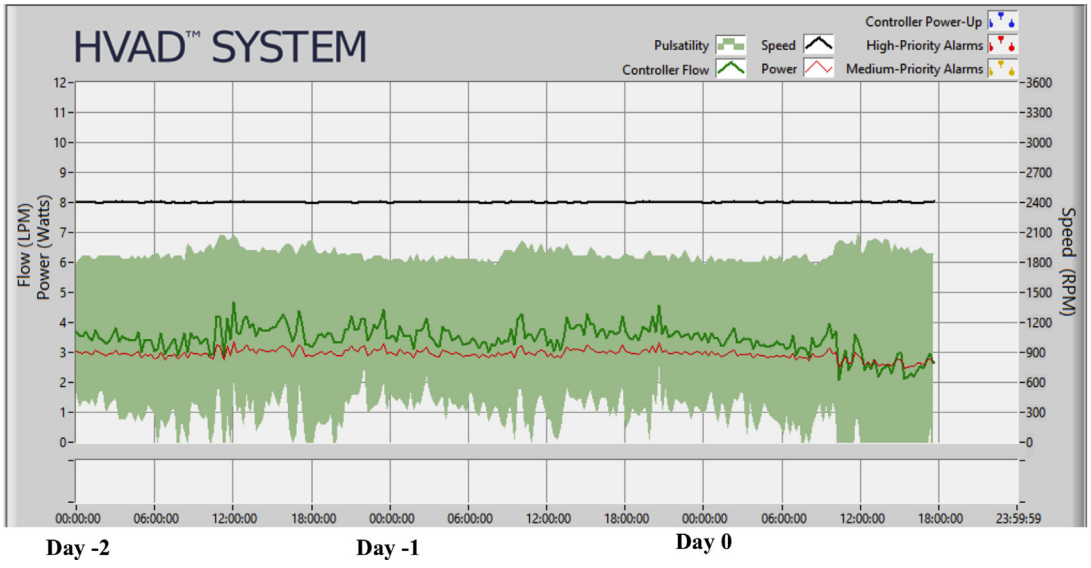
The flow trace shows a high amplitude waveform with low diastolic troughs.

platelets of 244 × 10^9, international normalized ratio of 2.3, lactate dehydrogenase of 194 U/L (range 120-250 U/L), plasma free hemoglobin of 5 mg/dL (range <44 mg/dL), and total bilirubin of 10 μmol/L.

MANAGEMENT

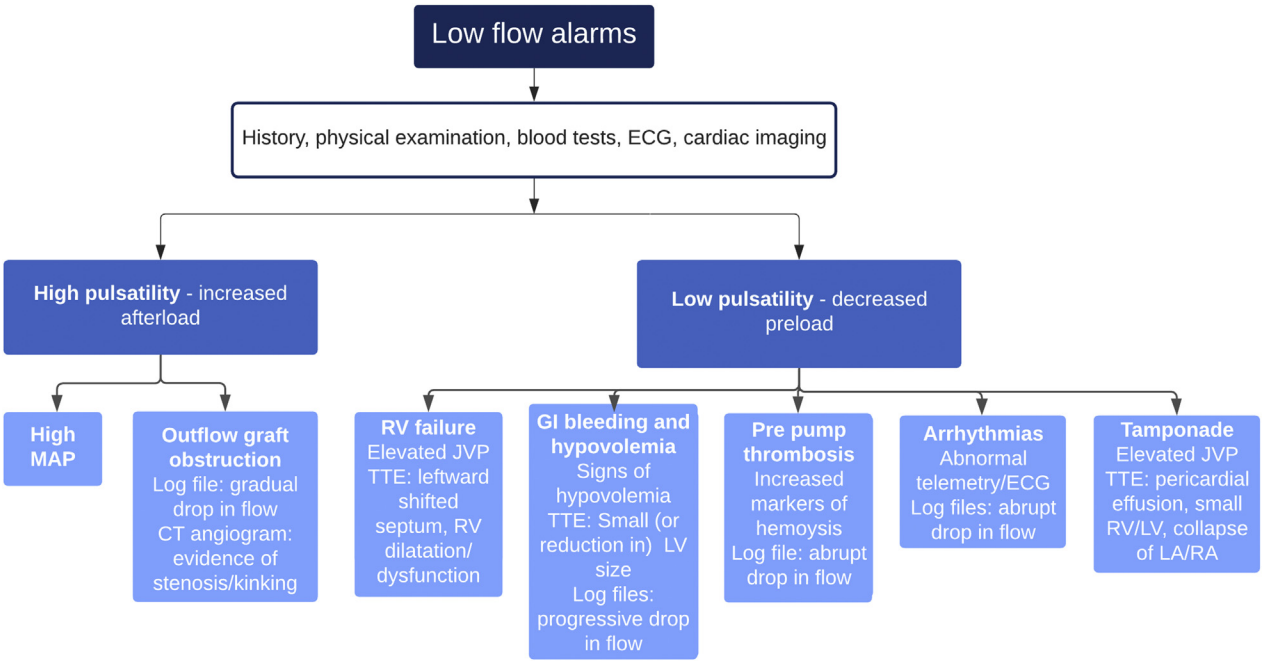
After fluid resuscitation and transfusion of packed red blood cells, the low flow and echocardiographic findings persisted. The patient was transferred to the

FIGURE 3 Log File



Logfile data show a slow and progressive decrease in flow over the 24 hours preceding the admission.

FIGURE 4 Low Flow Algorithm



*If the initial investigations are not diagnostic, patient-pump hematocrit discrepancies should be checked as it can cause pseudo low flow. CT = computerized tomography; ECG = electrocardiogram; JVP = jugular venous pressure; LA = left atrium; LV = left ventricle; MAP = mean arterial pressure; RA = right atrium; RV = right ventricle; TTE = transthoracic echocardiogram

operating room for surgical management. Redo median sternotomy was performed and cardiopulmonary bypass was established. The mitral valve was exposed via a trans-septal incision extending across the roof of the left atrium. A small, ovoid structure approximately 10 mm × 6 mm was found free within the left ventricular cavity and was subsequently identified as fibrin and necrotic debris on histologic examination. The anterior and posterior leaflets were both excised along with the subvalvular apparatus. The inflow portion of the LVAD was inspected using a thoracoscope and did not appear to contain any foreign body or thrombus. A 31-mm Epic mitral bioprosthesis (St Jude Medical) was implanted using pledgeted everting mitral annular sutures. The cardiac chambers were closed and weaning from cardio-pulmonary bypass was easily achieved, with improved LVAD flow. TEE demonstrated satisfactory function of the mitral prosthesis with no paravalvular or valvular leak and good filling of the LV. The patient recovered uneventfully in the intensive care unit and subsequent echocardiographic analysis revealed the LVAD inflow cannula was well positioned with no obstruction and a competent mitral valve replacement. A gastroscopy was performed during the admission and was essentially normal. The patient had a good recovery and had no further decrease in hemoglobin, being discharged a few days later.

DISCUSSION

Troubleshooting low flow alarms on an LVAD requires a thorough assessment of the patient, including laboratory tests, log files, and, often, cardiac imaging (4,5). A proposed algorithm is shown in **Figure 4**. Analysis of the logfiles is an essential tool in differentiating the cause of the low flow (6). Prepump thrombosis is associated with an acute onset (within hours) of low flow, whereas thrombosis of the outflow graft, kinking of the graft, anastomotic stenosis, and GI bleeding develop more slowly, over days to weeks. High pulsatility is associated with high afterload states, whereas low pulsatility is seen in low preload. Last, if the initial clinical assessment and intervention does not reveal the cause or normalizes the flow, one should check for mismatch between the patient/pump hematocrit as the flow calculation is affected by the hematocrit setting (1). Although auscultation of the pump sounds is not traditionally used as a

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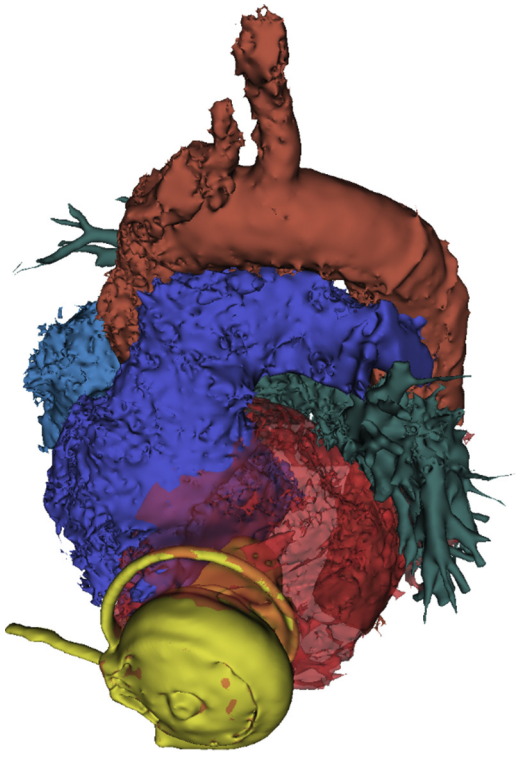
FIGURE 5 3-Dimensional Reconstruction of the Left Ventricular Assist Device and Surrounding Structures Showing Appropriate Inflow Cannula Alignment



diagnostic tool in LVAD malfunction, it raised the possibility of a mechanical issue.

In our case, our hypothesis of the most likely sequence of events was hypovolemia from GI bleeding leading to a reduction in an already small LV cavity size for a patient with severe heart failure. This, in turn, led to suction of the postero-lateral papillary muscle into the LVAD with partial obstruction of the inflow, causing low flow and a bizarre LVAD hum, likely due to turbulent flow through the impeller. A retrospective 3-dimensional reconstruction of the LVAD using a computerized tomography scan of the chest showed good alignment of the inflow cannula with the mitral valve, with an angle of 12.2° (**Figure 5**), ruling out poor cannula alignment as a contributing factor (7). The high amplitude flow trace can be explained by combination of intermittent diastolic obstruction of the cannula by the papillary muscle with zero flow, and papillary muscle contraction moving away from the cannula during systole resulting in high systolic flows.

FOLLOW-UP

The patient did not have any further issues with low flow alarms, melena, or hemoglobin decrease after discharge. Follow-up TTE showed satisfactory function of the mitral valve with no incompetence or a significant gradient.

CONCLUSIONS

This case illustrates the complexities of troubleshooting an LVAD, especially when the issue arises from complex patient-pump interactions. A multimodality approach including clinical assessment, blood tests, cardiac imaging, and analysis of the waveforms/log files is necessary for accurate diagnosis and management.

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KEY WORDS cardiac assist devices, chronic heart failure, mitral valve

APPENDIX For a supplemental video, please see the online version of this paper.