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CASE REPORT

INTERMEDIATE

CLINICAL CASE SERIES

Right Atrial Compression From Biodebris Associated With Long-Term Left Ventricular Assist Device Support

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ABSTRACT

Biodebris surrounding HVAD (Medtronic) intrapericardial centrifugal-flow left ventricular assist device outflow cannulas is common and appears to accumulate over time. We recently encountered 2 patients on long-term HVAD support with right atrial compression from such biodebris, prompting a review of our institution's HVAD cohort to better understand this phenomenon. (**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2022;4:101656) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

CASE 1

A 51-year-old woman with peripartum cardiomyopathy underwent left ventricular assist device (LVAD) placement (HVAD [HeartWare Ventricular Assist System, Medtronic]) as a bridge to transplantation. To prevent adhesions and facilitate future device explantation, the outflow graft was wrapped with a PRECLUDE (GORE) polytetrafluoroethylene pericardial membrane with interrupted sutures. Six years later, the woman presented to the emergency department with progressive dyspnea and generalized weakness.

LEARNING OBJECTIVES

- To recognize biodebris surrounding LVAD outflow grafts as a common occurrence with accumulation over time.
- To appreciate that despite differences in device design, biodebris accumulation remains a potential mechanism of hemodynamic compromise in patients on long-term HVAD support.

On presentation, she was afebrile with a mean arterial pressure of 80 mm Hg, was saturating appropriately on ambient air, and weighed 20 pounds less than her dry weight. Examination revealed a LVAD hum without S1 or S2 and nonpalpable peripheral pulses. Her lungs were clear, and she had no jugular venous distention, abdominal swelling, or lower extremity edema. The driveline exit site was clean and dry, with no erythema or drainage. Laboratory studies were significant for the following: hemoglobin, 9.4 g/dL; white blood cell count, 5.2 10^9/L; sodium, 128 mmol/L; potassium, 4.3 mmol/L; magnesium, 2.2 mg/dL; creatinine, 1.28 mg/dL (consistent with her baseline); lactate dehydrogenase, 340 U/L (reference range: 0-250 U/L); lactate, 0.5 mmol/L; and B-type natriuretic peptide,

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ABBREVIATIONS AND ACRONYMS

CTA = computed tomography angiography

LVAD = left ventricular assist device

RA = right atrium

TTE = transthoracic echocardiogram

225 pg/mL (typically exceeding 700 pg/mL with prior heart failure exacerbations). Her electrocardiogram showed normal sinus rhythm with no ischemic changes, and troponin levels were undetectable.

The patient underwent right heart catheterization, revealing severely elevated filling pressures (right atrium [RA] 25 mm Hg, pulmonary artery mean 52 mm Hg, wedge 38 mm Hg) and low cardiac index (by Fick, 2.17 L/min/m^2; by thermodilution, 1.80 L/min/m^2). With concern for impending cardiogenic shock, she was started on a furosemide infusion, and her LVAD speed was increased. Three days later, she began having low-flow alarms, which were not abated by fluids, necessitating initiation of inotropic and vasopressor support. The patient continued to worsen hemodynamically, ultimately culminating in cardiopulmonary arrest with successful resuscitation.

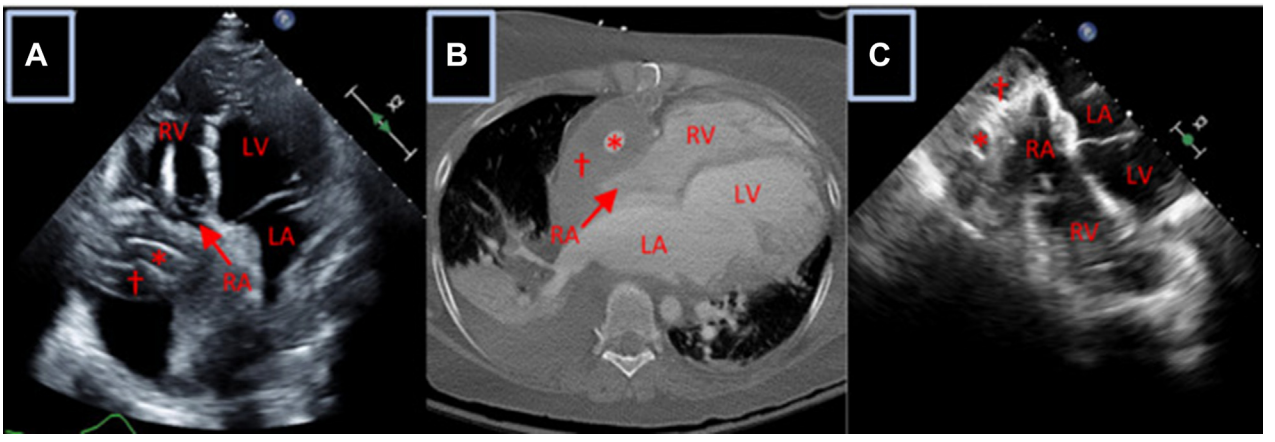
Urgent bedside transthoracic echocardiography (TTE) revealed nearly complete effacement of the RA by a large echogenic structure (**Figure 1,** Video 1). Subsequent computed tomography angiography (CTA) similarly demonstrated a fluid collection surrounding the LVAD outflow cannula, extending from the strain relief to the anastomosis with the native aorta. There was marked effacement of the RA, as well as compression of the inferior vena cava-RA confluence and superior vena cava (**Figure 1**, Video 1). The outflow graft lumen was of normal caliber with no evidence of compression. The patient was taken to the operating room for urgent surgical exploration, where removal of the polytetrafluoroethylene pericardial membrane encasing the outflow graft revealed a collection of fibrinous tissue and coagulated blood. Evacuation of this biodebris allowed immediate RA expansion and improvement in LVAD flows (**Figure 2,** Video 1).

CASE 2

A 64-year-old woman with nonischemic cardiomyopathy presented to the clinic 6 years after undergoing HVAD implantation as destination therapy. Her postimplantation course had been complicated by intracranial hemorrhage (2 months after implantation), pump thrombosis (1 year after implantation), and gastrointestinal bleeding (4 years after implantation). The successive 2 years proceeded without further complication, and she presented to the clinic for routine follow-up without reporting symptoms. Her physical examination was benign, and the laboratory workup was consistent with her baseline values, aside from an elevated lactate dehydrogenase of 593 U/L, increased from 227 U/L 2 months prior.

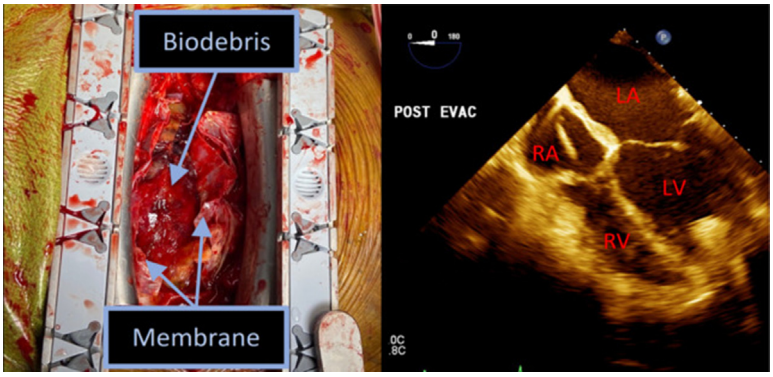
A TTE revealed unexpected compression of the RA from an unspecified extracardiac structure (**Figure 3**). The patient was referred for CTA, which showed a large fluid collection along the entire outflow cannula causing effacement of the RA (**Figure 3**). The outflow cannula and distal anastomosis themselves were both widely patent, as was the left ventricular outflow tract. Cardiothoracic surgery was urgently consulted

FIGURE 1 Echocardiography and Computed Tomography Showing RA Compression From Biodebris



Transthoracic echocardiography (TTE) **(A)**, computed tomography angiography (CTA) **(B)**, and transesophageal echocardiography (TEE) **(C)** demonstrating the HVAD outflow cannula **(*)** with a large amount of surrounding biodebris **(†)** compressing the right atrium (RA). LA = left atrium; LV = left ventricle; RV = right ventricle.

FIGURE 2 **Surgical Evacuation of Biodebris With Resolution of RA Compression**



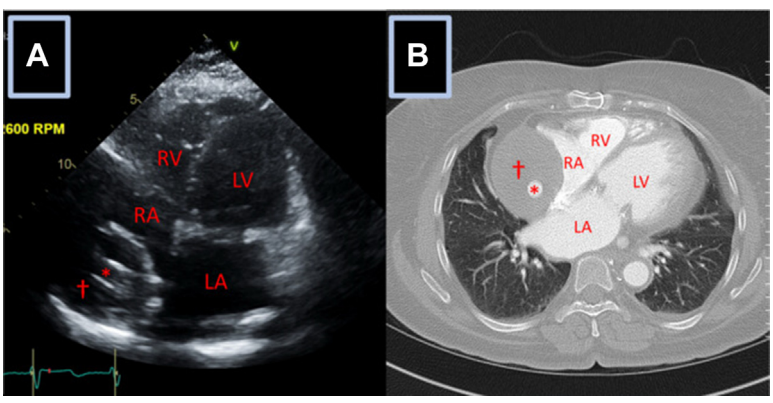
(Left) Surgical evaluation revealed a tense collection of biodebris beneath the polytetrafluoroethylene pericardial membrane. **(Right)** Evacuation of this biodebris allowed immediate relief of the RA compression, as assessed using TEE. Abbreviations as in Figure 1.

for possible surgical intervention. However, the patient was hesitant to commit to surgery in the absence of symptoms or current impacts on her hemodynamic status, so the decision was made for conservative management with close monitoring for any clinical deterioration. At the time of submission, she has been followed up for nearly 4 months since her CTA study, still with no evidence of clinical impact of her biodebris.

DISCUSSION

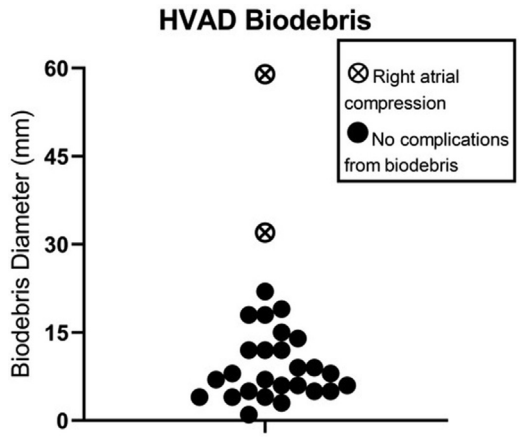
LVADs are increasingly used for patients with end-stage heart failure to improve quality of life and mortality. Advancements in device technology have allowed greater durations of LVAD support, with subsequent identification of potential long-term complications. We previously reported a case series of biodebris accumulation around LVAD outflow grafts.[1] If surrounded by a rigid structure (eg, bend relief in HeartMate models), extrinsic graft compression can occur and is often mistaken on computed tomography for intraluminal thrombus. In our original analysis, patients with HVAD models were not observed to have complications related to biodebris, albeit with a small number of patients studied.[1] However, these cases prompted us to reassess our expanded HVAD population.

FIGURE 3 **Echocardiography and Computed Tomography Showing RA Compression From Biodebris**



TTE **(A)** and CTA **(B)** demonstrating the HVAD outflow cannula **(*)** with a large amount of surrounding biodebris **(†)** compressing the RA. Abbreviations as in Figure 1.

FIGURE 4 Scatter Plot Depicting Varying Degrees of Biodebris Accumulation Among HVAD Recipients



In an updated retrospective review of patients supported by HVAD systems who had undergone CTA, mechanical complications were seen only in the 2 extreme examples described in this case report. Abbreviations as in Figure 1.

We subsequently updated our review of HVAD recipients at our center, identifying 27 patients (20 [74%] male; median age: 54 years [IQR: 45-63 years]; 12 [44%] African American; 20 [74%] with nonischemic cardiomyopathy) who underwent CTA of the chest a median 352 days (IQR: 77-1,015 days) after implantation. Using measurements previously described, the median biodebris diameter was 8 mm (IQR: 5-13 mm) (Figure 4).[1] Aside from the cases described herein with extreme amounts of biodebris accumulation, no other patients experienced clinical complications related to biodebris.

The presented cases and updated experience from our center suggest that although differences in device design typically protect patients with HVADs from outflow graft compression, severe cases of biodebris accumulation can cause complications through other mechanisms (eg, mass effect causing RA compression). A high level of awareness of this phenomenon was required to identify it from TTEs, primarily from the parasternal short axis and apical 4-chamber views. Furthermore, these events occurred in the setting of a technique used to facilitate future device explantation in patients supported with an LVAD over several years.

Other centers have reported HVAD outflow graft compression when closed tubes were placed around the outflow graft.[2,3] Surgical techniques have been proposed to reduce formation of adhesions and facilitate future surgeries while avoiding outflow graft obstruction.[2,4,5] Potapov et al[5] described covering the outflow cannula with a vascular graft, but shortening its length to only encompass the strain relief portion, splicing the side lengthwise to allow for biodebris escape. Jackson et al[4] suggested using a fenestrated or less-restrictive protective covering. Alnabelsi et al[2] reported eliminating the use of outflow graft coverings altogether; instead, the bare outflow tract is routed posteriorly along the diaphragmatic pericardium to avoid the development of any anterior adhesions that might complicate resternotomy.

Because survival on LVAD support continues to increase, the extended durations of device support may promote biodebris accumulation. The distribution and sale of HVAD models have been discontinued, but patients who have indwelling HVADs will still be observed in conditions of long-term device support—thus, a substantial population remains at risk for progressive biodebris accumulation.[6] Additionally, this complication may be possible in the free portion of the outflow cannula in HeartMate devices (Abbott), although not observed in our prior analysis. Finally, whether future LVAD designs may be vulnerable to similar phenomena remains to be seen. Further studies are needed to identify those at the highest risk of biodebris accumulation, establish whether routine surveillance CTAs are of benefit, and devise strategies toward minimizing its impact.

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KEY WORDS cardiomyopathy, chronic heart failure, tamponade

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