Clinical Research

The Impact of Intrapericardial versus Intrapleural HeartMate 3 Pump Placement on Clinical Outcomes

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Background: The integrated design of the HeartMate 3 (Abbott Laboratories, Chicago, IL, USA) affords flexibility to place the pump within the pericardium or thoracic cavity. We sought to determine whether the presence of a left ventricular assist device (LVAD) in either location has a meaningful impact on overall patient outcomes.

Methods: A retrospective cohort study was conducted of all 165 patients who received a HeartMate 3 LVAD via a median sternotomy from November 2014 to August 2019 at our center. Based on operative reports and imaging, patients were divided into intrapleural (n=81) and intrapericardial (n=84) cohorts. The primary outcome of interest was in-hospital mortality, while secondary outcomes included postoperative complications, cumulative readmission incidence, and 3-year survival.

Results: There were no significant between-group differences in baseline demographics, risk factors, or preoperative hemodynamics. The overall in-hospital mortality rate was 6%, with no significant difference between the cohorts (9% vs. 4%, p=0.20). There were no significant differences in the postoperative rates of right ventricular failure, kidney failure requiring hemodialysis, stroke, tracheostomy, or arrhythmias. Over 3 years, despite similar mortality rates, intrapleural patients had significantly more readmissions (n=180 vs. n=117, p<0.01) with the most common reason being infection (n=68/165), predominantly unrelated to the device. Intrapleural patients had significantly more infection-related readmissions, predominantly driven by non-ventricular assist device-related infections (p=0.02), with 41% of these due to respiratory infections compared with 28% of intrapericardial patients.

Conclusion: Compared with intrapericardial placement, insertion of an intrapleural HM3 may be associated with a higher incidence of readmission, especially due to respiratory infection.

Keywords: Left ventricular assist device, Heart failure, Pleural space

Introduction

Continuous-flow left ventricular assist devices (LVADs) have revolutionized survival and the quality of life for patients with end-stage heart failure. With the new United Network for Organ Sharing guidelines giving lower priority to patients with continuous-flow LVADs than in the previous schema, it is likely that LVADs will be used for long-term support irrespective of transplant eligibility [1]. Given this need to increase device longevity, coupled with a growing interest in minimally invasive intrapleural LVAD placement [2,3], whereby pumps must be placed into the pleural space, it is necessary to develop a more thorough understanding of optimal LVAD placement methods.

Historically, the large size of the older generation HeartMate II (HM2) pump required the creation of a preperitoneal pocket. This pocket had to be deep enough to ensure proper alignment of the inflow cannula and allow pump body placement below the diaphragm, perpendicular to the spine. Improper placement of the HM2 could lead to prob-

lems including pump migration, which is implicated in pump thrombosis and dysfunction [4-6].

The integrated design of the more recent fully-levitated centrifugal HeartMate 3 (HM3) may reduce the risk of in situ pump movement, as its lower profile negates the need to create a dedicated pocket and, therefore, facilitates flexibility in placement: either into the pericardium or into the thorax. In light of this flexibility, little is known regarding whether either location has a meaningful impact on overall patient outcomes. Conjectures could be made that the presence of a device within the constrained pericardial space may dynamically alter the orientation of the inflow cannula and lead to similar thrombosis issues as the HM2. Likewise, the greater mobility of the device within the thorax may predispose to cannula obstruction or kinking, as well as potentiate right ventricular (RV) failure in the absence of intact pericardium [7]. The purpose of this study was to retrospectively review whether the location of the HM3—within the pericardium or thorax—had any impact on overall patient outcomes.

Methods

Patients and procedures

A retrospective cohort study was conducted using clinical data of all patients who received an HM3 LVAD from November 2014 to August 2019 at the Columbia University Irving Medical Center (CUIMC). For each patient, using operative records or postoperative computed tomography (CT) scans, if available, it was determined whether LVAD placement was within the pericardium or in the left pleural space (intrapleural). This study was approved by the Institutional Review Board of CUIMC (IRB no., AAAE1866–4/13/2021) with a waiver of informed consent.

Patients' demographic and procedural characteristics were collected. The outcomes of interest included survival, postoperative RV failure according to the INTERMACS (Interagency Registry for Mechanically Assisted Circulatory Support) definition [8], and readmission due to any cause. The reasons for readmission included low flow alarms, inflow or outflow occlusion, stroke, infection, gastrointestinal bleeding, or non-elective admission for any other reason. Follow-up and survival data were obtained by chart review and defined as the date of the most recent clinic visit or death.

The standard approach for all patients was full median sternotomy. The inflow cannula was implanted into the apex and the outflow graft anastomosed to the mid-as-

cending aorta in all cases. The pump was placed either inside the pericardium or in the left pleural cavity at the surgeon's discretion. In general, pumps were placed into the pericardial space unless the patient had prior cardiac surgery where the pleural space had been opened at the last operation or if, on gross examination, the pericardial cavity appeared to be too tight due to either a small cavity or a suboptimal inflow angle/pump position detected by visual inspection or via transesophageal echocardiography (TEE) (i.e., the inflow cannula was not directed straight towards the mitral valve). In these instances, the pleural space was used because it permitted more liberal positioning of the pump in order to achieve a better inflow angle and pump position.

When the pump was placed in the intrapleural space, the pericardium at the diaphragm surface was incised towards the left ventricular apex. The left pleural space was entered and partially opened at the apex site and the pump was placed into the left pleural cavity. For pericardial placement, the pump was simply placed inside the pericardial cavity without opening the pleura.

In the operating room, after LVAD implantation, we began increasing the LVAD speed from 3,000 rpm until we achieved optimal hemodynamics (targeting a mean arterial pressure of 70–80 mm Hg and a central venous pressure of 8–13 mm Hg) without any evidence of septal distortion on TEE. We started dobutamine, milrinone, epinephrine, and inhaled nitric oxide for RV support if needed. If these adjuncts were insufficient in improving contractility and reducing RV afterload, then we considered placement of a concomitant right ventricular assist device (VAD).

Postoperatively, in the intensive care unit, we adjusted flows based on the patient's hemodynamics, targeting the aforementioned mean arterial pressure and central venous pressure goals. Additionally, if the pulmonary capillary wedge pressure exceeded 15 mm Hg, we increased the speed by 100 rpm. Anticoagulation was started when the chest tube output became more serous (typically on postoperative day 2), with the initiation of heparin at 300 U targeting a factor Xa goal of 0.1–0.2. Once adequate anticoagulation was achieved with heparin, oral coumadin was started with a goal international normalized ratio of 2–2.5.

Statistical methods

Clinical and demographic variables are presented using standard summary statistics and frequencies and proportions for categorical variables. Non-normally distributed continuous data, as determined by the Shapiro-Wilk test

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for normality, were reported as median (interquartile range) and compared using the Mann-Whitney test. Generalized estimating equations were used to identify differences in relative numbers of readmissions between the 2 groups, accounting for multiple readmissions for the same patient. The Fine-Gray sub-distribution method was used to plot the cumulative incidence of readmission between intrapleural and intrapericardial patients, with death as a competing risk. This method was chosen as it negates the impact of imbalances in the number of readmissions and their times-to-incidence between the 2 cohorts. The Fine-Gray sub-distribution method was also used in multivariable Cox regression to model readmissions due to non-VAD-related infections, where variables with p-values ≤0.10 in the univariable analyses were included along with an a priori decision to automatically include intrapleural VAD placement in the model. Kaplan-Meier curves were constructed to assess survival at 3 years, with patients censored at the point of documented mortality, transplant, or last-known follow-up. A p-value of <0.05 was considered statistically significant. All statistical analyses were performed with IBM SPSS ver. 25.0 (IBM Corp., Armonk, NY, USA) and SAS ver. 9.4M6 (SAS Institute Inc., Cary, NC, USA).

Results

Baseline outcomes

In total, 165 patients received an HM3 from November 2014 to August 2019. The median age was 61 years (interquartile range [IQR], 53–69 years), 16% (n=27) were women, and the median body mass index was 27.7 kg/m^2 (IQR, 24.7–32.5 kg/m^2). On a retrospective review of imaging and operative reports, 81 patients had HM3 devices implanted within the thorax and 84 had the devices implanted within the pericardium. Representative CT scans are presented in Fig. 1. There were no significant preoperative differences between these 2 cohorts in terms of demographics, left ventricular end-diastolic diameter, INTERMACS level, preoperative temporary mechanical support use, or prior surgery (Table 1). There were no significant differences in preoperative hemodynamics between the 2 groups (Table 2), with comparable preoperative central venous pressure/pulmonary capillary wedge pressure ratios, pulmonary pressures, pulmonary vascular resistance, and pulmonary arterial pulsatility index values [9]. The median follow-up was 409 days (approximately 1.12 years) post-discharge, with an 86% follow-up rate at 6 months.
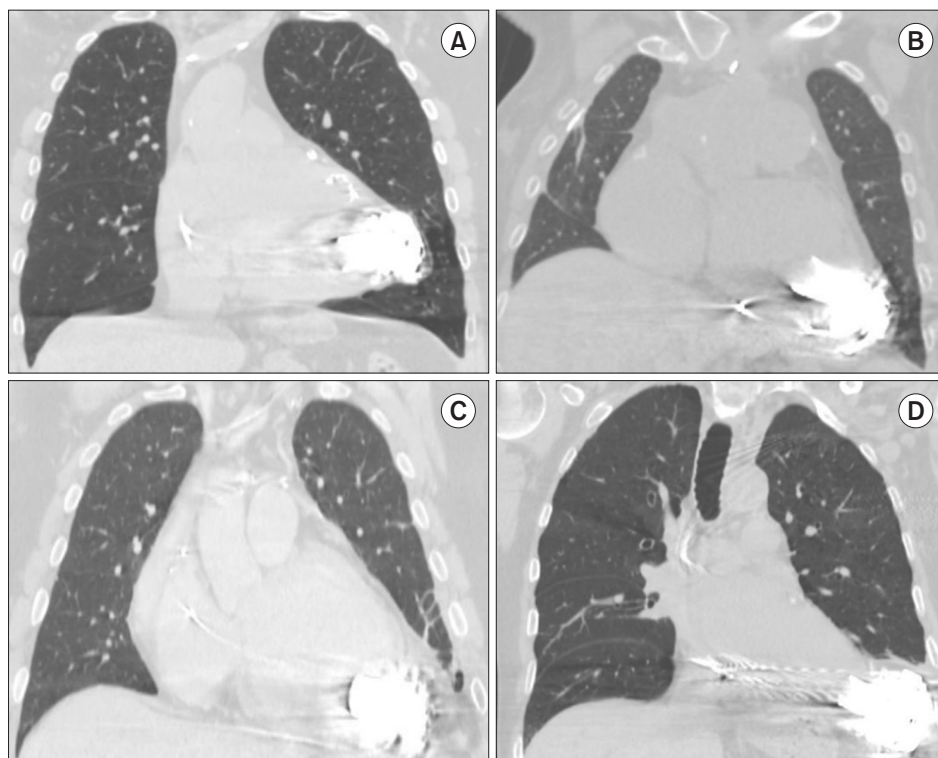
Fig. 1. Representative computed tomography (CT) images of intrapericardial (A, B) and intrapleural (C, D) left ventricular assist device (LVAD) patients. Panel C illustrates left lower lobe compressive atelectasis that may potentiate respiratory infections in intrapleural patients.

Table 1. Baseline characteristics

Characteristic	Total (n=165)	Intrapleural (n=81)	Intrapericardial (n=84)	p-value
Age (yr)	61 (53–69)	61.2 (54.6–69.2)	60.4 (44.6–68.9)	0.16
Female sex	27 (16)	15 (18)	12 (14)	0.30
Body mass index (kg/m²)	27.7 (24.7–32.5)	27.5 (23.9–31.4)	28.4 (24.8–32.8)	0.18
Body surface area (m²)	2.04 (1.87–2.19)	2.02 (1.84–2.18)	2.05 (1.89–2.31)	0.16
Hypertension	99 (62)	45 (56)	54 (68)	0.11
Known coronary disease	92 (56)	48 (59)	44 (52)	0.52
Diabetes mellitus	60 (54)	30 (37)	30 (38)	>0.99
Chronic obstructive pulmonary disease	18 (11)	10 (12)	8 (10)	0.55
Smoking history	90 (55)	46 (57)	44 (52)	0.87
Prior sternotomy	54 (33)	23 (28)	31 (37)	0.25
Preoperative LVEDd (mm)	6.76±1.01	6.78±1.00	6.74±1.03	0.82
INTERMACS				0.081
1	22 (20)	8 (10)	14 (17)	
2	85 (52)	46 (46)	39 (52)	
3	50 (30)	26 (32)	24 (29)	
4	8 (5)	1 (1)	7 (8)	
Preoperative MCS				
Intra-aortic balloon pump	61 (37)	30 (37)	31 (37)	>0.99
Impella	5 (3)	2 (2)	3 (4)	>0.99
Extracorporeal membrane oxygenation	11 (7)	4 (5)	7 (8)	0.54
Centrimag LVAD	4 (2)	2 (2)	2 (2)	>0.99
Centrimag RVAD	4 (2)	2 (2)	2 (2)	>0.99
Concomitant surgery				
Aortic valve repair	30 (18)	12 (15)	18 (21)	0.32
Mitral valve repair	26 (16)	13 (16)	13 (16)	>0.99
Tricuspid valve repair	9 (6)	5 (6)	4 (5)	0.90
Cardiopulmonary bypass time (min)	96 (73–124)	86 (65–117)	100 (82–130)	0.060

Values are presented as median (interquartile range), number (%), or mean±standard deviation, unless otherwise specified.
LVEDd, left ventricular end-diastolic diameter; INTERMACS, Interagency Registry for Mechanically Assisted Circulatory Support; MCS, mechanical circulatory support; LVAD, left ventricular assist device; RVAD, right ventricular assist device.

Table 2. Preoperative hemodynamics

Variable	Total (n=165)	Intrapleural (n=81)	Intrapericardial (n=84)	p-value
CVP (mm Hg)	9 (6–14)	11 (6–14)	9 (6–14)	0.21
CVP/PCWP	0.411 (0.30–0.58)	0.42 (0.30–0.60)	0.40 (0.30–0.58)	0.44
PA mean pressure (mm Hg)	34 (28–41)	35 (28–42)	33 (27–41)	0.45
PCWP pressure (mm Hg)	23 (17–29)	22 (17–30)	23 (17–27)	0.73
Fick PVR (Woods)	3.09 (1.94–4.37)	3.57 (2.14–4.50)	2.90 (1.64–4.16)	0.086
PAPi	3.00 (1.93–4.80)	2.95 (1.76–5.00)	3.00 (2.09–4.78)	0.64
Transpulmonary gradient (mm Hg)	11 (8–14)	11 (8–15)	11 (7–14)	0.64
Fick CI (L/min/m²)	1.80 (1.48–2.14)	1.82 (1.48–2.12)	1.77 (1.48–2.18)	0.94

Values are presented as median (interquartile range).
CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; PA, pulmonary arterial; PVR, pulmonary vascular resistance; PAPi, pulmonary arterial pressure index; CI, cardiac index.

Early outcomes

There were no significant differences between the 2 cohorts in adverse events, including RV failure, the need for continuous venovenous hemodialysis, cerebrovascular accidents, arrhythmias, or prolonged respiratory failure re-quiring tracheostomy (Table 3). There was also no significant between-group difference in the rate of hemi-diaphragm paralysis. One intrapleural patient experienced severe hemolysis, which self-resolved after a transient reduction in pump speed.

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Table 3. Early postoperative outcomes

Variable	Total (n=165)	Intrapleural (n=81)	Intrapericardial (n=84)	p-value
In-hospital mortality	10 (6)	7 (9)	3 (4)	0.20
Right ventricle failure	58 (35)	26 (32)	32 (38)	0.51
RVAD placement	45 (27)	17 (21)	28 (33)	0.09
Postoperative CVVH	10 (6)	7 (9)	3 (4)	0.20
Cerebrovascular accident	6 (4)	3 (4)	3 (4)	>0.99
Atrial fibrillation/flutter	76 (46)	41 (51)	35 (42)	0.24
Sustained ventricular tachycardia	33 (20)	16 (20)	17 (20)	0.84
Tracheostomy	15 (9)	10 (12)	5 (6)	0.12
Hemi-diaphragm paralysis	5 (3)	3 (4)	2 (2)	0.77

Values are presented as number (%).
RVAD, right ventricular assist device; CVVH, continuous venovenous hemodialysis.
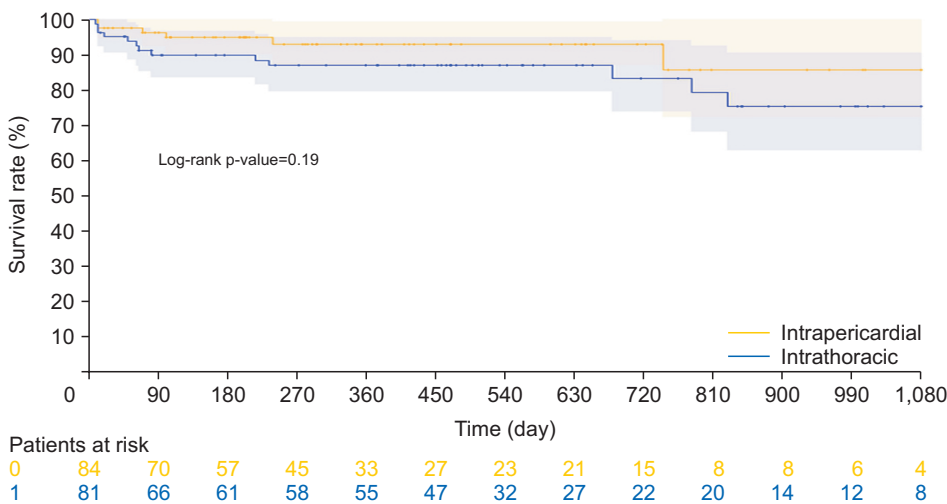
Fig. 2. Kaplan-Meier survival curves illustrating comparable mortality between the cohorts at 3 years.

Late survival

The overall cohort survival was 89.8% at 1 year (95% confidence interval [CI], 85.4%–94.2%) and 79.4% at 3 years (95% CI, 68.8%–90.0%) with a median survival of 434 days post-LVAD implantation (IQR, 201–714 days). When stratified by LVAD location, there was no significant difference in in-hospital mortality between the groups (intrapleural, 9%; intrapericardial, 4%; p=0.130) or survival over the first 3 years. Kaplan-Meier analysis presented in Fig. 2 (p=0.19). Although no significant differences were found, the intrapleural group showed lower survival rates at 6 months (89.9%; 95% CI, 83.4%–96.8% versus 94.9%; 95% CI, 90.2%–99.9%, respectively; p=0.22); 1 year (86.9%; 95% CI, 79.6%–94.8% versus 92.9%; 95% CI, 86.9%–99.3%, respectively; p=0.20), or 3 years (75.4%; 95% CI, 62.7%–90.6% versus 85.7%; 95% CI, 72.3%–99.9%, respectively; p=0.19).

Readmissions

Within the total cohort, there were 297 readmissions during the first 3 years post-device implantation, as presented in Table 4. Among those who survived to discharge (n=155), 102 patients (66%) were readmitted. This averaged 2.9 readmissions per patient (range, 1–13 readmissions), with a median of 135 days from implantation to first readmission (IQR, 65–278 days). There were 53 patients (34%) with no readmissions, 41 patients (40% of the readmitted cohort) with 1 readmission, 21 (21%) with 2, and 10 (10%), 13 (13%), and 4 (4%) with 3, 4, and 5 readmissions, respectively. One intrapleural LVAD patient, who had the most readmissions (n=14), was excluded from the analysis, as these readmissions were for photopheresis to treat cutaneous T-cell lymphoma–related mycosis fungoides.

Ninety-five of the 297 readmissions were within 6 months of device implantation, and 59 were between 6 months and 1 year; thus, a total of 154 readmissions (54%

Table 4. Causes of readmission

Variable	Total (n=165)	Intrapleural (n=81)	Intrapericardial (n=84)	p-value[a]
No. of patients readmitted	103 (63)	57 (70)	46 (55)	0.08
Readmissions (total)	297	180	117	
Readmission events by cause				
Gastrointestinal bleeding	31 (10)	18 (10)	13 (11)	0.12
Low-flow alarm	28 (9)	11 (6)	17 (15)	0.64
Right heart failure	18 (6)	9 (5)	9 (8)	0.24
Infections	68 (23)	46 (26)	22 (19)	0.02
Non-VAD-related[b]	41 (60)	27 (59)	14 (64)	0.02
VAD-related[b]	27 (40)	19 (41)	8 (36)	0.06
Miscellaneous	87 (29)	53 (29)	34 (29)	0.20
Neurologic	16 (5)	9 (5)	7 (6)	0.36
Arrhythmia	38 (13)	28 (16)	10 (9)	0.08
Inflow/outflow occlusion	9 (3)	5 (3)	4 (3)	0.25

Values are presented as number (%).
VAD, ventricular assist device.
[a]p-values calculated using generalized estimating equations to account for repeated admissions by individual patients. [b]Calculated as the percentage of total infections.
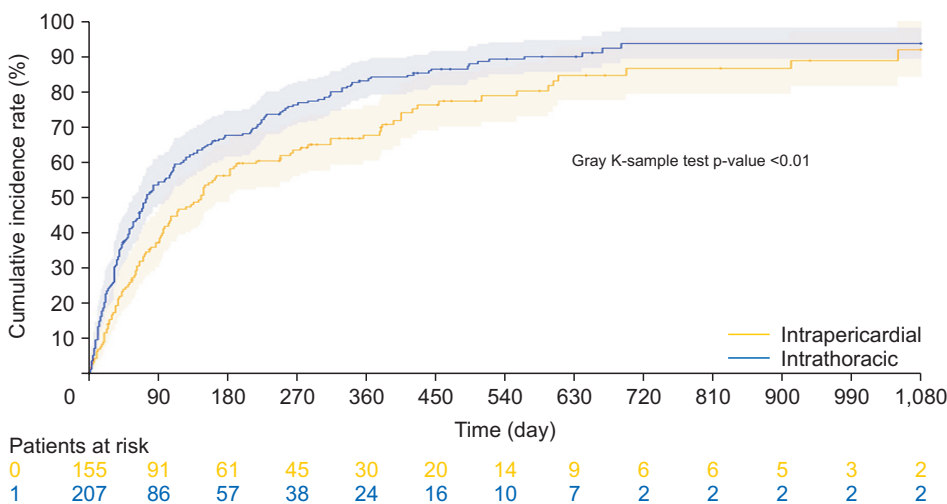
Fig. 3. Fine-Gray sub-distributional Cox regression demonstrating higher rates of readmission among intrapleural left ventricular assist device patients over 3 years (p<0.01).

of all readmissions) occurred within the first year after implantation. Over the course of the first year, the most common causes of readmission were low-flow alarms (16%, n=25), gastrointestinal bleeding (12%, n=19), non-surgical/non-device related infections (11%, n=17), arrhythmias (10%, n=15), and driveline or sternal wound infections (9%, n=14).

When comparing the cohorts, over the course of 3 years, there was a significantly higher rate of readmission among intrapleural patients (n=180 readmissions) compared with intrapericardial patients (n=117, p<0.01), as illustrated in Fig. 3.

The overall most common cause of readmission was infection. Among 68 infection-related readmissions, 41 (60%) were non-device-related (27 intrapleural, 14 pericardial),

such as influenza, pneumonia, and urinary sepsis, and 27 were for VAD-related infections, including 5 sternal wound infection-related readmissions between 2 intrapleural patients and 22 driveline infections. Intrapleural patients had significantly more readmissions for infectious etiologies (p=0.02), which were predominantly driven by non-VAD-related infections (p=0.02). The most common non-VAD-related infections among intrapleural patients were respiratory infections (n=11/27, 41%), which were found in a higher proportion than among intrapericardial patients (n=4/14, 28%). Influenza or viral respiratory infections were responsible for 7 of the 11 respiratory infections in intrapleural patients, but there was no difference in the rates of VAD implantation in the fall/winter compared with the spring/summer between the groups. Moreover, only 4 of

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these patients had evidence of left lower lobe atelectasis/opacification on imaging. On multivariable Cox regression, when controlling for chronic obstructive pulmonary disease, age, and body mass index, only intrapleural VAD placement was significantly associated with non-VAD-related infection development (hazard ratio, 1.42; 95% CI, 1.11–1.82; p<0.01). The intrapleural patients also had a higher prevalence of arrhythmia-related readmissions (n=28) than intrapericardial patients (n=10, p=0.08).

Nine patients were readmitted for inflow and outflow obstruction issues without evidence of pump thrombosis. Of these, 6 underwent late surgical intervention (5 intrapericardial and 1 intrapleural). These cases all presented with intermittent low-flow alarms that were not corrected with volume optimization, and ramp studies failed to demonstrate changes in left ventricular dimensions at different speeds. A more in-depth analysis of these cases has been previously reported [10].

Discussion

Pump thrombosis leading to pump dysfunction was a major limitation of the HM2 device [2]. Prior studies have found pump position and pump movement in situ to be predictors of pump-related complications, namely thrombosis [1,2]. This study sought to assess whether pump placement of the newer HM3 devices in the pericardial or intrapleural space had any effect on clinical outcomes. With the popularization of different approaches to intrapleural LVAD implantation, these results may have important implications for surgical approaches and considerations [2]. We hypothesized that intrapericardial placement of the pump in the relatively confined pericardial space may disrupt the heart geometry and result in adverse events such as inflow obstruction or RV failure. We further posited that in the intrapleural patients, the absence of intact pericardium may potentiate pump movement during support.

Our findings disproved both hypotheses in that were no significant differences in the rates of cannula obstruction, low flow alarms, or right heart failure between the cohorts. Moreover, there were no significant differences in postoperative complications or in-hospital mortality. Although intrapleural patients had similar demographic and risk factor profiles, they unexpectedly had an overall higher rate of readmission over 3 years when treating death as a competing risk. Furthermore, although the differences did not meet the threshold of statistical significance, intrapleural patients did appear to also have worse survival at the

6-month, 1-year, and 3-year time points.

The incidence of late inflow or outflow obstruction in these HM3 patients was 3%, with two-thirds requiring surgical intervention. The prior literature has demonstrated the deleterious effects of pump mobility and the inflow cannula angle, though much of the data involve the HM2 and therefore are unable to provide insights on location as a predictive variable [6,11-13]. We previously published our experience with 59 HM3 patients, demonstrating no difference in the rates of VAD malposition and misalignment regardless of pericardial or thoracic placement [14]. While there was no statistically significant difference between the 2 groups in this larger study, 5 of the 6 cases requiring reintervention were intrapericardial; raising the question of whether the lack of significant difference might have been because the study was underpowered to achieve significance. If this were to be the case, the higher rates of reintervention may negate the potential benefits of reduced readmission seen in the intrapericardial cohort. We have since adopted a strategy of placing the pump in the left thorax if the pericardial space appears to be too tight and may lead to inflow obstruction [10].

The pericardium provides a mutually restrictive chamber to balance biventricular outputs through limiting excessive acute dilation and ensures normal biventricular compliance [15]. However, the question arises of whether creating a wide opening in the pericardium could be implicated in the poorer outcomes seen in our intrapleural patients. While there were no differences in RV failure between the cohorts, the presence of an intrapleural device might have played a role in the differences that were observed.

Currently, there is no literature describing the physiologic effects of a VAD in the thoracic cavity. While our relatively high rates of readmission due to infections, particularly respiratory in nature, could be ascribed to unmeasured factors among the intrapleural patients or simply the result of type II error, it is possible that the presence of a device in the pleural cavity may potentiate these infections. Perhaps the marginal increase in mobility of a bulky pump in the thoracic cavity, unrestricted by the pericardium, can lead to localized left lower lobe atelectasis and, in turn, increase the risk of respiratory tract infections. However, as noted, only 4 intrapleural patients who developed respiratory infections had radiographic evidence of atelectasis on imaging.

Our practice has evolved to preferentially place LVADs within the pericardial space if possible, when implanting through a median sternotomy. Intrapleural placement is

reserved for select patients who had prior surgery in which the pleural space was opened in the last operation (such as a prior coronary artery bypass graft) or in those in whom the pericardium is too tight or the pump position/inflow angle is suboptimal on visual inspection or via TEE.

The observations in this study are particularly salient as new techniques for implanting durable LVAD devices are developed. The preservation of the pericardial cavity through sternotomy-sparing approaches is becoming more popular, as these procedures may reduce the number of adhesions encountered during a subsequent transplant [16,17]. This growing popularity, extending both to the bridge-to-transplant and destination therapy cohorts, will provide an opportunity to assess our findings in future investigations.

Our study is limited in that this is a single-center, retrospective study with a limited sample size. Furthermore, the pump placement location in almost all patients was chosen at the discretion of the surgeon based on surgical expertise, anatomic considerations, and other potential latent factors which may further limit generalizability. Nevertheless, both cohorts were balanced in their baseline demographics and hemodynamics, thereby allowing us to make meaningful comparisons. Finally, we did not have data on pump location and cannula angles to determine the relative rates of VAD malposition and misalignment in the 2 different locations, although flow-related readmissions were not significantly different between the 2 groups.

In conclusion, our cohort study suggests that intrapleural HM3 placement may be associated with a higher incidence of readmission, especially due to respiratory infection, compared with intrapericardial placement, although it remains an effective strategy for selected patients. Investigations into the physiologic rationales underlying these findings may yield insights on how to optimize LVAD placement in the future.

Conflict of interest

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