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Pulmonary artery pulsatility index predicts mechanical circulatory support following heart transplantation

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BACKGROUND: The incidence of mechanical circulatory support (MCS) for early graft dysfunction (EGD) following heart transplantation varies from 2.3% to 28.2%. Low pulmonary pulsatility index (PAPI) is associated with higher mortality in advanced heart failure and cardiogenic shock. We hypothesized that a lower pulmonary pulsatility index following heart transplantation is associated with MCS use for EGD

METHODS: Two-center study of consecutive heart transplantation from May 2018 to December 2022. Hemodynamic parameters and inotropic/vasoconstrictor data were investigated on admission to the intensive care unit (T0) and at 6 hours later (T6).

RESULTS: Of the 173 patients included in this study, 24 had MCS for EGD. PAPI in the group that required MCS was lower at T0 (1.21 (0.84) vs 1.67 (1.23), $p = 0.001$) and T6 (0.77 (0.52) vs 1.44 (0.82), $p = < 0.001$). There was no significant difference in recipient characteristics, donor characteristics (donor age and sex matching), and operative factors (warm/cold ischemic time, total ischemic time, cardiopulmonary bypass time) between the 2 groups. On multiple variable regression, PAPI at T6 was associated with delayed MCS independent of total donor organ ischemic time and short-term MCS bridge to transplantation (odds ratio, OR 0.1 (0.036-0.276), $p = < 0.001$). Receiver operating characteristic (ROC) analysis showed an area under the ROC curve of 0.694 for T0 PAPI and 0.832 for T6 PAPI; a cut-off T6 PAPI of 1.22 had sensitivity and specificity of 81% and 65%, respectively.

CONCLUSIONS: Lower PAPI at T6 (< 1.22) is independently associated with MCS use for severe EGD postheart transplantation.

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Background

Early graft dysfunction (EGD) whether primary or secondary (e.g., related to surgical/technical factors, immunological response, or pulmonary hypertension) within 24 hours of heart transplantation is associated with significant morbidity and mortality. Severe EGD is defined by the use of mechanical circulatory support (MCS) within 24 hours of heart transplantation.[1] The incidence of EGD following heart transplantation has been reported to be between 2.3% and 28.2% and in recipients who develop EGD, the overall mortality has been reported to be 28.4%, 38%, and 45.8% at 30 days, 1 year, and 5 years, respectively.[2] MCS is deployed early in the majority of cases of severe EGD due to failure or immediate hemodynamic instability following separation from cardiopulmonary bypass. However, in some cases, graft dysfunction may evolve over hours after transplantation, possibly compounded by other hemodynamic insults such as vasoplegia, leading to "delayed" MCS. There are little published data on the evolution of EGD and "delayed" MCS following heart transplantation. We hypothesized that delayed MCS, unlike immediate MCS in severe EGD, is related to right heart failure.[3]

Low pulmonary artery pulsatility index (PAPI, ratio of pulmonary artery pulse pressure to right atrial pressure) has been shown to be associated with an increased rate of adverse cardiac events, including left ventricular assist device (LVAD) implantation in patients with advanced heart failure.[4-6] Low PAPI prior to heart transplantation from right heart catheterization (median of 31 days for those with pulmonary hypertension and 41 days for those without pulmonary hypertension preceding heart transplantation) was also associated with graft failure following transplantation.[7]

This is a collaboration between 2 heart transplant centers with the aim of characterizing the hemodynamic changes and the "delayed" use of MCS following heart transplantation. We hypothesized that a low PAPI will be associated with "delayed" MCS use following heart transplantation.

Methods

A retrospective analysis was performed on 216 consecutive heart transplant recipients from 2 heart transplant centers between May 10, 2018, to December 20, 2022. We excluded patients who had "immediate" MCS on separation from cardiopulmonary bypass (n = 43, 19.9%) to focus on patients who underwent "delayed" MCS compared to patients without MCS (Figure 1). This analysis included 173 heart transplant recipients.

The transplant registry included donor and recipient data and early postoperative hemodynamic data from both centers. Hemodynamic data were obtained at T0 (immediately on return to the intensive care unit) and at T6 (6 hours following return to the intensive care unit). The main study endpoint was the use of MCS or death at 30 days. PAPI was derived from the ratio of pulmonary arterial pulse pressure to the right atrial pressure. This study had institutional approval to evaluate the use of MCS postheart transplantation and consent was waived (CARMS-18295).

Statistical analysis

For all variables, descriptive statistics were computed with histograms and Q-Q plots performed. Normally distributed data are presented as means and standard deviations and all nonparametric data as medians and interquartile range. Paired t-test was used for normally distributed related data and the Related Wilcoxon signed rank test was used for nonparametric data. Independent t-test was used for independent normally distributed data and the Mann-Whitney U test was employed for independent nonparametric data. Two-tailed tests of significance were considered to be significant at a $p < 0.05$. PAPI was analyzed with receiver operating characteristic (ROC) curves, and the total area under the ROC curve (AUC) values were considered to assess the performance of the variable. All statistical analyses were performed using SPSS (IBM Corp. SPSS Statistics for Macintosh, Version 29.0, Armonk, NY).

Univariate logistic regression and the Hosmer and Lemeshow goodness of fit tests were performed. If the Hosmer and Lemeshow goodness of fit test was significant, then the variable underwent log transformation prior to being included in the multivariable analysis. The models were built including PAPI, total ischemic time, and short-term MCS bridge to transplantation as these are widely reported to predict post-transplant MCS requirement. We chose total ischemic time as one of the variables as it has been reported each 10-minute increase in donor organ ischemic time is associated with a 5% greater odds of graft dysfunction and MCS following implantation,[2,8] furthermore pretransplant short-term MCS as bridge to transplantation has been associated with a 4-fold increase in post-transplant graft dysfunction.[9] In view of the low number of patients who required MCS (n = 24), we limited the number of variables to 3 to avoid overfitting. Multivariable logistic regression models were performed with both T0 and T6 PAPI as separate models.

Results

Of the 173 patients, 24 patients (13.9%) underwent delayed MCS (following return to the intensive care unit and within

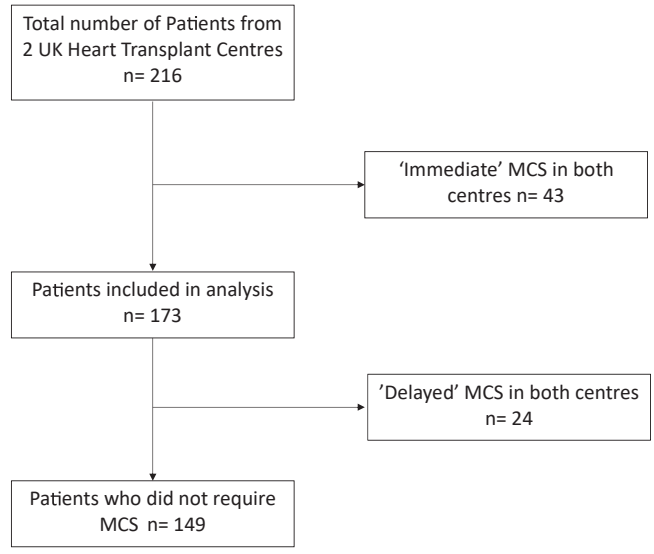

Figure 1 Consort diagram displaying the number of patients excluded from analysis. MCS, mechanical circulatory support.

Table 1 Donor and Recipient Characteristics

N = 173	All	No MCS N = 149	MCS N = 24	*p*
Recipient sex (male %)	69.4	70.5	62.5	0.433
Recipient age (years)	49 (21)	50 (20.5)	47.5 (24.3)	0.735
Recipient height (m)	1.71 ± 0.1	1.71 ± 0.1	1.69 ± 0.1	0.319
Recipient weight (kg)	75.7 ± 14	75.5 ± 14.2	77.3 ± 13.1	0.57
Recipient BMI (kg/m^2)	25.9 ± 4.33	25.7 ± 4.34	27.2 ± 4.14	0.128
Recipient BSA(m^2)	1.87 ± 0.20	1.87 ± 0.20	1.88 ± 0.19	0.982
Recipient LV mass (g)	147 ± 26.6	147 ± 26.7	146 ± 26.5	0.838
Recipient RV mass (g)	23.4 (5.41)	23.9 (5.43)	24.0 (5.48)	0.902
Recipient ventricular total mass (g)	171 ± 29.5	171 ± 29.7	170 ± 29.1	0.865
Donor sex (male %)	60.7	61.7	58.3	0.799
Donor age (years)	36(20)	36 (12.8)	38.5 (15.5)	0.514
Donor height (m)	175 ± 9.1	176 ± 8.92	171 ± 9.71	0.06
Donor weight (Kg)	80 ± 15.2	79.5 ± 14.4	83.8 ± 19.6	0.2
Donor BSA(m^2)	1.95 ± 0.19	1.95 ± 0.19	1.96 ± 0.23	0.858
Donor LV mass (g)	155 (41.1)	154 (41.0)	166 (36.2)	0.546
Donor RV mass (g)	27.0 (7.15)	27.0 (7.43)	26.3 (5.34)	0.315
Donor ventricular total mass (g)	184 (47)	183 (48.0)	193 (45.0)	0.696
Donor status DCD (*n*, %)	26 (15)	25 (16.8)	1 (4.2)	0.109
Cold ischemic time (minutes)	126 ± 39.9	126 ± 38.5	131 ± 47.0	0.549
Warm ischemic time (minutes)	58 (20.5)	58 (21.5)	58.5 (21.5)	0.245
Total ischemic time (minutes)	179 (70.5)	175 (70)	197 (64)	0.058
Cardiopulmonary bypass time (minutes)	189 (88.5)	190 (87.5)	186 (117)	0.814
Recipient etiology				
Dilated cardiomyopathy (%)	53.8	53	54.2	
Ischemic cardiomyopathy (%)	23.1	23.5	20.8	
Hypertrophic cardiomyopathy (%)	12.7	12.8	12.5	
Other	10.4	10.7	12.5	

Abbreviations: BMI, body mass index; BSA, body surface area; DCD, donation after circulatory death; LV, left ventricle; RV, right ventricle.

24 hours of transplantation). The median recipient age was 49 years, 69.4% were male and the most common pathology was idiopathic dilated cardiomyopathy. The median total ischemic time was 179 minutes and median cardiopulmonary bypass time was 189 minutes (Table 1). There were no deaths at or before 30 days in this cohort of patients. There were no significant differences in cold ischemic time, warm ischemic time, total ischemic time, or cumulative cardiopulmonary bypass time between the 2 groups. Of the 24 recipients who had "delayed" MCS following heart transplantation, the modality of support was central extra corporeal membrane oxygenation (ECMO) ($n = 4$), peripheral ECMO ($n = 8$), percutaneous right ventricular assist device (RVAD) ($n = 11$), central RVAD ($n = 1$). The median time to MCS from admission to intensive care unit was 11 (8-16) hours. A subanalysis of the pulmonary hemodynamic data for patients who had ECMO vs patients who had RVAD showed no significant difference in all hemodynamic parameters at both time points except for PAPI at T0. The group of patients who were supported with an RVAD had a lower PAPI compared to patients who were supported with ECMO, 0.96 (0.6) vs 1.41 (0.98), $p = 0.033$. There was no significant difference in the use of short-term MCS bridging in patients who had "delayed" MCS (6/24, 25%) compared to patients without severe EGD (41/149, 27.5%) ($p = 0.066$).

Right atrial pressure (RAP) was significantly higher in the "delayed" MCS group at both T0 and T6, compared to the no "delayed" MCS group (Table 2), and increased significantly from T0 to T6 in the "delayed" MCS group ($p = 0.026$). As a result, PAPI was significantly lower in the "delayed" MCS group at both T0 and T6 (T0: 1.67 vs 1.21 ($p = 0.001$) and T6: 1.44 vs 0.77 ($p < 0.001$)) (Figure 2). Cardiac indices were comparable between the 2 groups at both time points, but cardiac index dropped significantly in the "delayed" MCS group at T6 relative to T0, $p = 0.007$. Right ventricular stroke work index (RVSWi) was similar between the 2 groups at T0 but was significantly lower at T6 (3.53 (2.34) vs 1.80 (1.93), $p < 0.001$) which suggests the cause for EGD and the need for MCS is due to right ventricular failure.

At T0, the "delayed" MCS group was treated with significantly higher levels of Epinephrine, Norepinephrine, Phosphodiesterase inhibitors (PDEi), Vasopressin, resulting in significantly higher vasoactive inotropic score (VIS) to maintain comparable blood pressure (Table 3). Both SVR and SVRi were lower in the "delayed" MCS group at both time points, albeit not statistically significant ($p = 0.100$ and 0.066 for SVR and SVRi, respectively, at T0 and $p = 0.235$ and 0.332 at T6), and with significantly higher vasopressor use. At T6, the MCS group continued to require significantly higher levels of Norepinephrine and had a higher VIS. The changes in the inotropic/vasoconstrictor levels and the VIS score from T0 to T6 in the "delayed" MCS group was not statistically significant.

Table 2 Overall Cohort Hemodynamic Parameters Immediately Following Transplantation (T0) and at 6 hours Following Transplant (T6)

T0 hemodynamic parameters (N = 173)	*T0 all*	*T0 no MCS N = 149*	*T0 MCS N = 24*	*P*
RAP (mm Hg)	10.3 ± 3.71	9.95 ± 3.72	12.1 ± 3.15	0.009*
SPAP (mm Hg)	33.3 ± 6.52	33.5 ± 6.66	31.9 ± 5.42	0.281
DPAP (mm Hg)	16.7 ± 4.56	16.7 ± 4.68	17.0 ± 3.77	0.786
MPAP (mm Hg)	22.2 ± 5	22.3 ± 4.83	21.1 ± 5.97	0.256
MAP (mm Hg)	69.3 ± 9.19	69.9 ± 8.97	65.5 ± 9.86	0.03*
PAPI	1.64 (1.19)	1.67 (1.23)	1.21 (1.21)	0.001*
TPG (mm Hg)	5 (3)	5 (3)	5 (2)	0.120
PVR (WU)	1.05 (0.53)	1.11 (0.59)	0.92 (0.28)	0.122
RVSWi (g/m^2/beat)	3.83 (3.01)	3.85 (2.79)	3.02 (3.01)	0.115
CO (liter/min)	5.1 (2.48)	5.10 (2.50)	5.60 (1.89)	0.704
CI (liter/min/m^2)	2.67 (2.48)	2.67 (1.27)	2.87 (0.94)	0.596
SVR (dynes.sec.cm^{-5})	948 (472)	959 (481)	831 (579)	0.1
SVRi (dynes.sec.cm^{-5}.m^2)	1,770 (869)	1,842 (930)	1,597 (750)	0.066
T6 hemodynamic parameters (N = 173)	*T6 all*	*T6 no MCS N = 149*	*T6 MCS N = 24*	*P*
RAP (mm Hg)	11.3 ± 3.5	10.8 ± 3.11	14.7 ± 4.38	< 0.001*
SPAP (mm Hg)	31.7 ± 6.64	32.0 ± 6.83	29.5 ± 4.71	0.038*
DPAP (mm Hg)	16.5 ± 4.38	16.3 ± 4.42	17.8 ± 3.92	0.12
MPAP (mm Hg)	21.3 ± 5.21	21.5 ± 4.82	19.9 ± 7.18	0.164
MAP (mm Hg)	68.4 ± 8.35	69.3 ± 8.31	63.0 ± 6.47	< 0.001*
PAPI	1.38 (0.88)	1.44 (0.82)	0.77 (0.52)	< 0.001*
TPG (mm Hg)	5 (2)	5 (2)	4 (1.25)	< 0.001*
PVR (WU)	0.99 (0.51)	1.02 (0.5)	0.83 (0.23)	0.018*
RVSWi (g/m^2/beat)	3.36 (2.39)	3.53 (2.34)	1.80 (1.93)	< 0.001*
CO (liter/min)	4.77 (1.90)	4.80 (1.90)	4.60 (1.91)	0.17
CI (liter/min/m^2)	2.55 (0.88)	2.60 (0.91)	2.40 (0.82)	0.06
SVR (dynes.sec.cm^{-5})	947 (347)	949 (346)	858 (341)	0.235
SVRi (dynes.sec.cm^{-5}.m^2)	1,797 (589)	1,802 (622)	1,622 (558)	0.332

Abbreviations: CI, cardiac index; CO, cardiac output; DPAP, diastolic pulmonary arterial pressure; MAP, mean arterial pressure; MPAP, mean pulmonary arterial pressure; PAPI, pulmonary arterial pulsatility index; PVR, pulmonary vascular resistance; RAP, right atrial pressure; RVSWi, right ventricular stroke work index; SPAP, systolic pulmonary arterial pressure, TPG, transpulmonary gradient. SVR: systemic vascular resistance; SVRI: systemic vascular resistance index

On multivariable logistic regression analysis, both PAPI at T0 and T6 were associated with "delayed" MCS following transplantation independent of donor organ total ischemic time and short-term MCS bridge to transplantation ($p = 0.03$ and $p < 0.01$, respectively) (Table 4). ROC was performed for PAPI, central venous pressure (CVP), and

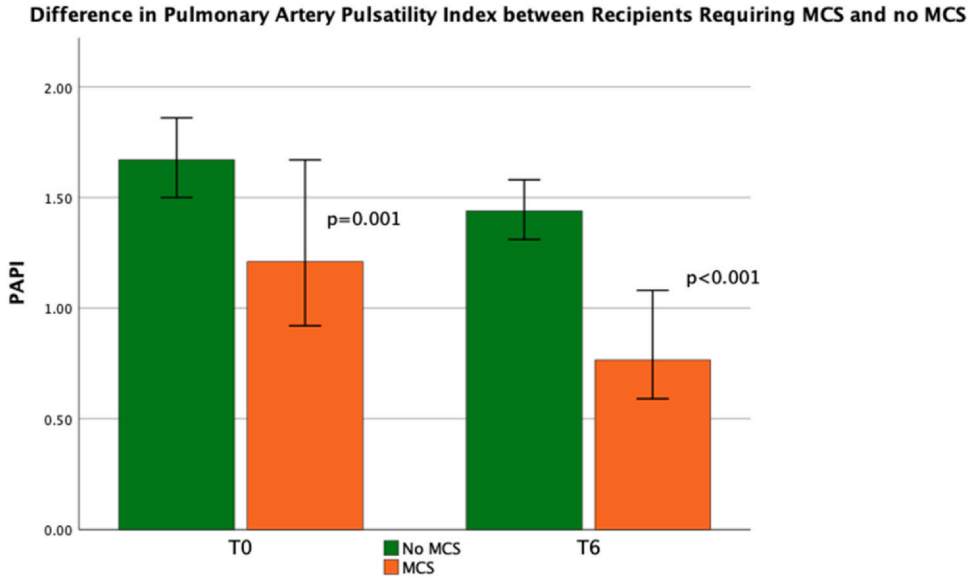

Figure 2 Column chart displaying the difference of PAPI between recipients who did not require MCS and those who did with the significant *p*-value displayed. MCS, mechanical circulatory support; PAPI, pulmonary arterial pulsatility index.

Table 3 Inotropic and Vasoconstrictor Data

T0 inotropes/vasoconstrictors	*T0 all*	*T0 no MCS N = 149*	*T0 MCS N = 24*	*p*
Epinephrine (mg/kg/min)	0.04 (0.05)	0.04 (0.05)	0.035 (0.07)	0.91
	77.50%	78.50%	70.80%	0.403
Norepinephrine (mg/kg/min)	0.07 (0.09)	0.06 (0.07)	0.15 (0.15)	< 0.001
	83.20%	80.50%	100%	0.018
Dopamine (mg/kg/min)	3.8 (5)	4 (5)	0 (5)	0.178
	64.20%	67.80%	41.70%	0.013
Milrinone (mg/kg/min)	0 (0.17)	0 (0.15)	0.225 (0.18)	< 0.001
PDEi (Enoximone or Milrinone)	57.20%	53%	83.30%	0.005
Vasopressin (units/kg/min)	0 (0.00033)	0 (0.00025)	0.00029 (0.00044)	0.034
	34.70%	31.50%	54.20%	0.031
Vasoactive inotropic score	16 (11.4)	15.4 (9.88)	23.8 (16.8)	< 0.001
T6 inotropes/vasoconstrictors	*T6 all*	*T6 no MCS N = 149*	*T6 MCS N = 24*	*p*
Epinephrine (mg/kg/min)	0.03 (0.06)	0.03 (0.06)	0.04 (0.07)	0.229
	71.70%	70.50%	79.20%	0.38
Norepinephrine (mg/kg/min)	0.07 (0.11)	0.06 (0.09)	0.17 (0.10)	< 0.001
	82.10%	79.20%	100%	0.014
Dopamine (mg/kg/min)	4.2 (5)	4.4 (5)	0 (5.07)	0.146
	64.70%	69.10%	37.50%	0.003
Milrinone (mg/kg/min)	0 (0.15)	0 (0.14)	0.14 (0.23)	0.005
PDEi (Enoximone or Milrinone)	57.80%	56.40%	66.70%	0.343
Vasopressin	0 (0.00039)	0 (0.00038)	0.000095(0.00044)	0.229
	35.80%	33.60%	50%	0.119
Vasoactive inotropic score	16.7 (11.7)	16.2 (11.7)	26 (15.8)	< 0.001

Abbreviation: PDEi, phosphodiesterase inhibitors (either Milrinone or Enoximone).

Vasoactive inotrope score was calculated as: 100 × Adrenaline dose (mg/kg/min) + 100 × Noradrenaline dose (mg/kg/min) + 10 × Milrinone dose (mg/kg/min) + 10,000 × Vasopressin dose (unit/kg/min).

Doses given in mg/kg/min and percentages represent the proportion of patients who were on each drug.

RVSWi, and T6 PAPI was found to be the best predictor for MCS with an AUC of 0.868. T0 CVP AUC 0.660, T6 CVP AUC 0.779, T0 RVSWi AUC 0.595, and T6 RVSWi 0.810. The optimal cut-off for T6 PAPI was determined as 1.22 for predicting MCS following heart transplantation with a sensitivity of 81% and 65% specificity (Figure 3).

Discussion

In this study, (i) 19.9% underwent "immediate" MCS and 13.9% of patients underwent "delayed" MCS for EGD; (ii) lower PAPI at both T0 and T6 were associated with "delayed" MCS; (iii) PAPI at T6 was independently associated with "delayed" MCS, with sensitivity and specificity of 81% and 65%, respectively, at a PAPI of < 1.22 (Figure 4).

Severe EGD, especially primary graft dysfunction often manifests at the time of surgery with difficulty in separating from cardiopulmonary bypass or low output syndrome shortly after separation from cardiopulmonary bypass. In the majority of cases, severe EGD is characterized by left ventricular dysfunction.[1,10] Vasoplegia is also a recognized cause of hemodynamic instability and may plausibly result in secondary graft dysfunction related to hypoperfusion. Secondary graft dysfunction in these cases may manifest over hours as progressive deterioration in right heart

Table 4 Multivariable Logistic Regression Analysis

T0	*Regression coefficient*	*p*	*Odds ratio*	*95% CI*
PAPI	−1.295	0.003*	0.274	0.117-0.643
Total ischemic time	0.008	0.125	1.008	0.998-1.018
Short-term MCS bridge	−0.391	0.476	0.676	0.231-1.982
Constant	−1.135	0.342	0.321	
T6	*Regression coefficient*	*p*	*Odds ratio*	*95% CI*
PAPI	−2.303	< 0.001*	0.1	0.036-0.276
Total ischemic time	0.007	0.193	1.007	0.996-1.018
Short-term MCS bridge	0.94	0.882	1.098	0.318-3.789
Constant	−2.956	0.005	0.052	

Abbreviations: MCS, mechanical circulatory support; PAPI, pulmonary arterial pulsatility index.

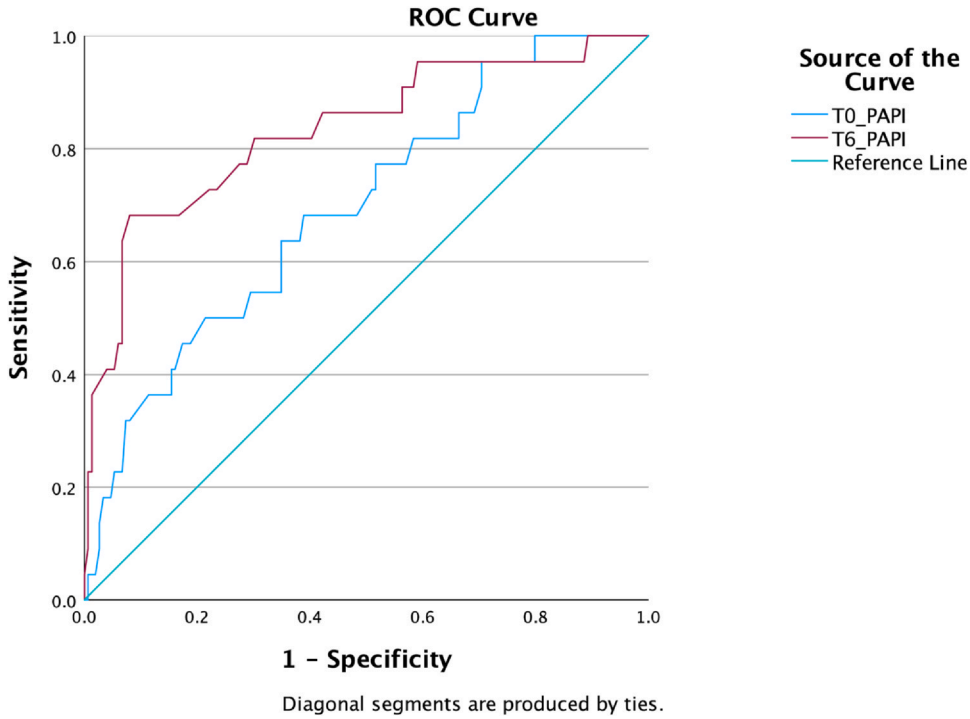

Figure 3 Receiver operating characteristics of T0 PAPI and T6 PAPI. T0 PAPI AUC = 0.694, T6 PAPI AUC = 0.832. AUC, area under the ROC curve; PAPI, pulmonary arterial pulsatility index; ROC, receiver operating characteristic.

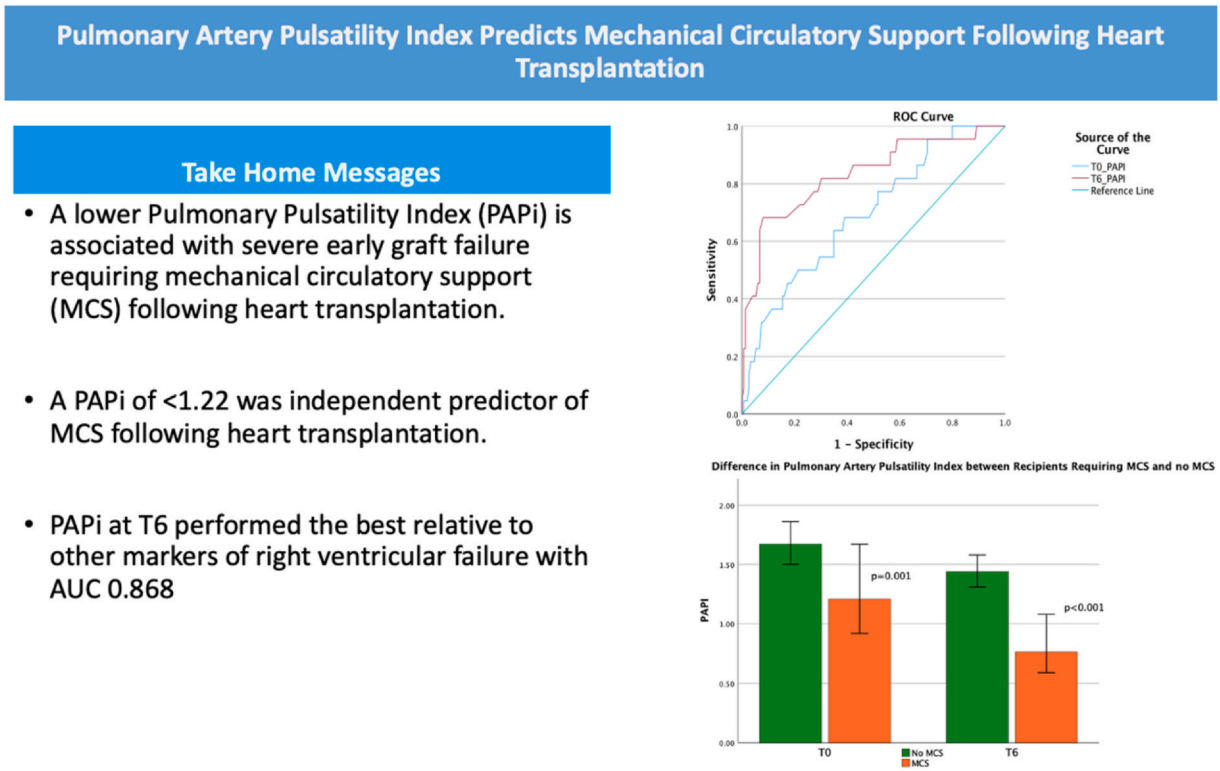

Figure 4 Visual take-home messages. AUC, area under the ROC curve; ROC, receiver operating characteristic.

function. As such, a measure of right heart function may identify patients at risk of severe graft dysfunction and "delayed" MCS.

There has been great interest in employing PAPI to predict right heart failure and indeed one original development of PAPI was in the context of cardiogenic shock following right ventricular infarction.[11] In patients with durable LVAD, a lower PAPI has been well documented to be associated with right heart failure and worse outcomes in the LVAD setting.[12-14] In the advanced heart failure cohort of patients, a lower PAPI has been shown to be associated with significant adverse cardiovascular events, including

death and rehospitalization.[15] There are no published reports of PAPI and severe EGD postheart transplantation.

Data from the Eurotransplant database suggested that a low pretransplant PAPI and high PVR predicted graft failure, although PAPI alone did not have any prognostic implications.[7] Furthermore, pretransplant hemodynamics of the right heart, that is, RAP, pulmonary capillary wedge pressure (PCWP), RAP:PCWP ratio, MPAP and PAPI, have also been shown to predict post-transplant acute kidney injury.[16] Pretransplant RAP has been shown to be associated with primary graft dysfunction and it has been used as part of the RADIAL score which predicts primary graft failure following heart transplantation.[17,18] However, pretransplant hemodynamic data have limited utility in guiding the use of post-transplant MCS, as postheart transplant management is primarily guided by post-transplant hemodynamic data. This is the rationale for studying the evolution of hemodynamic parameters in the hours following heart transplantation and emphasizes the value of serial hemodynamic assessment in guiding MCS use. Mortality was low (2/24, 8%, both deaths were > 120 days following transplant) in this cohort of patients undergoing "delayed" MCS, suggesting that a strategy of close hemodynamic monitoring and hemodynamic-guided MCS use may not compromise early post-transplant survival.

Pulmonary arterial pulsatility index has been used as an indicator of right heart function and it assumes that the failing right heart results in congestion (high RAP) and reduced stroke volume (reduced pulmonary artery pulse pressure). Pulmonary arterial pulse pressure is also intricately linked to pulmonary arterial capacitance (PAC) and pulmonary vascular resistance, as PAC decreases as MPAP increases.[19] In this study, the lower PAPI in the "delayed" MCS group was related to a combination of higher RAP and lower pulmonary artery pulse pressure at both T0 and T6. The ROC analysis showed that PAPI was good predictors of "delayed" MCS use following heart transplantation, especially at T6. The median PAPI was 1.21 at T0 and significantly dropped to 0.77 at T6 ($p = < 0.001$). In our cohort, a PAPI at T6 of 1.22 was the optimal cut-off for predicting impending MCS. It should be noted that PAPI threshold is highly dependent on the patient population under study, as PAC and pulmonary vascular resistance are determinants of PAPI. Thus, the PAPI cut-off cannot be extrapolated from studies of other patient groups.

Study limitations

This study has several limitations. First, this was an observational study with a relatively small number of patients in our cohort, who had "delayed" severe EGD. Second, our data are only relevant in the setting of heart transplantation and may not be applicable to other forms of cardiac surgery. Third, we only had data from 2 time points, at T0 and T6. It would have been desirable to have had data from additional time points for greater granularity. Finally, we limited the multivariable analysis in this study to avoid overfitting due to the relatively small numbers. The variables were selected based on reported evidence in the literature. In particular, we did not have data on preoperative medications, such as the use of amiodarone or neurohormonal antagonists (although none of the patients were on angiotensin receptor-neprilysin inhibitors), which may be relevant.

Conclusions

Low PAPI following heart transplantation especially at T6 is associated with "delayed" MCS. A T6 PAPI cut-off of < 1.22 had a sensitivity and specificity of 81% and 65%, respectively, for "delayed" MCS. PAPI immediately postheart transplant may be used to guide MCS use for severe EGD.

Disclosure statement

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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