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ORIGINAL RESEARCH

Mean Systemic Filling Pressure Was Associated with 28-Day Mortality in Patients with Constrictive Pericarditis After Pericardial Stripping: A Retrospective Cohort Study

Rongping Chen[1], Wei Du[2]

[1]Department of Critical Care Medicine, The First Affiliated Hospital, Sun Yat-Sen University, Guangzhou, People's Republic of China; [2]Department of Critical Care Medicine, Peking Union Medical College Hospital, Peking Union Medical College & Chinese Academy of Medical Sciences, Beijing, People's Republic of China

Correspondence: Wei Du, Peking Union Medical College Hospital, Peking Union Medical College & Chinese Academy of Medical Sciences, Beijing, 13691018276, People's Republic of China, Email duwpumch@126.com

Introduction: This study aimed to explore changes in the venous return system in patients with Constrictive pericarditis (CP) after pericardial stripping and examine their value in predicting mortality.

Methods: An 8-year single-center retrospective cohort study including patients with CP after pericardial stripping surgery. Hemodynamic parameters were analyzed in 90 patients at 11 time points including before and after surgery and every 4 to 9 hours in the first 48 hours in the ICU (pre-operation, post-operation, 0h, t1, t2, t3, 24h, t4, t5, t6, 48h).

Results: Mean systemic filling pressure (Pmsf) were significantly higher in patients who died (non-survival group) than survivors (P = 0.016, respectively). Pmsf at 24h, APACHE II score, and SOFA score were evaluated as predictors of 28-day mortality. APACHE II combined with Pmsf at 24h had the highest prediction (AUC 0.807; 95% confidence interval, 0.671–0.941; P = 0.003).

Discussion: In conclusion, Pmsf at 24h can be used as a valid indicator for prognostic assessment in patients with constrictive pericarditis admitted after pericardial stripping. Pmsf at 24h improves the performance of APACHE II scores in predicting 28-day mortality. Closely monitoring of Pmsf in patients after pericardial debridement may provide guidance for clinical management.

Keywords: venous return, hemodynamic monitoring, intensive care unit, pericardial stripping surgery, mortality

Introduction

Under steady conditions, cardiac output (CO) and venous return (VR) are equal, so any parameter that determines VR also determines CO.[1,2] VR is defined using three parameters: mean systemic filling pressure (Pmsf), right atrial pressure (RAP), and resistance to venous return (RVr).[1] The difference between Pmsf, which is the pressure that promotes blood return to the heart, and RAP, which represents central venous pressure (CVP), is defined as driving pressure of venous return.[3] Pmsf is defined as the pressure throughout the cardiovascular system when the heart is stopped and there is no fluid movement, or the upstream pressure of venous return, and is a functional indicator of effective intravascular volume.[4–6] Pmsf is also the pressure in any portion of the circulation during circulatory arrest.[5] Recently, in patients with acute circulatory failure, it has been further validated that Pmsf changes can indicate volume expansion.[7]

Pmsf can be measured using one of three methods: inspiratory-hold, stop-flow, and mathematical estimation.[8–10] The normal value of Pmsf in humans ranges between 2 and 10 mmHg.[5,11] Changes in Pmsf reflect changes in circulating blood volume and indicate changes in volume status.[12,13] Pmsf varies according to clinical condition, volume status, and vasodilatory tone[14] and increases after fluid infusion and vasopressor administration.[15,16] Two factors of equal importance modify Pmsf: volume of blood in the venous reservoir, which is increased by fluid administration, and venous system capacitance, which is under sympathetic control.[17]

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CP is a potentially curable form of diastolic heart failure that relies on elevated ventricular pressure to maintain cardiac filling and output, which are low because of decreased diastolic filling caused by pericardial stiffness.[18] CP is caused by the release of stress hormones and activation of the renin–angiotensin–aldosterone system, which leads to salt and water retention to increase and balance ventricular diastolic pressure. CP has clinical and hemodynamic features similar to those of restrictive cardiomyopathy and severe tricuspid regurgitation. The primary clinical manifestation is systemic venous stasis as a result of right heart insufficiency.[19] Pericardial stripping can restore satisfactory diastolic filling.

The venous reflux system may be a good indicator of effective volume status after pericardial stripping in patients with CP. Alterations in venous reflux in patients with constrictive pericarditis are currently unknown. We aimed to use a mathematical estimation of Pmsf to assess these alterations. We also examine the relationship of Pmsf with perfusion, organ function, and survival after pericardial stripping. In addition, APACHE II and SOFA are good predictors of disease severity.[20,21] Therefore, we will explore the joint effect of Pmsf on mortality with scores suggestive of disease severity in critically ill patients, such as the APACHE II and SOFA scores.

Methods

Study Design and Setting

We included patients with CP who were admitted to the intensive care unit (ICU) after pericardial stripping surgery at Peking Union Medical College Hospital, a tertiary hospital. The study was approved by the institutional review board of Peking Union Medical College Hospital (approval number, I-23PJ871). The informed consent was waived by the institutional review board of Peking Union Medical College Hospital. Since this study only uses de-identified or anonymized data, does not involve subjects' identity or private information, and does not pose any risk or harm to subjects, and since the study will not negatively affect the safety and rights of subjects and the risks that subjects may be subjected to are no more than minimal. All procedures were performed in accordance with the ethical standards of the local ethics committee on human experimentation and with the Helsinki Declaration of 1975.

Study populationPatients who were definitively diagnosed with constrictive pericarditis and underwent pericardial stripping surgery were included in the study. All included patients were aged 18 years or older and required continuous hemodynamic monitoring using the Pulse Contour Cardiac Output (PiCCO) system. Pericardial stripping surgery is a cardiac surgical procedure in which a small incision is made in the anterior wall of the pericardium to investigate for pericardial thickening, and after the pericardial thickening is clarified, the pericardium is progressively stripped of the thickened mural and visceral pericardium, and CO measurements are taken during the procedure by means of a PICCO, and CVP measurements are taken after anesthesia and at the time of completion of the surgery. Patients were excluded from the study if they: (i) had concomitant other heart conditions, such as coronary artery disease, heart valve disease, or pulmonary hypertension, or underwent other heart surgeries, such as coronary artery bypass grafting, valve replacement or repair, or resection of cardiac or pericardial tumors; (ii) had coexisting cancer, rheumatologic disease, cirrhosis, or end-stage renal disease requiring dialysis; (iii) had missing data.

Data Collection

The following data were recorded at the same time as the Pmsf-0h after ICU admission for analysis: individual patient data (age, gender, primary disease, height, weight), vital signs (blood pressure, heart rate), APACHE II score, SOFA score, ventilator parameters (PEEP), mechanical ventilation time, sedative drugs, vasoactive drugs, total volume balance, based on recorded intake and output, included blood products, oral fluids, etc, serum creatinine, total bilirubin, length of ICU stay, 28-day mortality, central venous-to-arterial carbon dioxide difference ($P_{(V\text{-}A)}CO_2$), central venous oxygen saturation ($ScvO_2$), lactate (Lac).

Hemodynamic Indicators and Calculation

CO, CVP, MAP, and Lac were recorded at 11 time points: immediately before and after surgery and every 4 to 9 hours in the first 48 hours in the ICU (pre-operation, post-operation, 0h, t1, t2, t3, 24h, t4, t5, t6, 48h) -. Pmsf was estimated using the following

$$Pmsf_{(analogue)} = a \times RAP + b \times MAP + c \times CO \quad (1)$$

where a + b = 1, a = 0.96, b = 0.04, and

$$c = 0.038 \times \frac{94.17 + 0.193 \times \text{age}}{\left(4.5 \times 0.99^{(\text{age}-15)}\right) \times 0.007184 \times (\text{height}^{0.725}) \times (\text{weight}^{0.425})} \quad (2)$$

RVr was estimated using the following

$$RVr = \frac{Pmsf - CVP}{CO} \quad (3)$$

Statistical Analysis

Statistical analyses were performed using SPSS software version 26.0 (IBM Corp., Armonk, NY, USA) and R software 4.2.2 (R Foundation, Vienna, Austria). Data normality was tested using the Shapiro–Wilk test. Normally distributed data are expressed as means with standard deviation and were compared using one-way analysis of variance or the *t* test. Data with a non-normal distribution were compared using the Kruskal–Wallis or Mann–Whitney *U*-test. Homogeneity of variance testing was also performed; when variances were uneven, non-parametric tests were used. Categorical data are expressed as numbers with percentage. Repeated observations were compared using repeated measures analysis of variance. Differences between multiple time points were determined using the method of least significant difference or Bonferroni's method. Correlation was determined using Pearson's or Spearman's method as appropriate. Receiver operating characteristic (ROC) curves were used to analyze diagnostic performance. $P < 0.05$ was considered significant.

Results

119 patients were initially reviewed. After excluding 29 based on criteria, 90 were included for analysis. Patients were grouped according to survival. Age, Lac, APACHE II and SOFA scores, epinephrine dose, and length of ICU stay were significantly higher, and MAP was significantly lower in patients who died (non-survival group). Patient characteristics are shown in Table 1.

Trends in Hemodynamic Parameters Over Time

Hemodynamic indicators before surgery and in the first 48 hours after are shown in Table 2 and Figure 1. CO was higher after surgery than that before surgery, and CVP was declined quickly after surgery (Figure 1a). Pmsf was highest before

Table 1 Patient Characteristics at the Time of ICU Admission

	Total (90)	Non-Survival Group (9)	Survival Group (81)	*P* value
Sex (male, %)	65(72.2%)	6 (66.6%)	57 (70.3%)	0.589
Age (years)	51.82±14.82	64.33±5.72	50.05±14.89	0.000*
Height (cm)	168.30±7.32	166.78±7.85	168.59±7.23	0.483
Weight (kg)	66.18±13.07	62.05±13.93	67.03±12.76	0.276
HR (bpm)	98.50±18.41	100.88±13.08	98.76±19.22	0.748
MAP (mmHg)	87±12.71	77.56±10.18	88.55±12.78	0.016*
$P_{(V\text{-}A)}CO_2$ (mmHg)	5.38±2.83	6.19±2.38	5.21±2.89	0.393
$ScvO_2$ (%)	77.4±9.10	79.97±8.71	77.15±9.35	0.448
Lac (mmol/L)	3.37±2.08	5.01±2.38	3.10±1.97	0.013*
APACHE II score	13.63±4.89	18.89±7.07	12.80±4.10	0.033*
SOFA score	8.75±3.43	11.56±2.35	8.48±3.430	0.011*
sCr (μmol/L)	98.64±21.38	84.56±16.54	79.37±23.64	0.526
TBil (μmol/L)	36.56±21.69	44.36±37.82	34.52±21.73	0.244

(*Continued*)

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Table 1 (Continued).

	Total (90)	Non-Survival Group (9)	Survival Group (81)	*P* value
Past medical history (n/%)				
Hypertension	8 (9.2%)	2 (25%)	6 (75%)	0.170
Diabetes	13(14.9%)	3 (25%)	9 (75%)	0.084
Cardiovascular disease	14(16.1%)	0 (0%)	12 (100%)	0.195
COPD	7 (8.0%)	1 (16.7%)	5 (83.3%)	0.625
Etiology, n, (%)				
Tuberculous	26(28.9%)	3(33.3%)	23(28.4%)	0.568
Idiopathic	55(61.1%)	4(44.4%)	51(63.0%)	0.612
immune	9(10.0%)	2(22.2%)	7(8.6%)	0.235
Diprivan (mg/h)	46.00±18.89	45.55±17.40	45.78±19.13	0.972
Fentanyl (ug/h)	46.90±44.78	41.66±12.50	47.64±48.50	0.715
PEEP (cmH_2O)	5.26±1.03	5.12±0.35	5.29±1.10	0.674
Time of MV (h)	70.81±89.10	294.33±125.43	56.47±60.72	0.085
NE (ug/kg/min)	0.11±0.28	0.30±0.58	0.09±0.25	0.390
E (ug/kg/min)	0.03±0.04	0.07±0.03	0.03±0.04	0.021*
Length of ICU days (day)	5(3.5,8)	20.5(17,21)	5(3,7)	0.000*

Notes: Values are expressed as means ± standard deviation or numbers (percentage). * indicates significance (P <0.05).
Abbreviations: HR, heart rate; MAP, mean arterial pressure; $P_{(V-A)}$ CO_2, central venous-to-arterial carbon dioxide difference; $ScvO_2$, central venous oxygen saturation; Lac, lactate; APACHE II, Acute Physiology and Chronic Health Evaluation II score; SOFA, Sequential Organ Failure Assessment Score; sCr, serum creatinine; TBil, total bilirubin; COPD, chronic obstructive pulmonary disease; PEEP, positive end-expiratory pressure ventilation; MV, mechanical ventilation; NE, norepinephrine; E, epinephrine.

Table 2 Hemodynamics Factors Over Time in All Patients

	Pmsf (mmHg)	CVP (mmHg)	CO (L/min)	MAP (mmHg)	Pmsf-CVP (mmHg)	RVr (Ω)
Pre	20.94±6.48	17.84 ±5.82	3.26 ±1.14	79.01±10.23	3.43±2.08	1.34±0.52
Post	14.45±6.15	9.43 ±4.45	5.11 ±1.61	75.11±6.94	4.66±2.46	1.23±0.34
0h	15.04±4.93	10.06±2.89	4.82 ±1.59	87.02±12.71	6.02±1.75	1.45±0.37
T1	15.41±2.77	9.38 ±3.01	4.51 ±1.33	80.87±10.04	6.03±1.50	1.42±0.36
T2	14.85±3.53	8.87 ±3.57	4.32 ±1.58	79.45±14.61	5.98±1.28	1.46±0.34
T3	14.29±2.91	8.09 ±3.33	4.21 ±1.29	81.22±14.03	6.10±0.99	1.50±0.30
24h	14.17±4.56	7.97 ±3.49	4.55 ±4.35	77.73±13.11	6.2±3.60	1.49±0.33
T4	14.21±3.03	8.07 ±3.27	4.31 ±1.30	80.84±14.64	6.13±1.25	1.46±0.26
T5	14.03±3.53	8.08 ±3.29	4.23 ±1.19	79.20±14.66	5.95±1.14	1.46±0.29
T6	13.64±4.40	8.54 ±3.43	4.05 ±1.07	78.35±15.27	5.60±1.33	1.48±0.32
48h	13.61±4.01	8.04 ±3.24	4.17 ±1.13	80.26±15.96	5.74±1.43	1.46±0.32

Notes: Values are expressed as mean ± standard deviation. P<0.05 were considered statistically significant. pre pre-operation, post post-operation, t1-t3 every 4–9h from 0h to 24h after admission to ICU, t4-t6 every 4–9h from 24h to 48h after admission to ICU.
Abbreviations: Pmsf Mean Systemic Filling Pressure, CVP central venous pressure, CO cardiac output, MAP mean artery pressure, Pmsf-CVP the difference between Mean Systemic Filling Pressure and central venous pressure, RVr the resistance to venous return.

surgery (20.94 ± 6.48 mmHg), reached its peak at t1 (15.41 ± 2.77 mm Hg), and then declined (Figure 1b). Pmsf−CVP reached its peak 24 hours after surgery (6.2 ± 3.60 mmHg, Figure 1b. RVr reached its peak at t3 (1.50 ± 0.30 mmHg, Figure 1b.

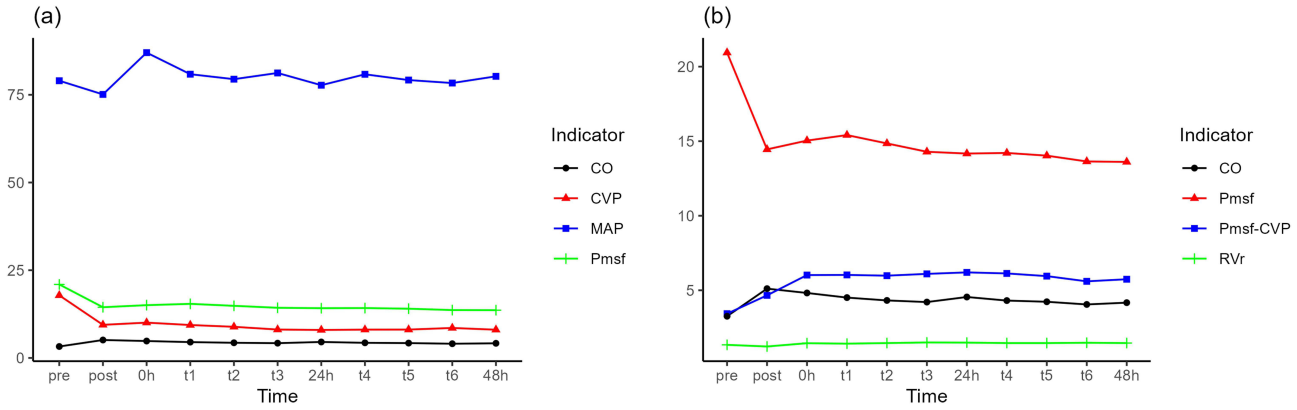

Figure 1 Trends in hemodynamic indicators. (**a**) The trend in Pmsf and the three calculation indicators. (**b**) The trend in the three factors determining venous return and CO.

Comparison of Hemodynamic Parameters Over Time Between the Survival and Non-Survival Groups

In the comparison of hemodynamic indicators between the survival group and the non-survival group, Pmsf was significantly higher in the non-survival group, and this higher level was consistent at 0h, t1, t2, t3, and 24h (Figure 2a). Pmsf−CVP was significantly higher in the non-survival group only at 0h (6.21 ± 0.86 vs 5.10 ± 2.73; P = 0.013, Figure 2b. RVr was significantly higher in the non-survival group at t2, t3, and 24h (Figure 2c). CVP was significantly higher in the non-survival group at 24h (Figure 2d). CO was significantly lower in the non-survival group at t1, t2, and t3 (Figure 2e). MAP was significantly lower in the non-survival group at 0h (Figure 2f). Lac was significantly higher in the non-survival group at 0h and t1 (Figure 2g).

As shown in Table 3, Pmsf (F 6.006, P = 0.016), RVr (F 5.611, P = 0.022), and CVP (F 5.390, P = 0.024) were significantly higher in the non-survival group than in the survival group, with no significant change in the difference

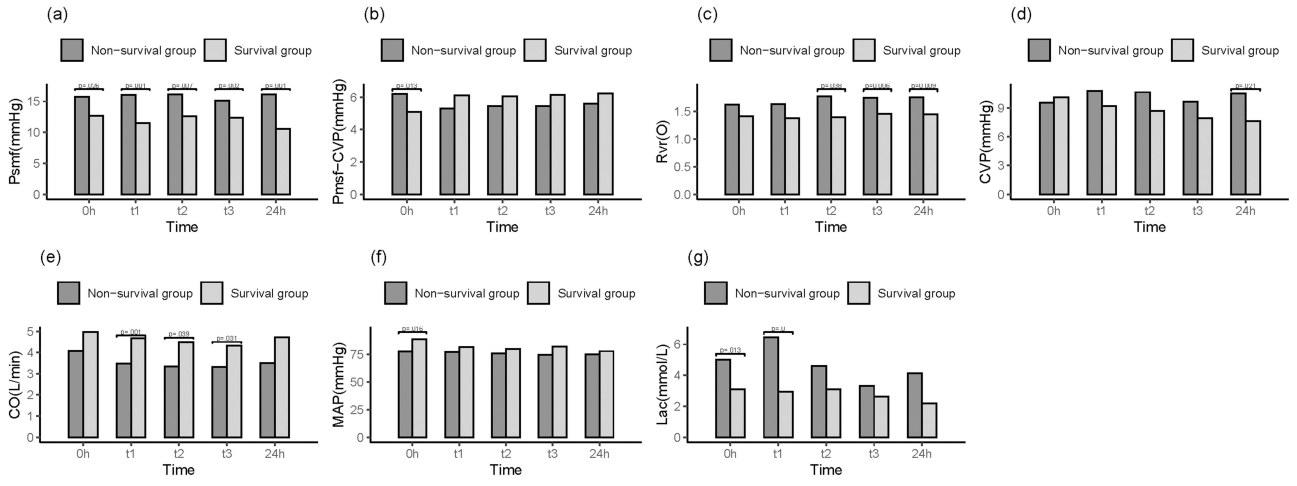

Figure 2 Comparison of hemodynamic parameters between the non-survival and survival groups: (**a**) Pmsf, (**b**) Pmsf−CVP, (**c**) RVr, (**d**) CVP, (**e**) CO (**f**) MAP, (**g**) Lac.

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Table 3 Hemodynamics Factors Over Time in the Non-Survival and Survival Groups

		Pmsf (mmHg)	Pmsf-CVP (mmHg)	RVr (Ω)	CVP (mmHg)	CO (L/min)	MAP (mmHg)	Lac (mmol/L)
Non-survival group	0h	15.77±2.97	6.21±0.86	1.62±0.36	9.56±3.12	4.07±1.39	77.56±10.18	5.01±2.38
	T1	16.08±2.77	5.30±1.34	1.63±0.48	10.78±2.68	3.48±1.27	77.00±11.09	6.46±2.25
	T2	16.11±2.74	5.44±0.92	1.77±0.45	10.67±3.04	3.33±1.36	75.56±8.60	4.60±1.57
	T3	15.12±1.56	5.45±1.15	1.74±0.36	9.67±2.17	3.32±1.22	74.44±11.80	3.31±1.80
	24h	16.15±3.27	5.60±1.08	1.75±0.50	10.56±4.06	3.51±1.39	75±8.68	4.12±3.90
Survival group	0h	12.69±7.13	5.1±2.73	1.41±0.37	10.12±2.92	4.97±1.61	88.55±12.78	3.10±1.97
	T1	11.48±7.11	6.10±1.52	1.37±0.31	9.21±3.08	4.68±1.26	81.45±10.11	2.94±1.88
	T2	12.63±6.22	6.04±1.33	1.39±0.29	8.72±3.63	4.49±1.57	79.83±15.46	3.08±2.37
	T3	12.36±5.62	6.14±0.95	1.45±0.27	7.97±3.44	4.32±1.28	82.03±14.35	2.62±1.6
	24h	10.6±7.26	6.22±3.92	1.44±0.27	7.67±3.28	4.71±4.74	77.68±13.69	2.17±1.31
Total analysis	HF	0.732	0.649	0.854	0.719	0.302	0.497	0.739
Group	*F, P*	6.006,0.016*	0.755,0.388	5.611,0.022*	5.390,0.024*	3.073,0.086	2.122,0.151	2.489,0.124
Time	*F, P*	0.195,0.887	0.409,0.709	2.299,0.078	1.914,0.137	0.561,0.484	2.433,0.096	4.080,0.013*
Time*group	*F, P*	0.410,0.731	0.249,0.825	1.047,0.375	3.623,0.019*	0.084,0.800	1.145,0.320	1.584,0.204

Notes: Values are expressed as means ± standard deviation.* indicates significance (*P* <0.05).HF, Huynh-Feldt; t1-t3 every 4–9 h from 0h to 24h after admission.
Abbreviations: Pmsf, mean systemic filling pressure; CVP, central venous pressure; Pmsf−CVP, difference between Pmsf and CVP; RVr, resistance to venous return; CO cardiac output; MAP, mean arterial pressure; Lac, lactate.

between the two groups over time. Pmsf-CVP, CO, and MAP were not significantly different between the non-survival and survival groups, with no significant change in the difference between the two groups over time. Lac was not significantly different between the non-survival and survival groups, but the difference between the two groups over time was significant.

Correlation Between the Venous System and Clinical Indicators

In patients with serum lactate >2.0 mmol/L at the time of ICU admission, there was a negative correlation between Pmsf-at 24h and lactate clearance rate at 6h (r^2=−0.596; P = 0.000; Figure 3a. The difference in Pmsf−CVP between 48h and 0h was positively correlated with total fluid balance 48h after surgery (r^2=0.751; P = 0.000; Figure 3b. In patients with renal insufficiency before surgery, the difference in Pmsf−CVP between 48h and 0h positively correlated with serum creatinine at the time of transfer out of the ICU (r^2=0.664; P = 0.001; Figure 3c.

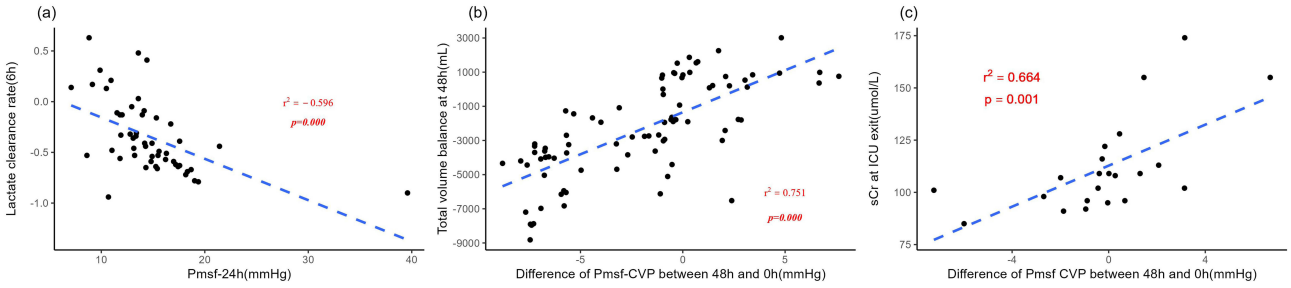

Figure 3 The correlation between (**a**) Pmsf-24h and lactate clearance rate at 6h; (**b**) Pmsf-CVP difference between 48h and 0h and total fluid balance 48h after surgery; (**c**) Pmsf-CVP difference between 48h and 0h and serum creatinine at the time of transfer out of the intensive care unit.

Table 4 The Risk Factors for 28-Day Mortality

	One-way Logistic Regression Analysis			Multifactor Logistic Regression Analysis		
	HR	***P***	**95% CI**	**HR**	***P***	**95% CI**
Pmsf-24h	1.125	0.049*	1.001–1.264	1.155	0.035*	1.010–1.321
SOFA	1.386	0.018*	1.057–1.917	1.392	0.019*	1.057–1.833
APACHE II	1.238	0.004*	1.071–1.430	1.205	0.016*	1.036–1.401

Notes: *indicates significance (P <0.05).
Abbreviations: HR, Hazard Ratio; CI, confidence interval; Pmsf, mean systemic filling pressure; APACHE II, Acute Physiology and Chronic Health Evaluation II score; SOFA Sequential Organ Failure Assessment score.

Table 5 Area Under the Receiver Operating Characteristic Curve for Various Indicators

	AUC±SE	***P***	**95% CI**	**Cut-off**	**Sensitivity**	**Specificity**
APACHE II	0.769±0.087	0.009*	0.598–0.939	19	55.6%	94.7%
SOFA	0.759±0.071	0.011*	0.620–0.899	9.5	88.9%	47.6%
Pmsf-24h	0.699±0.078	0.011*	0.607–0.913	14.9	66.7%	73.7%
APACHE II+Pmsf-24h	0.806±0.069	0.003*	0.671–0.941	0.06	100%	49.3%
SOFA+Pmsf-24h	0.782±0.064	0.001*	0.702–0.952	0.17	77.8%	84.0%

Notes: Values are expressed as means ± standard deviation. * indicates significance (P <0.05). AUC, area under the receiver operating characteristic curve;
Abbreviations: APACHE II, Acute Physiology and Chronic Health Evaluation II score; SOFA Sequential Organ Failure Assessment score; Pmsf, mean systemic filling pressure.

Prediction of 28-Day Mortality

Pmsf at 24h, APACHE II, and SOFA were evaluated as predictors of 28-day mortality (Table 4). Pmsf at 24h, respectively, combined with APACHE II and SOFA were significant predictors of 28-day mortality (Table 5, Figure 4a). APACHE II combined with Pmsf at 24h had the highest prediction (AUC, 0.806; 95% confidence interval, 0.671–0.941; P = 0.003; Table 5; Figure 4b.

Comparison of the Length of ICU Days Based on Pmsf-24h Predicted Mortality Cut-off Values

ICU day was significantly longer in the Pmsf-24h ≥14.9 mmHg group than in the <14.9 mmHg group (7 vs 5, p=0.010, Figure 5).

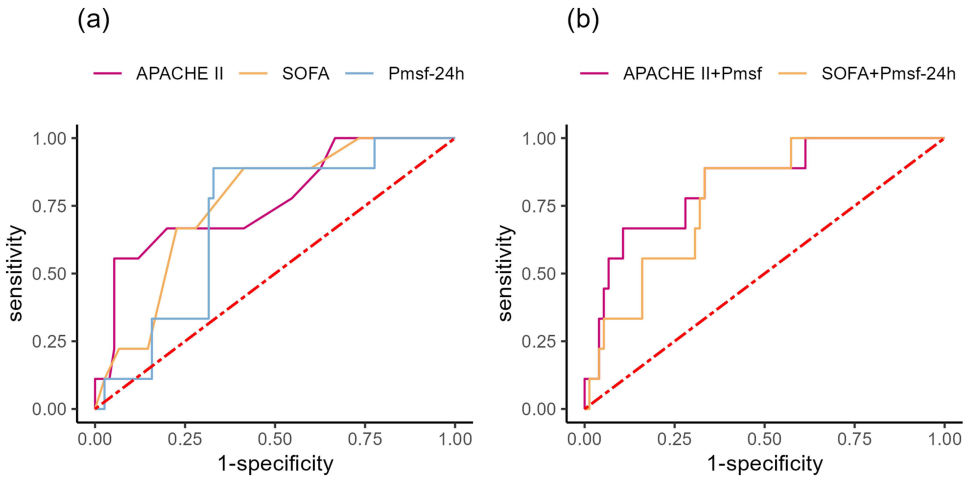

Figure 4 Predication of 28-day mortality: receiver operating characteristic curves of (**a**) single indicators and (**b**) combined indicators. APACHE II, Acute Physiology and Chronic Health Evaluation II score; SOFA Sequential Organ Failure Assessment score; Pmsf, mean systemic filling pressure.

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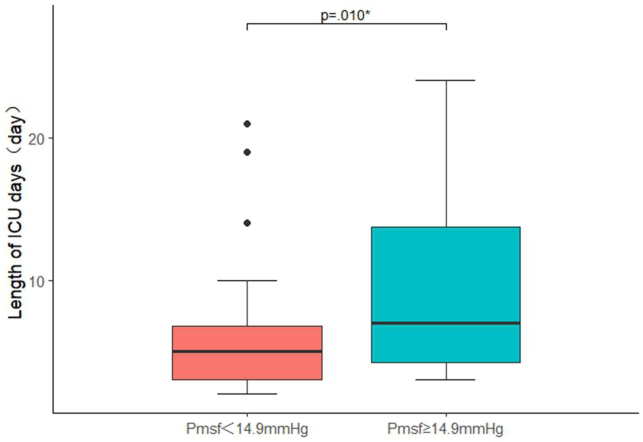

Figure 5 Comparison of the length of ICU days based on Pmsf-24h predicted mortality cut-off values. *indicates significance (*P* <0.05).

Discussion

To the best of our knowledge, this study is the first to describe changes in the venous return system and its significance in patients with constrictive pericarditis after pericardial stripping. We showed that Pmsf is associated with mortality in patients after pericardial stripping and that it improves the ability of the critically ill patient score to predict mortality.

In our study, mean Pmsf values ranged between 13 and 23 mmHg in the first 48 hours after surgery and trended downward over time. These values are higher than those reported in other populations. In a study of critically ill patients, Pmsf measured after cardiac arrest was 15 mmHg.[22] Another study reported that Pmsf was 12 mmHg 1 minute after cardiac arrest and 5 mmHg 8 min after death; use of norepinephrine at the time of death was associated with higher Pmsf.[15] Pmsf can increase from 16 to 18 mmHg during infusion of vasoactive drugs.[16] Systemic venous stasis is the main clinical manifestation of constrictive pericarditis and is caused by right heart insufficiency.[19] Pmsf is an indicator of effective volume and venous return.[23,24] Pmsf has been shown to be associated with venous return in post-cardiac surgery and cardiac arrest patients.[25,26]

In our study of CP patients after pericardial stripping, RVr was curvilinear, with the highest point at nearly 24 hours, and tended to decrease after negative equilibrium treatment. The original Guyton study showed that RVr decreased and venous return increased after an increase in intravascular volume.[27] Because cardiac obstruction and diastolic restriction were relieved and the high venous blood volume that had accumulated over a long period was rapidly returned to the heart. This is consistent with the pathophysiological features of CP (pericardial fibrosis leading to diastolic dysfunction and increased resistance).[19] We also found that Pmsf and RVr were significantly higher in the non-survival group than the survival group but Pmsf−CVP did not differ. In addition, none of these measures differed over time in either group. Previous studies have shown that in critically ill and postoperative cardiac patients, CVP and Lac are higher in patients who die.[28–30] In our study, CVP was higher in the non-survival group. Lac did not significantly differ between the groups, while it was higher in the non-survival group over time. CO and MAP were similar between the groups.

We found that the difference in Pmsf−CVP between 48 hours and the time of admission was positively correlated with total negative fluid balance at 48 hours, suggesting a gradual decrease in the differential venous return driving pressure in patients with negative fluid balance. Positive fluid balance in critically ill and postoperative cardiac patients is associated with higher Pmsf.[22,31,32] Pmsf can be used to assess volume responsiveness after cardiac surgery.[24] High blood lactate and low lactate clearance are considered indicators of poor prognosis.[30,33,34] In our study, Pmsf at 24 hours was negatively correlated with lactate clearance at 6 hours; therefore, we concluded that high Pmsf is detrimental. In addition, Pmsf−CVP increased with Pmsf in non-responding patients with no significant change in venous return driving pressure during the fluid load test in critically ill patients after surgery; however, venous return driving pressure increased in those with volume responsiveness.[35] Regarding the relationship between venous return and organ function, a study comparing MAP−CVP and MAP−Pmsf found that MAP−Pmsf correlated better with acute kidney injury and better represented the backward pressure of renal perfusion pressure.[36] Pmsf is an upstream indicator

of venous return. We investigated the relationship between Pmsf−CVP and renal function and found that in patients with preoperative pre-existing renal insufficiency, the differential venous return driving pressure at 48 hours versus that at 0 hours was positively correlated with serum creatinine at the time of transfer out of the ICU, suggesting that a large differential venous return driving pressure is not conducive to improved organ function. The venous return driving pressure difference was positively correlated with total negative fluid balance, suggesting that an appropriate negative balance is conducive to a decrease in venous return pressure difference, which may be beneficial for organ function recovery. Therefore, Pmsf and Pmsf−CVP can be used as prognostic indicators of perfusion and organ function. Our study also found that Pmsf is an independent risk factor for 28-day mortality in patients with constrictive pericarditis after pericardial stripping. Subgroup analysis based on Pmsf cut-off values showed that higher Pmsf was associated with longer length of ICU days. In previous studies, APACHE II and SOFA are good predictors of disease severity.[20,21] Pmsf at 24h had a diagnostic performance for predicting 28-day mortality. In addition, Pmsf at 24h combined with APACHE II had the best performance. Therefore, Pmsf appears to play a significant role in patients with constrictive pericarditis after pericardial stripping.

However, this study also has some limitations. First, it is a single-center study with a small sample size, and the number of mortality group is particularly limited. While our findings are promising, they require confirmation through further research with a larger cohort. In addition, Pmsf was estimated using three indicators: CVP, MAP, and CO.The disadvantage is that any changes in these measurements would affect Pmsf. However, studies comparing these three methods of measuring Pmsf have shown no significant difference in the results. Among these methods, the formula-based calculation of Pmsf is more clinically accessible because it relies on commonly used clinical indicators. Finally, our study was limited to patients with CP who underwent pericardial stripping. Our findings may not be generalizable to other populations.

Conclusions

Pmsf at 24h can be used as a valid indicator for prognostic assessment in patients with constrictive pericarditis admitted after pericardial stripping and improves the performance of APACHE II scores in predicting 28-day mortality. Close monitoring of Pmsf in patients after pericardial debridement may provide guidance for clinical management.

Data Sharing Statement

The dataset used and analyzed for the current study is available from the corresponding author upon reasonable request.

Ethics Approval and Consent to Participate

The study was approved by the institutional review board of Peking Union Medical College Hospital (approval number, I-23PJ871). The informed consent was waived by the institutional review board of Peking Union Medical College Hospital. The reasons for waiving the informed consent form include: This study does not involve additional interventions on patients. It only analyzes existing data, will not cause harm to patients, and the research team will strictly anonymize the collected data to ensure the security of patient information. All procedures were performed in accordance with the ethical standards of the local ethics committee on human experimentation and with the Helsinki Declaration of 1975.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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