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ORIGINAL ARTICLE

Predictive value of plasma volume status for contrast-induced nephropathy in patients with heart failure undergoing PCI

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Abstract

Aims Contrast-induced nephropathy remains a common complication of coronary procedure and increases poor outcomes, especially in patients with heart failure. Plasma volume expansion relates to worsening prognosis of heart failure. We hypothesized that calculated plasma volume status (PVS) might provide predictive utility for contrast-induced nephropathy in patients with heart failure undergoing elective percutaneous coronary intervention (PCI).

Methods and results We enrolled 441 patients with heart failure undergoing elective PCI from 2012 to 2018. Pre-procedural estimated PVS by the Duarte's formula (Duarte-ePVS) and Kaplan–Hakim formula (KH-ePVS) were calculated for all patients. CIN was defined as an absolute serum creatinine (SCr) increase ≥0.5 mg/dL or a relative increase ≥25% compared with the baseline value within 48 h of contrast medium exposure. We assessed the association between PVS and CIN in patients with heart failure undergoing elective PCI. In 441 patients, 28 (6.3%) patients developed CIN. The median Duarte-ePVS was 4.44 (3.87, 5.13) and the median KH-ePVS was −0.03 (−0.09, 0.05). The best cutoff values for Duarte-ePVS and KH-ePVS to predict CIN were 4.64 (with 78.6% sensitivity and 61.7% specificity) and 0.04 (with 64.5% sensitivity and 75.5% specificity), respectively. After adjusting for potential confounding variables, KH-ePVS > 0.04 [odds ratio (OR) 2.685, 95% confidence interval (CI) 1.012–7.123, P = 0.047] remained significantly associated with CIN whereas Duarte-ePVS was not.

Conclusions Pre-procedural KH-ePVS is an independent risk factor for CIN in patients with heart failure undergoing elective PCI. The best cutoff point of KH-ePVS for predicting CIN was 0.04.

Keywords Plasma volume status; Contrast-induced nephropathy; Heart failure; Elective percutaneous coronary intervention

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Introduction

Contrast-induced nephropathy (CIN) is a common complication following administration of iodinated contrast media (CM) during angiography or other medical procedures, which is one of the leading causes of nosocomial acute renal failure.[1,2] It is associated with increased morbidity, mortality, length of hospitalization, and acceleration towards end-stage renal disease.[3–5] Heart failure is a well-known risk factor for CIN,[3,6,7] but patients with heart failure cannot receive adequate hydration because fluid overload may worsen heart failure. Therefore, novel markers of plasma volume expansion might better stratify risk and guide hydration in patients with heart failure.

Plasma volume expansion is a significant complication which predicts poor prognosis in patients with heart failure.[8]

It can manifest with haemodynamic congestion and peripheral and pulmonary oedema but is difficult to quantify non-invasively.[9] Tracer-dilution techniques can measure plasma volume optimally but have not been readily available to clinicians because they are costly, time consuming and complex.[10,11] Recent studies showed calculated estimates of plasma volume status (PVS), which derived from a routine blood count and/or body weight, was moderately-to-highly correlated with the directly measured plasma volume and had prognostic value for heart failure.[12–15] However, the relationship between PVS and CIN in patients with heart failure undergoing elective percutaneous coronary intervention (PCI) has not been evaluated. The purpose of our study is to explore the predictive value of PVS on CIN in patients with heart failure undergoing elective PCI.

Method

Study population

We conducted a retrospective, single-centre observational study, enrolling consecutive patients with heart failure undergoing elective PCI at Fujian Provincial Hospital in China from January 2012 to December 2018. Exclusion criteria were (i) lacking anyone of the data on haematocrit, haemoglobin, weight and pre-procedural or post-procedural serum creatinine (SCr) levels; (ii) estimated glomerular filtration rate (eGFR) $<$15 mL/min/1.73 m^2 or dialysis; (iii) iodic contrast medium administration during the preceding 7 days; (iv) cancer with expectation of life less than 1 year; (v) died within 24 h after procedure; (vi) allergy to contrast medium.

Protocol

Haematocrit, haemoglobin and body weight were measured for each patient at admission and SCr was measured at admission and daily for the 2 days after contrast exposure. Data including baseline demographic, comorbidities, clinical risk factors, clinical treatment, and laboratory results were drawn from medical records. Elective PCI was performed by experienced interventional cardiologists and medication use was determined by clinicians according to current guidelines. The low-osmolar, non-ionic CM (either Iopamiron or Ultravist, both 370 mg I/mL) was administered during all procedures. Hydration therapy was at the clinician's discretion, and all patients received 0.9% normal saline (at a rate of 0.5 mL/kg/h) for 12 h during perioperative period except those who were intolerant. The study protocol was approved by the ethics committee of the Fujian Provincial Hospital, China (ethics approval number: K2019-07-011).

Plasma volume equations

The PVS was calculated in all study participants using both the Duarte's formula and Kaplan–Hakim formula. Estimated plasma volume assessed by the Duarte's formula (Duarte-ePVS) was calculated as follows: Duarte-ePVS = [(1 − haematocrit)/(haemoglobin)] × 100, measured in the unit of decilitres per gram (dL/g).[14]

Estimated plasma volume assessed by Kaplan–Hakim formula (KH-ePVS) was calculated by comparing actual plasma volume (aPV) to ideal plasma volume (iPV). The aPV was calculated by following equation[16] incorporating haematocrit and weight derived from curve-fitting techniques:

$$\mathrm{aPV} = (1 - \text{haematocrit}) \times [a + (b \times \text{body weight})]$$
$$(a = 1530 \text{ in males and a} = 864 \text{ in females},$$
$$b = 41.0 \text{ in males and b} = 47.9 \text{ in females}).$$

The iPV was calculated by following equation[17]:

$$\mathrm{iPV} = c \times \text{body weight } (c = 39 \text{ in males and c} = 40 \text{ in females}).$$

Therefore, relative PVS which reflecting the degree to which patients have deviated from their ideal plasma volume was calculated by following equation:

$$\mathrm{KH - ePVS} = [(\mathrm{aPV–iPV})/\mathrm{iPV}] \times 100\%.$$

Definition and study endpoint

The primary endpoint in the study was incidence of CIN, defined as an absolute SCr increase ≥0.5 mg/dL or a relative increase in serum creatinine ≥25% compared with the baseline value within 48 h of contrast medium exposure.[18] Additional endpoint was long-term mortality. The diagnosis of heart failure was based on the presence of appropriate symptoms (e.g. orthopnoea) and signs (e.g. bilateral oedema, increased jugular venous pressure), elevated level of brain natriuretic peptide (BNP) or N-terminal pro B-type natriuretic peptide (NT-proBNP), and a left ventricular ejection fraction (LVEF) ≤ 50%.[19] Anaemia was defined as haematocrit$<$0.39 (for male patients) or $<$0.36 (for female patients). The eGFR was calculated using the modified modification of diet in renal disease equation: 186.3 × SCr-1.154 × (age in years) − 0.203 × 1.212 (if patient was black) × 0.742 (if patient was female).[20]

Statistical analysis

All data were analysed with SPSS 25.0. The baseline characteristics were compared between two groups divided by CIN. Data are presented as mean ± standard deviation (SD) for normally distributed continuous variables, or medians

and interquartile ranges for unevenly distributed continuous variables. The Student's *t* test or Wilcoxon rank sum test was performed to determine the differences between groups. The categorical variables were represented as percentages and compared by chi-square test or Fisher exact test. Risk factors were initially screened for univariate association with CIN, and variables with $P < 0.20$ were then entered into a multivariate logistic analysis with forward stepwise algorithm. Multivariate logistic regression analysis was performed to identify independent risk factors for CIN. Receiver operator characteristic (ROC) curve analysis was performed to identify the optimal cutoff point for PVS to predict CIN. Areas under the curve (AUC) were calculated as measures of the accuracy of the test. The Kaplan–Meier curve assessed the survival between different estimated PVS groups (Duarte-ePVS $>$ 4.64 vs. Duarte-ePVS $\leq$ 4.64 and KH-ePVS $>$ 0.04 vs. KH-ePVS $\leq$ 0.04). A two-sided *P* value $<$0.05 was considered significant.

Results

Baseline characteristics

A total of 441 patients were included in this study, of whom 28 (6.3%) developed CIN. The median Duarte-ePVS was 4.44 (3.87, 5.13) and the median KH-ePVS was −0.03 (−0.09, 0.05). Baseline characteristics for patients with and without CIN are shown in *Table 1*. Patients with CIN were older, more

Table 1 Baseline variables in patients with and without CIN

	Total (*n* = 441)	CIN(−) (*n* = 413)	CIN(+) (*n* = 28)	*P* value
Demographics				
Age, years	64.61 ± 11.42	64.13 ± 11.35	71.14 ± 10.45	0.001
Age >75 years, *n* (%)	87 (19.7%)	74 (17.9%)	13 (46.4%)	<0.001
Sex, female, *n* (%)	80 (18.1%)	73 (17.7%)	7 (25.0%)	0.330
BMI, kg/m^2	23.88 (21.37–26.11)	23.88 (21.43–26.09)	23.85 (20.53–27.25)	0.810
Systolic blood pressure, mmHg	127.62 ± 21.32	127.57 ± 21.29	128.25 ± 22.10	0.854
Diastolic blood pressure, mmHg	74.29 ± 12.31	74.31 ± 12.30	73.93 ± 12.66	0.910
Medical history				
Smoker, *n* (%)	223 (50.6%)	211 (51.1%)	12 (42.9%)	0.399
Myocardial infarction, *n* (%)	195 (44.2%)	177 (42.9%)	18 (64.3%)	0.027
Hypertension, *n* (%)	278 (63.0%)	258 (62.5%)	20 (71.4%)	0.342
Diabetes, *n* (%)	182 (41.3%)	172 (41.6%)	10 (35.7%)	0.537
Atrial fibrillation, *n* (%)	53 (12.0%)	47 (11.4%)	6 (21.4%)	0.114
Anaemia, *n* (%)	184 (41.7%)	163 (39.5%)	21 (75.0%)	<0.001
Medication				
Antiplatelet agents, *n* (%)	440 (99.8%)	412 (99.8%)	28 (100.0%)	0.794
Statin use, *n* (%)	434 (98.4%)	406 (98.3%)	28 (100.0%)	0.487
ACEI/ARB, *n* (%)	372 (84.4%)	348 (84.3%)	24 (85.7%)	0.838
β-blocker, *n* (%)	388 (88.0%)	363 (87.9%)	25 (89.3%)	0.826
Diuretics, *n* (%)	278 (63.0%)	251 (60.8%)	27 (96.4%)	<0.001
Laboratory result				
NT-proBNP, pg/mL	1485.00 (426.00–3048.50)	1354.00 (417.05–2870.50)	5415.00 (2296.00–12046.75)	<0.001
Creatinine, mg/dL	0.95 (0.81–1.10)	0.94 (0.81–1.08)	1.06 (0.84–1.40)	0.032
WBC, 10^9/L	7.10 (5.80–8.69)	7.10 (5.80–8.55)	7.20 (5.65–10.20)	0.519
HGB, g/L	134.02 ± 17.62	135.02 ± 17.01	120.54 ± 20.34	<0.001
HCT	0.40 ± 0.05	0.40 ± 0.05	0.36 ± 0.05	<0.001
Cholesterol, mmol/L	4.14 ± 1.11	4.13 ± 1.11	4.25 ± 1.11	0.646
LDL-C, mmol/L	2.62 ± 1.05	2.61 ± 1.05	2.77 ± 0.98	0.502
eGFR, mL/min/1.73 m^2	83.45 ± 24.98	84.43 ± 24.52	70.27 ± 27.84	0.004
eGFR < 60 mL/min/1.73 m^2, *n* (%)	71(16.1%)	60(14.5%)	11(39.3%)	0.001
LVEF,%	42.25 ± 6.37	42.39 ± 6.26	40.34 ± 7.66	0.103
KH-ePVS	−0.03(−0.09–0.05)	−0.03(−0.10–0.04)	0.07(−0.02–0.11)	<0.001
Duarte-ePVS	4.44(3.87–5.13)	4.38(3.86–5.04)	5.12(4.65–6.72)	<0.001
Procedure performed				
Multi-vessel coronary artery disease, *n* (%)	379 (85.9%)	355 (86.0%)	24 (85.7%)	0.972
Number of stents, *n*	1.67 ± 0.93	1.69 ± 0.93	1.43 ± 0.88	0.149
Contrast volume, mL	194.14 ± 64.80	194.35 ± 63.93	191.25 ± 76.76	0.782
Iso-osmolar contrast media use, *n* (%)	150(34.0%)	138(33.4%)	12(42.9%)	0.307

Abbreviations: ACEI/ARB, angiotensin-converting enzyme inhibitor/angiotensin receptor blocker; BMI, body mass index; CIN, contrast induced nephropathy; NT-proBNP, N-terminal pro B type natriuretic peptide; HCT, haematocrit; HGB, haemoglobin; LDL-C, low-density lipoprotein; eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction; KH-ePVS, Kaplan–Hakim-estimated plasma volume status; Duarte-ePVS, Duarte-estimated plasma volume status; WBC, white blood cells.

likely to have myocardial infarction, anaemia, worse renal function, and higher levels of NT-proBNP, Duarte-ePVS, and KH-ePVS. A higher percentage of diuretics using during hospitalization was shown in patients who developed CIN (all $P < 0.05$).

Receiver operator characteristic analysis and multivariable factors for predicting contrast-induced nephropathy

According to the ROC curve analysis, the optimal cutoff value for KH-ePVS to predict CIN was measured as >0.04, with 64.5% sensitivity and 75.5% specificity, and the optimal cutoff value for Duarte-ePVS to predict CIN was measured as >4.64, with 78.6% sensitivity and 61.7% specificity [for KH-ePVS: C-statistic = 0.718; 95% confidence interval (CI) 0.674–0.760; for Duarte-ePVS: C-statistic = 0.720; 95% CI 0.618–0.823; *Figure 1*]. The rate of CIN was higher in patients with KH-ePVS > 0.04 (15.13% vs. 3.11%) and those with Duarte-ePVS > 4.64 (12.15% vs. 2.31%) than patients with lower estimated PVS (both $P < 0.001$, *Figure 2A,B*).

Results of multivariate logistic regression analysis have been displayed in *Table 2*. After adjusting for potential confounding risk factors including age >75 years, myocardial infarction, anaemia, lg (NT-proBNP) and eGFR <60 mL/min/1.73m^2, KH-ePVS > 0.04 remained significant independent predictor of CIN [odds ratio (OR) 2.685, 95% CI 1.012–7.123, $P = 0.047$], whereas there was no significant association between CIN and Duarte-ePVS either considered as a continuous ($P = 0.216$) or categorical variable ($P = 0.184$).

Plasma volume status and long-term outcomes

The median follow-up period was 637 days (interquartile range: 387 to 990 days). Compared with patients with KH-ePVS ≤ 0.04, the Kaplan–Meier curve showed that patients with KH-ePVS > 0.04 had a higher rate of all-cause long-term mortality ($P = 0.028$; *Figure 3A*). Similarly, the cumulative

Figure 1 Receiver operator characteristic (ROC) curves for estimated plasma volume status (ePVS) to predict contrast-induced nephropathy (CIN).

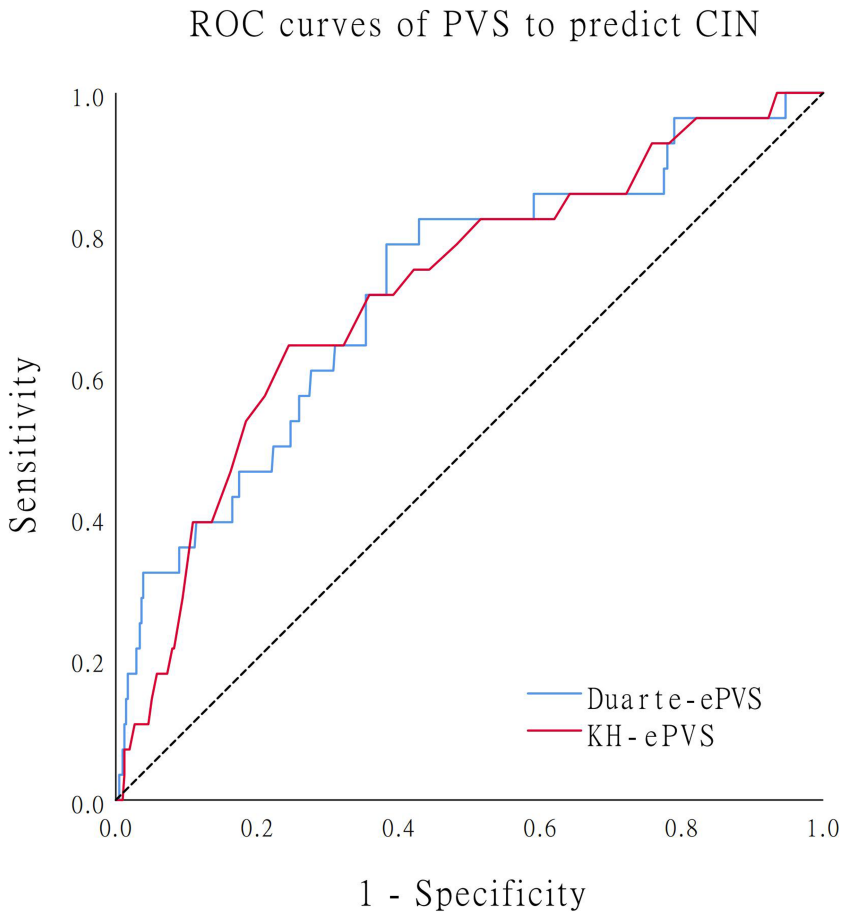
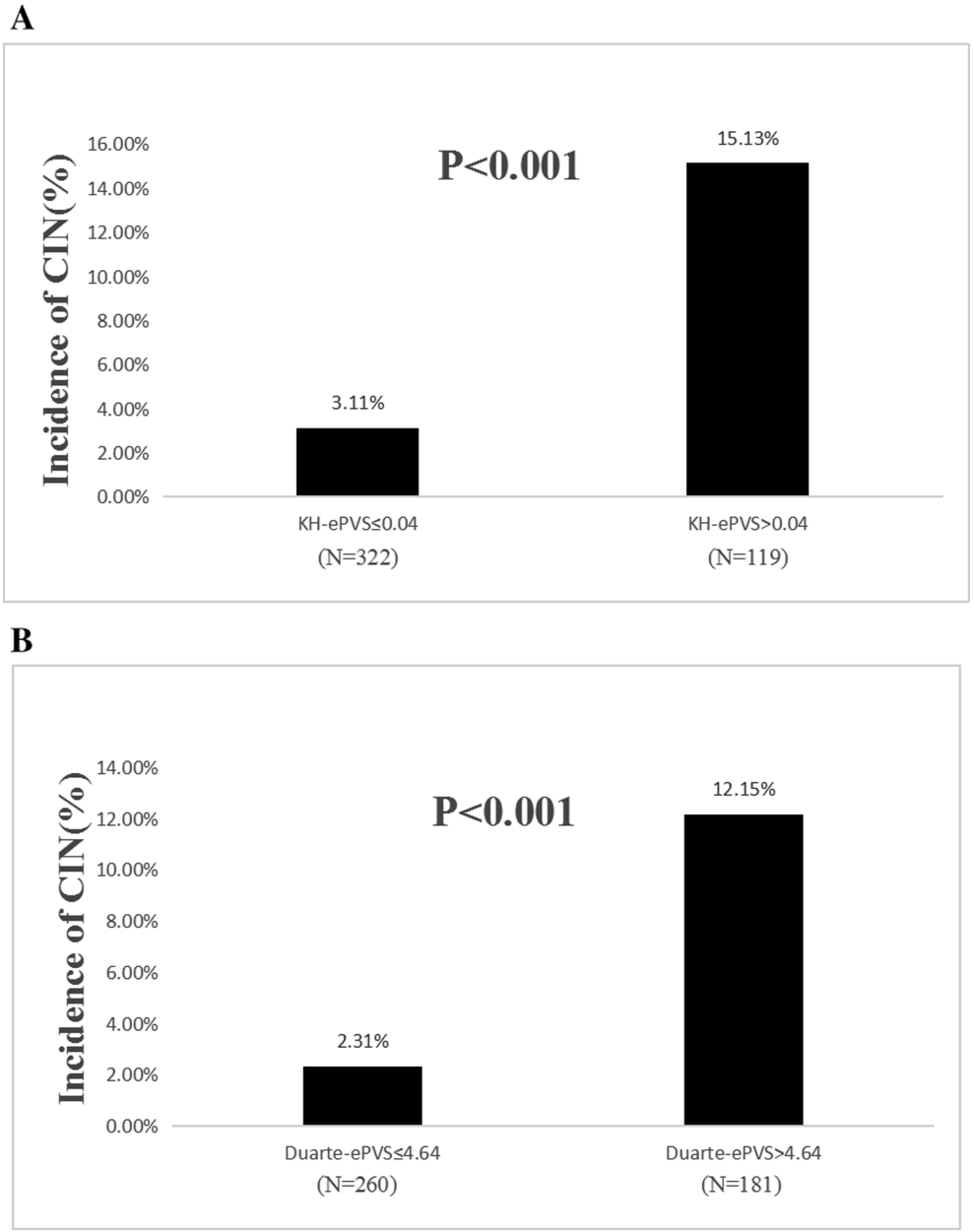


Figure 2 (A) Contrast-induced nephropathy (CIN) incidence between different KH-ePVS groups. (B) CIN incidence between different Duarte-ePVS groups.



rate for all-cause death was higher in patients with Duarte-ePVS $>$ 4.64 than those with Duarte-ePVS $\leq$ 4.64 (P = 0.035; *Figure 3B*).

Discussion

To the best of our knowledge, this is the first study that investigates the relationship between PVS and incidence of CIN. The present study showed that higher pre-procedural KH-ePVS, which obtained from Kaplan–Hakim formula was associated with increased risk of CIN in patients with heart failure undergoing elective PCI, and the best cutoff value for KH-ePVS to predict CIN was 0.04 with 64.5% sensitivity and 75.5% specificity according to the ROC analysis (C-statistic = 0.718; 95% CI, 0.674–0.760). Even after adjusting for potential confounding risk factors like myocardial infarction, anaemia, age $>$ 75 years, lg (NT-proBNP)

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Table 2 Association between estimated PVS and CIN

	Univariate logistic regression			Multivariate logistic regression		
	OR	95% CI	*P* value	OR	95% CI	*P* value
KH-ePVS > 0.04	5.560	2.486–12.436	<0.001	2.685	1.012–7.123	0.047
KH-ePVS, continuous (per 0.1-unit increase)	1.830	1.328–2.520	<0.001	1.198	0.751–1.910	0.449
Duarte-ePVS > 4.64	5.857	2.325–14.760	<0.001	4.505	0.490–41.395	0.184
Duarte-ePVS, continuous (per 1-unit increase)	1.912	1.431–2.554	<0.001	1.316	0.852–2.031	0.216

Abbreviations: CI, confidence interval; CIN, contrast induced nephropathy; NT-proBNP, N-terminal pro B type natriuretic peptide; HCT, haematocrit; HGB, haemoglobin; LDL-C, low-density lipoprotein; eGFR, estimated glomerular filtration rate; LVEF, left ventricular ejection fraction; KH-ePVS, Kaplan–Hakim-estimated plasma volume status; Duarte-ePVS, Duarte-estimated plasma volume status; WBC, white blood cells.
Adjusted by age >75 years, myocardial infarction, anaemia, lg (NT-proBNP), and eGFR < 60 mL/min/1.73 m^2.

and eGFR < 60 mL/min/1.73 m^2, KH-ePVS > 0.04 was still an independent risk factor for predicting CIN markedly. However multivariate logistic analysis did not show association between Duarte-ePVS which obtained from Duarte's formula and CIN. Meanwhile, in our study both KH-ePVS > 0.04 and Duarte-ePVS > 4.64 were associated with higher rate of long-term mortality.

Heart failure, as a known risk factor for CIN, has been included in the Mehran risk score for CIN.[3,6,7] Worsened cardiac function contributes to adverse haemodynamic state and reduced renal blood flow, which stimulates the renin–angiotensin system and sympathetic nervous system, increasing levels of inflammatory factors and ROS, and consequently promoting the development of CIN.[21,22] Plasma volume expansion is a significant complication in patients with heart failure and predicts poor prognosis.[8] Although hydration is effective in preventing CIN, excessive plasma volume expansion might cause more harm than benefit in patients with heart failure.[23,24] Therefore, it is significant to research the association between plasma volume and CIN in patients with heart failure.

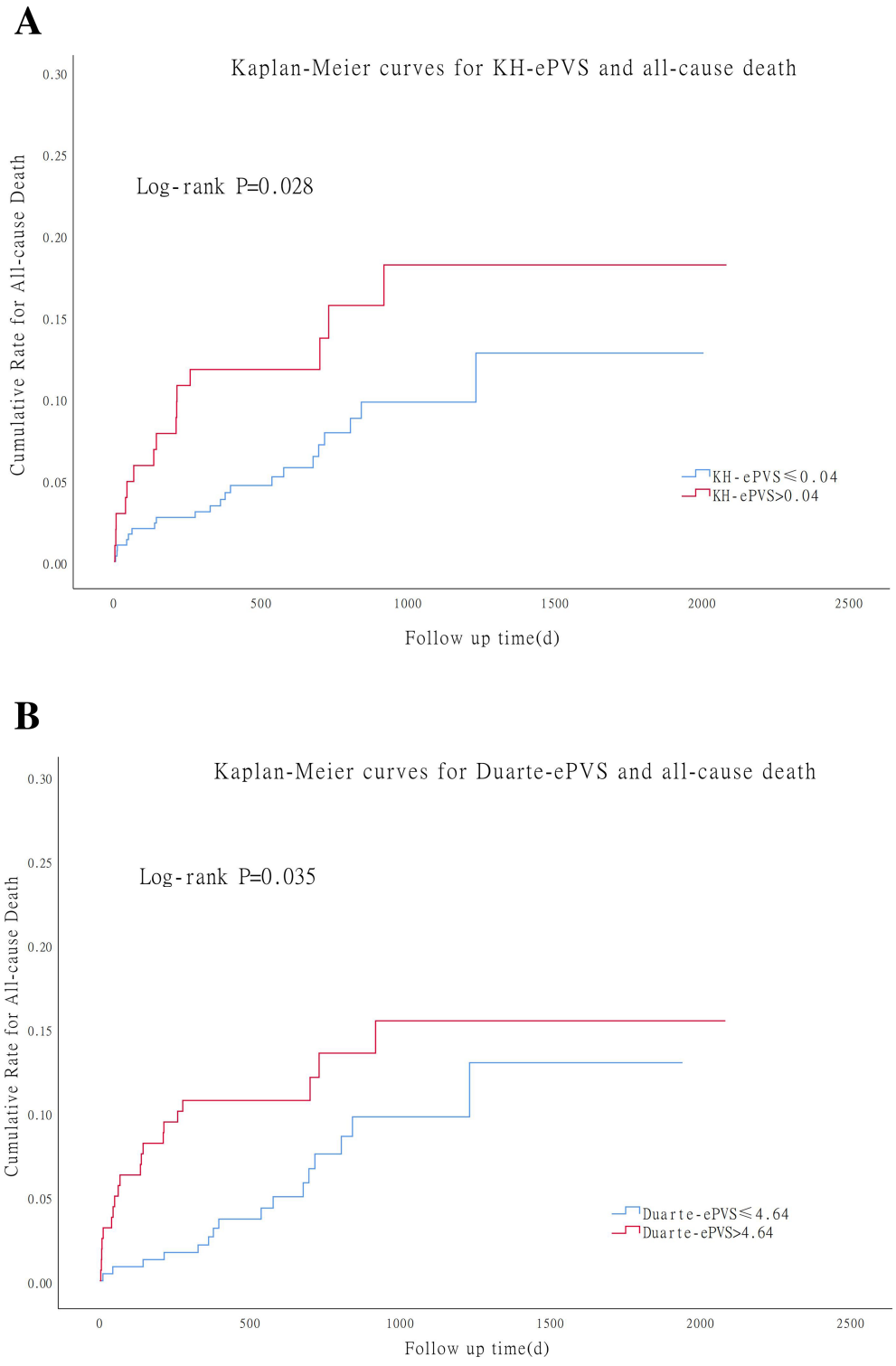
Plasma volume is classically measured by Tracer-dilution techniques, but these methods are expensive, time consuming and complex.[10,11] Estimated PVS, calculated from a routine blood count and/or body weight, has been validated as surrogate of intravascular filling using radiolabelled albumin.[12,13] Prior studies have demonstrated the independent association between PVS and poor outcome, consistently and across a wide heart failure spectrum, regardless of the formula used.[25] Duarte *et al*.[14] put forward Duarte's formula firstly and revealed prognostic value of Duarte-ePVS in the setting of heart failure complicating acute myocardial infarction. Subsequent researches further confirmed the association between Duarte-ePVS and poor outcome in patients with acute decompensated heart failure or chronic heart failure with preserved ejection fraction.[15,26–29] Ling *et al*.[13] found that KH-ePVS independently predicted death and first morbid events in chronic heart failure and KH-ePVS > −4% was associated with the worst prognosis. Similar prognostic value of KH-ePVS was shown in patients with acute heart failure syndromes[30] or preserved ejection fraction.[31] Furthermore, patients with a higher PVS often have poor renal function.[13,31] A KH-ePVS ≥ 5.6% independently predicted greater postoperative renal complication in patients undergoing coronary artery bypass graft.[32] Our study demonstrated the predictive value of PVS for CIN for the first time.

In fact, estimated PVS is also a congestion marker in patients with heart failure, which associated with E/e,[33] pulmonary artery pressure,[34] and BNP.[31] Mullens *et al*. found venous congestion was the most important haemodynamic factor for worsening of renal function in advanced decompensated heart failure.[35] Increased renal venous pressure from venous congestion reduces the arteriovenous gradient over renal circulation, increases renal interstitial pressure, and thereby impairs renal blood flow and destroy the architecture of the renal tissues, afterwards influencing local oxygen exchange and leading to diffuse metabolic dysfunction.[35–37] This may be the mechanism by which ePVS was associated with CIN in patients with heart failure.

Although Duarte's formula is now the dominating formula to calculate ePVS, it is KH-ePVS—not Duarte-ePVS—that showed predictive value for CIN in our study. Possible reasons for these differences in associations might rely on KH-ePVS incorporating body weight: KH-ePVS might reflect the degree to which patients have deviated from their ideal weight rather than a direct estimated measurement of plasma volume.

The findings of our study suggest the predictive value of PVS for CIN in patients with heart failure undergoing elective PCI, which is of great significance in clinical practice. Based on two readily available parameters, body weight and haematocrit, KH-ePVS can be calculated quickly to make effective measures taken for the certain higher-risk patients before procedure. Moreover, for the patients with KH-ePVS > 0.04, who suffer more from congestion, we suspect that hydration may not be a good strategy for preventing CIN and some decongestion treatment like diuresis may be better. The current study just puts forward consideration, and more prospective observational and interventional studies are needed to determine its value.

Figure 3 (A) Kaplan–Meier curves for KH-ePVS and long term all-cause death. (B) Kaplan–Meier curves for Duarte-ePVS and long term all-cause death.



This study has some limitations. First, this study was a single-centre, observational study, with a relatively small sample size, which potentially limit the universality of the findings. Second, data on peri-procedural fluid intake and output are lacking, which may influence the incidence of CIN. Third, although we had adjusted for many confounding

factors, there were still some potential confounders that cannot be controlled. Despite these limitations, our study revealed the association between volume expanding and CIN in patients with heart failure undergoing elective PCI, and provided novel insights into the PVS.

Conclusions

Pre-procedural PVS is an independent risk factor for CIN in patients with heart failure undergoing elective PCI. The best cutoff point for KH-ePVS to predict CIN was 0.04. These findings might guide use of preventive measures and therapy to prevent CIN.

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Conflict of interest

The authors have declared that no conflict of interest exists.

Informed consent

Informed consents were obtained from all participants included in the study.

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