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ORIGINAL PAPER

Prognostic significance of dilated inferior vena cava in advanced decompensated heart failure

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Abstract Dilated inferior vena cava (IVC) is prevalent among patients with heart failure (HF), but whether its presence predicts worsening renal function (WRF) or adverse outcomes is unclear. This cohort study analyzed patients with left ventricular ejection fraction <40 % and repeated hospitalizations (≥2 times) for HF between August 2009 and August 2011. The study endpoints were death and HF re-hospitalization. Among baseline parameters, IVC diameter was the most powerful predictor for the development of WRF (area under the curve = 0.795, cut-off value = 20.5 mm). During the 2-year follow-up, 36 patients (49 %) were re-hospitalized for HF and 14 patients (19 %) died. The event rates were significantly greater in the WRF group than in the non-WRF group (71 vs. 30 %, $P < 0.001$ for HF re-hospitalization; 29 vs. 10 %, $P = 0.03$ for death). In Cox regression model, the risk of combined end-points was increased in patients with aging, elevated blood urine nitrogen, IVC >21 mm, and WRF. When adjusted for confounding factors, IVC >21 mm [hazard ratio (HR) 3.73, 95 % confidence interval (CI) 1.66–8.34] and WRF (HR 2.68, 95 % CI 1.07–6.75) were significant predictors for adverse outcomes. In patients with advanced decompensated HF, dilated IVC (>21 mm) predicted the development of WRF and could be a predictor for adverse outcomes.

Keywords Heart failure · Inferior vena cava · Survival · Worsening renal function

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Introduction

Impaired renal function is highly prevalent among patients with heart failure (HF). Moreover, the coexistence of renal and cardiac dysfunction in the same patient, known as cardiorenal syndrome, has an extremely poor prognosis [1, 2]. For patients with chronic abnormalities in cardiac function (e.g., chronic HF), hypoperfusion alone cannot explain renal function decline in this setting. The presence of systemic venous congestion has been considered to be one of the mechanisms for the cardiorenal syndrome [3]. Systemic venous congestion may worsen renal function from the implication of experimental animal data [4, 5] and the evaluation study of congestive HF and pulmonary catheterization effectiveness (ESCAPE) trial [6]. Systemic venous congestion is highly prevalent among patients with advanced HF, but whether its presence predicts worsening renal function (WRF) or adverse outcomes is unclear. Echocardiography allows non-invasive evaluation of systemic venous congestion by measuring the size and collapsibility of the inferior vena cava (IVC) [7]. The aims of this study were to evaluate the relation between dilated IVC and WRF, and the prognostic significance of dilated IVC and WRF in patients with advanced decompensated HF.

Methods

Patient population

This cohort study analyzed consecutive patients aged 18 years or older with repeated hospitalizations (≥2 times) for decompensated HF, who visited our emergency room between August 2009 and August 2011. Patients were included if they had left ventricular (LV) ejection fraction (EF) <40 % and had echocardiography within 24 h after going to the emergency room to decrease the effects of intravenous diuretics that may interfere the measurements of the IVC size and collapsibility. Exclusion criteria included mechanical ventilation, end-stage renal disease under renal replacement therapy, intravenous inotropic support, congenital heart disease, prior valvular cardiac surgery, or poor echocardiographic image quality. The ischemic etiology of HF was defined by one of the following criteria: (1) significant epicardial coronary artery stenosis (≥50 %); or (2) history of myocardial infarction or coronary revascularization. The study conforms with the principles outlined in the Declaration of Helsinki. The research protocol was approved by the local Institutional Review Board.

Two-dimensional echocardiography

Conventional two-dimensional echocardiography was performed using commercially available equipment (Vivid 7, General Electric Vingmed Ultrasound, Horten, Norway) with a 2.5-MHz transducer. LV EF was determined by the biplane Simpson's method. According to the guidelines for the echocardiographic assessment of the right heart in adults, (1) dilated IVC is defined if diameter >2.1 cm; (2) the estimated right atrial (RA) pressure is 3 mmHg if an IVC diameter is ≤2.1 cm and collapse is >50 %; RA pressure is 15 mmHg if an IVC diameter is >2.1 cm and collapse is <50 %; RA pressure is 8 mmHg if the IVC diameter and collapsibility do not fit the above paradigm, respectively [7]. Peak velocity of tricuspid regurgitation (TR) was recorded by continuous wave Doppler and TR was graded qualitatively using the Framingham Heart Study criteria: mild if regurgitant jet area/RA area was <20 %, moderate if 20–40 %, or severe if >40 % [8]. The pressure gradient (PG) between right ventricle and right atrium was calculated by using the simplified Bernoulli equation [9, 10].

WRF

Serum creatinine in the emergency room was recorded. Glomerular filtration rate (GFR) was estimated using the four-variable modification of diet in renal disease study equation [11]. The development of WRF was defined as a rise in serum creatinine of >0.3 mg/dl, similar to prior studies [12–14]. The subjects were divided to whom developed WRF during hospitalization versus those who did not.

Study endpoints

The intermediate end-point was defined as the occurrence of WRF during hospitalization. The final end-point was the combined death and re-hospitalization during the 2-year follow-up. Re-hospitalization was defined as an unplanned overnight stay in our hospital because of progression of HF or as a direct result of HF. Patients had to have typical symptoms and signs of HF, using standard criteria. All events were evaluated and adjudicated by two independent observers.

Statistical analysis

Data are presented as mean ± standard deviation or as a count (percentage). Statistical analysis was performed using the Statistical Package for Social Sciences statistical software, version 19 for Windows. Chi square test for categorical variables and two-sample *t* test for continuous variables were used for comparisons between patients who developed WRF and those who did not. Receiver operating characteristic (ROC) curves were generated to determine the predictive ability of clinical and echocardiographic parameters for WRF. Stepwise logistic regression analysis was used to evaluate the independent effect of our aimed variables for WRF. Cox proportional model was used to evaluate the time-to-event associations with death and re-hospitalization for HF during the 2-year follow-up. Kaplan–Meier curves were plotted, and the log-rank test used to compare between groups. For all analysis, a P value <0.05 was considered statistically significant.

Results

Baseline characteristics

Baseline characteristics for the study patients are shown in Table 1. The mean age was 63.6 ± 16.2 years and approximately three fourths of the patients were male. No significant differences were found in age, sex, systolic blood pressure, heart rate, ischemic etiology, risk factors of coronary artery disease, atrial fibrillation, biochemistry data (serum creatinine, estimated glomerular filtration rate, hemoglobin, serum sodium), and medications for HF between patients with WRF and patients without WRF. The values of blood urine nitrogen (BUN) and brain natriuretic peptide (BNP) were significantly higher in patients with WRF than in those without WRF. Thirty-six patients (49 %) were re-hospitalized for decompensated HF and 14 patients

Table 1 Baseline characteristics

	Total (n = 74)	WRF (n = 34)	No WRF (n = 40)	*P* value
Age (years)	63.6 ± 16.2	64.7 ± 15.3	62.6 ± 17.0	0.08
Male [n (%)]	54 (73)	23 (68)	31 (78)	0.34
Ischemic etiology [n (%)]	27 (36)	10 (29)	17 (43)	0.24
Atrial fibrillation [n (%)]	22 (30)	10 (29)	12 (30)	0.96
SBP (mmHg)	131.1 ± 27.6	132.9 ± 32.2	129.6 ± 23.4	0.61
Heart rate (beats/min)	90.1 ± 17.7	92.3 ± 17.8	88.2 ± 17.7	0.32
Diabetes [n (%)]	28 (38)	14 (41)	14 (35)	0.59
Hypertension [n (%)]	41 (55)	18 (53)	23 (58)	0.69
Dyslipidemia [n (%)]	26 (35)	13 (38)	13 (33)	0.60
Smoking [n (%)]	33 (45)	16 (47)	17 (43)	0.69
Prior cardiac surgery [n (%)]	7 (9)	4 (12)	3 (8)	0.54
Past history of stroke [n (%)]	10 (14)	6 (18)	4 (10)	0.34
Blood results				
Hemoglobin (g/dl)	13.2 ± 2.4	13.4 ± 2.6	13.1 ± 2.1	0.52
Creatinine (mg/dl)	1.2 ± 0.8	1.4 ± 1.0	1.1 ± 0.7	0.09
GFR (ml/min/1.73 m^2)	78 ± 47	75 ± 57	80 ± 37	0.68
BUN (mg/dl)	24.8 ± 12.8	30.6 ± 14.0	19.8 ± 9.4	<0.01
Sodium (mEq/l)	137 ± 5	137 ± 6	137 ± 5	0.99
BNP (pg/ml)[a]	843 (27–4,910)	1,300 (67–4,910)	644 (27–4,460)	0.03
Medications [n (%)]				
ACEI/ARB	59 (80)	26 (76)	33 (83)	0.53
Digoxin	15 (20)	8 (24)	7 (18)	0.52
ACEI/ARB	59 (80)	26 (76)	33 (83)	0.53
Digoxin	15 (20)	8 (24)	7 (18)	0.52
Beta blocker	55 (74)	25 (74)	30 (75)	0.89
Spirolactone	23 (31)	13 (38)	10 (25)	0.22
Loop diuretic	65 (88)	32 (94)	33 (83)	0.13
Outcomes [n (%)]				
Re-hospitalization	36 (49)	24 (71)	12 (30)	<0.01
Death	14 (19)	10 (29)	4 (10)	0.03

Values are expressed as mean ± standard deviation or number (percentage)

SBP systolic blood pressure, *PH* past history, *CAD* coronary artery disease, *GFR* glomerular filtration rate, *BUN* blood urine nitrogen, *BNP* brain natriuretic peptide, *ACEI* angiotensin converting enzyme inhibitor, *ARB* angiotensin receptor blocker

[a] Median and interquartile range

(19 %) died during the follow-up period. The clinical event rate over 2 years was significantly greater in the WRF group than in the non-WRF group (71 vs. 30 %, $P < 0.001$ for re-hospitalization for worsening HF; 29 vs. 10 %, $P = 0.03$ for all-cause mortality).

Echocardiographic parameters

There were significant differences in LV EF, LV end-systolic volume (ESV), RA pressure, TR PG, IVC diameter, IVC >21 mm, and TR severity ≥moderate degree between patients with and without WRF (Table 2). In the logistic-regression model, dilated IVC was associated with WRF [odds ratio (OR) 1.37, 95 % confidence interval (CI) 1.18–1.59, $P < 0.01$], and IVC diameter remained significant after adjustment for the BUN, LV EF and RA pressure, or adjustment for the BNP, LV ESV and TR PG (Table 3). In the ROC curve analysis for the prediction of WRF, several indices including LV EF, LV ESV, BUN, BNP, RA pressure, TR PG, and IVC diameter were analysed. Among these factors, IVC diameter was the most powerful predictor for the development of WRF (area under the ROC curve = 0.795, cut-off value = 20.5 mm, 79 % sensitivity, 82 % specificity) (Fig. 1).

Clinical events and event-free survival

In Cox regression model, the risk of combined end-points including death and re-hospitalization for HF was increased in patients with aging, elevated BUN, IVC >21 mm, and WRF. When adjusted for age, BUN, and BNP, IVC >21 mm (HR 3.73, 95 % CI 1.66–8.34) and WRF (HR 2.68, 95 % CI 1.07–6.75) were significant predictors for adverse events (Table 4). Figure 2 shows the

Table 2 Echocardiographic characteristics

	Total (n = 74)	WRF (n = 34)	No WRF (n = 40)	*P* value
LV EF (%)	30.5 ± 7.6	28.0 ± 6.6	32.6 ± 7.9	0.01
LVEDV (ml)	167.7 ± 54.3	177.7 ± 58.1	159.3 ± 50.0	0.15
LVESV (ml)	108.2 ± 49.5	123.6 ± 95.1	95.1 ± 44.6	0.01
LA diameter (mm)	44.0 ± 8.0	45.9 ± 8.0	42.4 ± 7.7	0.06
RA pressure (mmHg)	9.2 ± 5.2	12.0 ± 4 .6	6.6 ± 4.3	<0.01
TR PG (mmHg)	30.8 ± 13.6	36.9 ± 12.4	25.0 ± 12.3	<0.01
PASP (mmHg)	40.6 ± 16.1	49.2 ± 12.2	32.0 ± 14.9	<0.01
IVC diameter (mm)	19.2 ± 4.4	21.7 ± 3.6	16.9 ± 3.7	<0.01
IVC > 21 mm [n (%)]	32 (43)	26 (76)	6 (15)	<0.01
TR ≥ moderate [n (%)]	22 (30)	16 (47)	6 (15)	<0.01

Values are expressed as mean ± standard deviation or number (percentage)

LV left ventricular, *EF* ejection fraction, *LVEDV* LV end-diastolic volume, *LVESV* LV end-systolic volume, *LA* left atrial, *RA* right atrial, *TR PG* tricuspid regurgitant pressure gradient, *PASP* pulmonary artery systolic pressure, *IVC* inferior vena cava

Table 3 Logistic-regression analysis for prediction of WRF

	Univariate analysis		Multivariate analysis (model 1)		Multivariate analysis (model 2)	
	OR	*P* value	OR (95 % CI)	*P* value	OR (95 % CI)	*P* value
BUN	1.09	0.01			1.10 (1.03–1.18)	<0.01
BNP[a]	2.16	0.02	2.88 (1.18–7.05)	0.02		
LV ejection fraction	1.09	0.01	1.00 (0.91–1.10)	1.00		
LVESV	1.01	0.02			1.01 (0.99–1.03)	0.15
TR PG	1.08	<0.01			1.08 (1.01–1.14)	0.02
RA pressure	1.26	<0.01	1.06 (0.85–1.31)	0.63		
IVC diameter	1.37	<0.01	1.33 (1.01–1.77)	0.05	1.44 (1.17–1.76)	<0.01

CI confidence interval, *OR* odds ratio, *BUN* blood urine nitrogen, *BNP* brain natriuretic peptide, *LV* left ventricular, *LVESV* LV end-systolic volume, *TR PG* tricuspid regurgitant pressure gradient, *RA* right atrial, *IVC* inferior vena cava

[a] A log transformation was done for the variable

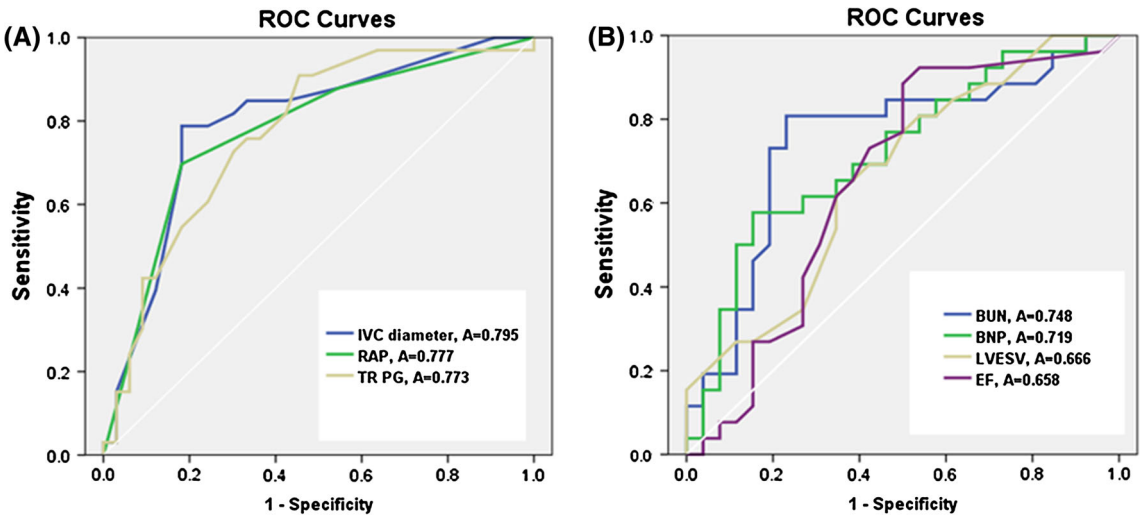

Fig. 1 Receiver operating characteristic curve analysis for the prediction of WRF including: **a** IVC diameter, right atrial pressure (RAP), and tricuspid regurgitant pressure gradient (TR PG) and **b** BUN, BNP, LVESV, and EF. A, area under the ROC curve. *All parameters had *P* values <0.05, except for EF (*P* = 0.05)

Table 4 Cox-regression analyses for predicting combined end-points (HF hospitalization and mortality) during 2-year follow-up

	Univariate analysis		Multivariate analysis (model 1)		Multivariate analysis (model 2)	
	HR	*P* value	HR (95 % CI)	*P* value	HR (95 % CI)	*P* value
Age	1.03	0.01	1.03 (1.01–1.06)	0.02	1.03 (1.00–1.06)	0.04
Male	0.69	0.29				
Ischemic HF	1.04	0.90				
LV ejection fraction	1.01	0.49				
BUN	1.03	0.02	1.02 (0.99–1.05)	0.25	1.01 (0.98–1.04)	0.64
TR PG	1.02	0.16				
BNP[a]	1.35	0.10	1.10 (0.70–1.75)	0.67	1.01 (0.66–1.55)	0.95
TR at least moderate	1.65	0.14				
IVC >21 mm	3.10	<0.01	3.73 (1.66–8.34)	<0.01		
WRF	2.98	<0.01			2.68 (1.07–6.75)	0.04

HR hazard ratio, *CI* confidence interval, *HF* heart failure, *LV* left ventricular, *BUN* blood urine nitrogen, *TR PG* tricuspid regurgitant pressure gradient, *BNP* brain natriuretic peptide, *TR* tricuspid regurgitation, *IVC* inferior vena cava, *WRF* worsening renal function

[a] A log transformation was done for the variable

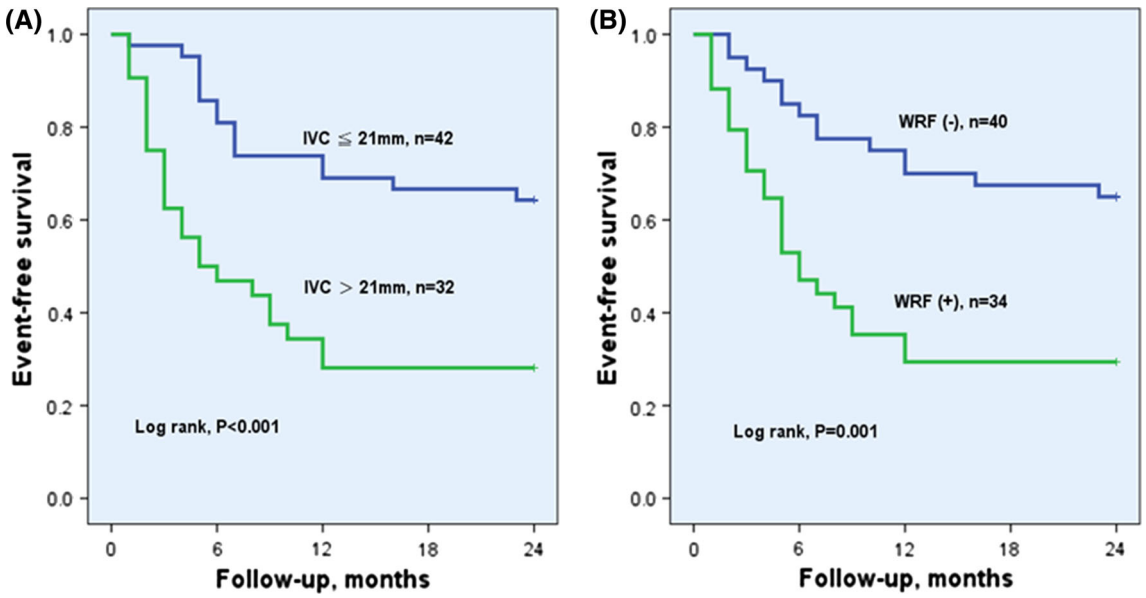

Fig. 2 Kaplan–Meier analyses for event-free survival in patients **a** with or without IVC dilatation, **b** with or without WRF

Kaplan–Meier event-free survival curves for patients with or without IVC dilatation and WRF.

Discussion

Our study shows that in patients with advanced decompensated HF, WRF during hospitalization is common and is associated with adverse outcomes. Dilated IVC identifies patients who may develop WRF during hospitalization, and is associated with increased risk of adverse events during 2-year follow up.

The mechanisms responsible for WRF in patients with HF are complex and not well-defined. Hemodynamic abnormalities, such as hypotension or low cardiac output, might be expected to play an important role. In addition, there is increasing evidence to support the role of systemic venous congestion in the development of WRF in patients with advanced decompensated HF [3]. In patients with advanced HF, LV systolic dysfunction causes increased left atrial pressure. The pressure is transmitted back through the pulmonary circulation to cause pulmonary arterial hypertension. The pulmonary artery hypertension can worsen pre-existing right ventricular dysfunction and exacerbate

TR, leading to systemic venous congestion [15]. If venous congestion and elevated central venous pressure are the hallmarks of HF, then distention of the IVC by echocardiography may be a good prognostic marker in patients with decompensated HF.

Increased BUN and natriuretic peptide have been associated with WRF and poor outcome in patients hospitalized for HF [16–20]. Increased urea reabsorption by proximal tubules or collecting ducts as a result of angiotensin II or vasopressin increase has been showed in HF patients with worsening symptoms and increased central venous pressure [4, 21–23]. Plasma BNP rises in various pathologic states, particular where there is increased cardiac wall stretch, an expanded fluid volume [24, 25] or reduced clearance [26]. Compatible with previous studies, the present study demonstrated that HF patients with WRF had higher BUN and BNP levels at hospital admission than those without WRF. The association between dilated IVC and WRF remains significant after adjustment for BUN, BNP or TR PG. These results implicate that HF patients with WRF had marked venous congestion. Actually, venous congestion had been demonstrated to be correlated with impaired renal function [6, 27–30]. Our findings confirm and support the venous congestion might be an important determinant of WRF in patients with HF. Moreover, our findings also contribute to the growing evidence that dilated IVC could be a marker of adverse outcomes in patients with decompensated HF [31]. The present study also indicated WRF was a significant prognostic factor for adverse events compatible with the results of a large body of literature [32].

Noninvasive measurement of the diameter of the IVC and the change in diameter with respiration by echocardiography has demonstrated fair to excellent correlation with RAP [33–38]. Instead of invasive nature of catheterization and complications such as pneumothorax, air embolism, or injury of great vessels [39, 40], when echocardiography is available, IVC diameter might provide similar information in HF patients. In addition, a rapid assessment of IVC physiology could be performed at the bedside by a non-cardiologist in the emergency department [41, 42].

Apart from the intrinsic limitations associated with a retrospective study and thereby possibly subject to selection bias, the other limitations were this work being a single-centre study and a small sample size. However, we applied rigorous inclusion criteria to ensure that our model is as valid as possible. Although we utilized IVC diameter >21 mm to define IVC dilatation according to the literature [7], the cut-off value of IVC diameter derived from the ROC analysis for predicting WRF was close to the guideline criteria for a dilated IVC. We also had attempt to avoid the influence of the use of medications influencing renal function; therefore, to limit the timing of echocardiography within 24 h, but we did not know how far the influence we could really avoid. In this study, we did not exclude patients with atrial fibrillation, prior cardiac surgery or nitrate use that may potentially affect the IVC diameter. However, they are frequently observed in patients with HF and the inclusion of these patients yields a realistic view of what is observed in daily clinical practice. Additional studies with the exclusion of these confounding factors could provide more specific information in this regard. Although we assessed EF and systolic blood pressure in our study, blood flow to the kidneys could not be assessed directly, and thus we cannot exclude reduced renal blood flow as an additional major contributor to impaired renal function.

Conclusions

In this cohort study, WRF was commonly observed and was associated with adverse outcomes. Dilated IVC predicted the development of WRF and provides similar prognostic information as WRF.

Conflict of interest None.

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