Original Article

Combined use of cardiopulmonary ultrasound in the diagnosis of pulmonary edema in patients with heart failure: a retrospective analysis

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Background: Heart failure (HF) is generally complicated with pulmonary edema (PE), the early diagnosis and treatment is essential. We aimed to evaluate the effects of combined use of cardiopulmonary ultrasound in the diagnosis of PE in HF patients, to provide reference for the management of HF.

Methods: HF patients treated in our hospital from January 1, 2019 to June 30, 2020 were included. All patients underwent echocardiography and lung ultrasonography, and analyzed the characteristics of patients and related detected results. Logistic regression analyses were conducted to identify the potential risk factors. And the receiver operating characteristic (ROC) curve was conducted to compare the predictive value of factors.

Results: A total of 183 HF patients were included, the incidence of PE in HF patients was 62.84%. Logistic regression analyses indicated that NT-proBNP (OR 2.24, 95% CI: 1.28–5.04), LAVI (OR 2.03, 95% CI: 1.02–4.45), E/e' (OR 1.57, 95% CI: 0.13–2.28), SPAP (OR 1.35, 95% CI: 0.02–2.84) were the independent risk factors for PE in patients with HF (all P<0.05). The AUC of NT-proBNP, LAVI, E/e' and SPAP were 0.705, 0.668, 0.674 and 0.691 respectively. NT-proBNP ≥8,842.37 ng/L, LAVI ≥42.14 mL/m^2, E/e' ≥19.20, SPAP ≥38.16 mmHg were the independent risk factors for PE in patients with HF (all P<0.05).

Conclusions: Combined use of cardiopulmonary ultrasound is beneficial to the early diagnosis of PE in patients with HF, and early interventions are needed for those patients with risk factors.

Keywords: Cardiopulmonary; ultrasound; pulmonary edema (PE); heart failure (HF); risk; treatment

Submitted Aug 21, 2020. Accepted for publication Sep 20, 2020.
doi: 10.21037/cdt-20-733
View this article at: http://dx.doi.org/10.21037/cdt-20-733

Introduction

Heart failure (HF) patients have high mortality and poor prognosis, and it has now become the leading cause of death in patients with cardiovascular disease (1). According to the survey (2), the current number of HF patients worldwide has reached about 24.5 million, and it is still increasing at a rate of over 2 million per year. It's been reported that the prevalence of HF in adult patients is about 0.9%, and the mortality of HF among hospitalized patients is about 8.9% (3). Clinically, the onset of HF is rapid and serious, and the symptoms and signs are often atypical. Therefore, the early diagnosis of HF is still difficult (4,5). It's necessary to identify the effective and convenient methods to promote the early diagnosis of HF.

Pulmonary edema (PE) refers to the imbalance of the production and return of tissue fluid in the lung due to some reasons, so that a large amount of tissue fluid cannot be absorbed by the pulmonary lymph and pulmonary

venous system in a short period of time. It leaks from the pulmonary capillaries and accumulates in the alveoli and lung interstitium, causing serious obstacles to lung ventilation and ventilation. Clinically, it is characterized by extreme dyspnea, sitting breathing, cyanosis, profuse sweating, paroxysmal cough accompanied by a large amount of white or pink foamy sputum, and the lungs are full of symmetrical wet rales. As an important sign of HF, PE has long been mainly dependent on chest X-ray or CT examination (6). Although bedside chest X-ray is more convenient, it also has its limitations. In addition to radiation, it is also affected by organs and tissues in the shadow of the mediastinum, such as the esophagus, trachea, and lymph nodes etc. (7). However, chest CT radiation is larger, which limits repeated use. Therefore, we still need to find new inspection methods to improve the sensitivity and specificity of the diagnosis of HF, and to determine the severity of the disease (8). With the continuous deepening of clinical research, the value of bedside cardiopulmonary ultrasound in detecting different lung diseases and pleural lesions has been reassessed. It has advantages of feasible bedside, non-radioactive, reusable and low cost. Therefore, we aimed to use cardiopulmonary ultrasound to compare the differences in clinical and echocardiographic parameters between HF patients with and without PE, and to evaluate the relationship between the patient's clinical manifestations and echocardiographic parameters, thereby providing evidence for the management of HF patients. We present the following article in accordance with the STARD reporting checklist (available at http://dx.doi.org/10.21037/cdt-20-733).

Methods

Ethical considerations

Our study is a retrospective study. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the ethics committee of our hospital (No. 2018026) and informed consent was taken from all the patients.

Patients

HF patients treated in our hospital from January 1, 2019 to June 30, 2020 were included. The inclusion criteria were as follows: (I) adult patients with age ≥18 years; (II) the diagnosis of HF was complied with related guidelines (9,10); (III) patients were informed and agreed to participants in this study. Patients were excluded if they were: (I) aged <18 years; (II) patients with atrial fibrillation, mitral stenosis and lung cancers; (III) patients who disagreed to participants in this study.

Ultrasound detection

The instrument used in this present study was Philips DX 150 ultrasonic diagnostic apparatus, the frequency of L5-1 phased array probe was 1–5 MHz, and the frequency of L12-3 linear array probe is 3–12 MHz.

Echocardiography was performed as follows: all patients were in a supine, semi-recumbent or lateral position. Echocardiographic measurement standards are based on the Heart Cavity Quantitative Guidelines M of the American Society of Echocardiography and the European Association of Cardiovascular Imaging (ASE/EACVE). The long axis view of the left ventricle was measured under M-mode echocardiography to measure left ventricular end-diastolic diameters (LVEDD) and left ventricular end-systolic diameters (LVESD). The apical two-chamber and four-chamber biplane Simpson method was used to measure left ventricular end-diastolic volumes (LVEDV), left ventricular end-systolic volumes (LVESV), and left ventricular evacuation. Left ventricular ejection fraction (LVEF) and left atrial volume (LAV), and normalized with body surface area to obtain left atrial volume index (LAVI). The apical four-chamber view with the right ventricle as the center was used to measure the diameter of right ventricle (RVD). The apical four-chamber view of the mitral valve measured the early diastolic flow velocity E peak and late diastolic flow velocity A peak. We placed a sample volume of 1.5 mm on the septal and lateral sides of the mitral annulus, and measured the early diastolic e', and calculated the average n/e'. The degree of mitral regurgitation was semi-quantitative with color Doppler, and the regurgitation was divided into mild, moderate and severe. The systolic pulmonary artery pressure (SPAP) was estimated by the tricuspid regurgitation pressure difference plus the right atrial pressure. The right atrial pressure was estimated based on the inner diameter of the inferior vena cava and the rate of inspiratory collapse. In addition, we classified the left ventricular diastolic function according to the 2016 updated ASE/EACVE left ventricular diastolic function guidelines (9,11).

The steps of lung ultrasound detection were as follows: the patient took the supine or semi-recumbent position, and the left and right scan ranges included the anterior and

lateral chest walls, that is, from the parasternal line to the posterior axillary line. And the probe was scanned along the intercostal space and perpendicular to the chest wall during scanning. In this study, the diagnosis of cardiogenic PE by pulmonary ultrasound was based on the results of previous studies, which showed bilateral multiple B-lines (spacing ≤7 mm) combined with smooth pleural lines.

Data collection

All patients underwent echocardiography and lung ultrasonography. The related ultrasonography parameters mentioned above were taken as the primary outcomes. Furthermore, we collected and recorded the patient's characteristics, including weight, age and other related information. And we collected the level of N-terminal pro-brain natriuretic peptide (NT-proBNP) and NYHA cardiac function classification.

Statistical analysis

We used SPSS 22.0 software for statistical analysis. Continuous data were expressed as mean ± standard deviation, and count data were expressed as percentage. Comparisons between groups were conducted with student *t*-test or chi-square test. Logistic regression analyses were conducted to identify the potential risk factors. According to the receiver operating characteristic (ROC) curve, the thresholds for predicting the occurrence of SHS were determined. And the area under the ROC curve (AUC) was used to compare the predictive value of factors, and to evaluate its sensitivity and specificity, positive and negative predictive values. We used the point with the largest Youden index for cutoff value calculation. In this present study, P<0.05 was considered statistically significant.

Results

The characteristics of included patients

A total of 183 HF patients were included, of which 115 patients were diagnosed as PE, the incidence of PE in HF patients was 62.84%. As presented in *Table 1*, there were no significant difference in the gender and age between two groups (all P>0.05), and there were significant differences in the classification of left ventricular diastolic function, NYHA classification, NT-proBNP, LVEDD, LVEDV, LVEF, LAVI, E/e', RVD and SPAP (all P<0.05).

The risk factors of PE in patients with HF

The significant variables in univariate analyses were further included for logistic regression analyses. As presented in *Table 2*, the logistic regression analyses indicated that NT-proBNP (OR 2.24, 95% CI: 1.28–5.04), LAVI (OR 2.03, 95% CI: 1.02–4.45), E/e' (OR 1.57, 95% CI: 0.13–2.28), SPAP (OR 1.35, 95% CI: 0.02–2.84) were the independent risk factors for PE in patients with HF (all P<0.05).

The predictive value of NT-proBNP, LAVI, E/e' and SPAP for the PE

Figure 1 presented the ROC of NT-proBNP, LAVI, E/e' and SPAP for the PE. As indicated in *Table 3*, the AUC of NT-proBNP, LAVI, E/e' and SPAP were 0.705, 0.668, 0.674 and 0.691 respectively. NT-proBNP ≥8,842.37 ng/L, LAVI ≥42.14 mL/m^2, E/e' ≥19.20, SPAP ≥38.16 mmHg were the independent risk factors for PE in patients with HF (all P<0.05).

Discussion

HF is a common clinical disease, which is generally complicated combined with pulmonary congestion (12). HF is the main syndrome of organic cardiovascular disease caused by different causes, and it is a common clinical critical illness (13). According to reports, the incidence of HF patients shows a significant increase with age (14,15). Despite the continuous improvement of its treatment methods, the annual mortality of patients with HF is still as high as 8.9%, and the 5-year mortality is as high as 50%, even exceeding the mortality of many malignant tumors (16). Therefore, how to diagnose HF more conveniently and timely is of great significance to the early prevention and treatment of HF. The results of this study have indicated that NT-proBNP ≥8,842.37 ng/L, LAVI ≥42.14 mL/m^2, E/e' ≥19.20, SPAP ≥38.16 mmHg were the independent risk factors for PE in patients with HF, which can provide significant implications for clinical treatment of HF.

Previous studies (17-19) have found that HF patients with PE have lower LVEF, more severe diastolic dysfunction, larger left and right ventricles, and higher SPAP, which is consistent with our findings. When the patient has left ventricular systolic and/or diastolic dysfunction, the left ventricular end diastolic pressure and left atrial pressure increase accordingly, which cause the

Table 1 The characteristics of included patients

Variables	No-PE group (n=68)	PE group (n=115)	t/χ^2	P
Male	49 (72.06%)	90 (78.26%)	1.346	0.087
Age (years)	64.42±9.33	65.01±10.28	15.301	0.061
Classification of left ventricular diastolic function			1.212	0.015
I level	43 (63.24%)	18 (15.65%)		
II level	19 (27.94%)	57 (49.57%)		
III level	6 (8.82%)	40 (34.78%)		
NYHA classification			1.168	0.008
I level	20 (29.41%)	0 (0%)		
II level	45 (66.18%)	37 (32.17%)		
III level	3 (4.41%)	74 (64.35%)		
IV level	0 (0%)	4 (3.48 %)		
NT-proBNP (ng/L)	1,190.47±395.08	9,822.16±895.41	84.104	0.001
LVEDD (mm)	58.24±12.10	62.17±13.45	17.299	0.036
LVEDV (mL)	152.37±44.03	169.54±53.14	37.469	0.063
LVEF (%)	38.40±11.28	34.01±10.12	10.664	0.033
LAVI (mL/m^2)	39.16±10.24	47.35±11.42	9.179	0.012
E/e’	15.29±3.05	22.16±4.65	3.268	0.017
RVD (mm)	31.36±4.41	37.74±4.97	5.177	0.042
SPAP (mmHg)	30.18±3.24	45.91±4.14	3.075	0.015

LVEDD, left ventricular end-diastolic diameters; LVEDV, left ventricular end-diastolic volumes; LVEF, left ventricular ejection fraction; LAVI, left atrial volume index; RVD, diameter of right ventricle; SPAP, systolic pulmonary artery pressure.

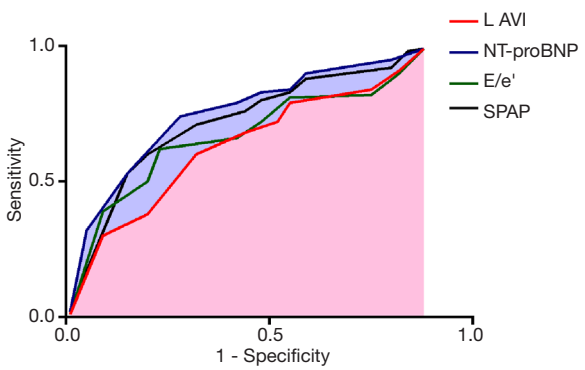

Figure 1 The ROC of NT-proBNP, LAVI, E/e’ and SPAP for the PE. ROC, receiver operating characteristic; LAVI, left atrial volume index; SPAP, systolic pulmonary artery pressure; PE, pulmonary edema.

Table 2 Logistic regression analysis on the risk factors of PE

Variables	β	SE	OR	95% CI	P
NT-proBNP	0.85	0.13	2.24	1.28–5.04	0.013
LAVI	0.94	0.25	2.03	1.02–4.45	0.021
E/e’	0.74	0.41	1.57	0.13–2.28	0.042
SPAP	0.92	0.32	1.35	0.02–2.84	0.030

PE, pulmonary edema; LAVI, left atrial volume index; SPAP, systolic pulmonary artery pressure.

Table 3 The predictive value of NT-proBNP, LAVI, E/e' and SPAP for the PE

Variables	Cutoff value	AUC	95% CI	Sensitivity (%)	Specificity (%)
NT-proBNP (ng/L)	8,842.37	0.705	0.628–0.784	80.24	83.18
LAVI (mL/m^2)	42.14	0.668	0.602–0.787	73.52	82.41
E/e'	19.20	0.674	0.524–0.753	72.45	81.22
SPAP (mmHg)	38.16	0.691	0.604–0.779	79.18	80.13

LAVI, left atrial volume index; SPAP, systolic pulmonary artery pressure; PE, pulmonary edema.

left heart to increase, and the pulmonary venous pressure and pulmonary capillary hydrostatic pressure increase accordingly (20). EVLW is composed of intracellular fluid, alveolar fluid and pulmonary interstitial fluid. It is a fluid distributed outside the pulmonary blood vessels (21). In general, the intracellular fluid is relatively fixed, and the alveolar fluid and interstitial fluid can change, reflecting the degree of PE (22,23). The B-line in the lung ultrasound signs is a reverberation artifact, derived from the edema thickened subpleural lobular septum and the alveoli with increased water content (16,24). The change in the number of B-line can reflect the change of EVLW, so lung ultrasound can be used to diagnose PE and monitor the effect of HF treatment.

The significant increase in pulmonary artery pressure and pulmonary capillary wedge pressure (PCWP) can be seen in patients with HF, it will cause changes in the ultrastructure of the pulmonary capillary wall, resulting in an increase in extravascular lung water (25). Through the quantitative monitoring and dynamic observation of extravascular pulmonary water, not only can the development and severity of PE be evaluated, but also the fluid therapy can be guided (26,27). This is useful for understanding the pathophysiological changes and pulmonary function of cardiopulmonary diseases (14). A study (28) evaluated the left heart function of patients admitted to the cardiovascular department through the application of echocardiography and pulmonary ultrasound, the results have showed that the number of B-lines is related to the patients' New York heart function classification and left ventricular radiography, and LVEF is closely related to the degree of diastolic insufficiency. Besides, it's been reported that lung ultrasound examination can have a better predictive effect on the right heart pressure and PVR of patients with HF (29). In addition, more and more studies (30-32) have shown that the index value that can clearly represent the decompensation of heart function in patients with HF is closely related to the B-line. Several studies (33,34) have found that lung ultrasound can detect PE and accurately detect changes in the amount of fluid in lung tissue before the early clinical symptoms of HF patients appear. Compared with traditional imaging techniques for displaying lungs such as chest X-ray or lung CT, pulmonary ultrasound needs to collect images of multiple parts of the lung, and the results of the examination need to be re-integrated (35). Therefore, it is necessary to select standardized operating procedures and make effective evaluations when performing pulmonary ultrasound examinations.

This study has certain limitations. Firstly, this study is a single-center study. Patients with atrial fibrillation and mitral stenosis were excluded from the study, but PE is not common among such patients (36). Secondly, due to the poor specificity of the B-line, it can only indicate the presence of pulmonary interstitial syndrome, but it cannot clearly show that the specific causes of PE. Therefore, it is necessary to combine specific etiology and medical history, patient clinical manifestations, and echocardiography in clinical application.

Conclusions

In conclusion, the results of this present study have indicated that the combined use of cardiopulmonary ultrasound is feasible and effective in the diagnosis of PE in patients with HF, which should be promoted in clinical settings. Furthermore, we have found that NT-proBNP ≥8,842.37 ng/L, LAVI ≥42.14 mL/m^2, E/e' ≥19.20, SPAP ≥38.16 mmHg were the independent risk factors for PE in patients with HF (all $P<0.05$), targeted strategies are needed for those patients.

Acknowledgments

Funding: None.

Footnote

Reporting Checklist: The authors have completed the STARD reporting checklist. Available at http://dx.doi.org/10.21037/cdt-20-733

Data Sharing Statement: Available at http://dx.doi.org/10.21037/cdt-20-733

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at http://dx.doi.org/10.21037/cdt-20-733). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). This present study had been verified and approved by the ethics committee of our hospital (No. 2018026), and written informed consents had been obtained from all included patients.

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Cite this article as: Shen M, Chen H, Cong Y. Combined use of cardiopulmonary ultrasound in the diagnosis of pulmonary edema in patients with heart failure: a retrospective analysis. Cardiovasc Diagn Ther 2020;10(5):1325-1331. doi: 10.21037/cdt-20-733

