Effect of transthoracic echocardiography on short-term outcomes in patients with acute kidney injury in the intensive care unit: a retrospective cohort study based on the MIMIC-III database

Caijie Liu[1], Shuying Wang[2], Xiuzhen Wang[3]

[1]Department of Ultrasound, The First Affiliated Hospital of Jinzhou Medical University, Jinzhou, China; [2]Department of Blood Transfusion, First Medical Center, PLA General Hospital, Beijing, China; [3]Department of Rheumatology and Immunology, The First Affiliated Hospital of Jinzhou Medical University, Jinzhou, China

Contributions: (I) Conception and design: C Liu; (II) Administrative support: S Wang; (III) Provision of study materials or patients: C Liu; (IV) Collection and assembly of data: C Liu, S Wang; (V) Data analysis and interpretation: All authors; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Xiuzhen Wang. Department of Rheumatology and Immunology, The First Affiliated Hospital of Jinzhou Medical University, Jinzhou, China. Email: 19223002@qq.com.

Background: Acute kidney injury (AKI) is a common problem in the intensive care unit (ICU). Transthoracic echocardiography (TTE) has become a common test in the ICU. But the effect of TTE on the short-term prognosis in ICU patients with AKI remains unclear. We aimed to analyze the relationship between TTE and short-term prognosis in ICU patients with AKI.

Methods: AKI patients in the Medical Information Mart for Intensive Care (MIMIC)-III database were retrospectively enrolled according to inclusion and exclusion criteria, and their clinical information was extracted. TTE was conducted during ICU stay. AKI was diagnosed according to KIDGO criteria. The primary endpoint was the outcome of 28-day ICU stay. The doubly robust estimation method was used to analyze the association of TTE with the primary and secondary outcomes of patients with AKI in ICU. The gradient boosted model (GBM) was used to estimate the propensity score of patients to undergo TTE examination, thereby minimizing the variable imbalance between the TTE and non-TTE groups. Weighted cohorts were built using the inverse probabilities weighting (IPW) model with estimated propensity scores as weights. The weighted cohort was analyzed using logistic regression and validated using other models.

Results: A total of 2,983 patients were included. In the original cohort, 28-day mortality was 37.9% in the TTE group (n=1,684) and 40.8% in the non-TTE group (n=1,299). In the propensity score model (PSM) cohort, 28-day mortality was 34.6% in the TTE group (n=702) and 45.6% in the non-TTE group (n=702). Doubly robust analysis showed that TTE was associated with lower 28-day mortality.

Conclusions: TTE examination might decrease the 28-day mortality in patients with AKI in the ICU and should be considered for critical patients when necessary.

Keywords: Acute kidney injury (AKI); death; intensive care unit (ICU); transthoracic echocardiography (TTE)

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Introduction

Acute kidney injury (AKI) is a common problem in clinical practice, defined as a rapid decline in kidney function over a short period of time due to a variety of causes. Studies have shown that AKI occurs in approximately 13.3 million patients worldwide each year, resulting in approximately 1.7 million deaths each year (1). Moreover, 85% of AKI patients are from developing countries (1). The incidence of AKI in the general hospitalized population is 10–15%,

and the incidence in the intensive care unit (ICU) can reach 30–50% (2-5). AKI increases mortality in hospitalized patients. A previous study found that among patients with septic shock, the 60-day mortality rate was 3–5-fold higher in those who developed AKI than in those who did not (6). Other studies have found that many factors are associated with the prognosis of patients with AKI, including urine output, comorbidities, and multiple biomarkers, such as urine interleukin-18 concentration, neutrophil gelatinase-associated lipocalin, tissue inhibitor of metalloproteinase-2 (TIMP2), insulin-like growth factor (IGF)-binding protein-7 (IGFBP7) and microRNA-210, 21, etc. (7-9). In order to have a full and timely understanding of the condition of critically ill patients, doctors often perform various examinations or tests to detect some potential problems or changes in the patient's condition over time, and this plays an important role in optimizing the treatment plan. However, many examinations and tests are overused, which does not benefit patients. Therefore, assessing the benefit of an examination or test to the patient has important clinical implications.

Ultrasound has become a common test in ICUs (10). Since heart is a common organ influenced by critical illness, transthoracic echocardiography (TTE) is often conducted to evaluate its changes of structure and function. In a previous study, Feng *et al.* conducted a retrospective analysis of the Medical Information Mart for Intensive Care (MIMIC) database and found that TTE was associated with a reduction in 28-day mortality in patients with sepsis (11). Therefore, the purpose of this study was to retrospectively analyze the relationship between TTE examination and the short-term prognosis of AKI patients using data from the MIMIC database. We present the following article in accordance with the STROBE reporting checklist (12) (available at https://atm.amegroups.com/article/view/10.21037/atm-22-3158/rc).

Methods

Study population

This study was a retrospective cohort study. Cases were obtained from the MIMIC-III, v1.4 database (13), which was developed and maintained by the Laboratory for Computational Physiology at Massachusetts Institution of Technology. This study only used the data of each AKI patient when they were first admitted to the ICU, and divided them into a TTE and non-TTE group according to whether or not TTE was performed during the first ICU admission. Inclusion criteria were: (I) age ≥18 years; (II) first admission to ICU; (III) TTE completed within 24 h of admission to ICU; (IV) complete clinical information (i.e., 28-day outcome). Exclusion criteria were: (I) repeat admission to ICU; (II) TTE completed before admission to ICU; (III) incomplete information. AKI was diagnosed according to KIDGO criteria (14). The study was conducted in accordance with the Declaration of Helsinki (as revised in 2013).

Data extraction

MIMIC-III contains the clinical data of 38,605 patients admitted to the ICU of Beth Israel Deaconess Medical Center from 2002 to 2011. In this study, Bigquery software (Google) was used to extract the following data from the MIMIC-III v1.4 database using SQL language: sex, age, weight, white blood cell count, hemoglobin, platelet count, blood urea nitrogen, blood creatinine, blood sugar, blood electrolytes (K^+, Na^+, Ca^{2+}), blood HCO^{3-}, sequential organ failure assessment (SOFA) score, ICU length of stay and death in ICU, etc., and comorbidities [hypertension, coronary artery disease (CAD), chronic obstructive pulmonary disease (COPD), chronic nephropathy] and whether mechanical ventilation, vasoactive drugs and renal replacement therapy were used during ICU stay, and whether there were complications such as ventilator-associated pneumonia, urinary tract infection, diabetic ketoacidosis, and acute myocardial infarction. All laboratory parameters were extracted from the data generated within the first 24 h after the patient was admitted to the ICU (i.e., baseline values) and the extreme values [i.e., maximum (max) and minimum (min)] during ICU stay.

Study outcomes

The primary outcome of this study was all-cause death 28 days from ICU admission. Secondary outcomes were mechanical ventilation-free days within the 28 days, vasopressor-free days within the 28 days, dopamine use, infusion volume during the first 3 days of ICU admission, and changes in SOFA score on the second and third days after ICU admission, maximum dose of norepinephrine, changes in serum creatinine at 48 h after admission to ICU, changes in serum lactate and creatinine at 24 h after admission in ICU.

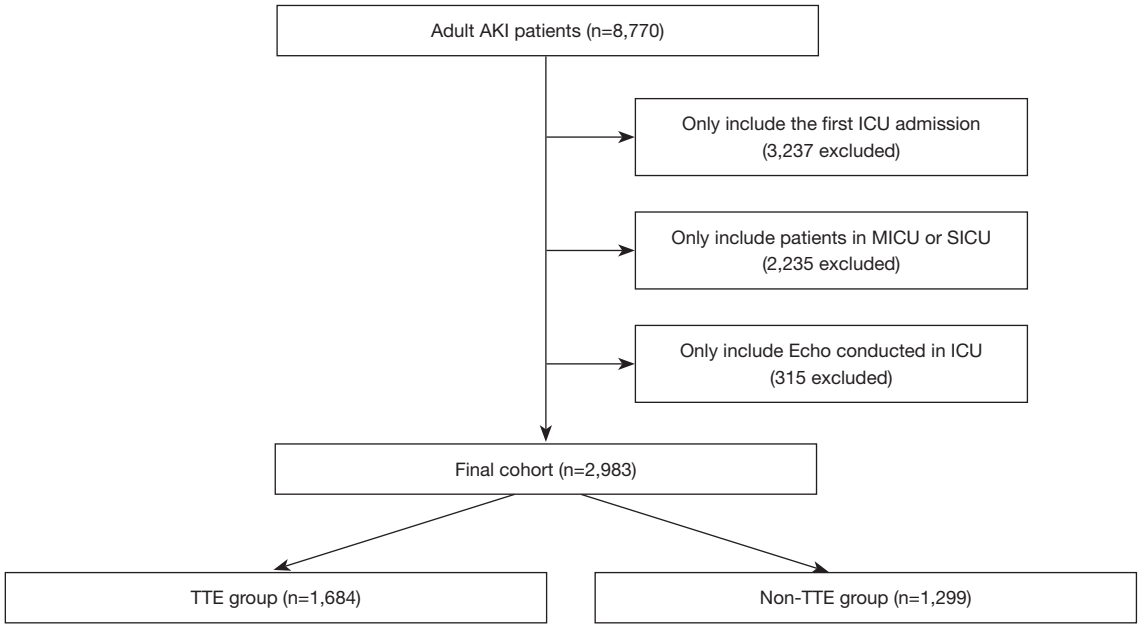

Figure 1 Flowchart of patients' inclusion in the study. AKI, acute kidney injury; ICU, intensive care unit; MICU, medical intensive care unit; SICU, surgical intensive care unit; TTE, transthoracic echocardiography.

Statistical analysis

R 3.6 statistical software was used to analyze the data. Continuous variables are expressed as mean ± standard deviation (SD), and independent samples *t*-test was used for comparison between two groups. Categorical variables are expressed as percentages (%), and comparisons between groups were performed using the chi-squared test. Referring to the study of Feng *et al.*, we also used the doubly robust estimation method to analyze the association of TTE with the primary and secondary outcomes of AKI patients in the ICU. This method combines multiple regression models with propensity score models (PSMs) to analyze the effect of exposure factors on outcomes (11). The gradient boosted model (GBM) model was used to estimate the propensity score of patients to undergo TTE examination, thereby minimizing the variable imbalance between the TTE and non-TTE groups. Weighted cohorts were built using the inverse probabilities weighting (IPW) model with estimated propensity scores as weights (15). This weighted cohort was then analyzed using logistic regression and adjusted for variables that remained unbalanced between the two groups in a PSM, resulting in a doubly robust analysis. To assess the role of the PSM in balancing the two groups of variables, we calculated standardized the mean differences (SMDs) for variables between the TTE and non-TTE groups. The Wilcoxon signed rank test was used to test the differences between continuous variables, and the χ^2 test was used to evaluate the differences between categorical variables. For sensitivity analysis, we used four models, comprising a doubly robust model adjusted for all variables, an IPW model based on the propensity score, a case-paired model based on the propensity score, and a multivariate analysis model based on logistic regression. P<0.05 was consider statistically significant.

Results

General information

According to the AKI diagnostic criteria, there were 8,770 AKI patients in the MIMIC-III v1.4 database, and according to our inclusion and exclusion criteria, 2,983 patients were finally included in the analysis (*Figure 1*): 1,684 patients (56.5%) in the TTE group and 1,299 (43.5%) in the non-TTE group. The clinical information of the two groups of patients is shown in *Table 1*. Patients in the TTE group had higher simplified acute physiology score (SAPS), SOFA scores, and Elixhauser scores, and higher rates of patients receiving mechanical ventilation, vasopressors, and sedatives. The ratios of patients with heart failure, atrial fibrillation (AFIB), COPD or CAD in the TTE group were

Table 1 Comparison of the basic demographics, comorbidity conditions, and day of ICU admissions between the original and matched (weighted) cohorts

Covariate	Original cohort (n=2,983)			Matched cohort (n=1,404)			Missing data (%)
	Non-TTE	TTE	SMD	Non-TTE	TTE	SMD	
N	1,299	1,684		702	702		
Age (years), mean (SD)	66.34 (16.29)	66.07 (16.17)	0.017	66.83 (16.22)	65.65 (16.51)	0.072	0.00
Sex (female, %)	51.30	48.60	0.054	49.00	49.70	0.014	0.00
Service unit (MICU *vs.* SICU, %)	70.40	76.70	0.141#	73.10	75.50	0.055	0.00
Weight (kg), mean (SD)	88.83 (39.42)	88.87 (30.98)	0.001	86.96 (31.62)	87.57 (29.87)	0.020	8.10
SAPS score, mean (SD)	21.34 (6.12)	22.28 (5.43)	0.162#	22.22 (5.84)	20.94 (5.55)	0.225#	0.00
SOFA score, mean (SD)	6.58 (4.09)	7.54 (4.03)	0.237#	7.26 (4.16)	6.50 (3.68)	0.195#	0.00
Elixhauser score, mean (SD)	11.05 (8.15)	13.76 (8.33)	0.329#	12.38 (8.00)	11.60 (8.08)	0.097	0.00
Mechanical ventilation use (1st 24 h, %)	56.90	73.80	0.360#	68.20	58.00	0.214#	0.00
Vasopressor use (1st 24 h, %)	28.20	40.20	0.256#	34.50	27.20	0.158#	0.00
Sedative use (1st 24 h, %)	42.50	52.00	0.190#	50.60	40.20	0.210#	0.00
CHF (%)	20.00	42.90	0.508#	30.80	23.10	0.174#	0.00
AFIB (%)	22.60	36.90	0.317#	32.50	27.50	0.109#	0.00
Renal (%)	21.90	21.90	0.002	24.20	22.40	0.044	0.00
Liver (%)	16.50	14.50	0.053	17.00	14.40	0.071	0.00
COPD (%)	12.70	16.60	0.111#	15.70	13.40	0.065	0.00
CAD (%)	12.30	16.30	0.115#	15.00	15.50	0.016	0.00
Stroke (%)	8.90	8.00	0.033	7.80	10.10	0.080	0.00
Malignancy (%)	27.50	22.40	0.117#	26.60	22.40	0.099	0.00
Day of ICU admission (%)			0.139			0.091	12.60
Sunday	0.00	0.00		0.00	0.00		
Monday	13.30	14.90		14.40	15.10		
Tuesday	18.20	16.40		17.10	17.10		
Wednesday	17.60	17.60		17.20	16.10		
Thursday	15.00	19.10		16.30	19.20		
Friday	17.60	16.70		17.40	15.60		
Saturday	18.30	15.20		17.60	16.80		
Hour of ICU admission (%)			0.166			0.182	0.00
0	3.50	4.30		3.70	2.80		
1	3.10	3.20		3.70	2.70		
2	4.20	3.10		3.40	3.80		
3	2.50	3.30		2.80	2.60		
4	2.70	3.30		3.60	2.70		

Table 1 (*continued*)

Table 1 (*continued*)

Covariate	Original cohort (n=2,983)			Matched cohort (n=1,404)			Missing data (%)
	Non-TTE	TTE	SMD	Non-TTE	TTE	SMD	
5	1.60	2.60		1.70	2.10		
6	2.30	2.10		1.70	2.40		
7	1.20	1.10		1.10	0.60		
8	1.50	1.50		1.60	2.00		
9	2.10	2.40		2.70	2.00		
10	2.40	2.60		2.00	1.70		
11	2.80	3.10		2.70	3.30		
12	2.70	3.40		2.70	3.10		
13	3.80	3.40		4.10	3.40		
14	4.10	5.60		3.70	4.40		
15	4.50	4.60		5.40	4.60		
16	6.50	5.50		6.70	6.80		
17	5.90	5.50		6.00	5.80		
18	8.40	7.10		6.60	7.40		
19	5.70	5.60		5.10	6.80		
20	8.20	7.40		7.80	8.70		
21	7.20	6.80		7.50	7.10		
22	7.30	7.10		7.40	7.40		
23	6.00	5.30		6.10	5.60		
MAP (mmHg), mean (SD)	80.40 (20.28)	78.87 (19.21)	0.077#	79.63 (19.80)	79.70 (19.66)	0.004	0.40
Heart rate (beats/min), mean (SD)	91.07 (19.95)	94.96 (22.47)	0.183#	92.30 (20.58)	90.21 (18.93)	0.106#	0.40
Temperature (℃), mean (SD)	36.61 (1.97)	36.63 (1.12)	0.011	36.58 (1.10)	36.59 (1.07)	0.010	0.70
CVP (cmH_2O), mean (SD)	13.34 (24.19)	15.46 (24.78)	0.086	14.35 (27.80)	11.71 (6.41)	0.131	60.00
WBC ($\times10^9$/L), mean (SD)	13.50 (14.03)	13.36 (9.73)	0.011	14.43 (16.57)	13.16 (10.20)	0.093	2.80
Hemoglobin (g/L), mean (SD)	10.59 (2.07)	10.53 (2.07)	0.028	10.54 (2.11)	10.53 (2.13)	0.004	2.60
Platelets ($\times10^9$/L), mean (SD)	214.62 (125.10)	207.44 (128.71)	0.057	213.51 (131.37)	216.16 (136.58)	0.020	2.70
Sodium (mmol/L), mean (SD)	138.71 (5.77)	138.19 (5.87)	0.089#	138.47 (5.78)	138.01 (5.91)	0.079	1.50
Potassium (mmol/L), mean (SD)	4.32 (0.86)	4.33 (0.87)	0.016	4.34 (0.84)	4.29 (0.84)	0.060	1.40
Bicarbonate, mean (SD)	22.23 (5.94)	21.84 (5.93)	0.066	22.15 (6.10)	22.46 (5.88)	0.051	1.80
Chloride (mmol/L), mean (SD)	104.71 (7.10)	104.23 (7.23)	0.067	104.49 (7.28)	103.71 (7.26)	0.107#	1.60
BUN (mmol/L), mean (SD)	38.02 (29.88)	41.14 (28.57)	0.107#	39.53 (28.66)	39.86 (30.22)	0.011	1.70
Lactate (mmol/L), mean (SD)	3.09 (2.75)	2.94 (2.79)	0.052	2.94 (2.63)	2.68 (2.49)	0.100	37.30
Creatinine (μmol/L), mean (SD)	2.55 (2.83)	2.47 (2.37)	0.029	2.60 (2.98)	2.58 (2.67)	0.008	1.70
pH, mean (SD)	7.33 (0.12)	7.33 (0.11)	0.022	7.33 (0.12)	7.34 (0.11)	0.113#	27.10

Table 1 (*continued*)

Table 1 (*continued*)

Covariate	Original cohort (n=2,983)			Matched cohort (n=1,404)			Missing data (%)
	Non-TTE	TTE	SMD	Non-TTE	TTE	SMD	
PO_2 (mmHg), mean (SD)	146.59 (100.29)	135.25 (95.09)	0.116#	141.86 (100.98)	143.63 (99.55)	0.018	29.30
PCO_2 (mmHg), mean (SD)	41.42 (13.38)	42.84 (14.86)	0.101#	42.67 (14.28)	42.44 (14.18)	0.016	29.30
BNP (tested, %)	1.20	3.60	0.159#	1.30	1.70	0.035	0.00
Troponin (tested, %)	23.80	45.20	0.463#	37.00	30.80	0.133#	0.00
Creatinine kinase (tested, %)	39.10	60.70	0.443#	52.10	44.40	0.154#	0.00

#P<0.05. ICU, intensive care unit; SD, standard deviation; MICU, medical intensive care unit; SICU, surgical intensive care unit; SAPS, simplified acute physiology score; SOFA, sequential organ failure assessment; CHF, chronic heart failure; AFIB, atrial fibrillation; COPD, chronic obstructive pulmonary disease; CAD, coronary artery disease; MAP, mean artery pressure; CVP, central venous pressure; WBC, white blood cell; BUN, blood urine nitrogen; BNP, B-type natriuretic peptide; TTE, transthoracic echocardiography; SMD, standardized mean difference.

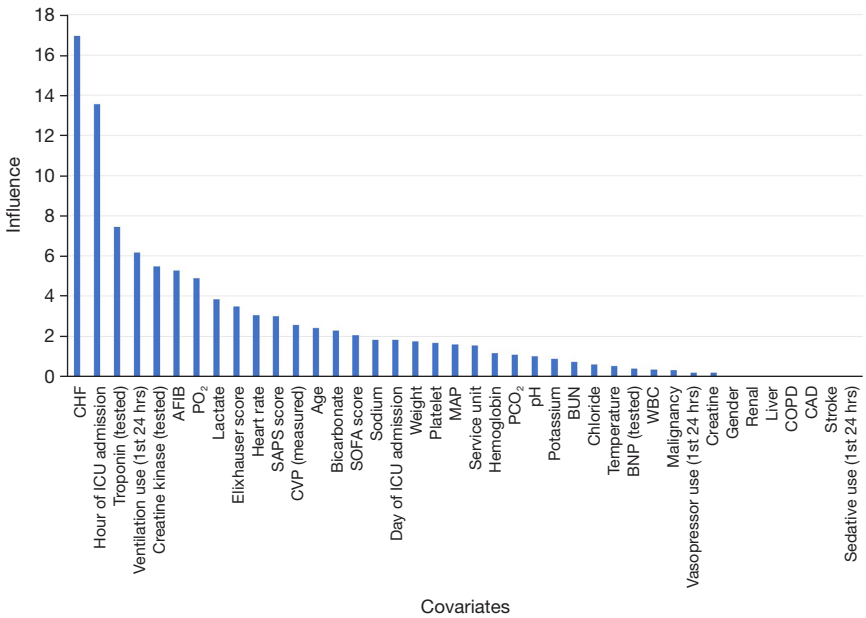

Figure 2 Relative influence factor of covariates. The relative influence factor measures how discriminative the 40 covariates of the PSM are when predicting the likelihood of echocardiography performance (influence over 1). CHF, chronic heart failure; ICU, intensive care unit; AFIB, atrial fibrillation; SAPS, simplified acute physiology score; CVP, central venous pressure; SOFA, sequential organ failure assessment; MAP, mean artery pressure; BUN, blood urine nitrogen; BNP, B-type natriuretic peptide; WBC, white blood cell; COPD, chronic obstructive pulmonary disease; CAD, coronary artery disease; PSM, propensity score model.

also significantly higher than those in non-TTE group. Relatedly, the proportion of patients with brain natriuretic peptide, troponin and creatinine kinase levels detected in the TTE group was significantly higher than in the non-TTE group.

Doubly robust analysis

GBM and 40 covariates were used to establish the PSM, and the contribution of each covariate to the PSM was significantly different (*Figure 2*). The top five covariates with the highest contribution were chronic heart failure (CHF),

Table 2 Primary outcome analysis with five different models

Models	OR	CI (2.5%)	CI (97.5%)
Doubly robust with unbalanced covariates	0.62	0.52	0.74
Doubly robust with all covariates	0.50	0.37	0.68
Propensity score IPW	0.73	0.65	0.81
Propensity score matching	0.66	0.54	0.81
Multivariate	0.53	0.40	0.72

IPW, inverse probabilities weighting; OR, odds ratio; CI, confidence interval.

hour of ICU admission, troponin (tested), ventilation use (1st 24 h) and creatinine kinase (tested). Further, after normalization by IPW, in the matched cohort the difference between the covariate TTE and non-TTE groups was no longer statistically significant (*Table 1*). However, there were still significant statistical differences in SAPS and SOFA scores, but compared with the original cohort, the opposite changes occurred. In the matched cohort, the average SAPS and SOFA scores of the non-TTE group were higher than those of the TTE group. Similarly, in the matched cohort, the non-TTE group used mechanical ventilation, vasopressor and sedative, and the ratio of detecting troponin and creatine kinase, and the ratio of CHF and AFIB were higher within the first 24 h.

Primary outcome

In the original cohort, 28-day mortality was 37.9% in the TTE group and 40.8% in the non-TTE group. In the PSM cohort, it was 34.6% in the TTE group and 45.6% in the non-TTE group. Doubly robust analysis showed that TTE was associated with lower 28-day mortality, and the analysis results of the five models were consistent (*Table 2*).

Secondary outcomes

Further secondary outcome analyses showed that patients in the TTE group lived longer without ventilation and vasopressor, and were infused lower fluid volumes on the first and second days of ICU admission. Other secondary outcomes had no statistical difference between the two groups of patients (*Table 3*).

Discussion

In our retrospective analysis of the MIMIC-III database, we found that performing TTE was associated with a reduced 28-day risk of death in patients with AKI hospitalized in the ICU. This benefit was consistent across multiple analytical models. At the same time, the results also showed that patients with AKI who underwent TTE spent longer without ventilation and vasopressor during their ICU stay and received less volume of fluid infusions on the first and second days of ICU stay. These results suggest that patients with AKI admitted to the ICU may receive more rational and precise treatment after TTE examination, thereby reducing the risk of death. In this study, doubly robust analysis showed that the top five covariates with the highest contribution were CHF, hour of ICU admission, troponin (tested), ventilation use (1st 24 h) and creatinine kinase (tested). In clinical practice, in CHF patients, troponin (tested) and creatine kinase (tested) are closely related to the clinician's decision to perform TTE examination.

The results of the present study of patients with AKI in the ICU are consistent with those of the study by Feng *et al.* (11), who focused on sepsis patients in the ICU. Similarly, in another study, Li *et al.* found that performing TTE was associated with decreased 28-day mortality in middle-aged and elderly patients in the ICU (16). In fact, all three categories (AKI, sepsis, older) are common patients in ICUs, and there is extensive overlap between the categories (17,18). In clinical practice, there is often a need for a certain basis to perform TTE examination of a patients (19), and commonly this includes patients with clinical manifestations of decreased cardiac function or myocardial injury [e.g., asthma, chest pain, limb edema, ST segment changes on electrocardiogram (ECG), etc.] or laboratory evidence [e.g., increased levels of B-type natriuretic peptide (BNP), troponin]; the patient may have cardiac structural changes (i.e., ECG or laboratory evidence of myocardial infarction, a history of valvular disease, or cardiac auscultation and abnormal heart murmurs, etc.) (19). Therefore, TTE is more often performed in patients with heart disease. In our study, patients who underwent TTE in the original cohort

Table 3 Secondary outcome analysis with propensity score matched cohorts

Secondary outcomes	Non-TTE group	TTE group	P value	Missing data (%)
Ventilation-free days in 28 days, mean (SD)	14.05 (13.77)	17.17 (32.55)	0.022*	0.00
Vasopressor-free days in 28 days, mean (SD)	15.19 (14.87)	18.30 (19.73)	0.002*	0.00
Dobutamine use (%)	1.90	3.40	0.100	0.00
IV fluids day 1 (mL), mean (SD)	3,143.93 (3,559.72)	2,554.96 (3,494.82)	0.003*	15.40
IV fluids day 2 (mL), mean (SD)	2,038.08 (2,672.23)	1,541.76 (2,743.58)	0.002*	20.60
IV fluids day 3 (mL), mean (SD)	1,110.59 (2,362.48)	1,054.15 (2,681.68)	0.485	30.60
SOFA reduction day 2, mean (SD)	0.10 (4.74)	0.05 (3.29)	0.813	0.00
SOFA reduction day 3, mean (SD)	0.25 (5.12)	0.26 (3.56)	0.972	0.00
Norepinephrine (maximum dosage mg/min), mean (SD)	1.64 (3.63)	1.41 (3.01)	0.197	0.00
Serum lactate reduction (48 h), mean (SD)	0.16 (2.76)	0.35 (1.93)	0.896	66.00
Serum creatinine reduction (48 h), mean (SD)	–0.10 (2.12)	0.03 (1.25)	0.115	21.20
Serum lactate reduction (24 h), mean (SD)	0.23 (2.29)	0.21 (1.55)	0.894	59.30
Serum creatinine reduction (24 h), mean (SD)	–0.11 (1.85)	–0.02 (1.03)	0.350	14.90

*, P<0.05. SD, standard deviation; IV, intravenous injection; SOFA, sequential organ failure assessment; TTE, transthoracic echocardiography.

were more ill than those who did not (SAPS, SOFA, and Elixhauser scores were higher in the TTE group than in the non-TTE group), but had a lower mortality rate than the patients without TTE. In the original cohort of our study, we did observe higher rates of heart failure, AFIB, and CAD in patients undergoing TTE (*Table 1*). However, in our matched cohort, patients who did not undergo TTE had higher rates of heart failure, AFIB, and increased troponin, and creatine kinase (*Table 1*). Meanwhile, in the matched cohort, there was no significant difference in the rate of CAD and the rate of detection of BNP between the two groups (*Table 1*). Therefore, in the matched cohort, there may have been some patients who should have undergone TTE but did not and this can lead to the clinician not fully understanding the patient's condition, which is not conducive to the formulation or adjustment of treatment plans. Based on the results of this and similar studies (11,16), for patients with clear indications for a TTE examination, it should be actively considered, and may help reduce the short-term risk of death of patients (11,20,21).

Further analysis showed that TTE was also associated with shorter duration of both mechanical ventilation and vasopressor use during the 28-day ICU stay, which may be related to accurate real-time cardiac ultrasonography. When doctors have enough information about the structure and function of the heart, they can more accurately judge the change and development of the patient's condition, thus more rationally use mechanical ventilation and vasopressor drugs (22). Furthermore, the patients who underwent TTE had less fluid input during the first 48 h of ICU admission than patients who did not undergo TTE, possibly because TTE more accurately detected cardiac functional status and provided more information on right heart pressure and the basis for the rational use of fluids. However, this result is inconsistent with other studies. In Feng *et al.*'s study, patients who underwent TTE had significantly higher fluid intake in the first 3 days after ICU admission than patients who did not undergo TTE (11). This discrepancy may relate to the different target populations of the studies. In our study, the subjects were patients with AKI who required strict control of fluid input when they were not receiving dialysis. We know that many cases of AKI are secondary to heart disease. Kidney function declines rapidly due to a sharp decline in cardiac function, resulting in severe hypoperfusion of the kidneys. TTE can provide accurate and real-time cardiac structural and functional information, which is helpful for timely identification of a cardiogenic factor in renal function changes (23,24). Improvement of cardiac function and thus renal perfusion can rapidly improve renal function (25).

This study has certain limitations. First, it was retrospective based on the MIMIC-III database, and many patients were excluded according to the inclusion and exclusion criteria. The reason for exclusion was mainly related to incomplete clinical data, so a patient selection bias may exist. Second, the MIMIC-III database used recorded case data before 2011, and 11 years have passed since. In that time, critical care medicine, especially for AKI, has developed significantly, and the current treatment plan may be significantly different from that of a decade ago. Therefore, the results of this study cannot be used as the final basis for clinical application, but have certain enlightening significance for clinical practice. Future research should be a multicenter real-world study, based on an electronic medical record system, to include a large sample of cases, and further analyze and verify the results of this study.

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Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://atm.amegroups.com/article/view/10.21037/atm-22-3158/rc

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://atm.amegroups.com/article/view/10.21037/atm-22-3158/coif). 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).

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