ORIGINAL ARTICLE

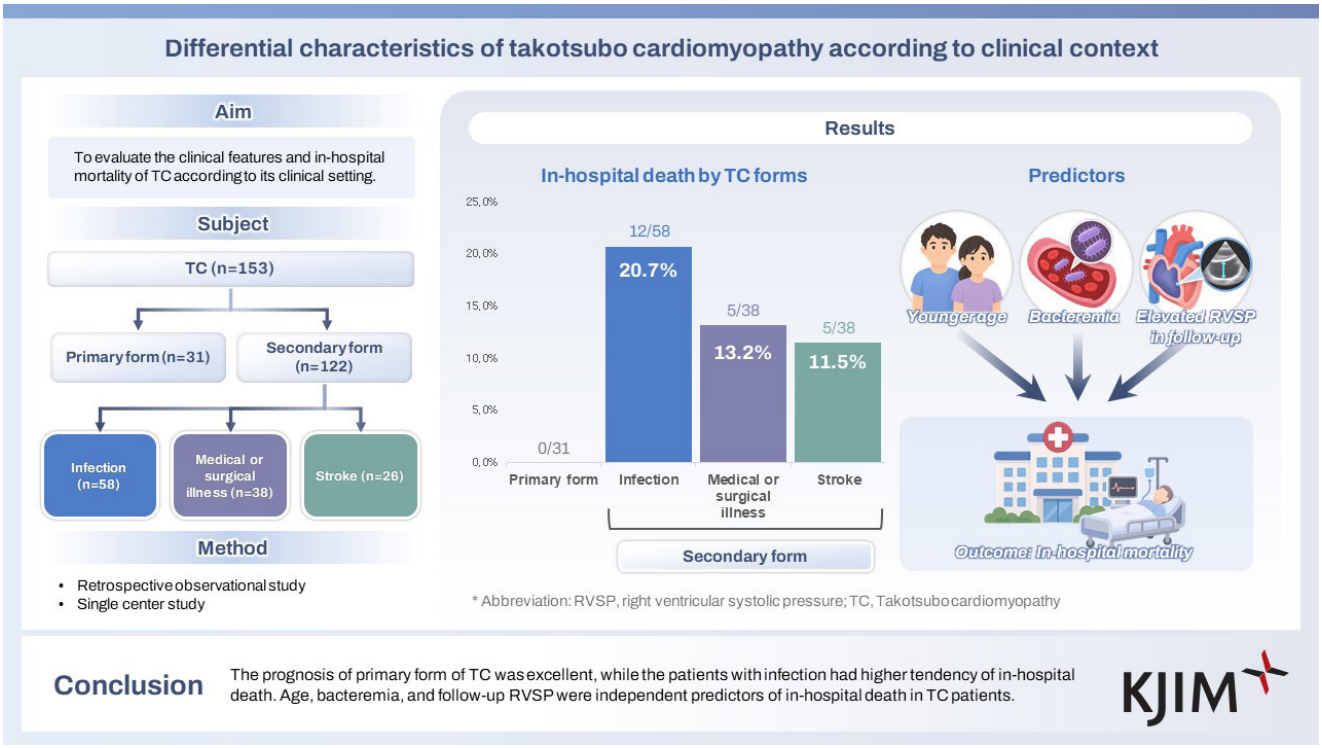
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Differential characteristics of takotsubo cardiomyopathy according to clinical context

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Background/Aims: Takotsubo cardiomyopathy (TC) occurs in diverse clinical settings and generally has a favorable prognosis; however, little is known about its characteristics and outcomes across different contexts. We evaluated the clinical features and in-hospital mortality of TC according to clinical setting.

Methods: A total of 153 patients diagnosed with TC from 2008 to 2020 were included. Patients were classified as having primary TC (presenting as the primary reason for care, n = 31) or secondary TC (n = 122), which was further subdivided into infection (n = 58), medical/surgical illness (n = 38), and stroke (n = 26).

Results: Prior cerebrovascular accident was more frequent in the infection and stroke groups. Length of hospital stay, inten-

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sive care unit stay, and ventilator use were greatest in the stroke group, whereas inotropic agent use was most frequent in the infection group. N-terminal pro–B-type natriuretic peptide levels differed across groups, with the highest levels observed in the primary form (p = 0.025). Echocardiographic parameters were generally comparable, although left atrial diameter was larger in the primary form and smaller in the stroke group. Overall in-hospital mortality was 13.1% and occurred exclusively in secondary TC. Mortality was highest in the infection group, but the difference was not statistically significant (p = 0.053). In multivariate regression analysis, age, bacteremia, and follow-up right ventricular systolic pressure (RVSP) were independently associated with in-hospital mortality.

Conclusions: The prognosis of primary form of TC was excellent, while the patients with infection had higher tendency of in-hospital death. Age, bacteremia, and follow-up RVSP were independent predictors of in-hospital death in TC patients.

Keywords: Echocardiography; Mortality; Takotsubo cardiomyopathy

INTRODUCTION

Takotsubo cardiomyopathy (TC), also known as Takotsubo syndrome, is characterized by transient left ventricular systolic dysfunction that mimics acute coronary syndrome in the absence of obstructive coronary artery disease [1,2]. Although the prognosis is generally favorable, a subset of patients experience severe complications, including cardiogenic shock and in-hospital death [3-6]. TC has been increasingly recognized in various clinical settings, not only following emotional or physical stress but also as a complication of other critical illnesses, such as infection, surgery, or stroke.

According to a recent review, TC can be classified into two major forms depending on the clinical context: the primary form, wherein TC constitutes the index cause for medical presentation, and the secondary form, wherein TC develops secondarily during the evaluation or treatment of another acute illness [1]. This distinction may have important clinical implications, because the underlying pathophysiological mechanisms, triggers, and outcomes likely differ between both forms [1,6]. However, comprehensive data comparing clinical and echocardiographic characteristics and outcomes of both forms remain scarce. Moreover, the secondary form encompasses a heterogeneous spectrum of precipitating conditions, and its prognosis may vary depending on the underlying comorbidity.

Therefore, we sought to evaluate the potential differences in the characteristics and in-hospital outcomes of patients with TC according to its clinical form and context, with particular focus on differences among secondary subtypes related to infection, medical or surgical illness, and stroke.

METHODS

Study design and population

Details of the participant selection and group allocation are shown in Figure 1. Between June 2008 and September 2020, 73,099 patients who underwent echocardiography during admission to Ilsan Paik Hospital, Inje University, Goyang, South Korea, were screened for eligibility in this retrospective study. Among the 358 patients with suspected TC, 131 without follow-up echocardiography and 74 with confirmed obstructive coronary artery disease or other forms of cardiomyopathy were excluded. Therefore, a total of 153 patients who met the following criteria were included in the final analysis: (1) typical shape of takotsubo or inverted takotsubo on echocardiography, or transient akinesia/dyskinesia beyond a single epicardial coronary artery territory, confirmed with follow-up echocardiography; (2) no evidence of obstructive epicardial coronary artery disease or angiographic evidence of acute plaque rupture, including no history of angina and a normal coronary angiography or myocardial perfusion scan; (3) no evidence of other forms of cardiomyopathy, including myocarditis or primary cardiomyopathy, based on clinical evaluation and imaging findings. TC was diagnosed based on the established diagnostic criteria, including the Mayo Clinic and InterTAK criteria [7,8]. In patients who did not undergo coronary angiography because of clinical instability or physician judgment, the diagnosis was based on an integrated clinical assessment, includ-

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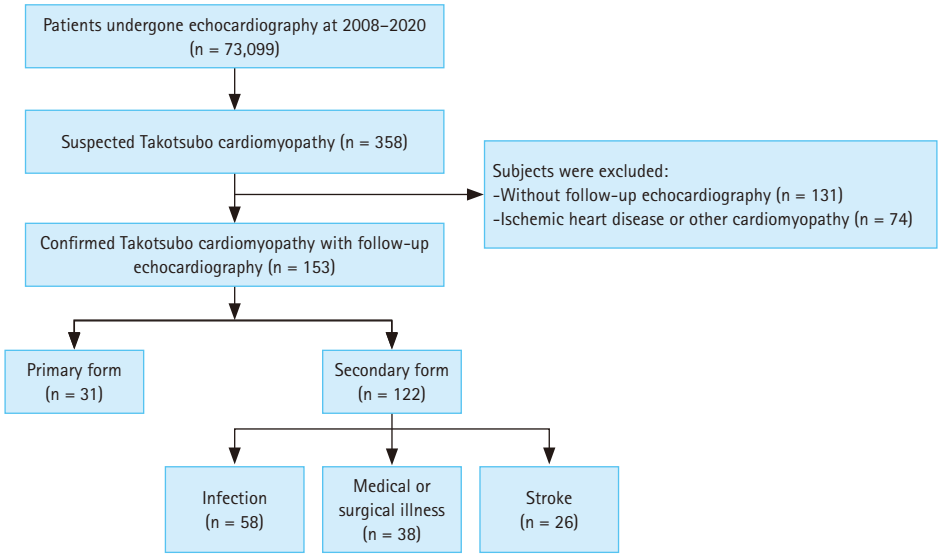


Figure 1. Flow diagram for selection of study population. Study population at each step and exclusion reasons are described in detail.

ing characteristic echocardiographic findings (regional wall motion abnormalities extending beyond a single coronary territory), clinical presentation, electrocardiographic (ECG) findings, cardiac biomarker patterns, presence of identifiable physical or emotional stressors, and the absence of clinical features suggestive of type 1 myocardial infarction. Recovery of left ventricular systolic function on follow-up echocardiography was required to confirm the diagnosis of TC. Patients with TC were classified into the primary or secondary form according to the clinical context as described in a previous review [1]. The primary form was defined as TC constituting the index cause for medical presentation, whereas the secondary form was defined as TC identified in the setting of ongoing evaluation or treatment for another critical illness. The secondary form was further subdivided into infection, medical or surgical illness, and stroke groups, according to the clinical context in which the patients were already receiving treatment. Experienced cardiologists confirmed all diagnoses based on a comprehensive clinical review. The study was performed in accordance with the ethical principles of the 1975 Declaration of Helsinki. The Institutional Review Board of Ilsan Paik Hospital approved the study (approval no. 2025-11-019) and waived the requirement for informed consent due to its retrospective design.

Covariates and definition

Data on demographic characteristics, medical history, laboratory findings, and ECG and echocardiographic parameters were retrospectively collected from a comprehensive review of electronic medical records and compared across TC subtypes. Information regarding the reason for echocardiography, duration of hospitalization, presence and length of intensive care unit (ICU) stay, use of ventilatory support or inotropes, and in-hospital mortality was also obtained through a review of electronic medical records.

Blood samples for analysis of cell count, liver function, creatinine, erythrocyte sedimentation rate (ESR), cardiac enzymes, and N-terminal pro-B-type natriuretic peptide (NT-proBNP) were obtained from all patients at the time of admission. Peak values of cardiac enzymes during admission were also collected. Bacteremia was confirmed when a viable microorganism was detected in the bloodstream via a positive blood culture.

ECG findings were assessed using the ECG obtained closest to the time of TC diagnosis. In patients with primary TC, this corresponded to the ECG obtained at initial presentation, whereas in patients with secondary TC, the ECG findings were based on the ECG obtained at the time of TC diagnosis, defined as the date of index echocardiography. The ECG findings included T-wave inversion, ST-segment depression, ST-segment elevation, Q waves, atrial tachyarrhythmias, and ventricular tachyarrhythmias. If none of these findings were observed, the ECG was classified as showing non-specific findings. For individuals with a previous baseline ECG, newly developed changes were identified by comparing with the prior tracing.

Coronary evaluation was left to the physician's discretion and was performed using either invasive or computed tomographic angiography, or myocardial perfusion scanning.

Echocardiographic examinations were conducted following the American Society of Echocardiography guidelines [9,10]. Digitally stored echocardiographic images were reviewed by experienced echocardiography specialists to confirm parameters and classify TC morphological subtypes according to the distribution of left ventricular wall motion abnormalities as described in the International Takotsubo Registry (apical, mid-ventricular, basal, focal, and diffuse types) [6,8]. Left ventricular ejection fraction (LVEF) was obtained using the modified biplane Simpson's method from the apical 4- and 2-chamber views. Right ventricular systolic pressure (RVSP) was calculated from the peak tricuspid regurgitant jet velocity (V) using the modified Bernoulli equation (RVSP = $4V^2$ + right atrial pressure). Follow-up echocardiography was performed at a median of 10 days (interquartile range, 6–42 days) to assess the left ventricular function.

In-hospital death was defined as death from any cause during the index hospitalization and was assessed among the groups according to the TC subtype.

Statistical analysis

Continuous variables were expressed as mean ± standard deviation or median [interquartile range], depending on their distribution, and categorical data as numbers (percentages). Baseline characteristics, laboratory findings, and echocardiographic parameters were compared across the TC forms using one-way analysis of variance or the Kruskal–Wallis test for continuous variables and Pearson's chi square test or Fisher's exact test for categorical variables. Comparisons between two groups were performed using Student's t-test or Wilcoxon rank-sum test, as appropriate. In-hospital mortality was also compared across the TC forms. A univariate logistic regression model was used to assess the association between covariates and in-hospital mortality in patients with TC. To determine the predictors of in-hospital mortality, multivariate logistic regression analysis was performed. All variables with a $p < 0.10$ in the univariate analysis and age were entered into the model. Given the limited number of outcome events, the number of variables included in the final model was restricted to avoid over-parameterization. Multicollinearity among the variables included in the multivariate model was assessed using variance inflation factors, and no significant collinearity was observed (all variance inflation factor values were < 3). To assess potential selection bias, baseline clinical characteristics were compared between patients with and without follow-up echocardiography; the latter were excluded from the study population. As an additional sensitivity analysis, we repeated the multivariate logistic regression analysis after excluding very elderly patients (age ≥ 85 years) to assess the robustness of the association between age and in-hospital mortality. Analyses of baseline medical conditions, laboratory and echocardiographic findings were reported using available data with no imputation for missing data given the low rate of missing data (< 3%). There were no missing data regarding the clinical events. Statistical analyses were performed using the R Statistical Software (version 4.5.1; R Foundation for Statistical Computing, Vienna, Austria). For all statistical tests, a two-tailed p value of < 0.05 was considered significant.

RESULTS

Baseline characteristics of study population

The baseline characteristics of the study population stratified according to the primary or secondary forms of TC are summarized in Table 1. The mean age of the population was 68.5 ± 17.3 years, and 98 (64.1%) participants were female; these characteristics did not differ significantly between the primary and secondary forms. The rates of comorbidities were generally comparable between the groups, except that previous cerebrovascular accident was more frequent in the secondary form due to infection (24.1%) or stroke (26.9%) than in the primary form (6.5%) or the secondary form due to medical or surgical illness (7.9%). The duration of hospital and ICU stay was longer in the secondary form than in the primary form, with the secondary form showing the longest stays for both due to stroke. Ventilator care was also more frequent in the secondary form, particularly in cases secondary to stroke. The use of inotropic agents was most frequent in the secondary form due to infection.

Laboratory findings and coronary evaluation

Blood chemistry and ECG findings according to the primary and secondary forms of TC are presented in Table 2. Blood test results, including complete blood counts and initial or peak cardiac enzyme levels, were generally similar across the TC forms. Creatinine levels were the highest and estimated

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Table 1. Baseline characteristics of patients with stress cardiomyopathy

Characteristics	Total (n = 153)	Primary form (n = 31)	Secondary form			*p* value
			Infection (n = 58)	Medical or surgical illness (n = 38)	Stroke (n = 26)	
Age, yr	68.5 ± 17.3	62.9 ± 17.8	73.6 ± 12.4	69.1 ± 17.4	62.7 ± 22.6	0.636
Female	98 (64.1)	21 (67.7)	31 (53.4)	26 (68.4)	20 (76.9)	0.158
Hypertension	80 (52.3)	17 (54.8)	31 (53.4)	20 (52.6)	12 (46.2)	0.918
Diabetes	51 (33.3)	10 (32.3)	24 (41.4)	14 (36.8)	3 (11.5)	0.058
Current smoker	20 (13.1)	5 (16.1)	4 (6.9)	5 (13.2)	6 (23.1)	0.213
Chronic kidney disease[a)]	6 (3.9)	0	2 (3.4)	4 (10.5)	0	0.080
Chronic lung disease	25 (16.3)	5 (16.1)	11 (19.0)	5 (13.2)	4 (15.4)	0.898
Any cancer	16 (10.5)	3 (9.7)	10 (17.2)	3 (7.9)	0	0.103
Previous PCI	11 (7.2)	2 (6.5)	2 (3.4)	4 (10.5)	3 (11.5)	0.399
Previous MI	6 (3.9)	2 (6.5)	1 (1.7)	2 (5.3)	1 (3.8)	0.693
Previous CVA	26 (17.0)	2 (6.5)	14 (24.1)	3 (7.9)	7 (26.9)	0.035
Previous AF	19 (12.4)	7 (22.6)	8 (13.8)	4 (10.5)	0	0.077
Reasons for echocardiography						< 0.001
Chest pain	8 (5.2)	7 (22.6)	0	1 (2.6)	0	
Dyspnea	53 (34.6)	15 (48.4)	27 (46.6)	10 (26.3)	1 (3.8)	
Others	92 (60.1)	9 (29.0)	31 (53.4)	27 (71.1)	25 (96.2)	
Hospital stay, days	18.0 [9.0–37.0]	8.0 [5.0–10.0]	23.0 [14.0–40.0]	15.0 [10.0–26.5]	52.0 [19.5–86.8]	< 0.001
ICU care	117 (76.5)	19 (61.3)	48 (82.8)	28 (73.7)	22 (84.6)	0.095
ICU stay, days	7.0 [4.0–16.3]	3.0 [1.5–3.5]	9.0 [6.5–22.5]	6.5 [3.0–14.3]	12.5 [5.3–28.0]	< 0.001
Ventilator care	70 (45.8)	5 (16.1)	31 (53.4)	16 (42.1)	18 (69.2)	< 0.001
Inotropic use	92 (60.1)	12 (38.7)	45 (77.6)	18 (47.4)	17 (65.4)	0.001

Values are presented as mean ± standard deviation, median [interquartile range], or number (%), as appropriate.
AF, atrial fibrillation; CVA, cerebrovascular accident; ICU, intensive care unit; MI, myocardial infarction; PCI, percutaneous coronary intervention.
[a)]Chronic kidney disease was defined as an estimated glomerular filtration rate of < 60 mL/min/1.73 m^2.

glomerular filtration rate was the lowest in the medical or surgical illness subgroup, whereas the stroke subgroup had the most preserved renal function (p = 0.006 and p = 0.002, respectively). ESR was the highest in the infection subgroup (p = 0.001). NT-proBNP levels were highest in the primary form (5,923.5 [1,564.5–9,381.3] pg/mL), followed by the secondary form due to infection (2,760.0 [1,428.0–11,878.0] pg/mL), medical or surgical illness (1,598.0 [591.8–5,147.0] pg/mL), and stroke (366.0 [55.0–1,175.3] pg/mL) (p = 0.025). On ECG, T inversion was the most common finding (60.7%), followed by ST segment elevation (13.1%), atrial tachyarrhythmia (3.9%), ST segment depression (2.6%), Q wave (1.3%), and ventricular tachyarrhythmia (0.7%). incidence of these findings was comparable between the TC forms (p = 0.173). Coronary artery evaluation was performed in 46.4% of the study population, most frequently in the primary form (77.4%), followed by the secondary form due to medical or surgical illness (44.7%), infection (36.2%), and stroke (34.6%) (Table 2).

Echocardiographic findings

Table 3 presents the echocardiographic parameters according to the TC form. The initial LVEF of the study population was 41.4 ± 8.4%, but the follow-up LVEF improved to 59.0 ± 11.1%. The initial and follow-up echocardiographic parameters were comparable between the TC forms, except that the left atrial diameter was larger in the primary form and smaller in the secondary form because of stroke. There

Table 2. Laboratory findings

Variables	Total (n = 153)	Primary form (n = 31)	Secondary form			*p* value
			Infection (n = 58)	Medical or surgical illness (n = 38)	Stroke (n = 26)	
Blood chemistry						
Hemoglobin, g/dL	11.7 ± 2.4	12.7 ± 1.7	11.0 ± 2.0	11.1 ± 3.0	13.0 ± 1.9	0.648
White blood cell count, k/mm^3	12.8 ± 7.2	11.6 ± 7.2	13.2 ± 7.5	13.5 ± 8.1	11.9 ± 5.3	0.828
Neutrophil, %	76.2 ± 17.0	71.1 ± 20.0	82.4 ± 14.7	74.9 ± 16.5	70.2 ± 15.0	0.335
Platelet count, k/mm^3	226.2 ± 95.5	241.5 ± 80.5	214.8 ± 109.0	215.4 ± 91.3	249.1 ± 80.9	0.795
Creatinine, mg/dL	0.9 [0.7–1.3]	0.9 [0.7–1.1]	1.0 [0.7–1.5]	1.1 [0.9–2.2]	0.8 [0.6–0.9]	0.006
eGFR, mL/min/1.73 m^2	62.1 [43.3–85.7]	68.6 [53.9–83.1]	57.4 [41.8–80.1]	47.0 [25.7–63.7]	81.4 [69.7–98.2]	0.002
ESR, mm/hr	18.0 [8.0–31.0]	6.0 [2.0–13.0]	25 [12.0–39.0]	19 [6.3–32.5]	17 [9.0–27.0]	0.001
Initial CKMB, ng/mL	3.5 [1.8–10.0]	4.4 [1.7–13.4]	3.0 [1.2–7.2]	4.9 [2.4–10.3]	3.2 [1.8–10.0]	0.139
Initial cTnI, pg/mL	5.0 [0.1–184.0]	2.7 [0.5–55.4]	12.5 [0.2–286.7]	3.9 [0.0–262.8]	8.5 [0.1–106.3]	0.723
Peak CKMB, ng/mL	11.5 [4.4–22.7]	14.4 [6.2–20.3]	9.4 [4.2–19.7]	13.5 [4.6–36.4]	12.7 [6.1–27.8]	0.381
Peak cTnI, pg/mL	22.6 [0.8–1,679.8]	6.2 [0.4–2,513.6]	105.0 [1.2–1,559.3]	3.9 [0.3–2,328.1]	71.6 [0.6–1,097.2]	0.816
NT-proBNP, pg/mL	2,571.0 [873.4–7,163.3]	5,923.5 [1,564.5–9,381.3]	2,760.0 [1,428.0–11,878.0]	1,598.0 [591.8–5,147.0]	366.0 [55.0–1,175.3]	0.025
ECG findings						0.173
T inversion	93 (60.7)	21 (67.7)	34 (58.6)	26 (68.4)	12 (46.2)	
ST segment depression	4 (2.6)	0	1 (1.7)	1 (2.6)	2 (7.7)	
ST segment elevation	20 (13.1)	5 (16.1)	10 (17.2)	2 (5.3)	3 (11.5)	
Q wave	2 (1.3)	1 (3.2)	0	1 (2.6)	0	
Atrial tachyarrhythmia	6 (3.9)	1 (3.2)	4 (6.9)	1 (2.6)	0	
Ventricular tachyarrhythmia	1 (0.7)	1 (3.2)	0	0	0	
Non-specific change	27 (17.6)	2 (6.5)	9 (15.5)	7 (18.4)	9 (34.6)	
Coronary artery evaluation	71 (46.4)	24 (77.4)	21 (36.2)	17 (44.7)	9 (34.6)	0.001

Values are presented as mean ± standard deviation, median [interquartile range], or number (%), as appropriate.
CKMB, creatine kinase MB; cTnI, cardiac troponin I; ECG, electrocardiography; eGFR, estimated glomerular filtration rate; ESR, erythrocyte sedimentation rate; NT-proBNP, N-terminal pro-B-type natriuretic peptide.

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Table 3. Echocardiographic parameters

Variables	Total (n = 153)	Primary form (n = 31)	Secondary form			*p* value
			Infection (n = 58)	Medical or surgical illness (n = 38)	Stroke (n = 26)	
Initial						
LVEF, %	41.4 ± 8.4	43.1 ± 8.3	38.3 ± 7.7	41.6 ± 7.0	45.8 ± 9.6	0.067
LVEDD, mm	48.2 ± 7.7	49.9 ± 6.1	48.8 ± 5.5	45.4 ± 11.4	48.8 ± 6.0	0.185
LVESD, mm	35.2 ± 7.5	36.5 ± 8.3	35.5 ± 7.0	34.3 ± 7.6	34.4 ± 7.3	0.192
IVSD, mm	8.7 ± 1.4	8.9 ± 1.9	8.4 ± 1.1	8.9 ± 1.3	8.7 ± 1.2	0.929
LVPWD, mm	8.8 ± 1.3	9.2 ± 1.4	8.5 ± 1.3	8.9 ± 1.3	8.8 ± 1.2	0.595
LA diameter, mm	36.3 ± 8.5	38.1 ± 9.7	36.8 ± 7.9	37.5 ± 8.3	31.6 ± 7.5	0.014
LAVI, mL/m^2	35.4 ± 16.6	41.1 ± 24.0	35.9 ± 15.1	31.8 ± 12.7	32.0 ± 12.7	0.093
E/A ratio	1.0 ± 0.7	1.0 ± 0.6	0.9 ± 0.7	1.0 ± 1.0	0.9 ± 0.5	0.689
DT, msec	188.5 ± 62.8	182.3 ± 49.3	192.6 ± 70.0	186.1 ± 54.9	189.9 ± 73.4	0.830
E/E'	15.3 ± 8.2	14.6 ± 8.1	13.8 ± 5.9	19.6 ± 10.8	14.1 ± 7.4	0.393
TR jet velocity, m/s	2.6 ± 0.6	2.6 ± 0.6	2.6 ± 0.6	2.6 ± 0.6	2.5 ± 0.5	0.600
RVSP, mmHg	38.9 ± 12.8	39.3 ± 13.4	39.1 ± 12.8	39.6 ± 14.3	36.8 ± 9.8	0.618
Follow-up						
LVEF, %	59.0 ± 11.1	63.3 ± 8.2	56.8 ± 12.7	56.6 ± 11.2	62.1 ± 8.0	0.640
LVEDD, mm	47.0 ± 7.8	48.9 ± 7.6	46.2 ± 6.7	46.9 ± 8.7	46.7 ± 9.3	0.416
LVESD, mm	30.4 ± 6.5	30.9 ± 6.6	30.0 ± 6.7	32.3 ± 6.6	28.0 ± 4.6	0.368
IVSD, mm	9.0 ± 1.2	8.8 ± 1.3	9.1 ± 1.0	8.9 ± 1.2	9.1 ± 1.2	0.270
LVPWD, mm	9.0 ± 1.2	8.8 ± 1.1	9.1 ± 1.2	8.8 ± 1.3	9.3 ± 1.1	0.306
LA diameter, mm	35.9 ± 8.3	38.6 ± 9.1	35.3 ± 8.6	36.9 ± 7.4	32.7 ± 6.9	0.033
LAVI, mL/m^2	34.2 ± 20.5	38.2 ± 23.3	34.7 ± 17.7	37.4 ± 25.3	23.5 ± 9.6	0.058
E/A ratio	0.9 ± 0.5	1.1 ± 0.8	0.7 ± 0.3	0.9 ± 0.5	0.8 ± 0.4	0.343
DT, msec	202.4 ± 78.0	221.7 ± 100.8	187.0 ± 74.8	205.3 ± 71.1	208.2 ± 57.4	0.845
E/E'	12.9 ± 6.8	12.3 ± 6.3	12.9 ± 6.2	14.1 ± 8.6	12.1 ± 6.0	0.871
TR jet velocity, m/s	2.5 ± 0.5	2.4 ± 0.5	2.6 ± 0.6	2.5 ± 0.4	2.2 ± 0.3	0.108
RVSP, mmHg	36.3 ± 12.0	35.0 ± 11.1	39.8 ± 14.3	35.7 ± 9.4	30.6 ± 6.7	0.069
TC types						0.374
Apical	124 (81.0)	24 (77.4)	50 (86.2)	31 (81.6)	19 (73.1)	
Mid	19 (12.4)	4 (12.9)	7 (12.1)	4 (10.5)	4 (15.4)	

Table 3. Continued

Variables	Total (n = 153)	Primary form (n = 31)	Secondary form			p value
			Infection (n = 58)	Medical or surgical illness (n = 38)	Stroke (n = 26)	
Basal	5 (3.3)	2 (6.5)	1 (1.7)	0	2 (7.7)	
Focal	3 (2.0)	1 (3.2)	0	1 (2.6)	1 (3.8)	
Diffuse	2 (1.3)	0	0	2 (5.3)	0	
Timing of follow-up echocardiography, days	10.0 [6.0–42.0]	13.0 [5.5–192.0]	9.0 [6.0–22.5]	16.0 [7.0–70.0]	12.0 [4.3–20.8]	0.291

Values are presented as mean ± standard deviation, median [interquartile range], or number (%), as appropriate.
DT, deceleration time; IVSD, interventricular septum diameter; LA, left atrium; LAVI, left atrial volume index; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic diameter; LVPWD, left ventricular posterior wall thickness in diastole; RVSP, right ventricular systolic pressure; TC, Takotsubo cardiomyopathy; TR, tricuspid regurgitation.

Figure 2. In hospital mortality according to the forms of stress cardiomyopathy. No deaths occurred in the primary form, whereas all in-hospital deaths occurred in the secondary form. Among the secondary subgroups, infection-related Takotsubo cardiomyopathy showed the highest mortality rate (20.7%), followed by medical or surgical illness (13.2%), and stroke (11.5%), although the difference was not statistically significant ($p = 0.053$).

were no differences in the TC morphological types among the TC forms ($p = 0.374$). The timing of follow-up echocardiography did not significantly differ across the etiologic subgroups ($p = 0.291$).

In-hospital death

There were 20 in-hospital deaths (13.1%) in our study population, and all cases occurred in the secondary form of TC. The in-hospital mortality rate was highest in the secondary form due to infection (20.7%), followed by the secondary form due to medical or surgical illness (13.2%), and the secondary form due to stroke (11.5%); however, these differences were not statistically significant ($p = 0.053$) (Fig. 2). Follow-up echocardiography tended to be performed earlier in patients who died during hospitalization, although the difference was not statistically significant (median 8.5 vs. 12.0 days, $p = 0.065$).

Predictors for in-hospital death

Table 4 shows the factors associated with in-hospital death among patients with TC. After adjustment for covariates, younger age (odds ratio [OR], 0.933; 95% confidence interval [CI], 0.887–0.981; $p = 0.007$), bacteremia (OR, 6.302; 95% CI, 1.097–36.199; $p = 0.039$), and follow-up RVSP (OR, 1.114; 95% CI, 1.040–1.193; $p = 0.002$) were identified as independent factors associated with in-hospital death. To further explore this unexpected association between young-

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Table 4. Predictors of in-hospital death

Variables	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*p* value	OR (95% CI)	*p* value
Age, yr	0.984 (0.960–1.001)	0.206	0.933 (0.887–0.981)	0.007
Female sex	0.511 (0.198–1.319)	0.165		
Hypertension	0.900 (0.351–2.305)	0.826		
Diabetes	0.630 (0.216–1.844)	0.400		
Chronic lung disease	1.333 (0.405–4.386)	0.636		
Any cancer	2.521 (0.725–8.763)	0.146		
Previous CVA	2.421 (0.832–7.045)	0.105		
Previous AF	1.290 (0.340–4.898)	0.708		
ICU stay, days	1.010 (0.989–1.032)	0.337		
Ventilator care	6.667 (1.455–30.544)	0.015	2.419 (0.337–17.347)	0.379
Inotropics use	5.440 (0.688–43.025)	0.108		
Apical type	0.926 (0.285–3.010)	0.898		
ESR	1.023 (1.000–1.047)	0.051	1.042 (0.997–1.088)	0.066
Bacteremia	4.320 (1.624–11.494)	0.003	6.302 (1.097–36.199)	0.039
LAVI	0.992 (0.940–1.047)	0.774		
Initial LVEF	0.892 (0.834–0.955)	< 0.001	0.899 (0.789–1.026)	0.114
Follow-up LVEF	0.966 (0.928–1.005)	0.088	0.967 (0.899–1.040)	0.366
Initial RVSP	0.997 (0.960–1.036)	0.882		
Follow-up RVSP	1.056 (1.019–1.094)	0.003	1.114 (1.040–1.193)	0.002
Change in RVSP[a)]	1.035 (1.002–1.069)	0.037		
ST segment elevation	0.710 (0.152–3.321)	0.663		

AF, atrial fibrillation; CI, confidence interval; CVA, cerebrovascular accident; ESR, erythrocyte sedimentation rate; ICU, intensive care unit; LAVI, left atrial volume index; LVEF, left ventricular ejection fraction; OR, odds ratio; RVSP, right ventricular systolic pressure.
[a)]Calculated as follow-up RVSP minus initial RVSP.

er age and mortality, we compared clinical severity markers and etiologic subgroups between younger (< 65 years) and older patients (≥ 65 years). Most clinical severity markers and etiologic subgroups were comparable between the two age groups, although follow-up RVSP was higher in older patients than in younger patients (37.7 ± 12.8 vs. 33.1 ± 9.0 mmHg, $p = 0.016$) (Supplementary Table 1). In crude comparisons, age did not significantly differ between survivors and non-survivors, whereas markers of illness severity such as ventilator support, bacteremia, lower initial LVEF, and higher follow-up RVSP, were more common among non-survivors. In addition, RVSP increased during follow-up in non-survivors but decreased slightly in survivors (Supplementary Table 2).

Sensitivity analysis

To assess the potential selection bias, we compared the baseline characteristics of patients with and without follow-up echocardiography. Patients without follow-up echocardiography were older and had significantly higher in-hospital mortality rates than those who underwent follow-up imaging (36.6% vs. 13.1%, $p < 0.001$). Despite this, they had shorter ICU stays and lower use of intensive supportive measures, including ventilator care and inotropic support, while demonstrating higher baseline RVSP values (Supplementary Table 3). In an additional sensitivity analysis that excluded very elderly patients (aged ≥ 85 years), younger age and follow-up RVSP remained associated with in-hospital mortality, and the overall direction of the association was preserved (Supplementary Table 4).

DISCUSSION

In this retrospective study of 153 patients with TC, the baseline characteristics were largely comparable across clinical settings; however, patients with secondary TC tended to have longer hospital and ICU stays and were more likely to require ventilator care or inotropic support, indicating greater overall illness severity. The prognosis of the primary form of TC was excellent, with no in-hospital deaths observed. All in-hospital deaths occurred in patients with the secondary form, most commonly infection-related cases, although the differences among the subgroups were not statistically significant. On multivariate analysis, younger age, bacteremia, and elevated follow-up RVSP were independently associated with in-hospital death. These findings suggest that systemic factors, especially infection-related stress and bacteremia, play crucial roles in determining adverse short-term outcomes in patients with TC.

Despite these differences in short-term outcomes being driven primarily by systemic illness severity, several baseline cardiac characteristics also varied across the TC subgroups. Primary TC showed higher NT-proBNP levels and larger left atrial size than the secondary TC subgroups. Previous atrial fibrillation was also more frequent in the primary TC group, whereas atrial tachyarrhythmia on the index ECG was not. These findings raise the possibility that differences in the underlying atrial remodeling or chronic cardiac substrates may have contributed to the observed subgroup differences. However, this interpretation remains speculative and does not explain the lack of in-hospital mortality in the primary TC group. In particular, NT-proBNP measurements were obtained at non-standardized clinical time points according to the context of TC diagnosis and may, therefore, reflect differences in illness stage or hemodynamic status rather than intrinsic subgroup-specific characteristics. In addition, follow-up NT-proBNP data were not systematically available, and neither previous atrial fibrillation nor left atrial volume index was significantly associated with in-hospital mortality in the univariate analysis.

Previous studies have reported that secondary TC tends to occur in critically ill patients and is associated with worse outcomes than the primary form [1,6,8]. In the International Takotsubo Registry, secondary TC was found to account for approximately one-third of all cases and shows markedly higher in-hospital mortality than the primary form, largely driven by comorbid medical conditions rather than the cardiomyopathy itself [6]. Similarly, de Chazal et al. [1] emphasized that secondary TC frequently develops in the setting of severe systemic illnesses such as sepsis, respiratory failure, or stroke, and that mortality in these patients is primarily attributable to the underlying disease. Consistent findings were reported by Citro et al. [11], who demonstrated significantly higher long-term mortality in secondary than in primary Takotsubo syndrome (13.1% vs. 2.8%) in a multicenter Italian registry. Consistent with these observations, our study demonstrated that all in-hospital deaths occurred in the secondary form, whereas patients with primary TC had an excellent short-term prognosis. Unlike previous studies that broadly categorized TC as primary or secondary, we further subdivided the secondary form according to its clinical context (infection, medical or surgical illness, and stroke) to provide a more granular understanding of outcome heterogeneity within secondary TC.

The highest mortality observed in infection-related cases likely reflects the systemic inflammatory and hemodynamic burden that accompanies critical illness, rather than intrinsic myocardial vulnerability. Systemic inflammation, cytokine release, and catecholamine surge associated with sepsis may exacerbate myocardial stunning, precipitate microvascular dysfunction, and increase pulmonary vascular resistance, which may explain the elevated RVSP observed in patients with poor outcomes [12-14]. Bacteremia, a marker of severe systemic inflammation [15,16], was also independently associated with in-hospital death, supporting the hypothesis that the severity of systemic stress, rather than cardiac structural damage, drives mortality in TC. Although older age is generally associated with adverse outcomes in cardiovascular disease [17], an inverse relationship between age and mortality was observed in the present study. A large nationwide registry reported that younger patients with Takotsubo syndrome exhibited more severe presentations, including a higher incidence of cardiogenic shock and mechanical support requirements but without increased in-hospital mortality [18]. In the present study, younger age was associated with in-hospital mortality in the multivariate analysis of secondary TC. However, these findings should be interpreted with caution. Patients excluded because of the absence of follow-up echocardiography were older and had substantially higher in-hospital mortality rates, raising the possibility of selection bias. In addition, age was not significantly associated with mortality in univariate analysis. Therefore, the observed association may reflect residual

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confounding factors, selection bias, or statistical instability related to the limited number of events. This finding should be considered hypothesis generating and requires confirmation in larger prospective studies.

Despite similar LVEF recovery across the TC forms, elevated follow-up RVSP was independently associated with in-hospital mortality. However, these findings should be interpreted with caution. The initial RVSP did not differ between survivors and non-survivors, whereas the follow-up RVSP was significantly higher in non-survivors. In addition, RVSP increased during follow-up in non-survivors but decreased slightly in survivors, and a greater increase in RVSP was associated with in-hospital mortality in the univariate analysis. These findings suggest that persistent or worsening elevation in RVSP during hospitalization may reflect an unresolved hemodynamic burden, severity of the underlying systemic illness, or incomplete cardiopulmonary recovery, rather than baseline pulmonary pressure alone. Elevated RVSP may reflect persistent pulmonary congestion, increased afterload from mechanical ventilation or sepsis-related pulmonary vasoconstriction, and right ventricular–pulmonary artery uncoupling [19-21], all of which may contribute to adverse outcomes in critically ill patients with TC. Previous studies have largely focused on left ventricular function in TC; the prognostic relevance of right-sided parameters has received limited attention [22]. Our results suggest that comprehensive echocardiographic assessment, including right ventricular and pulmonary hemodynamic indices, may provide additional prognostic information during follow-up evaluations, particularly in patients with secondary TC. Simultaneously, follow-up echocardiography was performed at nonstandardized time points, and factors that could influence RVSP measurements, such as ventilator settings, including positive end-expiratory pressure and diuretic treatment, were not systematically captured in this retrospective study. In addition, in secondary TC, particularly in patients with neurological or infectious triggers, clinical outcomes may be more strongly influenced by the severity of the underlying illness than by cardiac dysfunction alone. Therefore, the relationship between follow-up RVSP and mortality should be interpreted within a broader clinical context, and an elevated RVSP may serve as a marker of overall disease severity and cardiopulmonary interaction rather than as a direct determinant of mortality. Consistent with previous observations, these findings support the concept that secondary TC may represent a manifestation of systemic stress rather than a primary driver of mortality [6].

Collectively, these findings have important clinical implications. Differentiating between primary and secondary TC is clinically relevant because their pathophysiological mechanisms and prognostic profiles differ substantially. While primary TC, typically triggered by emotional or transient physical stress, usually follows a benign clinical course and requires supportive care, secondary TC develops in the context of severe systemic illnesses such as sepsis, respiratory failure, or neurologic injury, where management should primarily focus on treating the underlying condition rather than the cardiomyopathy itself. The observed association between elevated follow-up RVSP and mortality highlights the potential value of careful hemodynamic monitoring and right-sided cardiac assessment in the context of overall illness severity. Recognition of bacteremia as a marker of illness severity and systemic stress may help clinicians identify high-risk patients early and tailor management strategies accordingly.

This study had several limitations. First, this was a retrospective, single-center study with a relatively small sample size, which may have limited the generalizability of the findings. Second, the classification of TC into primary and secondary forms was based on the clinical context rather than predefined diagnostic criteria, and some degree of misclassification cannot be excluded. In addition, secondary TC subgroups were not independently associated with mortality after adjustment, possibly reflecting the limited statistical power and overlap with more specific markers of systemic illness severity. Third, echocardiographic follow-ups were not performed at standardized time intervals but according to clinical needs, which may have introduced a selection bias. Follow-up imaging tended to occur earlier in patients with clinical deterioration, suggesting that RVSP may partly reflect the overall illness severity rather than being an isolated cardiac determinant. Furthermore, the multivariate analysis was restricted to patients who underwent follow-up echocardiography, and exclusion of those without follow-up imaging may have led to an underestimation of mortality and distortion of the associations between follow-up echocardiographic parameters and outcomes. In particular, patients without follow-up imaging had a higher in-hospital mortality, suggesting that early death or limited treatment intensity may have reduced the opportunity for repeat echocardiography. Fourth, we focused on in-hospital outcomes and did not assess the long-term prognosis. Fifth, the limit-

ed number of outcome events raises the possibility of model overfitting, despite efforts to limit the number of predictors. Sixth, because coronary evaluation was not performed in all patients, particularly critically ill patients with secondary TC, misclassification of other causes of myocardial dysfunction cannot be fully excluded. Nevertheless, because the study included patients whose regional wall motion abnormalities, unrelated to a specific coronary artery territory, were completely resolved on follow-up echocardiography, the overall findings are likely to reflect real-world clinical practice. Seventh, the study period spanned more than a decade, during which advances in critical care and cardiovascular management may have influenced the outcomes, and residual confounding cannot be excluded. Eighth, the impact of heart failure medications on left ventricular recovery and mortality could not be assessed because medication data were unavailable. Finally, the study relied on routinely collected clinical and conventional echocardiographic parameters and did not incorporate advanced imaging modalities such as strain analysis or cardiac magnetic resonance imaging, nor did it incorporate any novel circulating biomarkers. Therefore, these findings should be interpreted with caution, and hypotheses should be considered, warranting confirmation in larger prospective studies.

In conclusion, our findings suggest that in this single-center cohort, in-hospital outcomes of secondary TC appear to be more closely related to markers of systemic illness severity and right ventricular–pulmonary hemodynamics than to left ventricular systolic function, warranting confirmation in larger prospective studies.

KEY MESSAGE

1. Clinical characteristics and in-hospital outcomes of takotsubo cardiomyopathy differed according to the clinical context.
2. In-hospital mortality occurred exclusively in secondary takotsubo cardiomyopathy, with the highest rate in infection-related cases, although the between-group difference was not statistically significant.
3. Age, bacteremia, and follow-up right ventricular systolic pressure were independently associated with in-hospital mortality.
4. Clinical characteristics and in-hospital outcomes of takotsubo cardiomyopathy differed according to the clinical context.

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Supplementary Table 1. Comparison of clinical severity markers between younger and older patients with Takotsubo syndrome

Variables	Age < 65 years (n = 46)	Age ≥ 65 years (n = 107)	*p* value
Age, yr	46.5 ± 14.5	77.9 ± 6.4	< 0.001
Female	30 (65.2)	68 (63.6)	0.989
Secondary form	32 (69.6)	90 (84.1)	0.067
Infection	12 (26.1)	46 (43.0)	0.073
Medical or surgical illness	9 (19.6)	29 (27.1)	0.432
Stroke	11 (23.9)	15 (14.0)	0.208
Hospital stay, days	15.0 [6.0–51.0]	19 [10.0–32.0]	0.997
ICU care	38 (82.6)	79 (73.8)	0.334
ICU stay, days	7.0 [3.3–17.8]	7.5 [4.0–15.0]	0.960
Ventilator care	25 (54.3)	45 (42.1)	0.477
Inotropic use	31 (67.4)	61 (57.0)	0.765
Bacteremia	12 (26.1)	23 (21.5)	0.682
Initial LVEF, %	41.4 ± 8.8	41.4 ± 8.2	0.962
Follow-up LVEF, %	61.5 ± 9.6	57.9 ± 11.6	0.070
Initial RVSP, mmHg	36.7 ± 11.7	39.8 ± 13.2	0.192
Follow-up RVSP, mmHg	33.1 ± 9.0	37.7 ± 12.8	0.016
In-hospital death	7 (15.2)	13 (12.1)	0.799

Data are presented as number (%), mean ± standard deviation, or median [interquartile range].
ICU, intensive care unit; LVEF, left ventricular ejection fraction; RVSP, right ventricular systolic pressure.

Supplementary Table 2. Comparison of clinical severity markers between in-hospital survivors and non-survivors

Variables	In-hospital survivor (n = 133)	In-hospital non-survivor (n = 20)	*p* value
Age, yr	69.2 ± 17.2	63.9 ± 18.1	0.203
Age ≥ 65 yr	94 (70.7)	13 (65.0)	0.799
Female	88 (66.2)	10 (50.0)	0.248
Secondary form	102 (76.7)	20 (100.0)	0.034
Infection	46 (34.6)	12 (60.0)	0.053
Medical or surgical illness	33 (24.8)	5 (25.0)	1.000
Stroke	23 (17.3)	3 (15.0)	1.000
Hospital stay, days	18.5 [8.8–40.5]	16.0 [10.8–24.3]	0.415
ICU care	99 (74.4)	18 (90.0)	0.212
ICU stay, days	7.0 [3.0–15.0]	12.0 [7.3–20.0]	0.051
Ventilator care	54 (40.6)	16 (80.0)	0.013
Inotropic use	75 (56.4)	17 (85.0)	0.142
Bacteremia	25 (18.8)	10 (50.0)	0.005
Timing of follow-up echocardiography, days	12.0 [6.0–54.0]	8.5 [5.5–11.8]	0.065
Initial LVEF, %	42.3 ± 7.7	35.2 ± 10.1	< 0.001
Follow-up LVEF, %	59.6 ± 10.2	55.0 ± 15.9	0.220
Initial RVSP, mmHg	39.0 ± 12.5	38.5 ± 15.3	0.883
Follow-up RVSP, mmHg	35.1 ± 10.6	45.2 ± 17.1	0.025
Change in RVSP, mmHg[a)]	-2.8 ± 15.1	5.8 ± 17.6	0.033

Data are presented as number (%), mean ± standard deviation, or median [interquartile range].
ICU, intensive care unit; LVEF, left ventricular ejection fraction; RVSP, right ventricular systolic pressure.
[a)]Calculated as follow-up RVSP minus initial RVSP.

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Supplementary Table 3. Comparison of clinical characteristics between patients with and without follow-up echocardiography

Variables	Without echocardiography follow-up (n = 131)	With echocardiography follow-up (n = 153)	*p* value
Age, yr	73.9 ± 12.8	68.5 ± 17.3	0.002
Female	82 (62.6)	98 (64.1)	0.896
Hospital stay, days	10.0 [5.0–23.5]	18.0 [9.0–37.0]	< 0.001
ICU care	93 (71.0)	117 (76.5)	0.361
ICU stay, days	4.0 [2.0–8.0]	7.0 [4.0–16.3]	< 0.001
Ventilator care	49 (37.4)	70 (45.8)	0.001
Inotropic use	65 (49.6)	92 (60.1)	< 0.001
Initial LVEF, %	42.7 ± 10.8	41.4 ± 8.4	0.244
Initial RVSP, mmHg	44.3 ± 15.3	38.9 ± 12.8	0.003
In-hospital death	48 (36.6)	20 (13.1)	< 0.001

Data are presented as number (%), mean ± standard deviation, or median [interquartile range].
ICU, intensive care unit; LVEF, left ventricular ejection fraction; RVSP, right ventricular systolic pressure.

Supplementary Table 4. Sensitivity analysis of predictors for in-hospital death after excluding patients aged ≥ 85 years

Variables	OR (95% CI)	*p* value
Age, yr	0.937 (0.890–0.986)	0.012
Ventilator care	2.165 (0.295–15.887)	0.448
ESR	1.042 (0.998–1.089)	0.063
Bacteremia	5.412 (0.938–31.234)	0.059
Initial LVEF	0.892 (0.781–1.019)	0.093
Follow-up LVEF	0.956 (0.888–1.030)	0.238
Follow-up RVSP	1.129 (1.044–1.221)	0.002

Values are presented as odds ratios (ORs) with 95% confidence intervals (CIs) derived from multivariable logistic regression analysis.
ESR, erythrocyte sedimentation rate; LVEF, left ventricular ejection fraction; RVSP, right ventricular systolic pressure.

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