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Original Article

A Comprehensive Evaluation of Clinical Variables and Their Association with in Hospital Mortality in Infective Endocarditis Cases

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Abstract

Objectives: Infective endocarditis (IE) remains a significant clinical challenge due to its high morbidity and mortality rates. Prognosis is influenced by patient characteristics, causative microorganisms, complications, and echocardiographic findings. The Barcelona Bio-Heart Failure (BCN Bio-HF) Risk Score, widely used in heart failure, has not yet been evaluated in IE. This study aimed to assess the association between the BCN score and in-hospital mortality in patients with IE.

Methods: This retrospective, single-center observational study included 108 patients diagnosed with IE. Patients were divided into two groups based on the occurrence of in-hospital mortality, which was defined as any death occurring during hospitalization. Clinical, demographic, laboratory, and echocardiographic data were compared between the groups.

Results: Among 108 patients, 29 (26.9%) experienced in-hospital mortality. Compared to survivors, non-survivors were older (p=0.046) and had a higher prevalence of chronic kidney disease (p=0.041). *Staphylococcus aureus* and methicillin-resistant strains were more common in the mortality group (p=0.043 and p=0.04, respectively). Echocardiographic findings showed a lower ejection fraction (p=0.03), higher pulmonary artery systolic pressure (p=0.017), and larger vegetation size (p=0.033) in non-survivors. Mechanical complications were also more frequent (p=0.017). Laboratory results revealed lower hemoglobin (p=0.012) and higher levels of WBC (p=0.035), CRP (p=0.01), procalcitonin (p=0.02), NT-proBNP (p=0.008), and BCN Bio-HF risk score (p=0.008) in patients with in-hospital mortality.

Conclusion: The BCN Bio-HF risk score showed potential in predicting in-hospital mortality in infective endocarditis by integrating clinical and biomarker data. Future studies should aim to develop IE-specific prognostic models incorporating dynamic clinical variables and novel biomarkers to enhance risk stratification and guide management.

Keywords: Barcelona bio-heart failure risk score; infective endocarditis; mortality.

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Enfektif Endokardit Olgularında Klinik Değişkenlerin Hastane İçi Mortalite ile İlişkisinin Kapsamlı Analizi

Özet

Amaç: Enfektif endokardit (EE), yüksek morbidite ve mortalite oranları nedeniyle önemli bir klinik sorun olmaya devam etmektedir. Prognoz; hasta özellikleri, etken mikroorganizmalar, gelişen komplikasyonlar ve ekokardiyografik bulgulara bağlıdır. Kalp yetersizliğinde yaygın olarak kullanılan Barcelona Bio-Heart Failure (BCN Bio-HF) Risk Skoru, EE hastalarında henüz değerlendirilmemiştir. Bu çalışmanın amacı, BCN Bio-HF risk skorunun EE hastalarında hastane içi mortalite ile ilişkisini incelemektir.

Gereç ve Yöntem: Bu retrospektif, tek merkezli gözlemsel çalışmaya EE tanısı alan 108 hasta dahil edilmiştir. Hastalar, hastane içi mortalite gelişimine göre iki gruba ayrılmıştır. Hastane içi mortalite, hastanede yatış süresince meydana gelen tüm ölüm olayları olarak tanımlanmıştır. Gruplar arasında klinik, demografik, laboratuvar ve ekokardiyografik veriler karşılaştırılmıştır.

Bulgular: Toplam 108 hastanın 29'unda (%26,9) hastane içi mortalite gelişmiştir. Mortalite gelişen hastalar, sağ kalanlara göre daha ileri yaşta (p=0,046) ve daha yüksek oranda kronik böbrek hastalığına sahipti (p=0,041). Mortalite grubunda *Staphylococcus aureus* ve metisiline dirençli suşlar daha sık izlenmiştir (sırasıyla p=0,043 ve p=0,04). Ekokardiyografik değerlendirmede, mortalite grubunda ejeksiyon fraksiyonu daha düşük (p=0,03), pulmoner arter sistolik basıncı daha yüksek (p=0,017) ve vejetasyon boyutu daha büyük bulunmuştur (p=0,033). Mekanik komplikasyonlar da bu grupta daha sık görülmüştür (p=0,017). Laboratuvar bulguları, mortalite gelişen hastalarda daha düşük hemoglobin düzeyi (p=0,012) ve daha yüksek lökosit (p=0,035), CRP (p=0,01), prokalsitonin (p=0,02), NT-proBNP (p=0,008) ve BCN Bio-HF skoru (p=0,008) göstermiştir.

Sonuç: BCN Bio-HF risk skoru, klinik ve biyobelirteç verilerini entegre ederek EE'de hastane içi mortaliteyi öngörmede potansiyel bir araç olarak ortaya çıkmıştır. Gelecekte yapılacak çalışmalar, dinamik klinik değişkenler ve yeni biyobelirteçleri içeren EE'ye özgü prognostik modellerin geliştirilmesine odaklanmalıdır.

Anahtar sözcükler: Barcelona bio-heart failure risk score; enfektif endokardit; mortalite.

Introduction

Infective endocarditis (IE) continues to pose a significant challenge in cardiology, with persistently high morbidity and mortality rates despite advancements in treatment. Hospital mortality rates in IE patients remain between 15% and 30%, influenced by factors such as underlying health conditions, the causative pathogen, and the presence of complications.[1,2] Early identification of patients at highest risk for mortality offers an opportunity for more aggressive treatment and closer monitoring, potentially improving outcomes. The prognosis of IE is primarily influenced by four key factors: patient characteristics, the presence of cardiac and non-cardiac complications, the causative microorganism, and echocardiographic findings. Critical prognostic indicators include prosthetic valve involvement, advanced age, septic shock, neurological complications, history of hemodialysis, heart failure (HF), periannular abscesses, and infections caused by *Staphylococcus aureus*.[1,3] Additionally, comorbidities such as insulin-dependent diabetes, impaired left ventricular function, and stroke have been identified as significant predictors of poor outcomes during hospitalization.[4–6]

Echocardiography has also been explored as a predictive tool in IE, particularly regarding the size of vegetations and their correlation with the risk of embolic events. In addition to these echocardiographic findings, several IE-specific risk scores have been developed and validated to further predict mortality risk. These include the STS (The Society of Thoracic Surgeons)-IE score.[7] and the risk prediction model proposed in the study by De Feo et al. (2012)[8] for native valve IE surgery, all of which integrate microbiological, clinical, and echocardiographic data. These scores have shown strong calibration in predicting outcomes by incorporating established factors that significantly affect surviva.[9]

In HF patients, several risk scores have been developed and extensively utilized to predict outcomes such as mortality, hospital admissions, and overall prognosis. These scores assess the severity and progression of HF, providing valuable insights into patient management. Additionally, clinical and laboratory parameters that reflect the condition's development contribute to the accuracy and utility of these scores in guiding treatment decisions and forecasting patient outcomes. Given the similarity in prognostic factors between HF and IE, the potential utility of general HF risk scores in predicting mortality for IE patients may become an important area of investigation, particularly considering their established value in HF prognosis.

In patients with HF, various scoring models have been utilized to predict adverse outcomes by considering a broad range of demographic, clinical, therapeutic, and laboratory parameters. The Barcelona Bio-Heart Failure (BCN Bio-HF) Risk Score is a widely accepted tool for stratifying mortality risk in advanced HF.[10] However, despite its proven value in HF, the BCN Bio-HF Risk Score has yet to be evaluated for its prognostic ability in IE. Given the overlap in prognostic factors between HF and IE, we aimed to assess the reliability of the BCN Bio-HF Risk Score in predicting in-hospital mortality among IE patients, expanding its potential application in this clinical context.

Materials and Methods

Study Population

A total of 129 patients diagnosed with IE according to the Duke criteria[11] at the Cardiology Clinic of Basaksehir Cam & Sakura City Hospital between 2020 and 2024 were included in the study. A single-center, retrospective observational study was performed. The study encompassed patients with IE affecting either natural or prosthetic valves, while cases related to cardiac implantable electronic devices were excluded. Patients under the age of 18 and those with incomplete laboratory data were also excluded from the study. The final group consisted of 108 individuals from whom fundamental demographic information, cardiovascular history, physical examination results, clinical risk factors, treatment characteristics, echocardiography findings, and laboratory results were obtained. Patients were divided into two groups based on the development of in-hospital mortality, and clinical, demographic, echocardiographic, and laboratory data were compared between the groups. The study adhered to the principles specified in the Helsinki Declaration for biomedical research involving human subjects. The study protocol received approval from the Clinical Research Ethics Committee (Basaksehir Cam and Sakura City Hospital Ethics Committee, Number and Date: 191/28.08.2024).

Imaging

All patients underwent transthoracic and transesophageal echocardiography. The diameters and functions of the cardiac chambers, as well as the evaluation of valve insufficiency and stenosis, were measured and calculated according to current guideline recommendations. Echocardiographically, the vegetation was described as an irregular and echogenic mass adhered to a valve or the myocardial surface.[12] Additionally, each patient was evaluated for mechanical complications such as paravalvular regurgitation, dehiscence, abscess, and leaflet perforation. A paravalvular abscess was defined as an infection and necrosis resulting in a purulent cavity capable of invading surrounding structures.[12] Dehiscence is typically identified by observing a prosthetic valve moving with an excursion of at least 15° in any direction.[13] Paravalvular regurgitation is the backward flow of blood around a valve due to structural damage caused by infection or other complications, typically observed as an eccentric regurgitant jet adjacent to the valve. Patients were evaluated with TTE and TEE as needed. FDG-PET/CT (18F-fluorodeoxyglucose positron emission tomography/computed tomography) was utilized in cases of possible PVE to identify valvular lesions and confirm the diagnosis of IE.

Laboratory Parameters

Hemogram, CRP, procalcitonin, sedimentation, and routine laboratory parameters were obtained multiple times from all patients at both admission and follow-up. Blood cultures were drawn from at least three distinct venipuncture locations. In patients with no growth in cultures, repeated blood cultures were obtained.

Treatment and Outcomes

All patients were initiated on appropriate antibiotic therapy and supportive treatment according to the guidelines. Treatment was adjusted based on culture results or clinical progression. In cases of AKI and heart failure development during the clinical course, patients were managed with appropriate treatment options. Clinical complications, including acute HF, acute renal insufficiency, and peripheral embolization, were meticulously documented. HF was diagnosed based on established guideline criteria.[14] New onset of septic emboli, including cerebral, pulmonary, or systemic embolism, and neurological complications, including ischemic stroke, intracerebral or subarachnoidal hemorrhage, were identified through computed tomography (CT) and/or magnetic resonance imaging (MRI). Surgical interventions were conducted in accordance with current guidelines.[14] In our study, we followed the guidelines and recommended cardiac surgery without delay in patients who experienced a transient ischemic attack, when indicated. In patients who suffered a stroke, surgery was performed promptly in the presence of heart failure, uncontrolled infection, abscess, or persistent high embolic risk, as long as coma was absent. Nevertheless, some patients received conservative pharmacological management due to either severe medical conditions or prohibitively high surgical risks.

Patients who had no culture growth after treatment, with laboratory parameters normalizing and no clinically significant valve dysfunction on echocardiography, were classified as having recovered. Additionally, patients whose laboratory and echocardiographic parameters returned to normal after surgery were also considered to have recovered. In-hospital mortality was defined as death occurring during the hospitalization for the treatment of IE, regardless of whether the cause of death was directly related to endocarditis or associated complications. This includes deaths resulting from septic shock, heart failure, stroke, or other severe complications arising during hospitalization.

Prognostic Score

The in-hospital mortality risk was assessed using the BCN Bio-HF Calculator in our study. The BCN Bio-HF Calculator comprises a model with 15 predictors, including 7 clinical and laboratory variables, 5 treatment-related variables, and 3 biomarker-related variables.[15] The BCN Bio-HF risk score was calculated for each patient using these factors, which were obtained from the patients' medical records. An online calculator is accessible at http://ww2.bcnbiohfcalculator.org/web/calculations. In our study, BCN Bio-HF risk scores were compared between patients who had in-hospital mortality and those who survived.

Statistical Analysis

The distributional characteristics of the variables were assessed using the Kolmogorov–Smirnov test and further verified through visual inspection of histograms and probability plots. Continuous variables were summarized as mean±standard deviation for normally distributed data and as median with interquartile range (IQR 25–75) for data not following a normal distribution. Between-group comparisons of continuous variables were conducted using either the independent Student's t-test or the Mann–Whitney U test, based on the distribution. Categorical variables were expressed as absolute numbers and percentages. Comparisons of categorical data across groups were carried out using the chi-square (χ^2) test or Fisher's exact test, as appropriate. A two-tailed P-value of less than 0.05 was considered indicative of statistical significance. All statistical analyses were performed using R statistical software (version 4.1.3, Vienna, Austria).

Results

The study included 108 patients, with a mean age of 54.8±15.2 years; 64.8% were male. Patients were divided into two groups based on the presence or absence of in-hospital mortality. Group 2 consisted of patients who experienced in-hospital mortality (n=29).

When comparing the two groups, age was significantly higher in Group 2 [52 (44–64) vs. 59 (53–67.5) years, p=0.046], as was the presence of chronic kidney disease (11.4% vs. 27.6%, p=0.041). In terms of causative microorganisms, *Staphylococcus aureus* and methicillin-resistant strains were more common in Group 2 (p=0.043 and p=0.04, respectively). Echocardiographic parame-

Table 1. Baseline demographic and clinic parameters of study population

	Group 1 (survivors) n=79		Group 2 (non-survivors) n=29		p
	n	%	n	%	
Age (years)	52 (44-64)		59 (53-67.5)		0.046
Gender, male	53	67.1	17	58.6	0.41
BMI, kg/m^2	27.4±3.8		26.7±4		0.47
Height, cm	164±8		166±9		0.39
Weight, kg	73.8±12		73.4±13.2		0.91
Medical history					
CAD	17	21.5	6	20.7	0.92
CHF	14	17.7	8	27.6	0.26
DM	15	19	10	34.5	0.091
Hypertension	45	57	15	51.7	0.63
Smoking	12	15.2	5	17.2	0.79
Hyperlipidemia	17	21.5	6	20.7	0.93
PAD	5	6.3	2	6.9	0.25
Atrial fibrillation	12	15.2	5	17.2	0.79
Prior neurological events	5	6.3	3	10.3	0.48
Chronic kidney disease	9	11.4	8	27.6	0.041
Hemodialysis	6	7.6	4	13.8	0.33
Intravenous drug users	8	10.1	4	13.8	0.59
HIV/AIDS	0	0	1	3.1	0.10
Involvement					
Prosthetic valve	19	24.4	10	34.5	0.295
Native valve	59	75.6	19	65.5	0.295
Aortic involvement	31	39.2	10	34.5	0.652
Mitral involvement	35	44.3	13	44.8	0.961
Right side involvement	5	6.3	3	10.3	0.480
Multivalvular involvement	10	8.1	5	17.2	0.314
Etiological risk factors					
Degenerative valvular disease	7	8.3	3	10.3	0.814
Rheumatic heart disease	9	11.4	3	10.3	0.878
Prosthetic heart disease	19	24.4	10	34.5	0.295
Previous infective endocarditis	8	10.1	4	13.8	0.591
Congenital heart disease	9	11.4	4	13.8	0.734
Unknown	34	43	9	31	0.259
Microorganisms					
Culture-negative	24	30.4	9	31	0.948
Staphylococcus spp.	21	26.6	12	41.4	0.139
Staphylococcus aureus	13	16.5	10	34.5	0.043
S.aureus Methicillin resistance	9	11.4	8	27.6	0.041
Streptococcus spp.	14	17.7	2	6.9	0.160
Enterococcus spp.	8	10.1	2	6.9	0.608
Candida	5	6.3	5	17.2	0.083
Pseudomonas	3	3.8	1	3.4	0.932
Brucella	1	1.3	0	0	0.543
Others	4	5.1	2	6.9	0.665

BMI: Body mass index; CAD: Coronary artery disease; CHF: Congestive heart failure; DM: Diabetes mellitus; PAD: Peripheral artery disease; HIV/AIDS: Human immunodeficiency virus / acquired immunodeficiency syndrome; *S. aureus: Staphylococcus aureus.*

ters revealed that Group 2 had a significantly lower ejection fraction [56% (45–66) vs. 45% (35–65), p=0.03], higher pulmonary artery systolic pressure (PASP) [30 mmHg (28–41) vs. 35 mmHg (30–41), p=0.017], and larger vegetation size [8 mm (7–13) vs. 13 mm (8.5–15), p=0.033]. Mechanical complications were also more frequently observed in Group 2 (p=0.017) (Table 1).

Regarding laboratory findings, patients in Group 2 had significantly lower hemoglobin levels [11.2 g/dL (10.5–12.2) vs. 10 g/dL (8.4–12.1), p=0.012], and significantly higher levels of white blood cell count [8.1 (5.5–12.5) vs. 13.5 (7.1–17.1), p=0.035], C-reactive protein (CRP) [52.9 mg/dL (30.6–134.2) vs. 105 mg/dL (88–145.5), p=0.01], procalcitonin [0.41 ng/mL

Table 2. Echocardiographic and laboratory findings of both groups

	Group 1 (survivors) n=79		Group 2 (non-survivors) n=29		p
	n	%	n	%	
Echocardiographic findings					
EF (%)	56 (45–66)		45 (35–65)		0.03
PAPs (mmHg)	30 (28–41)		35 (30–41)		0.017
Vegetation size (mm)	8 (7–13)		13 (8.5–15)		0.033
Complications					
Mechanical complications	15	19	12	41.4	0.017
Dehiscence	2	2.5	2	6.9	0.287
Fistula	2	2.5	2	6.9	0.287
Pseudoaneurysm	1	1.3	1	3.4	0.956
Abscess	3	3.8	2	6.9	0.497
Significant Valve dysfunction	8	10.1	7	24.1	0.062
Stroke	3	3.8	5	17.2	0.018
Laboratory findings					
Hg, g/dL	11.2 (10.5–12.2)		10 (8.4–12.1)		0.012
Platelet count, 10^3/μL	231±104		261±131		0.214
WBC count, 10^3/μL	8.1 (5.5–12.5)		13.5 (7.1–17.1)		0.035
BUN, mg/dL	49.9 (31.9–83)		60.5 (29–101.3)		0.264
Creatinine, mg/dL	0.9 (0.81–2.41)		1.3 (0.96–1.47)		0.073
AST (IU/mL)	15 (10–35)		16 (7–38)		0.997
ALT (IU/mL)	23 (16–39)		21 (14.5–54)		0.928
C-reactive protein, mg/dL	52.9 (30.6–134.2)		105 (88–145.5)		0.001
Procalcitonin (ng/mL)	0.41 (0.1–3.6)		2.45 (1.03–3.4)		0.020
NT-proBNP, pg/mL	1026 (409–6860)		5654 (2006–8254)		0.008
Troponin I, ng/mL	70 (22–198)		140 (41.5–278.5)		0.063
BCN Bio-HF risk score	15.3 (6.3–26.6)		21 (13.4–41.9)		0.008

EF: Ejection fraction; PAPs: Pulmonary artery systolic pressure; Hg: Hemoglobin; WBC: White blood cell; BUN: Blood urea nitrogen; AST: Aspartate aminotransferase; ALT: Alanine aminotransferase; NT-proBNP: N-terminal pro-B-type natriuretic peptide; BCN Bio-HF: Barcelona bio-heart failure.

(0.1–3.6) vs. 2.45 ng/mL (1.03–3.4), p=0.02], and NT-proBNP [1026 pg/mL (409–6860) vs. 5654 pg/mL (2006–8254), p=0.008]. Additionally, the median BCN Bio-HF risk score was significantly higher in the mortality group [15.3 (6.3–26.6) vs. 21 (13.4–41.9), p=0.008] (Table 2).

Discussion

In this study, in addition to the clinical, microbiological, echocardiographic, and laboratory parameters traditionally used to evaluate in-hospital mortality in patients with IE, the BCN Bio-HF risk score—originally developed for heart failure populations—was also found to be statistically significant in predicting in-hospital mortality in the IE group.

In recent years, numerous risk factors have been identified as significant predictors of both in-hospital and long-term mortality, as well as for evaluating surgical risk in patients with IE. However, no single factor has proven to be sufficiently predictive. This limitation arises due to the dynamic nature of IE, in which clinical, microbiological, and echocardiographic findings continuously evolve throughout the active phase of the disease. As a result, a comprehensive and multidimensional approach to risk assessment is essential.

Previously, several studies have combined multiple risk factors to create new scoring systems, with the goal of enhancing the accuracy of mortality predictions and improving clinical decision-making. For instance, one study evaluating 1,293 consecutive IE patients utilized the ACEF score, which combines age, creatinine levels, and left ventricular ejection fraction (LVEF).[16] The ACEF score was designed to estimate mortality risk in elective cardiac surgeries, and it was found that this score was independently associated with in-hospital and long-term mortality. The ACEF score was also considered suitable for assessing the risk of in-hospital mortality in IE patients undergoing surgery.

Another study assessed the EuroSCORE in predicting operative mortality in patients with native valve endocarditis.[17] The EuroSCORE showed strong discriminatory power and proved useful for identifying high-risk patients who may require surgical intervention. However, this score was originally formulated for general cardiac surgeries and was not specifically tailored to the unique challenges of IE.

In another study evaluating the severity of illness during the acute phase of IE, researchers used the Acute Physiology, Age, Chronic Health Evaluation II (APACHE II) score at the time of presentation to assess patient outcomes.[4] The results re-

vealed significant differences in APACHE II scores between in-hospital survivors and non-survivors, with patients diagnosed with *Staphylococcus aureus*-related IE exhibiting markedly higher scores compared to those with non-S. aureus etiology. The APACHE II score, primarily used to assess the severity of illness in critically ill patients, helps clinicians estimate mortality risk by integrating a variety of clinical data, offering a more comprehensive view of the patient's condition.

Similarly, HF prognostic scores take into account other comorbidities—such as renal dysfunction, diabetes, and prior cardiovascular events—which are essential for understanding the complex clinical picture of IE patients and guiding effective risk stratification. Despite the multitude of studies examining prognostic scores for IE, no single tool has proven to be ideal, and their clinical utility often falls short of expectations. In this study, we sought to investigate HF scores that incorporate a broader range of demographic, clinical, therapeutic, and laboratory data to determine their potential for more accurate risk stratification in IE patients.

The BCN Bio-HF risk calculator, developed by Lupón et al.[18] in 2014, was based on data from 864 consecutive outpatients at a multidisciplinary HF unit and is designed to predict all-cause mortality and hospitalization rates for HF patients over a 1- to 5-year period. The model incorporates 11 clinical variables, including age, gender, NYHA functional class, left ventricular ejection fraction (LVEF), serum sodium levels, hemoglobin, estimated glomerular filtration rate (eGFR), use of β-blockers, ACE inhibitors/angiotensin II receptor blockers, loop diuretic dosage, and statin therapy. Additionally, it integrates three key serum biomarkers: proBNP, high-sensitivity cardiac troponin T (hs-cTnT), and high-sensitivity soluble ST2 (sST2).[6]

Our study demonstrates that the BCN Bio-HF Risk Calculator effectively predicted in-hospital mortality with a high level of performance. This superior predictive capability may be attributed to the inclusion of biomarkers such as troponin and BNP, which have been increasingly recognized for their prognostic significance. Although cardiac troponins are traditionally used to diagnose myocardial infarction, recent studies utilizing high-sensitivity assays have highlighted their broader prognostic value in a variety of clinical settings. Elevated troponin levels are now acknowledged as reliable markers of cardiac injury and as predictors of adverse outcomes in conditions such as HF, valvular heart disease, septic shock, and non-cardiac surgeries.[19–21] Patients with IE represent a unique patient group, often presenting with a combination of these conditions. However, there is limited research on the role of troponin as an early prognostic biomarker in IE. Some studies have suggested that elevated troponin levels correlate with poorer outcomes in these patients.[22]

For instance, a study found that patients with high cTnI levels were less likely to have isolated right-sided IE and more likely to experience left ventricular systolic dysfunction or renal impairment. Elevated cTnI levels were also associated with a higher risk of severe complications, including mortality, abscess formation, and neurological events.[23] Similarly, elevated BNP levels have been linked to increased morbidity and mortality in IE patients, particularly when combined with elevated cTnI levels. One study demonstrated that high BNP levels were strongly associated with the composite outcome of mortality and the development of intracardiac abscesses, underscoring the importance of these biomarkers in predicting severe IE-related outcomes.[24]

The strength of the BCN Bio-HF Calculator lies not only in its comprehensive integration of clinical variables but also in its development using data from a cohort of patients actively monitored within multidisciplinary HF programs. Furthermore, the tool is regularly updated to reflect the prognostic benefits of new HF medications and devices, ensuring it aligns with current treatment practices. This ongoing refinement enhances the reliability and relevance of the BCN Bio-HF Calculator as a risk assessment tool, particularly in multidisciplinary clinics, where it integrates the latest clinical advancements.[25]

Limitations

This study has several limitations that should be acknowledged. Firstly, the single-center and retrospective design limits the generalizability of the findings, which may reduce the applicability of the results to a broader population. Additionally, the relatively small sample size may diminish the statistical power of the study and limit the robustness of the conclusions. The risk scores used in this study also do not incorporate some novel prognostic factors. Another limitation is the variability in clinical and echocardiographic characteristics during the fluctuating course of IE. Due to the disease's dynamic nature, changes over time may affect the accuracy of risk assessments, potentially impacting the reliability of the scoring systems used.

Conclusion

This study highlights the potential utility of the BCN Bio-HF score in predicting in-hospital mortality among patients with infective endocarditis (IE), demonstrating promising performance through its integration of clinical, therapeutic, and biomarker data. The dynamic and multifactorial nature of IE necessitates a more tailored approach to risk assessment. Therefore, future research should focus on developing and validating comprehensive, IE-specific prognostic models that incorporate evolving clinical variables and novel biomarkers to improve risk stratification and guide treatment strategies more effectively.

Disclosures

Ethics Committee Approval: The study was approved by the Basaksehir Cam and Sakura City Hospital Clinical Research Ethics Committee (no: 191, date: 28/08/2024).

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