

A COMPARATIVE STUDY OF RISK STRATIFICATIONS SCORES FOR ACUTE HEART FAILURE PATIENTS AND ITS OUTCOME IN THE EMERGENCY DEPARTMENT, EGYPT

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ABSTRACT Introduction: Diagnosis of heart failure according to Framingham criteria which are. A) Major criteria. B) Minor criteria for establishing a definite diagnosis of HF, two major or one major and two minor criteria had to be present. In this study two risk stratification models will be used among heart failure patients who present to the emergency room, The Seattle heart failure model and the Canadian predictor mortality of HF model. **Aim:** comparison will be done according to accuracy and easiness in predicting the risk of death in 48hr and 28day from hospital admission among HF patient. **Methods:** This study is cross-sectional, observational study on adults presenting to the emergency department with Acute Heart Failure with short-term follow-up in cardiology care unit (CCU) and Cardiology inpatient ward for MACE. Patients will be divided into two groups. Each group will include 30 patients their cardiac risk determined by one of the two methods of risk stratification utilizing findings on presentation with timeframe outcome 48hr and 28 day. **Results:** The studied patients in Canadian model were 20.0%, acute renal failure was 23.3%, stroke was 10.0%, the cardiogenic shock was 36.7%, and significant arrhythmia was 10.0%. While according to Seattle heart failure model, 57% of the patients were at very high risk, 16.7% were at high risk, 16.7% were at intermediate risk, and 10% were at low risk of mortality **Conclusion:** Sensitivity, specificity, and +PV of Seattle heart failure model were significantly higher than Canadian predictive model with a very high area under the ROC curve (AUC) and very low standard error (SE).

KEYWORDS: heart failure, risk stratifications

HOW TO CITE THIS ARTICLE

Elbaih AH, Elzeky EA, Elshaboury I, Oraby M. A Comparative Study of Risk Stratifications Scores for Acute Heart Failure Patients in the Emergency Department, Egypt. *Int J Surg Med.* 2017; 3(3): 140-149. doi:10.5455/ijsm.acute-heart-failure

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DOI:10.5455/ijsm.acute-heart-failure

First Received: December 24, 2016

Accepted: March 02, 2017

Manuscript Associate Editor: George Baitchev (BG)

Editor-in Chief: Ivan Inkov (BG)

Reviewers: Mehmet Ali Karaca (TR);

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Introduction

Heart failure (HF) can be defined as an abnormality of cardiac structure and function leading to failure of the heart to deliver oxygen at a rate commensurate with the requirement of the metabolizing tissues [1].

Approximately 1-2% of adult population in developed countries has HF with the prevalence raising to more than 10% among

persons 70 years of age or older. About 26 million adults worldwide are living with heart failure leading some to describe it as a global pandemic. In Egypt, the prevalence of heart failure increased dramatically in the second half of the 20th century as cardiovascular diseases have quadrupled in less than three decades and increased the prevalence of hypertension in adult Egyptians 26.3% and diabetes 9.3% and approximately 47% of the nation mortality due to HF and cardiovascular problems. As a primary diagnosis, heart failure accounts for about 1-4% of all hospital admissions in economically developed countries [2].

The definition of HF includes typical signs: elevated jugular venous pressure, hepatojugular reflux, third heart sound (gallop rhythm), laterally displaced apical impulse, cardiac murmur, and less typical signs: peripheral edema(ankle, sacral, scrotal.), pulmonary crepitations, reduced air entry and dullness to percussion at lung bases, tachycardia, irregular pulse , tachypnea >16 breaths/min, hepatomegaly, ascites,tissue wasting(cachexia) [3].

HF may be manifest clinically by typical symptoms: include breathlessness, orthopnea, paroxysmal nocturnal dyspnea, reduced exercise tolerance, fatigue, tiredness, increased time to recover after exercise, ankle swelling. Moreover, less typical symptoms include a nocturnal cough, wheezing, weight gain> 2kg/week or weight loss in advanced stages bloated feeling, loss of appetite, confusion, depression, palpitations and syncope [4].

Coronary heart disease and hypertension remain the most common causes of HF. Other causes include heart problems as valve defect, muscle defect, rhythm disorder and lung problems as COPD, asthma. Infection like rheumatic fever. Bad lifestyle like alcohol abuse. Other medical conditions as diabetes, obesity, anemia, thyroid dysfunction and kidney disorder [5].

Echocardiogram is the primary investigation to evaluate cardiac structure and function, including diastolic function and measure LV ejection fraction (EF) to make the diagnosis of heart failure and to provide information on left and right ventricular size, volume, wall thickness, ventricular systolic, diastolic function, valvular structure and function so detect severity of the case and cause of HF and also to obtain prognostic information's [2].

Any causes of acute, severe LV or RV dysfunction may lead to HF. Acute myopericarditis, takotsubo cardiomyopathy, and hypertrophic cardiomyopathy, myocardial infarction, and significant coronary artery disease. Severe valvular regurgitation, typically caused by endocarditis or chordal rupture due to trauma or degenerative disease, may also cause HF. Aortic dissection may lead to CS via acute, severe aortic insufficiency. Severe stress in the setting of aortic or mitral stenosis. Cardiac tamponade or pulmonary embolism, pulmonary edema, hypertensive crisis, severe anemia, all this are life threatening conditions may lead to HF complications like cardiac arrest and death, severe renal impairment or failure, cerebrovascular stroke and cardiogenic shock [5].

Identification of patients at risk

Include older age hypertension, diabetes mellitus, dyslipidemia, severe dyspnea, multivessel coronary artery disease, prior MI or angina, previous diagnosis of heart failure, and bundle-branch block. Tachyarrhythmia, there may be clues to impending shock: heart rate is higher and blood pressure lower on hospital presentation [8].

The aim of our study to improve the management process in patients with Acute Heart Failure and predicts their outcome at

the emergency department of Suez Canal University Hospital.

Patients and methods

Type of the study: This study will be conducted as cross-sectional, observational study on adults presenting to the emergency department with Acute Heart Failure.

Site of the study: The study will be carried out in the Emergency Department of Suez Canal University Hospitals .with short term follow up in cardiology care unit (CCU) and Cardiology inpatient ward for MACE

Patients: Inclusion criteria:

1. Age: more than 18 years old.
2. Presented with **Framingham criteria** for heart failure.

A) Major criteria paroxysmal nocturnal dyspnea or orthopnea. , neck veins distension, rales, cardiomegaly, acute pulmonary edema, s3 gallop, increased venous pressure >16 cm of water, circulation time >25 sec, hepatojugular reflux.

B) Minor criteria: ankle edema, night cough, dyspnea on exertion, hepatomegaly, pleural effusion, vital capacity decreased 1/3 from maximum, tachycardia >120/min, weight loss >4.5 kg in 5 days in response to treatment. For establishing a definite diagnosis of HF, two major or one major and two minor criteria had to be present [6].

3. Gender: males and females.

Exclusion criteria:

1. Cancer patients with chemotherapy-induced HF.
2. Patients with trauma
3. Cardiac arrest on arrival to Emergency Department.
4. Inability to provide informed consent.

Sample size estimation:The sample size is calculated using the following equation;

$$n = \left[\frac{Z_{\alpha/2} + Z_{\beta}}{p_1 - p_2} \right]^2 (p_1q_1 + p_2q_2)$$

Where

n= sample size

Z α /2 = 1.96 (The critical value that divides the central 95% of the Z distribution from the 5% in the tail).

P1 = Prevalence/proportion in the study group = 4%

P2 = Prevalence/proportion in the study group =96%

So, by calculation, the number of cases is equal to 30 individuals per group. So, the sample size will be 60 individuals [7].

Sampling method: Sampling will be conducted among ER patients presenting to the ER with Acute heart failure during the preceding 48 h and 28 days during follow-up timeframe according to the inclusion and exclusion criteria.

- **Investigations** to consider in all selected patients is:
 - . **transthoracic echocardiography** to evaluate cardiac structure and function, including diastolic function and measure LV ejection fraction (EF) to make the diagnosis of heart failure and to assist in planning and monitoring of treatment it provide information on left and right ventricular size, volume, wall thickness, ventricular systolic and diastolic function and valvular structure and function so detect

severity of the case and cause of HF e.g. (coronary heart disease, hypertension, valvular heart disease, myocardial damage —), and to obtain prognostic information.

- **A12 lead ECG** to determine heart rate and rhythm, QRS morphology and duration.

- **Measurement of blood chemistry** (sodium, potassium, calcium, blood urea nitrogen, creatinine level, liver enzymes and bilirubin-) and complete blood count to detect anemia.

- **Chest X-ray** to identify pulmonary congestion/edema and cardiomegaly.

Methods of the study: Patients included in the study will be divided into two groups. Each group will include 30 patients their cardiac risk determined by one of the two methods of risk stratification utilizing findings on presentation to the ER.

Data analysis & management

Data Collection: Data were collected by the researchers themselves.

Statistical analysis of data: The data were collected, organized and tabulated using SPSS (statistical package for social science) computer software version 16.0, on an IBM compatible computer. Receiver operating characteristic (ROC) curves used to evaluate its predictive performance. The area under the curve (AUC) utilized as a summary measure of the diagnostic accuracy of the prediction tools across the gamut of risk groups, with 95% confidence intervals (CI). For quantitative data, mean and standard deviation were calculated. For comparison between two means, students (t) test was used. For comparison between the values of a parameter at two different intervals, the paired (t) test was used. For categorical data, chi2 or Fisher's exact test were calculated. For interpretation of results, p-value was considered significant when it equal or less than 0.05.

Ethical consideration

1. Approval of authority.
2. Agreement of participant without obligation.
3. Confidentiality of data.
4. Explanation of our study to the participants. Informed consent will be taken from each patient or his relatives if comatose before taking any data or doing any intervention.
5. Explanation of the aim in a simple manner to be understood by the common people.
6. The right of the patient to refuse involving in the research and he will have his usual treatment.
7. The right of the participant to withdraw from the study at any time without giving any reason.
8. All participants will be announced by results of the study.
9. The right of the patient to have a copy from the informed consent.
10. Signature or fingerprints of the patient or his relatives.
11. The researcher phone number and all possible communicating methods will be identified to the participant.
12. Inform the patient the outcome of the research.

Results

This study includes 60 heart failure patients presented to ER and divided into two groups each includes 30 patients. For each group we used an accurate risk stratification score for the group (A) we used the Seattle heart failure model and for the group (B) we used the Canadian predictor model for heart failure. So this study was conducted to compare two risks stratification scores (the Seattle Heart failure model, and the Canadian Predictor of Heart Failure Mortality model) regarding major adverse cardiac events (MACE).

Demographic characteristics of the studied populations:

The mean age of the studied groups was comparable without any statistically significant differences ($p=0.74$). The average age of the total population was 61.7 ± 15.7 years with age ranged from 18 to 88 years. Totally, the frequency of males was 43.3%, while the frequency of females was 56.7% (Table 1).

The SBP and RR among the studied populations show that. One-quarter (25.0%) of the studied patients had SBP 100-119 mmHg, and 21.7% of them had SBP ≥ 180 mmHg. Meanwhile, 18.3% of the sample population had SBP <90 mmHg. Approximately 38% of the patients had RR between 30 to 39 breath and minute and 36.7% of them had RR between 29 to 29 breath/minute. In contrast, 15.0% of the sample population had RR <20 breath/minute. There were insignificant differences between both studied groups regarding vital signs ($p>0.05$).

The laboratory findings among the studied populations show that. The majority (80.0%) of the studied patients had sodium concentrations <136 meq/l. additionally, 70.0% of the sample population had BUN >20 mg/dL. Approximately 2% of the patients had severe anemia, 11.7% had moderate anemia, 38.3% had mild anemia, and 48.3% of them had normal hemoglobin levels. As regard to TLC was higher among the Seattle, risk score patient is in opposite to platelet count which was higher among the Canadian group. There were petty differences between both studied groups regarding laboratory findings ($p>0.05$).

Table (4) shows the Canadian predictive model of mortality in patients with heart failure. Cerebrovascular disease was present in 23.3% of the patients of Canadian group, COPD was present in 50%, and liver cirrhosis was present in 26.7% of them.

As regard the predicting survival using Seattle heart failure model. The majority of the patients (76.7%) of Seattle group had severe limitation of function (NYHA grade IV), 83.3% of them had EF% $<50\%$, hypercholesteremia was present in 43.3%, hyperuricemia was present in 90.0%, and lymphocytes $>1.5 \times 10^3$ was present in 50% of them.

From The 60 patients included in the study, only eight managed in the ER room represents 13.3%. Moreover, 52 patients were risky and admitted to CCU unit represents 86.7%.

As regard the risk stratification according to Seattle heart failure model. About 57% of the patients were at very high risk (survival% $<40\%$), 16.7% were at high risk, 16.7% were at intermediate risk, and 10% were at low risk of mortality.

The risk stratification according to Canadian predictive model of mortality in patients with heart failure shows that. Twenty percent of the patients were at very high risk (>150 points), 30% were at high risk (121-150 points), 26.7% were at intermediate risk (91-120 points), and 23.3% were at very low (≤ 60 points) to low risk of mortality (61-90 points). As regard the MACE in Seattle heart failure model group. The mortality rate among the studied patients was 66.7%, acute renal failure

Table 1 Group A: the Seattle heart failure risk score:[9].

Components:	calculation:	Risk score
- age by years -Gender, male or female - weight by kg -systolic blood pressure - NYHA classes -Ejection fraction (1%-75%) -basic laboratory values : (sodium.meq/l, creatinine.mg/l, total cholesterol mg/dl, white cell count, hemoglobin, % of lymphocytes, uric acid(.mg/dl) - heart failure medication: ACE inhibitor, beta blocker, angiotensin receptor blocker, K-sparring diuretic, statins, allopurinol use, diuretics dose.mg/kg -devices (eg, defibrillators and biventricular pacemakers)	(survival for score_0) was estimated by $Survival = e(-\lambda t)$ where t = time and $\lambda = (0.0405)$. Survival at time t (between 0 and 5 years for any score is estimated by the, following equation: $Survival = e(-\lambda t) e$ (SHFM Score)	High survival % = low mortality accompanied with: Statins use.High EF. Low NYHA class Low survival % = high mortality accompanied with: Increased daily diuretic dose. Low NYHA classes. Intracardiac devices Multiple medications use. Hyponatremia <138 meq/l, anemia, old age

Table 2 Group B: Canadian predictor of heart failure risk score: [10].

Components:	No of points	Risk score
age	+ age in years	
respiratory rate, in min	+rate (in/ min)	
systolic blood pressure, mmHg		
>180	-60	Very low: <60 POINTS
160-179	-55	
140-159	-50	Low risk: (61 90 points),
120-139	- 45	
100-119	-40	Intermediate risk (91-120)
90-99	-35	
<90	-30	High risk (121-150)
Urea nitrogen	+ level in mg/dl	POINTS
Sodium concentration <136meq/ l	+10	
Cerebrovascular disease	+10	Very high risk >150
Dementia	+20	POINTS
COPD	+10	
Hepatic cirrhosis	+25	

Table 3 Table(1) Demographic characteristics of the studied populations:

Variables	Canadian score (n=30)	Seattle score (n=30)	Total (n=60)	Used test	p-value
Age (years)					
Mean \pm SD	62.4 \pm 14.2	61.03 \pm 17.2	61.7 \pm 15.7	0.33	0.74
Range	34-86	18-88	18-88	0.33	0.74
Gender					
Male	11(36.7%)	15(50.0%)	26(43.3%)	1.1	0.30
Female	19(63.3%)	15(50.0%)	34(56.7%)	1.1	0.30
Insignificant p-value >0.05, *significant p-value \leq 0.05.					

Table 4 Table (2) SBP & RR among the studied populations:

Variables	Canadian score (n=30)	Seattle score (n=30)	Total (n=60)	χ^2 (p-value)
SBP(mmHg)				
<90	6(20.0%)	5 (16.7%)	11(18.3%)	0.11(0.74)
90-99	5(16.7%)	3(10.0%)	8(13.3%)	Fisher(0.71)
100-119	7(23.3%)	8(26.7%)	15(25.0%)	0.09(0.76)
120-139	1(3.3%)	2 (6.7%)	3(5.0%)	Fisher(0.99)
140-159	2(6.7%)	2 (6.7%)	4(6.7%)	Fisher(1.00)
160-179	4(13.3%)	2 (6.7%)	6(10.0%)	Fisher(0.67)
≥ 180	5(16.7%)	8(26.7%)	13(21.7%)	0.88(0.35)
RR/min				
<20	4(13.3%)	5(16.7%)	9(15.0%)	Fisher(0.99)
20-29	12(40.0%)	10(33.3%)	22(36.7%)	0.29(0.59)
30-39	13(43.4%)	10(33.3%)	23(38.3%)	0.63(0.43)
≥ 40	1 (3.3%)	5(16.7%)	6(10.0%)	Fisher(0.19)
Insignificant p-value>0.05, *significant p-value \leq 0.05, SBP=systolic blood pressure, RR=respiratory rate.				

Table 5 Table (3) Laboratory findings among the studied populations:

Variables	Canadian score (n=30)	Seattle score (n=30)	Total (n=60)	χ^2 (p-value)			
Sodium							
<136 meq/ l	25(83.3%)	23(76.7%)	48(80.0%)	0.42(0.52)			
≥ 136 meq/ l	5(16.7%)	7(23.3%)	12(20.0%)	0.42(0.52)			
BUN							
≤ 20mg/dL	10(33.3%)	8(26.7%)	18(30.0%)	0.32(0.57)			
>20mg/dL	20(66.7%)	22(73.3%)	42(70.0%)	0.32(0.57)			
Hemoglobin							
<8 g/dL	1(3.3%)	0(0.0%)	1(1.7%)	Fisher(1.00)			
8-9.5 g/dL	3(10.0%)	4(13.3%)	7(11.7%)	Fisher(1.00)			
9.5-11.9 g/dL	13(43.4%)	10(33.3%)	23(38.3%)	0.63(0.43)			
≥ 12 g/dL	13(43.4%)	16(53.4%)	29(48.3%)	0.60(0.44)			
TLC							
Mean	9.0	9.6	.70	0.48			
SD	2.97	3.5	.70	0.48			
minimum	4.5	3.4	.70	0.48			
maximum	14.8	19.00	.70	0.48			
Platelet count							
Mean	201.7	161.53	Paired sample statistics.				
Std. Deviation	55.692	73.204	Paired sample statistics.				
Std. Error mean	10.168	13.365	Paired sample statistics.				
Paired sample test							
Mean	Std. Deviation	Std. Error	95% confidence of the difference		t	df	Sig (2- tailed)
			upper	lower			
39.733	100.340	18.319	2.266	77.201	2.169	29	.038

was 10.0%, stroke was 3.3%, the cardiogenic shock was 13.3%, and significant arrhythmia was 6.7%.

The MACE in Canadian predictive model of mortality in patients with heart failure in (Table 6) shows that. The mortality rate among the studied patients was 20.0%, acute renal failure was 23.3%, stroke was 10.0%, the cardiogenic shock was 36.7%, and significant arrhythmia was 10.0%.

The coordinate's values of the ROC curve to Canadian predictive model of mortality in patients with heart failure in (Table 7) show that. The sensitivity of Canadian predictive model was 66.7%, specificity of 62.5%, +PV of 30.8% and -PV of 88.2%. The AUC was low (AUC: 0.559). As regard the coordinate's values of the ROC curve to Seattle model of mortality in patients with heart failure. The sensitivity of Seattle model was 75%, specificity of 90%, +PV of 93.7% and -PV of 64.3%. The AUC was significantly high (AUC: 0.840), and SE was significantly low (SE: 0.0866) (p-value <0.0001).

Discussion

Heart failure is often a progressive condition, beginning with predisposing factors and leading to the development and worsening of clinical illness. Despite improvements in therapy, the mortality rate in patients with heart failure has remained unacceptably high (Yancy et al., 2013) [11].

Heart failure presents enormous healthcare burdens, and the outcomes in heart failure are highly disappointed with an annual mortality rate reaching up to 75% (Levy et al., 2006, [12].

Despite a rich and growing history of predictive heart failure modeling in medicine, few such models have been successfully incorporated into routine practice and decision support at the point of care. This relative lack of applied prediction modeling in medicine is due in part to the insufficient adoption of available, thoroughly validated models (Alba et al., 2013) [13].

The aim of this study was to compare two risks stratification scores; Seattle Heart Failure Model and Canadian Predictor of

Table 6 Table (4) Canadian predictive model of mortality in patients with heart failure (n=30):

Variables	Canadian score (n=30)
Cerebrovascular disease	
Present	7 (23.3%)
Absent	23 (76.7%)
Chronic obstructive pulmonary disease	
Present	15 (50.0%)
Absent	15 (50.0%)
Liver cirrhosis	
Present	8 (26.7%)
Absent	22 (73.3%)

Table 7 Table (5) Predicting survival using the Seattle heart failure model (n=30):

Variables	Seattle score (n=30)
NYHA Functional Classification	
Marked limitation	7 (23.3%)
Severe limitation	23 (76.7%)
Ejection fraction (EF %)	
<50%	25 (83.3%)
≥ 50%	5 (16.7%)
Hypocholesteremia	
Present	13 (43.3%)
Absent	17 (56.7%)
Hyperuricemia	
Present	27 (90.0%)
Absent	3 (10.0%)
Lymphocytes / mm3	
<1.5 x10 ³	15 (50.0%)
>1.5 x10 ³	15 (50.0%)
NYHA=New York Heart Association.	

Table 8 Table (6) comparison between patients with the two risk scores according to admission or discharge from the emergency room.

		The Canadian model		The Seattle risk score		total	
		n	%	n	%	n	%
outcome	CCU	24	80.0%	28	93.3%	52	86.7%
outcome	discharge	6	20%	2	6.7%	8	13.3%
statistics	X=2.30, P= 0.19						

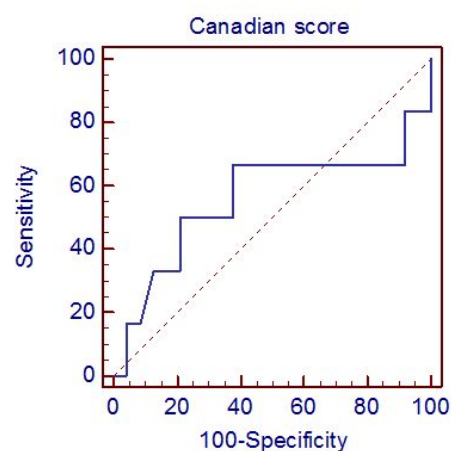


Figure 1: ROC curve to Canadian predictive model of mortality in patients with heart failure

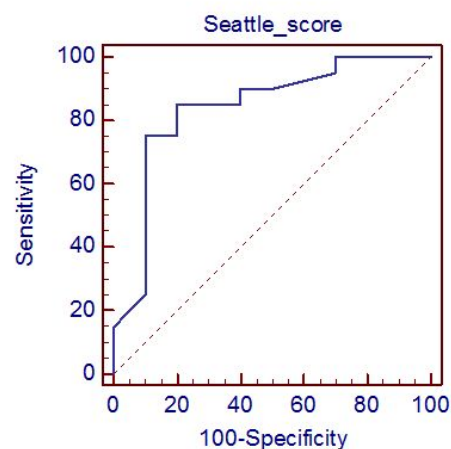


Figure 2: ROC curve of Seattle model of mortality in patients with heart failure.

Table 9 Table (7) comparison between two groups risks stratification results.

Variables	The canadian score (n=30)	Seattle score (n=30)
Risk stratification		
Very Low	6(20%)	
Low	1 (3.3%)	3(10.0%)
Intermediate	8 (26.7%)	5(16.7%)
High	9(30.0%)	5(16.7%)
Very high	6 (20.0%)	17(56.7%)
NYHA=New York Heart Association.		

Table 10 Table (8) Comparison between two groups as regard MACE.

Variables	The canadian score (n=30)	Seattle score (n=30)
MACE		
Mortality	6 (20%)	20 (66.7%)
Acute renal failure	7 (23.3%)	3(10.0%)
Stroke	3 (10.0%)	1 (3.3%)
Cardiogenic shock	11 (36.7)	4 (13.3%)
Significant arrhythmia	3 (10.0%)	2 (6.7%)
MACE=major adverse cardiac events.		

Heart Failure Mortality Model regarding mortality prediction. Two groups of patients were included, 30 cases in each group.

The mean age of the studied groups was comparable without significant difference. The mean age of the total population was 61.7 ± 15.7 years with age ranged from 18 to 88 years. The age is one of the prognostic variables included in the predictive models. This high mean age of the patients with heart failure also observed in several studies. [14].

The study of Levy et al. (2006) was performed to develop and validate a multivariate risk model to predict 1-, 2-, and 3-year survival in heart failure patients. The mean (\pm SD) age of the included cases was 63 ± 11 years. [12].

In similar prospect, Ketchum et al. (2012) investigated the incremental value of the Seattle Heart Failure Model for prediction of mortality and risk stratification in heart failure patients. The mean age of their patients was 62 ± 12 years. In these previous studies, older age groups were associated with increased mortality rate. [15].

In the present study, female gender predominates in the sample presented with acute HF (56.7%); on the other hand, this gender difference did not show any significance on the incidence of MACE. These findings agree with the results of ESC Heart Failure 2014. Who reported that elderly patients hospitalized with heart failure are mainly women and also agree with the ESC Guidelines for 2012 who indicated that patients with HF-PEF are more often females and obese [1].

Table 11 Table(9)comparison between the ROC curve of the two risk scores.

Coordinates	The Canadian score value	The Seattle score Value
Area under the ROC curve (AUC)	0.559	0.840
Standard Error (SE)	0.175	0.0866
95% Confidence interval	0.367 to 0.739	0.661 to 0.948
Significance level P-value	0.7365	<0.0001**
Sensitivity	66.7%	75.0%
Specificity	62.5%	90.0%
Positive predictive value	30.8%	93.7%
Negative predictive value	88.2%	64.3%

Our study revealed that 25.0% of the studied patients had SBP 100-119 mmHg and 18.3% of the sample population had SBP <90 mmHg. This low SBP was observed by many studies (Levy et al., 2006). [12].

The association between low blood pressure and prognosis in the general population has been controversial, with some reports suggesting an increased mortality for patients with the lowest blood pressures. In this prospect, Lee et al. (2016) concluded that patients with systolic dysfunction, lower SBP and DBP were associated with greater mortality (16). Also, Levy et al. (2006) reported that lower SBP was associated with poor prognostic outcome. [12].

Regarding laboratory findings, the majority (80.0%) of the studied patients had low sodium concentrations (<136 mEq/l), 70.0% had high BUN levels (>20 mg/dl), and approximately 52% of the patients had mild to severe anemia.

In correspondence with our data, Lee et al. (2016) reported high levels of BUN among their patients (29.4 ± 19.3 mg/dl) and low hemoglobin concentration were predictive of mortality in heart failure cases [16]. The same results of low sodium concentrations (135.8 ± 2.1 mEq/l) were estimated by Giamouzis et al. (2009). Where they found that, in the studied patients with major events, the mean sodium levels were low (135.9 ± 3.3 mEq/l), the mean BUN levels was high (26.5 ± 15.4 mg/dl), the mean platelet and TLC levels was higher in the high risk group (201.27 ± 55.6 for platelet) and (9.3 ± 10.5 for TLC) as was estimated by JAMA, (2005) [17].

In our study, ischemic causes like cerebrovascular disease were present in 23.3% of the patients. In agreement with our study, Levy et al. (2006) described that ischemic etiology had a significant impact on the mortality rate of patients with heart failure. [12].

Additionally, May et al. (2007) found that 59.7% of the studied heart failure patients had ischemic causes [18]. The study performed by Giamouzis et al. (2009) stated that ischemic events

in their patients were 38.2% [17]. Gorodeski et al. (2010) observed in their study that ischemic etiologies were present in 55.2% of the patients with heart failure. [19].

According to Canadian predictive model in our research, 20% of the patients were at very high risk, 30% were at high risk, 26.7% were at intermediate risk, and 23.3% were at very low to low risk of mortality. The sensitivity was 66.7%, specificity of 62.5%, +PV of 30.8% and -PV of 88.2%. The AUC was low (AUC: 0.559) [10].

Canadian predictive model was developed and validated by Lee et al. (2016) study. They estimated that 51.1% of the patients were at very high risk, 27.1% were at high risk, 14.8% were at intermediate risk, and 7% were at very low to low risk of mortality. The AUC was 0.80 for prediction of mortality in patients with heart failure. [16].

Consistency with our figures, Auble et al. (2007) detected that sensitivity Canadian predictive model was 62.2%, specificity of 55.2% and the AUC was 0.62. Similarly, Austin et al. (2010) found that the predictive model had AUC of 0.62 for predicting in-hospital mortality in patients hospitalized with heart failure. [20].

In our study, 23.3% and 76.7% of the patients among Seattle model group had marked to severe limitation of function (NYHA grades III and IV, respectively) and 83.3% of them had EF% <50%. [9].

Anad I and his colleagues (2007) conducted a study to determine the effectiveness of Seattle Heart Failure Model in a Pakistani heart failure cases in predicting mortality in this population. They found that the majority of their patients (66.1%) had marked limitation of function (NYHA grade III) and 31.4% of them had the severe limitation of function (NYHA grade IV). Similarly, Giamouzis et al. (2009) estimated that most of the patients had marked to severe limitation of function (NYHA grade III and IV) and the mean EF% among the patients was very low (18.2±7.9%). [17].

In our patients, hyperuricemia was present in 90.0%. In the same side, Giamouzis et al. (2009) reported that the mean level of serum uric acid in patients with heart failure was 8.4±0.9 mg/dl [17]. Also, Agha SA et al. (2009) found high serum uric acid (8.5±0.7 mg/dl) (22). In this way, Gorodeski et al. (2010) stated that the mean serum uric acid concentrations in the studied patients suffered from heart failure were high (9 mg/dl). [23].

Our findings indicated that the sensitivity of Seattle model was 75%, specificity of 90%, +PV of 93.7% and -PV of 64.3%. The AUC was significantly high (AUC: 0.840) (p-value <0.0001). [9].

Validation of the Seattle Heart Failure Model in several trials to predict survival showed this model to be highly accurate, with AUC for predicting one-year mortality being between 0.75 to 0.80 (Levy et al., 2006) [12].

In agreement with our findings, Hussain et al. (2014) found that the sensitivity of the model was 89.3% and specificity was 71.1% with 49% +PV and 95.5% -PV. The accuracy of the model was 75.4%. In ROC analysis, AUC for the Seattle Heart Failure Model was 0.802 (p < 0.001) [24].

This study shares the limitations of all observational studies; however, it was prospective nature in patient ascertainment and follow-up.

As regards results of the study we found that: The mean age of the studied groups was comparable without any statistically significant differences (p=0.74). The mean age of the total population was 61.7±15.7 years with age ranged from 18 to 88 years. There were insignificant differences between male and female

gender in the studied groups (p=0.30). Totally, the frequency of males was 43.3%, while the incidence of women was 56.7%.

One-quarter (25.0%) of the studied patients had SBP 100-119 mmHg, and 21.7% of them had SBP ≥ 180 mmHg. Meanwhile, 18.3% of the sample population had SBP <90 mmHg. Approximately 38% of the patients had RR between 30 to 39 breath/minute, and 36.7% of them had RR between 29 to 29 breath/minute. In contrast, 15.0% of the sample population had RR <20 breath/minute. There were petty differences between both studied groups regarding vital signs (p>0.05).

The majority (80.0%) of the studied patients had sodium concentrations <136 meq/l. additionally, 70.0% of the sample population had BUN >20 mg/dL. Approximately 2% of the patients had severe anemia, 11.7% had moderate anemia, 38.3% had mild anemia, and 48.3% of them had normal hemoglobin levels. There were petty differences between both studied groups regarding laboratory findings (p>0.05).

Cerebrovascular disease was present in 23.3% of the patients of Canadian group, COPD was present in 50%, and liver cirrhosis was present in 26.7% of them.

According to Canadian predictive model, 20% of the patients were at very high risk, 30% were at high risk, 26.7% were at intermediate risk, and 23.3% were at very low to low risk of mortality.

The mortality rate among the studied patients in Canadian model was 20.0%, acute renal failure was 23.3%, stroke was 10.0%, the cardiogenic shock was 36.7%, and significant arrhythmia was 10.0%.

The sensitivity of Canadian predictive model was 66.7%, specificity of 62.5%, +PV of 30.8% and -PV of 88.2%. The AUC was low (AUC: 0.559).

The majority of the patients (76.7%) of Seattle group had severe limitation of function (NYHA grade IV), 83.3% of them had EF% <50%, hypercholesterolemia was present in 43.3%, hyperuricemia was present in 90.0%, and lymphocytes >1.5 x10³ was present in 50% of them.

According to Seattle heart failure model, 57% of the patients were at very high risk, 16.7% were at high risk, 16.7% were at intermediate risk, and 10% were at low risk of mortality.

In Seattle heart failure model group, the mortality rate among the studied patients was 66.7%, acute renal failure was 10.0%, stroke was 3.3%, the cardiogenic shock was 13.3%, and significant arrhythmia was 6.7%.

The sensitivity of Seattle model was 75%, specificity of 90%, +PV of 93.7% and -PV of 64.3%. The AUC was significantly high (AUC: 0.840), and SE was significantly low (SE: 0.0866) (p-value <0.0001).

Conclusion

The Seattle Heart Failure Model allows prediction of survival of heart failure patients with the use of easily obtained clinical characteristics. The model provides an accurate estimate of the survival rate of the patients and allows estimation of effects of adding medications or devices to a patient's regimen. Use of this model by both healthcare providers and their patients may facilitate estimation of prognosis, enhance compliance, and increase the use of life-saving medications and devices; further investigation of such potential benefits is needed. Seattle Heart Failure Model is more accurate than Canadian predictive model in the prediction of mortality rate in patients with heart failure.

Recommendations

It is recommended to use the Seattle heart failure risk model for risk stratification and mortality prediction in patients with heart failure.

Increase health education about risk factors of ischemic heart diseases and heart failure (especially; modifiable risk factors).

To conduct health programs that increase knowledge about early manifestations of heart failure and the importance of healthy lifestyle, blood pressure control and monitoring of blood sugar level especially among high-risk group.

To supply more facilities to hospitals to increase their ability to manage acute and massive complications of heart failure.

Further, larger studies with higher sample size and more controlling of confounding factors are recommended to compare other risk stratifications scores for prediction of mortality in patients with heart failure.

Authors' Statements

Competing Interests

The authors declare no conflict of interest.

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