World Journal of Biology Pharmacy and Health Sciences
eISSN: 2582-5542
Cross Ref DOI: 10.30574/wjbphs
Journal homepage: https://wjbphs.com/

(RESEARCH ARTICLE)

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Comparatively tested potential risk factors across survivors and non-survivors critically patients

Basim Nawaf Alshwaheen [1,*], Mo'ath Musa Falah Rabab'ah [2], Suhaib Adel Mahmoud Albarbbarawi [1], Tariq Saber Mohammad Alkhrissat [1] and Amro Nayif Ghaleb Al-Rawashdeh [1]

[1] *Internal Medicine, Royal Medical Services, Amman, Jordan.*
[2] *Nephrologist, Royal Medical Services, Amman, Jordan.*

World Journal of Biology Pharmacy and Health Sciences, 2024, 18(01), 123–135

Publication history: Received on 01 April 2024; revised on 10 April 2024; accepted on 11 April 2024

Article DOI: https://doi.org/10.30574/wjbphs.2024.18.1.0173

Abstract

Background: The risk factors associated with mortality in critically ill patients have been the subject of numerous studies. However, a considerable amount of variation is evident when one considers the dispersion of patients, the geographical spread, and the classification of healthcare establishments.

Aim: The purpose of this study was to compare potential risk factors between Survivors and Non-Survivors, including demographics, anthropometrics, kidney and liver indices, complete blood counts, and biochemical assays. Patients whose condition is critical.

Methods: An ICU retrospective investigation was conducted in Jordan, where critically ill patients with surgical and medical conditions were examined. Using the Electronic Medical Record System (Hakeem), the study analysed data and classified patients into two cohorts according to their survival status. Demographic information, clinical characteristics, insulin administration rate, blood glucose levels, dosing schedules for vasopressors, and the burden of comorbidities were all included in the data. The research was granted approval by the Institutional Review Board committee of the Royal Medical Services, Jordan.

Results: The research examined the male-to-female ratios among COVID-19 patients and classified them into six distinct age groups. A significant proportion of critical patients, ranging in age from 50 to 60, exhibited elevated corrected sodium levels and a diminished normontraemia status. Higher albumin levels and mean arterial pressures were associated with survivors, whereas a greater proportion of non-survivors were underweight and had a low BMI. A normal temperature was observed in the majority of critically ill patients, whereas non-survivors exhibited elevated fractional blood glucose levels and estimated creatinine clearances. The research emphasises the significance of treatment outcomes and patient demographics.

Conclusion: SI may be a more accurate prognostic indicator than non-septic patients due to the effect of norepinephrine, a vasopressor, on heart rate and systolic blood pressure, which may account for the difference in SI values between the two groups.

Keywords: Comparatively tested; Potential risk factors; Survivors; Non-survivors; Critically ill patients

* Corresponding author: Basim Nawaf Alshwaheen

1. Introduction

A prevalent condition observed in intensive care unit (ICU) patients, hyperglycemia is correlated with heightened rates of both mortality and morbidity. Preliminary investigations revealed that strict adherence to glucose management protocols in surgical intensive care units and medical intensive care units could potentially mitigate mortality and morbidity rates, respectively. Subsequent investigations, nevertheless, have failed to corroborate these findings. Patients who underwent intensive glucose control had an increased risk of mortality, according to the NICE-SUGAR trial; however, the underlying cause of this phenomenon remains unexplained. Patients who are at an increased risk of death as a result of severe hypoglycemia are typically monitored with more stringent glucose control protocols.

The incident underscored the importance of adequate volume replacement in preventing acute kidney injury (AKI), despite the fact that rectifying fluid deficiency does not invariably avert renal failure. Continuous fluid challenges should be avoided if they fail to improve kidney function or exacerbate oxygen levels. Acute kidney injury (AKI) risk is increased by sepsis, cardiac surgery, age, diabetes, rhabdomyolysis, preexisting renal disease, hypovolemia, and shock. In comparison to hypooncotic colloids, crystalloid resuscitation is equally effective and safe; however, hyperoncotic solutions are contraindicated as they carry the potential for renal complications. Discontinuing the use of low-dose dopamine to improve renal function was recommended by the panel. Although conventional triggers for renal replacement therapy (RRT) in cases of kidney failure may not be appropriate for critically ill patients with AKI, the intervention is still capable of sustaining life.

Liver disease is a substantial global public health concern, wherein the most prevalent forms include hepatitis A, B, C, D, and E. Alcoholic liver disease is a close second in mortality rate in the United States, with cirrhosis being ranked as the twelfth most prevalent cause of death. Acute liver failure is predominantly attributed to paracetamol poisoning (46%), whereas hepatitis B stands as the most prevalent infectious cause. Acute hepatitis is caused by a metabolic, toxic, or infectious damage to liver cells, which ultimately results in inflammation, cell death, and scarring of the liver. The replacement of liver parenchyma with fibrous tissue as a result of chronic disease isolates hepatocytes into nodules. The aforementioned disruption in the customary configuration of tissues has the potential to intensify and lead to the characteristic manifestations of liver failure: cellular-level metabolic and synthetic function breakdown, progressive development of portal hypertension, fluid accumulation in the abdomen (ascites), and irregular blood circulation between the portal and systemic circulatory systems. Uncontrolled bleeding, a potentially severe complication of hepatic failure and a life-threatening consequence of liver disease, can result from inadequate production of these clotting factors. Portal hypertension is characterised by increased hydrostatic pressure in the portal vein and its tributary vessels as a result of reduced hepatic blood flow caused by cirrhosis. It results in the development of portal-systemic shunting and esophageal and gastric varices in the long run.

Liver failure is characterised by encephalopathy, a distinguishing characteristic of chronic liver disease. Although the precise pathophysiology remains unknown, ammonia is commonly thought to be the cause of perplexity and lethargy in encephalopathy patients. Ammonia produced by colonic microorganisms can infiltrate the bloodstream via portal-systemic shunting when portal hypertension is present in cirrhosis. An accumulation of bile pigment in the epidermis, sclerae, and mucous membranes can lead to jaundice at any stage of liver disease, which is caused by elevated bilirubin levels in the blood. Blood loss, viral infection, and the ingestion of pathogens are all potential causes of prehepatic jaundice. Elevated levels of conjugated bilirubin result from posthepatic jaundice.

In order to effectively manage hemodynamics in critically ailing and post-operative patients following high-risk surgery, vasopressor-inotropic support is indispensable. According to a study utilising data from the French and European Outcome Registry in Intensive Care Unit (FROG-ICU), critically ailing patients who require more vasopressor-inotropics have an increased risk of morbidity and mortality. It is of the utmost importance to provide substantial vasoactive-inotropic support to cardiac surgical patients throughout the peri-operative period. The findings of the study on the relationship between post-operative mortality and leucoglycemic index (LGI) were limited by the absence of concurrent post-operative vasopressor-inotropic requirements. When peri-operative inflammation commences, as indicated by a high LGI, post-operative patients have an increased requirement for vasopressor-inotropic agents owing to inflammatory myocardial depression and/or vascular hyporesponsiveness. The VIS, which was developed by Gaies et al., is an objective metric utilised to measure post-operative hemodynamic support. A duration component was included by Crow et al. in order to account for the fluctuating requirements for vasopressor-inotropic substances during the post-operative phase. A study discovered that a VIS index of three or greater was a more accurate predictor of adverse composite outcomes in infants undergoing cardiac surgery than the maximal VIS score alone.

The objective of this retrospective investigation was to contrast the rates of distribution of the designated concerns between two primary study cohorts: survivors and nonsurvivors. A range of surrogate variables were investigated in

this study, including demographics, the burden of comorbidities, rates of vasopressors and insulin, the blood glucose target range, estimated creatinine clearance, and the functional liver child index. The participants were critically ill patients who were admitted to the intensive care unit at King Hussein Medical Centre in Royal Medical Services, Jordan, from 2021 to 2022.

2. Material and Methods

This retrospective study centred on critically ill patients, including those with surgical and medical conditions, and was conducted in the Intensive Care Unit (ICU). pending approval by the local Institutional Review Board committee (IRB) of the Royal Medical Services in Jordan. The present investigation encompassed all admitted critically ill patients, including those undergoing mechanical ventilation and those whose information was accessible through the electronic medical record system of our institution (Hakeem). Our study excluded patients who possessed substantial lacking data for the primary parameters being examined or the variables being compared. On the basis of survival status, we classified all eligible admitted intensive care unit (ICU) patients into two cohorts: Cohort I (Survivors Cohort) and Cohort II (Non-Survivors Cohort). Statistical analysis was conducted on the comparative variables of the two cohorts that were categorised using the Chi Square Test (p-value < 0.05). The analysis comprised the following: Number (Percentage), Pearson chi-square statistic (χ 2), which computes the discrepancy squared between expected and observed frequencies, and Goodness of Fit (G-Test of independence), which employs the logarithm of the likelihood ratio to evaluate the correspondence between observed and expected frequencies. In order to quantify the strength of associations, odds ratios (OR) and their 95% confidence intervals were utilised. The correlation values were determined through the implementation of ordinal-by-ordinal correlations (Spearman, ρ) and interval-by-interval correlations (Pearson, r). However, the research conducted a retrospective analysis of various factors including demographic information of patients, clinical characteristics (e.g., norepinephrine rate in micrograms per minute), insulin administration rate in international units per hour, average blood glucose level relative to the target threshold of 180 mg/dL, demographic information and clinical characteristics of patients at admission days with regard to liver and kidney health, and the comorbidity burden of patients as assessed by the Charlson Com

3. Results

In this study, the male-to-female ratio was 2.28:1 (1,498 males, or 69.5 percent, versus 657 females, or 30.5%). The distribution of [293 (66.6%) and 147 (33.4%) was found to be insignificant in comparison to 1205 (70.3%) and 510 (29.7%), p-value=0.567. Ages of the patients were classified into six consecutive ranges, beginning with 18–30 and ending with >=70. The age groups of 50–60%, 40–50%, and 60–70% comprised the majority of the critical patients in our study [890 (41.3%), 531 (24.6%), and 473 (21.9%), respectively]. The Non-Survivors Cohort had considerably greater proportions of corrected sodium levels below 120 mEq/l than the Survivors Cohort [590 (34.4%) vs. 53 (12.0%), respectively]. As a result, the Non-Survivors had a lower percentage of individuals with relatively normonatraemia status [1116 (65.1%) vs. 387 (88.0%), respectively].

The Survivors Cohort exhibited a greater proportion of patients with albumin levels ranging from 2 to 2.49 g/dl [314 (71.4%)], in contrast to the Cohort II patients who presented with a more extensive distribution of albumin ranges. In comparison to the Survivors Cohort, the Non-Survivors Cohort exhibited a considerably greater proportion of albumin levels within the range of 1.5-1.99 g/dL [610 (35.6%) vs. 50 (11.4%), respectively]. In the Survivors Cohort, a considerably greater proportion of the evaluated patients had mean arterial pressures of 70 mmHg or higher than in the Non-Survivors Cohort [284 (64.5%) vs 591 (34.5%), respectively].

The anthropometric measurements of the patients' body mass indexes (BMIs) were systematically classified into six distinct categories, ranging from Obese III (≥40 kg/m2) to low BMI (<18.4 kg/m2). In comparison to the Non-Survivors Cohort, the proportions of individuals classified as over-weight and those classified as having a low BMI were notably different in the Survivors Cohort [12 (2.7%) versus 218 (12.7%) and 180 (40.9%) versus 503 (29.3%), respectively]. In contrast to patients in Cohort II, whose average temperatures were predominantly in the mild hyperthermia range [740 (43.1%)], the majority of critically ill patients in Cohort I had average temperatures within the normal range [285 (64.8%)].

While the proportion of patients in the non-survivor cohort (fBG180) of critically ill patients was greater than that of the survivors (387.3%) versus 1276 (74.4%), respectively, in the Survivors cohort and the non-survivors cohort, a greater number of patients (BGGlk) than 200 mg/dl and below 250 mg/dl were detected using glucometer-based blood glucose levels [1622 (94.6%) vs 284 (64.5%), respectively]. A greater proportion of patients in Cohort I exhibited estimated creatinine clearances falling within the range of (40-49) ml/min, followed by those in the (50-59) ml/min

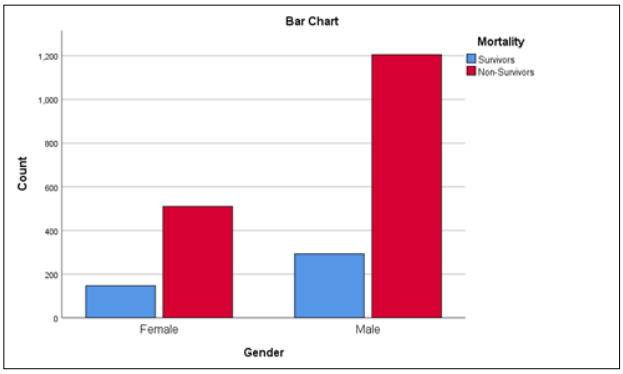
range, in contrast to patients in Cohort II who reported clearances falling within the range of (30-39) ml/min, followed by the (40-49) ml/min range [123 (28.3%) and 111 (25.6%) vs. 501 (29.3%) and 390 (22.8%), respectively]. A summary of the variables that were compared and the visual representations of the bar charts for the critically ill patients in Cohorts I-II were provided in Table 1-3 and Figure 1-3, respectively.

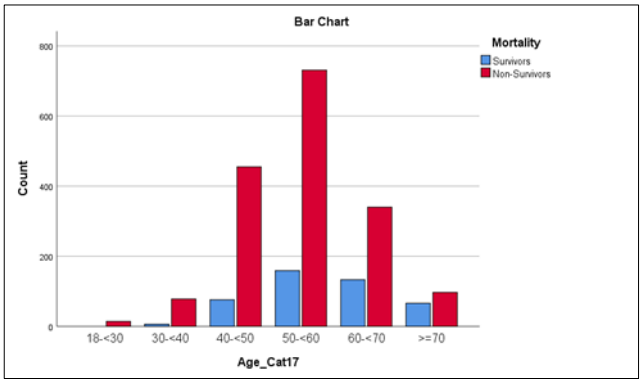
Table 1 Variables compared between the Survivors Cohort (Cohort I) and the Non-Survivors Cohort (Cohort II) of critically ill patients

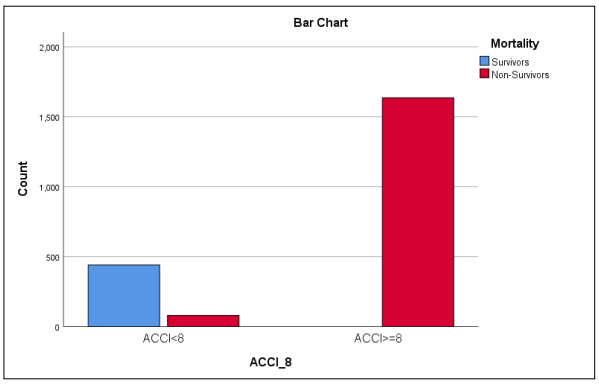
	Survivors Cohort I (N=440, 20.4%)	**Non-Survivors Cohort II (N=1715,79.6%)**	**Total (N=2155)**	**OR**	R ρ	χ^2 G-Test	**p-Value**
Gender							
Female	147 (33.4%)	510 (29.7%)	657 (30.5%)	1.185 (95% CI; 0.95-1.48)	0.032±0.022 0.032±0.022	2.227 2.200	0.136
Male	293 (66.6%)	1205 (70.3%)	1498 (69.5%)				
Male: Female	1.99: 1	2.36: 1	2.28: 1				
Age (Yrs)							
18-<30	0 (0.0%)	14 (0.8%)	14 (0.6%)	NA	-0.192±0.021 -0.185±0.021	86.133 84.717	0.000
30-<40	6 (1.4%)	78 (4.5%)	84 (3.9%)				
40-<50	76 (17.3%)	455 (26.5%)	531 (24.6%)				
50-<60	159 (36.1%)	731 (42.6%)	890 (41.3%)				
60-<70	133 (30.2%)	340 (19.8%)	473 (21.9%)				
>=70	66 (15.0%)	97 (5.7%)	163 (7.6%)				
AACCI							
<8	440 (100.0%)	79 (4.6%)	519 (24.1%)	0.152 (95% CI;0.124-0.186)	0.899±0.011* 0.899±0.011*	1742.817 1738.712	0.000
≥8	0 (0.0%)	1636 (95.4%)	1636 (75.9%)				
BMI (Kg/m^2)							
<18.4	12 (2.7%)	218 (12.7%)	230 (10.7%)	NA	-0.202±0.021* -0.202±0.020*	99.59 104.61	0.000
18.4-24.9	179 (40.7%)	885 (51.6%)	1064 (49.4%)				
25-29.9	180 (40.9%)	503 (29.3%)	683 (31.7%)				
30-34.9	69 (15.7%)	102 (5.9%)	171 (7.9%)				
35-39.9	0 (0.0%)	6 (0.3%)	6 (0.3%)				
>=40	0 (0.0%)	1 (0.1%)	1 (0.0%)				
ALB (g/dl)							
<1	3 (0.7%)	0 (0.0%)	3 (0.1%)	NA	-0.054±0.017	208.27	0.000

1-1.49	3 (0.7%)	35 (2.0%)	38 (1.8%)		-0.080±0.018	221.89	
1.5-1.99	50 (11.4%)	610 (35.6%)	660 (30.6%)				
2-2.49	314 (71.4%)	613 (35.7%)	927 (43.0%)				
2.5-2.99	70 (15.9%)	406 (23.7%)	476 (22.1%)				
3-3.49	0 (0.0%)	51 (3.0%)	51 (2.4%)				
Temp (˚C)							
36-36.9	87 (19.8%)	250 (14.6%)	337 (15.6%)	NA	0.201±0.019 0.213±0.019	129.09 146.09	0.000
37-37.9	285 (64.8%)	698 (40.7%)	983 (45.6%)				
38-38.9	68 (15.5%)	740 (43.1%)	808 (37.5%)				
39-39.9	0 (0.0%)	27 (1.6%)	27 (1.3%)				

Cohort I: Survivors studied critically ill patients; Cohort II: Non-Survivors studied critically ill patients; Temp: Measured body core temperatures; ALB: Serum albumin level.







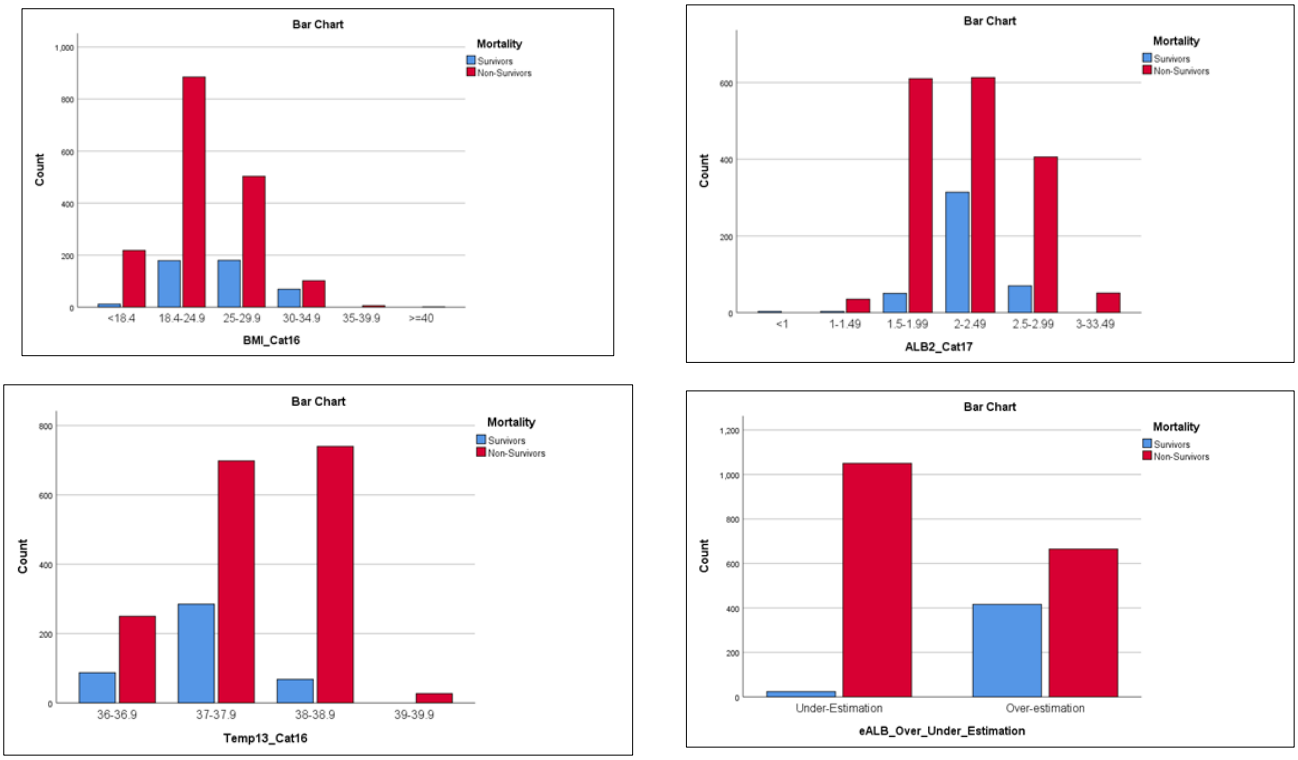


Figure 1 Bar charts representing critically ill patients as members of the Survivors Cohort (Cohort I) and the Non-Survivors Cohort (Cohort II)

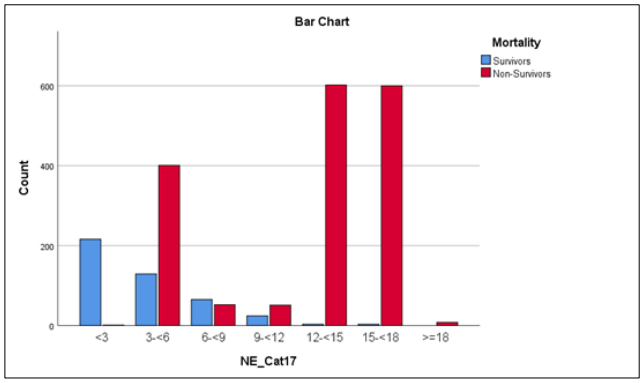
Table 2 Between January 2018 and May 2021, variables pertaining to critically ill patients from the Survivors Cohort (Cohort I) and the Non-Survivors Cohort (Cohort II) at the King Hussein Medical Centre, Royal Medical Services, Jordan were compared

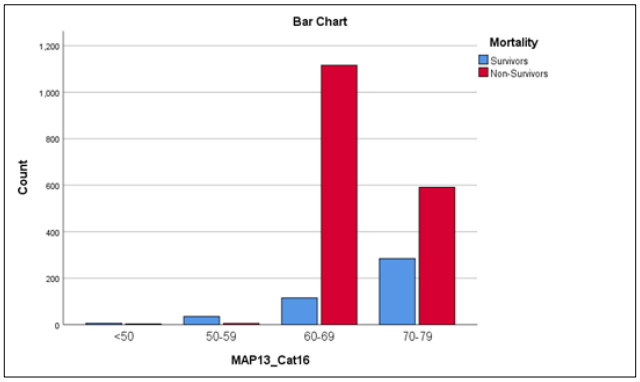
	Survivors Cohort I (N=440, 20.4%)	**Non-Survivors Cohort II (N=1715,79.6%)**	**Total (N=2155)**	**OR**	**R ρ**	**χ^2 G-Test**	**p-Value**
NE rate (mcg/min)							
<3	216 (49.1%)	1 (0.1%)	217 (10.1%)	NA	0.603±0.014* 0.583±0.013*	1233.23 1250.03	0.000
3-<6	129 (29.3%)	401 (23.4%)	530 (24.6%)				
6-<9	65 (14.8%)	52 (3.0%)	117 (5.4%)				
9-<12	24 (5.5%)	51 (3.0%)	75 (3.5%)				
12-<15	3 (0.7%)	602 (35.1%)	605 (28.1%)				
15-<18	3 (0.7%)	600 (35.0%)	603 (28.0%)				
>=18	0 (0.0%)	8 (0.5%)	8 (0.4%)				
MAP (mmHg)							
<50	6 (1.4%)	3 (0.2%)	9 (0.4%)	NA		293.615	0.000

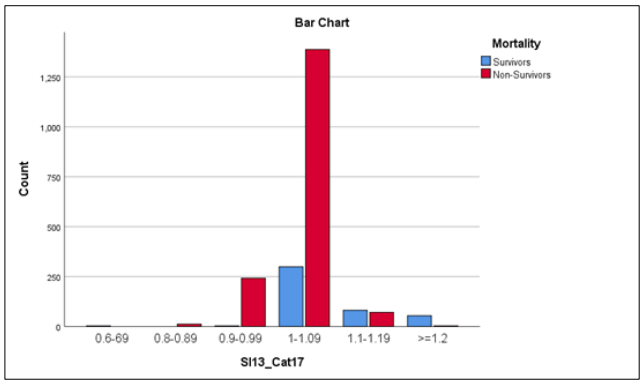
50-59	35 (8.0%)	5 (0.3%)	40 (1.9%)		-0.148±0.027* -0.195±0.024*	272.732	
60-69	115 (26.1%)	1116 (65.1%)	1231 (57.1%)				
70-79	284 (64.5%)	591 (34.5%)	875 (40.6%)				
Na (mEq/l)							
<120	0 (0.0%)	8 (0.5%)	8 (0.4%)	NA	-0.235±0.020* -0.238±0.020*	127.947 118.533	0.000
120-129	132 (30.0%)	1015 (59.2%)	1147 (53.2%)				
130-139	308 (70.0%)	686 (40.0%)	994 (46.1%)				
140-149	0 (0.0%)	6 (0.3%)	6 (0.3%)				
Na (mEq/l)							
<129.35	91 (20.7%)	936 (54.6%)	1027 (47.7%)	0.217 (95% CI; 0.169-0.279)	-0.274±0.019* -0.274±0.019*	161.275 171.082	0.000
≥129.35	349 (79.3%)	779 (45.4%)	1128 (52.3%)				
cNa (mEq/l)							
120-129	53 (12.0%)	590 (34.4%)	643 (29.8%)	NA	-0.189±0.017* -0.192±0.017*	87.25 100.74	0.000
130-139	387 (88.0%)	1116 (65.1%)	1503 (69.7%)				
140-149	0 (0.0%)	9 (0.5%)	9 (0.4%)				
cNa (mEq/l)							
131.05	73 (16.6%)	890 (51.9%)	963 (44.7%)	0.184 (95% CI; 0.141-0.241)	-0.286±0.018* -0.286±0.018*	176.568 192.634	0.000
131.05	367 (83.4%)	825 (48.1%)	1192 (55.3%)				
AAR							
0-1.79	168 (38.2%)	444 (25.9%)	612 (28.4%)	NA	0.217±0.019* 0.224±0.019*	148.482 101.261	0.000
1.8-2.19	208 (47.3%)	478 (27.9%)	686 (31.8%)				
>=2.2	64 (14.5%)	793 (46.2%)	857 (39.8%)				
CPS							
6	137 (31.1%)	0 (0.0%)	137 (6.4%)	NA	0.825±0.008* 0.729±0.012*	1912.146 1869.627	0.000
7	65 (14.8%)	0 (0.0%)	65 (3.0%)				
8	179 (40.7%)	1 (0.1%)	180 (8.4%)				
9	44 (10.0%)	51 (3.0%)	95 (4.4%)				

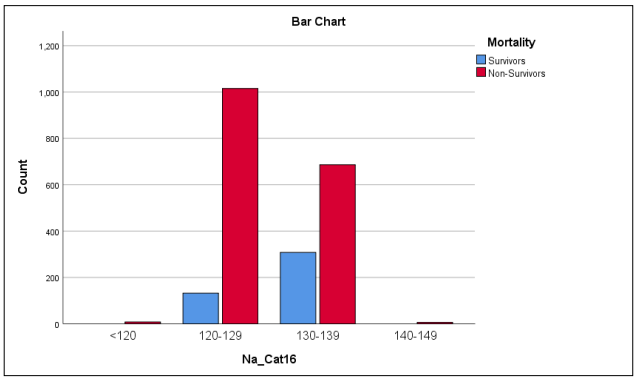
10	9 (2.0%)	1128 (65.8%)	1137 (52.8%)				
11	6 (1.4%)	426 (24.8%)	432 (20.0%)				
12	0 (0.0%)	86 (5.0%)	86 (4.0%)				
13	0 (0.0%)	20 (1.2%)	20 (0.9%)				
14	0 (0.0%)	3 (0.2%)	3 (0.1%)				

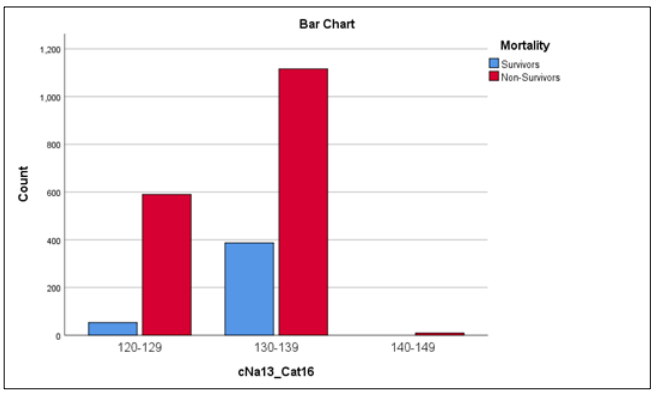
Cohort I: Survivors studied critically ill patients; Cohort II: Non-Survivors studied critically ill patients; NE: Norepinephrine; MAP: Mean arterial pressure; SI: Shock index; cNa: Corrected sodium level.











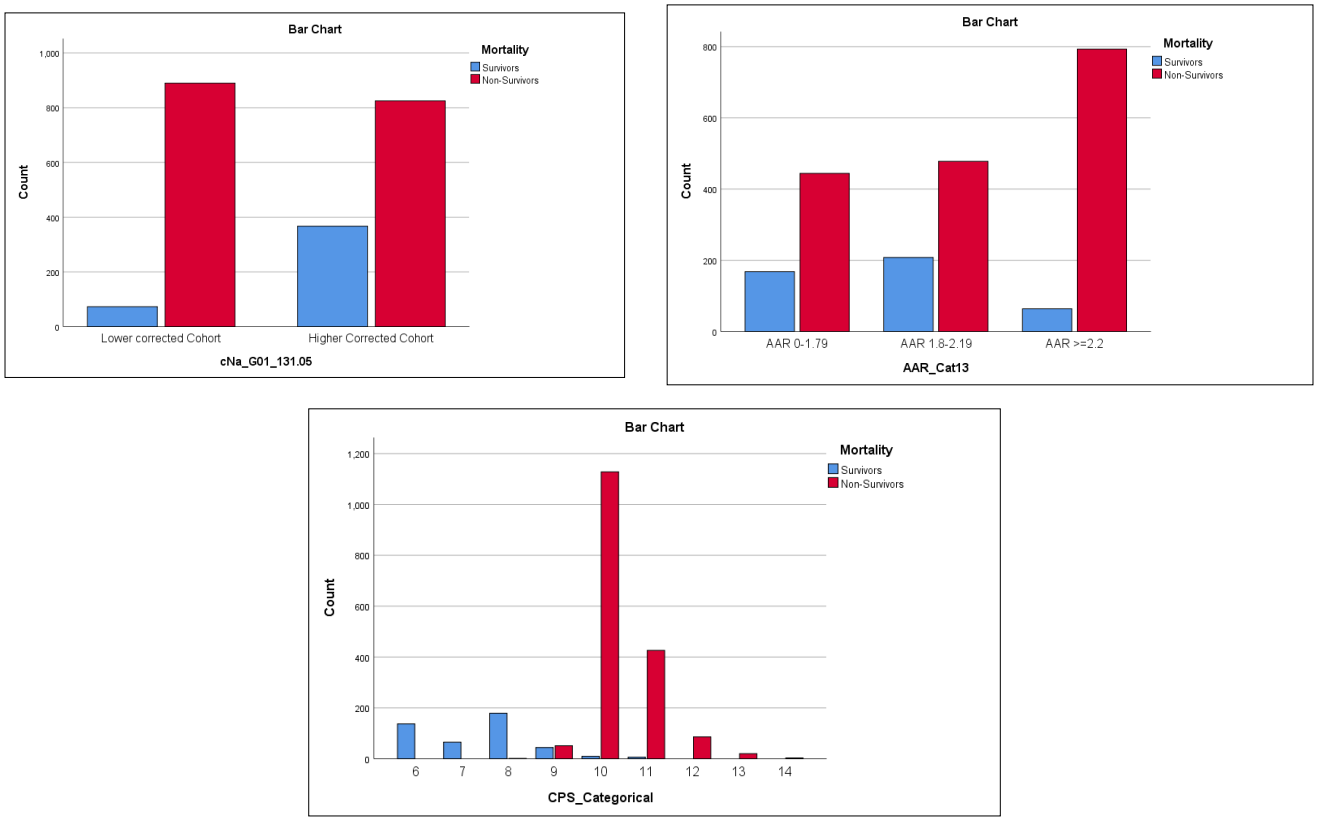


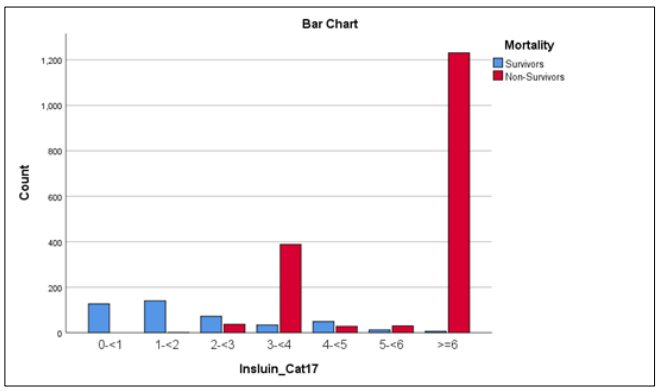
Figure 2 Between January 2018 and May 2021, bar charts were utilised to represent critically ill patients who were studied in comparison between the Survivors Cohort (Cohort I) and the Non-Survivors Cohort (Cohort II)

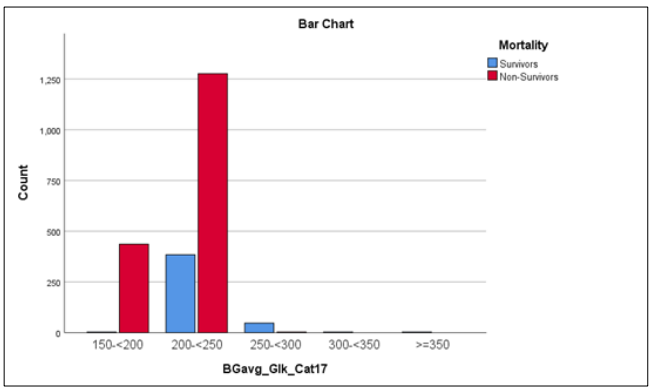
Table 3 (Subsequent). Between January 2018 and May 2021, variables pertaining to critically ill patients

	Survivors Cohort I (N=440, 20.4%)	**Non-Survivors Cohort II (N=1715,79.6%)**	**Total (N=2155)**	**OR**	**R ρ**	**χ^2 G-Test**	**p-Value**
Insulin (IU/hr)							
0-<1	127 (28.9%)	0 (0.0%)	127 (5.9%)	NA	0.729±0.012*	1606.93	0.000
1-<2	140 (31.8%)	1 (0.1%)	141 (6.5%)		0.690±0.013*	1566.33	
2-<3	72 (16.4%)	37 (2.2%)	109 (5.1%)				
3-<4	34 (7.7%)	388 (22.6%)	422 (19.6%)				
4-<5	49 (11.1%)	28 (1.6%)	77 (3.6%)				
5-<6	12 (2.7%)	30 (1.7%)	42 (1.9%)				
>=6	6 (1.4%)	1231 (71.8%)	1237 (57.4%)				
BG_{Glk} (mg/dl)							
150-<200	3 (0.7%)	436 (25.4%)	439 (20.4%)	NA	-0.336±0.014*	302.74	0.000
200-<250	384 (87.3%)	1276 (74.4%)	1660 (77.0%)		-0.320±0.013*	327.17	
250-<300	47 (10.7%)	3 (0.2%)	50 (2.3%)				
300-<350	3 (0.7%)	0 (0.0%)	3 (0.1%)				
>=350	3 (0.7%)	0 (0.0%)	3 (0.1%)				

BG_{Lab} (mg/dl)							
100-<150	0 (0.0%)	27 (1.6%)	27 (1.3%)	NA	-0.246±0.025*	285.08	0.000
150-<200	105 (23.9%)	429 (25.0%)	534 (24.8%)		-0.139±0.024*	130.385	
200-<250	256 (58.2%)	1251 (72.9%)	1507 (69.9%)				
250-<300	26 (5.9%)	5 (0.3%)	31 (1.4%)				
300-<350	15 (3.4%)	0 (0.0%)	15 (0.7%)				
>=350	38 (8.6%)	3 (0.2%)	41 (1.9%)				
fBG_{180}							
0.75-<1	0 (0.0%)	72 (4.2%)	72 (3.3%)	NA	-0.445±0.019*	554.53	0.000
1-<1.25	284 (64.5%)	1622 (94.6%)	1906 (88.4%)		-0.465±0.021*	450.09	
1.25-<1.5	147 (33.4%)	21 (1.2%)	168 (7.8%)				
1.5-<1.75	3 (0.7%)	0 (0.0%)	3 (0.1%)				
1.75-<2	3 (0.7%)	0 (0.0%)	3 (0.1%)				
>=2	3 (0.7%)	0 (0.0%)	3 (0.1%)				
CrCl (ml/min)							
<20	50 (11.5%)	48 (2.8%)	98 (4.6%)	NA		230.31	0.000
20-29	29 (6.7%)	252 (14.7%)	281 (13.1%)			241.00	
30-39	65 (15.0%)	501 (29.3%)	566 (26.4%)				
40-49	123 (28.3%)	390 (22.8%)	513 (23.9%)				
50-59	111 (25.6%)	163 (9.5%)	274 (12.8%)				
60-69	47 (10.8%)	112 (6.5%)	159 (7.4%)				
70-79	9 (2.1%)	124 (7.2%)	133 (6.2%)				
=>80	0 (0.0%)	122 (7.1%)	122 (5.7%)				

Cohort I: Survivors studied critically ill patients; Cohort II: Non-Survivors studied critically ill patients; BGavg_Glk: Blood glucose average based on Glucometer; fBG_180: Fractional blood glucose level based on 180 mg/dl; IU: International unit; CrCl: Creatinine clearance.





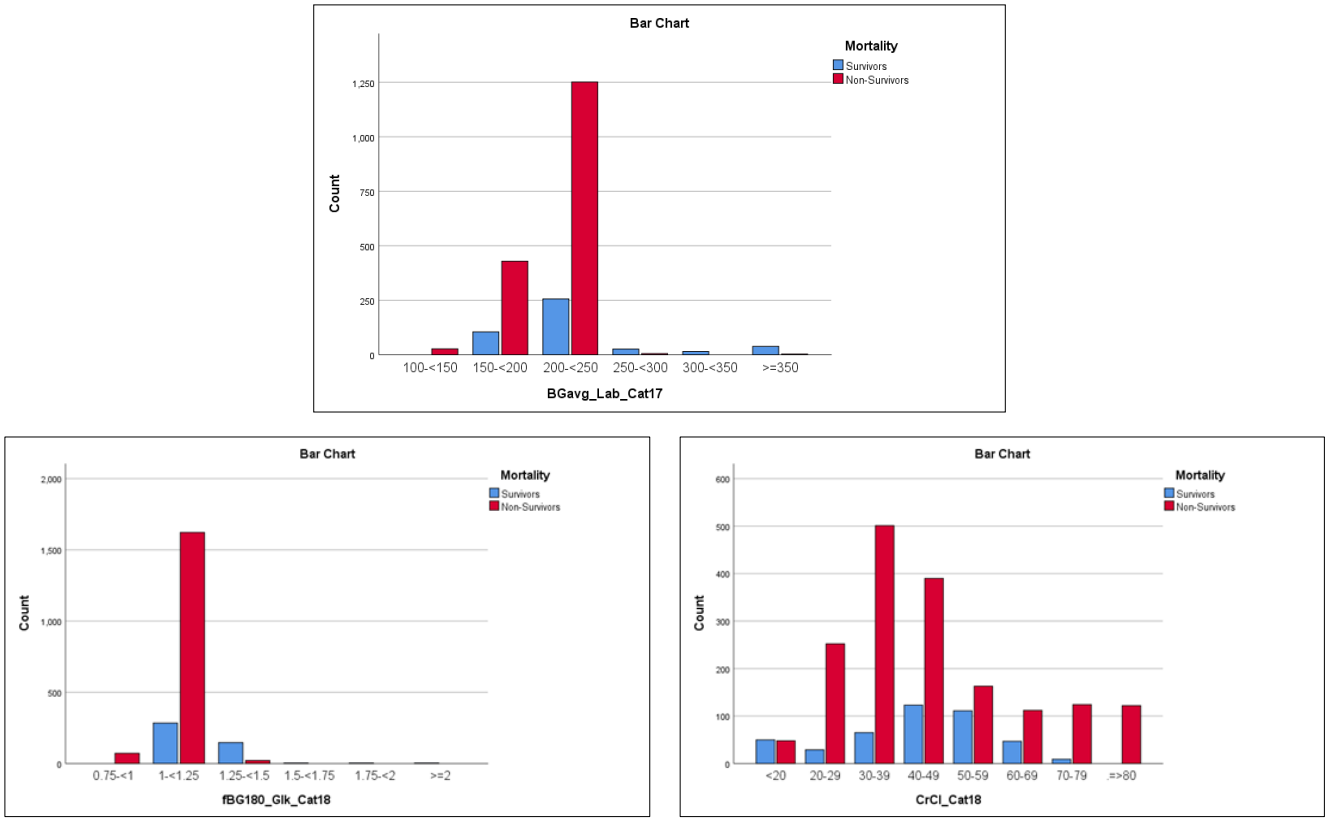


Figure 3 Between January 2018 and May 2021, bar charts were utilised to represent critically ill patients

4. Discussion

An unsponsored, retrospective observational study was undertaken for a period of sixty months in a multi-specialty intensive care unit (ICU) located at the preeminent tertiary medical centre in Jordan. The study's external validity is enhanced by the broad spectrum of selection criteria utilised to identify critically ill patients, which is the study's primary distinguishing feature. Surgical and medical intensive care unit (ICU) patients were included in this study, along with mechanically ventilated and non-ventilated patients. Patients who were placed on intensive glucose control frequently experienced moderate to severe hypoglycemia, according to the NICE-SUGAR study.

The identified risk factors for hypoglycemia were consistent with those identified in previous studies. The incidence of hypoglycemia was found to be higher among patients who adhered to intensive glucose control as opposed to those who followed conventional glucose control. In both groups, however, the association between hypoglycemia and mortality was comparable. While the risks were mitigated by controlling for potential variables that could have an impact on the initial and final results, the associations remained significant. Observational studies and smaller randomised controlled trials provide additional support for the findings. A prolonged QT interval is associated with both severe hyperglycemia and hypoglycemia, thereby elevating the risk of life-threatening cardiac arrhythmias.

However, our investigation did not investigate this aspect. In critically ill patients, blood glucose management strategies should prioritise the prevention of both moderate and severe hypoglycemia and the regulation of hyperglycemia. An ICU admission accompanied by elevated distribution rates of specific tested variables is an independent predictor of increased vasopressor-inotropic need and mortality probability, according to the study. There is a correlation between hyperglycemia (high blood sugar) and hypoglycemia (low blood sugar) and an increased mortality risk in specific circumstances. A variety of detrimental processes, including inflammation, blood clot formation, and an increase in oxidative stress, are linked to hyperglycemia, which increases the risk of mortality. Furukawa et al. established a correlation between hypoglycemia, hypoalbuminemia, and increased mortality rates among septic patients. Increasing data suggests that %BGvar is an indispensable indicator for mortality prediction. We restate our assertion and declare that our research represents the initial attempt to examine the correlation between the three hyperglycemic parameters that were assessed and mortality.

Early categorization utilising precise, readily accessible, and accurate predictive tools is of the utmost importance for the high severity and unpredictability of critically ill patients, given the limited resources at our disposal: to prevent insufficient prioritisation or delays in allocating higher care levels to those who require them. Sepsis-afflicted critically ill patients who were mechanically ventilated participated in the study. The vasopressant norepinephrine administered to these patients has an average flow rate of 9.53±1.79 mcg/min. As of the present, this is the first study to examine the associations between SI, CRP, and mortality. In the current context of limited resources, high acuity, and uncertainty among critically ill septic patients, early stratification utilising valid, reliable, and reasonably priced predictive tools is of the utmost importance. This helps prevent under-triage or delays and guarantees that patients who are ill are given priority.

When making triage decisions for septic patients, the Systematic Inflammatory Response recommends real-time physiological bedside triage tools over static ones [10–19]. This is especially true while awaiting the results of other diagnostic tests, including white blood cells (WBCs) with differential, CRP, and procalcitonin (PCT). SI exhibits enhanced sensitivity, performance, specificity, positive and negative predictive value, and accuracy in relation to ICU mortality at both the 28-day and late stages when compared to CRP. SI demonstrates superior performance, specificity, positive predictive value, and accuracy in relation to early mortality when compared to CRP. The findings of this research demonstrate a notable disparity between the predictive capabilities and significance of the Shock Index (SI) and C-reactive protein (CRP).

The observed disparity can be ascribed to the impact of norepinephrine, a vasopressor administered to mechanically ventilated critically ill patients with sepsis, on heart rate (HR) and systolic blood pressure (SBP). These effects enhance the reliability of SI as a prognostic indicator for septic patients and a more accurate indicator of heart rate (HR). SI is a cost-free, dependable, and efficient implement utilised at the bedside. When comparing norepinephrine as a vasopressor to CRP, it demonstrates superior sensitivity, specificity, and accuracy in predicting ICU mortality among mechanically ventilated critically ill patients who are septic. During bedside assessments, the systolic index (SI) can function as a practical and readily available indicator for severe illness.

This research is limited by its retrospective design, dependence on data collected at a single institution, and exclusive attention to mechanically ventilated septic patients in the intensive care unit. Nonetheless, the fact that our facility is seasoned and has a large capacity suggests that our data may be of use to other facilities. The present investigation is limited by its retrospective design, reliance on data from a single centre, and recruitment of hyperglycemic intensive care unit patients exclusively. Nevertheless, the proficiency and capacity of our facility render our data invaluable to other centres. Account for numerous confounding variables and establish the causal relationship between variables and mortality; this requires a prospective, multicenter, and exhaustive study.

5. Conclusion

SI may be a more accurate prognostic indicator than non-septic patients due to the effect of norepinephrine, a vasopressor, on heart rate and systolic blood pressure, which may account for the difference in SI values between the two groups.

Compliance with ethical standards

Acknowledgments

Our appreciation goes to staff of the departments of Royal Medical Services for their enormous assistance and advice.

Disclosure of conflict of interest

There is no conflict of interest in this manuscript

Statement of informed consent

Owing to the retrospective design of this study, the informed consent form was waived.

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