ORIGINAL ARTICLE

Impact of addition of Troponin I to APACHE II score on its Predictive Value of Mortality in Medical Intensive Care Patients

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Abstract

Background: ***In 1981, the APACHE scoring system was initially developed to forecast the outcome for patients admitted to the intensive care unit. And then, in the subsequent two decades, there were subsequent versions including APACHE II, III, and IV systems.***

Aim and objectives: ***The purpose of this study is to determine whether the Damanhur Medical National Institute in Damanhur, Egypt's non-surgical critical care unit patients' risk of death may be improved by including Troponin I as a criterion in the APACHE II score.***

Patients and methods: ***From March 2023 to March 2024, this prospective study was conducted in the intensive care department of the Damanhur Medical National Institute, involving 200 patients who were admitted to the intensive care unit(ICU) for medical reasons.***

Results: ***Around 75% of the cases that died had positive troponin levels, while 78% of the cases that lived had negative levels. This difference in troponin levels is statistically significant (p-value<0.05).***

Conclusion: ***In terms of APACHE score and APACHE percentage, there was a statistically significant distinction between cases where Troponin was positive and those where it was negative. The Addition of Troponin I levels to the APACHE II score results in an approximate 11.25% improvement in predictive value.***

Keywords: Troponin I; APACHE II; Mortality; Intensive care

1. Introduction

Acute physiology, chronic health, and age are the three components that make up the APACHE II criteria. To get the APACHE II score, add up all the points from all three sections. Respiratory illness, severe pancreatitis, acute pulmonary edema, and severe sepsis are among intensive care unit patients for whom this score has been shown to predict unfavorable outcomes.[1]

One component of the contractile machinery of cardiac myocytes is cardiac Troponin (cTn). Myocardial cell damage triggers its release into the bloodstream. There are two subtypes of cardiac Troponin: cTnT and cTnI.[2]

The high sensitivity(Hs) assay has reduced the particular function of cTn in the detection of acute myocardial damage. Even when acute cardiac injury is not present, the Hs-cTn assay can detect a low concentration of circulating protein.[3]

In addition to acute and chronic non-ischemic cardiac injuries, patients with sepsis, chronic kidney disease, stroke, subarachnoid hemorrhage, severe illness, side effects of chemotherapy, and even seemingly healthy individuals, particularly the elderly, may exhibit an increase in Hs-cTn levels.[4]

The present research intends to evaluate the prognostic power of the APACHE II score for mortality in non-surgical critical care patients at Damanhur Medical National Institute by including Troponin I as a metric.

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2. Patients and methods

From March 2023 to March 2024, this prospective study was conducted in the intensive care department of the Damanhur Medical National Institute, involving 200-patients who were admitted to the intensive care unit(ICU) for medical reasons.

Exclusion criteria:

Patients who were admitted for a surgical cause, patients with acute coronary syndrome, patients less than 18 years old, and patients who passed 24 hours or patients with readmission in less than 30 days.

All patients have been subjected to full history taking, initial clinical evaluation and laboratory investigations highlighting APACHE II score components, and Troponin I, also all patients had twelve leads resting electrocardiography and 2D trasnthoracic echocardiography.

APACHE II score:

We began by compiling the twelve most severe Acute Physiologic Assessment readings from the initial day. The metric with the highest correlation to points was considered the "worst" measurement. After the patient's age, the APACHE score moves on to the second point. Organ insufficiency and immunocompromised patients make up the last entity. Although patients seldom reach scores of 55 or higher on the APACHE II, the scale runs from 0 to 71 points.

The 12th values are:

Celsius temperature, the average pressure in the blood vessels during the two phases of a heartbeat, known as systole and diastole, is called mean arterial pressure(MAP). Heart rate in beats per minute, respiratory rate(whether ventilated or not). Oxygen saturation in the blood: FiO2 is the oxygen concentration in the breathed gas mixture, which is 21% times that of ambient air which is subdivided into two categorize if its less than 50% we measured PaO2 and if its equal or more than 50% we measured the A-a gradient which is the differential between the oxygen concentration in the alveoli and the arterial system. Arterial pH, serum sodium, potassium, and creatinine(in micromol/L or mg/dL), hematocrit, white blood cells, and the Glasgow coma score(GCS), where each point represents an APACHE score.

AGE point

Ages below 44-years old receive 0 points, 45–54 years old receive 2, 55–64 years old receive 3, 65–74 years old receive 5, and 75-years old and older receive 6 points.

Very impaired immune system function; severe organ system deficiency

Liver: Portal hypertension in biopsy-confirmed cirrhosis; history of upper gastrointestinal bleeding due to portal hypertension; or history of hepatic failure, encephalopathy, or coma

Cardiovascular: NYHA class IV heart failure

Respiratory: proven chronic hypoxia, hypercapnia, secondary polycythemia, severe pulmonary hypertension(>40 mmHg), dyspnea, significant exercise restriction (i.e., inability to climb stairs or do household activities), or respirator dependency.

The patient's immune system is weakened because they have had treatments that reduce their body's natural defenses against infections. These treatments may include immunosuppressants, radiation, chemotherapy, high-dose steroids, severe lymphoma, leukemia, or AIDS. Patients with immunocompromised immune systems or a history of severe organ insufficiency who are undergoing elective postoperative care will receive two points, whereas patients who are not undergoing surgery or who require emergency postoperative care will receive five points.

We collected Troponin I levels and took into account the worst value within the first 24 hours of admission after calculating the APACHE score and assessing the risk of mortality for each patient in the intensive care unit.

Lab Examination: The Troponin I level is measured using immunoassay techniques. Commonly used methods include enzyme-linked immunosorbent assay (ELISA) or chemiluminescent immunoassay.

Measuring Troponin I using different immunoassay methods, such as ELISA and chemiluminescent immunoassays(CLIA), with more consistent use of ELISA, as it offers varying advantages.

ELISA is a more cost-effective and reliable method, despite its lower sensitivity and longer processing time compared to more advanced techniques like CLIA but it still a valuable tool for batch testing and research applications, providing quantitative results that help in understanding cardiac biomarker levels.

In the first twenty-four hours after admission, we have also measured significant echocardiographic parameters in accordance with imaging guidelines established by the American Society of Echocardiography, placing special emphasis on: left ventricular ejection fraction(EF) as calculated by Simpson's technique, Systolic planar excursion of the tricuspid annulus(TAPSE) from M-mode Using the apical four-chamber view, align the lateral tricuspid annulus with the ventricular apex, and measure the RV fractional area change(FAC)-the displacement of the annulus relative to the free wall-during systole and diastole. This is accomplished by following the path of the RV endocardium along the interventricular septum from the annulus back to the apex. Consider the chamber's trabeculations and bands, as well as the left ventricle's diastolic activity using pulsed wave Doppler sample on the tips of the mitral valve.

Ethical Consideration:

All participant information is treated with the utmost confidentiality. In no publication or report will the names of the study's participants appear. We discussed the study's goals and methodology, as well as the risk-benefit analysis, with potential participants before we admitted them. A waiver of liability was signed.

Statistical Analysis:

Using SPSS for data processing, the data was reviewed, inputted, and analyzed. Quantitative data is shown as the mean±SD, whereas qualitative data is expressed as numbers and percentages. If the results were deemed significant, the p-value was set at less than 0.05.

3. Results

The study subjects were arranged according to demographic and baseline characteristics into age, sex, APACHE II scores and percentages, the survival and the qualitative Troponin I as in table 1.

Table 1. Baseline characteristics of the studied cases.

BASELINE CHARACTERISTICS (N=200)		DESCRIPTIVE STATISTICS
AGE	Mean±SD	59.7±15.4
	(Range)	(20:92)
AGE GROUP	<60 years	97(48.5%)
	>60 years	103(51.5%)
SEX	Male	132(66%)
	Female	68(34%)
APACHE SCORE	Mean±SD	14±6.3
	(Range)	0:38
APACHE PERCENTAGE	Mean±SD	21.8±13.9
	(Range)	4:85
SURVIVAL	Survived	144(72%)
	Died	56(28%)
TROPONIN	Positive	72(36%)
	Negative	128(64%)

The most frequent diagnosis among the studied cases was pulmonary embolism(11%) then chest infection(9.5%), then stroke(8.5%), then decompensated heart failure, AKI and DKA(7%) of the studied cases while the least frequent diagnosis is lung cancer, warfarin toxicity and infective endocarditis(1%) of the studied cases,(table 2;figure 1).

Table 2. Frequency and percentage of different diagnosis of the studied cases.

DIAGNOSIS(N=200)	FREQUENCY	PERCENTAGE
PULMONARY EMBOLISM	22	11%
CHEST INFECTION	19	9.5%
STROKE	17	8.5%
DECOMPENSATED HF	14	7%
ACUTE KIDNEY INJURY	14	7%
DKA	14	7 %
COPD EXACERBATION	13	6.5%
INTRACRANIAL HEMORRHAGE	12	6%
UTI SEPSIS	12	6%
CELLULITIS SEPSIS	10	5%
BED SORES SEPSIS	8	4%
DEHYDRATION	8	4%
HTN PULMONARY EDEMA	7	3.5%
HEPATIC ENCEPHALOPATHY	7	3.5%
ASTHMA EXACERBATION	6	3%
PERI MYOCARDITIS	5	2.5%
ENCEPHALITIS	3	1.5%
ANEMIA	3	1.5%
INFECTIVE ENDOCARDITIS	2	1%
LUNG CANCER	2	1%
WARFARIN TOXICITY	2	1%
TOTAL1	200	100%

There is statistically significant difference between survived and died cases as regard age, age group, APACHE score, and APACHE percentage(p-value<0.05) as mean age in died cases was 67 years compared to 57 in survived cases, also as mean APACHE score died cases was 19 compared to 12 in survived cases. While their non-significant difference between survived and died cases as regard sex(p-value>0.05), (table 3;figures 2,3).

Table 3. Comparison between survivors and died cases as regard baseline characteristics.

BASELINE CHARACTERISTICS		SURVIVORS (N=144)	DIED (N=56)	P-VALUE
AGE	Mean±SD	57±15.8	67±11.8	<0.001*
	Median (Range)	60(20:85)	70(38:92)	
AGE GROUP	<60-years	79(55.2%)	18(32%)	0.002*
	>60-years	65(44.8%)	38(68%)	
SEX	Male	98(61.8%)	34(67.6%)	0.44
	Female	47(38.2%)	21(32.4%)	
APACHE SCORE	Mean±SD	12±5.3	19±6	<0.001*
	Median (Range)	12(0:25)	19(4:38)	
APACHE PERCENTAGE	Mean±SD	19.3±9.8	33.6±16.2	<0.001*
	Median (Range)	17(4:55)	25(4:85)	
TROPONIN	Positive	30(20.8%)	42(75%)	<0.001*
	Negative	114(79.2%)	14(25%)	

*:Significant level at P-value<0.05

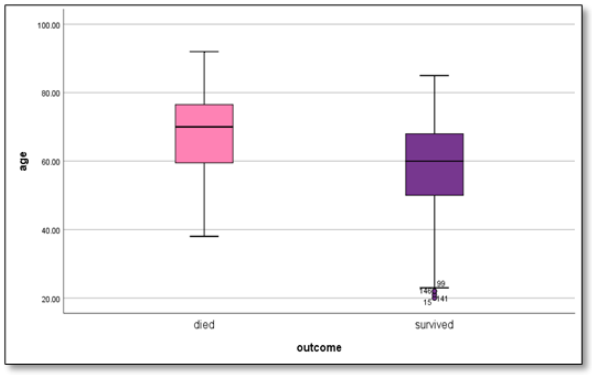

Figure 1. Comparison between died and survived cases as regard age.

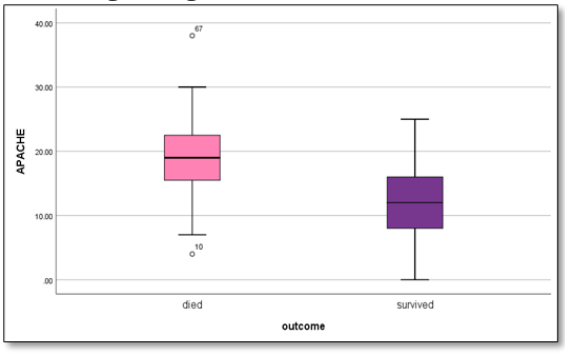

Figure 2. Comparison between died and survived cases as regard APACHE score.

There is statistically significant difference between troponin positive and negative cases as regard age, age group, APACHE score and APACHE percentage(p-value<0.05) as mean age in troponin positive cases was 62.5 years compared

to 58.2 in troponin negative cases, also as mean APACHE score in troponin positive cases was(16.4) compared to(12.5) in troponin negative cases. While their non-significant difference between troponin positive and negative cases as regard sex and GCS score(p-value>0.05),(table 4; figure 4).

Table 4. Comparison between Troponin positive and negative cases as regard baseline characteristics.

BASELINE CHARACTERISTICS		TROPONIN POSITIVE (N=72)	TROPONIN NEGATIVE (N=128)	P-VALUE
AGE	Mean±SD	62.5±15.5	58.2±15.3	0.049*
	Median(Range)	66(21:92)	60(20:85)	
AGE GROUP	<60 years	28(39%)	69(54%)	0.002*
	>60 years	44(61%)	59(46%)	
SEX	Male	48(66.7%)	84(65.5%)	0.88
	Female	24(33.3%)	44(34.5%)	
APACHE SCORE	Mean±SD	16.4±7.3	12.5 ±5	<0.001*
	Median (Range)	17.5(0:38)	13(2:26)	
APACHE PERCENTAGE	Mean±SD	27.8±16.8	18.4±10.7	<0.001*
	Median (Range)	25(4:85)	15(4:55)	
GCS SCORE	Mean±SD	14±1.8	14.3±1.5	0.41
	Median (Range)	15(7:15)	15(6:15)	

*:Significant level at P-value<0.05

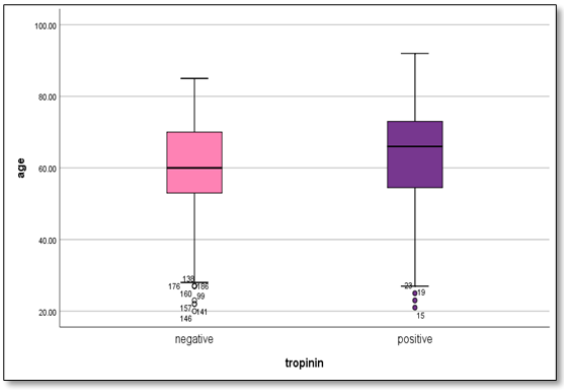

Figure 3. Comparison between troponin positive and negative cases as regard age.

There is statistically significant difference between survived and died cases as regard troponin(p value<0.05) as about 75% of died cases were troponin positive and 79% of survived cases were troponin negative,(table 5; figure 5).

Table 5. Comparison between troponin positive and negative cases as regard cardiac parameter.

CARDIAC PARAMETER		TROPONIN POSITIVE (N=72)	TROPONIN NEGATIVE (N=128)	P-VALUE
EF	Mean±SD	48.7±12	52.7±12	0.01*
	Median(Range)	50(20:60)	60(20:75)	
TAPSE	Mean±SD	16.4±3.2	18.1±2.5	0.001*
	Median(Range)	17(10:22)	18(12:24)	
RV-FAC	Mean±SD	37.8±8.1	39.5±6	<0.01*
	Median(Range)	35(25:60)	40(24:55)	
DIASTOLIC DYSFUNCTION	Normal	16(22.2%)	35(27.3%)	0.01
	Grade 1	32(44.4%)	69(53.9%)	
	Grade 2	6(8.3%)	7(5.5%)	
	Grade 3	5(6.9%)	0	
	Atrial fibrillation	13(18.1%)	17(13.3%)	

*:Significant level at P-value<0.05

As shown in previous table, there are statistically significant difference between troponin positive and negative cases as regard EF, TAPSE, RV-FAC and diastolic dysfunction(p-value<0.05) as EF, TAPSE and RV-FAC was higher in troponin negative cases(52.7& 18.1& 39.5) than troponin positive cases(48.7& 16.4& 37.8). For diastolic dysfunction 27% of troponin negative cases were normal compared to 22.2% of troponin positive cases also 5.5% of troponin negative cases had grade 2 and 3 DD compared to 17% of troponin positive cases.

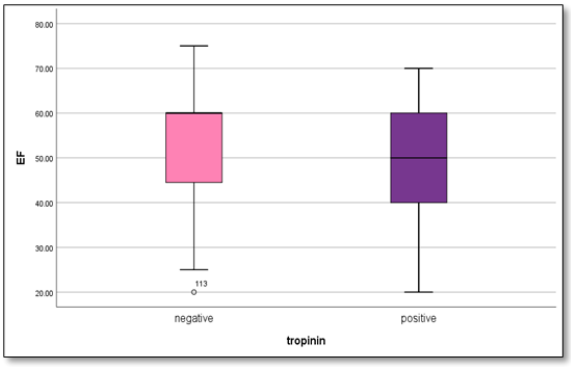

Figure 4. Comparison between troponin positive and negative cases as regard EF.

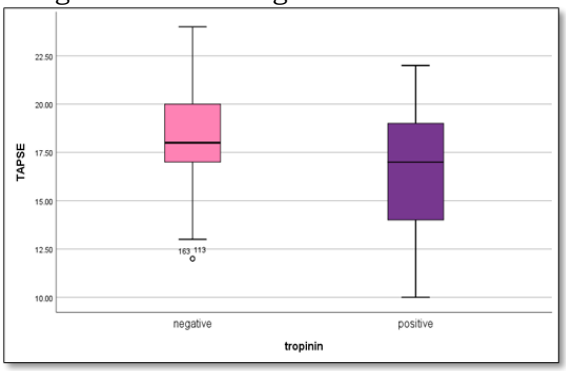

Figure 5. Comparison between troponin positive and negative cases as regard TAPSE.

Table 1. ROC curve analysis for prediction of mortality.

	APACHEII	TAPSE	CREATININE	GCS	TROPONIN
OPTIMAL CUT OFF POINT	>125	<18.5	>1.4	<13.5	0.05
AUC	0.80	0.71	0.64	0.69	0.234
955CI	(0.73:87)	(0.62:0.79)	(0.55:72)	(0.60:0.78)	0.157:0.311
P VALUE	<0.001*	<0.001*	0.002*	0.61	<0.001
SENSITIVITY	89%	76%	60%	36%	98%
SPECIFICITY	51%	50%	60%	91%	57%
PPV	89%	76%	60%	36%	98%
NPV	51%	50%	60%	91%	57%

AUC:Area Under the Curve, CI:Confidence Interval, PPV:Positive Predictive Value

NPV:Negative Predictive Value, *:Significant level at P-value<0.0

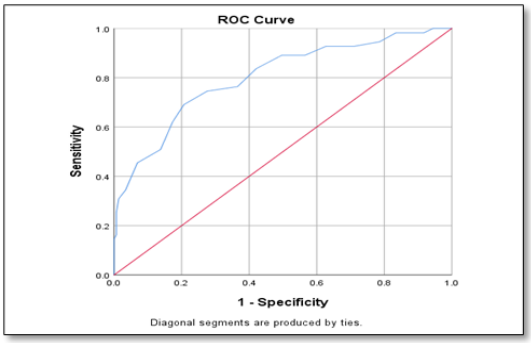

Figure 6. ROC curve for APACHE II score for prediction of mortality.

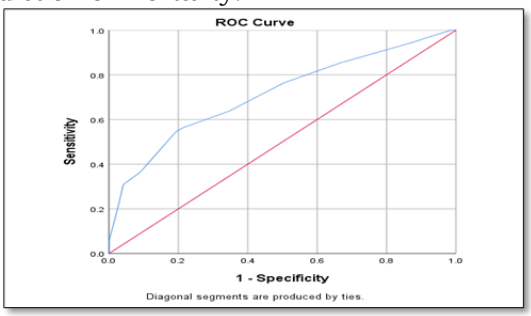

Figure 7. ROC curve for TAPSE for prediction of mortality.

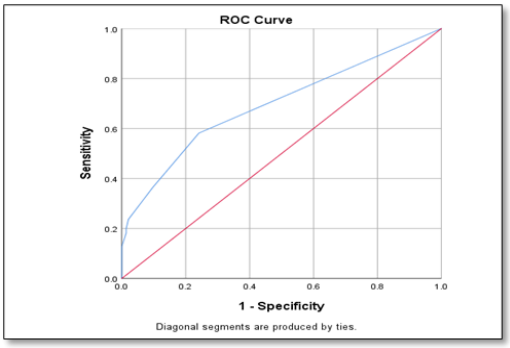

Figure 8. ROC curve for GCS score for prediction of mortality.

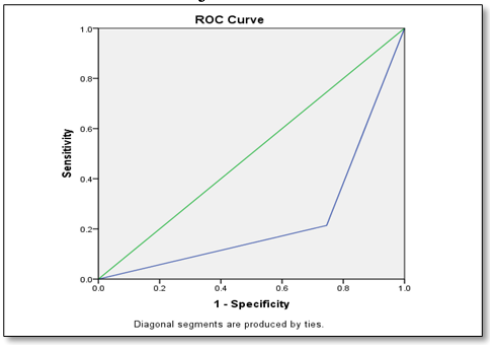

Figure 9. ROC curve for troponin for prediction of mortality.

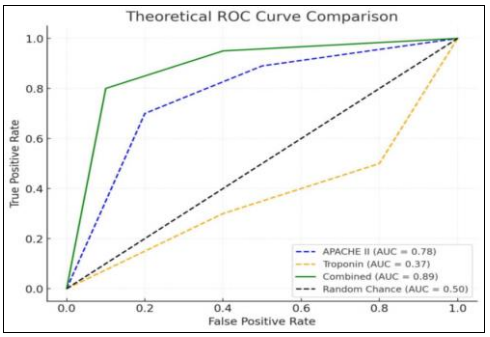

Figure 10. Area under the curves for predictive values of APACHE II, Troponin I and their combination.

ROC curve comparison illustrates the predictive performance of APACHE II alone, Troponin alone, and their combination, APACHE II AUC=0.78, showing strong predictive power Troponin AUC=0.37, indicating weak predictive ability on its own. Combined model AUC=0.89, showing significant improvement when combining Troponin with APACHE II. Percentage Improvement=[(AUC combined-AUC APACE II)/AUC APACHE II]×100 which will represent after calculation[(0.89-0.80)/0.80]×100 Percentage Improvement 11.25%.

4. Discussion

In order to anticipate the mortality and morbidity of critically ill patients, clinical assessment of illness severity is a crucial component of medical treatment, particularly in intensive care units(ICUs).[5]

A number of factors that may greatly affect the prognosis of critically sick patients are included by the APACHE, which was first introduced in 1981 and upgraded in 1985 into APACHE II, which includes twelve physiological variables and vital signs, in addition to age and co-morbid disorders.[6]

Myocytes in the heart include the protein cTn as part of their contractile machine. Myocardial cell damage triggers its release into the bloodstream. There are two subtypes of cardiac Troponin: cTnT and cTnI.[7]

This study was carried out on 200-patients with a mean age of 59.7 and a standard deviation of 20.92 years, 48.5% of the cases in this study were under 60-years old and 51.5% were over 60-years old. Sixty-three percent of the cases tested positive for Troponin and 64% tested negative, 72% of the cases lived and 28% died. The average APACHE score was 14, with a standard deviation of 0.38.

In agreement with our results, Babuin et al.,[8] we sought to ascertain, using the acute physiology and chronic health evaluation III prognostic system, if troponin elevations indicate in-hospital, short-term, and long-term death in patients receiving medical intensive care unit

treatment, regardless of the severity of the underlying disease. The statistical analysis revealed the following: an average age of 67.3±15.6 years, 524 males (56.4% of the total), an average APACHE III score of 63.7±29.5 points, and an average predicted risk of in-hospital death of 22.7±23.5 points. Heart troponin levels were ≥0.01g/L in 570 cases(61.4%), ≥0.03g/L in 439-cases(47.3%), and ≥0.1g/L in 203 cases(21.9%).

In our study, we found that the most frequent diagnosis among the studied cases was pulmonary embolism(11%) then sepsis(9.5%), then stroke and chest infection(8.5%), then COPD exacerbation(7.5%) of the studied cases while the least frequent diagnosis is marivan toxicity, infective endocarditis and lung cancer (1%) of the studied cases.

In line with our results, King et al.,[9] They set out to examine a diverse sample of critically sick medical patients with the goal of discovering whether cTnI level assessed upon admission is a standalone predictor of mortality. Regarding the reason for admission, 46-patients(34.9%) were admitted due to sepsis, 35-patients(27.3%) to respiratory failure, 10-patients(7.8%) to poisoning or drug overdose, 6-patients(4.7%) to gastrointestinal bleeding, and 31-patients(24.2%) for other reasons.

In our study, we reported that there was a statistically significant difference between survived and died cases with regard to age, age group, APACHE score, APACHE percentage, and GCS(p-value<0.05), as the mean age in died cases was 67 years compared to 57 years in survived cases. Also, the mean APACHE score and GCS score in the deceased cases were (19 & 13) compared to(12 & 14.6) in the surviving cases. There was no significant difference between survivors and deceased cases with regard to sex(p-value>0.05).

In support of our results, King et al.[9] they found that in the Alive(n=104) group, age(years) was 51.6±19.4, male was 57(54.8%), and APACHE II was 13.6±7.5. While, in dead(n=24) group, age(years) was 64±13.6, male was 11(45.8%) and APACHE II was 22.7±10.9. There was a highly significant difference regarding age and APACHE II between the alive group and the dead group.

In our results, there was a statistically significant difference between troponin-positive and negative cases with regard to age, age group, APACHE score, and APACHE percentage(p-value<0.05), as the mean age in troponin-positive cases was 62.5 years compared to 58.2 years in troponin-negative cases. Also, the mean APACHE score in troponin-positive cases was 16.4 compared to 12.5 in troponin-negative cases.

In agreement with our results, Babuin et al.,[8] they reported that in the cTnT positive(n=570) group, age, mean(SD), years were 71.5(13.1), the number of men was 331(58.1%), and APACHE score, mean (SD) was 71.4(27.8). In cTnT negative (n=359) group, age, mean(SD), years were 60.7(17.4), men numbers were 193(53.8%) and APACHE score, mean(SD), was 51.5(28.0). The age and APACHE score of the cTnT-positive and negative groups were significantly different. However, neither group differed significantly in terms of sex.

In our results, there was a statistically significant difference between survived and died cases regarding troponin(p-value<0.05), as about 75% of died cases were Troponin positive and 79% of survived cases were Troponin negative.

In supporting our results, King et al.,[9] they found that in the alive group, cTnI>0.7 in 10(19.2%), while in the dead group, cTnI>0.7 in 15(62.5%). There was a highly significant difference regarding Troponin between the alive group and the dead group(P<0.001).

We discovered a statistically significant distinction between the cases that survived and those that did not in terms of MAP, SBP, DBP, and PO2 score(p-value<0.05) in the current study. The mean MAP in the perished group was 81, while the mean MAP in the survived group was 87, and the mean systolic blood pressure and diastolic blood pressure were 110 and 66.5, respectively, while the mean systolic blood pressure and diastolic blood pressure in the survived group were 117 and 71.5. However, there was not a statistically significant distinction between the cases that survived and those that did not in terms of temperature, heart rate, or the AA Gradient score(p>0.05).

King et al.,[9] they found that in alive group, maximal temperature was 37.8±1.1 and minimal mean arterial pressure was 73.1±22.1. While, in dead group, maximal temperature was 38.1±1.4 and minimal mean arterial pressure was 59.5±15.9. There was significant difference as regard minimal mean arterial pressure between a live group and dead group while there was no significant difference as regard maximal temperature.

By comparing the mean heart rates of troponin-positive and troponin-negative patients, we found a statistically significant difference(p-value<0.05) in terms of heart rate and PO2 score, with troponin-positive cases having a mean heart rate of 112 and troponin-negative cases having a mean heart rate of 104.8. In terms of PO2 score grade-0, 47.7% of troponin-negative cases and 28% of troponin-positive cases were similar. Regarding MAP, SBP, DBP, temperature, and the AA Gradient score, there was no statistically significant difference between the positive and

negative cases of Troponin (p>0.05).

In our findings, there was a statistically significant difference between troponin-positive and negative cases with regard to heart rate and PO2 score(p-value<0.05), as the mean heart rate in troponin-positive cases was 112, compared to 104.8 in troponin-negative cases. For the PO2 score, 47.7% of troponin-negative cases had a PO2 score grade 0 compared to 28% of troponin-positive cases. There was no significant difference between troponin-positive and negative cases regarding MAP, SBP, DBP, and temperature, as well as the AA Gradient score(p-value>0.05).

In consistent with our results, Wu et al.,[10] they found that in the Elevated cTnl (n=49) group, mean arterial blood pressure (mmHg) was 84.2±15.5, Heart rate (beat/min) was 112.2±25.8, Respiratory rate(times/min) was 22.9±6.8, PaO2/FiO2(mmHg) was 216.5±133.2, and Temperature(°C) was 37.4±1.2. In Normal cTnl(n=59) group, mean arterial blood pressure (mmHg) was 88.9±12.7, Heart rate(beat/min) was 112.2±25.8, Respiratory rate (times/min) 22.9±6.8, PaO2/FiO2(mmHg) was 253.1±130.9 and Temperature(°C) was 37.1±1.2. There was no significant difference regarding Mean arterial blood pressure, Respiratory rate, PaO2/FiO2, and Temperature(°C).

In our study, we assessed the area under the curve (AUC) for the predictive performance of the APACHE II score, Troponin I, and their combination. We found that the APACHE II score had a strong predictive value, with an AUC of 0.78. In contrast, Troponin I alone showed weak predictive ability, with an AUC of 0.37. However, the combination of APACHE II and Troponin I significantly improved predictive performance, demonstrating the added value of incorporating Troponin I into mortality prediction models.

Comparing to the results of the current study, Babuin et al.[8] examined the role of troponin elevation in ICU mortality using APACHE III. Found that high troponin levels correlated with worse outcomes, similar to our study. However, their troponin levels were elevated in a higher proportion of ICU patients (61.4% vs. 36% in our study).

Ghorbani et al.[5] assessed the predictive value of APACHE-IV. Found a higher AUC (~0.85) than APACHE II alone in your study (0.78), but their model did not incorporate Troponin.

Even in the absence of acute coronary syndrome (ACS), critically ill patients can experience myocardial ischemia due to a supply-demand mismatch. Conditions such as hypoxia, anemia, and tachycardia can lead to myocardial oxygen imbalance, contributing to troponin elevation. This is particularly relevant in patients with sepsis, respiratory failure, or shock.

Inflammation and cytokine storms in critically ill patients, especially in sepsis or viral infections, can lead to myocarditis, resulting in myocardial injury and elevated troponin levels. Stress-induced cardiomyopathy (Takotsubo syndrome) is another possibility, where high levels of catecholamines cause transient myocardial dysfunction and troponin release.

In conditions such as septic shock or cardiogenic shock, prolonged hypotension and systemic inflammation can lead to myocardial dysfunction. Heart failure, whether pre-existing or developed due to critical illness, can cause myocardial strain and troponin leakage.

Sepsis induces a systemic inflammatory response that can lead to direct myocardial depression, microvascular dysfunction, and increased troponin release. Studies suggest that troponin elevation in sepsis correlates with worse outcomes, independent of coronary artery disease.

Patients with chronic kidney disease, pulmonary embolism, and stroke may also have elevated Troponin due to non-ischemic mechanisms like endothelial dysfunction, small-vessel disease, or increased left ventricular strain.

The results of the present study indicate a significant difference in mortality based on Troponin I levels, with 75% of non-survivors having positive Troponin versus 21% of survivors. This aligns with Babuin et al.[8] who stated that elevated troponin levels were independently associated with higher mortality in ICU patients.

Limitations of the Study:

First: The study included only the patient who admitted in the intensive care for a medical reason excluding any admission for a surgical cause, limiting the prognostic value of the score for these patients, which actually dependent on the interventions they will have.

Second: The qualitative value of Troponin I overestimates the rate of mortality for patients who may have had a positive basic Troponin, as patients had chronic kidney disease.

Third: The cost of follow-up Troponin and other investigation during the first 24-hour of admission to record the worst value wasn't available for all the patients.

Finally: Although the study included 200-patients, but a larger scope of studies is required to determine whether the addition of Troponin I to the APACHE II score is of a true beneficial value.

Recommendations: It's recommended to add Troponin I to the Apache II score to for the intensive care medical patients to assess their predication of mortality.

It is recommended that future studies be conducted using well-designed randomized controlled trials or large, comparative observational studies.

Inclusion of a representative sample of patients with similar age, gender, and disease severity. The sample size of future studies should be large enough to provide meaningful conclusions and to control for confounding factors.

To accurately assess long-term outcomes, studies should have a longer follow-up period. We recommended that future research should include multicenter studies to validate our findings.

4. Conclusion

The findings of this study demonstrate that incorporating Troponin I levels into the APACHE II score significantly enhances its predictive accuracy for mortality in critically ill non-surgical ICU patients. The observed 11.25% improvement in predictive value suggests that Troponin I provides additional prognostic information beyond the traditional APACHE II scoring system.

From a clinical practice perspective, these results highlight the potential benefits of integrating Troponin I testing into routine ICU risk assessments. By identifying high-risk patients more accurately, healthcare providers can implement earlier and more targeted interventions, optimize resource allocation, and improve patient outcomes. Future guidelines for ICU risk stratification may consider the inclusion of Troponin I as a standard biomarker in severity scoring systems.

Further research is warranted to validate these findings in larger and more diverse populations and to explore the potential for refining critical care protocols based on Troponin I levels.

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