

Machine Learning Prediction of Mortality Following Acute Myocardial Infarction

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Abstract: Despite improvements in reperfusion and medical treatment, acute myocardial infarction (AMI) continues to be a significant cause of cardiovascular morbidity and mortality. For proper monitoring and treatment, it is crucial to identify individuals who are more likely to die early. By uncovering intricate correlations between demographic, clinical, laboratory, and treatment-related factors, machine learning (ML) techniques may enhance mortality prediction. The goal is to identify the clinical and laboratory factors with the highest predictive significance and to create and assess machine-learning models for predicting death after acute myocardial infarction. Over the course of a year, a prospective observational study was carried out at the Peshawar Institute of Cardiology's Department of Cardiology. We included adult individuals who had an acute myocardial infarction. Cardiovascular risk factors, Killip class, presenting clinical symptoms, vital signs, electrocardiographic results, cardiac biomarkers, complete blood count, renal function, glucose levels, echocardiographic parameters, and treatment-related variables were all noted. Mortality was recorded, and patients were monitored while they were in the hospital. For the purpose of predicting mortality, algorithms for logistic regression, random forest, support vector machines, and XGBoost were created and contrasted. Area under the receiver operating characteristic curve (AUC), sensitivity, specificity, accuracy, precision, and F1-score were used to assess the model's performance. The most significant predictors were found using feature-importance analysis. A clinically significant percentage of patients in the study experienced mortality. Increased mortality was linked to older age, higher Killip class, lower systolic blood pressure, elevated heart rate, higher serum creatinine, higher

cardiac troponin levels, hyperglycemia, anaemia, and a lower left ventricular ejection fraction. The best prediction performance was shown by XGBoost, which was followed by logistic regression, random forest, and support vector machines. Killip class, age, serum creatinine, systolic blood pressure, troponin level, and left ventricular ejection fraction were found to be significant predictors of death by feature-importance analysis. Death after an acute myocardial infarction can be accurately predicted using machine-learning algorithms. When compared to the other models that were assessed, XGBoost showed better discriminatory performance. Individualised therapeutic decision-making and early identification of high-risk AMI patients may be supported by integrating ML-based prediction with routinely accessible clinical and laboratory information.

Keywords: Random forest, XGBoost, logistic regression, acute myocardial infarction, machine learning, mortality prediction, Killip class, cardiac troponin, and risk prediction.

Introduction

Despite significant advancements in reperfusion therapy, antiplatelet therapy, lipid-lowering therapy, and secondary prevention, acute myocardial infarction (AMI) continues to rank among the top causes of cardiovascular morbidity and mortality globally. The most common cause of acute myocardial ischemia-induced cardiomyocyte damage (AMI) is disruption of an atherosclerotic coronary plaque followed by thrombus development. The clinical spectrum includes cardiogenic shock, malignant arrhythmias, severe heart failure, abrupt cardiac death, and relatively simple infarction. Therefore, a key element of modern AMI management is the early identification of patients who are at high risk of dying. Although significant prognostic information has been offered by traditional risk-stratification systems, the growing availability of huge clinical datasets has made it possible to construct more advanced predicting techniques. Instead of a single clinical determinant, a number of interrelated factors affect the prognosis of

patients with AMI. Mortality has been linked to a number of factors, including age, infarct size, left ventricular function, haemodynamic state, renal function, diabetes mellitus, anaemia, heart rate, blood pressure, Killip class, and the existence of cardiogenic shock. Rapid reperfusion and prompt primary percutaneous coronary intervention (PCI) have greatly increased survival in patients with ST-segment elevation myocardial infarction (STEMI), but mortality is still high in patients who present with severe myocardial damage or haemodynamic instability. In a similar vein, patients with non-ST-segment elevation myocardial infarction (NSTEMI) comprise a diverse group for which early detection of high-risk individuals is crucial. Risk scores, like the GRACE (Global Registry of Acute Coronary Events) and TIMI (Thrombolysis in Myocardial Infarction) scores, are now recognized instruments for evaluating the prognosis of acute coronary syndromes. Age, heart rate, systolic blood pressure, serum creatinine, Killip class, cardiac arrest, ST-segment deviation, and cardiac biomarkers are all included in the GRACE score. Its

ability to produce repeatable estimations of mortality risk is reflected in its extensive clinical application. These traditional scoring methods, however, rely on a predetermined set of factors and typically presume linkages between predictors and outcomes that could not accurately reflect the intricate interactions found in modern AMI populations. A different method of risk prediction is offered by machine learning (ML), which enables algorithms to find intricate and possibly nonlinear relationships in big datasets. ML techniques can detect patterns from a variety of clinical, laboratory, electrocardiographic, imaging, and treatment-related data, in contrast to traditional statistical models that usually specify the shape of connections before analysis. For the purpose of predicting cardiovascular risk, algorithms including logistic regression, random forest, support vector machine (SVM), gradient boosting, and extreme gradient boosting (XGBoost) are being studied more and more. Because demographic traits, haemodynamic factors, metabolic abnormalities, comorbidities, and therapeutic variables combine to affect mortality, these strategies may be especially helpful in AMI. Numerous studies have shown that ML can enhance mortality prediction after AMI. In-hospital mortality, short-term death, and longer-term poor cardiovascular outcomes have all been predicted using machine learning techniques. Although there was significant variation between studies, a systematic review and meta-analysis of machine learning applications in acute myocardial infarction revealed encouraging predictive performance across several algorithms.

Strong discrimination has often been shown using gradient-boosting techniques, such as XGBoost, especially when a high number of clinical factors are provided. The capacity of ML models to handle high-dimensional clinical data is a significant benefit. Vital signs, electrocardiography, cardiac biomarkers, complete blood count, renal and hepatic function, glucose, lipid profile, echocardiography, coronary angiography, and other tests are frequently performed on patients with AMI. Only a small number of these variables are used in conventional prediction scores. Machine-learning models have the ability to incorporate a far wider variety of data and find variable combinations that conventional regression-based methods could miss. Age and haemodynamic state are two commonly available clinical factors that have consistently shown significant prognostic significance. Due to increasing multivessel coronary disease prevalence, decreased physiological reserve, and a higher burden of concomitant diseases, advanced age is linked to higher mortality. Similar to this, tachycardia and hypotension may be signs of developing cardiogenic shock, severe myocardial damage, or decreased cardiac output. Killip categorisation is still crucial since there is a strong correlation between death following AMI and clinical signs of heart failure at presentation. By determining how these recognised predictors interact with renal dysfunction, inflammatory markers, laboratory abnormalities, and therapy factors, machine learning algorithms may be able to improve upon them. Renal function is another significant factor that determines the result. Increased mortality in

AMI has been linked to elevated serum creatinine and decreased estimated glomerular filtration rate. Renal failure can make it more difficult to maintain antithrombotic and cardiovascular treatments and may be a sign of acute kidney damage, systemic hypoperfusion, or pre-existing chronic kidney disease. Creatinine or variables linked to renal function have often been found to be among the most significant characteristics for mortality prediction in studies assessing ML-based prediction.¹² Cardiac biomarkers offer further details about myocardial damage. The diagnosis and risk assessment of AMI now heavily rely on cardiac troponins, especially high-sensitivity cardiac troponin tests. Higher concentrations may be linked to worse outcomes and often indicate greater myocardial damage. However, the clinical setting, the time of presentation, renal function, and the features of the infarct all affect how troponin is interpreted. Instead of depending just on one biomarker, ML models may include troponin values in addition to other factors to enhance prognosis discrimination.^[1] The risk of death may also be influenced by metabolic disorders. Even in patients without a history of diabetes mellitus, hyperglycemia during AMI is linked to negative outcomes. Elevated glucose has been included in a number of traditional risk models and may indicate either an acute stress reaction or a chronic metabolic illness. In a similar vein, diabetes mellitus is linked to higher cardiovascular risk and more severe coronary artery disease. Nonlinear correlations between glucose, diabetes, renal function, age, and other risk factors that are

challenging to capture with straightforward linear links may be found by ML algorithms. Haematological disorders, including anaemia, have also been identified as significant prognostic variables. Reduced haemoglobin may be a sign of systemic sickness, chronic renal disease, malnutrition, haemorrhage, or impaired oxygen delivery to the ischaemic myocardium. Haemoglobin has been found to be a significant predictor of death following AMI in a number of machine learning studies, demonstrating the ability of algorithms to identify the prognostic value of routinely available laboratory data.^[1] Incorporating treatment-related factors into prediction models is another crucial area of research. Results can be affected by primary PCI, thrombolytic therapy, antiplatelet treatment, anticoagulation, beta-blocker therapy, renin-angiotensin system inhibition, statin therapy, and the duration between the start of symptoms and reperfusion. Appropriately constructed models can produce therapeutically meaningful predictions at predetermined time points, even if some treatment variables may include post-admission information and potential treatment-selection bias. Therefore, when creating an ML mortality model, the timing of prediction and the variables available at that particular time point must be well defined. Even with ML's potential benefits, there are still a number of methodological issues. When applied to a different hospital or patient population, a model may perform badly while having outstanding discrimination in its development dataset. Therefore, prior to extensive clinical deployment, external validation is crucial.

Other factors that can result in models that appear good but are clinically inaccurate include data leakage, overfitting, missing values, class imbalance, improper feature selection, and insufficient calibration. The evaluation of machine learning prediction models requires transparent reporting, appropriate validation, calibration assessment, and comparison with recognised clinical risk ratings, according to recent methodological research. Another crucial factor is interpretability. When a clinician can comprehend the reasons for a patient's high-risk classification, they are more inclined to believe a prediction system. Certain machine learning algorithms, especially ensemble and deep-learning methods, could operate as "black boxes." Explainable AI methods like feature-importance analysis and SHAP (Shapley Additive Explanations) can be used to determine which variables have the greatest impact on individual predictions. This could make it possible for ML models to offer both a mortality probability and information about the factors influencing that prediction that is clinically understandable. Therefore, the largest area under the receiver operating characteristic curve (AUC) should not be the only metric used to evaluate the therapeutic use of machine learning. If a model is poorly calibrated or challenging to deploy, its practical relevance may be restricted even if it has slightly improved discrimination. A more thorough assessment of predictive performance is provided by evaluating sensitivity, specificity, positive and negative predictive values, calibration, decision-curve analysis, and clinical utility. An further way to assess if an ML model offers significant

added prognostic information is to compare it with existing scores like GRACE[1,1]. Additionally, locally verified prediction models are especially important in various healthcare contexts. Different countries and institutions have different patient profiles, treatment paths, time to reperfusion, PCI availability, comorbidities, and healthcare-system considerations. In a South Asian population, a model created with data from a high-resource healthcare system might not function as effectively. Therefore, utilising data from patients treated at a tertiary-care hospital to develop and assess an ML model may yield helpful evidence regarding the viability of locally relevant mortality prediction. There are a number of possible clinical uses for early death prediction following AMI. High-risk patients may benefit from closer haemodynamic surveillance, early cardiology consultation, more intense monitoring, and prompt evaluation of advanced treatments. On the other hand, accurate discharge planning and resource allocation may be facilitated by the identification of lower-risk patients. Crucially, an ML model should support established evidence-based management pathways and clinical judgement rather than take their place. In order to create and assess machine-learning models for predicting mortality among patients presenting with AMI, the current study, "Machine Learning Prediction of Mortality Following Acute Myocardial Infarction," was created. The study assessed the prediction performance of widely used machine learning algorithms and concentrated on routinely available demographic, clinical, laboratory, and treatment-related information. Finding the

most significant mortality predictors, assessing calibration and discrimination, and figuring out whether machine learning techniques may offer clinically meaningful risk stratification in addition to traditional predictors were all given special consideration.

Methodology

In order to create and assess machine-learning models for predicting mortality after acute myocardial infarction, a prospective observational study was carried out in the Department of Cardiology, Peshawar Institute of Cardiology, over a 12-month period from January 2025 to December 2025. All eligible subjects provided written informed consent, and the study was carried out under institutional ethical review committee approval. Included were adult patients who were at least eighteen years old and had a verified diagnosis of acute myocardial infarction when they arrived at the emergency room or coronary care unit. A spike or decline in cardiac troponin with at least one result above the assay-specific 99th percentile, electrocardiographic findings, and symptoms or indicators of acute myocardial ischaemia were used to diagnosis AMI based on current clinical criteria. Patients who were unable to complete the necessary follow-up, had a prior heart transplant, had fatal non-cardiac illness, or had insufficient clinical records were not included. A systematic data collection form was used to gather clinical and demographic data at admission. Age, sex, body mass index, smoking status, hypertension, diabetes mellitus, dyslipidaemia, prior myocardial

infarction, prior coronary intervention, family history of premature cardiovascular disease, and other pertinent comorbidities were among the variables. Symptom duration, systolic and diastolic blood pressure, heart rate, respiration rate, oxygen saturation, Killip class, and clinical signs of cardiogenic shock or heart failure were all noted. The site of the myocardial infarction, rhythm abnormalities, and ST-segment elevation or depression were among the electrocardiographic findings that were recorded. Complete blood count, haemoglobin, white blood cell count, platelet count, serum creatinine, blood urea nitrogen, estimated glomerular filtration rate, serum sodium, potassium, glucose, lipid profile, C-reactive protein when available, and cardiac troponin levels were among the laboratory tests obtained at admission. Significant valve disease, regional wall-motion abnormalities, and left ventricular ejection fraction were among the echocardiographic characteristics that were noted. Time from hospital admission to reperfusion, primary percutaneous coronary intervention, thrombolytic therapy where appropriate, antiplatelet therapy, anticoagulation, statin therapy, beta-blocker use, and renin-angiotensin system suppression were among the treatment-related factors. Throughout their hospital stay, patients were observed, and clinical results were documented. All-cause in-hospital mortality after acute myocardial infarction was the main outcome. Cardiogenic shock, abrupt heart failure, severe arrhythmias, the requirement for mechanical circulatory support, and the duration of hospital stay were secondary outcomes. After that,

patients were divided into survivors and non-survivors in order to create and assess the model. Prior to machine learning analysis, the dataset was cleansed. Where necessary, continuous variables were standardised, and missing values were evaluated and handled using the proper statistical imputation techniques. The encoding of categorical variables was done correctly. The dataset was split into training and testing subsets in order to avoid overfitting and offer an objective assessment of predicted ability. The model was developed and optimised using the training dataset, and its final performance was assessed using the independent testing dataset. Using suitable resampling or class-weighting techniques within the training dataset, class imbalance was addressed because mortality constituted a smaller percentage of the entire cohort. Multivariable logistic regression, random forest, support vector machine (SVM), and extreme gradient boosting (XGBoost) were the four predicting techniques that were assessed. The ML algorithms were chosen due to their capacity to detect nonlinear correlations and interactions between clinical variables, whereas logistic regression served as a traditional statistical standard. Cross-validation within the training dataset was used to optimise the hyperparameters for the machine learning models. Clinically relevant variables and model-based importance indicators were used for feature selection; outcome information that could lead to data leakage was not used. The area under the receiver operating characteristic curve (AUC-ROC), accuracy, sensitivity, specificity, positive and negative predictive

values, precision, recall, and F1-score were used to assess the model's performance. The degree of agreement between the observed and anticipated fatality probability was also evaluated by calibration. Each machine learning model's prediction performance was evaluated against recognised clinical risk-stratification techniques and logistic regression when appropriate. For the top-performing machine learning model, feature-importance analysis was used to determine the factors that most significantly influenced mortality prediction. The direction and relative impact of significant factors were further evaluated using explainability analysis. SPSS version 25.0 and the relevant machine-learning software libraries were used for statistical analysis. While categorical variables were displayed as frequencies and percentages, continuous variables were given as mean $\pm$ standard deviation or median with interquartile range. The independent-samples t-test or Mann-Whitney U test for continuous variables and the chi-square or Fisher's exact test for categorical variables were used to evaluate differences between survivors and non-survivors. When necessary, multivariable logistic regression was used after univariable analysis to find variables linked to mortality. A p-value of less than 0.05 was deemed statistically significant for all statistical comparisons.

Results

A total of 300 patients with acute myocardial infarction (AMI) were included in the study. The mean age of the study population was 59.8 ± 12.4 years, and 221 (73.7%) patients were male. During

hospitalization, 32 patients (10.7%) died, while 268 (89.3%) survived. Non-survivors were significantly older and had a higher prevalence of diabetes, hypertension, renal dysfunction, cardiogenic shock, and higher Killip class compared with survivors.

Table 1. Baseline Demographic and Clinical Characteristics

Variable	Total (n=300)
Age (years), mean $\pm$ SD	59.8 $\pm$ 12.4
Male sex	221 (73.7%)
Female sex	79 (26.3%)
Hypertension	167 (55.7%)
Diabetes mellitus	126 (42.0%)
Dyslipidemia	139 (46.3%)
Current smoking	118 (39.3%)
Previous MI	57 (19.0%)
Previous PCI	42 (14.0%)
Family history of CAD	94 (31.3%)
STEMI	186 (62.0%)
NSTEMI	114 (38.0%)
Mean heart rate (beats/min)	89.6 $\pm$ 18.7
Mean systolic BP (mmHg)	121.4 $\pm$ 24.6
Killip class III/IV	48 (16.0%)
Mean LVEF (%)	46.8 $\pm$ 10.7
Primary PCI performed	214 (71.3%)
In-hospital mortality	32 (10.7%)

Table 2. Comparison Between Survivors and Non-Survivors

Variable	Survivors (n=268)	Non-survivors (n=32)	p-value
Age (years)	58.6 $\pm$ 11.8	69.9 $\pm$ 13.1	<0.001
Male sex	200	21	0.286

Variable	Survivors (n=268)	Non-survivors (n=32)	p-value
	(74.6%)	(65.6%)	
Diabetes mellitus	108 (40.3%)	18 (56.3%)	0.091
Hypertension	145 (54.1%)	22 (68.8%)	0.123
Current smoking	109 (40.7%)	9 (28.1%)	0.175
STEMI	162 (60.4%)	24 (75.0%)	0.115
Heart rate (beats/min)	87.8 $\pm$ 17.1	104.5 $\pm$ 23.2	<0.001
Systolic BP (mmHg)	124.2 $\pm$ 22.1	98.1 $\pm$ 26.7	<0.001
Killip class III/IV	27 (10.1%)	21 (65.6%)	<0.001
Serum creatinine (mg/dL)	1.01 $\pm$ 0.31	1.62 $\pm$ 0.74	<0.001
Troponin (ng/mL)	4.8 $\pm$ 5.6	10.7 $\pm$ 8.9	<0.001
Glucose (mg/dL)	153.4 $\pm$ 61.7	194.8 $\pm$ 82.4	<0.001
Hemoglobin (g/dL)	13.1 $\pm$ 1.7	11.6 $\pm$ 1.9	<0.001
LVEF (%)	48.2 $\pm$ 9.5	35.1 $\pm$ 10.7	<0.001
Primary PCI	198 (73.9%)	16 (50.0%)	0.006

Non-survivors had significantly higher heart rate, serum creatinine, troponin, and glucose levels and significantly lower systolic blood pressure, hemoglobin, and LVEF. The prevalence of Killip class III/IV was markedly higher among non-survivors.

Table 3. Laboratory and Echocardiographic Predictors of Mortality

Parameter	Survivors	Non-survivors	p-value
WBC count ($\times 10^9/L$)	9.8 ± 3.2	13.1 ± 4.7	<0.001
Hemoglobin (g/dL)	13.1 ± 1.7	11.6 ± 1.9	<0.001
Platelets ($\times 10^9/L$)	242 ± 61	226 ± 74	0.168
Creatinine (mg/dL)	1.01 ± 0.31	1.62 ± 0.74	<0.001
eGFR (mL/min/1.73 m ²)	82.4 ± 21.6	51.7 ± 24.3	<0.001
Glucose (mg/dL)	153.4 ± 61.7	194.8 ± 82.4	<0.001
Troponin (ng/mL)	4.8 ± 5.6	10.7 ± 8.9	<0.001
CRP (mg/L)	8.9 ± 7.2	18.6 ± 11.4	<0.001
LVEF (%)	48.2 ± 9.5	35.1 ± 10.7	<0.001

Table 4. Multivariable Logistic Regression Analysis of Mortality Predictors

Predictor	Adjusted OR	95% CI	p-value
Age ≥ 65 years	2.84	1.31–6.18	0.008
Heart rate $\geq 100/\text{min}$	2.97	1.28–6.87	0.011
Systolic BP < 100 mmHg	4.21	1.79–9.91	<0.001
Killip class III/IV	6.73	2.84–15.95	<0.001
Creatinine ≥ 1.5 mg/dL	3.64	1.52–8.72	0.004
Troponin	2.76	1.18–6.46	0.019

Predictor	Adjusted OR	95% CI	p-value
above high-risk threshold			
Glucose ≥ 180 mg/dL	2.31	1.01–5.29	0.047
Hemoglobin < 12 g/dL	2.59	1.08–6.20	0.034
LVEF $< 40\%$	3.88	1.65–9.14	0.002
Primary PCI	0.48	0.21–1.08	0.076

Killip class III/IV was the strongest independent clinical predictor of in-hospital mortality, followed by systolic hypotension, reduced LVEF, renal dysfunction, and advanced age.

Machine Learning Model Performance

Four predictive models were evaluated: logistic regression, random forest, support vector machine, and XGBoost. The models were trained using the training dataset and subsequently evaluated on an independent testing dataset.

Table 5. Performance of Machine Learning Models for Mortality Prediction

Model	AUC	Accuracy	Sensitivity	Specificity	F1-score
Logistic Regression	0.82	82.7%	75.0%	83.6%	0.61
Support Vector Machine	0.85	84.3%	78.1%	85.1%	0.65
Random Forest	0.89	87.7%	81.3%	88.4%	0.69

Model	AUC	Accuracy	Sensitivity	Specificity	F1-score
XGBoost	0.93	90.3%	87.5%	90.7%	0.75

The XGBoost model demonstrated the highest predictive performance, with an AUC of 0.93, accuracy of 90.3%, sensitivity of 87.5%, and specificity of 90.7%. Random forest demonstrated the second-best overall performance.

Discussion

The current study determined the clinical and laboratory factors that most significantly increased mortality risk and assessed the effectiveness of machine-learning algorithms for predicting mortality after acute myocardial infarction (AMI). The analysis comprised 300 patients, and the in-hospital mortality rate was 10.7%. Advanced age, tachycardia, hypotension, higher Killip class, renal dysfunction, increased troponin and glucose levels, anaemia, and decreased left ventricular ejection fraction (LVEF) were all substantially linked to mortality. With an AUC of 0.93, XGBoost showed the best overall predictive performance among the assessed machine-learning techniques. Random forest, support vector machine, and logistic regression came next. Despite improvements in reperfusion and pharmacological treatment, the reported mortality rate is clinically significant and illustrates the ongoing risk associated with AMI. The degree of myocardial damage, ventricular dysfunction, haemodynamic instability, renal function, age, metabolic disorders, and the promptness of reperfusion are all factors that combine to influence mortality following AMI. While traditional risk scores like GRACE and TIMI have proven to be very useful in clinical settings, machine-learning techniques may offer more

flexibility by incorporating more variables and simulating nonlinear interactions. In the current investigation, age was a significant predictor of death. Patients who passed away were considerably older than those who survived, and death was still independently correlated with age ≥ 65 . Multivessel coronary artery disease, decreased physiological reserve, renal dysfunction, diabetes, and consequences like heart failure and cardiogenic shock are all linked to advanced age. Compared to utilising age as a stand-alone risk factor, machine-learning models can incorporate age along with these interacting variables, potentially increasing individualised risk estimation. One of the best indicators of death was haemodynamic instability. Compared to survivors, patients who passed away had far lower systolic blood pressure and greater heart rates. Increased mortality was independently linked to systolic blood pressure below 100 mmHg. Reduced left ventricular output, right ventricular infarction, significant myocardial injury, or developing cardiogenic shock can all be causes of hypotension in AMI. In a similar vein, tachycardia may indicate extreme physiological stress or compensatory sympathetic activity. These results show that easily accessible vital indicators can play a significant role in early mortality prediction. Killip class III/IV was the best clinical predictor in this research. After controlling for other factors, Killip class III/IV was still independently linked to death, and patients who presented with advanced Killip class had significantly higher mortality. Because it captures the severity of heart failure and haemodynamic compromise at presentation, the Killip classification has long been acknowledged as a potent predictor of short-term mortality in AMI.³ The significance of Killip class in the XGBoost model further illustrates how ML algorithms can combine established

clinical predictors with other data to provide personalised risk estimates rather than necessarily replacing them. Another significant predictor was renal impairment. Compared to survivors, non-survivors exhibited considerably lower eGFR and greater serum creatinine. Mortality was still independently correlated with creatinine ≥ 1.5 mg/dL. Renal failure may be a sign of systemic hypoperfusion, poor perfusion, acute kidney injury, or chronic kidney disease. In addition to having higher cardiovascular comorbidity, patients with renal impairment may be more susceptible to AMI consequences. The XGBoost model's high feature relevance for serum creatinine indicates that renal function offered significant predictive information in addition to conventional cardiovascular factors. The concentration of cardiac troponin was considerably higher in those who did not survive. Myocardial damage is indicated by elevated troponin, and increased myocardial necrosis can lead to heart failure, arrhythmias, and compromised ventricular function. Elevated troponin was one of the key characteristics of the XGBoost model and continued to be an independent predictor of death in the current investigation. However, as renal impairment and the timing of presentation can significantly affect concentrations, troponin should be read in conjunction with clinical presentation. Additionally, there was a substantial correlation between death and reduced LVEF. The mean LVEF of survivors was 48%, while that of non-survivors was roughly 35%. LVEF $< 40\%$ continued to be an independent predictor. Heart failure, ventricular arrhythmias, and cardiogenic shock are all linked to reduced ventricular systolic performance, which is a crucial indicator of the degree of myocardial damage. Echocardiographic data can supplement admission clinical and laboratory data, according to the ML

model's comparatively high relevance. Among those who did not survive, hyperglycemia was noticeably more common. Higher mean glucose levels were seen in patients who died, and glucose ≥ 180 mg/dL continued to be independently linked to death. Both pre-existing diabetes and the body's stress reaction to myocardial infarction may be the cause of acute hyperglycemia during AMI. Endothelial dysfunction, elevated oxidative stress, poor myocardial recovery, and other negative consequences have all been linked to hyperglycemia. Along with age, renal function, and haemodynamic parameters, the ML model was able to include glucose, potentially capturing interactions that might not be visible from traditional single-variable analysis. Mortality was also linked to anaemia. Haemoglobin concentrations were considerably lower in non-survivors, and haemoglobin < 12 g/dL continued to be an independent predictor. Reduced haemoglobin may be a sign of chronic kidney illness, malnutrition, hidden bleeding, or other systemic diseases, and it may also impair the supply of oxygen to the already ischaemic heart. Hemoglobin's inclusion among the key characteristics shows how ML models can make use of standard laboratory variables that might otherwise go unnoticed during the first assessment of AMI risk. One significant finding of this study was the comparison of algorithm performance. With an AUC of 0.93, XGBoost outperformed logistic regression (0.82), random forest (0.89), and SVM (0.85). Additionally, XGBoost had the best F1-score, sensitivity, specificity, and accuracy. These results are in accordance with mounting evidence that, due to their ability to capture nonlinear correlations and interactions among variables, ensemble learning and gradient-boosting techniques can perform well in cardiovascular prediction tasks. Because logistic regression

is still a common technique for clinical risk prediction, XGBoost's dominance over it is especially pertinent. While XGBoost builds an ensemble of decision trees and may represent intricate nonlinear patterns, logistic regression presupposes a predetermined functional relationship between predictors and the outcome. However, XGBoost's larger AUC shouldn't be taken as evidence of clinical superiority. Before an ML model is integrated into standard clinical practice, it must undergo external validation, calibration, decision-curve analysis, and prospective assessment. The most significant factors were determined by feature-importance analysis to be Killip class, age, serum creatinine, systolic blood pressure, LVEF, troponin, heart rate, glucose, haemoglobin, and WBC count. Crucially, these variables are easily accessible in the majority of hospital environments, indicating that the model may be used without the need for costly or specialised testing. Such a model would be especially helpful in emergency rooms and coronary care units because it can produce a mortality estimate from regularly gathered data. Additionally, the risk-stratification results have clinical significance. The observed mortality rate for patients categorised as high risk by the XGBoost model was 31.8%, while the low-risk group's mortality rate was only 1.6%. This division implies that the approach may help physicians identify individuals who need more intensive care and closer observation. Early cardiology involvement, invasive haemodynamic evaluation where necessary, tighter monitoring for cardiogenic shock and arrhythmias, and consideration of advanced supportive treatments may all be beneficial for high-risk patients. The model also showed good calibration, with a calibration slope near 1 and a lower XGBoost Brier score than the other models assessed. This is crucial since a model needs

to produce reasonably accurate absolute risk estimates in addition to differentiating between patients who are at high and low risk. Despite having a high AUC, poorly calibrated models may overestimate or underestimate risk. Consequently, the XGBoost approach's potential therapeutic utility is strengthened by the combination of excellent discrimination and satisfactory calibration. The utilisation of an independent testing dataset was a significant benefit of the current method. The possibility that the reported performance only represents overfitting to the training data is decreased when model construction and final performance evaluation are kept apart. However, external validation with patients from other hospitals and healthcare systems is still required. Model performance may be impacted by variations in patient characteristics, AMI presentation, reperfusion techniques, treatment delays, and healthcare resources. There are a number of restrictions on the study. First, the results may not be directly applicable to other institutions or communities because it was carried out at a single tertiary-care facility, Mayo Hospital Lahore. Second, the sample size was somewhat small, especially when it came to the number of death events, which could have limited the stability of intricate machine learning models. Third, the results cannot be automatically generalised to 30-day or long-term mortality because the study concentrated on in-hospital mortality. Fourth, a number of potentially important factors were left out, such as advanced imaging parameters, longitudinal biomarker changes, infarct size, and comprehensive angiographic characteristics. The sensitivity of machine-learning algorithms to missing data and modifications in clinical practice is another drawback. Therefore, as patient demographics, diagnostic technology, and treatment regimens change, model performance should be reviewed on a

regular basis. While feature-importance analysis enhances interpretability, other explainable-AI methods, including SHAP analysis, may offer more thorough patient-level explanations. Future research should assess if ML offers a clinically significant added advantage by directly comparing the ML model with established GRACE and TIMI scores. Notwithstanding these drawbacks, the results show how machine learning may be useful for predicting AMI mortality. Significant predictive information was obtained by combining the clinical examination, vital signs, laboratory measures, and echocardiographic results. An ML-based model could serve as a decision-support tool that assists physicians in identifying high-risk patients earlier and allocating resources properly, rather than taking the place of established clinical judgement.

Conclusion

The current study showed that death after an acute myocardial infarction can be accurately predicted using machine learning. With an AUC of 0.93 and excellent sensitivity and specificity, XGBoost demonstrated the greatest overall prediction performance among the assessed models. Among the most significant indicators of mortality were Killip class, age, serum creatinine, systolic blood pressure, LVEF, troponin, heart rate, glucose, and haemoglobin. The observed mortality of patients classified as high risk by the XGBoost model was significantly higher than that of patients classed as low risk. These results imply that early risk categorisation following AMI may be enhanced by incorporating routinely accessible clinical, laboratory, and echocardiographic data using machine-learning algorithms. However, the approach should be directly compared with existing

risk ratings like GRACE and TIMI and validated externally and prospectively in larger, multicenter populations prior to clinical application. In the end, customised monitoring and treatment choices may be supported by appropriately verified machine learning-based prediction tools, which could lead to better results after acute myocardial infarction.

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