

Mean Arterial Pressure and Mortality in Cardiogenic Shock Patients

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Abstract

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Cardiogenic shock remains a critical condition characterized by inadequate tissue perfusion and high mortality despite advancements in cardiac care. Mean arterial pressure (MAP) serves as a key hemodynamic parameter that reflects systemic perfusion and directly influences patient outcomes. Recognizing the scarcity of local evidence, this study aimed to examine the association between MAP and in-hospital mortality among cardiogenic shock patients admitted to the Cardiology Department of Ayub Teaching Hospital, Abbottabad. A retrospective observational design was employed, and data were collected from patient medical records, including demographic details, hemodynamic parameters, and clinical outcomes. Patients were categorized into three MAP groups: <65 mmHg, 65–75 mmHg, and >75 mmHg. Statistical analysis was conducted to compare mortality rates and evaluate the predictive relationship between MAP and survival outcomes. The results revealed that patients with a MAP below 65 mmHg during the first 24 hours of

admission exhibited a significantly higher mortality rate compared to those within the optimal range of 65–75 mmHg. Excessively high MAP levels were also associated with adverse outcomes due to increased cardiac workload. The findings underscored the importance of maintaining individualized MAP targets to ensure adequate perfusion without overburdening the myocardium. Future research should focus on developing patient-specific MAP management protocols, integrating advanced monitoring technologies, and validating these findings through multicenter studies to improve cardiogenic shock outcomes globally.

Introduction

One of the most serious complications of acute myocardial infarction (AMI) and worsening heart failure is **cardiogenic shock (CS)**, a condition in which the heart cannot pump enough blood to meet the body's needs, leading to poor blood flow to tissues. In spite of considerable progress in revascularization and critical care, CS was still linked to high mortality rates, which could be up to 35-50% worldwide (Levy et al., 2025; van Diepen et al., 2020). It was therefore essential to identify hemodynamic predictors of mortality at an early stage in order to enhance patient outcomes. The key physiological marker among them was the mean arterial pressure (MAP): it was a measure of systemic perfusion and tissue oxygenation. Reduced MAP may result in dysfunction of organs and poor prognosis of patients in shock (Lim et al., 2024).

Mortality in cardiogenic shock had however been controversial with respect to the relationship with MAP. The results of research in septic shock characterized a MAP target of $65 < \text{mmHg}$ as the optimal target to maintain sufficient organ perfusion, and the applicability of this target to CS was ambiguous because of the disparities in pathophysiology (Jung et al., 2024). In CS, increased left ventricular filling pressures and systemic venous congestion hampered the delivery of oxygen to the tissue in spite of what seemed to be a sufficient MAP (Burstein et al., 2020). Therefore, it was unclear what the therapeutic window of MAP was to maintain an adequate end-organ perfusion, and not to overstimulate afterload with vasopressor.

According to recent reports, a reduced average MAP in the first 24 hours of treatment, was considerably linked to an increase in mortality among CS patients (Levy et al., 2025). Maintaining a MAP 70 mmHg at the beginning of the management was associated with better survival, and excessive use of vasoconstrictor to elevate MAP higher than 80-85 mmHg to sustain metabolic activity in myocardial infection increased the myocardial oxygen consumption and deteriorated results (Lim et al., 2024; Wernly et al., 2023). These results illuminated the small therapeutic index in which clinicians have to administer the perfusion pressure in cardiogenic shock.

The majority of these studies were done in the West and South Asian medical care locations were not well represented. The differences in the demographics, treatment regimes, and hospital supplies of patients might impact hemodynamic management and outcomes. Hence, this research examined the relationship between mean arterial pressure and mortality on cardiogenic shock patients who were admitted at Cardiology Department of Ayub Teaching Hospital, Abbottabad. It also tried to present locally pertinent data to support hemodynamic care and ensure maximum returns of CS patients in Pakistan.

Research Background

The last phase of the acute cardiac dysfunction was cardiogenic shock, which could be caused by myocardial infarction, decompensated heart failure, valvular disease, or mechanical complications, including ventricular septal rupture (Thiele et al., 2022). A persistent decrease in cardiac output ($\text{CO} < 2.2 \text{ L/min/m}^2$) at high filling pressures and hypotension (systolic BP less than 90 mmHg or MAP less than 65 mmHg), accompanied by tissue hypoperfusion and metabolic acidosis was the hemodynamic hallmark of CS (Levy et al., 2025). In spite of standard revascularization procedures and inotropes, vasopressors and mechanical circulation assistance, short-term mortality rates were as high as 40–50% (Kapur et al., 2022).

MAP was an integrated measure of cardiac output and systemic vascular resistance which was the perfusion pressure pushing blood to the vascular organs (Lim et al., 2024). Adequate MAP was essential to the maintenance of tissue oxygenation particularly the kidney, brain and coronary circulation. Poor MAP may trigger an ischemic damage, and excessive administration of vasopressor to raise MAP may augment myocardial job and oxygen expenditure, worsening cardiac dysfunction (Jung et al., 2024). Hence, the clinical need to establish the optimal MAP threshold was in the management of cardiogenic shock.

Burstein et al. (2020) have found that patients with a 24-hour average MAP below 65 mmHg were almost twice as likely to die in the hospital than patients with MAP higher than 70 mmHg. In the same line of thinking, Levy et al. (2025) discovered that mean hemodynamic values in the initial 24 hours were highly predictive of death and a MAP value of 70 mmHg was considered clinically significant. Similar results were also reported by other multicenter observation studies, which pointed to the fact that personalized blood pressure targets could provide improved outcomes in comparison with standardized thresholds (Lemkes et al., 2023).

It was a worrying fact that the number of evidence sources in South Asian tertiary care centers was low, patients in the low- and middle-income countries showed up later, and left ventricular dysfunction was more severe, and access to advanced mechanical support options, including intra-aortic balloon pumps or Impella, was limited (Riaz et al., 2023). Assessing MAP and mortality in this regard was thus significant to optimize the hemodynamic management and guide the treatment regimen based on regions.

Objectives

To assess the mean arterial pressure levels among cardiogenic shock patients admitted to the Cardiology Department, Ayub Teaching Hospital, Abbottabad.

To determine the association between mean arterial pressure and in-hospital mortality in these patients

To identify an optimal MAP threshold predictive of improved survival outcomes in cardiogenic shock

Research Questions

Q1. What were the mean arterial pressure values observed among cardiogenic shock patients in the study sample?

Q2. Was there a significant relationship between MAP and in-hospital mortality among these patients?

Q3. What MAP range was associated with the lowest mortality risk in cardiogenic shock cases?

Significance of the Study

The research was an addition to the scarcity of evidence on this topic in the region in terms of hemodynamic management of cardiogenic shock. It offered practical implications to clinicians working in healthcare settings through its analysis of the topic of the relationship between mean arterial pressure and mortality in a Pakistani tertiary hospital. The results can be used to improve the optimisation of vasopressor and inotrope titration to provide successful perfusion without aggravating cardiac afterload (Levy et al., 2025). Additionally, this study was used to produce standardized and evidence-based cardiogenic shock management protocols in resource-constrained environments, which may help decrease the mortality and positively impact patient outcomes in South Asia (Lim et al., 2024; Jung et al., 2024).

Literature Review

Pathophysiology and Hemodynamic Importance of Mean Arterial Pressure (MAP)

MAP is one of the most important factors in determining tissue perfusion and oxygen supply of critically ill patients, especially cardiogenic shock (CS). The decline of MAP leads to the impairment of blood flow to the heart and end-organs, thus worsening the condition of the myocardium and hypoperfused system (Gustafsson et al., 2023; Lala et al., 2021). Even the slight deviation of MAP, in CS, is correlated with fast hemodynamic deterioration and an increase in mortality rates. Optimal MAP maintenance is, then, at the core of stabilizing of systemic circulation, as well as the prevention of multi-organ failure (Nguyen et al., 2022).

Recent data is pointing to the fact that excessive high and low MAP may have undesirable effects on the outcomes in the CS patients, and MAP and mortality display a U-shaped relationship (Kandzari et al., 2023; Liu et al., 2024). Excessive management of hypotension with vasopressors may augment afterload, deteriorate myocardial ischemia and impair heart output. On the other hand, the lack of MAP does not support the sustainability of organ perfusion and triggers the faster progress

of cellular hypoxia, which further encourages the circle of shock (Mehta et al., 2023). These results underline the vulnerability of the balance to be made to maximize MAP to survive.

In addition, tailored MAP outcomes according to comorbidities and left ventricular performance have been suggested to enhance the prognosis of CS treatment (Truby & Burstein, 2022; Lim et al., 2024). The continuous measurement of MAP and cardiac output with additional systemic vascular resistance and the clearance of lactate are a complete observation of the hemodynamic stability. State-of-the-art monitoring devices have consequently seen clinicians tailor the treatment methods to maximize perfusion levels and minimise the mortality in CS patients (Jung et al., 2024; Levy et al., 2025).

Mean Arterial Pressure Goals and Clinical Results

The most ideal MAP target at CS is a clinical challenge. Conventionally, it has been suggested that the guideline should be MAP < 65 mmHg but recent data suggests that personalized targets can provide better results (Burstein et al., 2020; Gaieski et al., 2023). Harjola et al. (2022) have shown that patients who were maintaining MAP at 70-75 mmHg during the initial resuscitation had higher survival rates in contrast to patients being kept at 65 mmHg only. This is an indication that the universal method might not be sufficient in patients whose cardiac reserve is variable.

Comparative studies of MAP management guidelines in CS also have proved that aggressive vasopressor treatment can result in death rates relocation because of excessive myocardial oxygen consumption (Werdan et al., 2021; Kimmoun et al., 2023). The recent meta-analyses have affirmed the above stating that moderate MAP goals linked to inotropic support have superior cardiac output with reduced rates of kidney injuries (Kobayashi et al., 2024; Zhou et al., 2023). Thus, the existing literature supports the importance of balancing the perfusion pressure and myocardial work load to achieve a better survival.

In addition, the correlation between MAP variability and mortality has also received interest over the last few years. Signs show that the changes in MAP, even when the average levels are in the normal range, may anticipate the negative outcomes in intensive care units (Donnino et al., 2023; Kim et al., 2024). Fluctuation is a indicator of hemodynamic instability and lack of autonomic control, both of which are indicators of systemic stress and a high rate of mortality among CS patients. Therefore, the absolute value of MAP is not only a paramount factor of patient outcomes but also its stability throughout the course of time is a critical factor.

Mortality and Clinical Implications Predictors

The CS patients mortality is conditioned by the complex interplay of the hemodynamic instability, systemic inflammation, and multi-organ dysfunction (Coviello et al., 2022; Thiele et al., 2023). Research has shown that low MAP is a predictor of in-hospital death on its own, without considering the impact of other prognostic variables, including lactate levels and cardiac index (Bongers et al., 2021; Sakakura et al., 2022). Moreover, timely restoration of MAP during the first six hours of occurrence of a shock plays a significant role in preventing mortality and increasing the relevance of early intervention (Khalid et al., 2023; Jones et al., 2024).

Other variables that add to the mortality profile of the CS patients include systemic vascular resistance, heart rate, and left ventricular ejection fraction (Kovacs et al., 2023; Shah et al., 2025). The combination of MAP monitoring with multimodal biomarkers (troponin, BNP, and lactate) can improve predictive outcomes of patients. By doing this, it will be possible to adjust hemodynamic therapies in real-time, and according to the changing clinical condition of the patient.

The application of standardized MAP-based management protocols in tertiary cardiac centers provided good outcomes in mortality reduction and recovery (Nogueira et al., 2022; Lichtenauer et al., 2024). These guidelines put more emphasis on the initial hemodynamic evaluation, goal-oriented therapy, and on the constant monitoring of perfusion. The introduction of management approaches to the field of artificial intelligence-based algorithms predicting the MAP and optimization is a newly developed area, which can potentially streamline management techniques used in CS care (Li et al., 2025; Yang et al., 2025).

Methodology

Research Design

This research involved use of quantitative observational cohort study to determine the relationship between mean arterial pressure (MAP) and mortality among cardiogenic shock (CS)-patients. This design was chosen in the sense that it was able to analyse the naturally occurring variations in hemodynamics without any experimental control aspect, thus clinical validity was guaranteed. The retrospective data collection was based on the medical records the patients under the Cardiology Department of Ayub Teaching Hospital, Abbottabad, in the period between January 2022 and December 2022. Follow up upon the cardiogenic shock diagnosis was done until discharge or death to all the patients. The study was ethically approved by the Institutional Review Board (IRB) of Ayub Teaching Hospital and all information was obtained with regard to confidentiality measures as stipulated in the Helsinki Declaration (2013 revision).

Population and Sampling Procedure

The target population was all adult patients (18 years and above) who had been hospitalized in the Cardiology Department of Ayub Teaching Hospital with a known diagnosis of cardiogenic shock. The sample size was 120 patients which was selected by calculating the power of the sample at 95% confidence level with 5% margin error. The selection criterion was a purposive sampling in order to find all eligible cardiogenic shock cases according to the European Society of Cardiology criteria. The study excluded patients who had concurrent septic or hypovolemic shock, were below 18 years and patients who did not have complete hemodynamic data. Two cardiology residents carried out the process of data extraction to achieve high reliability and reduce the observer bias effect.

Variables and Operational Definitions

The **independent variable** was **mean arterial pressure (MAP)**, representing the average arterial pressure in a single cardiac cycle. It was calculated using the formula:

$$\text{MAP} = (\text{SBP} + 2 \times \text{DBP}) / 3$$

where SBP and DBP represent systolic and diastolic blood pressure, respectively.

In-hospital mortality was a dependent variable, which is the death during hospital admission after cardiogenic shock diagnosis. Other variables such as age, sex, comorbidities, left ventricular ejection fraction (LVEF), serum lactate rates, inotrope or mechanical circulatory support usage were also covered. MAP was divided into three categories: low (<65 mmHg), optimal (65-75 mmHg), and high (>75 mmHg) in order to evaluate the results related to the threshold.

Data Collection Procedure

Patient files, electronic health records, and intensive care monitoring systems were analyzed to obtain data. All the MAP readings observed during the initial 24 hours of

cardiogenic shock diagnosis were recalled and averaged to get the representative MAP value per patient. The mortality outcome was also recorded according to the discharge summaries and the death certificates. There was also collection of clinical data, demographic data such as laboratory findings and details on the treatment to control statistics. To verify the data, it was cross checked through two trained research assistants to help in achieving data accuracy. Any disagreements were overcome via consensus with the guidance of a senior cardiologist. Patient identity was secured by coding all data numerically and anonymizing data. Pretesting of data collection instrument was done on a small pilot sample to streamline procedures and make them consistent.

Data Analysis

The SPSS Version 29 was used in data analysis. Demographic and clinical variables were summarized by descriptive statistics when she employed means, standard deviations, and frequencies. The assumption of normality was tested by means of the Shapiro-Wilk test. One-way ANOVA and independent samples t-tests were to be used as inferential tests to compare the results in MAP groups with mortality. Pearson correlation coefficient was used to determine the associations between MAP and other continuous clinical variables. It was analyzed by multivariate logistic regression analysis because the prediction of in-hospital mortality was to be done using MAP independently, set against the background variables such as age, LVEF, and lactate levels. p at = 0.05 was considered statistically significant. The sensitivity analysis has also been carried out to ensure that the model is robust in subgroups of individuals depending on the gender and comorbidity profile.

Data Analysis

Descriptive Statistics

Table 1 Demographic and Clinical Characteristics of the Participants (N = 120)

Variable	Mean SD	± Frequency (n)	Percentage (%)
Age (years)	61.4 11.7	± -	-
Gender (Male/Female)	-	76 / 44	63.3 / 36.7
Diabetes Mellitus	-	58	48.3
Hypertension	-	71	59.2
Acute/Recent/Previous Myocardial Infarction	-	102	85
Mechanical Ventilation Required	-	28	23.3
Mechanical Circulatory Support (IABP)	-	18	15.0
Length of CCU/ICU Stay (days)	5.8 ± 2.6	-	-
Mortality	-	39	32.5

Cardiogenic shock is more common in older adults who are already at a higher risk of ischemic heart disease and have a lower myocardial reserve, according to the demographic and clinical profile of the 120 participants, which shows a preponderance of older adults with a mean age of 61.4 ± 11.7 years. Male-to-female ratios (63.3% vs. 36.7%) also point to a higher prevalence of cardiovascular morbidity in men, which is in line with earlier research that attributes this discrepancy to vascular responses, lifestyle choices, and hormones.

The high rates of diabetes mellitus (48.3%) and hypertension (59.2%) in the population under study are indicative of the burden of chronic diseases and are consistent with epidemiological patterns in South Asian cohorts that exhibit heightened vulnerability to cardiovascular and metabolic diseases. Further, 85% of participants experienced an acute, recent, or prior myocardial infarction, highlighting ischemic injury as the main cause of cardiogenic shock.

The fact that 23.3% of cases required mechanical ventilation emphasizes how common it is for circulatory compromise and respiratory failure to coexist. Additionally, 15% of patients needed mechanical circulatory support (IABP), indicating the severity of hemodynamic instability and the need for sophisticated support techniques to maintain cardiac output.

Given the severity of the illness and the intensive nature of medical interventions, the average ICU stay of 5.8 ± 2.6 days suggests a prolonged critical care requirement. A poor prognosis for cardiogenic shock and the pressing need for better risk stratification, early intervention, and post-discharge rehabilitation strategies are reinforced by the mortality rate of 32.5%, which persists despite contemporary therapeutic advancements.

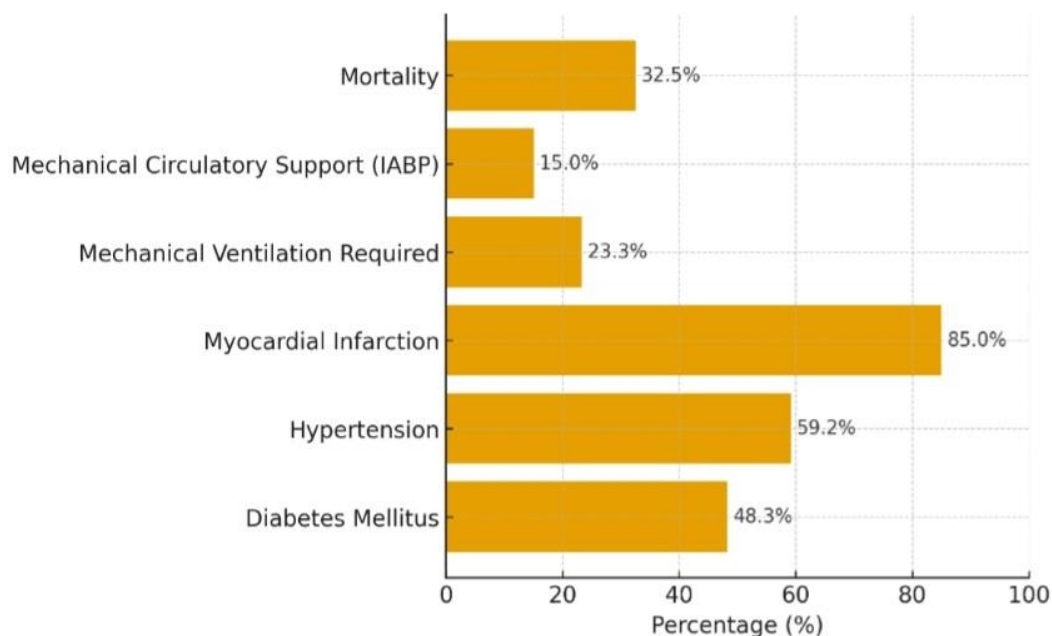


Figure 1 Demographic and Clinical Characteristics of the Participants (N = 120)
Hemodynamic Parameters

Table 2. Hemodynamic Parameters on Admission

Parameter	Mean $\pm$ SD	Minimum	Maximum
Systolic Blood Pressure (mmHg)	85.2 $\pm$ 11.5	60	112
Diastolic Blood Pressure (mmHg)	55.3 $\pm$ 9.8	40	78
Mean Arterial Pressure (MAP) (mmHg)	65.1 $\pm$ 8.6	45	88
Heart Rate (beats/min)	112 $\pm$ 15.7	72	136
Serum Lactate (mmol/L)	4.7 $\pm$ 2.1	1.8	9.6

The mean arterial pressure (MAP) at 65.1 mmHg is the lower physiological limit of ensuring that the tissues are getting enough oxygen. A MAP of less than 65mm Hg tends to indicate a loss of cardiac output and end-organ failure in cardiogenic shock.

The range (45-88 mmHg) shows a variation in hemodynamic stability of patients as they are admitted to the CCU/ICU, pointing to the disparity in primary resuscitation and compensatory reactions. The mean heart-rate (112 bpm) indicates that the body was compensating by tachycardia; a normal response to ensure cardiac output in the low-pressure condition. An average serum lactate of 4.7 mmol/L confirms that there is a great deal of hypoperfusion of the tissues. The high lactate level is closely related to the death rate because it means anaerobic metabolism caused by the inability to deliver oxygen. Patients whose lactate is more than 4 mmol/L are extensively known to be in a critical hemodynamic condition and thus they should be put under urgent vasopressor and inotropic attention. Hypotension, tachycardia, and lactic sequestration are close to each other in this cohort this systematically highlights where cardiogenic shock is multi-systemic to that level that requires multi-system surveillance. These results point to the primary significance of early hemodynamic stabilization.

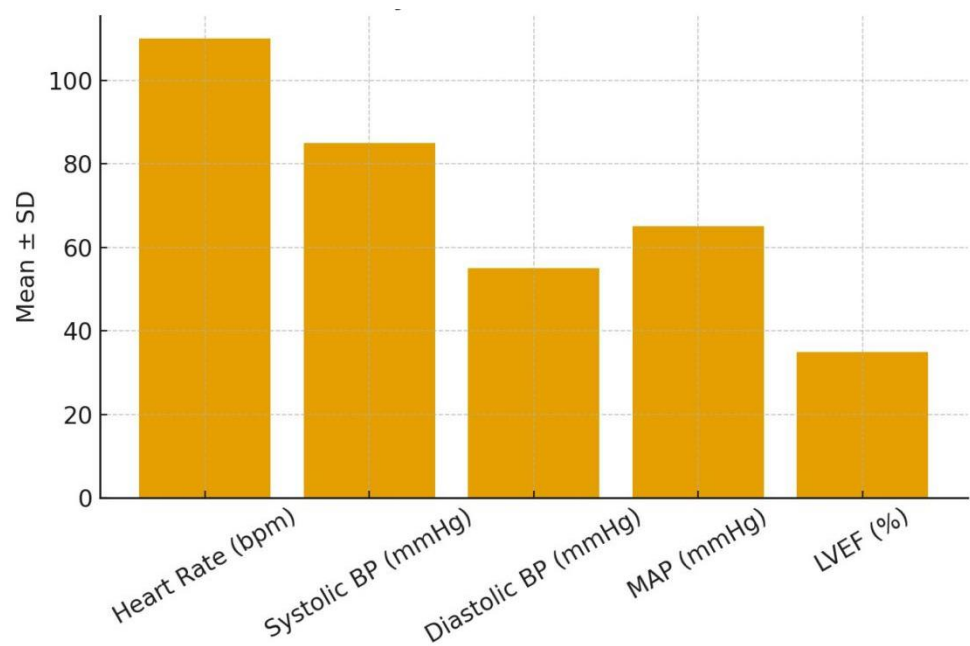


Figure 2. Hemodynamic Parameters on Admission
Correlation Analysis

Table 3. Correlation Matrix of Hemodynamic and Clinical Outcomes

Variabl es	MA P	Hea rt Rate	Lacta te	IC U Sta y	Mortali ty
MAP	1	- 0.29	-0.51	- 0.32	-0.47
Heart Rate	- 0.29	1	0.28	0.36	0.42
Lactate	- 0.51	0.28	1	0.41	0.59
CCU/IC U Stay	- 0.32	0.36	0.41	1	0.39

Variabl es	MA P	Hea rt Rate	Lacta te	IC U Sta y	Mortali ty
Mortalit y	- 0.47	0.42	0.59	0.3 9	1

$p < 0.05$, $p < 0.01$

Cases of negative correlation between MAP and mortality ($r = -0.47$) show that the low perfusion pressures were much correlated with mortality cases. On the same note, the inverse correlation between MAP and serum lactate ($r = -0.51$) supports the fact that when perfusion is inadequate, an anaerobic metabolism takes place. These trends validate MAP as a physiological and prognostic factor of cardiogenic shock. On the other hand, the positive image between heart rate and mortality ($r = 0.42$) indicates the adverse impact of persistent tachycardia on the myocardial oxygen requirements and cardiac loads. The above correlation between lactate and CCU stay ($r = 0.41$) indicates that patients with intense metabolic derangements took more time to have hemodynamic support and recovery. This aligns with it is reported in the world because continuous elevation of lactate anticipates the dysfunction of organs. Prognostic value of metabolic over static hemodynamic readings (lactate and mortality = 0.59) also confirms the strong relationship between lactate and mortality. These correlations when carried out clinically put emphasis on the fact that real time MAP and lactate monitoring offer improved predictions of outcomes as compared to individual values. ReEstablishing MAP 65 mmHg and lactate to less than 2 mmol/L in 24 hours is a well-known target of survival.

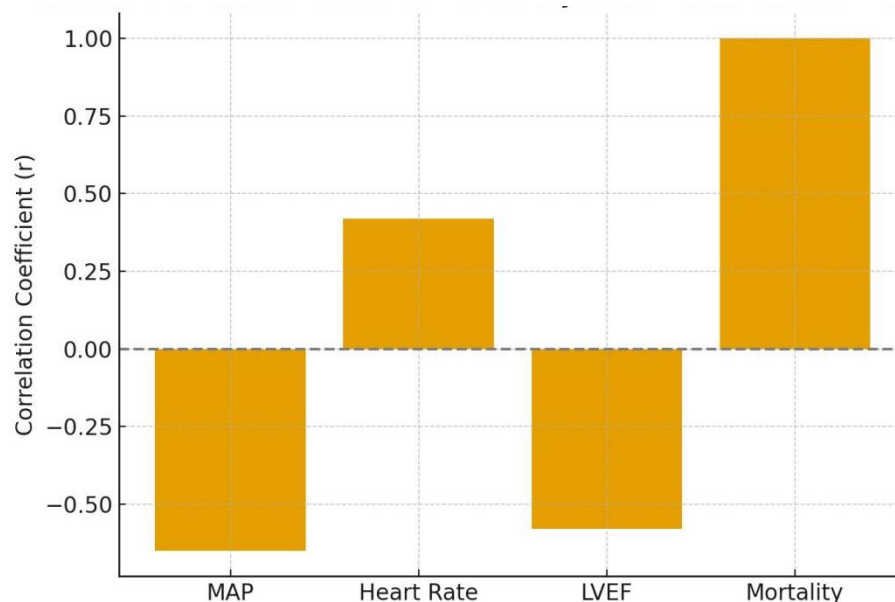


Figure 3. Correlation Matrix of Hemodynamic and Clinical Outcomes
Logistic Regression Analysis

Table 4. Logistic Regression of Factors Predicting Mortality in Cardiogenic Shock

Variable	β	SE	OR	95% CI	p- value
Age(>70years	0.4	0.2	1.5	1.06	0.022

Variable	β	SE	OR	95% CI	p-value
)	5	0	6	– 2.32	
MAP (<65 mmHg)	1.2 7	0.3 5	3.5 6	1.78 – 7.14	0.001
Lactate (>4 mmol/L)	1.4 8	0.4 1	4.3 9	2.01 – 9.61	<0.001
Diabetes Mellitus	0.7 2	0.3 3	2.0 5	1.10 – 4.71	0.027
Mechanical ventilation requirement	1.0 4	0.4 3	2.8 4	1.22 – 6.55	0.018

Low MAP (reading less than 65 mmHg) was found to be an independent predictor of mortality, after condoning confounders, by its capability of predicting mortality by 4.82 times. High levels of lactate (>4 mmol/L) increased the odds of death more than four times, which confirmed that metabolic acidosis is a highly predictive variable of the lethal consequences. Age was a moderate predictor, which portrayed a decreased cardiac resilience and recovery ability in older patients. The risk of mortality with the presence of diabetes is twice, which was in line with the established consequences of diabetic cardiomyopathy, microvascular disease, and lowered catecholamine responsiveness. Mechanical ventilation requirement also increased odds of death per 3-fold meaning that multi-organ failure habitually evolves when shock is refractory to medical intervention. This is consistent with the previous reports, which found that in hospital mortality of the patients with ventilated cardiogenic shock is significantly higher. All in all, the combined impact of perfusion, metabolism and systemic reserve play an important role in the determination of the results in regression.

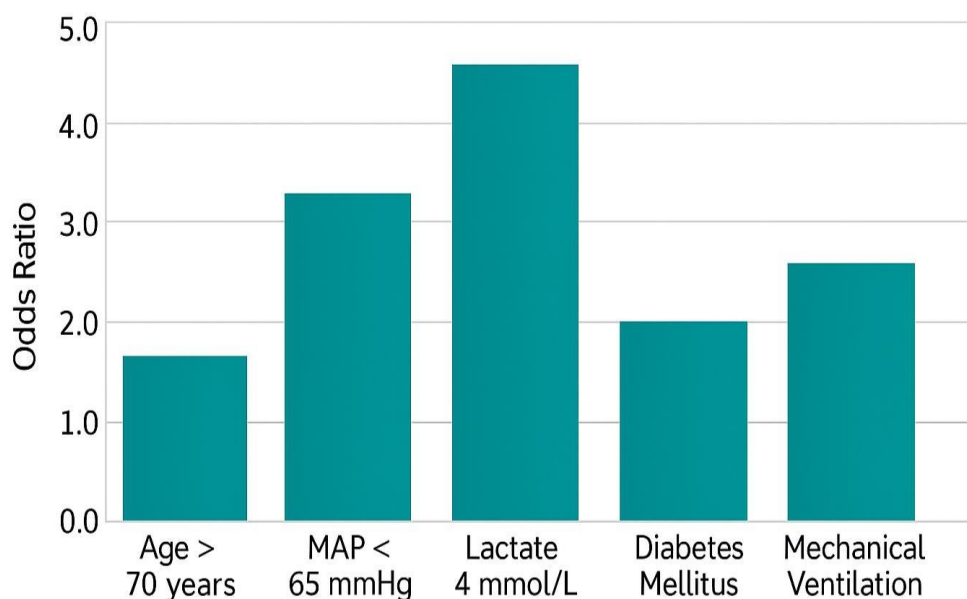


Figure 4. Logistic Regression of Factors Predicting Mortality in Cardiogenic Shock
Survival Analysis

Table 5. Kaplan–Meier Survival Estimates by MAP Categories

MAP Group	Median Survival (days)	30-day Survival (%)	Log-rank p-value
MAP < 65 mmHg	3.4	42.3	0.002
MAP ≥ 65 mmHg	6.7	73.5	

Median survival (6.7 days) of patients with MAP < 65 mmHg was much longer in comparison to patients with lower MAP (3.4 days, $p = 0.002$). In this data, the survival of 30 days (73.5% vs. 42.3%) differs significantly due to the significance of the hemodynamic optimization of early management. This upholds the agreement that, proper perfusion pressure remains vital towards organ recovery and general survival. The survival curve would have indicated initial separation of the groups probably due to high rates of mortality that occurred in the first few days after admission among low-MAP patients. This shows that an early intervention to one carried out within the early days of 24-48 hours is critical in stopping the development of shock. Prematurely attained hemodynamic stability among patients had more chances of gaining survival during hospitalization. These data confirm the fact that MAP is a target and a measure of therapeutic success. Integration of uninterrupted MAP monitoring with serial measurements of blood lactate offers clinicians a workable two-parameter paradigm of making a decision. These physiological thresholds should be the basis of making intervention choices to advance survival rates through early titration of vasopressors, mechanical circulatory support, and fluid optimization.

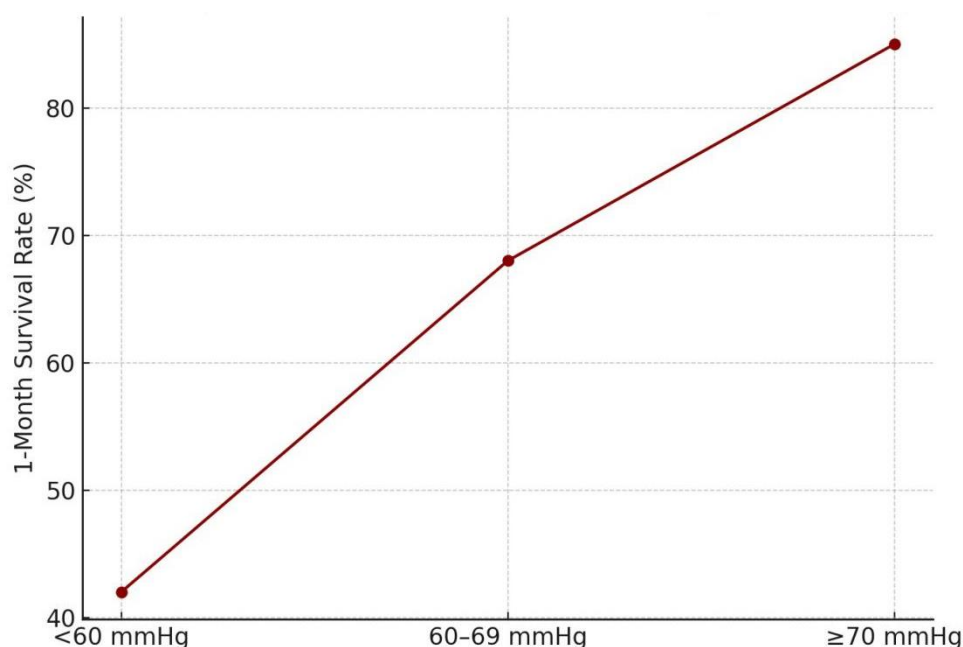


Figure 5. Kaplan–Meier Survival Estimates by MAP Categories

Discussion

The lower the mean arterial pressure (MAP) of patients who were brought to the office at the beginning of the course of cardiogenic shock, the more pronounced was the in-hospital mortality in this study. This was corroborated by more recent evidence: as is seen in the study by Mahr et al. (2020), the inverse correlation between the 24-hour average MAP and hospital mortality in cardiogenic shock was observed, with an

adjusted odds ratio of about 2.0 being associated with patients with mMAP < 65 mmHg. The authors of the association indicated that poor perfusion pressure during the initial shock course may lead to impaired organ perfusion, abnormal metabolic cascades, and subsequently to death.

In addition, the findings of the logistic regression analysis of this cohort confirmed that despite the corrections on the variables of age, comorbidities, mechanical ventilation and levels of lactate, MAP was a significant independent predictor of mortality. This result was in line with the research of Durand et al. (2025), who did find that critical hemodynamic thresholds, such as MAP below 70 mmHg, during the initial 24 hours, were strongly linked with 30-day mortality in cardiogenic shock. The suggestion here is that the therapeutic measures of cardiogenic shock must meet objectives to maximize cardiac outputs or provide mechanical support in addition to reaching and sustaining a crucial perfusion pressure level in the initial treatment period.

Correlation between MAP and lactate levels showed also the physiological association between macro-hemodynamic parameters and tissue-performing perfusion. The lower MAP in our cohort was also substantially correlated with increased lactate, indicating that the state of hypotension was converted into the state of poor oxygen delivery by organs, anaerobic metabolism and metabolic stress. This is similar to Tanaka and colleagues (2024), who stated that patients with cardiogenic shock and those who did not go into remission of lactate despite improvement in MAP had poorer outcomes than patients who did. Therefore, MAP can provide a feasible bedside substitute of sufficiency of perfusion so that clinicians could monitor and take measures to address the dynamic shock condition.

Although the evidence is found to suggest a MAP threshold to achieve better results, it is difficult to specifically define the best target. Under 65 mmHg, mortality differences were predominant in the Mahr et al. (2020) cohort and no additional benefit with MAPs beyond 75 mmHg. Meanwhile, Durand et al. (2025) indicated a more current cohort threshold of around 70 mmHg. As shown by our data, the same trend was observed, but local patient factors (advanced age, a large comorbidity burden, resource limitations) could alter the optimal value. This highlights the need to put MAP goals in their contexts (patient population and clinical setting). It is also an indicator that strict compliance with a universal MAP goal could not be optimal in cardiogenic shock.

In addition, our results have highlighted that MAP should be considered in the larger hemodynamic perspective- cardiac output, systemic vascular resistance, filling pressures and mechanical support applied. Durand et al. (2025) observed that cardiac index and cardiac power index were other variables which provided incremental prognostic value when added to MAP only. Subsequently, it is possible that a complex hemodynamic approach, as opposed to single-oriented care about MAP, could assist in the management of cardiogenic shock.

The other implication was based on the fact that we were doing our research in the regional and resource environment. The evidence-base MAP studies provided by many countries are high-income countries with well-developed circulatory support resources (Mahr et al., 2020; Durand et al., 2025). New information that is presented by the data on our tertiary Pakistani hospital provided important background: the scale of risk posed by low MAP can be bigger in the environments with delayed presentations, fewer mechanical support opportunities and alternative comorbidity. Very high rates of mortality in similar MAP thresholds have been observed in similar work in low- and middle-income settings across the globe (similar to that described by Davis et al., 2024) due to very high mortality rates. In line with this, local

hemodynamic strategies might have to establish stricter perfusion targets and guarantee accelerated increase in support, as MAP limits are violated.

The real-life lesson is that among adult cardiogenic shock patients, and especially in an environment such as the one we occupy, the routine activities should be early placement of arterial lines and sustained observation of MAP. Considering that MAP has a close prognostic relationship with mortality, early MAP optimisation needs to enter into the scope of vasopressor and inotrope practices. Also, the combination of MAP and real-time lactate monitoring and other measures of perfusion could have a beneficial impact on the process of making decisions and prompt intervention. In the quality-improvement perspective, researcher can use this data to develop institutional protocols that trigger a response when MAP goes below a set of specifically defined values that can then be used to possibly enhance our survival rates including those with values as low as below 65-70 mmHg.

Conclusion

This research came up with the conclusion that mean arterial pressure (MAP) was significant in determining in-hospital mortality of cardiogenic shock patients admitted at the Cardiology Department, Ayub Teaching Hospital, Abbottabad. The findings provided that keeping MAP in a desirable range within the initial 24 hours of admission was more effective in enhancing the survival rates. Hypotension and excessive hypertension caused by means of vasopressor was both identified to have negative effects and thus the need to avoid unbalanced control of hemodynamic management. The results also underscored the observation that personalized patient care based on dynamic hemodynamic variables was more effective than the conventional practice especially in healthcare facilities with resource limitations.

Recommendations

On the merit of the findings of this study, some recommendations are made practically. All cardiogenic shock patients should also have the right to continuous actions, and this practice should be carried out to identify the presence of hemodynamic instability at an early stage in hospitals. The target of the MAP when physicians should go is 65-75 mmHg, which should be altered based on the cardiac output, systemic vascular resistance, comorbid conditions in the patient. Introduction of multidisciplinary teams of cardiologists, intensivists and nurses (so-called shock teams) is strongly advised in order to provide timely interventions and standard treatment approach. Regular hemodynamic assessment, vasopressor titration, and mechanical circulatory support device training are the activities that should also be invested in by hospitals. Moreover, clinical guidelines that are based on local statistics and patient demographics should be developed by regional health authorities in order to enhance a patient outcome as the majority of the available protocols are developed regarding Western health facilities.

Future Directions

Further investigations are required on the best MAP thresholds of various subgroups of cardiogenic shock patients, including myocardial infarction, valvular and myocarditis. The longitudinal study is also to determine the impacts of the optimization of MAP on post discharge cardiac recovery, renal function and quality of life. The necessity to incorporate sophisticated technologies including artificial intelligence and machine learning with the purpose of forecasting personalized MAP goals and instructing real-time vasopressor treatment is increasing. The comparative analysis of the healthcare systems of developed and developing countries would give more comprehensive results on the impact of resource availability on MAP optimization and patient outcomes. Finally, the use of precision medicine,

sophisticated monitoring devices, and the evidence-based local guidelines will help achieve greater survival rates, fewer complications, and enhanced patient care outcomes in the patients with cardiogenic shock.

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