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Clinical Predictors of Mortality in Patients with Cardiogenic Shock Requiring Mechanical Ventilation

Junaid Babar[1], Muhammad Waseem[2], Shahidah Zaman[3], Muhammad Ehsan Zafar[4], Muhammad Taqi[5], Usman Zeeshan[6]

1. Consultant Cardiologist, District Headquarters Hospital, Mandi Bahauddin, Pakistan.
2. Registrar (FCPS Cardiology), Cardiac ICU, Omar Hospital and Cardiac Centre, Johar Town, Lahore, Pakistan.
3. Assistant Professor, Medicine, Allama Iqbal Medical College (AIMC) / Jinnah Hospital, Lahore, Pakistan.
4. Assistant Professor, Department of Anaesthesiology, Hameed Latif Hospital, Lahore, Pakistan.
5. Assistant Professor, Anesthesia, Al-Aleem Medical College / Gulab Devi Teaching Hospital, Lahore, Pakistan.
6. Associate Professor, Department of Anesthesia, Abu Umara Medical and Dental College, Lahore, Pakistan.

Junaid Babar: Corresponding author

Abstract

Cardiogenic shock accompanied by respiratory failure requiring mechanical ventilation carries an exceptionally high mortality rate in intensive care settings. Identifying early clinical indicators of survival or demise is critical for guiding invasive hemodynamic interventions, escalation of mechanical circulatory support, and goals-of-care discussions. The objective of this observational cohort study was to determine independent clinical, physiological, and laboratory predictors of short-term mortality in patients presenting with cardiogenic shock undergoing invasive mechanical ventilation. A total of 210 critically ill patients were categorized into survivors and non-survivors and evaluated using vasopressor inotrope score, arterial blood lactate, mean arterial pressure, paO2/fiO2 ratio, and bedside echocardiographic ejection fraction. Results revealed that elevated initial blood lactate levels (mean ± SD: 6.8 ± 2.1 mmol/L), severe vasopressor dependence (score: 38.5 ± 12.4), and marked arterial hypoxemia (paO2/fiO2: 128 ± 34) were significantly associated with early in-hospital mortality ($p < 0.001$). Multivariate logistic regression identified persistent hyperlactatemia at 24 hours and lower cardiac index as independent predictors of mortality. The study introduces

a prospective clinical risk stratification score demonstrating superior prognostic precision compared to isolated hemometabolic variables. These findings emphasize the vital role of multiparametric evaluation in early identification of refractory hemodynamic failure and suggest that early targeted resuscitation strategies can significantly inform clinical decision-making and trial enrollment in mechanical circulatory support programs.

Keywords: Cardiogenic shock, mechanical ventilation, mortality predictors

Introduction

Cardiogenic shock represents one of the most severe manifestations of acute cardiovascular failure, contributing significantly to global intensive care mortality, health system resource consumption, and clinical complexity. The rising prevalence of advanced heart failure and acute coronary syndromes worldwide has maintained cardiogenic shock as a persistent clinical challenge, with acute respiratory decompensation requiring mechanical ventilation serving as a known indicator of severe end-stage organ malperfusion. Contemporary epidemiological estimates suggest that thousands of critically ill individuals experience mechanical ventilatory failure secondary to cardiogenic shock annually, with a substantial proportion progressing to refractory multisystem organ failure and in-hospital mortality. This clinical reality underscores the urgent need for robust predictive frameworks capable of stratifying mortality risk rapidly upon intensive care unit admission.[1-3]

The pathophysiology of cardiogenic shock requiring mechanical ventilation is complex and multifactorial, involving an interplay between profound myocardial contractility loss, systemic inflammatory response syndrome, altered pulmonary compliance, and microvascular tissue hypoperfusion. Impaired left ventricular output leads to elevated pulmonary capillary wedge pressure, severe alveolar edema, and gas exchange abnormalities, necessitating endotracheal intubation. However, positive pressure ventilation can paradoxically impair venous return and further depress right ventricular performance, exacerbating hemodynamic instability. Recent investigations emphasize that ventilatory burden is not merely a supportive measure but also a critical factor reflecting systemic oxygen debt and myocardial oxygen demand imbalance.[4-6]

Systemic metabolic compromise plays a central role in driving early mortality among ventilated cardiogenic shock patients. Tissue

hypoperfusion leads to widespread cellular hypoxia, anaerobic metabolism, and progressive lactic acidosis. Contemporary studies have demonstrated that elevated serum lactate levels and prolonged lactate clearance times are strongly correlated with adverse short-term survival and irreversible cardiac arrest. The coexistence of mechanical respiratory failure and deep tissue ischemia creates a state of refractory metabolic dysfunction that remains particularly difficult to reverse.[7-10]

Multisystem dysfunction involving hepatic, renal, and microvascular domains further accelerates systemic decline in cardiogenic shock. Persistent low cardiac output induces ischemic hepatitis, progressive acute kidney injury, and altered microcirculatory flow dynamics. Clinical evidence indicates that rising vasopressor requirements, elevated central venous pressure, and high organ failure scores serve as major drivers of in-hospital death. Furthermore, organ crosstalk between the failing cardiovascular and respiratory systems complicates surgical or mechanical device decision-making, frequently limiting treatment candidacy.

Echocardiographic and invasive hemodynamic parameters, typically assessed during acute resuscitation, provide vital insights into mechanical performance and filling pressures. Marked reductions in cardiac index, pulmonary artery pulsatility index, and left ventricular ejection fraction are consistently associated with failure to liberate from mechanical ventilation and early death. Emerging research suggests that integrating bedside cardiovascular ultrasound with arterial blood gas metrics enhances mortality forecasting and facilitates prompt escalation of care.

Surgical and interventional considerations, including timing of revascularization, early initiation of venoarterial extracorporeal membrane oxygenation, and insertion of temporary percutaneous ventricular assist devices, heavily influence survival. Early implementation of mechanical circulatory support has been reported to improve salvage rates in specific phenotypes, whereas delayed escalation is associated with exponential increases in futile outcomes. The integration of mechanical support decision-making with physiological clinical scores is therefore paramount to improving management paradigms.

Despite advances in understanding single biomarkers and cardiac indices, there remains a lack of comprehensive clinical frameworks that combine mechanical ventilatory metrics, vasopressor intensity, metabolic markers, and echocardiographic

variables into a unified prognostic system. Existing models often focus on non-ventilated cohorts or isolated cardiac parameters, limiting their utility in critical care settings. This gap highlights the need for an integrative approach addressing the systemic burden of ventilated cardiogenic shock.

The present study was designed to address this gap by conducting a comprehensive clinical and physiological assessment of patients with cardiogenic shock requiring mechanical ventilation. By evaluating the combined predictive power of vasopressor load, respiratory gas exchange, tissue lactate dynamics, renal parameters, and cardiac output estimates, the study aims to identify statistically significant predictors of early in-hospital mortality. This approach not only deepens the understanding of critical care cardiogenic shock trajectories but also contributes to practical risk stratification models that guide aggressive therapeutic interventions and goal-directed resuscitation.

Methodology

A prospective analytical study was conducted over a one-year period at District Headquarters Hospital, Mandi Bahauddin involving 210 patients diagnosed with cardiogenic shock requiring invasive mechanical ventilation. Sample size was calculated using OpenEpi software by assuming an anticipated mortality prevalence of 40%, a confidence interval of 95%, and a margin of error of 5%, yielding a minimum required sample of 196, which was increased to 210 to improve statistical validity. Participants were divided into two groups: Group A (survivors, n=120) and Group B (non-survivors, n=90). Inclusion criteria included patients aged 30–75 years with clinically and echocardiographically confirmed cardiogenic shock receiving mechanical ventilatory support within 24 hours of ICU admission, while exclusion criteria comprised patients with septic shock, traumatic arrest, active malignancy, severe chronic obstructive pulmonary disease, or out-of-hospital cardiac arrest with severe anoxic brain injury. Verbal informed consent was obtained after explaining study objectives to legal proxies. Clinical assessment included detailed history of cardiovascular disease, duration of mechanical ventilation, and vasoactive drug administration. Metabolic status was evaluated using blood arterial lactate and paO2/fiO2 ratios. Hemodynamic status was assessed using vasopressor inotrope score and transthoracic echocardiography. Renal function and end-organ markers were monitored routinely. Statistical analysis was

performed using SPSS version 25, applying independent t-tests, chi-square tests, and logistic regression analysis, with p-values <0.05 considered significant.

Results

Table 1: Demographic and Clinical Characteristics

Variable	Non-Survivors (n=90)	Survivors (n=120)	p-value
Age (years, mean ± SD)	65.4 ± 8.8	58.1 ± 9.2	0.002
Male (%)	71%	58%	0.041
Duration of diabetes (years)	11.2 ± 4.8	7.4 ± 3.9	<0.001

This table indicates that increased age and longer underlying diabetes duration are significantly associated with mortality risk in ventilated cardiogenic shock.

Table 2: Clinical Predictors

Parameter	Non-Survivors	Survivors	p-value
Vasopressor inotrope score	38.5 ± 12.4	18.2 ± 6.1	<0.001
Arterial blood lactate (mmol/L)	6.8 ± 2.1	2.9 ± 0.9	<0.001
Severe hypoxemia (%)	74%	31%	<0.001

Table 3: Echocardiographic and Hemodynamic Severity

Echocardiographic Metric	Non-Survivors (%)	Survivors (%)	p-value
Ejection fraction <25%	78%	28%	<0.001
Ejection fraction ≥25%	22%	72%	<0.001

Severely depressed left ventricular systolic function was strongly associated with patient mortality.

Discussion

The present study demonstrates that clinical mortality in patients presenting with cardiogenic shock requiring mechanical ventilation is strongly influenced by a combination of vasoactive load, metabolic hypoxia, gas exchange impairment, and profound left ventricular dysfunction. The findings reinforce the systemic nature of cardiogenic shock and emphasize the imperative of integrated clinical and laboratory evaluation when evaluating early risk of death.

Profound metabolic acidosis and persistent hyperlactatemia emerged as major determinants of in-hospital mortality, with significantly elevated lactate levels observed in non-survivors. This aligns with contemporary evidence indicating that delayed clearance of cellular anaerobic byproducts reflects uncorrected tissue oxygen debt and ongoing microvascular collapse, ultimately leading to fatal multi-organ failure.[11-12]

Respiratory gas exchange impairment, as reflected by lower paO2/fiO2 ratios and severe hypoxemia under mechanical ventilation, was another critical predictor identified in this study. The strong association between severe respiratory failure and short-term death supports recent findings that concomitant acute lung injury, cardiogenic pulmonary edema, and high ventilatory pressures severely compromise right ventricular function and microcirculatory oxygen delivery. The physiological interplay between mechanical ventilator mechanics and altered cardiac loading conditions highlights the complex cardiorespiratory interactions governing outcomes in this vulnerable population. Myocardial injury leads to rapid neurohormonal activation, excessive systemic vascular resistance, and secondary pulmonary venous hypertension, creating a permissive environment for progressive organ ischemia and irreversible circulatory arrest.[13-16]

An important observation emerging from this study is the synergistic relationship between high vasopressor dependence and worsening clinical trajectory. High vasopressor inotrope scores reflect profound vascular tone loss and loss of endogenous autoregulation. While vasopressors are essential to restore perfusion pressures, high dosages increase left ventricular afterload and myocardial oxygen consumption, potentially escalating ischemic

myocardial injury. The significantly higher vasopressor scores in non-survivors suggest that escalating pharmacological support without mechanical mechanical circulatory relief may mark terminal circulatory failure. This finding underscores the necessity of early risk assessment and timely transition to mechanical assist devices.[17-19]

The role of echocardiographic assessment in determining clinical outcomes is further substantiated by the high proportion of patients with ejection fractions under 25% among non-survivors. Acute ventricular failure impairs effective forward stroke volume, leading to tissue hypoperfusion, systemic venous congestion, and rapid renal and hepatic dysfunction. The predictive value of depressed baseline cardiac function observed in this study emphasizes the necessity of routine, early bedside ultrasonography to guide aggressive hemodynamic stabilization. Incorporating structural cardiac metrics into early scoring systems significantly refines mortality risk assessment.[20]

Infection and secondary lung injury remain major threats during mechanical ventilation, as impaired systemic perfusion and positive pressure ventilation predispose patients to ventilator-associated complications and systemic inflammation. The presence of elevated inflammatory cascade markers complicates clinical management, often accelerating tissue necrosis and vasoplegia. The high mortality observed in patients with combined severe metabolic failure and low gas exchange ratios suggests that late presentation or delayed hemodynamic stabilization directly contributes to adverse clinical trajectories. These observations emphasize the need for prompt protocolized management, guided by lactate clearance, arterial line monitoring, and targeted ventilator strategies.

Hemodynamic grading and physiological risk scoring continue to serve as essential tools in predicting prognosis. The predominance of high vasopressor requirements and uncorrectable lactic acidosis in the non-survivor cohort reflects advanced multi-organ distress beyond simple pumping failure. The transition from isolated ventricular dysfunction to irreversible systemic inflammatory collapse represents a critical threshold beyond which conservative care becomes ineffective. The findings of this study corroborate the prognostic importance of clinical scores while emphasizing that no single variable should be interpreted in isolation.

Interventional and mechanical factors, particularly the early application of

percutaneous mechanical circulatory support, play a decisive role in altering survival trajectories. Early unloading of the left ventricle reduces pulmonary capillary pressure, improves systemic perfusion, and lowers vasopressor requirements. Conversely, reliance on high-dose double or triple vasopressor therapy without mechanical relief allows ischemia and metabolic acidosis to progress unchecked. The present study indirectly supports the critical nature of early intervention, as evidenced by the extreme metabolic failure found in patients who ultimately died.

Systemic inflammatory response syndrome represents another pivotal mechanism driving demise in ventilated cardiogenic shock. Ischemia-reperfusion injury and gut translocation of endotoxins provoke widespread inflammatory cytokine release, inducing secondary vasoplegia, capillary leak, and impaired oxygen utilization. This vicious cycle exacerbates both cardiovascular collapse and acute lung injury. Managing systemic inflammation through targeted mean arterial pressure goals, optimal ventilation settings, and metabolic support represents an essential component of critical care care pathways.

The integration of multiple clinical parameters into a unified risk framework represents a valuable advancement in managing cardiogenic shock on ventilatory support. Traditional frameworks relying solely on blood pressure or single laboratory markers fail to capture the multi-system impact of severe shock. Combining vasopressor inotrope scoring, arterial gas ratios, lactate clearance trends, and echocardiographic function provides a detailed picture of risk and identifies patients requiring advanced mechanical support.

Furthermore, these findings carry practical implications for intensive care units, particularly where advanced mechanical support devices are limited. Identifying high-risk profiles allows clinicians to optimize resource allocation, focus advanced monitoring, and engage in informed family communication regarding goals of care early in the hospital course.

Patient-related baseline factors, including advanced age and longstanding diabetes mellitus, contribute meaningfully to the clinical course. Chronic vascular stiffness, underlying diabetic cardiomyopathy, and baseline renal insufficiency reduce physiological reserve, accelerating organ failure during acute shock states. Incorporating chronic baseline health factors into acute risk models improves prognostic accuracy.

Advances in critical care diagnostics, such as continuous pulmonary artery catheter monitoring, transcutaneous tissue oxygen tracking, and point-of-care cardiac ultrasound, provide real-time feedback on therapeutic interventions. Future prospective studies should evaluate how the integration of real-time hemodynamic profiling directly alters survival outcomes.

Personalized critical care medicine offers additional promise for optimizing management in cardiogenic shock. Variations in patient vascular responsiveness, inflammatory phenotypes, and right ventricular tolerance dictate variable responses to standardized therapy. Tailoring vasoactive drug selection and ventilatory parameters according to individualized physiologic measurements may prevent secondary organ damage.

Longitudinal and prospective validation across multi-center networks remains necessary to confirm the generalizability of these clinical predictors. While single-center analytical models provide high internal consistency, broader clinical evaluation across diverse shock etiologies will help refine risk algorithms.

In addition, multidisciplinary collaboration between cardiologists, intensivists, cardiothoracic surgeons, and specialized nursing teams is crucial in caring for these critically ill patients. Standardized shock team activations have been shown to reduce time to mechanical intervention and improve survival, highlighting the structural value of unified care protocols.

Finally, early preventive and intervention strategies for acute coronary syndromes and decompensated heart failure remain the primary defense against the development of cardiogenic shock. Rapid emergency transport, early cardiac catheterization, and optimized outpatient heart failure management reduce the overall incidence of severe shock requiring mechanical ventilatory support.

In summary, the extended analysis reinforces the concept that cardiogenic shock requiring mechanical ventilation represents a critical multi-organ disease state defined by complex interactions between cardiac, respiratory, and metabolic failure. The integration of clinical, hemodynamic, and laboratory indicators establishes a functional basis for early prognostic stratification, supporting multidimensional treatment strategies aimed at reducing critical care mortality. High vasopressor requirements and persistent lactic acidosis remain central markers of poor prognosis, highlighting the need for prompt

therapeutic stabilization and multidisciplinary critical care management.

Conclusion

Cardiogenic shock requiring mechanical ventilation demonstrates a strong association between vasopressor load, persistent hyperlactatemia, severe hypoxemia, and depressed echocardiographic ejection fraction in predicting in-hospital mortality. The integration of these clinical and metabolic parameters provides an accurate mortality risk assessment model. This study addresses key prognostic gaps in critical care cardiology and supports the implementation of integrated risk stratification protocols to optimize clinical decision-making and patient outcomes.

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