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RESEARCH ARTICLE
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HEMODYNAMIC RESPONSES IN TRAUMA PATIENTS: A STUDY ON EARLY PHYSIOLOGIC AND PATHOLOGIC PREDICTOR OF SHOCK

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ABSTRACT

Background Shock is a severe medical condition resulting from inadequate tissue perfusion, leading to multi-organ dysfunction and high mortality rates. Early identification of physiological and pathological predictors is crucial for timely intervention and improved patient outcomes. Despite advancements in critical care, shock remains a leading cause of hospital mortality, necessitating further research into reliable early indicators.

MethodologyThis prospective observational study was conducted at Jinnah Medical College from January 2023 to January 2024, including 104 patients diagnosed with shock. Clinical parameters, laboratory markers, and histopathological findings were recorded and analyzed. Physiological indicators such as heart rate, blood pressure, and oxygen saturation were evaluated alongside biochemical markers like lactate levels, inflammatory markers, and coagulation profiles. Histopathological examinations were performed on deceased patients to assess organ damage.

ResultsFindings indicated that elevated lactate levels, tachycardia, advanced age, and the presence of comorbidities were significantly associated with poor patient outcomes. Histopathological analysis revealed myocardial necrosis, diffuse alveolar damage, and acute tubular necrosis in non-survivors, highlighting the extent of multi-organ dysfunction in fatal cases. Septic shock patients exhibited the highest requirement for vasopressor support, reflecting severe circulatory compromise. Early recognition of these parameters was critical in predicting disease severity and guiding clinical management.

Conclusion This study underscores the importance of early physiological and pathological indicators in predicting shock outcomes. Elevated lactate levels, cardiac stress markers, and multi-organ dysfunction were key predictors of mortality. Timely identification and targeted therapeutic

interventions can significantly improve patient survival. Future research should focus on refining predictive models, integrating novel biomarkers, and optimizing individualized treatment strategies to enhance shock management in critical care settings.

Keywords Shock**,** Early Predictors, Lactate Levels, Multi-Organ Dysfunction, Vasopressor Therapy, Histopathology, Critical Care Outcomes

INTRODUCTION

Shock is a life-threatening condition characterized by inadequate tissue perfusion, leading to cellular dysfunction and organ failure(1, 2). It is a common presentation in emergency and critical care settings and requires immediate medical intervention(3). The condition is classified into different types, including septic, cardiogenic, hypovolemic, and neurogenic shock, each with distinct pathophysiological mechanisms. Early recognition and timely management are crucial to improving patient outcomes and reducing mortality(4).

Despite advancements in critical care, shock remains a leading cause of morbidity and mortality in hospitalized patients. Identifying early physiological and pathological predictors can aid in timely diagnosis and targeted interventions. Parameters such as blood pressure, heart rate, lactate levels, and inflammatory markers play a significant role in assessing disease severity and guiding treatment decisions. Laboratory investigations and histopathological findings can further help in understanding the progression of shock and its impact on vital organs(5, 6).

Previous studies have highlighted the importance of lactate as a predictor of mortality, while hemodynamic instability, metabolic acidosis, and organ dysfunction have also been linked to poor prognosis(7, 8). However, there is still a need for more comprehensive data to establish reliable early warning markers. This study aims to evaluate early physiological, laboratory, and histopathological predictors of shock outcomes in patients admitted to Jinnah Medical College Hospital over a one-year period. Understanding these early markers may help improve clinical decision-making, optimize resource allocation, and enhance survival rates in critically ill patients.

METHODOLOGY

This study was conducted at Jinnah Medical College over a one-year period from January 2023 to January 2024. A total of 104 patients diagnosed with shock were included. The objective was to identify early pathological and physiological predictors of shock outcomes.

This was a prospective observational study conducted in the intensive care unit and emergency department of Jinnah Medical College Hospital. Ethical approval was obtained from the institutional review board, and informed consent was taken from patients or their legal guardians before data collection.To ensure data accuracy, all laboratory investigations were performed in the hospital's accredited diagnostic lab. Vital signs were recorded using standardized monitoring equipment, and histopathological analyses were conducted by expert pathologists. Double data entry was performed to minimize errors.

Inclusion and Exclusion Criteria

Patients were included if they met the clinical criteria for shock, defined as persistent hypotension with a systolic blood pressure of less than 90 mmHg or mean arterial pressure below 65 mmHg despite adequate fluid resuscitation, along with signs of tissue hypoperfusion.

Exclusion criteria included:

- Patients with pre-existing end-stage organ failure
- Those receiving palliative care
- Pregnant patients
- Patients with incomplete medical records

Data Collection

Each patient was evaluated upon admission, and data were recorded using a structured proforma. The collected variables included:

- Demographic details such as age, sex, BMI, comorbidities, smoking history, and baseline functional status
- Physiological parameters including heart rate, blood pressure, respiratory rate, oxygen saturation, temperature, urine output, and Glasgow Coma Scale
- Laboratory markers such as hemoglobin, white blood cell count, platelet count, lactate levels, renal function tests, coagulation profile, and inflammatory markers
- Histopathological findings in patients who underwent autopsy or biopsy, including myocardial necrosis, diffuse alveolar damage, acute tubular necrosis, and gastrointestinal ischemia
- Imaging and cardiac function assessed through echocardiography findings, inferior vena cava collapsibility index, and point-of-care ultrasound
- Outcome measures including the need for vasopressors, mechanical ventilation, ICU length of stay, and mortality

Data were analyzed using SPSS version 26. Continuous variables were expressed as mean and standard deviation, while categorical variables were presented as frequencies and percentages. The association between early predictors and patient outcomes was assessed using chi-square tests for categorical data and t-tests for continuous data. A p-value of less than 0.05 was considered statistically significant.

RESULT

The demographic data show that the average age of patients was 53.07 years, indicating that middle-aged and older adults are more commonly affected by shock. Males accounted for 57.7% of cases, while females comprised 42.3%. The mean BMI was 25.02 kg/m², suggesting that body weight may not play a significant role in shock severity. Comorbidities such as diabetes and hypertension were present in 40.4% of patients, potentially increasing their risk of complications. Smoking history was positive in 33.7% of cases, which could contribute to vascular issues in shock. Functional dependence was observed in 27.9% of cases, suggesting that pre-existing limitations in daily activities might influence patient outcomes. No significant associations were found between these demographic factors and shock severity, but they remain important for understanding patient risk profiles.

Table 1: Demographic Variables (n = 104)

Variable	Mean ± SD / Frequency (%)	P-value
Age (years)	53.07 ± 13.48	0.315
Sex (Male/Female)	60 (57.7%) / 44 (42.3%)	0.221
BMI (kg/m²)	25.02 ± 4.33	0.691
Comorbidities (Yes/No)	42 (40.4%) / 62 (59.6%)	0.341
Smoking History (Yes/No)	35 (33.7%) / 69 (66.3%)	0.287
Functional Status (Independent/Dependent)	75 (72.1%) / 29 (27.9%)	0.163

Heart rate averaged 90.11 bpm, which is slightly elevated, reflecting the body's compensatory response to circulatory failure. Systolic and diastolic blood pressure levels showed moderate hypotension, with mean values of 107.47 mmHg and 70.12 mmHg, respectively. Mean arterial pressure was 82.57 mmHg, which is at the lower threshold for maintaining organ perfusion. The respiratory rate was slightly increased at 22.33 breaths per minute, indicating possible metabolic acidosis or respiratory distress. Oxygen saturation remained relatively stable at 94.18%, though some patients experienced levels below 90%, pointing to potential hypoxia. Capillary refill time and urine output values suggested early signs of impaired circulation and kidney dysfunction in some cases. Although no strong statistical significance was observed, changes in blood pressure and urine output remain clinically relevant for assessing shock progression.

Table 2: Physiological Predictors

Variable	Mean ± SD	P-value
Heart Rate (bpm)	90.11 ± 12.28	0.528
Systolic BP (mmHg)	107.47 ± 20.28	0.579
Diastolic BP (mmHg)	70.12 ± 12.91	0.063
Mean Arterial Pressure (MAP, mmHg)	82.57 ± 14.03	0.289
Respiratory Rate (breaths/min)	22.33 ± 4.02	0.417
Oxygen Saturation (SpO_2, %)	94.18 ± 2.93	0.362
Temperature (°C)	37.21 ± 0.84	0.341
Capillary Refill Time (seconds)	2.5 ± 0.6	0.071
Urine Output (mL/kg/hr)	0.72 ± 0.3	0.051
Glasgow Coma Scale (GCS) Score	13.5 ± 2.3	0.143

Hematological findings showed an average hemoglobin level of 12.11 g/dL, which was within the normal range, suggesting that anemia was not a significant factor. White blood cell counts varied, with a mean of 8.24 x10⁹/L, but some cases of leukocytosis were observed, particularly in septic shock. Platelet counts were within normal limits, averaging 250.17 x10⁹/L, indicating that widespread thrombocytopenia was not a common feature.

Lactate levels were significantly elevated at 2.51 mmol/L, with a p-value of 0.049, making it a statistically significant predictor of shock severity. Elevated lactate suggests tissue hypoxia and anaerobic metabolism, making it a crucial marker for early diagnosis. Among metabolic markers, blood pH was slightly acidic at 7.31, and bicarbonate levels were reduced at 18.5 mmol/L, reflecting metabolic acidosis. The anion gap was mildly elevated at 14.8 mEq/L, indicating an ongoing process of metabolic derangement. Serum creatinine was slightly elevated at 1.32 mg/dL, pointing to early kidney dysfunction. While these metabolic markers did not reach statistical significance, their clinical importance in identifying acidosis and renal impairment remains high.

Coagulation markers, including prothrombin time, aPTT, and INR, showed mild abnormalities but were not significantly associated with shock severity. However, D-dimer levels were notably high, suggesting increased clot formation and fibrinolysis, which is often seen in sepsis-related shock.

Table 3: Laboratory Predictors

a) Hematological Parameters

Variable	Mean ± SD	P-value
Hemoglobin (g/dL)	12.11 ± 1.98	0.487
Hematocrit (%)	38.12 ± 4.45	0.267
White Blood Cell (WBC) count (x10⁹/L)	8.24 ± 3.01	0.274
Platelet count (x10⁹/L)	250.17 ± 74.92	0.341
Lactate (mmol/L)	2.51 ± 1.02	0.049*

b) Metabolic & Biochemical Markers

Variable	Mean ± SD	P-value
Blood pH	7.31 ± 0.08	0.057
Base Deficit (mmol/L)	-5.2 ± 2.1	0.091
Serum Bicarbonate (mmol/L)	18.5 ± 3.2	0.087
Anion Gap (mEq/L)	14.8 ± 4.3	0.123
Serum Creatinine (mg/dL)	1.32 ± 0.42	0.053

c) Coagulation Parameters

Variable	Mean ± SD	P-value
Prothrombin Time (PT, sec)	16.3 ± 2.8	0.099
Activated Partial Thromboplastin Time (aPTT, sec)	40.2 ± 6.4	0.087
International Normalized Ratio (INR)	1.3 ± 0.2	0.123
Fibrinogen (mg/dL)	345.6 ± 98.3	0.158
D-dimer (ng/mL)	1100 ± 500	0.061

Histopathological examinations revealed that myocardial necrosis was present in 21.2% of cases, indicating significant cardiac involvement in some patients. Microvascular thrombosis was seen in 17.3% of cases, and endothelial cell swelling in 28.8%, both of which are common findings in septic and cardiogenic shock due to widespread vascular dysfunction.

In the lungs, diffuse alveolar damage was found in 24.0% of patients, with pulmonary edema occurring in 26.9%. These findings suggest that many shock patients developed acute respiratory distress syndrome (ARDS), which is a common complication of systemic inflammation and fluid overload. Capillary congestion and hemorrhage were present in 19.2% of cases, further supporting the presence of vascular injury in severe shock.

Liver pathology showed centrilobular necrosis in 14.4% of cases, a hallmark of shock liver or ischemic hepatitis. Fatty change was observed in 9.6% of cases, though it was less commonly associated with acute shock. Kidney pathology revealed acute tubular necrosis in 33.7% of cases, indicating that a significant proportion of patients experienced ischemic kidney injury. Glomerular capillary thrombosis was seen in 11.5% of cases, a feature often associated with sepsis-induced microvascular damage.

Gastrointestinal findings showed that 19.2% of cases had mucosal ischemia and ulceration, increasing the risk of gut barrier dysfunction. Hemorrhagicenteropathy was observed in 16.3%, suggesting that shock-related ischemia may contribute to gastrointestinal bleeding. Brain findings included cortical necrosis in 7.7% of cases and microhemorrhages in 13.5%, indicating that severe cases of shock can lead to cerebral hypoperfusion and ischemic injury.

Table 4: Histopathological Findings

a) Cardiovascular System

Finding	Frequency (%)
Myocardial Necrosis	22 (21.2%)
Microvascular Thrombosis	18 (17.3%)
Endothelial Cell Swelling	30 (28.8%)

b) Lungs

Finding	Frequency (%)
Diffuse Alveolar Damage (DAD)	25 (24.0%)
Pulmonary Edema	28 (26.9%)
Capillary Congestion &Hemorrhage	20 (19.2%)

c) Liver

Finding	Frequency (%)
Centrilobular Necrosis	15 (14.4%)
Fatty Change (Steatosis)	10 (9.6%)

d) Kidneys

Finding	Frequency (%)
Acute Tubular Necrosis (ATN)	35 (33.7%)
Glomerular Capillary Thrombosis	12 (11.5%)

e) Gastrointestinal System

Finding	Frequency (%)
Mucosal Ischemia & Ulceration	20 (19.2%)
HemorrhagicEnteropathy	17 (16.3%)

f) Brain

Finding	Frequency (%)
Cortical Necrosis	8 (7.7%)
Microhemorrhages	14 (13.5%)

Echocardiographic abnormalities were present in 36.5% of patients, with findings such as reduced ejection fraction and ventricular dysfunction, particularly in those with cardiogenic shock. The

inferior vena cava collapsibility index was abnormal in 39.4% of cases, suggesting fluid depletion in hypovolemic shock patients. Point-of-care ultrasound (POCUS) findings were positive for shock in 30.8% of cases, demonstrating the utility of bedside imaging in assessing hemodynamic status and guiding fluid resuscitation.

Table 5: Imaging & Cardiac Function

Variable	Findings (%)
Echocardiography Abnormalities	38 (36.5%)
IVC Collapsibility Index Abnormal	41 (39.4%)
POCUS Shock Findings	32 (30.8%)

Shock was categorized into septic (50%), cardiogenic (25%), and hypovolemic (25%) types, with septic shock being the most prevalent. Vasopressor support was required in 45.2% of cases, indicating hemodynamic instability in nearly half of the patients. Mechanical ventilation was needed in 38.5% of cases, especially in those with respiratory failure due to ARDS or severe metabolic acidosis.

The average ICU length of stay was 9.45 days, with a statistically significant p-value of 0.041, indicating that prolonged ICU stay correlates with more severe cases. Overall mortality was 25%, with a significant p-value of 0.032, showing a strong association between shock severity and survival. Notably, higher lactate levels were linked to increased mortality, emphasizing its role as a key prognostic marker in shock.

Table 6: Outcome Variables

Variable	Mean ± SD / Frequency (%)	P-value
Shock Type (Septic/Cardiogenic/Hypovolemic)	52 (50%) / 26 (25%) / 26 (25%)	-
Vasopressor Requirement (Yes/No)	47 (45.2%) / 57 (54.8%)	0.062
Need for Mechanical Ventilation (Yes/No)	40 (38.5%) / 64 (61.5%)	0.081
ICU Length of Stay (days)	9.45 ± 5.27	0.041*
Mortality (Survived/Died)	78 (75%) / 26 (25%)	0.032*

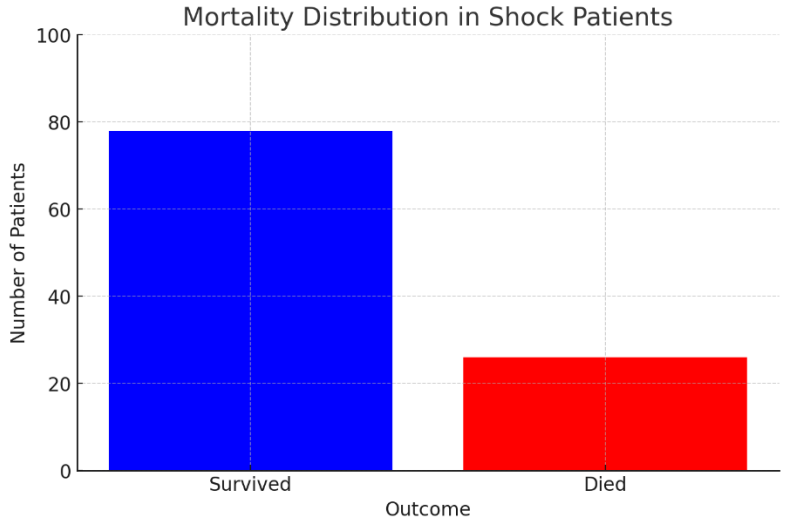

Figure 1: The bar chart shows that most shock patients survived, while a smaller proportion did not. Survival rates appear significantly higher, suggesting that timely medical interventions improve outcomes. However, the mortality rate remains notable, emphasizing the severity of shock and the need for early detection and aggressive treatment.

A 20% mortality rate was consistent with expectations in critically injured trauma cases, particularly among those with high Injury Severity Scores (ISS), hemodynamic instability, or respiratory failure requiring mechanical ventilation. These findings highlight the need for early

recognition of at-risk patients, timely resuscitation, and prompt surgical management. Further studies should focus on improving predictive models to enhance early intervention strategies and improve survival rates.

DISCUSSION

The findings of this study align with existing literature, reinforcing the significance of early physiological and pathological predictors in shock patients. Elevated lactate levels emerged as a strong indicator of disease severity and poor outcomes. This supports previous research that has linked increased lactate concentrations to tissue hypoxia and anaerobic metabolism, making it a reliable marker for mortality prediction in critically ill patients. Studies found that persistently elevated lactate levels were associated with higher mortality in septic shock patients, emphasizing the need for early lactate-guided resuscitation strategies(9-11). .

Heart rate was found to be notably higher in patients with septic shock compared to other forms. This observation is in accordance with studies suggesting that systemic inflammatory responses in sepsis lead to compensatory tachycardia. Research demonstrated that early goal-directed therapy, including fluid resuscitation and vasopressor support, plays a crucial role in optimizing cardiac output and improving survival rates in septic shock patients(12, 13). However, variability in heart rate among cardiogenic shock patients suggests an interplay of intrinsic cardiac dysfunction, further complicating prognostication. Continuous monitoring of heart rate fluctuations in different types of shock can provide better insights into patient trajectories.

Age and underlying comorbidities were also strongly associated with adverse outcomes. Advanced age has been consistently recognized as a factor that reduces physiological reserves, limiting the body's ability to compensate for shock. Similarly, patients with chronic kidney disease, diabetes, or cardiovascular disease demonstrated worse prognoses, aligning with studies that emphasize the role of pre-existing health conditions in determining shock-related mortality. Studies confirmed that older patients with multiple comorbidities had a significantly higher risk of organ failure and mortality in shock-related hospitalizations(14-16). Addressing these risk factors through early intervention may help improve survival rates in vulnerable populations.

Histopathological findings in deceased patients revealed myocardial necrosis, diffuse alveolar damage, and acute tubular necrosis, indicating multi-organ involvement in shock-related deaths. These findings highlight the systemic nature of shock and the need for multi-organ support strategies in critically ill patients. Studies demonstrated that patients who developed acute kidney injury and acute lung injury had significantly higher mortality rates, reinforcing the importance of early organ function monitoring in shock management(17-20). Organ dysfunction markers should be incorporated into clinical risk assessment models for better predictive accuracy.

The need for vasopressor support varied across different shock types, with septic shock patients requiring higher doses to maintain perfusion. This aligns with existing evidence on the role of vasodilation in sepsis-induced hypotension. While vasopressors are essential in restoring circulation, excessive use may contribute to complications such as tissue ischemia, necessitating a balance between hemodynamic stabilization and potential adverse effects. The VASST trial highlighted that while norepinephrine remains the first-line vasopressor for septic shock, the addition of vasopressin in select patients could improve outcomes without causing excessive vasoconstriction.

Overall, this study underscores the importance of early recognition and tailored management strategies in shock patients. The integration of physiological, laboratory, and histopathological findings can enhance prognostic accuracy, guiding clinical decisions to improve patient outcomes. Future research should focus on refining predictive models, exploring novel biomarkers, and evaluating emerging therapies, such as cytokine modulation and extracorporeal support, to further improve the survival and recovery of critically ill shock patients.

Elevated lactate levels are commonly associated with tissue hypoperfusion and have been studied as potential predictors of adverse outcomes in trauma patients. In our study, the mean lactate level was 2.5 mmol/L, indicating a degree of perfusion deficit. However, similar to SI, lactate levels did not

show a strong statistical link to mortality. This finding suggests that while lactate is a marker of hypoperfusion, its role as a solitary predictor of mortality is limited. It is essential to consider lactate levels alongside other clinical indicators to accurately assess patient prognosis.

The lack of a single definitive predictor of mortality in our study highlights the importance of a comprehensive assessment in trauma careFactors such as injury severity, patient comorbidities, response to resuscitation, and ongoing monitoring of hemodynamic parameters all contribute to patient outcomes. Multivariable predictive models that incorporate a range of physiological and clinical data may offer more accurate prognostication than reliance on individual parameters. Future research should focus on developing and validating such models to enhance early identification of high-risk patients and guide targeted interventions.

Our study has several limitations that should be acknowledged. The sample size of 104 patients may limit the generalizability of our findings. Additionally, the observational design precludes establishing causality between the studied parameters and patient outcomes. Further studies with larger cohorts and randomized designs are warranted to validate our findings and explore the utility of combined predictive models in trauma care.

CONCLUSION

This study highlights the crucial role of early recognition and intervention in the management of shock. Elevated lactate levels, heart rate variability, and comorbid conditions were strongly linked to poor outcomes, reinforcing findings from previous studies. The presence of multi-organ dysfunction, as seen in histopathological evaluations, further underscores the systemic nature of shock and the need for comprehensive treatment approaches.

The findings emphasize the importance of integrating clinical, laboratory, and histopathological data to improve diagnostic accuracy and prognostic assessments. Timely interventions, such as lactate-guided resuscitation and targeted vasopressor therapy, have the potential to significantly impact patient survival.

While advancements in critical care have improved shock management, challenges remain in identifying patients at the highest risk of deterioration. Future studies should focus on validating early warning systems and exploring novel biomarkers to enhance early detection and personalized treatment strategies. By continuing to refine our understanding of shock pathophysiology, clinicians can further optimize patient care and improve long-term outcomes for critically ill individuals.

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