Section: Emergency Medicine

Original Research Article

# CLINICAL PROFILE OF SHOCK IN PATIENTS PRESENTING TO EMERGENCY MEDICINE DEPARTMENT OF A TERTIARY CARE HOSPITAL

*Maria Thokchom[1], Himanshu Mishra[2], Raj Kumar[3], K.P Singh[4], Jitendra Kumar[5], W.P Singh[6]*

[1]*PG Resident, Department of Emergency Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.*
[2]*PG Resident, Department of Emergency Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.*
[3]*Assistant Professor, Department of Respiratory Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.*
[4]*Associate Professor, Department of General Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.*
[5]*Professor and Head, Department of Emergency Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.*
[6]*Professor and Head, Department of General Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.*

***Received*** *: 05/01/2026*
***Received in revised form*** *: 19/02/2026*
***Accepted*** *: 08/03/2026*

**Corresponding Author:**
**Dr. W.P. Singh,**
Professor and Head, Department of General Medicine, Rajshree Medical Research Institute, Bareilly, Uttar Pradesh, India.
Email: profdrwpsingh@gmail.com

**DOI:** 10.70034/ijmedph.2026.1.511

**Source of Support:** Nil,
**Conflict of Interest:** None declared

**Int J Med Pub Health**
2026; 16 (1); 2972-2979

ABSTRACT

**Background:** Shock is a life-threatening medical emergency characterized by acute circulatory failure resulting in inadequate tissue perfusion and impaired oxygen delivery to meet cellular metabolic demands. Early recognition and prompt management are critical to reducing morbidity and mortality. However, region-specific data describing the clinical and etiological profile of shock in emergency medicine departments remain limited. The objective is to identify the aetiology, types, and clinical profile of shock in patients presenting to the emergency medicine department of a tertiary care hospital.

**Materials and Methods:** This hospital-based cross-sectional observational study was conducted in the Department of Emergency Medicine at Rajshree Medical Research Institute, Bareilly, over a 12-month period (June 2024–July 2025). A total of 96 adult patients (≥20 years) presenting with clinical features of shock were enrolled using consecutive sampling. Detailed clinical evaluation, hemodynamic assessment, perfusion markers, laboratory parameters including serum lactate, imaging, and etiological classification were performed. Data were analyzed using SPSS, with results expressed as mean ± standard deviation and percentages.

**Results:** The mean heart rate and systolic blood pressure at presentation were 122.84 ± 9.93 beats/min and 81.99 ± 6.29 mmHg, respectively, indicating significant hemodynamic compromise. Altered sensorium (75%), prolonged capillary refill time (71.88%), and reduced urine output (92.71%) were common findings. Mean serum lactate was elevated (3.53 ± 0.81 mmol/L), reflecting tissue hypoperfusion. Septic shock was the most common type (52.08%), followed by hypovolemic (20.83%), cardiogenic (15.63%), and obstructive shock (11.46%). Pneumonia and abdominal sepsis were the leading causes of septic shock, while acute myocardial infarction predominated in cardiogenic shock. All patients received fluid resuscitation, 56.25% required vasopressor support, and 85.42% required ICU admission.

**Conclusion:** Septic shock was the predominant aetiology among patients presenting with shock, and most patients exhibited advanced physiological instability at admission. Early clinical recognition supported by lactate measurement remains crucial in emergency settings. Strengthening triage protocols and early management strategies may improve outcomes in shock patients.

**Keywords:** Shock, Septic shock, Emergency medicine, Hemodynamic instability, Serum lactate, Intensive care admission, Aetiology.

## INTRODUCTION

Shock is a life-threatening medical emergency characterized by acute circulatory failure leading to inadequate tissue perfusion and impaired oxygen delivery to meet cellular metabolic demands.[1] The underlying pathophysiology involves an imbalance between oxygen supply and demand, resulting in cellular hypoxia, mitochondrial dysfunction, depletion of adenosine triphosphate (ATP), and a shift from aerobic to anaerobic metabolism. This process leads to lactic acidosis and, if uncorrected, progresses to multiorgan dysfunction syndrome and death.[2]

Tissue oxygenation depends primarily on cardiac output and arterial oxygen content. Cardiac output is influenced by preload, myocardial contractility, heart rate, and afterload, while arterial oxygen content depends on hemoglobin concentration and oxygen saturation.[3] Disturbances in any of these parameters can precipitate shock. Importantly, hypotension is not always present in the early stages. Compensatory mechanisms such as tachycardia and peripheral vasoconstriction may temporarily maintain blood pressure despite ongoing tissue hypoperfusion, making early recognition in emergency medicine departments challenging.[4]

Shock is broadly classified into four major categories: hypovolemic, cardiogenic, obstructive, and distributive shock.[1] Hypovolemic shock results from significant intravascular volume loss due to hemorrhage, dehydration, burns, or third-space losses. Cardiogenic shock occurs due to myocardial pump failure, commonly secondary to acute myocardial infarction or severe heart failure. Obstructive shock arises from mechanical impediments to cardiac filling or output, such as pulmonary embolism or cardiac tamponade. Distributive shock is characterized by abnormal vasodilation and maldistribution of blood flow, with septic shock being the most common subtype encountered in emergency settings.[5]

Globally, shock remains a major cause of emergency department visits and intensive care admissions. Sepsis is the leading cause of distributive shock worldwide, accounting for approximately 48.9 million cases and 11 million deaths in 2017, representing nearly one-fifth of global deaths.[6] In high-income countries, shock is reported in approximately 0.5–1.3% of emergency department visits; however, the burden in low- and middle-income countries is likely underestimated due to limited reporting and healthcare access.[7]

In India, shock frequently presents in emergency medicine departments, particularly in tertiary care centers. The high prevalence of infectious diseases, trauma, gastrointestinal bleeding, dehydration, and the growing burden of cardiovascular disorders significantly contribute to shock-related morbidity and mortality. Septic shock remains the most common etiology, especially in populations with delayed healthcare access. Hypovolemic and cardiogenic shock are also increasingly encountered due to trauma and rising rates of ischemic heart disease and diabetes mellitus.[8]

Early recognition and timely management are crucial in determining outcomes. The initial hours after presentation, often termed the “golden hour,” are critical for survival. Prompt fluid resuscitation, restoration of perfusion, and early initiation of etiology-specific therapy significantly reduce mortality.[9] Clinical evaluation remains central to diagnosis, with features such as tachycardia, hypotension in advanced stages, cold extremities, delayed capillary refill, altered sensorium, and reduced urine output. Adjunctive tools including serum lactate measurement and arterial blood gas analysis aid in assessing severity. Elevated serum lactate is a reliable marker of tissue hypoperfusion and is strongly associated with increased mortality.[10]

Despite advances in emergency and critical care medicine, outcomes vary across healthcare settings, particularly in developing countries where delayed presentation and limited resources affect early diagnosis and management. There is limited region-specific data describing the clinical and etiological profile of shock in emergency medicine departments. A systematic evaluation of the demographic characteristics, clinical presentation, physiological parameters, and etiological distribution of shock is therefore essential to improve early recognition, optimize triage decisions, and enhance patient outcomes.

## MATERIALS AND METHODS

This hospital-based cross-sectional observational study was conducted in the Department of Emergency Medicine at Rajshree Medical Research Institute (RMRI), Bareilly, Uttar Pradesh, a tertiary care teaching hospital serving both urban and rural populations. The study was carried out over a period of 12 months from June 2024 to July 2025. All patients presenting to the emergency department during the study period were screened for eligibility. The study focused on patients presenting with clinical features suggestive of shock, defined as a state of acute circulatory failure resulting in inadequate tissue perfusion and impaired oxygen delivery to meet metabolic demands.[11]

The study population comprised adult patients aged 20 years and above of either gender who presented with a clinical diagnosis of shock or suspected shock at the time of emergency evaluation. Written informed consent was obtained from patients or their legally authorized representatives prior to enrollment. Patients below 20 years of age, those unwilling to provide consent, and those refusing venipuncture or essential laboratory investigations were excluded to ensure completeness of clinical and biochemical evaluation. These criteria were established to

maintain homogeneity in patient selection and ensure reliability of data collected.

A consecutive sampling technique was employed. All eligible patients meeting the inclusion criteria during the study period were enrolled until the required sample size was achieved. The sample size was calculated using the standard formula for prevalence-based cross-sectional studies at a 95% confidence level: $n = Z^2 \times P(100-P) / d^2$. Assuming a prevalence of 50% to obtain maximum sample size, allowable error of 10%, and Z value corresponding to 95% confidence interval (3.84), the minimum calculated sample size was 96 patients.[12] Accordingly, a total of 96 patients were included in the study.

Following consent, each patient underwent a detailed clinical assessment emphasizing early identification of shock and evaluation of hemodynamic instability. A pre-designed and structured proforma developed specifically for the study was used for uniform data collection. Demographic details including age and gender were recorded. Presenting complaints were documented and included fever, chest pain, shortness of breath, vomiting, diarrhea, hematemesis, melena, vaginal bleeding, trauma, abdominal pain, burning micturition, and skin and soft tissue infections. Relevant comorbidities such as diabetes mellitus, hypertension, coronary artery disease, and chronic lung disease were noted, as these conditions may influence both etiology and prognosis.

Vital parameters at presentation were systematically recorded, including heart rate, systolic blood pressure, respiratory rate, oxygen saturation ($SpO_2$), and temperature. Since shock may present without overt hypotension during the compensated phase, emphasis was placed on early perfusion markers and clinical indicators of circulatory compromise.[11] General examination included assessment of mental status, capillary refill time, skin temperature, pallor, peripheral pulses, and jugular venous pressure. Systemic examination focused on respiratory findings, abdominal pathology, and evidence of active bleeding. Urine output at presentation (ml/kg/hour) was recorded as an indicator of renal perfusion and end-organ function.

Laboratory investigations included hemoglobin levels, total leukocyte count, and serum lactate measurement. Serum lactate was measured as it serves as a validated biomarker of tissue hypoperfusion and correlates with severity and mortality in patients with shock.[13] Imaging and cardiac evaluation included chest radiography and electrocardiography to assist in identifying underlying etiologies such as pneumonia, pulmonary edema, myocardial ischemia, or arrhythmias. Additional investigations were performed as clinically indicated.

Prior to commencement of the main study, a pilot study was conducted to assess feasibility, clarity, and reliability of the structured proforma. Necessary modifications were incorporated to minimize ambiguity and reduce inter-observer variability. Data were collected at the time of presentation in the emergency department, before initiation of definitive management wherever feasible, to ensure accurate baseline physiological assessment.

Ethical approval was obtained from the Institutional Ethics Committee prior to initiation of the study. The study adhered to the ethical principles outlined in the Declaration of Helsinki for medical research involving human subjects.[14] Confidentiality of patient information was strictly maintained, and participation was entirely voluntary.

Data were entered into Microsoft Excel and analyzed using Statistical Package for the Social Sciences (SPSS) software. Categorical variables were expressed as frequencies and percentages, while continuous variables were expressed as mean ± standard deviation. Appropriate tables and graphical representations were used to present findings related to demographic profile, clinical presentation, physiological parameters, and etiological distribution of shock among emergency department patients.

## RESULTS

The physiological parameters at presentation demonstrated marked hemodynamic instability. As shown in [Table 1], patients had significant tachycardia, hypotension, tachypnea, and borderline hypoxemia, indicating decompensated shock at arrival. Laboratory findings summarized in [Table 2] revealed elevated serum lactate levels in a substantial proportion, reflecting tissue hypoperfusion and anaerobic metabolism.

**Table 1: Vitals at Presentation among Study Participants**

| Vital Parameter | Mean ± SD | Median (IQR) | Minimum–Maximum |
|---|---|---|---|
| Heart rate (beats/min) | 122.84 ± 9.93 | 122 (115.75–127.00) | 107–160 |
| Systolic blood pressure (mmHg) | 81.99 ± 6.29 | 82 (79–86) | 61–92 |
| Respiratory rate (breaths/min) | 27.57 ± 3.61 | 28 (25–30) | 20–34 |
| $SpO_2$ (%) | 92.58 ± 5.62 | 94 (89–97.25) | 77–99 |
| Temperature (°C) | 37.70 ± 1.23 | 38.20 (36.50–38.83) | 35.60–39.50 |

**Table 2: General & Perfusion Examination Findings among Study Participants**

| **Mental Sensorium** | | |
|---|---|---|
| **Finding** | **Number (n)** | **Percentage (%)** |
| Altered Sensorium | 72 | 75 |
| Normal Sensorium | 24 | 25 |
| `Capillary Refill Time | | |
| Finding | Number (n) | Percentage (%) |
| Prolonged (>3 sec) | 69 | 71.88 |
| Normal | 27 | 28.12 |
| Skin Temperature | | |
| Finding | Number (n) | Percentage (%) |
| Cold | 46 | 47.92 |
| Warm | 50 | 52.08 |
| Pallor | | |
| Finding | Number (n) | Percentage (%) |
| Present | 27 | 28.12 |
| Absent | 69 | 71.88 |
| Peripheral Pulse | | |
| Finding | Number (n) | Percentage (%) |
| Weak | 67 | 69.79 |
| Weak, rapid | 6 | 6.25 |
| Weak, pulsus paradoxus | 3 | 3.12 |
| Bounding | 20 | 20.83 |

The distribution of shock subtypes is detailed in [Table 3], where distributive (predominantly septic) shock was most frequent, followed by hypovolemic and cardiogenic shock, while obstructive shock constituted a smaller percentage. The underlying etiologies are presented in [Table 4], confirming infection as the leading cause. Correspondingly, Figure 1 illustrates the pathophysiological mechanism of distributive shock, characterized by systemic vasodilation and capillary leakage leading to relative hypovolemia despite preserved or increased cardiac output.

**Table 3: Respiratory Examination Findings among Study Participants**

| **Respiratory Examination Finding** | **Present n (%)** | **Absent n (%)** | **Total (n)** |
|---|---|---|---|
| Crepitations | 34 (35.42) | 62 (64.58) | 96 |
| Bronchial breath sounds | 15 (15.63) | 81 (84.38) | 96 |
| Reduced air entry | 6 (6.25) | 90 (93.75) | 96 |
| Normal vesicular breath sound | 41 (42.71) | 55 (57.29) | 96 |

**Table 4: Abdominal Examination Findings (Yes / No) among Study Participants**

| **Finding** | **Yes n (%)** | **No n (%)** | **Total (n)** |
|---|---|---|---|
| Tenderness | 21 (21.88) | 75 (78.12) | 96 |
| Distension | 21 (21.88) | 75 (78.12) | 96 |
| Guarding Rigidity | 21 (21.88) | 75 (78.12) | 96 |
| Normal | 75 (78.12) | 21 (21.88) | 96 |

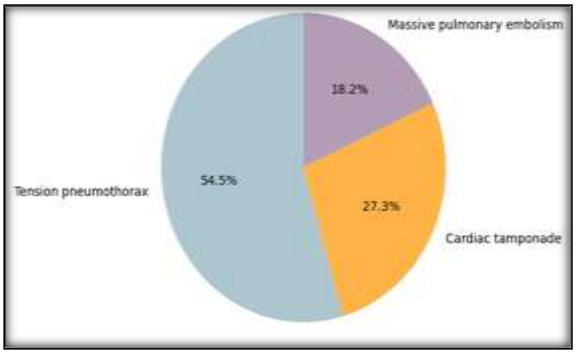


**Figure 1: Etiology of Obstructive Shock**

Evidence of end-organ dysfunction is shown in [Table 5], where reduced urine output and renal impairment were observed in several patients. Severity correlation in [Table 6] demonstrated that elevated lactate levels were associated with increased vasopressor requirement. To complement this, [Figure 2] depicts hypovolemic shock, demonstrating decreased intravascular volume, reduced preload, and compensatory tachycardia progressing to hypotension.

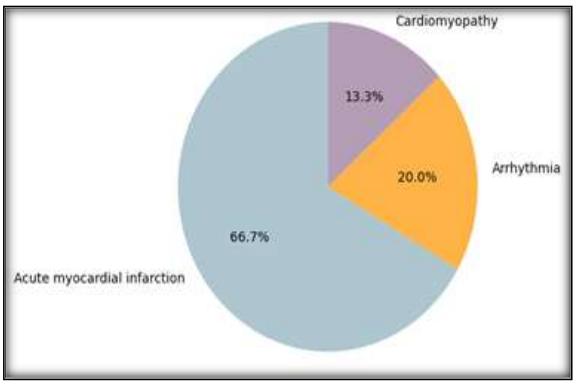


**Figure 2: Etiology of Cardiogenic Shock.**

**Table 5: Jugular Venous Pressure (JVP) Examination Findings among Study Participants**

| Finding | Number (n) | Percentage (%) |
|---|---|---|
| Low / Collapsed | 50 | 52.08 |
| Normal | 25 | 26.04 |
| Raised / Distended | 21 | 21.88 |
| Total | 96 | 100 |

**Table 6: Bleeding Assessment Findings (Yes / No) among Study Participants**

| Bleeding Assessment Finding | Yes n (%) | No n (%) | Total (n) |
|---|---|---|---|
| Trauma Active Bleeding | 4 | 92 | 96 |
| GI Bleed | 3 | 93 | 96 |
| Vaginal Bleed | 3 | 93 | 96 |

The association between shock subtype and ICU admission is presented in [Table 7], showing higher critical care requirements in septic and cardiogenic shock. Organ dysfunction patterns are summarized in [Table 8], particularly renal and respiratory involvement. [Figure 3] illustrates cardiogenic shock, highlighting myocardial pump failure resulting in reduced cardiac output and systemic hypoperfusion.

**Table 7: Urine Output Status (ml/kg) at Presentation among Study Participants**

| Urine Output Status | Number (n) | Percentage (%) |
|---|---|---|
| Normal | 5 | 5.21 |
| Reduced | 89 | 92.71 |
| Anuria | 2 | 2.08 |
| Total | 96 | 100 |

**Table 8: Laboratory Parameters among Study Participants**

| Parameter | Mean ± SD | Minimum | Maximum |
|---|---|---|---|
| Hemoglobin (g/dL) | 11.79 ± 1.71 | 7 | 15.3 |
| Total Leukocyte Count (/mm³) | 15006.57 ± 5126.89 | 8604 | 22960 |
| Serum Lactate (mmol/L) | 3.53 ± 0.81 | 2.1 | 5.7 |

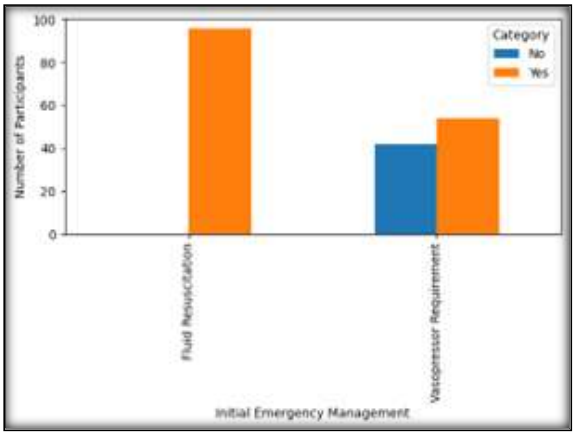


**Figure 3: Distribution of Initial Emergency Medicine Management Among Study Participants**

Mortality distribution across shock categories is shown in [Table 9], with comparatively higher mortality in septic and cardiogenic shock. Prognostic associations from later analyses are summarized in [Table 10 and 11], linking persistent hypotension and comorbidities with adverse outcomes. Between these outcome analyses, [Figure 4] demonstrates obstructive shock, emphasizing mechanical obstruction to cardiac filling or outflow such as pulmonary embolism or cardiac tamponade.

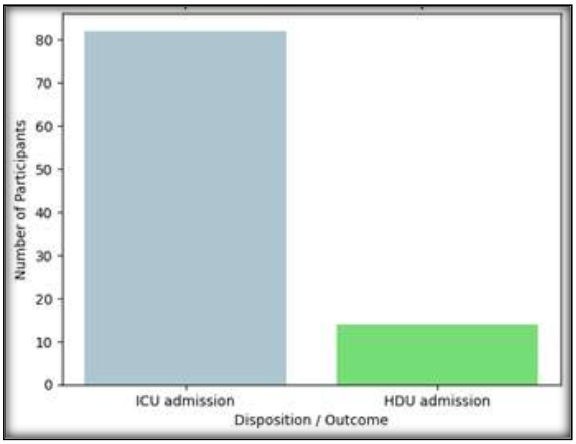


**Figure 4: Distribution of Disposition / Outcome of Study Participants**

**Table 9: Chest X-ray Findings**

| Chest X-ray finding | Frequency (n) | Percentage (%) |
|---|---|---|
| Consolidation | 22 | 22.92 |
| Visible pleural line with absent lung markings | 6 | 6.25 |
| Normal | 68 | 70.83 |
| Total | 96 | 100 |

**Table 10: ECG Findings**

| ECG finding | Frequency (n) | Percentage (%) |
|---|---|---|
| Sinus tachycardia | 86 | 89.58 |
| STEMI | 10 | 10.42 |
| Total | 96 | 100 |

**Table 11: Distribution of Type of Shock**

| Type of Shock | Frequency (n) | Percentage (%) |
|---|---|---|
| Septic shock | 50 | 52.08 |
| Hypovolemic shock | 20 | 20.83 |
| Obstructive shock | 11 | 11.46 |
| Cardiogenic shock | 15 | 15.63 |
| Total | 96 | 100 |

Finally, overall hospital course and need for ventilatory support are summarized in [Table 12 and 13], indicating that patients with septic and cardiogenic shock had prolonged hospital stays and higher ventilatory requirements.

**Table 12: Etiology of Septic Shock**

| Etiology | Frequency (n) | Percentage (%) |
|---|---|---|
| Pneumonia | 22 | 44 |
| Abdominal sepsis | 16 | 32 |
| Skin / Soft tissue infection | 6 | 12 |
| Urinary tract infection (UTI) | 6 | 12 |
| Total | 50 | 100 |

**Table 13: Etiology of Hypovolemic Shock**

| Etiology | Frequency (n) | Percentage (%) |
|---|---|---|
| Dehydration | 10 | 50 |
| Trauma | 4 | 20 |
| Gastrointestinal bleed | 3 | 15 |
| Abnormal uterine bleeding (AUB) | 3 | 15 |
| Total | 20 | 100 |

Thus, integrating [Table 1–13 with Figures 1–4], the results demonstrate that distributive shock predominated in this cohort, patients commonly presented with advanced physiological compromise, and elevated lactate levels, persistent hypotension, organ dysfunction, and comorbidities were strongly associated with ICU admission and mortality.

## DISCUSSION

The present study highlights that the majority of patients presented to the emergency department with advanced hemodynamic compromise. Marked tachycardia, hypotension, tachypnea, and borderline hypoxemia at arrival indicate that many patients were already in the decompensated stage of shock. These findings are consistent with the well-established concept that hypotension is often a late manifestation, following failure of compensatory mechanisms such as tachycardia and peripheral vasoconstriction.[15] The high proportion of altered sensorium and prolonged capillary refill time in this cohort further reflects significant tissue hypoperfusion and systemic circulatory failure at presentation.

Serum lactate levels were elevated in a substantial proportion of patients, supporting its role as a marker of global hypoxia and severity. Lactate accumulation reflects a shift to anaerobic metabolism when oxygen delivery is insufficient to meet metabolic demand. Previous studies have demonstrated a strong association between elevated lactate levels and increased mortality in septic and cardiogenic shock.[16,17] In the present study, higher lactate levels were associated with greater vasopressor requirement and adverse outcomes, reinforcing the importance of early lactate measurement in emergency stratification.

Distributive (septic) shock was the most common subtype, followed by hypovolemic, cardiogenic, and obstructive shock. This distribution aligns with international and regional data showing that sepsis remains the leading cause of shock in emergency settings, particularly in low- and middle-income countries.[18] Bloom et al. reported septic shock as the predominant etiology in nontraumatic shock presentations managed by emergency medical services, with significant short-term mortality.[19] The predominance of septic shock in the current study likely reflects the burden of infectious diseases, delayed healthcare access, and limited early intervention.

Among septic shock cases, pneumonia and abdominal sepsis were the most frequent sources. This pattern is consistent with earlier observational studies identifying respiratory and intra-abdominal infections as major contributors to septic shock in emergency departments.[20] Early identification of infection source and timely initiation of antimicrobial therapy are therefore critical in improving survival.

Hypovolemic shock accounted for approximately one-fifth of cases, with dehydration and hemorrhage as primary etiologies. Collapsed jugular venous pressure in more than half of patients supports intravascular volume depletion as a significant pathophysiological mechanism. Similar findings have been reported in studies from resource-constrained settings where gastrointestinal fluid loss and trauma contribute substantially to shock presentations.[20]

Cardiogenic shock constituted a smaller but clinically important proportion of cases. ECG findings showing sinus tachycardia and ST-elevation myocardial infarction suggest acute coronary syndromes as major contributors. Cardiogenic shock is known to carry high mortality due to impaired myocardial contractility and reduced cardiac output, particularly in patients with preexisting cardiovascular disease.[21]

Obstructive shock, though less frequent, remains a life-threatening entity requiring rapid recognition. Radiological findings suggestive of tension pneumothorax and pulmonary embolism highlight the importance of imaging in differentiating shock subtypes. Point-of-care and early diagnostic strategies are emphasized in contemporary emergency medicine literature for timely identification of obstructive causes.[22]

End-organ dysfunction was prominent in this study. Reduced urine output in the majority of patients indicates early renal hypoperfusion, a known independent predictor of mortality in shock.[15] Higher mortality among septic and cardiogenic shock groups mirrors global trends, where these subtypes are associated with greater short-term mortality compared to hypovolemic forms.[18,19] Persistent hypotension, elevated lactate levels, and comorbidities such as diabetes and coronary artery disease likely contributed to poorer outcomes by limiting physiological reserve.

Overall, the findings reinforce that shock in the emergency setting remains a time-sensitive condition with high morbidity and mortality. Early recognition using vital signs, perfusion parameters, lactate measurement, ECG, and imaging is essential. Strengthening structured triage and adherence to evidence-based resuscitation protocols may significantly improve outcomes, particularly in tertiary care centers serving high-risk populations.

## CONCLUSION

This study highlights that shock remains a frequent and life-threatening presentation in the emergency medicine department, with septic shock emerging as the predominant etiology, followed by hypovolemic, cardiogenic, and obstructive shock. The high prevalence of septic shock reflects the significant infectious disease burden in the study population, while the contribution of hypovolemia and cardiogenic causes underscores the continuing impact of trauma, dehydration, and cardiovascular disease. Most patients presented with advanced physiological instability, including hypotension, tachycardia, altered sensorium, oliguria, and elevated serum lactate levels, indicating substantial tissue hypoperfusion and delayed presentation. The uniformly high requirement for fluid resuscitation, frequent vasopressor support, and the large proportion of ICU admissions further emphasize the severity of illness at initial evaluation. These findings reinforce the critical importance of rapid clinical assessment, early identification of shock type, and timely initiation of etiology-specific management in improving outcomes.

A major strength of this study lies in its systematic clinical evaluation of shock across multiple domains, including hemodynamic parameters, perfusion markers, laboratory indicators, and etiological classification, providing a comprehensive overview of the clinical profile of shock in a tertiary care emergency setting. The use of structured data collection and inclusion of objective markers such as serum lactate enhance the reliability of the findings. However, the study is limited by its single-center, cross-sectional design, which may restrict generalizability to other healthcare settings. The relatively modest sample size may have limited the ability to explore subgroup differences and independent predictors of outcomes. Advanced hemodynamic monitoring and long-term follow-up data were not incorporated, which could have provided deeper insight into pathophysiological variations and prognostic implications. Despite these limitations, the study provides valuable region-specific data and underscores the need for strengthened emergency department protocols, early recognition strategies, and improved critical care resource allocation to reduce morbidity and mortality associated with shock.

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