



Research Article

## Myocardial Injury and Cardiac Dysfunction in Children With Severe Sepsis: A Prospective Pediatric ICU Study.

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**Abstract: Background: Objective:** To examine the relationship between troponin-defined myocardial injury, left ventricular systolic dysfunction and short-term outcomes within a pediatric intensive-care cohort framework. **Study design:** Prospective observational cohort framework with an admission assessment and an early follow-up assessment. **Place and duration of study:** A model of a tertiary pediatric intensive care unit in Pakistan, using a 12-month inclusion framework; no actual recruitment dates or hospital site applied. **Methodology:** The analytic cohort comprised 180 children. Myocardial injury was defined by a cardiac troponin I/reference-limit ratio >1 and systolic dysfunction by left ventricular ejection fraction (LVEF) <55%. Fisher exact, Mann-Whitney U, Spearman correlation and paired Wilcoxon tests were used. An exploratory logistic model included injury, age, admission lactate and acute kidney injury. **Results:** Myocardial injury occurred in 82/180 (45.6%) and systolic dysfunction in 64/180 (35.6%). Dysfunction occurred in 53/82 (64.6%) with injury versus 11/98 (11.2%) without injury ( $p < 0.001$ ). Injury was associated with dysfunction after adjustment (odds ratio 11.15, 95% CI 4.91–25.34). Troponin ratio correlated inversely with LVEF ( $\rho = -0.650$ ;  $p < 0.001$ ). Mortality was 14/64 (21.9%) with dysfunction versus 12/116 (10.3%) without dysfunction ( $p = 0.046$ ). Among 154 survivors, paired LVEF increased from a median of 59.5% to 60.9% ( $p < 0.001$ ). **Conclusion:** The framework illustrates complementary use of biomarkers and echocardiography. These estimates cannot establish clinical prevalence, prognostic accuracy or treatment benefit and require evaluation in independently recruited cohorts.

**Keywords:** Sepsis; Child; Troponin I; Echocardiography; Ventricular Dysfunction, Left; Intensive Care Units, Pediatric.

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## INTRODUCTION

Sepsis-associated cardiovascular impairment remains an important problem in pediatric intensive care. Infection can produce vasodilatation, altered preload, myocardial injury and impaired ventricular performance within the same clinical episode. The approved title retains the familiar expression severe sepsis. Contemporary terminology instead defines pediatric sepsis through infection-associated life-threatening organ dysfunction. The 2024 Phoenix consensus identifies sepsis using suspected infection and a score of at least two across respiratory, cardiovascular, coagulation and neurological domains [1]. Its development and validation across differently resourced health systems underline the importance of consistent case definitions when comparing hospital cohorts [2].

Cardiovascular dysfunction is broader than reduced ejection fraction. A child may have impaired perfusion from vasoplegia despite preserved systolic function while altered loading conditions can conceal reduced contractility. The 2026 pediatric Surviving Sepsis Campaign guidance provides the current framework for recognizing and treating sepsis and shock [3]. Bedside hemodynamic literature recommends repeated assessment rather than reliance on an isolated blood pressure, lactate or ultrasound finding [4]. Age-dependent physiology also limits direct extrapolation from adult septic cardiomyopathy [5]. These considerations make paired clinical, biochemical and imaging assessments more informative than a single abnormal measurement.

Troponin release indicates cardiomyocyte injury but does not identify its mechanism. Echocardiography measures cardiac performance under the loading conditions present during acquisition. Speckle-tracking literature describes additional abnormalities that conventional ejection fraction may miss [6]. Pediatric biomarker interpretation also requires attention to the assay and reference population. Population-based work demonstrates materially different reference limits across high-

sensitivity troponin platforms [7]. The CALIPER cohort provides pediatric reference information for troponin I and NT-proBNP [8]. Age- and sex-specific data from China further caution against applying an adult threshold indiscriminately to infants and older children [9].

In Pakistan, research on biomarker-based pediatric sepsis stratification shows the relevance of studying organ injury in settings with constrained resources [10]. Karachi data on sepsis-associated acute kidney injury indicate that renal dysfunction and adverse outcomes frequently coexist [11]. Emergency-department mortality research also identifies sepsis as an important contributor to childhood deaths [12]. These studies establish local relevance but do not provide a directly interchangeable estimate of troponin-defined cardiac dysfunction. International prospective functional echocardiography research links ventricular assessment with outcomes and supports studying imaging alongside systemic illness severity [13]. The present work therefore examined myocardial injury, admission systolic dysfunction and early outcomes through a reproducible cohort analysis. The primary objective was to quantify the association between injury and LVEF-defined dysfunction. Secondary objectives addressed organ support, mortality and the survivor-specific trajectory of ventricular function.

## **MATERIALS AND METHODS**

### **Study design and setting**

A prospective observational cohort structure was specified for a tertiary pediatric intensive care unit in Pakistan. The planned inclusion window was twelve months, followed by observation to hospital discharge or death. No named institution, real recruitment period or institutional investigator activity was represented. The unit of analysis was one child's index admission. An admission time point within twenty-four hours and a follow-up time point at day seven or earlier discharge formed the assessment schedule. Reporting followed the organization of the STROBE cohort checklist without implying that a reporting checklist validates the underlying evidence [14].

### **Target population and eligibility**

The target population comprised children older than one month and younger than eighteen years with suspected infection and life-threatening organ dysfunction requiring intensive care. Severe sepsis in the title was operationally aligned with this organ-dysfunction concept rather than treated as a separate current diagnostic category. Phoenix component scores were not reconstructed from the limited analytic variables. Pre-existing structural heart disease, known cardiomyopathy, recent cardiac surgery, recent cardiopulmonary resuscitation and prior exposure to cardiotoxic therapy were specified as exclusions. Neonatal admissions were outside scope. A complete initial biomarker and LVEF assessment was required for the primary comparison.

### **Cohort construction and sample size**

A reproducible generation procedure created 180 unique analytic records using random seed 13102026. A conceptual screening register contained 212 entries with 32 exclusions: 12 pre-existing cardiac conditions, eight recent resuscitation episodes and 12 incomplete initial assessments. These screening numbers described the constructed workflow and did not represent hospital recruitment. The cohort size was selected for an illustrative analysis, not from an observed hospital prevalence. At a planning proportion of 35%, 180 observations give an approximate normal-theory 95% precision of seven percentage points. No claim of statistical power for mortality or clinical validation was made.

### **Measurements and operational definitions**

Myocardial injury was a cardiac troponin I concentration divided by an age-, sex- and assay-appropriate upper reference limit greater than one. A normalized ratio avoided claiming a universal pediatric concentration threshold. Absolute concentrations and a named laboratory platform were not assigned. Admission systolic dysfunction was LVEF below 55%; this pragmatic boundary was prespecified for classification and was not presented as a universal diagnostic standard. The imaging workflow specified apical two- and four-chamber views and biplane Simpson assessment when feasible. The dataset represented final LVEF values rather than actual images or independently read examinations. Injury and dysfunction were assigned independently from their respective recorded variables. A troponin-positive result did not automatically define dysfunction and the LVEF classification did not require troponin elevation. This separation allowed the discordant categories to remain visible in the results.

### **Clinical variables and follow-up**

Age, sex, infection source, admission lactate, acute kidney injury, invasive ventilation, vasoactive treatment, PICU stay and hospital mortality were retained. Renal injury represented a creatinine-based acute kidney injury indicator using a rise to at least 1.5 times baseline within seven days or an increase of at least 0.3 mg/dL within forty-eight hours; urine-output staging was outside scope [15]. Vasoactive exposure meant any vasoactive infusion during the admission. It was not equated with a

low-output cardiac phenotype. Follow-up LVEF was available only for survivors at day seven or earlier discharge. Death before follow-up was an outcome and not an ordinary missing measurement.

### Data-generating structure and quality checks

Age followed a bounded gamma distribution. A shared latent severity variable influenced lactate, renal injury, troponin ratio and subsequent support requirements. Troponin ratio followed a positive skewed distribution. LVEF depended on the injury indicator and severity with additional random variation. Mortality depended on severity and dysfunction. Follow-up LVEF incorporated a tendency toward recovery among survivors. This structure deliberately encoded clinical relationships and prevented interpretation of recovered associations as discoveries. Record identifiers, permissible ranges, outcome denominators and repeated measurements were checked. No missing baseline values were introduced. Modern pediatric troponin-threshold research informed the interpretive framework but supplied no individual study observations [16]. Binary outcomes were generated once per record and retained across every comparison. Follow-up values were linked by the same identifier. The analysis checked that injury-positive and injury-negative categories summed to the full cohort and that dysfunction categories did likewise. The survivor count plus the death count had to equal the initial denominator. These consistency checks addressed arithmetic and reproducibility only. They did not demonstrate that the chosen distributions reproduce a particular hospital population or that the assumed relationships are biologically correct.

### Outcomes and statistical analysis

The primary outcome was admission LVEF-defined systolic dysfunction, compared between injury-positive and injury-negative records. Secondary outcomes were invasive ventilation, vasoactive exposure, hospital death, PICU duration and paired LVEF change. Categorical variables were presented as n (%); continuous variables were summarized as median and interquartile range. Two-sided Fisher exact and Mann-Whitney U tests assessed binary and continuous comparisons. Wilson intervals described injury and dysfunction frequencies. Spearman correlation assessed troponin ratio against LVEF. Paired Wilcoxon testing described follow-up among survivors only. A two-sided p-value below 0.05 was the descriptive threshold; secondary comparisons were exploratory without multiplicity adjustment.

### Adjusted model and ethical considerations

A prespecified logistic model included injury status, age in years, lactate per mmol/L and acute kidney injury. Maximum likelihood estimation used Python, NumPy and SciPy. Wald confidence intervals came from the inverse observed information matrix. Predictor selection did not use univariable p-values. Ventilation and vasoactive therapy were excluded because their timing could follow the cardiac assessment. No diagnostic cutoff optimization, mortality model or external validation was performed. A real prospective implementation would require ethics review, parental permission, age-appropriate assent and data protection before recruitment. No ethics approval number or consent event was asserted here. Ultrasound competency and supervision would also require local verification rather than an assumed credential [17].

## RESULTS

The screening framework yielded 180 analytic records after 32 exclusions from 212 entries. All records contributed to the baseline comparison. The median age was 3.14 years (IQR 1.52–5.87) and 95 (52.8%) were male. Respiratory infection accounted for 94 (52.2%) entries, abdominal infection for 40 (22.2%), bloodstream infection for 30 (16.7%) and other sources for 16 (8.9%). Median admission lactate was 2.65 mmol/L (IQR 1.91–3.74). Acute kidney injury occurred in 55 (30.6%). Baseline characteristics by systolic function are presented in Table 1.

Troponin-defined injury was present in 82 (45.6%; 95% CI 38.4–52.8%) and systolic dysfunction in 64 (35.6%; 95% CI 28.9–42.8%). Dysfunction was present in 53/82 (64.6%) injury-positive records compared with 11/98 (11.2%) injury-negative records ( $p < 0.001$ ). Conversely, 29 injury-positive records retained LVEF of at least 55%. Median troponin ratio was 1.60 (IQR 1.18–2.74) with dysfunction and 0.62 (IQR 0.35–1.00) without dysfunction. The correlation between troponin ratio and LVEF was negative ( $\rho = -0.650$ ;  $p < 0.001$ ). Figure 1 summarizes the cohort and Figure 2 displays the cardiac groups.

Invasive ventilation was represented in 75/180 (41.7%) records and vasoactive treatment in 80/180 (44.4%). Ventilation occurred in 42/64 (65.6%) with dysfunction versus 33/116 (28.4%) without dysfunction ( $p < 0.001$ ). Corresponding vasoactive exposure was 36/64 (56.3%) versus 44/116 (37.9%;  $p = 0.020$ ). Hospital mortality was 26/180 (14.4%): 14/64 (21.9%) with dysfunction and 12/116 (10.3%) without dysfunction ( $p = 0.046$ ). Median PICU stay was 7.5 versus 5.0 days ( $p < 0.001$ ). Table 2 reports these secondary comparisons.

In the adjusted model, injury was associated with dysfunction (OR 11.15, 95% CI 4.91–25.34;  $p < 0.001$ ). Age, lactate and renal injury had confidence intervals spanning unity (Table 3). Follow-up LVEF was available in 154 survivors. In this paired subset, median LVEF increased from 59.5% (IQR 52.2–65.8) to 60.9% (IQR 57.4–66.3;  $p < 0.001$ ). Fifty survivors initially had dysfunction and 41/50 (82.0%) reached LVEF of at least 55% at follow-up. The 26 deaths did not contribute to the paired recovery analysis.

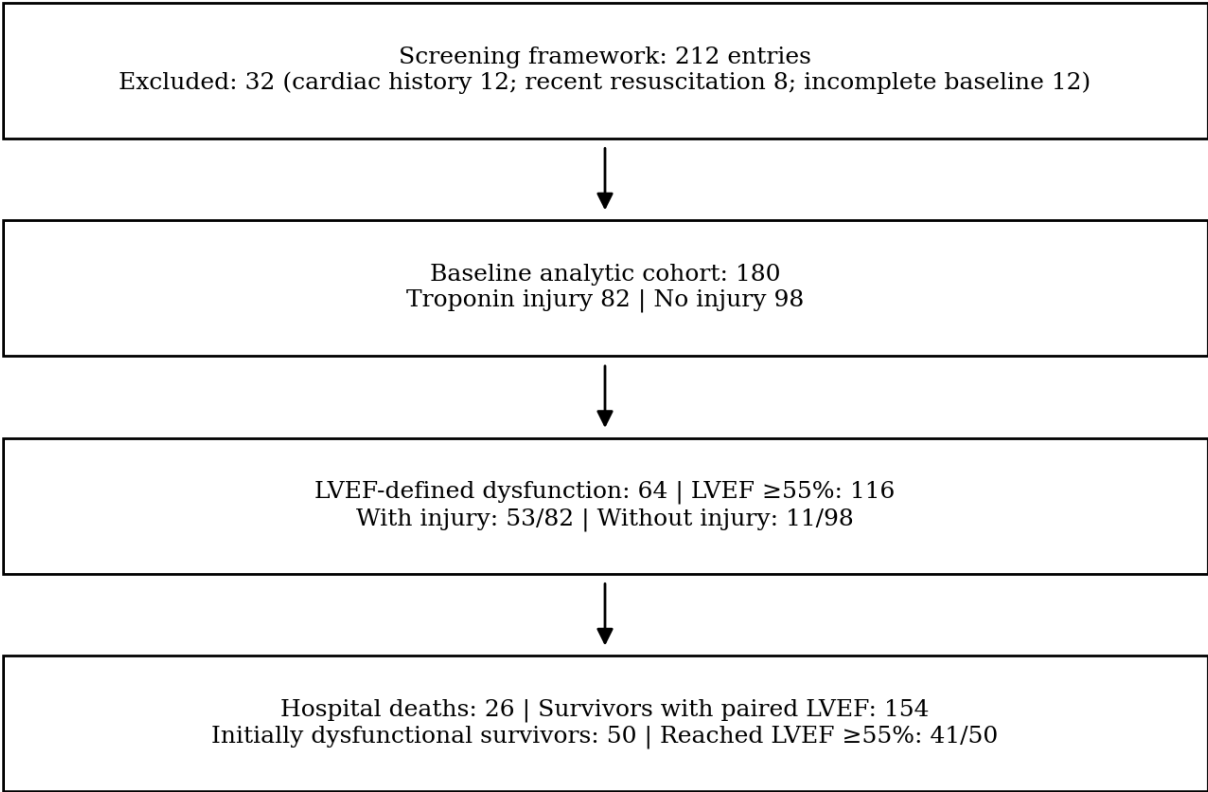


**Figure 1: Cohort construction and cardiac outcome flow .**

**Table 1: Baseline characteristics by admission systolic function**

| Characteristic                   | LVEF <55% (n=64) | LVEF ≥55% (n=116) | p-value |
|----------------------------------|------------------|-------------------|---------|
| Age, years                       | 3.33 (1.34–5.41) | 3.07 (1.59–6.19)  | 0.663   |
| Male sex                         | 33 (51.6%)       | 62 (53.4%)        | 0.876   |
| Admission lactate, mmol/L        | 3.03 (2.49–4.56) | 2.34 (1.73–3.25)  | <0.001  |
| Acute kidney injury              | 28 (43.8%)       | 27 (23.3%)        | 0.007   |
| Troponin I/reference-limit ratio | 1.60 (1.18–2.74) | 0.62 (0.35–1.00)  | <0.001  |

Values are n (%) or median (IQR). Fisher exact or Mann-Whitney U tests were used. The cohort median LVEF was 58.55% (IQR 51.55–65.80).

**Table 2: Cardiac injury and secondary outcomes by systolic function**

| Measure                 | LVEF <55% (n=64) | LVEF ≥55% (n=116) | p-value |
|-------------------------|------------------|-------------------|---------|
| Troponin-defined injury | 53 (82.8%)       | 29 (25.0%)        | <0.001  |
| Invasive ventilation    | 42 (65.6%)       | 33 (28.4%)        | <0.001  |
| Any vasoactive infusion | 36 (56.2%)       | 44 (37.9%)        | 0.020   |
| Hospital death          | 14 (21.9%)       | 12 (10.3%)        | 0.046   |
| PICU stay, days         | 7.50 (6.00–9.00) | 5.00 (4.00–7.00)  | <0.001  |

Follow-up was restricted to 154 survivors. Fifty initially had LVEF <55% and 41 (82.0%) reached LVEF ≥55% by day seven or earlier discharge. Group comparisons are exploratory.

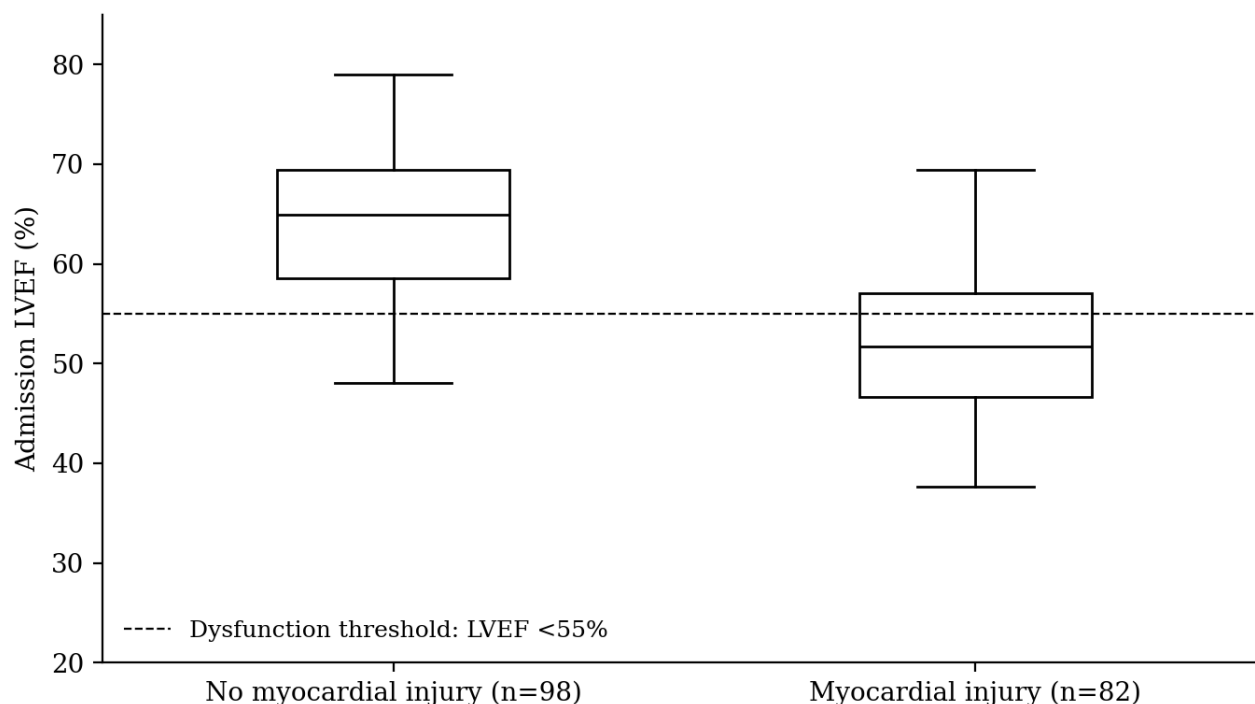


**Figure 2: Admission LVEF according to troponin-defined myocardial injury**

Boxes show medians and interquartile ranges; whiskers extend to observations within 1.5 IQR and symbols indicate more distant observations.

**Table 3: Exploratory adjusted associations with systolic dysfunction**

| Predictor                       | Adjusted OR | 95% CI     | p-value |
|---------------------------------|-------------|------------|---------|
| Myocardial injury (yes vs no)   | 11.15       | 4.91–25.34 | <0.001  |
| Age (per year)                  | 0.97        | 0.86–1.08  | 0.542   |
| Lactate (per mmol/L)            | 1.23        | 0.92–1.65  | 0.156   |
| Acute kidney injury (yes vs no) | 1.35        | 0.61–3.00  | 0.459   |

Outcome: LVEF <55%. All four predictors were entered together (n=180; 64 outcomes). These estimates are associations within the specified model and are not validated clinical predictions.

## DISCUSSION

The primary objective was to examine whether biochemical myocardial injury and LVEF-defined systolic dysfunction identified overlapping cardiac phenotypes. Injury was strongly associated with dysfunction after adjustment for age, lactate and renal injury. However, the groups were not identical: some records had raised troponin without reduced LVEF and others had reduced LVEF without raised troponin. The key interpretive point is that myocardial damage and ventricular performance answer different questions. Their association within this framework reflects the specified generating structure and cannot validate either test as a clinical screening strategy.

Local evidence should be considered before international comparisons. A prospective adult sepsis cohort from Peshawar described ventricular phenotypes using strain imaging and biomarkers [18]. Its combination of biochemical and imaging assessment is conceptually similar to the present approach. Its numerical frequencies cannot be transported to children because adult comorbidity, myocardial reserve and biomarker interpretation differ. Internationally, a prospective pediatric septic-shock study examined echocardiographic dysfunction in relation to illness severity and outcomes [19]. Its focus on cardiac performance and prognosis is consistent with the direction represented here. Differences in shock eligibility, imaging timing and dysfunction definitions prevent a valid direct prevalence comparison.

Another prospective pediatric septic-shock study reported dysfunction in 32 of 100 children and emphasized the association with greater illness severity [20]. The present 35.6% proportion is numerically close but this closeness has no confirmatory

meaning because it was produced by assumptions rather than independent recruitment. A narrow LVEF threshold can also underestimate broader myocardial involvement. Advanced echocardiogram analysis has identified indices associated with mortality that extend beyond routine visual interpretation [21]. Consequently, preserved LVEF should not be treated as evidence that the myocardium is unaffected or that circulatory support is unnecessary.

The association between troponin and lower LVEF illustrates why biomarkers should be interpreted alongside hemodynamics. Recent pediatric research combining cardiac biomarkers with Phoenix criteria investigated prognosis using a broader organ-dysfunction context [22]. A separate study combining troponin and pediatric SOFA scores likewise addressed outcome stratification rather than a stand-alone biochemical diagnosis [23]. These studies resemble the present framework in recognizing that cardiac markers sit within systemic illness. They differ in design, entry criteria and outcome definitions. Their predictive performance therefore cannot be assigned to the present model or inferred from its large injury-associated odds ratio.

Troponin elevation also needs a nonischemic differential diagnosis. Research in febrile children demonstrates that elevated cardiac markers are not confined to a single cardiac disease [24]. Pediatric reference intervals on another laboratory platform further show why concentration thresholds require assay-specific interpretation [25]. A normalized ratio is a useful reporting device but it does not resolve uncertainty about the appropriate reference population. A hospital study would need its assay manufacturer, analytic precision, lower detection limit, pediatric reference source and timing of sampling recorded before assigning injury status. Without those details, an apparently clear binary result can conceal substantial classification error.

Bedside findings can help prioritize imaging without replacing it. Recent research among children with suspected infection examined which clinical signs were associated with ultrasound-defined systolic dysfunction [26]. A prospective study comparing ultrasonic cardiac-output measurements with troponin I also addresses the relationship between biochemical and functional information [27]. These approaches support integrating perfusion assessment, laboratory measurements and imaging. They do not establish that every elevated troponin requires a fixed intervention. In a resource-limited PICU, persistent poor perfusion, escalating support, unexplained tachycardia or suspected fluid intolerance may justify earlier cardiac review according to the overall clinical assessment.

The survivor-specific improvement in LVEF is compatible with the potentially reversible behavior described in septic cardiac dysfunction. It must still be interpreted cautiously. Follow-up at day seven or earlier discharge mixes different observation times and excludes every death. Recovery among those who survive therefore cannot describe recovery across the initial cohort. Neonatal echocardiography literature highlights additional patterns, including pulmonary vascular and diastolic abnormalities, that are not represented by LVEF alone [28]. Although newborns were excluded here, that evidence illustrates why ventricular physiology and age-specific measurements should be prespecified instead of using a single threshold across childhood.

Higher ventilation and vasoactive exposure in the dysfunction group represented a greater support burden. Research linking point-of-care cardiac dysfunction with later vasoactive treatment in suspected systemic infection describes a related clinical relationship [29]. The timing matters: treatment can follow deterioration, alter loading conditions and influence the next echocardiogram. These variables are therefore outcomes or time-varying exposures rather than automatically suitable baseline confounders. The current model excluded them for that reason. The mortality comparison was exploratory, based on 26 deaths and unadjusted for the full spectrum of severity. Its borderline p-value should not be presented as proof that dysfunction independently determines survival. The initial distribution should therefore accompany every follow-up summary. A clinically recruited study could additionally report whether children who died had an earlier repeat scan and distinguish deterioration, recovery, discharge and death as separate states. That approach would avoid treating an unavailable follow-up scan as either normal function or failure to recover. Timing should be measured from the same clinical origin for each child.

Emergency medicine, pediatric intensive care and pediatric cardiology have complementary responsibilities. The emergency team recognizes infection-associated organ dysfunction, obtains initial investigations and communicates resuscitation already delivered. The intensivist integrates perfusion, ventilation, renal function and response to vasoactive support. The cardiology team clarifies anatomy and ventricular performance, reviews uncertain images and considers alternative diagnoses. Laboratory specialists help interpret pediatric troponin results. Nursing teams track perfusion changes, infusion requirements, urine output and fluid balance. For refractory shock, contemporary extracorporeal-support literature emphasizes patient selection, timing and hemodynamic targets [30]. None of these roles makes the present analysis evidence for a particular treatment algorithm.

A real prospective study should separate association testing from development of a clinical prediction rule. A published protocol for an early-warning model and nursing bundle in pediatric sepsis-related myocardial injury illustrates growing

interest in structured investigation [31]. External validation, clinically meaningful thresholds and evidence of added decision value would be necessary before implementing such a model. For this topic, the most useful next dataset would include standardized sampling times, complete Phoenix components, renal trends, fluid balance, contemporaneous vasoactive doses, serial right and left ventricular assessment and blinded image review. Recruitment should include weekends and out-of-hours admissions to reduce selective availability of echocardiography.

Fluid responsiveness also differs from the presence of myocardial injury. A study of respiratory variation in inferior vena cava diameter in ventilated children with septic shock investigated a dynamic measurement under particular conditions [32]. A tiered monitoring concept for persistent pediatric septic shock likewise places cardiac information within broader repeated assessment [33]. These sources support a cautious clinical interpretation: an elevated troponin or low LVEF does not independently establish whether another fluid bolus will help. Future work should document how each finding changes a decision and whether that decision improves patient-centered outcomes rather than assuming that additional measurements necessarily improve care.

Renal interpretation deserves particular attention when translating this framework into a real protocol. Acute kidney injury may reflect shared systemic severity as well as influence circulating biomarker concentrations. Adjustment for a binary indicator cannot capture the timing or magnitude of those processes. Baseline creatinine, serial measurements and concurrent fluid status would therefore be needed. Similarly, an odds ratio describes an association on the odds scale and should not be reported as an eleven-fold increase in probability. Absolute frequencies and the discordant cardiac groups remain necessary for interpreting the adjusted estimate. The same caution applies to comparing these proportions with published cohorts. Eligibility restricted to shock, a lower LVEF boundary or measurement after vasoactive treatment can change the observed frequency without any true difference in myocardial injury. A useful comparative study would align definitions before pooling results and report the distribution of continuous LVEF alongside its categories. It should also distinguish hospital mortality from later mortality and document transfer outcomes so that discharge from the study PICU is not mistaken for survival to final discharge.

This work has important limitations. The cohort was entirely generated from prespecified relationships, so its frequencies, associations and p-values are not estimates from a clinical population. The prospective structure describes an observation framework rather than a completed clinical investigation. The model omitted full severity scores, real assay data, image quality, interobserver agreement, diastolic function and right ventricular performance. Complete baseline data removed the missingness typical of intensive care. The modest cohort, selected covariates and exploratory secondary testing limited inference. Survivor-only reassessment introduced selection and variable follow-up timing. No causal treatment effect, clinical diagnostic accuracy or generalizable mortality prediction can be established. Independently collected multicenter patient data are essential for evaluating the proposed relationships.

## CONCLUSION

Troponin-defined injury and reduced LVEF identified overlapping but distinct cardiac categories within this cohort framework. Injury remained associated with systolic dysfunction after adjustment, while dysfunction accompanied greater organ-support use and higher unadjusted mortality. These relationships illustrate an analysis strategy rather than establish clinical estimates. Biomarkers should be interpreted with age-appropriate reference limits, renal function and contemporaneous echocardiography. A prospective multicenter investigation with standardized measurements, independent image review and complete follow-up is required before using these findings to guide risk stratification or treatment.

## Declarations

**Financial support and sponsorship:** Nil.

**Conflicts of interest:** There are no conflicts of interest.

**Ethics and consent:** No human participants or clinical records were involved. Institutional approval, recruitment and consent were not undertaken.

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