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ORIGINAL ARTICLE

# The Prognostic Value of Plasma Brain Natriuretic Peptide as a Predictor of Weaning Outcome from Mechanical Ventilation in Critically Ill Children

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## ABSTRACT

**Background:** Safe weaning from mechanical ventilation in children remains challenging. Conventional weaning indices often lack accuracy. B-type natriuretic peptide (BNP) may reflect cardiovascular stress during spontaneous breathing trials (SBTs) and provide prognostic value in weaning from mechanical ventilation. This study aimed to evaluate the predictive value of BNP levels for weaning outcomes in critically ill pediatric patients.

**Methods:** A prospective cohort study was conducted in the pediatric intensive care unit (PICU) in Zagazig University Pediatric Hospital on forty-two intubated children meeting SBT readiness. BNP was measured at SBT initiation (BNP I) and completion (BNP II). Clinical, laboratory, and outcome data were recorded. The primary endpoint was weaning outcome (success vs. failure, defined as failed SBT or reintubation ≤48 h post extubation.

**Results:** Weaning succeeded in 73.8% and failed in 26.2%. BNP I and BNP II were significantly higher in failures vs. successes (954.3 ± 44.6 vs. 824.0 ± 71.2 pg/mL; 939.6 ± 42.1 vs. 773.0 ± 155.3 pg/mL; both $p<0.001$). BNP I independently predicted weaning failure (Adjusted Odds Ratio (AOR) of 1.059; 95% CI 1.006–1.115; $p=0.029$). BNP I correlated positively with PICU stay duration ($r=0.515$, $p<0.001$). BNP II correlated positively with respiratory rate ($r=0.329$, $p=0.033$), pH ($r=0.319$, $p=0.033$), BNP I ($r=0.445$, $p=0.003$), and PICU stay ($r=0.491$, $p<0.001$), and negatively correlated with Glasgow coma scale (GCS) ($r=-0.466$, $p=0.002$). ROC analysis showed BNP I ≥892.7 pg/mL predicted weaning failure with area under curve (AUC) 0.962, sensitivity 90.9%, specificity 87.1%, positive predictive value (PPV) 71.4%, negative predictive value (NPV) 96.4% ($p<0.001$). BNP II ≥905.2 pg/mL also performed well (AUC 0.921, sensitivity 81.8%, specificity 87.1%, PPV 69.2%, NPV 93.1%, $p<0.001$). Survivors showed greater BNP reduction than non-survivors (6.13% vs. −0.39%, $p<0.001$).

**Conclusion:** BNP, particularly baseline BNP I, could be an independent predictor of weaning failure in critically ill children. It outperformed conventional indices and was associated with prolonged PICU stay and mortality.

**Keywords:** Brain Natriuretic Peptide, Predictor, Weaning Outcome, Mechanical Ventilation, Critically Ill Children.

## INTRODUCTION

Mechanical ventilation remains a cornerstone of supportive care for critically ill children admitted to the pediatric intensive care unit (PICU). While it provides essential respiratory support, its prolonged use carries risks, including airway trauma, inflammation, and secondary

complications. Therefore, discontinuing ventilatory support at the earliest safe opportunity is critical to improve outcomes and reduce morbidity **[1].**

Weaning from mechanical ventilation continues to be one of the most challenging aspects of intensive care. It requires substantial attention from PICU staff, and both premature and delayed extubation can result in harm. Early removal of ventilatory support may cause extubation failure and hypoxemia, whereas unnecessary prolongation increases the risk of complications. Currently, no universally standardized approach exists to assess extubation readiness in children **[1,2].**

Extubation failure, typically defined as the need for reintubation within 48 hours following planned extubation, remains a significant concern in pediatric critical care. Reported rates range between 2% and 20%, depending on patient characteristics and institutional practices. Such failures are linked to prolonged PICU stay, extended duration of invasive ventilation, higher risk of tracheostomy, and increased mortality **[3,4].**

The mechanisms underlying weaning failure are multifactorial and include abnormalities in lung mechanics, respiratory muscle fatigue, cardiovascular dysfunction, neurological impairment, and metabolic derangements. Among these, cardiac dysfunction plays a particularly important role. Transitioning from positive pressure ventilation to spontaneous breathing increases cardiac workload and may precipitate subclinical myocardial dysfunction. Unfortunately, conventional diagnostic methods such as echocardiography are often operator-dependent and less sensitive in critically ill patients **[5–7].**

Biomarkers such as B-type natriuretic peptide (BNP) and its inactive fragment, N-terminal proBNP (NT-proBNP), are well-established indicators of cardiac strain and heart failure. Both are secreted by ventricular myocytes in response to increased wall stress. BNP has a relatively short half-life of approximately 20 minutes, whereas NT-proBNP persists longer, with a half-life of nearly 120 minutes. During weaning, especially during SBTs, transient elevations in left ventricular afterload may lead to subclinical pulmonary congestion or overt pulmonary edema. These changes can be detected by monitoring BNP and NT-proBNP levels, making them potentially valuable tools in predicting extubation outcomes **[8,9].**

Despite growing evidence in adult populations, pediatric data remain limited. Current studies provide important insights, but heterogeneity in patient groups and methodologies restricts their generalizability. Therefore, this study aimed to evaluate the predictive value of BNP levels for weaning outcomes in critically ill pediatric patients.

## METHODS

This prospective cohort study was conducted at the Pediatric Intensive Care Unit (PICU) of the Children's Hospital, and Clinical Pathology Department at Zagazig University, over a 13-month period from August 2023 to September 2024 after obtaining approval from the Institutional Review Board (IRB#10593) and written informed consent from all cases' relatives. Guardians were informed about the study aims, potential benefits/risks, and their right to decline or withdraw at any time without impact on clinical care. The research was conducted under the World Medical Association's Code of Ethics (Helsinki Declaration) for human research.

All intubated patients aged between 1 month and 16 years who were admitted to the PICU and required mechanical ventilation were eligible.

We included children who met the following criteria:

1. Duration of ventilation: Intubated and mechanically ventilated for more than 48 hours.
2. Hemodynamic stability: Demonstrated stable cardiovascular parameters (no requirement for escalating inotropic support) and were considered clinically suitable for a Spontaneous Breathing Trial (SBT) — a structured period of

spontaneous ventilation lasting 30–120 minutes, during which the child breathes spontaneously without ventilator assistance (e.g., on CPAP or T-piece) to assess readiness for extubation.

3. Identified etiology of ventilation: The underlying cause of mechanical ventilation was classified as one of the following categories: Respiratory causes (e.g., pneumonia, respiratory distress, or respiratory failure). Cardiac causes (e.g., heart failure, postoperative cardiac dysfunction). Central/neurological causes (e.g., apnea, neuromuscular dysfunction, disturbance of consciousness)

Patients were excluded if they met any of the following criteria:

1. Early mortality: Died within 48 hours of PICU admission.
2. Cardiac or renal conditions: Had a history of congenital heart disease, significant arrhythmias, congestive heart failure, or cardiogenic shock, as well as acute or chronic renal failure.
3. Chronic or neuromuscular diseases: Children with known neuromuscular disorders or severe chronic lung disease were also excluded.

**Clinical Evaluation and Baseline Assessment**

A detailed history was obtained, including demographic data, nutritional status, and socioeconomic background. Each child underwent thorough physical examination. Anthropometric measurements (weight and height), vital signs (heart rate, respiratory rate, blood pressure, and temperature), and Glasgow Coma Scale (GCS) were recorded. Routine laboratory tests (arterial blood gases, electrolytes, hemoglobin, renal and liver function tests) and imaging (chest radiography) were performed when indicated.

**Extubation Readiness Criteria**

Extubation readiness was assessed daily based on the following criteria: clinical stability with minimal or no inotropic support; adequate neurological status (Glasgow Coma Scale score > 10 and an intact cough reflex); acceptable ventilatory parameters, including exhaled tidal volume ≥ 5 mL/kg, pressure support ≤ 10 cm $H_2O$, fraction of inspired oxygen ($FiO_2$) ≤ 0.4, positive end-expiratory pressure (PEEP) ≤ 5 cm $H_2O$, and peripheral oxygen saturation ($SpO_2$) ≥ 94%; sufficient oxygenation with a $PaO_2/FiO_2$ ratio > 200; appropriate arterial blood gases (pH 7.35–7.45, $PaCO_2$ 35–50 mmHg, $PaO_2$ ≥ 80 mmHg); and hematologic and electrolyte stability, defined as hematocrit ≥ 30% with serum sodium 135–145 mEq/L and potassium 3.5–5 mEq/L.

**Spontaneous Breathing Trial (SBT) and Weaning Process**

Children who met these criteria underwent a 2-hour SBT using pressure support ventilation (≤10 cm $H_2O$) with CPAP at 5 cm $H_2O$. At the start of SBT, tidal volume, respiratory rate, rapid shallow breathing index (RSBI), minute ventilation, and $PaO_2/FiO_2$ ratio were recorded as predictors of extubation outcome [10].Extubation was performed in those who successfully tolerated SBT. In patients with neurological impairment, sedatives and analgesics were tapered before extubation. Post-extubation stridor was managed with nebulized adrenaline (0.5 mL/kg, maximum 4 mL of 1:1000 solution) and intravenous dexamethasone (0.2 mg/kg/dose every 6 hours for 2 days) when reintubation was necessary. Weaning failure was defined as either inability to tolerate SBT or requirement for reintubation within 48 hours after extubation.

**Severity Assessment**

The Pediatric Risk of Mortality (PRISM) score was calculated for each patient. Physiologic variables were measured in the first 4 hours of PICU admission, while laboratory values were obtained within 2 hours before admission through the initial 4 hours. Neurologic score was derived from GCS and pupillary reflexes, and non-neurologic components were calculated from remaining parameters [11].

**Laboratory investigations**

Electrolyte levels, including sodium ($Na^+$) and potassium ($K^+$), were determined using the Cobas ISE module (Roche Diagnostics, Mannheim, Germany). Hematological markers were assessed by complete blood count (CBC) with the Sysmex XN-2000 analyzer (Sysmex, Kobe, Japan) and C-reactive protein (CRP) measurement using the Cobas analyzer (Roche Diagnostics, Mannheim, Germany). Arterial blood gases (ABG) were analyzed from freshly collected arterial samples with the Cobas b 221 system (Roche Diagnostics, Mannheim, Germany). Kidney function was evaluated by measuring serum creatinine (SCr) and blood urea nitrogen (BUN) at admission and at 24 and 48 hours using the Roche Cobas 8000 c702 module. Liver function, specifically serum albumin levels, was determined with the Cobas auto analyzer (Roche Diagnostics, Mannheim, Germany).

**BNP measurement**

Plasma BNP concentrations were determined using a ready-to-use Human Brain Natriuretic Peptide (BNP) ELISA Kit (Catalog No. DLR-BNP-Hu, DEVELOP®, a multinational Chinese–Canadian company; www.dldevelop.com). It is based on the sandwich enzyme-linked immunosorbent assay (ELISA) principle and is specifically designed for the quantitative measurement of BNP in human serum and plasma.

According to the manufacturer's specifications, the assay has a detection range of 2–2000 pg/mL, with a minimum detectable concentration of 1.5 pg/mL. Blood samples were collected into EDTA tubes, centrifuged within 30 minutes, and plasma aliquots were stored at –80 °C until analysis.[8, 12].

**Outcome Measures**

Primary outcome was weaning success or failure. Secondary outcomes included length of PICU stay and patient disposition (discharge or death).

**Statistical Analysis**

Data were analyzed using SPSS version 28 (IBM Corp., Armonk, NY). Categorical variables were presented as counts and percentages, compared with chi-square or Monte Carlo tests. Continuous variables were expressed as mean ± SD or median (IQR), depending on distribution. Between-group comparisons were made using independent sample t-test or Mann–Whitney U test. Paired sample t-test assessed within-group changes. ROC curves were applied to determine cut-off values for predictors. Binary logistic regression was used to identify independent risk factors for weaning failure. A p-value <0.05 was considered statistically significant.

## RESULTS

Among 42 children (median age 12 months), males were 52.4%. Respiratory distress grade 3 (40.5%) and pneumonia with respiratory failure (38.1%) were main causes of admission, while septic shock (40.5%) was the leading cause of ventilation. The median duration of mechanical ventilation was 4 days (IQR 3–6), PICU stay 7 days (IQR 6–9), with 73.8% achieving successful weaning compared to 26.1% failure **(Table 1).**

**Table 2** demonstrates that BNP I levels at initiation of the spontaneous breathing trial (SBT) ranged from 651.67 to 1049.67 pg/mL, with a mean ± SD of 858.11 ± 86.91 pg/mL. By the end of SBT, BNP II levels showed a non-significant decrease to a mean of 816.6 ± 153.56 pg/mL ($p = 0.059$). The median percent decrease in BNP was 4.43%, indicating a slight overall decline during the SBT period. Weaning success was achieved in 73.8% of patients compared to 26.2% who failed weaning. No statistically significant associations were observed between weaning outcome and demographic or physiological parameters, including age ($p = 0.439$), respiratory rate ($p = 0.756$), Glasgow Coma Scale (GCS) ($p = 0.614$), arterial blood gas values, RSBI ($p = 0.383$), or hematological and metabolic indices. In contrast, a significant relationship was detected between C-reactive protein (CRP) positivity and weaning outcome: the failure group showed 81.8% CRP positivity compared to 38.7% in the success group ($p = 0.014$). These findings suggest that

inflammatory activity, rather than demographic or mechanical factors, may be more closely linked to weaning failure among the studied pediatric patients (Table 2).

**Table 3** shows that BNP I levels were significantly higher in the failure group (954.3 ± 44.57 pg/mL) compared to the success group (823.98 ± 71.17 pg/mL, $p < 0.001$). Similarly, BNP II levels remained higher in failed weaning (939.61 ± 42.05 pg/mL) versus successful weaning (772.96 ± 155.31 pg/mL, $p < 0.001$). Within-group comparison revealed no significant reduction between BNP I and BNP II in either success ($p = 0.082$) or failure ($p = 0.413$) groups (Table 3).

Higher BNP I significantly independently increased risk of mortality by 1.059 folds while high BNP II non-significantly independently increased risk of mortality by 1.031 folds. BNP I showed a positive correlation with PICU stay duration ($r = 0.515$, $p < 0.001$). BNP II demonstrated significant positive correlations with respiratory rate ($r = 0.329$, $p = 0.033$), pH ($r = 0.319$, $p = 0.033$), PICU stay duration ($r = 0.491$, $p < 0.001$), and BNP I significant ($r = 0.445$, $p = 0.003$), while it had a significant negative correlation with GCS ($r = –0.466$, $p = 0.002$) **(Table 4).**

Among factors significantly correlated to BNP II, only GCS (unstandardized β=-64.235, p=0.006) and BNP I (unstandardized β=0.635, p=0.01) significantly independently associated with it **(Table 5).**

**Table 6** and Supplementary Figure 1 demonstrate strong predictive performance of BNP for failed weaning. BNP I at a cutoff ≥892.667 pg/mL achieved an AUC of 0.962, with sensitivity 90.9%, specificity 87.1%, PPV 71.4%, NPV 96.4%, and accuracy 88.1% ($p < 0.001$). BNP II at a cutoff ≥905.167 pg/mL showed AUC 0.921, sensitivity 81.8%, specificity 87.1%, PPV 69.2%, NPV 93.1%, and accuracy 85.7% ($p < 0.001$).

The percent decrease in BNP correlated negatively with respiratory rate ($r = –0.434$, $p = 0.004$), pH ($r = –0.600$, $p < 0.001$), $PCO_2$ ($r = –0.378$, $p = 0.014$), and RSBI ($r = –0.382$, $p = 0.013$), and positively with GCS ($r = 0.489$, $p = 0.001$). For reintubation, BNP I was significantly lower in non-survivors ($p = 0.019$), while BNP II showed no difference. Survivors had a significant BNP reduction ($p = 0.009$) with a greater percent decrease (6.13% vs. –0.39%, $p < 0.001$) **(Table 7).**

**Table1 :** Demographic, Laboratory, and Clinical Characteristics of the Studied Patients (N = 42)

| Variable | N | % |
|---|---|---|
| **Gender: Female** | **20** | **47.6%** |
| **Gender: Male** | **22** | **52.4%** |
| **Clinical Characteristics** | | |
| **Cause of Admission** | | |
| **DCL** | **3** | **7.1%** |
| **Pneumonia with Respiratory Failure** | **16** | **38.1%** |
| **Respiratory distress grade 3** | **17** | **40.5%** |
| **Respiratory distress grade 4** | **6** | **14.3%** |
| **Cause of Ventilation** | | |
| **Apnea** | **2** | **4.8%** |
| **Neuromuscular dysfunction** | **1** | **2.3%** |
| **Pneumonia with Respiratory Failure** | **16** | **38.1%** |
| **Septic shock** | **17** | **40.5%** |
| **Cardiac arrest** | **6** | **14.3%** |
| **CRP: Negative** | **21** | **50 %** |

| Variable | N | % |
|---|---|---|
| CRP: Positive | 21 | 50 % |
| Parenteral Nutrition | 35 | 83.3% |
| TPN | 7 | 16.7% |
| Weaning Success | 31 | 73.8% |
| Weaning Failure | 11 | 26.1% |
| | Mean ± SD / Median (IQR) | Range |
| Age (months) | 12 (5.75 – 55.5) | 1.5 – 180 |
| Sodium (mg/dl) | 141.55 ± 4.27 | 135 – 149 |
| Potassium (mg/dl) | 4.03 ± 0.46 | 3.1 – 4.8 |
| Hemoglobin (g/dl) | 12.92 ± 1.91 | 7.5 – 15.3 |
| WBCs ($10^3/mm^3$) | 13.29 ± 3.19 | 7.3 – 20 |
| Platelet count ($10^3/mm^3$) | 362.05 ± 91.72 | 187 – 500 |
| RBG (mg/dl) | 90.69 ± 12.32 | 70 – 113 |
| PRISM III Score | 11 (6 – 14.25) | 2 – 18 |
| Duration of MV (days) | 4 (3 – 6) | 2 – 10 |
| LOS in PICU (days) | 7 (6 – 9) | 4 – 15 |

IQR: Interquartile range, SD: Standard deviation, WBCs: White blood cells, RBG: Random blood glucose, CRP: C-reactive protein, DCL: Disturbance of conscious level, RF: Respiratory failure, PRISM III: Pediatric Risk of Mortality Score III, MV: Mechanical ventilation, LOS: Length of stay, PICU: Pediatric intensive care unit, TPN: Total parenteral nutrition.

**Table 2:** BNP Levels in Studied Patients (N = 42)

| Variable | Category / Subgroup | Test | p-value |
|---|---|---|---|
| BNP I (Initiation of SBT) | 858.11 ± 86.91 (range 651.67 – 1049.67) | 0.059 | (paired t test) |
| BNP II (End of SBT) | 816.6 ± 153.56 (range=71.7 – 1005.33) | | |
| % Decrease in BNP | 4.43 (-1.02 – 7.53%) (range=-40.2 – 91.4%) | | |

BNP: Brain natriuretic peptide, SBT: Spontaneous breathing trial, *p < 0.05 considered statistically significant.

**Table 3:** Relation Between Outcome of Weaning and BNP I & BNP II Levels in Studied Patients (N = 42)

| Variable | Succeed Weaning (n=31) | Failed Weaning (n=11) | Test | p-value |
|---|---|---|---|---|
| BNP I (pg/mL) | 823.98 ± 71.17 | 954.3 ± 44.57 | t = -5.566 | <0.001** |
| BNP II (pg/mL) | 772.96 ± 155.31 | 939.61 ± 42.05 | t = -3.488 | <0.001** |
| Paired-sample p-value (BNP I vs II) | 0.082 | 0.413 | | |
| % Decrease in BNP | 4.28 (-0.49 – 9.02) | 4.58 (-4.36 – 5.27) | Z = -0.873 | 0.383 |

BNP I: Brain natriuretic peptide at initiation of spontaneous breathing trial (SBT), BNP II: Brain natriuretic peptide at end of SBT, SD: Standard deviation, IQR: Interquartile range. Statistical tests: t = Independent sample t test, Z = Mann-Whitney test, § = Paired sample t test. p < 0.05 considered statistically significant; p < 0.001 highly significant.

**Table 4**: Multivariate Analysis and Correlation of BNP with Clinical and Laboratory Parameters in Studied Patients (N = 42)

| Variable | β / r | p-value | AOR/ 95% CI (Lower–Upper) |
|---|---|---|---|
| **BNP I** | **β = 0.058** | **0.029*** | **1.059 (1.006 – 1.115)** |
| **BNP II** | **β = 0.031** | **0.069** | **1.031 (0.998 – 1.066)** |
| **Age** | **r = 0.016§** | **0.921** | |
| **Respiratory rate** | **r = -0.116** | **0.464** | |
| **GCS** | **r = -0.221** | **0.159** | |
| **pH** | **r = -0.059** | **0.713** | |
| **PCO2** | **r = -0.284** | **0.068** | |
| **HCO3** | **r = -0.260** | **0.096** | |
| **PO2** | **r = 0.101** | **0.523** | |
| **Sodium (mg/dl)** | **r = 0.077** | **0.626** | |
| **Potassium (mg/dl)** | **r = -0.002** | **0.991** | |
| **Hemoglobin (g/dl)** | **r = 0.016** | **0.920** | |
| **WBCs ($10^3$/$mm^3$)** | **r = -0.059** | **0.711** | |
| **Platelets ($10^3$/$mm^3$)** | **r = -0.065** | **0.684** | |
| **Random blood sugar (mg/dl)** | **r = -0.114** | **0.470** | |
| **LOS of PICU stay (days)** | **r = 0.515§** | **<0.001**** | |
| **Tidal volume (L)** | **r = -0.051§** | **0.747** | |
| **RSBI** | **r = -0.043§** | **0.789** | |
| **Age** | **r = -0.184§** | **0.244** | |
| **Respiratory rate** | **r = 0.329** | **0.033*** | |
| **GCS** | **r = -0.466** | **0.002*** | |
| **pH** | **r = 0.319** | **0.033*** | |
| **PCO2** | **r = 0.124** | **0.434** | |
| **HCO3** | **r = -0.247** | **0.114** | |
| **PO2** | **r = -0.268** | **0.087** | |
| **Sodium (mg/dl)** | **r = 0.033** | **0.837** | |
| **Potassium (mg/dl)** | **r = -0.138** | **0.384** | |
| **Hemoglobin (g/dl)** | **r = -0.224** | **0.154** | |
| **WBCs ($10^3$/$mm^3$)** | **r = -0.020** | **0.900** | |
| **Platelets ($10^3$/$mm^3$)** | **r = -0.009** | **0.959** | |
| **Random blood sugar (mg/dl)** | **r = -0.017** | **0.913** | |
| **Duration of PICU stay (days)** | **r = 0.491§** | **<0.001**** | |
| **Tidal volume (L)** | **r = -0.260§** | **0.097** | |
| **RSBI** | **r = 0.249§** | **0.112** | |
| **BNP I** | **r = 0.445** | **0.003*** | |

BNP I: Brain natriuretic peptide at initiation of SBT, BNP II: Brain natriuretic peptide at end of SBT, GCS: Glasgow Coma Scale, RSBI: Rapid shallow breathing index, TV: Tidal volume, LOS: Length of stay, PICU: Pediatric intensive care unit, WBCs: White blood cells, AOR: Adjusted odds ratio, CI: Confidence interval. Statistical tests: β = Logistic regression coefficient, r = Pearson correlation coefficient, § = Spearman rank correlation coefficient. *$p < 0.05$ statistically significant; **$p \leq 0.001$ highly significant.

**Table 5:** Linear stepwise regression analysis of factors associated with BNP by end of study

| | Unstandardized Coefficients | | Standardized Coefficients | t | p | 95.0% Confidence Interval | |
|---|---|---|---|---|---|---|---|
| | β | Std. Error | Beta | | | Lower | Upper |
| (Constant) | 1092.617 | 383.010 | | 2.853 | 0.007* | 317.907 | 1867.327 |
| GCS | -64.235 | 22.138 | -0.387 | -2.902 | 0.006* | -109.012 | -19.457 |
| BNP II | 0.635 | 0.236 | 0.360 | 2.697 | 0.010* | 0.159 | 1.112 |

*p<0.05 is statistically significant GCS: Glasgow Coma Scale

**Table 6**: Predictive Performance of BNP I and BNP II in Failed Weaning Among Studied Patients (N = 42)

| BNP Marker | Cutoff (pg/mL) | AUC | Sensitivity | Specificity | PPV | NPV | Accuracy | p-value |
|---|---|---|---|---|---|---|---|---|
| BNP I | ≥892.667 | 0.962 | 90.9% | 87.1% | 71.4% | 96.4% | 88.1% | <0.001** |
| BNP II | ≥905.167 | 0.921 | 81.8% | 87.1% | 69.2% | 93.1% | 85.7% | <0.001** |

BNP I: Brain natriuretic peptide at initiation of SBT, BNP II: Brain natriuretic peptide at end of SBT, AUC: Area under the curve, PPV: Positive predictive value, NPV: Negative predictive value. **p ≤ 0.001 statistically highly significant.

**Table 7:** Correlation of Percent Decrease in BNP with Clinical-Laboratory Parameters and Relation Between Reintubation and BNP I & BNP II (N = 42)

| Parameter / Variable | Statistic | Value (r / Mean ± SD / Median [IQR]) | p-value | Test |
|---|---|---|---|---|
| **Correlation of Percent Decrease in BNP with Clinical-Laboratory Parameters** | | | | |
| Age | r | 0.247 | 0.115 | Spearman |
| Respiratory rate | r | -0.434 | 0.004* | Spearman |
| GCS | r | 0.489 | 0.001** | Spearman |
| pH | r | -0.600 | <0.001** | Spearman |
| PCO2 | r | -0.378 | 0.014* | Spearman |
| HCO3 | r | -0.039 | 0.804 | Spearman |
| PO2 | r | -0.268 | 0.087 | Spearman |
| Sodium (mg/dl) | r | 0.071 | 0.654 | Spearman |
| Potassium (mg/dl) | r | 0.010 | 0.951 | Spearman |
| Hemoglobin (g/dl) | r | 0.265 | 0.090 | Spearman |
| WBCs ($10^3$/$mm^3$) | r | -0.106 | 0.504 | Spearman |
| Platelets ($10^3$/$mm^3$) | r | 0.001 | 0.998 | Spearman |
| Random blood sugar (mg/dl) | r | -0.201 | 0.201 | Spearman |
| LOS of PICU stay (days) | r | -0.212 | 0.177 | Spearman |
| Tidal volume (L) | r | 0.282 | 0.071 | Spearman |
| RSBI | r | -0.382 | 0.013* | Spearman |
| **Relation Between Reintubation and BNP I & BNP II** | | | | |
| BNP I (pg/mL) | Mean ± SD | Survivors: 883.63 ± 81.13 Non-survivors: 820.59 ± 83.48 | 0.019* | t-test |
| BNP II (pg/mL) | Mean ± SD | Survivors: 797.41 ± 180.58 Non-survivors: 844.82 ± 100.44 | 0.332 | t-test |
| Paired BNP I vs II | §p | Survivors: 0.009* Non-survivors: 0.238 | | Paired t-test |
| % Decrease in BNP | Median (IQR) | Survivors: 6.13 (3.45 – 12.02) Non-survivors: -0.39 (-8.95 – 3.72) | <0.001** | Mann-Whitney Z=-3.447 |

BNP I: Brain natriuretic peptide at initiation of SBT, BNP II: Brain natriuretic peptide at end of SBT, GCS: Glasgow Coma Scale, RSBI: Rapid shallow breathing index, TV: Tidal volume, LOS: Length of stay, PICU: Pediatric intensive care unit, WBCs: White blood cells, SD: Standard deviation, IQR: Interquartile range. Statistical tests: t = Independent sample t test, Z = Mann-Whitney test, r = Spearman rank correlation coefficient, § = Paired sample t test. *p < 0.05 statistically significant; **p ≤ 0.001 highly significant.

## DISCUSSION

B-type natriuretic peptide (BNP) and its inactive fragment N-terminal proBNP (NT-proBNP) are important cardiac biomarkers secreted in response to ventricular wall stretch and myocardial stress. With half-lives of approximately 20 and 120 minutes respectively, they may reflect cardiovascular changes during weaning, including subclinical congestion and increased ventricular afterload [9].

In our study, 42 patients were enrolled with a median age of 12 months (IQR: 5.75–55.5 months), and males constituted 52.4% of the cohort. Similar demographic findings were reported by Liu et al. **[13]**, who observed that younger age and male sex were associated with prolonged mechanical ventilation in a large pediatric cohort. Pisitcholakarn et al. **[14]** also identified younger age and male gender as predictors of prolonged ventilation in PICU settings. Likewise, Egbuta and Easley **[15]** highlighted that mechanical ventilation is required in up to 63% of critically ill pediatric admissions, with younger children being more vulnerable.

Our laboratory findings showed hemoglobin levels averaging 12.9 ± 1.9 g/dL, sodium levels of 141.6 ± 4.3 mEq/L, and equal distribution of positive and negative CRP results (50% each). These findings are consistent with Santschi et al. **[16]**, who observed similar baseline hemoglobin and electrolyte levels in ventilated children. Yehya et al. **[17]** also reported stable laboratory trends in children considered ready for weaning trials, supporting their utility in extubation readiness assessment. In addition, Konuksever et al. **[18]** demonstrated strong interchangeability of hemoglobin and electrolyte measurements in critically ill children, reinforcing the reliability of these values. Sood et al. **[19]** further emphasized the importance of close laboratory monitoring, noting that more than 20% of PICU patients require invasive mechanical ventilation.

In our study, the most frequent causes of PICU admission were respiratory distress grade 3 (40.5%) and pneumonia with respiratory failure (38.1%). The median duration of mechanical ventilation was 4 days (IQR: 3–6 days), with a weaning success rate of 73.8%. These findings were not in agreement with Liu et al. **[9]**, who reported that respiratory disorders account for the majority of indications for ventilation, with success rates typically between 70–80%. Similarly, Hysinger and Ahlfeld **[20]** confirmed that respiratory illnesses remain the leading cause of mechanical ventilation in pediatric populations, particularly among neonates and infants. The Pediatric Acute Lung Injury and Sepsis Investigators (PALISI) Network **[21]** also identified pneumonia and acute respiratory failure as the predominant causes of mechanical ventilation in children.

Our study demonstrated that BNP levels decreased non-significantly from the initiation to the end of SBT (858.1 ± 86.9 vs 816.6 ± 153.6 pg/mL, p=0.059), with a median reduction of 4.4%. Zaky et al. **[22]** reported a similar pattern, where NT-proBNP levels remained largely unchanged during SBT in successfully weaned patients. Akella et al. **[23]** also emphasized that cardiovascular stability during weaning, reflected by stable natriuretic peptide levels, may indicate adequate adaptation to spontaneous breathing. In contrast, Zheng et al. **[24]** observed greater BNP fluctuations during weaning in postsurgical patients, with changes up to 23.3%. This discrepancy may reflect differences in patient populations, as postsurgical cohorts often exhibit more dynamic cardiovascular responses compared with medical PICU patients.

We observed a weaning success rate of 73.8% and failure rate of 26.2%. Abu-Sultaneh et al. **[25]** reported lower failure rates of 4.6–5.8% in a large multicenter database, while Poletto et al. **[26]** found that extubation failure occurs in up to 20% of pediatric cases, similar to our results. Alsolami et al. **[27]** reported slightly lower rates (5–15%), suggesting that variability across studies may reflect differences in PICU practices and patient case mix.

Our findings showed no significant association between weaning outcome and either gender (p=0.592) or age (p=0.439). In agreement, Abu-Sultaneh et al. **[25]** noted that while younger age (<6 months) was associated with higher extubation failure, gender was not predictive. Saengsin et al. **[28]** also showed that demographic variables had limited predictive value for extubation outcomes in pediatric cardiac patients. These findings suggest that physiological and disease-related factors are more important determinants of weaning success than demographic characteristics.

We found no significant relationship between weaning outcome and conventional clinical predictors such as respiratory rate (p=0.756), GCS (p=0.614), arterial blood gas parameters, or RSBI (p=0.383). Egbuta and Easley **[15]** similarly reported that traditional indices alone have limited predictive value in pediatric populations. Poletto et al. **[26]** also emphasized the lack of a single reliable clinical predictor, while Menguy et al. **[29]** demonstrated that conventional weaning parameters lack sufficient sensitivity and specificity, supporting the need for novel biomarkers such as BNP.

Our results showed that CRP status was significantly associated with proper weaning outcomes (p=0.014), with positive CRP more common among failed cases (81.8% vs 38.7%). Other laboratory values were not predictive. Akella et al. **[23]** highlighted that systemic inflammation contributes to cardiovascular instability during weaning attempts, which may explain our findings. In contrast, Liu et al. **[9]** found in their review that most routine laboratory parameters, including inflammatory markers, were not consistent predictors. The difference may be related to variation in patient populations and thresholds used for inflammatory markers.

We found significant associations between failed weaning and both longer PICU stay (median 10 vs 7 days, p<0.001) and cause of admission (p=0.048). Similarly, Abu-Sultaneh et al. **[25]** observed that extubation failure was linked with longer mechanical ventilation, PICU, and hospital stays. Alsolami et al. **[27]** reported prolonged ICU stays (25.5 days) in failed extubation cases, while Saengsin et al. **[28]** found similar patterns in pediatric cardiac ICUs, though mortality was not affected. These findings reinforce that extubation failure not only prolongs critical care but also worsens resource utilization.

Our study demonstrated that both BNP I and BNP II were significantly associated with proper weaning outcomes, with higher levels observed in failed cases (BNP I: 954.3 ± 44.6 vs 824.0 ± 71.2 pg/mL, p<0.001; BNP II: p<0.001). Similarly, Zaky et al. **[22]** reported significantly higher NT-proBNP levels in failed weaning patients compared with successful cases. Liu et al. **[9]**, in their systematic review, also confirmed that elevated BNP levels were consistently linked to weaning failure, with strong pooled predictive estimates. Zheng et al. **[24]** found that baseline NT-proBNP levels were higher in failed weaning cases in postsurgical patients. In contrast, some individual studies included in systematic reviews showed weaker associations, possibly due to differences in assay type, cutoff thresholds, or patient heterogeneity.

We also found that higher BNP I significantly and independently increased the risk of weaning failure by 1.059-fold (β=0.058, p=0.029), whereas BNP II showed only a non-significant trend (p=0.069). Liu et al. **[9]** similarly reported that BNP levels were independent predictors of weaning failure in multivariate analyses across several studies. Zaky et al. **[22]** confirmed NT-proBNP as a strong independent predictor with excellent discriminative performance. Zheng et al. **[24]** demonstrated that BNP percentage change was the only independent predictor in their cohort, while Menguy et al. **[29]** showed that natriuretic peptides remained important features within artificial intelligence models, although multiple physiological variables contributed to prediction.

Our study also found a significant positive correlation between BNP I and PICU length of

stay (r=0.515, p<0.001), while no significant correlations were noted with other parameters. This finding aligns with Liu et al. **[9]**, who observed consistent correlations between BNP levels and length of stay across multiple studies. Akella et al**. [23]** explained that elevated natriuretic peptides reflect cardiovascular dysfunction that prolongs mechanical ventilation and critical care stay. Yu et al. **[30]** also demonstrated in pediatric septic patients that higher BNP levels were associated with longer PICU stay and worse outcomes. Similarly, Wang et al. **[31]** found that elevated NT-proBNP levels in a large pediatric cohort were associated with increased mortality and prolonged PICU admission. In contrast, Zaky et al. **[22]** observed additional correlations between NT-proBNP and respiratory parameters in adults, suggesting that biomarker–outcome relationships may differ between pediatric and adult populations due to differing pathophysiology.

Our study demonstrated that BNP II correlated significantly with several parameters, including respiratory rate (r=0.329, p=0.033), GCS (r=-0.466, p=0.002), pH (r=0.319, p=0.033), duration of PICU stay (r=0.491, p<0.001), and BNP I (r=0.445, p=0.003). Zaky et al. **[22]** reported similar associations between NT-proBNP and clinical variables such as respiratory and acid–base status. Liu et al. **[9]** also noted that end-trial BNP values correlated more strongly with physiological parameters than baseline values across multiple studies. Trudzinski et al. **[32]** emphasized the multifactorial pathophysiology of weaning failure, supporting our observation that BNP integrates multiple physiological signals. In contrast, some studies found weaker correlations with end-trial BNP, likely due to variability in patient populations and acute fluctuations during weaning trials.

Among these correlated factors, only GCS (β=-64.2, p=0.006) and BNP I (β=0.635, p=0.010) were independent predictors of BNP II. This supports the concept that baseline cardiac function, reflected by initial BNP, drives subsequent biomarker responses during weaning. Liu et al. **[9]** found similar associations between baseline and end-trial BNP values, while Akella et al. **[23]** emphasized the role of neurological status in cardiovascular adaptation. In contrast, Zheng et al. **[24]** reported additional independent predictors, including respiratory parameters, which may reflect differences in patient populations and regression models.

We found that BNP I ≥892.7 pg/mL predicted weaning failure with excellent accuracy (AUC 0.962, sensitivity 90.9%, specificity 87.1%, PPV 71.4%, NPV 96.4%). Liu et al. **[9]** reported comparable pooled diagnostic accuracy in their meta-analysis (sensitivity 89%, specificity 82%). Zaky et al. **[22]** also showed strong discrimination (AUC 0.89), while Zheng et al. **[24]** highlighted percentage change in BNP as a predictor (AUC 0.744). In contrast, Menguy et al. **[29]** found that artificial intelligence models integrating multiple physiological variables outperformed individual biomarkers, though BNP remained a key predictor.

BNP II ≥905.2 pg/mL also predicted weaning failure with high accuracy (AUC 0.921, sensitivity 81.8%, specificity 87.1%, PPV 69.2%, NPV 93.1%). These results align with Liu et al. [9], who found high specificity for end-trial BNP across multiple studies, as Zaky et al. **[22]**, who reported similar performance for NT-proBNP. In contrast, Zheng et al. **[24]** observed that baseline BNP occasionally outperformed end-trial levels in postsurgical patients, suggesting that optimal timing of measurement may vary by patient group.

BNP percentage decrease was significantly correlated with respiratory rate (r=-0.434, p=0.004), pH (r=-0.600, p<0.001), and GCS (r=0.489, p=0.001). Liu et al. **[9]** confirmed that greater BNP reductions are associated with successful cardiovascular adaptation. Zheng et al. **[24]** also found that percentage change was a stronger predictor than absolute levels. In contrast, some studies reported weaker associations, which may reflect differences in

adaptation patterns between pediatric and adult populations.

Finally, we observed that BNP I (p=0.019) and BNP 2 percentage decrease (p<0.001) were significantly related to survival, with survivors showing greater BNP decline (6.1% vs -0.4%). Liu et al. **[9]** also reported that declining BNP was linked to successful weaning and better outcomes. Zaky et al. **[22]** found that survivors had lower BNP levels and greater decreases during successful extubation. Dadam et al. **[33]** highlighted that reintubation within 48 hours significantly increased mortality, underscoring the importance of accurate weaning prediction. In contrast, some reviews reported weaker mortality associations, possibly due to small sample sizes or heterogeneous patient populations **[33].**

A key strength of this study is its prospective design and the standardized assessment of BNP at both the initiation and completion of spontaneous breathing trials, allowing for consistent evaluation of biomarker dynamics during weaning. Another strength is the inclusion of comprehensive clinical, laboratory, and outcome measures, which provided a robust dataset for correlating BNP levels with weaning outcomes in critically ill children.

In a prior ZUMJ publication, El-Beheidy et al. **[34]** studied 53 pediatric patients undergoing a spontaneous breathing trial and found that the rapid shallow breathing index (RSBI) was significantly elevated in those who failed weaning, with a cutoff ≥3.5 breaths/min·ml/kg yielding 100% sensitivity and 75% specificity for predicting failure. That study emphasized that classical clinical weaning parameters, although useful, may not provide sufficient accuracy alone in pediatric settings. Our results extend and refine this local evidence by demonstrating that baseline BNP levels, a biomarker of cardiovascular stress, outperform conventional indices (including RSBI) in discriminating weaning success versus failure in a comparable PICU population.

This study has some limitations that should be acknowledged. First, the relatively small sample size (n=42) and single-center design restrict the generalizability of the findings and may not fully represent broader pediatric intensive care populations. Second, the absence of long-term follow-up limited evaluation of the prognostic value of BNP beyond the acute phase, particularly regarding post-discharge morbidity, late complications, and overall survival.

## CONCLUSION

BNP could be an independent and accurate predictor of weaning outcomes in critically ill children. Elevated baseline BNP was strongly associated with weaning failure, longer PICU stay, and higher mortality. BNP thresholds of ≥892.7 pg/mL (BNP I) and ≥905.2 pg/mL (BNP II) demonstrated excellent diagnostic performance for predicting extubation failure. Conventional indices such as respiratory rate, RSBI, and ABG values had limited predictive value, while CRP positivity correlated with failed weaning. Overall, BNP provided superior prognostic utility, supporting its role as a reliable biomarker to guide safe ventilator weaning in pediatric intensive care.

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**Data availability**: The data, or files generated as well as the analyzed during the present study are available from the corresponding author upon request.

**Author contribution**: N.A.K.A. and S.Z. were responsible for the conceptualization of the study and overall supervision of the research process. E.E.H.E. contributed to the acquisition of clinical data, patient follow-up, and drafting of the initial manuscript. A.M.A. handled the laboratory investigations, data analysis, and interpretation of results. All authors participated actively in reviewing the final draft, provided critical intellectual input, and approved the manuscript in its current form.

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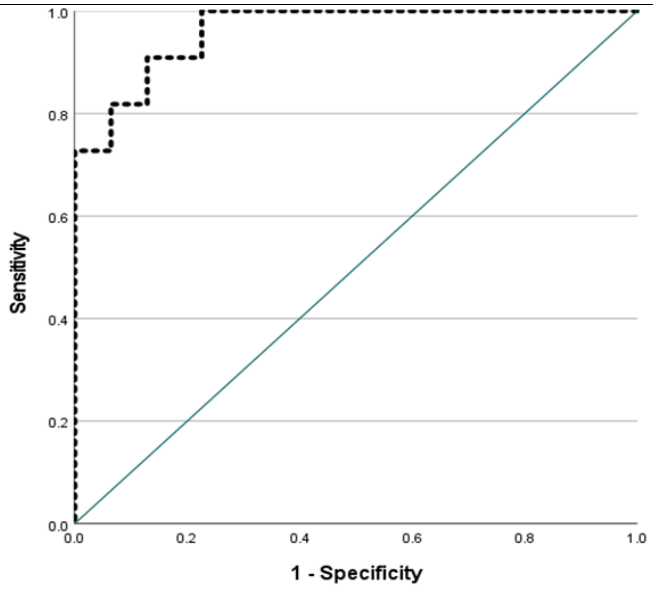
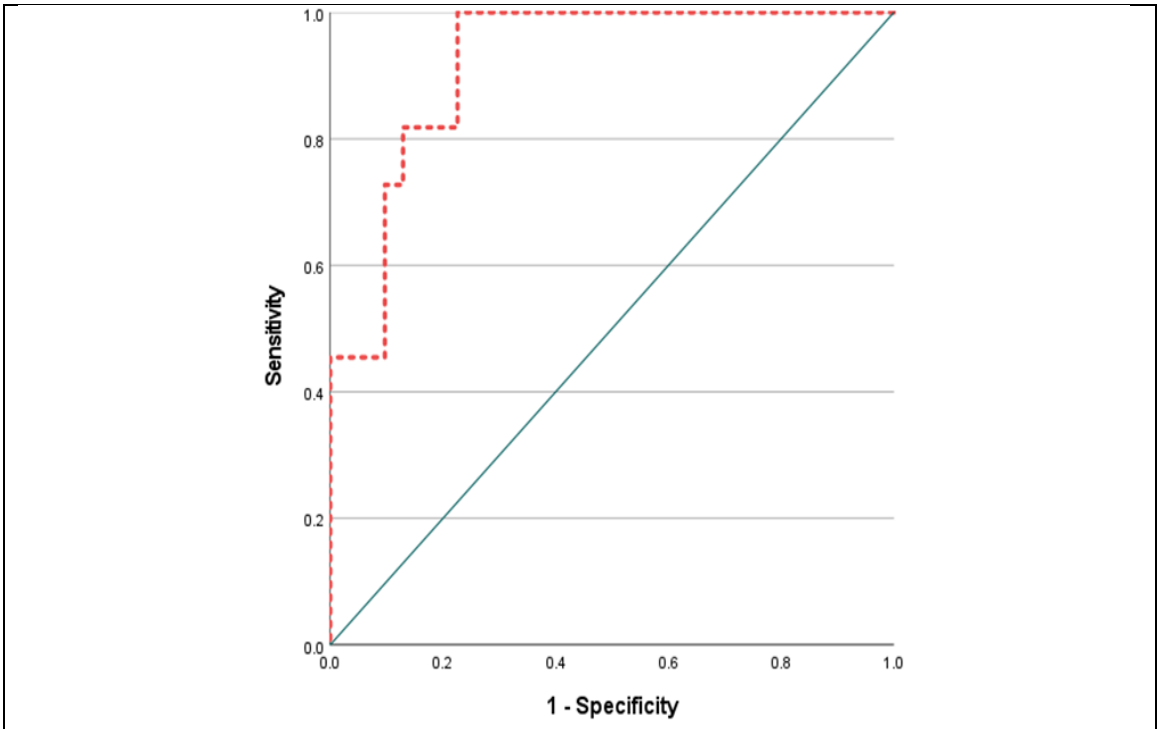



(A)




(B)

**Figure (S1):** (A): ROC curve showing performance of BNP I in prediction of mortality among the studied patients, (B): ROC curve showing performance of BNP I in prediction of failed weaning among the studied patients

**Table (S1):** Weaning Outcomes, and Relation to Clinical and Laboratory Parameters in Studied Patients(N= 42)

| Variable | Succeed Weaning (n=31) / Mean ± SD | Failed Weaning (n=11) /Mean ± SD | Test | p-value |
|---|---|---|---|---|
| **Outcome of Weaning** | **31 (73.8%)** | **11 (26.2%)** | | |
| **Relation Between Outcome and Demographics** | | | | |
| **Gender: Female** | **14 (45.2%)** | **6 (54.5%)** | | |
| **Gender: Male** | **17 (54.8%)** | **5 (45.5%)** | **$\chi^2$=0.287** | **0.592** |
| **Age (months)** | **11 (5 – 48)** | **18 (6 – 108)** | **Z=-0.773** | **0.439** |
| **Relation Between Weaning and Clinical Parameters** | | | | |
| **Respiratory rate** | **41.42 ± 13.9** | **39.91 ± 13.3** | **t=0.313** | **0.756** |
| **GCS** | **12.84 ± 0.82** | **12.64 ± 1.21** | **t=-0.516** | **0.614** |
| **pH** | **7.37 ± 0.03** | **7.38 ± 0.04** | **t=-0.496** | **0.622** |
| **PCO2 (mmHg)** | **41.29 ± 4.58** | **40.09 ± 4.59** | **t=0.746** | **0.460** |
| **HCO3 (mmol/L)** | **23.81 ± 1.33** | **23.73 ± 1.85** | **t=0.131** | **0.898** |
| **PO2 (mmHg)** | **89.23 ± 4.93** | **87.91 ± 5.05** | **t=0.757** | **0.453** |
| **Tidal volume (L)** | **0.063 (0.035 – 0.126)** | **0.08 (0.042 – 0.133)** | **Z=-0.301** | **0.764** |
| **RSBI** | **651 (164.2 – 1342)** | **420 (190.4 – 979.5)** | **Z=-0.873** | **0.383** |
| **Relation Between Weaning and Laboratory Parameters** | | | | |
| **Sodium (mg/dl)** | **141.29 ± 4.22** | **142.27 ± 4.52** | **t=-0.651** | **0.518** |
| **Potassium (mg/dl)** | **4.02 ± 0.49** | **4.06 ± 0.38** | **t=-0.271** | **0.788** |
| **Hemoglobin (g/dl)** | **12.98 ± 1.7** | **12.75 ± 2.5** | **t=0.352** | **0.727** |

| Variable | Succeed Weaning (n=31) / Mean ± SD | Failed Weaning (n=11) /Mean ± SD | Test | p-value |
|---|---|---|---|---|
| WBCs ($10^3/mm^3$) | 13.32 ± 3.06 | 13.23 ± 3.69 | t=0.079 | 0.938 |
| Platelets ($10^3/mm^3$) | 357.71 ± 91.43 | 374.27 ± 95.87 | t=-0.510 | 0.613 |
| Random blood sugar (mg/dl) | 91.9 ± 12.44 | 87.27 ± 11.85 | t=1.073 | 0.290 |
| CRP Negative | 19 (61.3%) | 2 (18.2%) | | |
| CRP Positive | 12 (38.7%) | 9 (81.8%) | $\chi^2$=6.035 | 0.014* |

GCS: Glasgow Coma Scale, RSBI: Rapid shallow breathing index, TV: Tidal volume, WBCs: White blood cells, CRP: C-reactive protein, SD: Standard deviation, IQR: Interquartile range. Statistical tests: $\chi^2$: Chi-square test, t: Independent sample t test, Z: Mann-Whitney test, *p < 0.05 considered statistically significant.

## citation

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