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**Original Research Article**

# Association of serum procalcitonin level with acute respiratory distress syndrome in patients following cardiopulmonary bypass

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## ABSTRACT

**Background:** Acute respiratory distress syndrome is a potentially life-threatening complication after cardiac surgery. Systemic inflammatory response is activated during cardiopulmonary bypass time which may cause acute respiratory distress syndrome and serum procalcitonin level also increases during systemic inflammatory response. This study aimed to see the association of serum procalcitonin level with acute respiratory distress syndrome in patients following cardiopulmonary bypass.
**Methods:** This was a prospective observational study conducted in the Department of Cardiac Surgery, National Heart Foundation Hospital and Research Institute, Dhaka, Bangladesh during the period from September, 2020 to August, 2022. In our study, we included 140 patients who underwent cardiac surgery with cardiopulmonary bypass. Patients were divided into two groups- group A included 70 patients with serum procalcitonin levels <7 ng/ml and group B included 70 patients with serum procalcitonin levels>7 ng/ml.
**Results:** The mean (±SD) age of the patients was 43.20 (±13.18) years and 46.61 (±13.75) years in group A and group B respectively. In group A, 41.4% of patients were male and 58.6% were female; in group B, 52.9% were male and 47.1% were female. On 1st postoperative day, in group, A serum procalcitonin levels were 1.36(±0.97) ng/ml and in group B serum procalcitonin levels were 27.09(±26.11) ng/ml ($p<0.001$). The incidence of ARDS was significantly higher in group B than in group A (35.7% versus 8.5%, $p= 0.002$).
**Conclusion:** This study concluded that there was an association of serum procalcitonin level with acute respiratory distress syndrome in patients following cardiopulmonary bypass.

**Keywords:** Serum procalcitonin level, Acute respiratory distress syndrome, Association, Cardiopulmonary bypass

## INTRODUCTION

Cardiac surgery with cardiopulmonary bypass is considered a highly sterile type of surgery but it could lead to a systemic inflammatory response syndrome.[1] This systemic inflammatory response syndrome is a result of several stimuli, such as exposure of blood to non-physiological surfaces, surgical trauma, and myocardial ischemia-reperfusion due to aortic clamping and endotoxin release which may lead to acute respiratory distress syndrome (ARDS).[2] Independent predictors of post-operative ARDS are – sepsis, high-risk

aortic vascular surgery, high-risk cardiac surgery, emergency surgery, cirrhosis, increased respiratory rate, a fraction of inspired oxygen (Fio2)> 35 %, and oxygen saturation < 95%.[3]

Acute respiratory distress syndrome is a life-threatening pulmonary condition that can lead to rapidly progressive acute hypoxemic respiratory failure and bilateral pulmonary infiltrates.[4] The occurrence of acute respiratory distress syndrome after cardiac surgery is also unpredictable and little is known about risk factors for the development of this complication.[5]

Acute respiratory distress syndrome is currently defined as hypoxemia that occurs within one week of a known clinical insult or new, worsening respiratory symptoms. This syndrome is characterized by bilateral opacities on chest imaging that are not fully explained by effusions, lobar/lung collapse, or nodules, and are not fully explained by cardiac failure or fluid.[6] The terms "acute lung injury" (ALI), which was previously used in the AECC (American European Consensus Conference) definition, has been eliminated. There are now only three mutually exclusive subgroups of ARDS that are described - a. Mild ARDS:200 mmHg<PaO2/FiO2 ≤300 mmHg with positive end- expiratory pressure (PEEP) or continuous positive airway pressure ≥5 cmH2O; b. Moderate ARDS: 100 mmHg <PaO2/FiO2 ≤200 mmHg with PEEP ≥5 cmH2O; c. Severe ARD: PaO2/FiO2 ≤100 mmHg with PEEP ≥5 cmH2.[7]

Procalcitonin is a propeptide of calcitonin that is produced by the thyroid gland. No procalcitonin is released into the bloodstream because the majority of the procalcitonin produced by thyroid C cells is converted into calcitonin. Consequently, procalcitonin's serum concentration in healthy individuals is incredibly low (0.05 ng/ml).[8] It has been considered as one of the most effective markers of bacterial sepsis, providing a new tool for early diagnosis and prognostic assessment of infection in various clinical conditions.[9] However, in the presence of infection, serum concentration of procalcitonin rises due to increased production from ectopic tissues such as the lung, intestine, liver and kidney.[10] Several studies have reported a moderate increase in procalcitonin levels postoperatively in cardiac patients as well as other conditions associated with systemic inflammation as severe trauma and different types of surgery.[11] The maximum peak level found different in different studies reported at a range of 0.5–7.0 ng/ml by immunoluminometric study.[12]

Patients were assigned to the procalcitonin - elevated cohort or control cohort according to serum procalcitonin concentration on the first postoperative day with a cut-off value of 7.0 ng/ml. This cut-off value was obtained from a previous retrospective case-control study in a similar study population and more than 100 cases were enrolled in that study between 2010 and 2014.[13]

Cardiac surgery with cardiopulmonary bypass triggers an inflammatory response involving proinflammatory cytokines such as TNF-a, IL-6, and IL-8 as well as activation of the complement system because of exposure of the blood to artificial surface causes systemic inflammatory response syndrome which lead to acute respiratory distress syndrome. This endotoxin release as well as the cascade of proinflammatory cytokines also affect procalcitonin secretion. Therefore, serum procalcitonin might serve as an indicator of the inflammatory response caused by cardiopulmonary bypass and thereby warning the development of acute respiratory distress syndrome.

Therefore, this study aimed to see the association of patients with higher serum procalcitonin concentration have a higher incidence of acute respiratory distress syndrome in postoperative period following cardiac surgery under cardiopulmonary bypass.

## METHODS

### *Study place*

This was a prospective observational study conducted in the Department of Cardiac Surgery, National Heart Foundation Hospital and Research Institute, Dhaka, Bangladesh.

### *Study duration*

The study was conducted during the period from September, 2020 to August, 2022.

### *Inclusion criteria*

In our study, we included 140 patients who underwent cardiac surgery with cardiopulmonary bypass meeting the inclusion and exclusion criteria in the cardiac surgery department at the National Heart Foundation Hospital and Research Institute. Patients were divided into two groups- group A included 70 patients with serum procalcitonin levels <7 ng/ml and group B included 70 patients with serum procalcitonin levels>7 ng/ml.

These are the following criteria to be eligible for enrollment as our study participants: a) Patients aged more than 18 years; b) Patients with elective cardiac surgery under cardiopulmonary bypass; c) Patients being free from active preoperative infection or inflammatory disease; d) Patients with leukocyte count <12×10^9/l, body temperature <37.5℃ were included in the study and a) Patients with a previous history of lung surgery. b) Patients with emergency and radio cardiac surgery. c) Patients with any history of acute illness (e.g., renal or pancreatic diseases, ischemic heart disease, asthma, COPD etc). d) Patients who were not willing to participate were excluded from our study.

***Surgical procedure***

All patients included in this study were operated on through a median sternotomy approach and using a cardiopulmonary bypass. A standard CPB circuit was used and a mean arterial pressure was kept 50 to 60 mm of Hg. Myocardial protection was achieved with intermittent antegrade cold blood cardioplegia with moderate systemic hypothermia (30℃ to 32℃). After completing the operating procedure, protamine sulfate (100:1 ratio) was used to reverse the heparin effect at completion of the surgical procedure. Following the surgical procedure, all the patients were brought to the Cardiovascular Intensive Care Unit (ICU) where they were monitored until the patients were extubated.

***Data collection procedure***

A thorough history and physical examination were performed and recorded on the questionnaire. Informed consent was taken from each subject before enrollment. Detailed history, clinical examination, and relevant investigation reports of all patients were recorded in the data collection sheet preoperatively. Demographic data were noted. Type of surgery, duration of operation, CPB time, aortic cross clamp time, blood transfusion, and FFP transfusion during the operation were recorded. Serum concentration of procalcitonin on 1st postoperative day was recorded. Patients were divided into two groups; group A included 70 patients with serum procalcitonin level <7 ng/ml and group B included 70 patients with serum procalcitonin level >7 ng/ml according to serum procalcitonin level on the 1st postoperative day with a cut-off value of 7.0 ng/ml. First postoperative day fluid balance and postoperative NT-Pro BNP were also recorded. Patients were followed daily up to the 7th postoperative day to determine whether the patient developed ARDS. Blood gas analysis was tested at least once per day when the patients were in the ICU. All data were collected, summarized, and statistically analyzed.

***Statistical analysis***

All data were recorded systematically in preformed data collection form. Quantitative data was expressed as mean and standard deviation and qualitative data was expressed as frequency distribution and percentage. Statistical analysis was done with independent Student's t-test for continuous data, Chi-square for categorical data, and Mann-Whitney U test for nonparametric data. Receiver-operating characteristics curve assessing the predictive performance of serum procalcitonin on the development of ARDS. A p-value <0.05 was considered as significant. Statistical analysis was performed by using SPSS 26 (Statistical Package for Social Sciences) for Windows version 10. The study was approved by the Ethical Review Committee of National Heart Foundation Hospital and Research Institute.

## RESULTS

Table 1 shows the distribution of the patients by age, gender, and BMI. The mean age of the patients was 43.20 (±13.18) years and 46.61 (±13.75) years in group A and group B respectively. In group A, 41.4% of patients were male and 58.6% were female; in group B, 52.9% were male and 47.1% were female. Among all patients, 40 (57.1%) and 33 (47.1%) patients of groups A & B respectively had normal body mass index (BMI). Only 6 (8.6%) patients in group A and 8 (11.4%) patients in group B were obese. The mean BMI of group A was 23.34 (±4.14) kg/m2 and group B was 23.53 (±4.41) kg/m2. There was no statistically significant ($p > 0.05$) difference observed in age, gender & BMI between the two groups.

Table 2 shows the distribution of the patients by preoperative clinical parameters. In group A mean (±SD) WBC counts was 8477.14 (±1295.72)/$mm^3$ of blood and in group B mean (±SD) WBC count was 8251.43(±1336.74)/$mm^3$ of blood. In groups A & B mean (±SD) NT pro-BNP was 251.86(±205.16) pg/ml and 217.01(±157.55) pg/ml respectively. There were no statistically significant ($p > 0.05$) differences observed in preoperative WBC and NT-pro-BNP between the two groups.

Table 3 shows the distribution of the patients by per-operative variables. In group A mean (±SD) total operative time was 4.46 (±0.98) hours, the mean (±SD) cardiopulmonary bypass (CPB) time was 105.01 (±33.95) minutes, and the mean (±SD) aortic cross-clamp time was 69.03 (±27.54) minutes and in group B mean (±SD) total operative time were 5.56 (±1.80) hours, mean (±SD) cardiopulmonary bypass (CPB) time were 164.04 (±78.67) minutes and mean (±SD) aortic cross-clamp time was 95.83 (±34.11) minutes.

Table 4 shows the distribution of the patients by postoperative variables. In group A mean (±SD) serum procalcitonin level on 1st postoperative day was 1.36 (±0.97) ng/ml and in group B mean (±SD) serum procalcitonin level on 1st postoperative day was 27.09 (±26.11) ng/ml. The mean(±SD) 1st postoperative day fluid balance in group A was -300.7 (±100.9) ml, postoperative NT-Pro- BNP was 316.6 (±204.2) pg/ml, and in group B was -309.9 (±109.9) ml, post-operative NT-Pro- BNP were 264.54 (±138.28) pg/ml.

Table 5 shows the distribution of the patients by severity of ARDS. In group A, 64 (91.4%) patients had no ARDS, mild ARDS (5.7%), moderate ARDS (1.4%), severe ARDS (1.4%), and in group B, no ARDS (64.3%), mild ARDS (17.1%), moderate ARDS (8.6%), severe ARDS (10.0%). There was a statistically significant ($p<0.05$) difference observed between the two groups.

Table 6 shows the mean (±SD) mechanical ventilation time in group A was 8.39 (±1.97) hours, duration of ICU

stay were 23.09 (±3.54) hours, postoperative hospital stay was 7.20 (±0.67) days and in group B were 17.97 (±16.53) hours, duration of ICU stay were 40.79 (±32.66) hours, postoperative hospital stay was 8.49 (±2.64) days. There were statistically significant ($p<0.05$) differences between the two groups.

**Table 1: Comparison of demographic variables of the patients between two groups (n=140).**

| Demographic variables | Group A (n=70) | Group B (n=70) | P value |
|---|---|---|---|
| **Age group (in years)** | | | |
| 18-27 | 11 (15.7%) | 9 (12.9%) | |
| 28-37 | 18 (25.7%) | 10 (14.3%) | |
| 38-47 | 13 (18.6%) | 17 (24.3%) | |
| 48-57 | 17 (24.3%) | 17 (24.3%) | |
| >57 | 11 (15.7%) | 17 (24.3%) | |
| **Mean±SD** | 43.20±13.18 | 46.61±13.75 | $0.136^{NS}$ |
| **Gender** | | | |
| Male | 29 (41.4%) | 37 (52.9%) | $0.176^{NS}$ |
| Female | 41 (58.6%) | 33 (47.1%) | |
| **BMI** | | | |
| Underweight (<18.50) | 8 (11.4%) | 9 (12.9%) | |
| Normal weight (18.50-24.99) | 40 (57.1%) | 33 (47.1%) | |
| Overweight (>25.0-29.99) | 16 (22.9%) | 20 (28.6%) | |
| Obese (>30.0) | 6 (8.6%) | 8(11.4%) | |
| **Mean±SD** | 23.34±4.14 | 23.53±4.41 | $0.796^{NS}$ |

NS =Not Significant.

**Table 2: Comparison of preoperative clinical parameters between two groups (n=140).**

| Pre-operative variables | Group A (n=70) | Group B (n=70) | P value |
|---|---|---|---|
| **Preoperative WBC (per mm3 of blood)** | 8477.14±1295.72 | 8251.43±1336.74 | $0.312^{NS}$ |
| **Range (min-max)** | (5700.0–11200.0) | (5100.0–12000.0) | |
| **Preoperative NT-pro-BNP (pg/ml)** | 251.86±205.16 | 217.01±157.55 | $0.262^{NS}$ |
| **Range (min-max)** | (20.0–875.0) | (25.0–778.0) | |

NS =Not Significant.

**Table 3: Comparison of per-operative variables between two groups (n=140).**

| Variables | Group A (n=70) | Group B (n=70) | P value |
|---|---|---|---|
| **Total operative time (hrs)** | 4.46±0.98 | 5.56±1.80 | $<0.001^{S}$ |
| **Range (min-max)** | (3.0–9.3) | (3.3–15.0) | |
| **CPB time (min)** | 105.01±33.95 | 164.04±78.67 | $<0.001^{S}$ |
| **Range (min-max)** | (48.0–252.0) | (43.0–660.0) | |
| **Aortic cross-clamp time (min)** | 69.03±27.54 | 95.83±34.11 | $<0.001^{S}$ |
| **Range (min-max)** | (26.0–180.0) | (19.0–244.0) | |
| **Blood transfusion (unit)** | 1.89±0.67 | 2.07±0.89 | $0.166^{NS}$ |
| **Range (min-max)** | (1.0–4.0) | (1.0–8.0) | |
| **FFP transfusion (unit)** | 1.90±0.30 | 2.00±0.42 | $0.107^{NS}$ |
| **Range (min-max)** | (1.0–2.0) | (1.0–4.0) | |

NS =Not Significant, S=Significant.

**Table 4: Comparison of postoperative variables between two groups (n=140).**

| Variables | Group A (n=70) | | Group B (n=70) | | P value |
|---|---|---|---|---|---|
| | Mean±SD (range) | Median | Mean±SD (range) | Median | |
| **Serum procalcitonin level on 1st postoperative day (ng/ml)** | 1.36±0.97 (0.0–5.3) | 1.10 | 27.09±26.11 (8.0–94.0) | 14.2 | $<0.00^{S}$ |
| **1st postoperative day fluid balance (ml)** | -300.7±100.9 (-474.0– -42.0) | -297.0 | -309.9±109.9 (-475.0–-110.0) | -321.5 | $0.554^{NS}$ |
| **Postoperative NT-Pro BNP (pg/ml)** | 316.6±204.2 (74.0–1050.0) | 255.0 | 264.54±138.28 (77.0–896.0) | 243.5 | $0.286^{NS}$ |

**Table 5: Comparison of severity of ARDS between two groups (n=140).**

| Severity of ARDS | Group A (n=70) | Group B (n=70) | P value |
|---|---|---|---|
| **No ARDS** | 64 (91.4%) | 45 (64.3%) | 0.002[S] |
| **Mild ARDS** | 4 (5.7%) | 12 (17.1%) | |
| **Moderate ARDS** | 1 (1.4%) | 6 (8.6%) | |
| **Severe ARDS** | 1 (1.4%) | 7 (10.0%) | |
| **Total** | 70 (100.0%) | 70 (100.0%) | |

**Table 6: Comparison of mechanical ventilation time, duration of ICU stays, and postoperative hospital stay between two groups (n=140).**

| Variable | Group A (n=70) | Group B (n=70) | P value |
|---|---|---|---|
| **Mechanical ventilation time (hours)**<br>**Range (min-max)** | 8.39±1.97<br>(6.0–16.0) | 17.97±16.53<br>(6.0–80.0) | <0.001[S] |
| **Duration of ICU stay (hours)**<br>**Range (min-max)** | 23.09±3.54<br>(18.0–36.0) | 40.79±32.66<br>(18.0–144.0) | <0.001[S] |
| **Postoperative hospital stay (days)**<br>**Range (min-max)** | 7.20±0.67<br>(7.0-10.0) | 8.49±2.64<br>(4.0-16.0) | <0.001[S] |

**Table 7: Area under the curve.**

| Test result variable (s): Serum procalcitonin (ng/ml) | | | | | | | |
|---|---|---|---|---|---|---|---|
| **AUC** | **SE** | **Cut of value** | **Sen.** | **Spe.** | **P value** | **95% CI** | |
| | | | | | | Lower | Upper |
| 0.891 | 0.036 | 10.0 | 80.6% | 76.1% | 0.000 | 0.820 | 0.962 |

After ROC analysis, procalcitonin had an AUC of 0.891 (95% CI 0.820 to 0.962, p<0.001) for ARDS prediction. The optimal cut-off value of procalcitonin was 10 ng/ml, yielding a sensitivity of 80.6% and a specificity of 76.1% for ARDS.

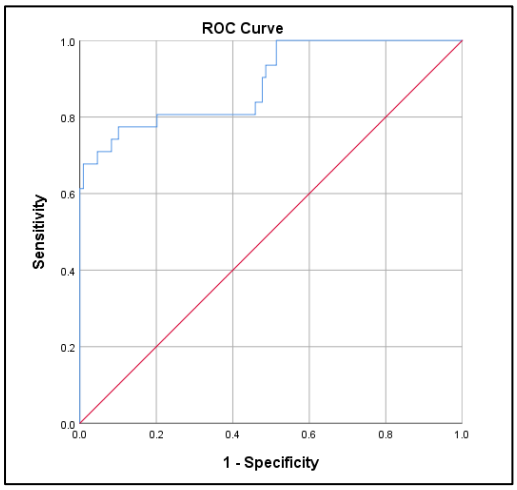


**Figure 1: Receiver-operating characteristics curve assessing the predictive performance of pct on the development of ARDS.**

## DISCUSSION

In this study, the mean (±SD) serum procalcitonin level on 1st post-operative day in group A was 1.36 (±0.97) ng/ml and in group B was 27.09 (±26.11) ng/ml. These findings were consistent with the study conducted by Cheng and Chen, 2020, where procalcitonin concentration was 16.23±5.9 ng/ml in the procalcitonin elevated cohort, and 2.70±1.43 ng/ml in the procalcitonin control cohort (p<0.001).[13]

Meisner et al. reported that patients with PCT above 2 ng/ml on day 1 or 2 (n=55) had postoperative abnormalities (95%) than patients with lower PCT (59%). Specifically, the incidence of three or more criteria of the "systemic inflammatory response syndrome" was 45% versus 4% (area under the curve of the receiver operating characteristic 0.866).[14] Aouifi et al, reported peak procalcitonin was 1.79 (1.64) ng/ ml in SIRS patients vs 0.34 (0.32) ng/ ml in patients without SIRS. Procalcitonin levels were measured in 10 patients who had postoperative complications. Serum procalcitonin concentrations in these patients ranged from 6.2 to 230 ng/ml. SIRS caused by cardiac surgery, with and without cardiopulmonary bypass, influenced serum PCT concentrations with a moderate and transient postoperative peak on the first postoperative day, and procalcitonin concentrations greater than 5 ng/ml are highly suggestive of a postoperative complication.[2]

In group A, 91.4% of patients, and in group B, 64.3% of patients had no ARDS. The incidence of ARDS was significantly higher in group B than in group A (35.7% versus 8.5%, p=0.002, p< 0.05). There was a statistically significant (p<0.05) difference between the two groups.

These results were in line with research by Cheng and Chen (2020), who found that the procalcitonin-elevated cohort had a considerably greater incidence of ARDS (21.9% versus 5.6%, p<0.001) than the procalcitonin control cohort. In addition, there was a substantial increase in the incidence of moderate-to-severe ARDS (10.9% versus 0.4%, p<0.001) in the procalcitonin-raised cohort compared to the procalcitonin control cohort.[13]

Milot et al, on the other hand, conducted a retrospective, case-control study on 3,278 patients who had cardiac surgery and cardiopulmonary bypass between January 1995 and December 1998, and 13 of them developed ARDS during the postoperative period. ARDS occurred at a rate of 0.4%, with a mortality rate of 15%.[5]

Chen et al, conducted a retrospective chart review on 457 patients who underwent isolated valvular heart surgery. The primary outcome was postoperative ARDS, according to the 2012 Berlin definition of ARDS. A total of 37 patients (8.1%) developed postoperative ARDS, with a mortality rate of 29.7%.[15] In current study showed after ROC analysis, procalcitonin had an AUC of 0.891 (95% CI 0.820 to 0.962, p<0.001) for ARDS prediction. The optimal cut-off value of procalcitonin was 10 ng/ml, yielding a sensitivity of 80.6% and a specificity of 76.1% for ARDS. In accordance this study Cheng and Chen, 2020 reported a cut-off value 6.5 ng/ml and the sensitivity 59.3% and specificity 81.4%.[13]

Prat et al, studied 151 patients, of whom 136 recovered normally and 15 experienced severe postoperative complications. Except for the control group, all groups' mean procalcitonin levels were significantly higher on the first postoperative day. The area under the ROC curve (complicated vs. uncomplicated) for procalcitonin after surgery (PCT 0), 0.978 (SE 0.015, p=0.0001) for procalcitonin on the 1st postoperative day (PCT 1), and 0.927 (SE 0.066, p=0.0001) for procalcitonin on the second postoperative day was 0.726 (SD 0.079, p=0.004). (PCT 2).[11]

The rise in serum PCT levels following cardiac surgery with CPB is supported by the work of Meisner and colleagues.[16] They found an increase in PCT in 59% of patients undergoing CPB. In contrast to both our results and those of Meisner and colleagues, Boeken and colleagues found no significant increase in serum PCT after cardiac surgery and concluded that cardiac surgery with CPB did not influence PCT.[17] In a previous study, Kilger and colleagues, comparing peak PCT concentrations after conventional CABG with CPB and after direct coronary artery bypass without CPB, showed that peak serum PCT concentrations were significantly greater in conventional CABG than after surgery without CPB.[12]

Regardless of the surgical method, there seems to be a connection between postoperative SIRS and the rise in serum PCT concentration following heart surgery. This implies a connection between PCT and the endotoxin release and cytokine cascade brought on by the surgical operation. Dandona and colleagues established a chronology between endotoxin, cytokine, and PCT production, supporting the concept of a link between PCT and cytokine cascade.[18] We speculate that the postoperative inflammatory cascade is probably responsible for the increase in serum PCT after CPB. Fransen and colleagues showed that cytokine production is present with the same magnitude with or without CPB. So, the increase in PCT concentrations after cardiac surgery without CPB may also be related to the cytokine cascade.[19]

The typical range of serum PCT levels following heart surgery has yet to be identified. In our study, serum PCT concentrations remained less than 7 ng/ml in all patients with fewer complications and were 8.0–94.0 ng/ml in the patients with moderate to severe complications. This supports the study by Hensell and colleagues in which serum PCT concentrations of 5.1–14.3 ng/ml were measured on day 1 after cardiac surgery in nine patients who developed acute lung injury after the operation. This suggests that serum PCT concentrations of more than 5 ng/ml are strongly indicative of a postoperative complication. PCT seems to be an important marker of impending complications after cardiac surgery, particularly when conventional clinical and biological signs can be difficult to interpret.[20]

The limitations of the study were that it was a single-center study. We took a small sample size due to our short study period. We did not measure serum procalcitonin level pre-operatively. After evaluating those patients, we did not follow up with them for the long term and did not know other possible interference that may happen in the long term with these patients.

## CONCLUSION

In our study, we found that there was an association of high serum procalcitonin levels with acute respiratory distress syndrome in patients following cardiopulmonary bypass. The findings from our study showed that patients undergoing CPB cardiac surgery who have moderate to severe acute respiratory distress syndrome (ARDS) had greater serum PCT concentrations than those who do not or have mild ARDS. Serum PCT level may be a valuable biomarker to predict the onset of moderate to severe ARDS.

### *Recommendations*

So further study with a prospective and longitudinal study design including a larger sample size needs to be done to validate the findings of our study.

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## REFERENCES


1. Chen H, Cheng ZB, Yu RG. Procalcitonin as a predictor of moderate to severe acute respiratory distress syndrome after cardiac surgery with cardiopulmonary bypass: a study protocol for a prospective cohort study. BMJ open. 2014;4(10):6344.
2. Aouifi A, Piriou V, Blanc P, Bouvier H, Bastien O, Chiari P, et al. Effect of cardiopulmonary bypass on serum procalcitonin and C-reactive protein concentrations. British J Anaest. 1999;83(4):602-7.
3. Sanfilippo F, Palumbo GJ, Bignami E, Pavesi M, Ranucci M, Scolletta S, et al. acute respiratory distress syndrome in the perioperative period of cardiac surgery: predictors, diagnosis, prognosis, management options, and future directions. J Cardiothor Vasc Anest. 2022;36(4):1169-79.
4. Song JH, Woo WK, Song SH, Kim HH, Kim BJ, Kim HE, et al. Outcome of veno-venous extracorporeal membrane oxygenation use in acute respiratory distress syndrome after cardiac surgery with cardiopulmonary bypass. Journal of Thoracic Disease. 2016;8(7):1804.
5. Milot J, Perron J, Lacasse Y, Létourneau L, Cartier PC, Maltais F. Incidence and predictors of ARDS after cardiac surgery. Chest. 2001;119(3):884-8.
6. Ranieri VM, Rubenfeld GD, Taylor Thompson B, Ferguson ND, Caldwell E, et al. Acute respiratory distress syndrome: the Berlin Definition. JAMA: J American Med Assoc. 2012;307:23.
7. Rong LQ, Di Franco A, Gaudino M. Acute respiratory distress syndrome after cardiac surgery. Journal of thoracic disease. 2016;8(10):1177.
8. Soreng K, Levy HR. Procalcitonin: an emerging biomarker of bacterial sepsis. Clinical microbiology newsletter. 2011;33(22):171-8.
9. Kallel S, Jmel W, Jarraya A, Abdenadher M, Frikha I, Karouia A. The role of procalcitonin and N-terminal pro-B-type natriuretic peptide in predicting outcome after cardiac surgery. Perfusion. 2012;27(6):504-11.
10. Assicot M, Bohuon C, Gendrel D, Raymond J, Carsin H, Guilbaud JJ. High serum procalcitonin concentrations in patients with sepsis and infection. The Lancet. 1993;341(8844):515-8.
11. Prat C, Ricart P, Ruyra X, Domínguez J, Morillas J, Blanco S, et al. Serum concentrations of procalcitonin after cardiac surgery. J Cardiac Surg. 2008;23(6):627-32.
12. Kilger E, Pichler B, Goetz AE, Rank N, Welte M, Morstedt K, et al. Procalcitonin as a marker of systemic inflammation after conventional or minimally invasive coronary artery bypass grafting. Thorac Cardiovasc Surg. 1998;46:130-3.
13. Cheng ZB, Chen H. Higher incidence of acute respiratory distress syndrome in cardiac surgical patients with elevated serum procalcitonin concentration: a prospective cohort study. Eur J Med Res. 2020;25:1-8.
14. Meisner M, Rauschmayer C, Schmidt J, Feyrer R, Cesnjevar R, Bredle D, et al. Early increase of procalcitonin after cardiovascular surgery in patients with postoperative complications. Int Care Med. 2002;28:1094-102.
15. Chen SW, Chang CH, Chu PH, Chen TH, Wu VC, Huang YK, et al. Risk factor analysis of postoperative acute respiratory distress syndrome in valvular heart surgery. J Cri Care. 2016;31(1):139-43.
16. Meisner M, Tschaikowsky K, Hutzler A, Schick C, Schuttler J. Postoperative plasma concentrations after different types of surgery. Intensive Care Med. 1998;24:680–4.
17. Boeken U, Feindt P, Petzold T. Diagnostic value of procalcitonin: the influence of cardiopulmonary bypass, aprotinin, SIRS, and sepsis. Thorac Cardiovasc Surg. 1998;46:348–51.
18. Dandona P, Nix D, Wilson MF. Procalcitonin increase after endotoxin injection in normal subjects. J Clin Endocrinol Metab. 1994;79:1605–8.
19. Fransen E, Maessen J, Dentener M, Senden N, Geskes G, Buurman W. Systemic inflammation present in patients undergoing CABG without extracorporeal circulation. Chest. 1998;111:1290–5
20. Hensel M, Volk T, Docke WD. Hyperprolactinemia in patients with non-infectious SIRS and pulmonary dysfunction associated with cardiopulmonary bypass. Anesthesiol. 1998;89:93–104.


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