

Comparison of fluid bolus and fluid bolus plus low dose noradrenaline infusion on cardiovascular collapse among critically ill adults before tracheal intubation: a randomized trial

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Abstract

Background: Endotracheal intubation of critically ill patients is associated with various complications, mainly hypotension. The administration of an intravenous fluid bolus before induction of anesthesia has been used to prevent hypotension during endotracheal intubation in the intensive care unit (ICU). However, some patients still experience hemodynamic instability. Therefore, this study was conducted to compare the effect of a fluid bolus alone and a fluid bolus plus low-dose noradrenaline infusion on cardiovascular collapse among critically ill adults before tracheal intubation.

Methods: The randomized trial enrolled 136 critically ill patients who required endotracheal intubation. Patients were allocated to the intravenous fluid (group A) and the intravenous fluid plus low-dose noradrenaline group (group B). Cardiovascular collapse, hypotension, and other secondary outcomes were recorded.

Results: Vasopressor requirement at 2 minutes postinduction was lower in group B (39.7%) than in group A (44.1%). Cardiac arrest within 1 hour occurred in 2.9% of patients in group A. Death within 1 hour occurred in 2.9% patients of group A and 1.5% of group B. The requirement for renal replacement therapy, duration of vasopressor requirement and in-hospital mortality were higher in group B. The duration of ICU stay and the duration of ventilator requirement were higher in group A. All these differences were statistically insignificant except for the total duration of hospital stay, which was longer in group A than in group B ($P = 0.036$).

Conclusion: Among critically ill patients undergoing tracheal intubation, the administration of intravenous fluid bolus plus low-dose noradrenaline, compared with a fluid bolus alone, did not significantly reduce the incidence of cardiovascular collapse.

Keywords: Cardiovascular collapse, Critically ill patients, Endotracheal intubation, Fluid bolus, Low-dose noradrenaline

Introduction

Tracheal intubation is one of the most frequently performed procedures in the management of critically ill patients. It is, however, associated with a high incidence of complications such as hemodynamic instability, decreased oxygen saturation, failed

tracheal intubation, esophageal intubation, airway trauma, aspiration, cardiac arrest, and even death.^[1–3] These complications occur at an increased rate in critically ill patients compared with those undergoing elective endotracheal intubations (ETIs).^[4, 5]

Hypotension occurs during 25%–42% of tracheal intubations in the intensive care unit (ICU) and can lead to cardiac arrest and death.^[6–11] Cardiovascular collapse, defined as a profound drop in blood pressure requiring intervention, can occur in up to 42% of cases.^[6] This peri-intubation cardiovascular collapse is associated with a significant increase in the risk of mortality and longer length of stay in both the hospital and the ICU.^[12, 13]

Therefore, it is important to optimize the patient before tracheal intubation in order to maintain hemodynamic stability and decrease further complications during tracheal intubation.

The administration of an intravenous fluid bolus beginning before induction of anesthesia has been proposed to prevent cardiovascular collapse during tracheal intubation in the ICU.^[14–18] Given the fact that despite this fluid bolus administration, a large proportion of patients not only experience hypotension but also cardiovascular collapse, some researchers have recently proposed administering a fluid bolus combined with low-dose noradrenaline infusion as a strategy to enhance cardiovascular stability and reduce the risk of cardiovascular collapse among critically ill adults during procedures such as tracheal intubation.^[19] This approach addresses both preload and afterload, thereby optimizing cardiac output and maintaining adequate blood pressure. With this concept in mind, some researchers have proposed

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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evaluating the role of prophylactic vasopressor use either alone or in combination with fluid bolus in order to evaluate their effect on postintubation hypotension and cardiovascular collapse.^[20, 21]

However, most of the evidence is still in an evolutionary phase, and there is a lack of adequate data to establish the efficacy of combined vasopressor and fluid bolus use.

Therefore, the present study was designed to compare whether a fluid bolus plus low-dose noradrenaline infusion reduces the incidence of cardiovascular collapse among critically ill adults before tracheal intubation compared with a fluid bolus alone.

Materials and methods

The study was performed in the medical adult ICU of our center between July 2023 and July 2024. The study followed the principles of the Declaration of Helsinki as revised in 2013. This study was approved by the Institutional Ethics Committee of Era University (reference number: ELMC&H/R-Cell/2023/37 on February 28, 2023), and written informed consent was obtained from the patient or their next kin. The trial was registered with Clinical Trials Registry—India (CTRI number: CTRI/2023/07/054773, URL: <https://ctri.nic.in/Clinicaltrials/main1.php?EncHid=99458.25924>, date of registry: July 5, 2023).

The primary outcome of the study was the occurrence of cardiovascular collapse, defined as a decrease in mean arterial pressure (MAP) to less than 65 mm Hg between the induction of anesthesia and 2 minutes after tracheal intubation. The secondary outcomes included a decrease in systolic blood pressure (SBP) to less than 90 mm Hg; new or increased noradrenaline use between induction and 2 minutes after tracheal intubation; cardiac arrest within 1 hour of tracheal intubation; death within 1 hour of tracheal intubation; ventilator-free days; noradrenaline-free days; need for renal replacement therapy postintubation in the ICU; length of ICU stay; length of hospital stay; ICU mortality; and hospital mortality.

Critically ill patients aged 18 years or older who were admitted to the ICU of our center and required ETI were included in the study. Patients were excluded if any of the following criteria were met: ETI performed outside the ICU or hospital setting; tracheal intubation during cardiac arrest; use of vasoactive medications at the time of screening; hypotension (SBP <90 mm Hg) at screening; hypertension (SBP >140 mm Hg) at screening; urgency of tracheal intubation precluding performance of study procedures; or pregnancy..

Study design

This was a pragmatic, single-center, parallel-group, randomized trial comparing the efficacy of fluid bolus plus low-dose noradrenaline with a fluid bolus alone in preventing cardiovascular collapse among critically ill patients before tracheal intubation.

Sample size

Sample size was calculated using the formula^[6]:

$$n = \frac{(Z_{\alpha/2} + Z_{\beta})^2 \cdot [p_1(1-p_1) + p_2(1-p_2)]}{(p_1 - p_2)^2}$$

Control event rate, $p_1 = 0.29$

Assumed intervention event rate, $p_2 \approx 0.10$

Absolute risk reduction = 19%

$\alpha = 0.05 \rightarrow Z_{\alpha/2} = 1.96$

Power = 80% $\rightarrow Z_{\beta} = 0.84$

Required sample size ≈ 62 per group

Total = 124

With 10% attrition $\rightarrow 136$

The sample size was calculated for the comparison of 2 independent proportions. Based on prior literature reporting an incidence of cardiovascular collapse of approximately 29% during ETI in critically ill patients, and assuming that prophylactic low-dose noradrenaline could reduce this to 10% (absolute risk reduction 19%), with a 2-sided α of 0.05 and power of 80%, the required sample size was 62 patients per group. After accounting for 10% attrition, the final sample size was 136 patients.

Methodology

A total of 146 patients were screened for enrollment, of whom 7 didn't meet the inclusion criteria, and 3 declined to participate in the study. A total of 136 patients were finally included in the study after obtaining written informed consent from the caregiver.

Patients were randomly divided into 2 parallel groups using the Sequentially Numbered Opaque Sealed Envelope method in a 1:1 ratio, with 68 patients in each group. The airway operator was blinded to the randomization and was therefore sent out of the intervention room. Monitors were attached to the patients. Group A received 500 mL of fluid bolus (Ringer lactate) over 10 minutes, along with normal saline infusion to ensure blinding for vasopressor before the administration of procedural medications. Group B received a 500 mL fluid bolus (Ringer lactate) plus low-dose noradrenaline (0.05 $\mu\text{g/kg/min}$) infusion over 10 minutes prior and 5 minutes after tracheal intubation. Three milligrams of noradrenaline were diluted in 47 mL of 5% dextrose solution, and the infusion was started according to the decided dose. Any incidence of hypertension during this period was recorded, and noradrenaline was discontinued if SBP >140 mm Hg. Patients in both groups continued to receive the intravenous fluid as part of routine clinical care before enrollment. The airway operator then entered the room to proceed with the intubation. Patients were preoxygenated for 3 minutes; rapid sequence tracheal intubation was performed using fentanyl, ketamine, or etomidate, and succinylcholine or rocuronium. Apneic oxygenation was performed between induction and laryngoscopy. However, gentle positive pressure was applied during this period if the operator felt the need to do so. Patients were intubated using a video laryngoscope. Following confirmation of tube position and fixation, patients were placed on a ventilator using protective lung ventilation.

Relevant demographic details and clinical data were obtained from the patients' case files, including age, sex, comorbidities, indications for ICU admission, vital signs, and ICU events. These data were recorded on a separate case sheet for every patient and entered in Microsoft Excel (Microsoft Corporation, Redmond, WA), which was used for analysis.

Statistical analysis

The statistical analysis was performed using IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY). The values were represented in number (%), mean $\pm$ standard deviation, or median (interquartile range, IQR). To compare the proportional difference between the 2 groups chi-square test was used, Student t test was used for normally distributed continuous variables, Mann–Whitney U test was used for nonnormally distributed continuous variables, and Fisher exact test was used for categorical variables.

The level of significance was kept at $P < 0.05$.

The details of the protocol can be checked at the following link: <https://ctri.nic.n/Clinicaltrials/rmaindet.php?trialid=84380&EncHid=99458.25924&modid=1&compid=19>.

Results

The study was conducted as a randomized comparative study at the Department of Anaesthesiology and Critical Care Medicine of our center. Figure 1 shows the patient flowchart. A total of 136 patients were included in the study, with 68 patients in each group.

Baseline demographic characteristics were comparable between the fluid bolus group and fluid bolus plus low-dose noradrenaline group, as shown in Table 1. Hypertension was the most common comorbidity in both groups, followed by diabetes, stroke, chronic kidney disease, asthma, cirrhosis, and malignancy. The distribution of patients in 2 study groups did not show bias for any of the comorbidities ($P > 0.05$). The most common indication for admission to ICU in group A was hypoxic respiratory failure, followed by altered mental status, hypercapnic respiratory failure, and other indications. In group B, the most common indication was altered mental status, followed by hypoxic respiratory failure, hypercapnic respiratory failure, and other indications. No significant difference was found between the 2 study groups ($P = 0.331$). Simplified Acute Physiology Score II was comparable between the 2 groups, indicating no difference in disease severity (Table 1).

The results of primary and secondary outcomes are shown in Table 2. The proportion of patients experiencing MAP < 65 mm Hg was comparable between group A and group B (44.1% vs. 39.7%, respectively; $P = 0.687$). Similarly, there were no statistically significant differences in MAP or SBP between the 2 groups ($P > 0.05$). One patient in group A developed SBP < 90 mm Hg at 2 minutes postinduction. Vasopressor requirement at 2 minutes postinduction, incidence of cardiac arrest within 1 hour, and mortality within 1 hour were comparable between the groups, with no statistically significant differences observed. Likewise, renal replacement therapy requirement, duration of ICU stay,

duration of mechanical ventilation, and duration of vasopressor support did not differ significantly between group A and group B (all $P > 0.05$). The differences of these outcomes were not clinically significant. The total duration of hospital stay of patients of group A (16.4 [11.2–21.6] days) was significantly higher than that of group B (13.8 [9.2–18.3] days) ($P = 0.036$). ICU/hospital mortality was comparable in both groups ($P > 0.05$).

Discussion

ETI is a critical procedure for the management of critically ill patients, ensuring adequate ventilation and oxygenation. However, ETI is associated with several concerns and potential complications. Several mechanisms contribute to the incidence of hemodynamic instability and cardiovascular collapse during intubation. Some of the mechanisms are underlying shock, respiratory failure, metabolic acidosis, other patho-physiological changes along with additional effects of fluid deficit, anesthetic drugs suppressing the endogenous activation of sympathetic response, and the increased intrathoracic pressure due to positive pressure mechanical ventilation. Among various post-ETI complications seen in critically ill patients, post-ETI hypotension is the most important one, as it can often lead to adverse outcomes, including cardiac collapse. Administering a fluid bolus before ETI can play a crucial preventive role in mitigating the risk of postintubation hypotension and subsequent cardiac collapse in critically ill patients. In addition to a fluid bolus, the concurrent use of vasopressors is often recommended for patients at high risk of hypotension. This combination strategy of fluids and vasopressors can be particularly effective in patients with significant cardiovascular instability or those who do not respond adequately to fluid resuscitation alone.^[22] While fluids alone can be sufficient for many patients, the addition of vasopressors ensures a more robust and rapid response to hypotension, significantly lowering the risk of cardiac collapse by maintaining perfusion pressure to vital organs.^[23] With this background, the present study was planned to compare the effects of a fluid bolus alone versus a fluid bolus plus low-dose noradrenaline infusion on cardiovascular collapse among critically ill patients before

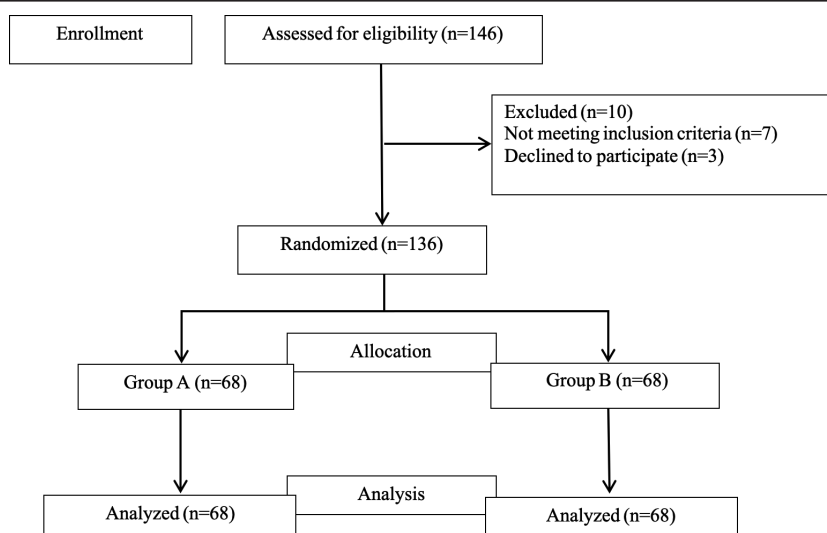


Figure 1. Flow chart of patient enrollment. A total of 146 patients were assessed for eligibility. Seven did not meet the inclusion criteria, and 3 refused to participate in the study. A total of 136 patients were included in the study (68 in each group).

Table 1**Baseline Patient Characteristics**

Characteristics of the Patient	Group A (Fluid Bolus) (n = 68)	Group B (Fluid Bolus + Noradrenaline) (n = 68)	P Value
Age (years), mean ± SD	53.37 ± 18.75	51.09 ± 17.90	0.470
Male, n (%)	32 (47.1)	31 (45.6)	0.863
Female, n (%)	36 (52.9)	37 (54.4)	0.863
Comorbidities			
Diabetes, n (%)	29 (42.6)	33 (48.5)	0.491
COPD, n (%)	12 (17.5)	11 (16.2)	0.819
Asthma, n (%)	3 (4.4)	4 (5.9)	0.698
Stroke, n (%)	5 (7.4)	7 (10.3)	0.545
CAD, n (%)	17 (25.0)	13 (19.1)	0.408
Hypertension, n (%)	38 (55.9)	41 (60.3)	0.602
Malignancy, n (%)	2 (2.9)	0	0.154
Cirrhosis, n (%)	1 (1.5)	1 (1.5)	>0.999
CKD, n (%)	5 (7.4)	5 (7.4)	>0.999
Indications for ICU admission			
Altered mental status, n (%)	26 (38.2)	33 (48.5)	0.331
Hypercapnic respiratory failure, n (%)	2 (2.9)	2 (2.9)	
Hypoxic respiratory failure, n (%)	30 (44.1)	29 (42.6)	
Others, n (%)	10 (14.7)	4 (5.9)	
SAPS II, mean ± SD	39.6 ± 8.72	37.48 ± 10.71	0.446

Data are reported as n (%) or mean ± SD. No significant difference was found between the 2 groups with respect to the baseline characteristics.

CAD, coronary artery disease; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; ICU, intensive care unit; SAPS II, Simplified Acute Physiology score II; SD, standard deviation.

Table 2**Primary and Secondary Outcomes**

End Points	Group A (Fluid Bolus) (n = 68)	Group B (Fluid Bolus + Noradrenaline) (n = 68)	P Value
Number of patients with MAP, 65 mm Hg between induction and 2 minutes postintubation, n (%)	30 (44.1)	27 (39.7)	0.687
MAP at 2 minutes postinduction (mm Hg), mean ± SD	64.88 ± 2.40	64.75 ± 4.02	0.816*
SBP at 2 minutes postinduction (mm Hg), mean ± SD	112.59 ± 7.27	112.62 ± 7.22	0.981*
Decrease in SBP to <90 mm Hg, n (%)	1 (1.5)	0	>0.999†
Vasopressor need 2 min postinduction, n (%)	32 (47.1)	29 (42.6)	0.489‡
Cardiac arrest within 1 hour, n (%)	2 (2.9)	0	0.497‡
Death within 1 hour, n (%)	2 (2.9)	1 (1.5)	>0.999‡
RRT need, n (%)	8 (11.8)	14 (20.6)	0.162
Duration of ICU stay (days), median (IQR)	11.0 (8.0–16.0)	10.0 (7.0–15.0)	0.110‡
Duration of mechanical ventilation (days), median (IQR)	8.0 (5.0–12.0)	7.0 (5.0–11.0)	0.206‡
Duration of vasopressor support (days), median (IQR)	7.0 (5.0–9.0)	7.3 (5.9–8.8)	0.398‡
Duration of hospital stay (days), median (IQR)	16.4 (11.2–21.6)	13.8 (9.2–18.3)	0.036‡
In hospital mortality/ICU mortality, n (%)	26 (38.2)	37 (54.4)	0.059

Data are reported as n (%) or mean ± SD or median (IQR).

ICU, intensive care unit; IQR, interquartile range; MAP, mean arterial pressure; RRT, renal replacement therapy; SBP, systolic blood pressure; SD, standard deviation.

*Student *t* test was used for normally distributed continuous variables.

†Fisher exact test was used for categorical variables.

‡Mann–Whitney *U* test was used for nonnormally distributed continuous variables. The duration of hospital stay was significantly lower in group B than in group A. No significant difference was found in other parameters between the 2 groups.

tracheal intubation. The present study, however, did not find any significant difference in cardiovascular collapse between the group receiving only fluid bolus and the group receiving fluid bolus along with low-dose noradrenaline. The only significant difference was found in the length of hospital stay, which was lower in the combination group.

The previous studies by Russell et al.^[11] and Janz et al.^[10] compared fluid bolus to no-fluid bolus, while Fuchita et al.^[19] compared prophylactic vasopressor to no vasopressor. The use of vasopressors in studies by Russell et al.^[11] and Janz et al.^[10] was

an additional intervention that was done as per the needs of the patients in both the groups (12% and 17% of cases, respectively) as compared with the present study where it was part of standard care in one of the 2 groups of study.

In our study, groups were statistically matched for age, sex, comorbidity, and other baseline characteristics similar to previous studies.^[11, 19] Hypoxic respiratory failure and altered mental status were the most common indications for ICU admission, responsible in our study accounting for >80% of cases. There was no significant difference between 2 groups for indications

for ICU admission. This was similar to the observation of Russell et al.,^[11] Fuchita et al.,^[19] and Janz et al.^[10] who also observed these 2 conditions to be the dominant indications.

Incidence of postintubation hypotension was non significantly higher in group A than in group B. Interestingly, in the study by Fuchita et al.,^[19] the secondary analysis of 2 multicenter randomized clinical trials reported incidence of peri-intubation hypotension to be higher in vasopressor group (41%) as compared with that in no-vasopressor group (32%) but did not find this difference to be statistically significant, thus showing vasopressor use did not help to reduce the peri-intubation incidence of hypotension. The study by Russell et al.^[11] was a multicenter randomized controlled trial to study the effect of fluid bolus administration on cardiovascular collapse among critically ill patients undergoing tracheal intubation had enrolled 538 patients in fluid group who received 500 mL of intravenous fluid bolus and 527 patients in no-fluid group. However, they reported a much lower incidence of cardiovascular collapse in both fluid bolus group (21%) as well as in no-fluid bolus group (18.2%), thus showing no benefit of fluid bolus on the cardiovascular collapse events. In the multicentric randomized controlled trial by Janz et al.^[10] which enrolled 337 patients to study the effect of a fluid bolus on cardiovascular collapse among critically ill adults undergoing tracheal intubation, the incidence of cardiovascular collapse was 20% in fluid bolus group as compared with 18% in no-fluid bolus group, and the difference was not statistically significant. Both these studies did not find a significant effect of fluid bolus on the risk of cardiovascular collapse.

In the current study, cardiac arrest was seen in only 2 (2.9%) patients in group A within 2 hours of intubation (one of these 2 patients died within one hour), whereas no cardiac arrest occurred in group B. For other outcomes such as renal replacement therapy need and ICU/inhospital mortality, no significant difference was found between the 2 groups, though the inhospital mortality rate was nonsignificantly higher in group B as compared with that in group A. A significant difference was observed only in the total duration of hospital stay, which was lower in the fluid plus vasopressor group. The shorter hospital stay may be more related to the nonsignificant increase in mortality observed in the fluid bolus plus norepinephrine group.

In their study, Russell et al.^[11] also did not find a significant difference between the 2 groups for events such as cardiac arrest (1.7% vs. 1.5%), early death (0.7% vs. 0.6%), and inhospital mortality rates (40.5% vs. 42.3%). Similar observations were also made by Janz et al.^[10] in their study. In both of these studies, additional use of vasopressor did not alter any of the targeted events in either group. In the study by Fuchita et al.,^[19] vasopressor group as compared with no vasopressor group had relatively higher events of cardiac arrest within 1 hour (4% vs. 3%) and inhospital mortality (57% vs. 47%) and failed to find a significant difference between the 2 groups, thus ruling out any benefit of additional vasopressor use in reducing these adverse events.

Limitations

The study had a few limitations. First, the trial was a single-center trial. Second, the volume of 500 mL was selected based on the previous trials to improve cardiac output in critically ill patients. It is not known whether the results would have been different if different doses of crystalloid or noradrenaline had been used. Third, the study used noradrenaline in the dose of 0.05 µg/kg/min. The

results might have been different if a higher dose of noradrenaline had been used. Finally, the sample size was based on an anticipated large treatment effect; therefore, the study may be underpowered to detect smaller, yet clinically meaningful differences.

Conclusion

The findings of the present study thus show that preemptive administration of noradrenaline in combination with a fluid bolus during ETI in the critically ill patients did not reduce the incidence of cardiovascular collapse. Among the secondary outcomes, only the need for a new or increased dose of noradrenaline within 2 minutes was reduced in the intervention group. Therefore, a comprehensive approach involving careful assessment of fluid status, appropriate use of vasopressors, and consideration of underlying cardiac function is essential to prevent postintubation cardiovascular collapse in critically ill patients. The findings suggest that there is need to understand the significance of fluid and vasopressor requirements in individualized context rather than standard care plans. Further studies on a larger sample size are recommended in order to unravel other facets of this problem.

Conflict of interest statement

The authors declare no conflict of interest.

Author contributions

Jahan SM contributed to concept and design of study; acquisition, analysis, and interpretation of data; drafting the manuscript; and final approval of the version to be published. Dubey M contributed to concept and design of study; acquisition, analysis, and interpretation of data; manuscript preparation, manuscript editing and review, and final approval of the version to be published. Mustahsin M contributed to concept of study, data interpretation, manuscript preparation, manuscript review, and final approval of the version to be published. Choubey S contributed to concept of study, manuscript editing, manuscript review, and final approval of the version to be published. Singh S contributed to data interpretation, manuscript review, and final approval of the version to be published. Amir M contributed to Data interpretation, manuscript editing and review, and final approval of the version to be published.

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Ethical approval of studies and informed consent

The study followed the principles of the Declaration of Helsinki as revised in 2013. This study was approved by the Institutional Ethics Committee of Era University (reference number: ELMC&H/R-Cell/2023/37 on February 28, 2023), and written informed consent was obtained from the patient or their next kin. The trial was registered with Clinical Trials Registry—India (CTRI number: CTRI/2023/07/054773, URL: <https://ctri.nic.in/Clinicaltrials/main1.php?EncHid=99458.25924>, date of registry: July 5, 2023). Written informed consent was obtained from all individual participants or their next kin included in the study.

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