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ORIGINAL RESEARCH

Risk factors of post-intubation hypotension in severe pneumonia patients

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Abstract

Post-intubation hypotension (PIH) was one of the serious complications after Endotracheal intubation (ETI) in Severe pneumonia (SP) patients. The risk factors of PIH were investigated in SP patients to provide a theoretical basis of early intervention. This was a retrospective study wherein 420 SP patients undergone ETI from December 2019 to December 2022 were selected as the study subjects. Patients were divided into the hypotension and normal blood pressure groups based on the blood pressures recorded after intubation. Two groups were compared for the general data and various physicochemical indicators before the intubation. The independent risk factors of hypotension after tracheal intubation were explored in SP patients through univariate and multivariate logistic regression. PIH was developed in 170 (40.47%) SP patients. Univariate logistic regression analysis exhibited that age (Odd Ratio (OR) = 1.021, p = 0.001), weight (OR = 0.976, p = 0.015), inducer usage before intubation (OR = 1.221, $p < 0.001$), chronic obstructive pulmonary disease (OR = 1.768, p = 0.013), sepsis (OR = 1.870, p = 0.006), and hyperelastic acidemia (OR = 1.665, p = 0.012) were associated with PIH. Multivariate logistic regression analysis indicated that age (OR = 1.015, p = 0.033), weight (OR = 0.974, p = 0.012), inducer usage before intubation (OR = 1.228, $p < 0.001$), chronic obstructive pulmonary disease (OR = 1.660, p = 0.037), sepsis (OR = 1.733, p = 0.035), and hyperelastic acidemia (OR = 1.679, p = 0.018) were the independent risk factors of PIH. The study demonstrated that SP patients were prone to PIH with advanced age, low body weight, inducer usage before intubation, increased lactic acid levels before intubation, and high risk factors of chronic obstructive pulmonary disease (COPD) or sepsis. The findings had potential of advanced patient care, and refined medical practices, and could stimulate further investigations in the field.

Keywords

Severe pneumonia; Endotracheal intubation; Hypotension; Risk factors

1. Introduction

Severe pneumonia (SP) has the main clinical manifestations of respiratory failure and multiple organ dysfunction resulting from various etiologies, pathogens and circumstances. SP incidence is high and ranges from 30% to 50% [1]. The disease poses health challenge because of its severity and mortality rates. SP is not restricted to any particular pneumonia type, and community-acquired pneumonia (CAP), hospital-acquired pneumonia (HAP), medical-associated pneumonia (healthcare-associated pneumonia, HeAP) and ventilator-associated pneumonia (VAP) can progress to SP. The timely and accurate diagnosis along with antimicrobial therapy and supportive care are vital for managing SP and improving patient outcomes. The available statistics underscore the importance of addressing this health challenge.

Endotracheal intubation (ETI) with mechanical ventilation is an effective, rapid and widely employed treatment for respiratory tract problems in SP patients [2]. It has role in airway protection and respiratory support. Advancements have improved the safety and efficacy of this procedure, particularly with the emergence of ETI visualization technology [3]. However, ETI is an invasive procedure and has complications such as airway injury, local hemorrhage, laryngeal edema, hypertension, hypotension and arrhythmia [4]. These complications in severe cases may create hemodynamic instability to threaten the patient lives.

Post-intubation hypotension (PIH) is a serious complication after ETI, and is linked with increased morbidity and mortality. It can lead to inadequate organ perfusion and multiple organ failure if not managed in time. There are limited studies addressing the risk factors associated with PIH in SP patients despite their importance in maintaining the hemodynamic stability during and after ETI. Understanding these risk factors can improve patient outcomes and optimize healthcare strategies.

This study investigated the risk factors associated with hypotension following the ETI in SP patients. The study outcomes could improve patient care and medical practices, and innovate the treatments. It could spur investigations into the pathophysiological mechanisms underlying PIH in SP patients for its broader understanding. Additionally, the findings could guide future studies to develop therapeutic interventions for severe pneumonia.

2. Methods

2.1 Study design and samples

The study was designed using retrospective case-control method. SP patients undergone ETI in the emergency department of our hospital from December 2019 to December 2022 were selected as the study subjects. Three years period was selected to limit the changes in intubation practice which might confuse the relationship between analyzed risk factors and postoperative hypotension. Inclusion criteria: (1) patient age >18 years; (2) SP diagnostic criteria: simplified diagnosis of Chinese adult severe pneumonia in 2015 as per The American Society of infectious diseases (IDSA) and the American Thoracic Society (ATS), IDSA/ATS standard: SP diagnosed by either meeting the major criteria or more than 3 secondary criteria; major criteria: ETI required mechanical ventilation; secondary criteria: respiratory rate >30/min; oxygenation index (OI) <250 mmHg; multiple lobar infiltration; disturbance in consciousness and/or disorientation; and blood urea nitrogen ≥7 mmol/L; and (3) hypotension after intubation: blood pressure decreased within 1 hour of intubation and its duration was above 10 min. Hypotension criteria referred to 2021 septic shock rescue guidelines: systolic blood pressure (SBP) <90 mmHg or SBP decreased by >40 mmHg or above in the absence of other hypotension causes [5]. Exclusion criteria: (1) age <18 years; (2) patients with hypotension before ETI; and (3) fluid resuscitation or vasoactive drugs before ETI. Ethics committee approved the study.

2.2 Definition of Post-intubation hypotension (PIH)

A consistent definition of postoperative hypotension was lacking. PIH in this study was defined based on literature review and expert opinions. PIH referred to a decrease in blood pressure shortly after ETI and initiation of mechanical ventilation and any vasopressor medication within 60 min of emergency intubation. This study defined PIH as: (1) mean arterial pressure (MAP) of <65 mmHg; (2) SBP of <80 mmHg or 40% decrease in SBP from baseline; and (3) increase in initiation or infusion rate of vasoactive drug in 60 min of ETI.

2.3 Data collection and outcome measurements

The participants data were obtained from the electronic medical records of our hospital. The demographic information was collected regarding age, weight, gender, collected usage of inducer before intubation (propofol and midazolam), chronic disease (hypertension, diabetes, COPD and chronic renal failure) and sepsis. Laboratory assessments were made pertaining to systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate, respiratory frequency, oxygen saturation, white blood cells (WBC), neutrophil count (N), hematocrit (Hct), hemoglobin (Hb), creatinine (Cr), albumin (Alb), serum potassium (K), blood sodium (Na), blood sugar, blood pH, oxygen partial pressure (PO_2), carbon dioxide partial pressure (PCO_2), lactate (Lac), and bicarbonate radical (HCO_3^-).

2.4 Statistical methods

Shapiro-Wilk test was employed to test the normal distributions of all continuous data. The variable following normal distribution was expressed as mean ± standard deviation. The difference was compared *via* independent sample *t* test, and expressed as median and quartile spacing (median (interquartile range)). Differences between the two groups of independent samples were compared by non-parametric test (Mann-Whitney test). The qualitative data were expressed as n (%), and Pearson chi-square test was employed to compare differences between the groups. The subjects were divided into hypotension and non-hypotension groups according to PIH occurrence. The age, body weight, chronic disease, inducer usage before intubation, and physical and chemical indexes before intubation were compared. Univariate and multivariate logistic regression analyses were performed to explore the independent related factors of hypotension. The statistical analyses were made with SPSS 22.0 software (IBM, Armonk, NY, USA). p value of < 0.05 was considered statistically significant.

3. Results

This study included 420 patients wherein 40.47% (170/420) experienced PIH. Patients were divided into hypotension (n = 170) and normal blood pressure (n = 250) groups based on whether PIH occurred. Table 1 revealed that the average age of patients in hypotension group (66.81 ± 16.09) was higher than that in non-hypotension (61.04 ± 17.56) with statistical difference ($t = -3.417$, $p = 0.001$). The patients' weight in hypotension group (58.72 ± 10.38) was lower than that in non-hypotension (61.19 ± 9.97) with statistical difference ($t = 2.457$, $p = 0.014$). The patients in hypotension group using inducers before intubation (31.2%) were higher than those in non-hypotension (20.4%) with statistical difference ($\chi^2 = 6.308$, $p = 0.012$). COPD patients in hypotension group (32.9%) were higher than those in non-hypotension (20.8%) with statistical difference ($\chi^2 = 7.809$, $p = 0.005$). The sepsis patients in hypotension group (47.1%) were higher than those in non-hypotension (34.8%) with statistical difference ($\chi^2 = 6.349$, $p = 0.012$). Other indexes depicted no statistical significance ($p > 0.05$).

Table 2 exhibited the comparison of two groups regarding physical and chemical indicators before the intubation. The lactate levels in hypotensive group (3.59 ± 2.41) were higher than those in non-hypotensive (2.77 ± 1.68) and the difference was statistically significant ($t = -3.835$, $p < 0.05$). Other physicochemical indicators had no difference for the two

TABLE 1. Comparison of baseline characteristics before intubation between the two groups.

Variables	hypotension group (n = 170)	non-hypotension group (n = 250)	t/χ^2	p
Age (yr)	66.81 ± 16.09	61.04 ± 17.56	−3.417	0.001
Weight (kg)	58.72 ± 10.38	61.19 ± 9.97	2.457	0.014
Male, n (%)	112 (65.9)	158 (63.2)	0.317	0.573
Use inducer before intubation, n (%)	53 (31.2)	51 (20.4)	6.308	0.012
Hypertension, n (%)	57 (33.5)	85 (34.0)	0.010	0.920
Diabetes, n (%)	42 (24.7)	53 (21.2)	0.711	0.399
Chronic obstructive pulmonary disease, n (%)	56 (32.9)	52 (20.8)	7.809	0.005
Chronic renal failure, n (%)	35 (20.6)	66 (26.4)	1.871	0.171
Sepsis, n (%)	80 (47.1)	87 (34.8)	6.349	0.012

Note: t, the statistical value of the independent sample t-test; χ^2, the statistical value of the chi-square test.

groups.

Table 3 depicted the univariate and multivariate logistic regression of whether postoperative hypotension occurred after intubation. The univariate logistic regression analysis revealed that age (OR = 1.021, p = 0.001), weight (OR = 0.976, p = 0.015), lactic acid (OR = 1.221, $p < 0.001$), inducer usage before intubation (OR = 1.768, p = 0.013), COPD (OR = 1.870, p = 0.006), and sepsis (OR = 1.665, p = 0.012) were associated with PIH. The multivariate logistic regression analysis indicated that age (OR = 1.015, p = 0.033), weight (OR = 0.974, p = 0.012), lactic acid (OR = 1.228, $p < 0.001$), inducer usage before intubation (OR = 1.660, p = 0.037), COPD (OR = 1.733, p = 0.035), and sepsis (OR = 1.679, p = 0.018) were the independent risk factors of PIH.

4. Discussion

Main finding of this study was that the postoperative hypotension was common, predictable and clinically important in SP patients. High age, low body weight, high lactate levels, inducers usage before intubation, COPD and sepsis were the risk factors of postoperative PIH.

Studies had shown the PIH incidence as 20%–52% [6, 7]. This study found that 40.47% (170/420) patients experienced PIH which was consistent with the previous studies. Tracheal intubation with mechanical ventilation was an important treatment for airway protection and respiratory support in SP patients. The morbidity and mortality of SP patients had increased with COVID-19 [8]. ETI was widely employed in rescuing such patients. It provided with the necessary time to treat patients by connecting ventilator for assistance in breathing. ETI was an invasive operation which might lead to airway injury, local bleeding, laryngeal edema, hypertension, hypotension, arrhythmia and other complications after intubation, especially upon hemodynamic instability [9].

Limited studies were available on the influencing factors of PIH in SP patients. This study showed that age was an independent risk factor after ETI in SP patients. Advanced age was associated with the physiological changes which increased the vulnerability in this population. With increasing age, there was decrease in pulmonary elasticity, respiratory muscle strength, and respiratory reserve capacity. The age related changes could impair the respiratory system to hamper the gases exchange and leading to decreased lung function. Moreover, aging was accompanied by the alterations in immune function. The activity of immune cells was declined which resulted in the impaired immune defense, immune self-stability, and immune surveillance [10]. This weakened immune response made elderly patients more susceptible to infections and less capable of immune defense against pathogens including those of severe pneumonia [11]. Furthermore, aging affected the cardiovascular function. Elderly individuals often had reduced cardiac output which decreased the blood supply to organs, slowed the blood flow, and impaired the microcirculation. This systemic decline in cardiac function combined with reduced capacity for compensatory responses could lead to hemodynamic instability in response to the stressors such as ETI [11]. Upcoming studies should strategize in reducing risks associated with tracheal intubation in elderly population such as optimizing pre-intubation assessments, utilizing alternative airway management techniques, and personalizing methods for ventilation and hemodynamic support.

This study had identified low weight as an independent risk factor for hypotension following ETI. This finding was contrary to the notion that higher body weight was often associated with negative health outcomes. The “obesity paradox” theory proposed that overweight and obese people with severe infections might have lower mortality rates compared to those with normal or low weight [12]. This study was aligned with the obesity paradox theory by suggesting that adipose tissue being abundant in higher body weight individuals had protective role of stabilizing blood pressure in severely infected patients [13]. The epicardial adipose tissue had physiological functions beyond its role as fat storage depot. It could synthesize and secrete bioactive molecules called adipokines [14, 15]. The adipokines exerted local effects on adjacent myocardium through paracrine pathway. The adipose tissue and secreted adipokines might provide protective effect during hemodynamic instability such as after ETI [16]. These molecules maintained blood pressure by influencing vascular tone, vascular remodeling and inflammatory processes [17]. This protective effect of adipose tissue might help in explaining

TABLE 2. hypotension *vs.* non-hypotension group, comparison of the differences of physical and chemical indexes before intubation.

Variables	Hypotension group (n = 170)	Non-hypotension group (n = 250)	*t*/*Z*	*p*
SBP (mmHg)	128.78 ± 33.32	126.35 ± 23.54	−0.820	0.413
DBP (mmHg)	74.62 ± 21.43	75.23 ± 17.10	0.310	0.757
Heart rate (beats/min)	121.51 ± 25.95	119.29 ± 28.49	−0.814	0.416
Respiratory frequency (beats/min)	33.04 ± 10.33	33.17 ± 9.93	0.126	0.899
SPO_2 (%)	85.49 ± 11.37	85.83 ± 11.96	0.291	0.771
WBC ($\times 10^9$/L)	10.52 (6.10, 15.52)	10.46 (7.06, 15.78)	−0.621	0.534
N ($\times 10^9$/L)	8.99 (5.29, 13.45)	9.07 (5.56, 13.37)	−0.472	0.637
Hct (L/L)	0.34 ± 0.08	0.33 ± 0.10	−1.000	0.318
Hb (g/L)	109.66 ± 28.74	105.44 ± 30.28	−1.434	0.152
Cr (μmol/L)	81 (55, 143)	88 (56, 156)	−0.701	0.483
Alb (g/L)	30.81 ± 6.55	29.85 ± 5.91	−1.571	0.117
K (mmol/L)	4.05 ± 0.92	3.96 ± 0.81	−0.968	0.334
Na (mmol/L)	135.32 ± 7.72	135.28 ± 10.83	−0.045	0.964
Blood sugar (mmol/L)	9.48 ± 4.51	9.49 ± 4.99	0.031	0.975
pH	7.331 ± 0.139	7.347 ± 0.134	1.072	0.284
PO_2 (mmHg)	69.2 (57.8, 89.9)	64.1 (55.5, 83.0)	−1.563	0.118
PCO_2 (mmHg)	39.4 (29.8, 62.7)	37.7 (30.0, 53.1)	−1.057	0.290
Lac (mmol/L)	3.59 ± 2.41	2.77 ± 1.68	−3.835	<0.001
HCO_3^-	23.92 ± 9.74	22.62 ± 8.13	−1.283	0.201

Note: t, the statistical value of the independent sample t-test; the statistical value of Z, Mann-Whitney Test.
Abbreviations: SBP, Systolic blood pressure; DBP, diastolic blood pressure (mmHg); SPO_2, Pulse Oxygen Saturation; WBC, White blood cell; N, Neutrophils; Hct, Hematocrit; Hb, Hemoglobin; Cr, Creatinine; Alb, albumin; K, Serum potassium; Na, Blood sodium; PO_2, Oxygen partial pressure; PCO_2, Partial pressure of carbon dioxide; Lac, lactic acid; HCO_3^-, Bicarbonate radical.

TABLE 3. Univariate and multivariate logistics regression analysis of hypotension after intubation.

Variables	Univariate analysis		Multivariate analysis	
	OR (95% CI)	*p* value	OR (95% CI)	*p* value
Age	1.021 (1.008, 1.033)	0.001	1.015 (1.001, 1.029)	0.033
Weight	0.976 (0.957, 0.995)	0.015	0.974 (0.954, 0.994)	0.012
lactic acid	1.221 (1.104, 1.351)	<0.001	1.228 (1.103, 1.367)	<0.001
Use inducer before intubation	1.768 (1.130, 2.764)	0.013	1.660 (1.032, 2.671)	0.037
Chronic obstructive pulmonary disease	1.870 (1.202, 2.911)	0.006	1.733 (1.039, 2.892)	0.035
Sepsis	1.665 (1.119, 2.480)	0.012	1.679 (1.092, 2.583)	0.018

OR: Odd Ratio; CI: Confidence Interval.

the lower PIH incidence in higher body weight patients. More studies were required to understand the underlying mechanisms of relationships between weight, adipose tissue and hemodynamic stability.

This study provided evidence that elevated lactate levels after tracheal intubation in SP patients and concurrent sepsis were associated with higher mortality. Lactic acid was an important biomarker used in sepsis treatment [5]. In SP, elevated lactate levels were caused by the factors including decreased oxygen saturation, tissue hypoxia, mitochondrial dysfunction, and increased anaerobic glycolysis [18]. The relationship between lactic acid and severe infection was not fully understood whether it acted as a cause or consequence. Brooks proposed that hyperlactatemia was a manifestation of reduced systemic perfusion [19]. It might play a protective role during cytokine storm, however hyperlactatemia in later stages of severe infection could be a pathological factor exacerbating the disease [20]. Moreover, lactate and lactate ions inhibited the cellular metabolism and immune cell function induced by lipopolysaccharides which impaired the antimicrobial de-

fense mechanisms [21]. Acidosis had also hindered pathogens clearance such as Streptococcus pneumoniae and Escherichia coli. The current clinical observations supported the theory that high lactic acid levels in early stages of sepsis might inhibit the immune cell glycolysis and function. However, this negative regulation was detrimental as it impaired the pathogen clearance and contributed to secondary immunosuppression in sepsis [22–25]. Previous studies being consistent with this study demonstrated that in hemodynamically stable patients with severe infections, blood pressure decreased when blood lactate levels exceeded 4 mmol/L [26]. The association between increased lactate levels and mortality highlighted the importance of monitoring lactate levels in the clinical management of SP and sepsis [27]. Successive lactate measurements could provide information of patient's response to treatment and guide therapeutic interventions. Strategies of optimizing tissue oxygenation, improving perfusion, and addressing underlying infection could reduce lactate levels and improve patient outcomes.

This study revealed that COPD in SP patients was an independent risk factor for PIH. COPD was characterized by persistent dyspnea and airflow limitation. Smoking was a contributing factor to COPD development [28]. Long-term smoking could lead to cellular damage, decreased production of pulmonary surfactant, impaired ciliary function, imbalance between proteases and antiproteases, vascular endothelial damage, reduced secretion of nitric oxide (NO) and endothelin (ET), and impaired vasodilation and vasoconstriction. These factors increased the hypertension risk in COPD individuals [29]. It was observed in this study that SP patients with COPD history were more susceptible to HIP. This increased risk could be attributed to the factors including impaired circulatory function during intubation, infection, steroids usage, and drug-induced dysfunction of adrenal cortex [30]. However, the specific mechanisms underlying these relationships required further investigations. The coexistence of COPD and SP could result in complex clinical presentation and response to interventions. COPD complicated the SP management, and interaction between these two conditions contributed to the heightened PIH risk [31]. Further studies were required to understand the underlying mechanisms and to develop strategies of minimizing the hypotension occurrence in this patient population.

This study addressed the risk factors associated with PIH development in SP patients. This was considered a new topic in this area of research. Previous studies had focused on the efficacy and safety of intubation by the emergency physicians. Therefore, complications after intubation such as hypoxemia, hypotension, and arrhythmia were focused. Few studies, if any, had worked on the risk factors of PIH in SP patients. Results herein indicated that PIH development was a multifactorial model for SP patients. However, this study had some limitations. First, it was a retrospective study that could not draw causal conclusions about PIH in SP patients. Second, the data source was electronic medical record of our hospital. There was missing data like measurements of heart function, right ventricular function, PEEP anesthesia, corticosteroid usage, *etc*. These important indicators would be considered in future studies.

5. Conclusions

This study finds that high age, low body weight, high lactate levels, inducers usage before intubation, COPD and sepsis are the risk factors of postoperative hypotension after intubation. In future clinical practice, when ETI is performed in SP patients, the hypotension is likely to occur after intubation if there are risk factors such as advanced age, low body weight, inducers usage before intubation, increased lactic acid levels, COPD and sepsis. For such high risk patients, signs and hemodynamic changes should be monitored, and timely intervention be carried out.

AVAILABILITY OF DATA AND MATERIALS

The data presented in this study are available on reasonable request from the corresponding author.

AUTHOR CONTRIBUTIONS

YC and PP—designed the research study; wrote the manuscript. PP—performed the research. PP, QH, YWY and TC—analyzed the data. All authors read and approved the final manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study was approved by the Biomedical Ethics Review Committee of West China Hospital of Sichuan University, Grant number: 493 in 2022. All participants provided consent to participate in the study. All methods were performed according to the relevant guidelines and regulations.

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Not applicable.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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