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Original Contribution

Sedative dose and patient variable impacts on postintubation hypotension in emergency airway management

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ABSTRACT

Purpose: Postintubation hypotension (PIH) is an adverse event associated with poor outcomes in emergency department (ED) endotracheal intubation. This study aimed to evaluate the association between sedative dose adjustment and PIH during emergency airway management. We also investigated the impact of patient and procedural factors on the incidence of PIH.
Materials and methods: This was a single-center, retrospective study that used a prospectively collected registry of airway management performed at the ED from April 2014 to February 2017. Adult patients who received emergency endotracheal intubation were included. Multivariable logistic regression models were used to evaluate the association of PIH with sedative dose, patient variables, and procedural variables.
Results: Overall, 689 patients were included, and 233 (33.8%) patients developed PIH. In the patients overall, multivariable logistic regression demonstrated that age > 70 years, shock index >0.8, arterial acidosis (pH < 7.2), intubation indication, and use of non-depolarizing neuromuscular blocking agent were significantly related to PIH. In patients overall, the sedative dose was not related to PIH (overdose; OR: 1.09, 95%CI: 0.57–2.06), (reduction; OR: 0.93, 95%CI: 0.61–1.42), (none used; OR: 1.28, 95%CI: 0.64–2.53). In subgroup analysis, ketamine dose was not related to PIH (overdose; OR: 0.81, 95%CI: 0.27–2.38, reduction; OR: 1.41, 95%CI: 0.78–2.54). Reduction of etomidate dose was significantly associated with decreased PIH (reduction; OR: 0.46, 95%CI: 0.22–0.98, overdose; OR: 1.77, 95%CI: 0.79–3.93).
Conclusions: PIH was mainly related to predisposing patient-related factors. Only adjustment of etomidate dose was associated with the incidence of PIH.

1. Introduction

Critically ill patients who visit the emergency department (ED) often require emergency airway management due to various indications, including respiratory failures in oxygenation or ventilation, airway obstruction, metabolic derangements, and a requirement for airway protection. Endotracheal intubation (ETI) is an essential, life-saving procedure for critically-ill or injured patients who require a definitive airway in the ED. Rapid sequence intubation (RSI), which involves administration of a potent sedative followed by a rapidly acting neuromuscular blocking agent to increase the success rate and reduce adverse events, is a widely used technique to facilitate ETI in the ED [1-3].

During or immediately after RSI, various complications including hypotension, hypoxia, arrhythmia, and death can occur [4-10]. Among these, postintubation hypotension (PIH) is known to be the most common undesirable hemodynamic change [11]. Previous studies in the ED and intensive care unit (ICU) have reported that hypotension after endotracheal intubation is associated with an increased incidence of acute myocardial infarction, renal failure, increased duration of hospitalization, and mortality [12-17].

According to previous studies on the risk factors of PIH, hypotension before endotracheal intubation, use of neuromuscular blocking agent, pre-intubation shock index, chronic renal failure, endotracheal intubation indications (respiratory failure), diabetes, chronic obstructive pulmonary disease, sepsis, albumin level, body weight, and age are related to PIH [13-15,18,19]. However, most of the risk factors identified in these previous studies are difficult to modify prior to ETI, and studies for deriving strategies to reduce the incidence of PIH are lacking.

Considering that the dose of the drug administered for RSI can be adjusted at the time of endotracheal intubation, this study aimed to

Abbreviations: ED, emergency department; PIH, Postintubation hypotension; CI, confidence interval.

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investigate whether the dose of sedative used during endotracheal intubation could affect PIH incidence and to identify clinical factors that affect PIH.

2. Subject and methods

2.1. Study subjects

Adult (≥19 years old) ED patients who received emergency endotracheal intubation at a tertiary hospital ED (annual ED patients of approximately 70,000) from April 1, 2014 to February 28, 2017 (2 years and 11 months) were included. Patients who received endotracheal intubation due to cardiac arrest or for whom incomplete data were available were excluded.

This study was approved by the relevant institutional review board, and the need to obtain informed consent was waived due to the retrospective nature of this study.

2.2. Study method and data collection

2.2.1. Data collection

For this study, data were collected by retrospectively reviewing medical records. Data from the Airway Quality Improvement (QI) registry, which was prospectively collected from April 2014 for quality improvement of endotracheal intubation, was analyzed. Endotracheal intubation-related information was collected, and an airway management QI team acted to improve the success rate of endotracheal intubation and reduce complications. One independent emergency medical technician used a checklist to collect data for each attempted endotracheal intubation. After endotracheal intubation was performed, the operator completed the registry by filling in additional information. The registry included the patient's age, sex, height, weight, endotracheal intubation indication, vital signs before and after endotracheal intubation, and the medication and doses used for endotracheal intubation. Pre-intubation vital signs were collected at the time of attempted intubation. Post-intubation vital signs were collected for 30 min after intubation. The shock index prior to intubation was computed based on the equation of pulse rate/systolic blood pressure (sBP).

The primary endpoint of this study was PIH; this was defined as sBP < 90 mmHg or mean blood pressure (mBP) < 65 mmHg, requiring vasopressor administration or an increased dose of vasopressor, or a decrease in BP by >20%, using the lowest vital signs during 30 min after intubation. PIH was classified into five categories, as summarized in Table 1.

2.2.2. Endotracheal intubation process

Endotracheal intubation was performed in the ED by an emergency medicine resident or attending physicians, and other residents from the departments of internal medicine, neurology, general surgery, neurosurgery, thoracic surgery, otorhinolaryngology, intensive care medicine, family medicine, and anesthesiology participated. The standard RSI process was followed. The preoxygenation target was peripheral oxygen saturation (SpO_2) of >90%; patients were categorized on the basis of SpO_2 after at least 3 min of oxygen supply with a facial mask or bag-mask (15 L/min) at the time of decision to intubate: 1) low risk, SpO_2 > 95%; 2) high-risk, SpO_2 91–95%; 3) hypoxemic, SpO_2 < 91%. Noninvasive positive pressure ventilation and apneic oxygenation were considered in high-risk or hypoxemic patients. Depending on the patient, pre-treatment drugs, sedative, and neuromuscular blocking agents were administered prior to endotracheal intubation. The sedative dose was determined by the attending doctor, based on the recommended dose for the patient's hemodynamic status and age. The recommended dose for sedatives were as follows: etomidate 0.3 mg/kg, ketamine 1.5 mg/kg, and midazolam 0.1 mg/kg. The sedative dose was categorized as a dose reduction when <75%, as overdose when >125%, and as standard dose when >75% but <125%, relative to the recommended dose. For neuromuscular blocking agents, succinylcholine (1–1.5 mg/kg), rocuronium (1 mg/kg), and vecuronium (0.1 mg/kg) were used. For sedation after intubation, remifentanil, midazolam, and fentanyl were continuously administered.

Table 1
Postintubation hypotension categories.

(1) Before intubation[a]: sBP[b] ≥ 90 and mBP[c] ≥ 65 without vasopressor
After intubation[d]: sBP < 90 or mBP < 65 or vasopressor use
(2) Before intubation[a]: Any BP[e] with vasopressor
After intubation[d]: Any BP[e] with increased dose of vasopressor
(3) Before intubation[a]: sBP ≥ 90 and mBP ≥ 65 with vasopressor
After intubation[d]: sBP < 90 or mBP < 65 with the same dose of vasopressor
(4) Before intubation[a]: sBP < 90 or mBP < 65 without vasopressor
After intubation[d]: reduction of 20% of sBP or mBP without vasopressor
(5) Before intubation[a]: sBP < 90 or mBP < 65 without vasopressor
After intubation[d]: Any BP[e] with vasopressor use

[a] Blood pressure before intubation was that recorded at the time of intubation.
[b] sBP: systolic blood pressure.
[c] mBP: mean blood pressure.
[d] Blood pressure after intubation was the lowest value within 30 min of intubation, or the nearest record if no record within 30 min was available.
[e] BP: blood pressure.

2.3. Statistical analysis

For statistical analysis, STATA version 14.0 (Stata Corp LP, College Station, TX, USA) was used. Continuous variables are presented as means and standard deviation, and Student's *t*-test was used for comparisons. Categorical variables are presented as numbers and percentages, and the chi-square test was used for comparison. In order to investigate the association with PIH, univariate regression was performed for age, weight, body mass index (BMI), intubation indication, shock index, arterial acidosis, type of neuromuscular blocking agent, pre-treatment drug use, pre-intubation hypoxia, and type and dose of sedative, and results are presented as odds ratio (ORs) and 95% confidence intervals (CIs). For multivariable logistic regression models, sedative dose and variables with p values <0.1 in the univariate analysis were included. In order to analyze subgroups based on the drug type, the incidence of PIH according to drug dose was compared between the ketamine and etomidate group, and multivariate regression analysis was performed using adjustment for variables identified as statistically significant in univariate regression analysis. p values <0.05 were considered statistically significant.

3. Results

3.1. Patient characteristics

During the study period, from April 1, 2014 to February 28, 2017, a total of 1087 endotracheal intubations were performed in the ED. Among them, 398 cases, including 373 intubations performed due to cardiac arrest and 25 cases with incomplete data, were excluded; thus, a total of 689 intubations were analyzed. Of these 689 cases, 233 cases (33.8%) developed PIH, and according to the predefined PIH criteria (Table 1), 144 cases (20.9%) were classified as category (1), 43 cases (6.2%) as category (2), 8 cases (1.2%) as category (3), 6 cases (0.9%) as category (4), and 32 cases (4.6%) as category (5).

The baseline characteristics of the patients are shown in Table 2. The mean age was 64.1 ± 15.9 years, and most patients (61.4%) were male. The intubation indications were primarily for decreased level of consciousness (32.4%) and respiratory failure (52.0%). Most commonly used sedatives during endotracheal intubation were etomidate (49.6%) and ketamine (35.1%). There was a significant difference between the PIH and non-PIH groups in terms of age, weight, BMI, shock index prior to intubation, arterial blood pH, intubation indication, types of sedative and neuromuscular blocking agent used, fentanyl

Table 2
Baseline characteristics.

	Total (n = 689)	PIH[a] (n = 233)	Non-PIH[a] (n = 456)	p value
Age (years)	64.1 ± 15.9	67.5 ± 14.6	62.3 ± 16.3	<0.001
Age > 70	292 (42.4%)	121 (51.9%)	171 (37.4%)	<0.001
Sex (male)	423 (61.4%)	144 (61.8%)	279 (61.2%)	0.875
Weight (kg)	62.8 ± 13.7	60.2 ± 12.5	64.2 ± 14.1	<0.001
Weight < 60 kg	249 (36.1%)	99 (42.5%)	150 (32.9%)	0.013
BMI[b] (kg/m^2)				0.019
Underweight (<18.5 kg/m^2)	83 (12.1%)	39 (16.7%)	44 (9.7%)	
Normal weight (18.5–25 kg/m^2)	459 (66.6%)	151 (64.8%)	308 (67.5%)	
Overweight (>25 kg/m^2)	147 (21.3%)	43 (18.5%)	104 (22.8%)	
Intubation indication				<0.001
Altered mental status	223 (32.4%)	53 (22.8%)	170 (37.3%)	
Respiratory failure	350 (50.1%)	126 (54.1%)	222 (49.1%)	
Shock	49 (7.1%)	28 (12.0%)	21 (4.6%)	
Drug intoxication	13 (1.9%)	6 (2.6%)	7 (1.5%)	
Airway obstruction	8 (1.2%)	5 (2.2%)	3 (0.7%)	
Trauma	34 (4.9%)	9 (3.9%)	25 (5.5%)	
Others	12 (1.8%)	6 (2.6%)	6 (1.3%)	
Shock index	0.87 ± 0.39	1.04 ± 0.44	0.78 ± 0.33	<0.001
Shock index > 0.8	356 (52.9%)	155 (69.8%)	201 (44.6%)	<0.001
Arterial acidosis (pH < 7.2)	148 (21.5%)	64 (27.5%)	84 (18.4%)	0.006
Sedative				0.003
Ketamine	242 (35.1%)	99 (42.5%)	143 (31.4%)	
Etomidate	342 (49.6%)	95 (40.8%)	247 (54.2%)	
Others	45 (6.5%)	13 (5.6%)	32 (7.0%)	
None	60 (8.7%)	26 (11.2%)	34 (7.5%)	
NMB[c]				<0.001
Succinylcholine	435 (63.1%)	154 (35.4%)	281 (64.6%)	
Non-depolarizing NMB[c]	184 (26.7%)	43 (23.4%)	141 (76.6%)	
None	70 (10.2%)	36 (51.4%)	34 (48.6%)	
Pretreatment (fentanyl)	75 (10.9%)	17 (7.30%)	58 (12.7%)	0.031
Post-intubation continuous sedative	241 (35.0%)	87 (37.3%)	154 (33.8%)	0.353
Low SpO_2 before intubation[d]	198 (28.7%)	83 (35.6%)	115 (25.2%)	0.004
Sedative dose				0.281
Overdose (>125%)	61 (8.9%)	23 (9.9%)	38 (8.3%)	
Standard (75%–125%)	342 (49.6%)	107 (45.9%)	235 (51.5%)	
Reduction (<75%)	226 (32.8%)	77 (33.1%)	149 (32.7%)	
No use	60 (8.7%)	26 (11.2%)	34 (7.5%)	
Multiple attempts of intubation, ≥3 times	53 (7.7%)	18 (7.7%)	35 (7.7%)	0.981
In-hospital mortality (%)	233 (33.8%)	93 (49.2)	140 (28.0)	<0.001

[a] PIH: postintubation hypotension.
[b] BMI: body mass index.
[c] NMB: neuromuscular blocking agent.
[d] SpO_2 was <90% at the time of intubation, although the preoxygenation method was applied.

administration status, and SpO_2 below 90% prior to intubation. There was no statistically significant difference between the PIH and non-PIH groups in terms of the distribution of the sedative dose used during intubation (p = 0.281). In-hospital mortality was significantly higher in the PIH group (49.2%), compared with the non-PIH group (28.0%) (p < 0.001).

3.2. Multivariable logistic regression analysis for variables associated with PIH

In multivariate logistic regression analysis, after adjusting for confounding variables, dose reduction (adjusted OR: 0.93, 95% CI: 0.61–1.42, p = 0.735) or overdose (adjusted OR: 1.09, 95% CI: 0.57–2.06, p = 0.794) of sedative, as compared to the standard dose, and no use of sedative (adjusted OR: 1.28, 95% CI: 0.64–2.53, p = 0.483) demonstrated no significant association with PIH (Table 3). However, the following clinical factors were associated with PIH after endotracheal intubation: age over 70 (adjusted OR: 2.16, 95% CI: 1.50–3.10, p < 0.001), shock index over 0.8 (adjusted OR: 2.43, 95% CI: 2.05–4.05, p < 0.001), arterial blood pH below 7.2 (adjusted OR: 1.58, 95% CI: 1.04–2.41, p = 0.033), use of a non-depolarizing neuromuscular blocking agent (adjusted OR: 0.53, 95% CI: 0.34–0.81, p = 0.003).

3.3. Analysis according to the type of sedative used

For the most commonly used drugs, ketamine and etomidate, an additional analysis was performed to assess association between the drug dose and PIH (Tables 4, 5). In the group that used ketamine, the mean dose was 1.20 ± 0.54 mg/kg. Reduced dose (adjusted OR: 1.41, 95% CI: 0.78–2.54, p = 0.261) or overdose (adjusted OR: 0.81, 95% CI: 0.27–2.38, p = 0.697) of ketamine was not significantly associated with PIH. In the group that used etomidate, the mean dose was 0.29 ± 0.23 mg/kg. Reduced dose (adjusted OR: 0.46, 95% CI: 0.22–0.98, p = 0.044) of etomidate was statistically significantly associated with a decreased incidence of PIH.

4. Discussion

Endotracheal intubation is a core, life-saving technique in the treatment of critically ill patients. In a considerable number of patients, hypotension that can affect the patient's prognosis may occur after endotracheal intubation. In this study, the incidence of PIH was 33.8%, which is similar to that of previous studies [11-17,19,20]. Reduction or increase of the sedative dose prior to intubation did not show a significant association with PIH in the patients overall, but in the etomidate group, dose reduction was significantly associated with decreased PIH

Table 3
Logistic repression analysis for postintubation hypotension.

Variables	Univariable analysis			Multivariable analysis		
	OR[a]	95% CI[b]	p value	OR[a]	95% CI[b]	p value
Age > 70	1.80	1.31–2.48	<0.001	2.16	1.50–3.10	<0.001
Weight < 60 kg	1.51	1.09–2.09	0.013	1.11	0.71–1.73	0.652
BMI[c]						
Normal weight (18.5–25 kg/m^2)	Reference					
Underweight (<18.5 kg/m^2)	1.81	1.13–2.90	0.014	1.24	0.68–2.23	0.482
Overweight (>25 kg/m^2)	0.84	0.56–1.26	0.410	0.94	0.59–1.52	0.814
Intubation indication						
AMS[d]	Reference					
Respiratory failure	1.85	1.27–2.70	0.001	1.20	0.78–1.86	0.409
Others	2.66	1.63–4.34	<0.001	2.43	1.39–4.24	0.002
Shock index >0.8	2.88	2.05–4.05	<0.001	2.67	1.84–3.87	<0.001
Arterial acidosis (pH < 7.2)	1.68	1.16–2.43	0.007	1.58	1.04–2.41	0.033
Sedative						
Etomidate	Reference			Reference		
Ketamine	1.80	1.27–2.55	0.001	1.48	0.98–2.24	0.065
Others	1.06	0.53–2.10	0.876	1.10	0.49–2.49	0.810
None	0.39	1.13–3.49	0.017			
NMB[e]						
Succynylcholine	Reference					
Non–depolarizing NMB[e]	0.56	0.38–0.83	0.004	0.53	0.34–0.81	0.003
None	1.93	1.16–3.21	0.011	1.48	0.81–2.70	0.199
Pretreatment (fentanyl)	0.54	0.31–0.95	0.033	0.67	0.34–1.29	0.227
Post–intubation sedative	1.17	0.84–1.62	0.353			
Low SpO_2 before intubation	1.64	1.17–2.31	0.004	1.35	0.92–1.98	0.128
Sedative dose						
Standard (75%–125%)	Reference					
Overdose (>125%)	1.33	0.75–2.34	0.324	1.09	0.57–2.06	0.794
Reduction (<75%)	1.13	0.79–1.62	0.488	0.93	0.61–1.42	0.735
No use	1.68	0.96–2.94	0.069	1.28	0.64–2.53	0.483

[a] OR: odds ratio.
[b] CI: confidence intervals.
[c] BMI: body mass index.
[d] AMS; Altered mental status.
[e] NMB: neuromuscular blocking agent.

incidence. Patient baseline characteristics, such as hemodynamic status prior to intubation were more strongly associated with PIH.

Hypotension in endotracheal intubation can be explained by the effect of the administered drug and positive pressure ventilation. Notably, positive pressure ventilation can directly or indirectly cause PIH. Medications used during intubation could decrease or block sympathetic outflow. Moreover, they directly affect the cardiovascular system [11,21]. Previously, such phenomena were considered to be a temporary and benign process, but recently, its association with poor prognosis has been confirmed through a number of studies [12-17]. The present study showed an association between a reduced etomidate dose (as compared to the recommended dose, based on the patient's weight), and a decreased incidence of PIH. This seems reasonable considering the mechanism of PIH. Furthermore, it suggests that an appropriate reduction of etomidate dose according to the patient's condition may be a strategy for preventing PIH.

Unlike etomidate, the ketamine dose used was not significantly associated with PIH, and a tendency for a relatively higher PIH incidence in the ketamine group was observed. This may be due to the following factors. First, ketamine is a sedative with relatively stable hemodynamics, as compared to other drugs, due to its sympathetic nerve system stimulation. Based on our additional analysis, the ketamine group showed a significantly higher shock index than the etomidate group, and its use was more frequent in hemodynamically unstable patients. Second, for airway management QI, it has been recommended to reduce the sedative dose in hemodynamically unstable patients; thus, the dose reduction group tended to have unstable hemodynamics prior to intubation. In fact, the PIH group had received a lower dose of ketamine. Considering that previous studies reported ketamine administration to cause considerable PIH, the results of this study cannot be interpreted as indicating no association between ketamine dose adjustment and PIH.

Table 4
Comparisons of ketamine and etomidate doses according to the occurrence of postintubation hypotension.

Ketamine	Total (n = 242)	PIH[a] (n = 99)	Non-PIH[a] (n = 143)	p value
Dose (mg/kg)	1.20 ± 0.54	1.13 ± 0.49	1.25 ± 0.57	0.035
Overdose (>125%)	22	7 (31.8%)	15 (68.2%)	0.461
Standard (75%–125%)	91	35 (38.5%)	56 (61.5%)	
Reduction (<75%)	129	57 (44.2%)	72 (55.8%)	
Etomidate	**Total (n = 342)**	**PIH[a] (n = 95)**	**Non-PIH[a] (n = 247)**	**p value**
Dose (mg/kg)	0.30 ± 0.20	0.31 ± 0.10	0.29 ± 0.23	0.787
Overdose (>125%)	35	16 (45.7%)	19 (54.3%)	0.008
Standard (75%–125%)	237	67 (28.3%)	170 (71.7%)	
Reduction (<75%)	70	12 (17.1%)	58 (82.9%)	

Values with % show the proportion of PIH or non-PIH cases.
[a] PIH: postintubation hypotension.

Table 5
Results of logistic repression analysis for postintubation hypotension in subgroups according to ketamine and etomidate use.

	Univariable analysis			Multivariable analysis		
	OR[a]	95% CI[b]	p value	OR[a]	95% CI[b]	p value
Ketamine						
Standard (75%–125%)	Reference					
Overdose (>125%)	0.75	0.28–2.01	0.564	0.81	0.27–2.38	0.697
Reduction (<75%)	1.27	0.73–2.19	0.397	1.41	0.78–2.54	0.261
Etomidate						
Standard (75%–125%)	Reference					
Overdose (>125%)	2.14	1.04–4.40	0.039	1.77	0.79–3.93	0.163
Reduction (<75%)	0.52	0.27–1.04	0.064	0.46	0.22–0.98	0.044

[a] OR: odds ratio.
[b] CI: confidence intervals.

This study identified clinically important PIH risk factors. First, the association between older age and PIH is consistent with the findings of previous studies, and may be due to the higher possibility of comorbidities and lower hemodynamic reservoir with increasing age [13,14].

Shock index, which considers blood pressure and pulse prior to endotracheal intubation, is known to be an important predictor of PIH, and it was also identified as a significant factor in this study [13]. Arterial acidosis was also significantly correlated with PIH, and is likely to be related to decreased cardiac contractility, arterial vasodilation, and decreased catecholamine response. Additional studies regarding PIH prevention strategies, such as appropriately adjusting the sedative dose or rapidly correcting the aforementioned PIH risk factors prior to attempted intubation, including vasopressor administration, fluid challenge, and acidosis correction, are necessary.

The results of this study showed that the incidence of PIH was lower in the non-depolarizing neuromuscular blocking agent group than in the succinylcholine group. This is in contrast to the theory that the vasodilatory effect of non-depolarizing neuromuscular blocking agents may cause blushing, with lowering of blood pressure and increasing of the pulse rate [22]. Additional research comparing depolarizing and non-depolarizing neuromuscular blocking agents in terms of PIH incidence is necessary. Furthermore, there is still controversy regarding the use of neuromuscular blocking agents and PIH. Studies by Heffner et al. and Greene et al. have reported that the use of neuromuscular blocking agents decreased hemodynamic instability [13,14], but a study by Smischney et al. reported an association between the use of neuromuscular blocking agents and PIH [15].

The limitations of this study are as follows. First, this study was a single-center, observational study, and thus, the results cannot be generalized and should be cautiously interpreted. In particular, the drugs administered for endotracheal intubation can vary, depending on the preference of the operator and medical institution. Second, most data were prospectively collected, but the incidence of PIH was investigated by retrospectively reviewing the medical records. We collected vital signs immediately after intubation, but they were not recorded with a predefined interval. Third, the criteria used to define PIH in this study were broader than those used in previous studies. The definition of PIH has not yet been firmly established and has been reported to vary from 19% to 52% [12-17,19,20]. Heffner et al. conducted a study in which patients with confirmed hypotension prior to endotracheal intubation were excluded [13,17], while Green et al. investigated the incidence of PIH in a broader sense by including patients whose blood pressure was decreased by >20% and patients who required vasopressor or required increased dose of vasopressor [12,14,16]. The results of this study may have been different if PIH had been defined differently. Fourth, this study investigated the sedative type and dose based on the administered dose clinically determined by the endotracheal intubation operator. Thus, although multivariable regression models were used to correct the confounding variables as much as possible, bias may have affected the results. Intubation-related factors, including the time to successful intubation or preoxygenation, were not included. Fifth, the sedative dose adjustment range was not segmented and analyzed in subgroups due to the small number of samples. Sixth, we did not evaluate the association between mortality and PIH. Seventh, we used patient-related variables as adjusting factors rather than the experience of the physician performing the intubation; however, there might have been a strong selection bias in that senior physicians were more often responsible for additional analysis of patients with unstable vital signs, and in that attending EM physicians supervised every intubation procedure to ensure adherence to standardized protocols during the airway QI program.

5. Conclusion

Among sedatives used during endotracheal intubation, dose reduction of etomidate had a significant association with decreased PIH. In addition, the clinical condition of the patient prior to intubation, including age, shock index, and arterial acidosis were risk factors of PIH. Based on these findings, additional research on preventive strategies for reducing PIH prevention is necessary.

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Previous presentations

None.

Conflicts of interest

The authors have no potential conflicts of interest or funding sources to declare.

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