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Risk factors for ventilator-associated pneumonia in trauma patients with torso injury: a retrospective single-center study

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Abstract

Objective: We aimed to identify the risk factors for ventilator-associated pneumonia in patients admitted to critical care after a torso injury.

Methods: We retrospectively evaluated 178 patients with torso injury aged >15 years who were intubated in the emergency room and placed on a mechanical ventilator after intensive care unit (ICU) admission, survived for >48 hours, had thoracic and/or abdominal injuries, and had no end-stage renal disease. We compared clinico-laboratory variables between ventilator-associated pneumonia (n = 54, 30.3%) and non-ventilator-associated pneumonia (n = 124, 69.7%) groups. Risk factors for ventilator-associated pneumonia were assessed using multivariable logistic regression analysis.

Results: Ventilator-associated pneumonia was associated with a significantly longer stay in the ICU (11.3 vs. 6.8 days) and longer duration of mechanical ventilation (7 vs. 3 days). Injury Severity

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Score (adjusted odds ratio [AOR]: 1.048; 95% confidence interval [CI]: 1.008–1.090), use of vasopressors (AOR: 2.541; 95% CI: 1.121–5.758), and insertion of a nasogastric tube (AOR: 6.749; 95% CI: 2.397–18.999) were identified as independent risk factors of ventilator-associated pneumonia.

Conclusion: Ventilator-associated pneumonia in patients with torso injury who were admitted to the ICU was highly correlated with Injury Severity Score, use of vasopressors, and insertion of a nasogastric tube.

Keywords

Intensive care, risk factor, torso injury, ventilator-associated pneumonia, Injury Severity Score, retrospective study

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Introduction

Ventilator-associated pneumonia (VAP) is the most common hospital-acquired infection among critically ill patients requiring mechanical ventilation.[1] Trauma itself has been reported as an independent risk factor for VAP in large cohort studies.[2,3] Among intubated patients in the intensive care unit (ICU), patients with trauma have a four-fold higher rate of VAP than do non-trauma patients.[4] Many studies have shown that trauma-related factors, such as the Injury Severity Score (ISS), pulmonary contusion, rib fracture, sternal fracture, spinal cord injury, and traumatic brain injury, are associated with the development of VAP.[5–7]

VAP is defined as pneumonia in a patient who is on mechanical ventilation for more than 48 hours, with radiological evidence of new or progressive infiltrate, symptomatic evidence of systemic infection, and laboratory detection of a causative agent.[8] Although the standard diagnostic criteria for VAP remain under debate,[9] VAP is associated with the requirement for prolonged ventilatory support, ICU admission, hospital length of stay, and increased health care costs.[4,10]

The identification of VAP risk factors in patients with trauma is important for risk stratification and the provision of optimal ICU treatment. However, although many studies have reported the incidence and risk factors for VAP in patients with trauma who are admitted to critical care,[5–7,11,12] there is marked heterogeneity regarding patient characteristics and trauma centers among such studies. To address these issues, we aimed to identify the risk factors for VAP in patients admitted to the ICU after a torso injury at a single trauma center.

Methods

Study design and patients

This study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). The study was approved by the Institutional Review Board of Chungbuk National University Hospital (approval number: 2020-02-005). Individual consent for this retrospective analysis was waived because all patient details were de-identified, and the data were handled confidentially. The reporting of this study

conforms to The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) Statement.[13]

This retrospective observational cohort study was conducted at a single, level I trauma center at Chungbuk University Hospital, South Korea. Among patients with trauma who were admitted to the ICU after thoracic and/or abdominal injury between January 2016 and December 2019, we evaluated those who met the following inclusion criteria: intubation in the emergency room and mechanical ventilation at least 48 hours after ICU admission, age >15 years, having survived for >48 hours, and no end-stage renal disease.

Patients were divided into two groups according to whether they were diagnosed with VAP. The diagnosis of VAP was determined according to radiologic evidence of new or progressive infiltrate of more than 48 hours and laboratory detection of a causative agent.[8] The management and diagnosis of all patients was performed by a trauma specialist.

Study variables and definitions

Patients' baseline characteristics included age; sex; underlying disease; Glasgow Coma Scale score; and trauma-related variables, such as the ISS, Revised Trauma Score (RTS), Abbreviated Injury Scale (AIS) score, and mechanism of injury. We analyzed clinical and laboratory variables collected after arrival to the emergency room. Hypotension was defined as systolic blood pressure <90 mmHg. Injuries such as hemothorax, pneumothorax, rib fracture(s), abdominal solid organ injury, intestinal injury, and thoracolumbar spine fracture (s) were identified using computed tomography images. Abdominal solid organ injury was defined as injury of the liver, spleen, pancreas, adrenal glands, kidneys and urinary tract, or bladder. Intestinal injury was defined as abnormal findings of free air, mesenteric contusion or bleeding, and bowel edema or ischemia.

Information on the quantity of fluids administered, except for the number of blood transfusions, during the first 24 hours after hospital arrival was extracted from the medical records. The cumulative fluid balance for 24 hours was computed by subtracting all outputs from the total volume of infused fluids in 24 hours. A cutoff value of 2.5 L for the 24-hour cumulative fluid balance was estimated using the area under the receiver operating characteristic curve. Information on exposure to hydroxyethyl starch within the first 24 hours was also extracted from the medical records. An indwelling nasogastric (NG) tube was identified using chest or abdominal X-ray within the first 48 hours. Drugs such as vasopressors, sedatives, diuretics, and anti-ulcer medications administered within the first 48 hours were recorded.

Emergency surgery was defined as a thoracic- and/or abdominal-related operation performed within the initial 24 hours. Acute kidney injury was diagnosed based on the current Kidney Disease: Improving Global Outcomes criteria.[14] The clinical outcomes of interest included the length of ICU or hospital stay, days of mechanical ventilation, and in-hospital mortality.

Statistical analyses

Categorical variables are presented as frequency (%) and were compared using the chi-square or Fisher's exact test. Continuous variables are expressed as mean ± standard deviation or median and interquartile range and were compared using the Student *t*-test or Mann–Whitney *U* test. Factors found to be significantly associated with VAP ($p < 0.05$) in the univariable analysis were included in the multivariable analysis in addition to the predefined factors of hypotension; ISS; hemothorax; abdominal intestinal injury;

cumulative fluid balance during the initial 24 hours >2.5 L; use of vasopressors, sedatives, or diuretics; Levin tube insertion; enteral feeding within 7 days; and initial lactate level. Multivariable regression analysis was performed using a logistic regression model with the maximum likelihood method and backward stepwise selection. Goodness-of-fit was assessed using the Hosmer–Lemeshow test. All statistical analyses were performed using IBM SPSS Statistics version 23.0 (IBM Corp., Armonk, NY, USA).

Results

Baseline patient characteristics

Overall, 428 patients with trauma were admitted to the ICU after thoracic and/or abdominal injury between January 2016 and December 2019. Of these, we evaluated 178 patients who met the inclusion criteria. The VAP group comprised 54 patients (30.3%) and the non-VAP group included 124 patients (69.7%).The mean patient age was 54 years (56 years vs. 53 years, respectively), and there were no significant between-group differences in patient characteristics at baseline. The patient characteristics are listed in Table 1. Hypertension was the most common underlying disease; there were no significant differences in the distribution of underlying diseases between the two groups. The most common type and mechanism of injury was blunt trauma and car accident, respectively. Trauma scores such as the ISS and RTS

Table 1. Baseline patient characteristics.

	Total (N = 178)	VAP (n=54)	Non-VAP (n=124)	*p* value
Age (y) mean ± SD	54.6 ± 18.4	56.2 ± 20.0	53.9 ± 17.7	0.469
Male sex, n (%)	122 (69)	35 (65)	87 (70)	0.488
Underlying disease				
HTN, n (%)	50 (28)	13 (24)	37 (30)	0.473
DM, n (%)	17 (10)	6 (11)	11 (9)	0.782
CAoD, n (%)	6 (3)	2 (4)	4 (3)	1.000
CVA, n (%)	5 (3)	1 (2)	4 (3)	1.000
PTB, n (%)	4 (2)	2 (4)	2 (2)	0.586
Blunt trauma, n (%)	174 (98)	53 (98)	121 (98)	1.000
Injury mechanism				0.570
Pedestrian TA, n (%)	42 (24)	12 (22)	30 (24)	
Auto TA, n (%)	56 (32)	17 (32)	39 (32)	
Motorcycle TA, n (%)	28 (16)	13 (24)	15 (12)	
Fall, n (%)	36 (20)	10 (19)	26 (21)	
Other, n (%)	16 (9)	2 (4)	14 (11)	
GCS, median [IQR]	11 [5.0–15.0]	7.5 [3.0–14.0]	12.5 [6.0–15.0]	0.001
ISS, mean ± SD	30.2 ± 9.8	33.7 ± 9.53	28.6 ± 9.54	0.001
RTS, median [IQR]	6.085 [4.094–7.550]	5.439 [2.935–6.817]	6.376 [5.030–7.841]	0.001
AIS score				
Head and neck, median [IQR]	2 [0.0–3.0]	3 [0.0–4.0]	0 [0.0–3.0]	0.034
Chest, median [IQR]	3 [3.0–3.0]	3 [3.0–4.0]	3 [3.0–3.0]	0.127
Abdomen, median [IQR]	3 [2.0–3.0]	3 [2.0–3.0]	3 [2.0–3.0]	0.538

Hypotension is defined as systolic blood pressure <90 mm Hg.

VAP, ventilator-associated pneumonia; SD, standard deviation; HTN, hypertension; DM, diabetes mellitus; CAoD, coronary artery occlusive disease; CVA, cerebrovascular accident; TA, traffic accident; GCS, Glasgow Coma Scale; ISS, Injury Severity Score; AIS, Abbreviated Injury Scale; PTB, pulmonary tuberculosis; RTS, Revised Trauma Score; IQR, interquartile range.

were higher in the VAP group than those in the non-VAP group, indicating worse injury severity.

Clinical and initial laboratory variables

Patients' clinical and initial laboratory variables are presented in Tables 2 and 3. In the initial 24 hours, patients in the VAP group were more likely to receive packed red blood cell transfusion (median: 2 units vs. 0 units, $p = 0.001$); fresh frozen plasma (median: 2 units vs 0 units, $p = 0.009$); and a greater volume of infused fluids (7.2 L vs. 6.2 L, $p = 0.047$) than those in the non-VAP group. There were no significant differences in the 24-hour cumulative fluid balance. However, the proportion of patients with 24-hour cumulative fluid balance >2.5 L was significantly higher in the VAP group (78% vs. 60%, $p = 0.026$). More than half of patients (66%) had a Levin tube inserted, and the rate of tube insertion was significantly higher in the VAP group (91% vs. 56%, $p < 0.001$). The proportion of patients with enteral feeding within 7 days after admission was significantly higher in the non-VAP group than in the VAP group (58% vs. 33%, $p = 0.003$).

Table 2. Clinical parameters.

	Total (N = 178)	VAP group (n = 54)	Non-VAP group (n = 124)	*p* value
Hypotension (SBP <90 mmHg) n (%)	74 (42)	31 (57)	43(35)	0.008
Hemothorax, n (%)	77 (43)	29 (54)	48 (39)	0.072
Pneumothorax, n (%)	93 (52)	30 (56)	63 (51)	0.626
Pulmonary contusion	103 (58)	34 (63)	69 (56)	0.411
Rib fracture(s), n (%)	133 (75)	41 (76)	92 (74)	0.853
Abdominal solid organ injury, n (%)	91 (51)	29 (54)	62 (50)	0.745
Abdominal intestinal injury, n (%)	43 (24)	18 (33)	25 (20)	0.085
Thoraco-lumbar spine fractures, n (%)	87 (49)	27 (50)	60 (48)	0.872
pRBC transfusions (U) in 24 h, median [IQR]	1 [0–4]	2 [0–5]	0 [0–3]	0.001
FFP transfusions (U) in 24 h, median [IQR]	1 [0–4]	2 [0–5]	0 [0–4]	0.009
Total volume of infused fluid in 24 h (L), median [IQR]	6.6 [4.9–8.6]	7.2 [5.3–9.0]	6.2 [4.7–8.4]	0.047
Cumulative fluid balance in 24 h (L), median [IQR]	3.5 [1.9–5.1]	4.3 [2.7–5.7]	3.1 [1.7–5.0]	0.064
Cumulative fluid balance in 24 h >2.5 (L), n (%)	116 (65)	42 (78)	74 (60)	0.026
HES administered 24 h, n (%)	81 (46)	24 (44)	57 (46)	0.871
Nasogastric tube, n (%)	118 (66)	49 (91)	69 (56)	<0.001
Enteral feeding within 7 days, n (%)	90 (51)	18 (33)	72 (58)	0.003
Drugs administered during the first 48 h				
Vasopressors, n (%)	103(58)	47 (65)	61 (20)	<0.001
Sedatives, n (%)	134 (75)	46 (85)	88 (71)	0.058
Diuretics, n (%)	75 (42)	32 (44)	73 (24)	0.001
Anti-ulcer medications, n (%)				
H2 blockers	139 (78)	41 (76)	98 (79)	0.695
PPIs	36 (20)	14 (26)	22 (18)	0.227

VAP, ventilator-associated pneumonia; PPI, proton pump inhibitor; pRBC, packed red blood cells; FFP, fresh frozen plasma; SBP, systolic blood pressure; HES, hydroxyethyl starch; IQR, interquartile range; VAP, ventilator-associated pneumonia.

Table 3. Laboratory findings.

	Total (N = 178) Median [IQR]	VAP group (n = 54) Median [IQR]	Non-VAP group (n = 124) Median [IQR]	*p* value
Hemoglobin (g/dL)	12.7 [10.6 to 13.8]	11.3 [9.7 to 13.5]	12.8 [11.3 to 13.8]	0.034
WBC	11.4 [8.1 to 16.9]	11.7 [7.9 to 18.9]	11.7 [8.2 to 16.0]	0.815
hsCRP	0.1 [0.05 to 0.195]	0.1 [0.60 to 0.25]	0.1 [0.05 to 0.19]	0.312
BUN (mg/dL)	16.2 [12.1 to 20.4]	15.5 [12.7 to 19.4]	16.4 [11.9 to 20.8]	0.608
Creatinine (μmol/L)	0.88 [0.76 to 1.08]	0.88 [0.76 to 1.10]	0.88 [0.75 to 1.07]	0.844
CPK (g/dL)	419 [233 to 720]	447 [233 to 709]	409 [234 to 749]	0.799
Sodium (mmol/L)	139 [137 to 141]	139 [137 to 141]	139 [137 to 141]	0.952
Potassium (mmol/L)	3.8 [3.5 to 4.2]	3.9 [3.5 to 4.5]	3.8 [3.5 to 4.1]	0.951
Chloride (mmol/L)	108 [106 to 110]	108 [104 to 110]	108 [106 to 110]	0.455
pH	7.34 [7.26 to 7.39]	7.33 [7.25 to 7.39]	7.34 [7.27 to 7.39]	0.540
pO2	80 [64 to 131]	72[56 to 131]	87 [70 to 132]	0.047
pCO2	36 [31 to 42]	34 [29 to 43]	37 [33 to 42]	0.155
Base deficit (mmol/L)	−6.2 [−9.2 to −2.9]	−7.1 [−10.7 to −3.7]	−5.3 [−8.7 to −2.6]	0.054
Lactate (mmol/L)	3.5 [2.5 to 6.0]	4.5 [2.9 to 6.7]	3.3 [2.2 to 5.2]	0.024

IQR, interquartile range; WBC, white blood cell, hsCRP, high-sensitivity C-reactive protein; BUN, blood urea nitrogen; CPK, creatine phosphokinase; eGFR, estimated glomerular filtration rate; VAP, ventilator-associated pneumonia; pO2, partial pressure of oxygen; pCO2, partial pressure of carbon dioxide.

Table 4. Clinical outcomes.

	Total (N = 178)	VAP group (n = 54)	Non-VAP group (n = 124)	*p* value
LoH (d), median [IQR]	38.0 [20.0–72.0]	55.0 [35.0–106.5]	33.0 [16.0–55.7]	<0.001
LoICU (d), median [IQR]	9.0 [4.8–20.0]	21.0 [14.0–37.0]	6.0 [4.0–11.0]	<0.001
DoMV (d), median [IQR]	5.0 [2.0–9.0]	9.0 [6.5–18.5]	3.0 [1.0–5.8]	<0.001
Emergency operation	101 (57)	32 (59)	69 (56)	0.743
AKI, n (%)	53 (30)	21 (39)	32 (26)	0.108
ARDS, n (%)	5 (3)	5 (9)	0	
VAP rate per 1000 ventilator days, (%)	38.8			
In-hospital mortality, n (%))	31 (17)	13 (24)	18 (15)	0.136

LoH, length of hospital stay; IQR, interquartile range; LoICU, length of intensive care unit stay; DoMV, duration of mechanical ventilation; AKI, acute kidney injury; ARDS, acute respiratory distress syndrome; VAP, ventilator-associated pneumonia.

Clinical outcomes

The median length of hospital stay (55.0 days vs. 33.0 days, $p < 0.001$), length of ICU stay (21.0 days vs. 6.0 days, $p < 0.001$), and duration of mechanical ventilation (9.0 days vs. 3.0 days, $p < 0.001$) were longer in the VAP group than in the non-VAP group. In contrast, there were no significant between-group differences for the proportion of patients who underwent emergency surgery or for development of VAP and in-hospital mortality. The VAP incidence rate was 38.8 cases per 1000 ventilator days (Table 4).

Logistic regression analysis for VAP risk factors

The multivariable logistic analysis identified ISS (adjusted odds ratio [aOR]: 1.048, 95% confidence interval [CI]: 1.008–1.090, $p = 0.018$), vasopressor use (aOR: 2.541, 95% CI: 1.121–5.758, $p = 0.026$), and insertion of a Levin tube (aOR: 6.749, 95% CI: 2.397–18.999, $p < 0.001$) as independent risk factors for VAP (Table 5).

Discussion

Thoracic injury accounts for 10% to 15% of all cases of trauma,[15] and approximately 15% of patients with thoracic injury also have an abdominal injury.[16] These injuries are leading causes of death, hospitalization, and long-term disability.[17,18] Therefore, we evaluated mechanically ventilated patients with torso injuries admitted to the ICU and found a VAP incidence rate of 33.8 per 1000 ventilator days. Furthermore, 30% of patients on mechanical ventilation developed VAP. This rate is in line with reports of previous studies, which range from 22% to 44%.[11,12,19]

Consistent with previous studies, we also found that severe injury was associated with a higher risk of VAP.[11,20,21] Severely injured patients might require earlier airway acquisition and longer mechanical ventilation than other patients. Moreover, many severely injured patients have a compensatory anti-inflammatory response phase after an increased systemic inflammatory response, which is characterized by diminished resistance to infection.[22] Unlike in previous studies,[19,21] however, higher head and neck injury score was not an independent predictor of VAP in our study population. The mean ISS in our patients was >30, although the two groups showed a significant difference, which meant that they had an AIS ≥3 or 4 in two or more body regions. Head and neck injury alone did not account for the injury severity and higher risk of VAP. European guidelines stipulate that vasopressors can be used to maintain the target arterial pressure and reduce the volume of fluid resuscitation among severely injured patients who are in critical condition.[23] In the present study, the use of vasopressors was identified to be an independent risk factor for VAP.

Table 5. Univariate and multivariate analyses of risk factors for ventilator-associated pneumonia.

	Univariate analysis		Multivariate analysis	
Variables	OR (95% CI)	*p* value	OR (95% CI)	*p* value
Hypotension	2.539 (1.320–4.883)	0.005		
ISS	1.055 (1.020–1.092)	0.002	1.048 (1.008–1.090)	0.018
AIS score in the head and neck	1.194 (1.009–1.414)	0.039		
pRBC transfusions (U) in 24 h	1.125 (1.019–1.241)	0.019		
Cumulative fluid balance >2.5 L in 24 h	2.365 (1.134–4.932)	0.022		
Use of vasopressors	4.170 (1.969–8.828)	<0.001	2.541 (1.121–5.758)	0.026
Use of sedatives	2.352 (1.010–5.476)	0.047		
Nasogastric tube	7.812 (2.914–20.939)	<0.001	6.749 (2.397–18.999)	<0.001
Enteral feeding within 7 d	0.361 (0.185–0.705)	0.003		
Initial lactate level	1.131 (1.000–1.278)	0.049		

The *p* value for the Hosmer-Lemeshow goodness-of-fit-test was 0.868.

ISS, Injury Severity Score; AIS, Abbreviated Injury Scale; pRBC, packed red blood cells; SBP, systolic blood pressure; OR, odds ratio; CI, confidence interval.

Enteral nutrition is preferred in critically ill patients because it is inexpensive, helps maintain the integrity of the intestinal mucosal barrier, and prevents the translocation of intestinal bacteria.[24] Initiating enteral nutrition within the first 24 to 48 hours after ICU admission is generally recommended, except in cases of uncontrolled shock or hemodynamic instability.[25] Enteral nutrition is preferentially provided through an NG tube into the stomach within the first 24 hours.[26] However, insertion of an NG tube may also cause inadequate functioning of the upper and lower gastroesophageal sphincter, maxillofacial sinusitis, and increased pharyngeal colonization.[27] In addition, a malpositioned feeding tube can cause aspiration of gastric contents into the respiratory tract, resulting in pneumonia.[26,28,29] In our study, NG tube insertion was identified to be an independent risk factor for VAP. However, we could not assess malpositioning of the NG tube. Further research is required to clarify whether the insertion of an NG tube itself or its malpositioning increases the risk of VAP.

Our study had some limitations. First, this was a retrospective, single-center study. Our cohort was very specific and included a relatively small number of patients with thoracic and/or abdominal trauma. This could have resulted in selection bias, limiting the generalizability of our findings to other patients with trauma. Second, there were no data available regarding a history of vomiting, aspiration, or use of a supraglottic airway device before intubation in the emergency room. These factors might also be potential risk factors of VAP. Third, the optimal diagnostic criteria for VAP remain under debate. Fourth, the use of antimicrobial agents for surgical prophylaxis or open wounds and fractures might affect the incidence of VAP; use of these was not assessed in this study.

In summary, the development of VAP in patients with torso injury admitted to the ICU was significantly associated with ISS, vasopressor use, and NG tube insertion. Recognizing risk factors at an early stage could aid in risk stratification and the provision of optimal ICU care in patients with trauma.

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Data availability

The datasets and analysis in the current study are available from the corresponding author on reasonable request.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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