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Evaluation of the Infection-Related Ventilator-Associated Events Algorithm for Ventilator-Associated Pneumonia Surveillance in a Trauma Population

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BACKGROUND: The Centers for Disease Control and Prevention have recently introduced new ventilator-associated pneumonia (VAP) surveillance on the basis of the infection-related ventilator-associated complication (IVAC) definition. We aim to evaluate the accuracy of this new IVAC algorithm for detecting VAP according to the 2008 Centers for Disease Control and Prevention/National Healthcare Safety Network (NHSN) definition as the reference diagnosis (VAP-NHSN) in high-risk trauma patients. METHODS: This retrospective single-center study included all trauma subjects who were admitted to the ICU, required mechanical ventilation for >48 h, and received a blood transfusion. The new IVAC surveillance and the criteria for VAP-NHSN diagnosis were applied. The accuracy of the new IVAC surveillance for detecting VAP-NHSN was determined, and the clinical outcomes were compared between groups. RESULTS: The sensitivity, specificity, and positive and negative predictive values of IVAC for VAP-NSHN identification were 28.12%, 91.45, 58.06%, and 75.14%, respectively. Subjects with IVAC, VAP-NHSN, or both had higher morbidity when compared with those without IVAC and VAP-NHSN. Subjects with IVAC only had lower morbidity compared with those with VAP-NHSN only or those with both IVAC and VAP-NHSN. There was no significant difference in clinical outcomes between subjects with VAP-NHSN only and those with both IVAC and VAP-NHSN. CONCLUSIONS: IVAC criteria had a low accuracy for identifying VAP-NHSN in subjects with high-risk trauma. *Key words: accuracy; critical care; trauma; ventilator-associated events; ventilator-associated pneumonia.* [Respir Care 0;0(0):1–•. © 0 Daedalus Enterprises]

Introduction

Ventilator-associated pneumonia (VAP) is one of the most common hospital-acquired infections among mechanically ventilated patients.[1] Patients with VAP carry significantly higher morbidities, including longer duration of mechanical ventilator support as well as longer ICU and hospital stay[1] and higher mortality[2] than those without VAP.

According to the Centers for Disease Control and Prevention (CDC) and National Healthcare Safety Network (NHSN) surveillance definition of healthcare-associated infection published in 2008,[3] signs and symptoms such as

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The authors have disclosed no conflicts of interest.

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fever, leukocytosis, or leukopenia; deterioration in oxygenation; changes in characteristics of respiratory secretions; and microbiological evidence of pulmonary infection are used in combination with changes in serial chest radiographs to make the diagnosis of VAP. These criteria are widely accepted as a clinical standard for VAP diagnosis. However, they have been criticized as lacking objective definitions and having a high degree of intra-observer and inter-observer variability.[4] Recently, the CDC has published a new algorithm for surveillance of ventilator-associated events.[5] This algorithm mainly focuses on identifying mechanically ventilated patients who have worsening respiratory status resulting from various causes[6-11] on the basis of changes in support, namely PEEP and/or F_{IO_2}.[5] The term infection-related ventilator-associated complications (IVACs) is applied when a ventilator-associated event is associated with leukocytosis/leukopenia or hypothermia/hyperthermia plus administration of new antimicrobial agents.[5] Furthermore, the terms possible and probable VAP are applied to IVAC patients if there is evidence of pulmonary infection defined as purulent respiratory secretions and/or positive culture of lower respiratory tract specimens.[5] The main objectives of this newly developed algorithm are to increase the reliability, reproducibility, comparability, and efficiency of the surveillance and to improve patient safety in the ICU.[12,13] Despite this, recent studies in mechanically ventilated subjects have shown that the IVAC definition has poor accuracy in identifying patients with VAP.[6,8,10]

Trauma patients are known to carry a higher incidence of VAP compared with the nontrauma population.[1,14,15] Risk factors for VAP in these patients are well-described, including site and severity of trauma,[16,17] duration of mechanical ventilator support,[16] and transfusion of packed red blood cells.[18-20] To the best of our knowledge, there are very limited data regarding ventilator-associated event surveillance in the high-risk trauma patient population. We hypothesize that, on the basis of the IVAC algorithm, some high-risk trauma patients with VAP would be not identified and, on the other hand, patients who are identified having IVAC actually do not have VAP. We therefore aimed to determine the accuracy of the IVAC surveillance for detecting VAP as identified with the VAP-NHSN criteria in subjects with high-risk trauma and to describe characteristics and clinical outcomes in subjects with and without IVAC.

QUICK LOOK

Current knowledge

Ventilator-associated pneumonia (VAP) is one of the most common hospital-acquired infections. Patients with VAP have longer durations of mechanical ventilation, longer ICU and hospital stay, and higher mortality than those without VAP. The Centers for Disease Control and Prevention has published a new algorithm for surveillance of ventilator-associated events. This algorithm focuses on identifying mechanically ventilated patients with worsening respiratory status, on the basis of changes in PEEP and F_{IO_2}, to aid in identifying infection-related ventilator-associated complications (IVACs).

What this paper contributes to our knowledge

In a group of subjects with high-risk trauma, the IVAC surveillance algorithm had a low sensitivity and a low positive predictive value for identification of VAP. The algorithm failed to diagnose nearly 75% of subjects with VAP. The IVAC algorithm also identified subjects requiring increasing ventilatory support due to a range of conditions unrelated to VAP. The use of ventilator-associated event monitoring in this population requires further refinement.

Methods

Setting and Patient Population

This retrospective study was approved by the institutional review board at Massachusetts General Hospital, Boston, Massachusetts with a waiver of informed consent as a retrospective design of the study. Massachusetts General Hospital is a large university-affiliated, tertiary care referral and level-1 trauma center. Since 2005, data of all trauma patients admitted to our hospital have been prospectively collected in the Massachusetts General Hospital trauma registry database as a part of the National Trauma Data Bank. They include volume, acuity, age, stay, average ICU stay, ventilator days, and mortality. Patients in the Massachusetts General Hospital trauma registry database admitted to the hospital between July 1, 2009, and December 31, 2013, were eligible for inclusion in this study. They were included if they were at least 18 y old, admitted to the ICU after trauma, required endotracheal intubation and mechanical ventilator support for at least 48 h, and received a minimum of 1 unit of packed red blood cell transfusion during their mechanical ventilator support. Patients were excluded if they had tracheostomy placed upon arrival to ICU, were transferred from other hospitals after trauma, or died within 48 h after ICU admission.

Data Collection

Prospectively collected data of each included subject were retrieved from both electronic and paper medical records. Demographic data, including age, sex, weight,

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height, race, ABO blood group, past medical history and Charlson comorbidity index (CCI)[21] were collected. Acuity of illness scores, including the Acute Physiology and Chronic Health Evaluation (APACHE) II score,[22] the Sequential Organ Failure Assessment (SOFA) score,[23] mechanism of injury, the injury severity score,[24] and laboratory values were also recorded. Daily data, including vital signs, laboratory values, chest radiographs, modes of mechanical ventilator support, levels of PEEP, and F_{IO_2}, were recorded until patients were transferred to floor or died. At our institution, a chest radiograph was routinely taken every morning in all intubated patients. A sputum sample was collected for bacterial cultures when a new infiltrate was seen on the chest radiograph and/or fever associated with leukocytosis (white blood cell count >10,000 cells/mm^3) was observed. Surveillance cultures were not implemented as part of the routine care at our institute. Clinical outcomes, including duration of mechanical ventilator support, ventilator-free days at 28 d, requirement of re-intubation and/or tracheostomy, length of ICU and hospital stay, hospital mortality, and causes of death were also collected. For subjects with IVAC, onset of the event, timing and appropriateness of initial antimicrobial therapy, and microbiologic results were additionally recorded. Appropriate therapy was defined as proper antibiotic coverage of all of the identified pathogens.

Definition of IVAC

The definition of IVAC in this study was based on the recent publication from the CDC.[5] In brief, subjects would meet the criteria for the diagnosis of IVAC if they had a sustained increase of daily PEEP levels ≥3 cm H_2O or a sustained increase of daily F_{IO_2} ≥0.20 for at least 2 consecutive days after 2 or more consecutive days of stable or decreasing daily minimum PEEP levels or F_{IO_2} values plus had (1) temperature of >38°C or <36°C or white blood cell counts of ≥12,000 cells/mm^3 or ≤4,000 cells/mm^3 and (2) new antimicrobial agents started and continued for ≥4 consecutive days. If IVAC subjects had pulmonary infection as evidenced by purulent respiratory secretions and/or positive culture of lower respiratory tract specimens, they were classified as possible VAP or probable VAP, respectively, according to the definition.[5] To make a diagnosis of IVAC, first, ventilator-associated events were captured by using CDC-designed software[5] and the dedicated database from the Respiratory Care Department, in which the lowest F_{IO_2} and the lowest PEEP in every 12 h of all invasively ventilated patients were listed. This information was then sent to the Infection Control Department, who continued the analysis of IVAC and possible and probable VAP. None of the individuals from the Respiratory Care Department or the Infection Control Department were involved in this paper.

Definition of VAP

Subjects were considered to have VAP if they met the criteria according to the CDC and NHSN definition published in 2008,[3] and we considered these criteria as the reference diagnosis in this study. The criteria consisted of (1) ≥2 serial chest x-rays with new or progressive and persistent infiltration, consolidation, and/or cavitation; (2) signs/symptoms of fever (>38°C) and/or leukopenia or leukocytosis (white blood cell counts of <4,000 or ≥12,000 cells/mm^3, respectively) plus new onset of purulent sputum, change in character of sputum, or increased respiratory secretions; new onset or worsening cough, dyspnea, or tachypnea; rales or bronchial breath sounds; and/or worsening gas exchange; and (3) positive growth in blood culture, culture of pleural fluid, or culture from minimally contaminated lower respiratory tract specimens. The decision that VAP was present was determined by the infection control committee, which included a respiratory therapist, an infectious disease physician, and 2 infection control nurses. To make the diagnosis of VAP, the committee reviewed all of the subject's medical records, including daily chest x-rays interpreted by board-certificated radiologists. None of the infection control committee members were involved in this paper.

Study End Points

Our primary end point was the accuracy of the IVAC definition to identify VAP in subjects with high-risk trauma. We used the VAP-NHSN definition from the CDC/NHSN in 2008[3] as the reference diagnosis of VAP. Our secondary end point was to describe outcomes and incidence of respiratory complications of subjects with IVAC alone, with VAP-NHSN alone, with both IVAC and VAP-NHSN, or without IVAC and VAP-NHSN.

Statistical Analysis

The IVAC definition was tested for sensitivity, specificity, and positive and negative predictive values for the diagnosis of VAP as defined by the 2008 CDC/NHSN definition[3] in subjects with high-risk trauma. Continuous variables were compared between groups using the Mann-Whitney U test. Categorical variables were compared between groups using the chi-square test or the Fisher exact test when appropriate. All comparisons were unpaired, all tests of significance were 2-tailed, and a P value of <.05 was considered statistically significant. Statistical analyses were performed using SPSS 16.0 for Windows (SPSS, Chicago, Illinois).

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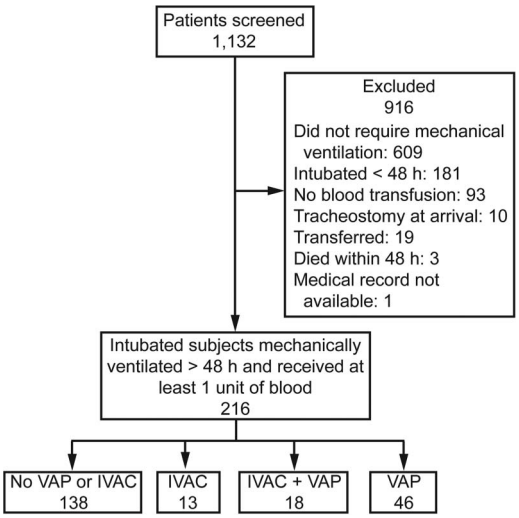

Fig. 1. Flow chart. VAP = ventilator-associated pneumonia; IVAC = infection-related ventilator-associated complication.

Table 1. Test Characteristics of Infection-Related Ventilator-Associated Complications for the Diagnosis of Ventilator-Associated Pneumonia

	VAP	No VAP	Total
IVAC	18	13	31*
No IVAC	46	139	185†
Total	64‡	152§	216

* Positive predictive value 58.1% (95% CI, 17.6–75.4%).
† Negative predictive value 75.1% (95% CI, 68.3–81.2%).
‡ Sensitivity 28.1% (95% CI, 17.6–40.8%).
§ Specificity 91.5% (95% CI, 85.8–95.4%).
VAP = ventilator-associated pneumonia
IVAC = infection-related ventilator-associated complications

Results

From the Massachusetts General Hospital trauma registry database between July 1, 2009, and December 31, 2013, there were 1,132 trauma patients screened for inclusion (Fig. 1). Of these, 916 patients did not meet the inclusion criteria (609 did not require mechanical ventilator support; 181 were intubated for <48 h; 93 did not receive a packed red blood cell transfusion during mechanical ventilator support; 10 had tracheostomy placed upon ICU arrival; 19 were transferred from other hospitals after trauma; 3 died within 48 h after ICU admission; and from 1 patient, the medical record could not be retrieved), leaving 216 subjects for analysis. Overall, there were 77 (35.6%) subjects who met the definition of IVAC and/or VAP-NHSN. Of these, 31 (14.4%) subjects were identified as IVAC, 64 (29.6%) as VAP-NHSN, and 18 (8.3%) as both IVAC and VAP-NHSN (Fig. 1). These corresponded to incidences of IVAC and VAP-NHSN of 16.6 and 34.2 events per 1,000 ventilator days, respectively. The sensitivity, specificity, and positive and negative predictive values of IVAC for VAP-NHSN diagnosis were 28.1% (95% CI, 17.6–40.8%), 91.5% (95% CI, 85.8–95.4%), 58.1% (95% CI, 17.6–75.4%), and 75.1% (95% CI, 68.3–81.2%), respectively (Table 1).

Table 2 shows the demographic data of all subjects. There was no significant difference in the demographic data between subjects with VAP-NHSN only, with IVAC only, with both VAP-NHSN and IVAC, and without VAP-NHSN and IVAC except that subjects with both IVAC and VAP-NHSN had higher injury severity scores than those with VAP-NHSN only and those without VAP-NHSN and IVAC (median 38 [interquartile range 27–58] vs 34 [29–38], $P = .044$ and 38 [27–58] vs 29 [22–38], $P = .007$, respectively).

In the cohort of 21 subjects with IVAC (Table 3), IVAC subjects with VAP-NHSN had antibiotics initiated later than IVAC subjects without VAP-NHSN (1.7 ± 3.6 d vs −0.8 ± 1.3 d, $P = .01$). There was no difference in appropriateness and duration of antibiotic administration as well as microbiology results between groups.

Table 4 shows the clinical outcomes of all subjects. Overall, subjects with IVAC and/or VAP-NHSN had higher morbidity when compared with those without IVAC and VAP-NHSN. Subjects with IVAC only had a lower tracheostomy rate compared with those with VAP-NHSN only (23.1% vs 67.4%, $P = .004$) and those with both IVAC and VAP-NHSN (23.1% vs 61.1%, $P = .036$). They also had shorter mechanical ventilator duration compared with those with both IVAC and VAP-NHSN (median 207 [interquartile range 124–295] h vs 258 [214–422] h, $P = .045$) and shorter hospital length of stay compared with those with VAP-NHSN (21 [13–25] d vs 25 [20–39] d, $P = .033$). There was no significant difference in clinical outcomes between subjects with VAP-NHSN only and those with both IVAC and VAP-NHSN.

Discussion

The main finding of our study is that, in subjects with high-risk trauma, the IVAC criteria had poor sensitivity and poor positive predictive value for the diagnosis of VAP according to the 2008 CDC/NHSN definition.[3] Our study confirmed the low sensitivity of IVAC to detect VAP-NHSN, with a value comparable with what has been already reported in large heterogenic critically ill populations.[6,8,10] Incidences of IVAC and VAP-NHSN in our homogeneous high-risk trauma patient population were 16.6 and 34.2 events per 1,000 ventilator days, respectively. These were higher than those reported in heteroge-

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Table 2. Demographic Data of All Subjects

	VAP Only (n = 46)	IVAC Only (n = 13)	Both IVAC and VAP (n = 18)	No IVAC or VAP (n = 139)
Age, median (IQR) y	50.7 (27.0–66.4)	37.2 (25.4–62.0)	50.0 (33.3–62.7)	45.4 (28.9, 61.8)
Male sex, n (%)	36 (78.3)	11 (84.6)	17 (94.4%)*	94 (67.6%)
Weight, median (IQR) kg	77 (70–87)	86 (82–100)	82 (77–92)	77 (66–87)
Height, median (IQR) cm	173 (168–180)	175 (168–183)	177 (171–183)	173 (165–178)
ABO type O, n (%)	21 (45.7)	8 (61.5)	13 (72.2)	75 (54.0)
Caucasian, n (%)	25 (54.3)	9 (69.2)	10 (55.6)	82 (59.0)
Past medical history, n (%)				
Cardiovascular	7 (15.2)	1 (7.7)	2 (11.1)	8 (5.8)
Pulmonary	4 (8.7)	3 (23.1)	0 (0.0)	12 (8.6)
Neurological	3 (6.5)	1 (7.7)	0 (0.0)	2 (1.4)
Renal	1 (2.2)	1 (7.7)	0 (0.0)	3 (2.2)
Diabetes mellitus	5 (10.9)	1 (7.7)	3 (16.7)	11 (7.9)
CCI, median (IQR) score	0 (0–1)	0 (0–1)	0 (0–1)	0 (0–1)
APACHE II, median (IQR) score	17 (13–21)	14 (12–25)	14 (10–21)	15 (11–19)
SOFA, median (IQR) score	8 (5–10)	7 (4–9)	6 (5–9)	7 (4–8)
Blunt mechanism, n (%)	42 (91.3)	11 (84.6)	18 (100.0)	125 (89.9)
ISS, median (IQR)	34 (29–38)†	34 (29–41)	38 (27–58)*	29 (22–38)
AIS head, median (IQR) score	1 (0–3)	2 (2–4)	3 (0–5)	1 (0–3)
AIS chest, median (IQR) score	3 (3–4)	3 (3–4)	4 (0–4)	3 (0–4)
WBC, median (IQR) thousand/mm^3	11.8 (8.9–18.2)	15.4 (13.0–19.5)	14.3 (9.4–16.4)	13.9 (10.0–18.3)
Hemoglobin, median (IQR) g/dL	10.4 (8.8–12.0)‡	9.1 (8.1–9.8)	9.5 (7.8–12.6)	10.5 (8.7–12.4)
Platelet, median (IQR) thousand/mm^3	162 (135–224)	227 (114–326)	172 (69–209)	167 (121–221)
PRBC, median (IQR) units	2 (1–8)	5 (3–13)	3 (2–6)	3 (2–8)

* $P < 0.05$, compared with group with no infection-related ventilator-associated complications or ventilator-associated pneumonia.
† $P < 0.05$, compared with group with both infection-related ventilator-associated complications and ventilator-associated pneumonia.
‡ $P < 0.05$, compared with group with infection-related ventilator-associated complications only.
VAP = ventilator-associated pneumonia
IVAC = infection-related ventilator-associated complications
CCI = Charlson comorbidity index
APACHE = Acute Physiology and Chronic Health Evaluation
SOFA = Sequential Organ Failure Assessment
ISS = injury severity score
AIS = abbreviated injury scale
PRBC = packed red blood cells

neous mechanically ventilated subjects (IVAC, 3.6–8.8 and VAP, 3.0–10.0 events/1,000 ventilator days).[6,8,10,12] The deviation of our finding from other reports can be justified by the difference in patient population. Our study focused on critically ill subjects with trauma with a high risk of respiratory compromise as well as VAP.

In our study, there were 13 (41.9%) of 31 IVAC subjects who did not meet the 2008 CDC/NHSN criteria for VAP diagnosis[3] (Table 1 and Fig. 1). This was mainly due to the absence of any change in chest radiographs consistent with the diagnosis of VAP-NHSN. The possible causes of worsening oxygenation and subsequent manipulation of mechanical ventilator support in these 13 subjects were pulmonary edema in 6 cases, atelectasis in 5 cases, pulmonary contusion in 1 case, and underlying pulmonary fibrosis in 1 case. Our findings were supported by the fact that the ventilator-associated event surveillance potentially identifies mechanically ventilated patients who have worsening respiratory status due not only to VAP but possibly to different pulmonary complications, such as ARDS, atelectasis, pulmonary embolism, and pulmonary edema as well as extrapulmonary causes, such as abdominal compartmental syndrome or sepsis.[6-11]

On the other hand, there were 46 (71.9%) of 64 subjects with VAP diagnosed according to the 2008 CDC/NHSN definition[3] that had no IVAC identified (Table 1 and Fig. 1). Of these, 38 did not meet the criterion of increase in daily minimum PEEP levels or F_{IO_2} values for ≥2 consecutive days, and 8 did not have a previous stable period of PEEP levels or F_{IO_2} values for ≥2 consecutive days. Our finding was consistent with recent literature.[8,10] It is noteworthy that the IVAC criteria did not detect almost three quarters of subjects with VAP-NHSN, although this group of subjects showed higher morbidities and worse clinical outcomes than the other groups. This should be considered as an important limitation of the usefulness of the IVAC definition in identifying patients with VAP-NHSN.

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Table 3. Antibiotics and Microbiology Results in Subjects With Infection-Related Ventilator-Associated Complications With Versus Without Ventilator-Associated Pneumonia

	Subjects with IVAC		*P*
	VAP (*n* = 18)	No VAP (*n* = 13)	
Time from IVAC to antibiotics, d			
Mean ± SD	1.7 ± 3.6	−0.8 ± 1.3	.01
Median (IQR)	0.5 (−0.3 to 3.0)	−1.0 (−2.0 to 0.0)	.006
Appropriateness, *n* (%)	18 (100.0)	11 (84.6)	.19
Initial antibiotics, *n* (%)			
Vancomycin	13 (72.2)	12 (92.3)	.36
Cefepime	9 (50.0)	12 (92.3)	.02
Metronidazole	0 (0.0)	3 (23.1)	.06
Tobramycin	2 (11.1)	0 (0.0)	.50
Piperacillin/Tazobactam	2 (11.1)	0 (0.0)	.50
Duration of antibiotics, d			
Mean ± SD	8.7 ± 3.4	10.2 ± 6.7	.44
Median (IQR)	9 (6–11.5)	10 (5–13)	.75
Microbiology results, *n* (%)			
1 organism isolated	12 (66.7)	9 (69.2)	>.99
>1 organism isolated	6 (33.3)	2 (15.4)	.41
MSSA	3 (16.7)	2 (15.4)	>.99
MRSA	1 (5.6)	1 (7.7)	>.99
Enterobacter spp.	4 (22.2)	0 (0.0)	.12
Klebsiella spp.	2 (11.1)	2 (15.4)	>.99
Escherichia coli	3 (16.7)	0 (0.0)	.25
Stenotrophomonas spp.	2 (11.1)	1 (7.7)	>.99
Haemophilus influenzae	2 (11.1)	0 (0.0)	.50
Pseudomonas aeruginosa	2 (11.1)	0 (0.0)	.50
Other GNR	2 (11.1)	3 (23.1)	.63
Others	3 (16.7)	4 (30.8)	.41

IVAC = infection-related ventilator-associated complications
VAP = ventilator-associated pneumonia
IQR = interquartile range
MSSA = methicillin-sensitive Staphylococcus aureus
MRSA = methicillin-resistant Staphylococcus aureus
GNR = Gram-negative rod

Table 4. Clinical Outcomes of All Subjects

	VAP Only (*n* = 46)	IVAC Only (*n* = 13)	Both IVAC and VAP (*n* = 18)	No IVAC or VAP (*n* = 139)
Re-intubation, *n* (%)	7 (15.2)	5 (38.5)*	2 (11.1)	18 (12.9)
Tracheostomy, *n* (%)	31 (67.4)*‡	3 (23.1)†	11 (61.1)*	49 (35.3)
Mechanical ventilation duration, median (IQR) h	263 (219–395)*	207 (124–295)*†	258 (214–422)*	139 (80–229)
Mechanical ventilator-free days, median (IQR) d	15 (10–19)*	16 (13–23)	10 (0–18)*	20 (9–24)
ICU LOS, median (IQR) d	16 (10–20)*	12 (5–16)	14 (10–21)*	8 (6–13)
Hospital LOS, median (IQR) d	25 (20–39)*‡	21 (13–25)	28 (17–40)	19 (11–31)
Hospital mortality, *n* (%)	8 (17.4)	4 (30.8)	7 (38.9)	31 (22.3)

* $P < 0.05$, compared with group with no IVAC or VAP.
† $P < 0.05$, compared with group with both IVAC and VAP.
‡ $P < 0.05$, compared with group with IVAC only.
VAP = ventilator-associated pneumonia
IVAC = infection-related ventilator-associated complications
IQR = interquartile range
LOS = length of stay

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In addition, our study showed that subjects with IVAC who also had VAP-NHSN had antibiotics initiated later than those who had IVAC only, for which we do not have an explanation. Those subjects also required longer duration of mechanical ventilator support as well as more often requiring tracheostomy for ventilatory management and weaning. It has been clearly demonstrated that early appropriate therapy improves outcome in critically ill patients and that delayed initiation of appropriate antibiotics for VAP has been associated with increased mortality.[25,26] However, it has also been suggested that it would be advisable to initiate targeted antimicrobial therapy after microbiological evidence and identification of infection.[27] Interestingly, a recent study, regarding the timing of antibiotic administration after identification of a ventilator-associated event, demonstrated that there was no association between timing of antibiotics and mortality, superinfection, or treatment failure in critically ill subjects.[28] Nevertheless, we could not conclude from our study whether delay in the initiation of antibiotics resulted in these adverse events or was only a mere statistical association. In addition, the surveillance culture protocol has not been implemented as routine care at our institution. The results of surveillance cultures might influence the appropriateness of antibiotic choice.

Our study had some limitations. First, as a single-center study focusing on homogeneous very critically ill subjects with trauma as well as a very high risk of VAP, our study results might not be applicable to other critical care settings. Second, VAP in our study was defined according to the 2008 CDC/NHSN definition,[3] which is generally considered as an accepted diagnosis of VAP. Currently, a standard diagnosis of VAP does not exist. However, almost all of the existing data on VAP prevention are based on this traditional definition.[12] Third, as a retrospective study, some risk factors that might potentiate the development of ventilator-associated events, such as overly positive fluid balance, mode of mechanical ventilation, or medication administered,[7] were not considered in our analysis. However, these factors would not be expected to significantly change the accuracy of the IVAC definition in identifying patients with VAP, which was our primary end point. Fourth, we chose to test the accuracy of the IVAC definition in identifying VAP according to the 2008 CDC/NHSN definition in our study despite the fact that the IVAC definition is likely to identify patients who have either pulmonary or extrapulmonary causes that result in severe deterioration of respiratory function.[5] Finally, our sample size might be insufficient to detect significant differences in major clinical outcomes, such as mortality.

Conclusions

Our study demonstrated that the IVAC surveillance algorithm has a low sensitivity and a low positive predictive value for the identification of VAP in subjects with high-risk trauma. The algorithm failed to diagnose about three quarters of subjects with VAP-NHSN. Additionally, the IVAC algorithm identified subjects requiring increasing ventilator support due to a range of conditions unrelated to VAP. Further investigations are required to determine the usefulness of the ventilator-associated event surveillance algorithm in detecting respiratory infections in mechanically ventilated patients.

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