Acinetobacter baumannii Nosocomial Pneumonia in Tertiary Care Hospitals in Thailand

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Background: *Nosocomial pneumonia (NP) is an important cause of morbidity and mortality in hospitalized patients. Acinetobacter baumannii is one of the common causative pathogens in NP. The prevalence of multi-drug resistance in A. baumannii has been increasing. The information on clinical features and clinical courses of A. baumannii NP in Thai patients are limited.*

Objective: *To determine the clinical features, risk factors and clinical courses of A. baumannii NP in Thai patients hospitalized in tertiary care hospitals in Thailand.*

Material and Method: *This was a prospective, hospital-based, active surveillance study on hospital-acquired pneumonia (HAP) and ventilator-associated pneumonia (VAP) in adults hospitalized in 12 tertiary care hospitals in Thailand between 2008 and 2009.*

Results: *There were 651 NP patients. A. baumannii was the most common cause of NP in 198 patients (30.4%). Most of NP patients were males with median age of 71 years. About 80% had late onset NP with the median duration of 10 days after admission in both A. baumannii and non-A. baumannii NP. Most of NP occurred in patients hospitalized in general medical wards. Most of the features of NP in A. baumannii NP and non-A. baumannii NP were not significantly different. The initial antibiotics prescribed were concordant in about 50% of the patients in both groups. Colistin was usually prescribed to the patients who received antibiotic modifications. The initial clinical responses in A. baumannii NP were less favorable than those in non-A. baumannii NP. The mortality rate in A. baumannii NP seemed to be more than that in non-A. baumannii NP. There was a trend of more persistence of pathogen in A. baumannii NP. Most isolates of A. baumannii were resistant to antibiotics including carbapenems. The patients with extensive drug resistant A. baumannii NP had less favorable responses than NP due to other bacteria, including non-extensive drug resistant A. baumannii. VAP, NP developed in medical ICU and NP with bilateral lung involvements on chest X-ray were associated with A. baumannii as the isolated pathogen.*

Conclusion: *A. baumannii is the most common causative pathogen for NP in tertiary care hospitals in Thailand and most of A. baumannii isolates were resistant to many antibiotics including carbapenems. The hospitalized patient in tertiary care hospitals with VAP, or NP that was developed in medical ICU, or NP with bilateral lung involvements on chest x-ray was likely to be due to A. baumannii. Many NP patients received inappropriate initial antibiotic regimens leading to a high mortality.*

Keywords: *Acinetobacter baumannii, Nosocomial pneumonia, Hospital-acquired pneumonia, Ventilator-associated pneumonia*

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Nosocomial pneumonia (NP) is an important cause of morbidity and mortality in hospitalized patients despite advances in antimicrobial therapy and better supportive care modalities[1-9]. NP is the most common site of nosocomial infections in Thailand with the prevalence being about one third of all nosocomial infections[10]. The impacts of NP include prolonged intensive care unit (ICU) and hospital stay, additional healthcare costs and an increase in mortality[1-9]. Gram negative bacteria are common pathogens for NP, especially in Asian countries[12,13]. The most common gram negative bacteria of NP was *A. baumannii*[12,14]. *Acinetobacter* spp is now emerging as a prominent complication of mechanical ventilation. The resistance rate of *A. baumannii* to multiple classes of antibiotics, especially carbepenems, in Thailand has been rapidly increasing[15,16]. The prognosis associated with *A. baumannii* NP is worse than that associated with other bacteria[17-19]. However, the information on clinical features and clinical courses of *A. baumannii* NP in Thai patients is limited.

The objective of the present study was to determine clinical features, risk factors and clinical courses of *A. baumannii* NP in Thai patients hospitalized in tertiary care hospitals in Thailand.

Material and Method

This was a prospective, hospital-based, active surveillance study on NP in hospitalized adults in 12 tertiary care hospitals in Thailand between 2008 and 2009. The patients who developed NP were enrolled. The diagnosis of nosocomial pneumonia was made by the Centers for Disease Control and Prevention (CDC) definition of nosocomial pneumonia, the American Thoracic Society and the Infectious Diseases Society of America Guidelines for the management of adults with hospital-acquired, ventilator-associated and healthcare-associated pneumonia and the Thai Clinical Practice Guidelines for Prevention and Management of Adults with Hospital-acquired and Ventilator-associated Pneumonia[1,2,10]. The data on patients' characteristics, types and antibiotic susceptibility of isolated pathogens, antibiotic therapy and treatment outcomes were collected and analyzed. The subject was included if he/she had all of the following criteria; 1) age $\geq$ 18 years, 2) had infection that was developed $\geq$ 48 hours after admission that was not incubating at the time of admission, 3) two or more serial chest radiographs with at least one of the following features - a. new or progressive and persistent infiltrate, b. consolidation, c. cavitation, 4) at least one of the following features -a. temperature > 38°C with no other recognized cause, b. leukocytosis ($\geq$ 12,000 WBC/mm^3) or leukopenia (< 4,000 WBC/mm^3), c. altered mental status with no other recognized cause in adults $\geq$ 70 years), 5) at least two of the following features - a. new onset of purulent sputum, or change in character of sputum, or increased respiratory secretions, or increased suctioning requirements, b. new onset or worsening cough, or dyspnea, or tachypnea, c. rales or bronchial breath sounds, d. worsening gas exchange (*e.g.* O_2 desaturations [$PaO_2/FiO_2 \leq 240$], increased oxygen requirements, or increased ventilation demand). The patient with any one of the following criteria was excluded: 1) immunocompromised patient (absolute neutrophil < 500 cell/mm^3, leukemia, lymphoma, known HIV infection with CD4 count < 200 cell/mm^3, splenectomy, one who is in his/her transplant hospital stay, cytotoxic chemotherapy, high dose steroids daily for > 2 weeks, daily use of immunosuppressive agent for >2 weeks), 2) patient on a clinical trial which does not allow the data to be used, 3) infection caused by confirmed non-bacterial pathogens such as fungus and virus. Each eligible patient was followed until he/she left the hospital or expired. The data collected included age, gender, underlying illnesses, severity of illness, type of isolated bacteria, antibiotic susceptibility profiles of the isolated bacteria, antimicrobial regimens, clinical courses and outcomes of therapy. Assessment of clinical response was performed at 72 hours after initial antibiotic treatment, at the end of treatment (EOT) and at 7-14 days (test-of-cure, TOC) after the end of treatment. Microbiological outcome was assessed at the end of treatment. Overall 30-day mortality and infection (pneumonia)-related mortality were also determined. The data were collected and enter into the case report forms.

Definitions

Hospital-acquired pneumonia (HAP) is defined as pneumonia that occurred $\geq$ 48 hours after admission to the hospital in non-ventilated patients[1,2,11]. Ventilator-associated pneumonia (VAP) is defined as pneumonia that occurs 48 hours or more after endotracheal intubation or within 48 hours after endotracheal tube removal[1,2,11]. Early onset HAP/VAP is defined as HAP/VAP that occurs within 4 days of hospitalization or tracheal intubation[1,2,11]. Septic shock is defined as sepsis associated with evidence of organ hypoperfu-sion and a systolic blood pressure < 90 or > 30 mmHg less than the baseline or a requirement for the use of vasopressor to maintain the blood pressure.

Etiologic bacteria of HAP/VAP were determined according to the results of microbiological evaluation performed before initiation of antibiotic. The antimicrobial therapy is considered 'concordant' if no isolated bacteria are resistant to the empirical antibiotics used in the case. Pan-drug resistance, extensive drug resistance and multi-drug resistance is defined as resistance of a pathogen to all available antibiotics, resistance to all but 1 or 2 antibiotics and resistance to $\geq$ 3 classes of antimicrobial agents, respectively[20].

Statistical analyses

Data were prepared and analyzed using PASW statistics 18.0 (SPSS Inc., Chicago, IL, USA). Number and percentage were described for categorical variables, whereas continuous variables were summarized by mean, standard deviation, median, range. Chi-square test or Fisher's exact test was used to compare the proportions of categorical variables. Student's t-test or Mann-Whitney U test, as appropriate, was accessed to compare continuous variables. The magnitude of association was measured using Odds Ratio (OR) and its 95% CI. Multiple logistic regression was performed to adjust for the effects of confounders. All statistical tests were two-sided and considered statistically significant for p-value < 0.05.

Results

There were 651 NP patients enrolled to the present study. *A. baumannii* was the most common cause of NP in 198 patients (30.4%). Other isolated pathogens were *P. aeruginosa* (29.2%), *K. pneumoniae* (22%), *S. aureus* (12.7%), *E. coli* (8%) and others (5.8%). The characteristics of the patients in both groups are shown in Table 1. Most of the patients were males. The median age was 71 years. About 80% of the patients had late onset of NP with the median duration of 10 days after admission in both *A. baumannii* and non-*A. baumannii* NP. Most of NP developed in the patients hospitalized in general medical wards. VAP was more common in *A. baumannii* NP than in non-*A. baumannii* NP. The severity of sepsis, extent of pneumonia and other clinical parameters were not significantly different between *A. baumannii* NP and non-*A. baumannii* NP. Bilateral lung involvement and ARDS were significantly more common in *A. baumannii* NP. Prior antibiotic usage within 72 hours especially cephalosporins, carbapanems and glycopeptides was significantly associated with development of *A. baumannii* NP. Treatments of the patients with NP are shown in Table 2. For the initial antibiotics, colistin was more commonly used in the patients with *A. baumannii* NP. The initial antibiotics were concordant in about a half of the patients in both groups. Initial antibiotics were modified in about a half of patients in both groups and colistin was usually prescribed to the patients who received antibiotic modifications. The duration of antibiotic treatment was not significantly different in both groups with the median duration being 12 days. More patients in the *A. baumannii* NP group received mechanical ventilators and vasopressor agents.

The outcomes of treatment of NP are shown in Table 3. The initial clinical responses of *A. baumannii* NP in terms of improvement and failure was less favorable than those of non-*A. baumannii* NP. The mortality rate of NP in *A. baumannii* NP seemed to be more than that in non-*A. baumannii* NP. The time to cure in *A. baumannii* NP was longer than non-*A. baumannii* NP but there was no significant difference in the time to death. Use of mechanical ventilator was longer in the *A. baumanii* NP group. Length of ICU stay, length of hospital stay, 30-day survival and 30-day mortality from NP were not significantly different between both groups. For the microbiological outcomes, there was a trend in more persistence of the pathogen in *A. baumannii* NP.

The antibiotic susceptibility profiles of *A. baumannii* in 198 patients revealed that 14.6% were resistant to 1-2 classes of antibiotics, 15.7% were multi-drug-resistant, 69.7% were extensive-drug-resistant, and 81.8% were resistant to carbapenems.

Comparisons of treatments and outcomes of the patients with extensive drug resistant *A. baumannii* NP and NP due to other bacteria, including non-extensive drug resistant *A. baumannii*, are shown in Table 4. Concordance of initial antibiotics was higher in the extensive drug resistant *A. baumannii* NP. However, the patients with extensive drug resistant *A. baumannii* NP had less favorable responses. Extensive drug resistant *A. baumannii* NP also had a significantly lower cure rate and tended to have higher mortality due to pneumonia at the end of treatment, lower 30-day survival, and higher 30-day mortality due to pneumonia. The duration of antibiotics, duration of mechanical ventilator, length of ICU stay and length of hospital stay were not significantly different between both groups. Extensive drug resistant *A. baumannii* NP had significantly higher persistence of pathogen at the end of treatment.

Univariate analyses of the risk factors for NP due to *A. baumannii* revealed that many factors were associated with *A. baumannii* NP, as shown in Table 5.

Table 1. Characteristics of the patients with *A. baumannii* and non-*A. baumannii* NP

	A. baumannii NP (n = 198)	Non-*A. baumannii* NP (n = 453)	p-value
Male	124 (63.3%)	306 (67.8%)	0.296
Age, years Median (min, max)	70.5 (18, 97)	71.0 (18, 99)	0.059
HAP/VAP	41 (20.7%)/157 (79.3%)	171 (37.7%)/282 (62.3%)	<0.001
Severity of illness			
Non-fatal	62 (31.3%)	120 (26.6%)	0.466
Ultimately fatal	81 (40.9%)	195 (43.2%)	
Rapidly fatal	55 (27.8%)	136 (30.2%)	
Onset of nosocomial pneumonia			
Early/Late	37 (18.7%)/161 (81.3%)	94 (20.8%)/359 (79.2%)	0.619
Duration of NP after admission, days			
Median (min, max)	10 (2, 341)	10 (1, 226)	0.798
Duration of NP after mechanical ventilation, days			
Median (min, max)	8 (1, 84)	7 (2, 171)	0.833
Patient's location at diagnosis of NP			
General medical ward	79 (39.9%)	231 (51.0%)	0.012
Medical ICU	71 (35.9%)	95 (21.0%)	<0.001
General surgical ward	22 (11.1%)	56 (12.4%)	0.748
Surgical ICU	14 (7.1%)	43 (9.5%)	0.393
Other wards	12 (6.1%)	28 (6.2%)	1.000
Underlying disease			
Chronic pulmonary disease	43 (21.7%)	70 (15.5%)	0.067
Congestive heart failure	23 (11.6%)	48 (10.6%)	0.805
Acute myocardial infarction	8 (4.0%)	19 (4.2%)	1.000
Hemi- or quadriplegia	21 (10.6%)	86 (19.0%)	0.011
Cerebrovascular diseases	43 (21.7%)	112 (24.7%)	0.466
Liver cirrhosis	12 (6.1%)	28 (6.2%)	1.000
Diabetes mellitus	51 (25.8%)	108 (23.8%)	0.671
Chronic Renal Failure	26 (13.1%)	60 (13.2%)	1.000
Acute Renal Failure	26 (13.1%)	33 (7.3%)	0.025
Hemodialysis	13 (6.6%)	14 (3.1%)	0.067
Enteral feeding	36 (18.2%)	71 (15.7%)	0.497
Connective tissue disease	1 (0.5%)	2 (0.4%)	1.000
Antacid usage	32 (16.2%)	96 (21.2%)	0.168
CA lung	1 (0.5%)	10 (2.2%)	0.121
Solid tumor other than lung	9 (4.5%)	27 (6.0%)	0.589
Metastasis to lung	3 (1.5%)	6 (1.3%)	1.000
Post thoracic surgery	2 (1.0%)	10 (2.2%)	0.296
Post abdominal surgery	12 (6.1%)	16 (3.5%)	0.210
Prior antibiotic usage within 72 hours	171 (86.4%)	319 (70.4%)	<0.001
Penicillin and derivatives	43 (21.7%)	87 (19.2%)	0.528
Cephalosporins	82 (41.4%)	145 (32.0%)	0.026
Fluoroquinolones	22 (11.1%)	48 (10.6%)	0.954
Carbapanems	35 (17.7%)	51 (11.3%)	0.036
Glycopeptides	21 (10.6%)	22 (4.9%)	0.011
Macrolides	12 (6.1%)	13 (2.9%)	0.084
Category of sepsis			
Sepsis	122 (64.9%)	286 (70.4%)	0.180
Severe sepsis	29 (15.4%)	64 (15.8%)	
Septic shock	37 (19.7%)	56 (13.8%)	

Table 1. Cont.

	A. baumannii NP (n = 198)	Non-*A. baumannii* NP (n = 453)	p-value
Extent of pneumonia			
Bronchopneumonia	45 (22.8%)	136 (30.3%)	0.136
Lobar pneumonia	80 (40.6%)	172 (38.3%)	
Multilobar pneumonia	72 (36.5%)	141 (66.2%)	
Lung involvement			
Unilateral	101 (51.3%)	281 (62.3%)	0.011
Bilateral	96 (48.7%)	170 (37.7%)	
Pathogen isolated from blood	10 (5.1%)	6 (1.3%)	0.005
WBC, cells per mm^3			
Median (min, max)	14,585 (2,260, 51,000)	13,910 (1,600, 82,000)	0.137
Hematocrit, % Mean (SD)	31.4 (5.3)	31.4 (5.9)	1.000
BUN, mg/dl Median (min, max)	26.0 (5, 138)	26.0 (1, 155)	0.741
Creatinine, mg/dl Median (min, max)	1.1 (0.3, 10.58)	1.1 (0.25, 47.0)	0.469
Albumin, mg/dl Median (min, max)	2.6 (1.0, 10.8)	2.6 (1.0, 13.9)	0.593
Complications of pneumonia			
Septic shock	87 (43.9%)	171 (37.7%)	0.162
ARDS	22 (11.1%)	15 (3.3%)	<0.001
Renal failure	33 (16.7%)	49 (10.8%)	0.052
Parapneumonic effusion	6 (3.0%)	11 (2.4%)	0.860

Table 2. Treatments of the patients with *A. baumannii* NP and non-*A. baumannii* NP

	A. baumannii NP (n = 198)	Non-*A. baumannii* NP (n = 453)	p-value
Initial antibiotics			
Penicillin derivatives	57 (28.8%)	128 (28.3%)	0.965
Cephalosporins	80 (40.4%)	154 (34.0%)	0.139
Aminoglycosides	16 (8.1%)	27 (6.0%)	0.406
Fluoroquinolones	21 (10.6%)	56 (12.4%)	0.613
Carbapenems	52 (26.3%)	123 (27.2%)	0.889
Glycopeptides	20 (10.1%)	52 (11.5%)	0.704
Colistin	69 (34.8%)	61 (13.5%)	< 0.001
Concordance of initial antibiotics	103 (52.0%)	224 (49.6%)	0.622
Modification of initial antibiotics	103 (52.0%)	207 (45.7%)	0.161
Modified antibiotics			
Penicillin derivatives	27 (13.6%)	41 (9.1%)	0.105
Cephalosporins	48(24.2%)	54 (11.9%)	< 0.001
Aminoglycosides	8 (4.0%)	15 (3.3%)	0.816
Fluoroquinolones	7 (3.5%)	38 (8.4%)	0.038
Carbapenems	30 (15.2%)	67 (14.8%)	1.000
Glycopeptides	12 (6.1%)	23 (5.1%)	0.747
Colistin	58 (29.3%)	58 (12.8%)	< 0.001
Concordance of modified antibiotics	83 (82.2%)	159 (76.8%)	0.433
Duration of antibiotics, days			
Median (min, max)	12 (1, 51)	12 (1, 42)	0.156
Mechanical ventilator use	185 (93.4%)	378 (83.4%)	0.001
Use of vasopressor agents	68 (34.3%)	115 (25.4%)	0.025

Table 3. Treatment outcomes of the patients with *A. baumannii* NP and non-*A. baumannii* NP

	A. baumannii NP (n = 198)	Non-*A. baumannii* NP (n = 453)	p-value
Initial clinical response			
Improvement	87 (43.9%)	266 (58.7%)	0.001
Failure	94 (47.5%)	136 (30.0%)	< 0.001
Death	13 (6.6%)	32 (7.1%)	0.95
End of treatment response			
Cure	96 (48.5%)	255 (56.3%)	0.080
Failure	17 (8.6%)	35 (7.7%)	0.830
Death due to pneumonia	60 (30.3%)	104 (23.0%)	0.059
Time to cure, days			
Median (min, max)	14 (3, 53)	14 (2, 42)	0.042
Time to death, days			
Median (min, max)	11 (1, 130)	9 (1, 172)	0.233
Mechanical ventilator days			
Median (min, max)	11 (1, 44)	9 (1, 32)	0.013
Length of ICU stay, days			
Median (min, max)	12 (1, 70)	10 (1, 174)	0.119
Length of hospital stay, days			
Median (min, max)	14 (1, 130)	14 (1, 174)	0.612
30-day survival	75 (37.9%)	182 (40.2%)	0.642
30-day mortality from NP	67 (33.8%)	129 (28.5%)	0.201
Microbiological outcomes			
Eradication	93 (47.0%)	217 (47.9%)	0.893
Persistence	67 (33.8%)	122 (26.9%)	0.091
Super-infection	19 (9.6%)	47 (10.4%)	0.871

Multivariate analysis showed only VAP, NP developed in medical ICU and bilateral lung involvements on chest x-ray were significantly associated with *A. baumannii* as the isolated pathogen as shown in Table 5.

Discussion

The present findings revealed that *A. baumannii* NP is an important nosocomial infection causing high morbidity and mortality especially in the elderly. Most of *A. baumannii* NP patients had multiple comorbidities leading to a difficulty in management and resulting in high complications and mortality due to NP. Prior use of antibiotics especially carbapenems was associated with *A. baumannii* NP as observed in the present study and was similar to other series[21-24]. Most of the clinical features of *A. baumannii* NP, were not significantly different from those of non-*A. baumannii* NP. Although the authors found many factors associated with *A. baumannii* NP including prior use of cephalosporins and carbapenems, only VAP, NP developed in medical ICU and bilateral lung involvements on chest x-ray were shown to be associated with *A. baumannii* NP in a multivariate analysis. Therefore, any patients who developed VAP or NP in medical ICUs in tertiary care hospitals in Thailand or the patient with NP who had bilateral lung involvements on chest x-ray should be suspected of having *A. baumannii* NP and antibiotics effective against *A. baumannii* should be initiated.

The authors observed that the initial antibiotics given to NP patients were not active against isolated pathogens in approximately 50% of the patients indicating that many causative pathogens of NP were multi-drug-resistant. The outcomes of initial therapy of *A. baumannii* NP were less favorable that those of non-*A. baumannii* NP and the mortality associated with pneumonia in *A. baumannii* NP was somewhat higher than that in non-*A. baumannii* NP. The authors also observed that the patients with extensive-drug-resistant *A. baumannii* NP had less favorable responses, had significant lower cure rate, tended to have higher mortality due to pneumonia at the end of treatment, had lower 30-day survival and had higher 30-day mortality due to pneumonia than those with NP due to other bacteria including non-extensive drug resistant *A. baumannii*. The 30-day

Table 4. Comparisons of treatment and outcomes of the patients with extensive drug resistant *A. baumannii* NP and NP due to other bacteria including non-extensive drug resistance *A. baumannii*

	Extensive drug resistant *A. baumannii* (n = 138)	Non-Extensive drug resistant *A. baumannii* and non-*A. baumannii* NP (n = 453)	p-value
Concordance of initial antibiotics	74 (53.6%)	253 (49.4%)	< 0.001
Concordance of modified antibiotics	55 (82.1%)	187 (77.6%)	0.548
Initial clinical response			
Improvement	54 (39.1%)	299 (58.3%)	< 0.001
Failure	69 (50.0%)	161(31.4%)	< 0.001
Death	11 (8.0%)	34 (6.6%)	0.716
End of treatment response			
Cure	63 (45.7%)	288 (56.1%)	0.036
Failure	14 (10.1%)	38 (7.4%)	0.381
Death due to pneumonia	44 (31.9%)	120 (23.4%)	0.054
Duration of antibiotics, days			
Median (min, max)	11.5 (1, 51)	12 (1, 44)	0.510
Time to cure, days			
Median (min, max)	14 (4, 53)	14 (2, 42)	0.139
Time to death, days			
Median (min, max)	11 (1, 130)	9 (1, 172)	0.643
Mechanical ventilator days			
Median (min, max)	10 (1, 39)	9 (1, 44)	0.194
Length of ICU stay, days			
Median (min, max)	11 (1, 70)	11 (1, 174)	0.957
Length of hospital stay, days			
Median (min, max)	14 (1, 130)	14 (1, 174)	0.764
30-day survival	49 (35.5)	208 (40.5)	0.329
30-day mortality from NP	49 (35.5)	174 (28.7)	0.146
Microbiological outcomes			
Eradication	58 (42)	252 (49.1)	0.166
Persistence	54 (39.1)	135 (26.3)	0.005
Super-infection	12 (8.7)	54 (10.5)	0.636

mortality of NP in our series was similar to that from other series[4-8,21-29].

The present study results recognized that *A. baumannii* NP was common among NP in tertiary care hospitals in Thailand, that most of *A. baumannii* isolates were multidrug-resistant (including resistant to carbapenems) and that *A. baumannii* NP had a high mortality. Therefore, appropriate antibiotics against multidrug-resistant pathogens should be prescribed at the onset of NP in order to diminish morbidity and mortality due to NP[25-33]. More importantly, intensive infection control measures including VAP bundles should be emphasized and implemented in order to prevent occurrence of NP. Such infection control measures were shown to be effective in many studies[34-40].

The present study had several limitations. Many patients were immunocompromised patients who were excluded from the present study according to the present study protocol. Therefore, the present study results might not be applicable for the immunocompromised patients. Some clinical parameters were not evaluated at the time of diagnosis of NP, such as APACHE II score and arterial blood gas. Therefore, the authors were unable to confidently compare the severity of NP between groups.

Conclusion

A. baumannii is the most common causative pathogen for NP in tertiary care hospitals in Thailand. *A. baumannii* causing NP had a very high prevalence of antibiotic resistance including resistance to carbapenems. The hospitalized patient in tertiary care hospitals with VAP, or NP that was developed in medical

Table 5. Univariate and multivariate analysis of risk factors for NP due to *A. baumannii*

	Crude odds ratio (95% CI)	p-value	Adjusted odds ratio (95% CI)	p-value
Age ≤ 54 years	1.614 (1.094-2.381)	0.016	1.353 (0.873-2.096)	0.176
VAP	2.322 (1.568-3.439)	< 0.001	2.144 (1.240-3.706)	0.006
Previous cephalosporin use within 72 hours	1.502 (1.064-2.120)	0.021	1.316 (0.886-1.953)	0.174
Previous carbapenem use within 72 hours	1.693 (1.061-2.700)	0.027	1.256 (0.718-2.196)	0.424
Previous glycopeptide use within 72 hours	2.324 (1.247-4.334)	0.008	1.647 (0.795-3.411)	0.179
NP developed in medical ICU	4.858 (1.062-22.214)	0.042	1.815 (1.171-2.811)	0.008
Hemiparesis as comorbidity	0.506 (0.304-0.843)	0.009	0.603 (0.342-1.062)	0.080
Acute renal failure	1.924 (1.117-3.314)	0.018	1.133 (0.610-2.103)	0.693
Hemodialysis	2.203 (1.016-4.779)	0.045	1.825 (0.741-4.496)	0.191
Central venous catheterization	1.627 (1.084-2.442)	0.019	0.951 (0.579-1.561)	0.842
Endotracheal Intubation	2.723 (1.830-4.052)	< 0.001	1.230 (0.672-2.252)	0.501
Tracheostomy	0.589 (0.365-0.952)	0.031	0.626 (0.327-1.201)	0.159
Urinary catheterization	1.774 (1.207-2.608)	0.004	1.124 (0.674-1.874)	0.655
Nasogastric intubation	1.480 (1.015-2.160)	0.042	1.125 (0.685-1.849)	0.642
Bilateral lung involvement	1.571 (1.120-2.203)	0.009	1.535 (1.049-2.246)	0.027
Late NP	1.139 (0.746-1.740)	0.546	1.449 (0.900-2.332)	0.127
Septic shock	1.549 (0.972-2.469)	0.066	1.274 (0.760-2.136)	0.358

ICU, or NP with bilateral lung involvements on chest x-ray was likely to be due to *A. baumannii*. Many NP patients received inappropriate initial antibiotic regimens leading to a high mortality.

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Potential conflicts of interest

None.

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ปอดอักเสบติดเชื้อ *Acinetobacter baumannii* ที่เกิดในโรงพยาบาลระดับตติยภูมิในประเทศไทย

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ภูมิหลัง: *เชื้อ Acinetobacter baumannii เป็นเชื้อก่อโรคที่เป็นสาเหตุที่พบบ่อยของปอดอักเสบติดเชื้อที่เกิดในโรงพยาบาล ข้อมูลเกี่ยวกับลักษณะทางคลินิกและการดำเนินโรคของปอดอักเสบติดเชื้อ A. baumannii ในผู้ป่วยไทยยังมีจำกัด*

วัตถุประสงค์: *เพื่อทราบลักษณะทางคลินิก ปัจจัยเสี่ยง และการดำเนินโรคของปอดอักเสบติดเชื้อ A. baumannii ที่เกิดในโรงพยาบาล*

วัสดุและวิธีการ: *การศึกษานี้เป็นการเก็บข้อมูลไปข้างหน้าจากผู้ป่วยอายุมากกว่า 18 ปีที่เป็นปอดอักเสบติดเชื้อในโรงพยาบาล จากโรงพยาบาลระดับตติยภูมิจำนวน 12 แห่งในประเทศไทยระหว่าง พ.ศ. 2551 และ พ.ศ. 2552*

ผลการศึกษา: *มีผู้ป่วยปอดอักเสบติดเชื้อที่เกิดในโรงพยาบาลทั้งหมดจำนวน 651 ราย ผู้ป่วยส่วนมากเป็นชายโดยมีอายุมัธยฐาน 71 ปี เชื้อ A. baumannii ก่อโรคปอดอักเสบติดเชื้อที่สัมพันธ์กับการได้รับเครื่องช่วยหายใจประมาณร้อยละ 80 และเชื้ออื่นๆ ร้อยละ 62 ประมาณร้อยละ 80 ของผู้ป่วยเป็นปอดอักเสบที่เกิดช่วงหลังโดยมีระยะเวลามัธยฐานหลังรับไว้รักษาในโรงพยาบาลประมาณ 10 วัน ผู้ป่วยส่วนมากเกิดปอดอักเสบติดเชื้อในโรงพยาบาลขณะอยู่ที่หอผู้ป่วยสามัญอายุรศาสตร์ ในจำนวนนี้มีสาเหตุจาก A. baumannii จำนวน 198 ราย (ร้อยละ 30.4) ลักษณะทางคลินิกของปอดอักเสบที่เกิดจาก A. baumannii และกลุ่มที่ไม่ได้เกิดจาก A. baumannii ไม่แตกต่างกันอย่างมีนัยสำคัญ ผู้ป่วยทั้งสองกลุ่มประมาณร้อยละ 50 ได้รับยาต้านจุลชีพเริ่มแรกตรงกับความไวของเชื้อก่อโรค ยาโคลิสตินมักเป็นยาที่ผู้ป่วยได้รับเมื่อมีการปรับเปลี่ยนยา การตอบสนองต่อการรักษาในช่วงแรกของปอดอักเสบที่เกิดจาก A. baumannii มักด้อยกว่ากลุ่มที่ไม่ได้เกิดจาก A. baumannii อัตราตายและการคงอยู่ของเชื้อในผู้ป่วยปอดอักเสบที่เกิดจาก A. baumannii มีแนวโน้มว่ามากกว่ากลุ่มที่ไม่ได้เกิดจาก A. baumannii เชื้อ A. baumannii ส่วนมากดื้อยาต้านจุลชีพหลายขนานรวมทั้งยากลุ่ม carbapenems ผู้ป่วยที่ติดเชื้อ A. baumannii ที่ดื้อยาอย่างกว้างขวางตอบสนองต่อการรักษาน้อยกว่าผู้ป่วยกลุ่มที่ติดเชื้ออื่นหรือติดเชื้อ A. baumannii ที่ไม่ดื้อยาอย่างกว้างขวาง ปัจจัยอิสระที่สัมพันธ์กับปอดอักเสบจากเชื้อ A. baumannii คือ ปอดอักเสบติดเชื้อที่สัมพันธ์กับการได้รับเครื่องช่วยหายใจ ผู้ป่วยที่เกิดปอดอักเสบติดเชื้อในหออภิบาลอายุรศาสตร์ และปอดอักเสบติดเชื้อที่พบความผิดปกติที่ปอดทั้งสองข้างจากภาพรังสีทรวงอก*

สรุป: *เชื้อ A. baumannii เป็นสาเหตุที่พบบ่อยที่สุดของปอดอักเสบติดเชื้อที่เกิดในโรงพยาบาล ระดับตติยภูมิในประเทศไทย เชื้อ A. baumannii ส่วนมากดื้อยาต้านจุลชีพหลายขนานรวมทั้งยากลุ่ม carbapenems ผู้ป่วยปอดอักเสบติดเชื้อในโรงพยาบาลที่สัมพันธ์กับการได้รับเครื่องช่วยหายใจ หรือปอดอักเสบติดเชื้อในผู้ป่วยที่อยู่ในหออภิบาลอายุรศาสตร์ หรือและปอดอักเสบติดเชื้อที่พบความผิดปกติที่ปอดทั้งสองข้างจากภาพรังสีทรวงอกมักเกิดจากเชื้อ A. baumannii ผู้ป่วยปอดอักเสบติดเชื้อในโรงพยาบาลจำนวนมากได้รับการรักษาด้วยยาต้านจุลชีพที่ไม่เหมาะสมทำให้ผู้ป่วยมีโอกาสเสียชีวิตสูง*