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ORIGINAL RESEARCH

Correlation Between the Distribution of Virulence Genes and Drug Resistance Genes and Clinical Characteristics of Lower Respiratory Tract Infections with *Acinetobacter baumannii* and *Klebsiella pneumoniae*

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Objective: To analyze the relationship between the distribution of virulence genes and resistance genes and clinical features of lower respiratory tract infections with *Acinetobacter baumannii* (AB) and *Klebsiella pneumoniae* (KP).

Methods: Lower respiratory tract specimens from patients with lung infections in the intensive care unit of the Affiliated Hospital of Guizhou Medical University were collected in December 2023, and the study population contained 32 strains of patients with AB infections and 22 strains of patients with KP infections. Target next generation sequencing (tNGS) was used to detect the pathogenic organisms, virulence genes, and drug-resistance genes, and to analyze the changes in the clinical detection indexes of different subgroups of patients.

Results: The highest detection rate of *adeG* and *adeF* virulence genes of AB was 62.50%; the highest detection rate of *ybtE* virulence gene of KP was 54.55%. Among the AB with detected virulence genes, the resistance genes *OXA23* and *TEM* had the highest carriage; among the KP with detected virulence genes, the resistance genes *KPC, TEM* and *SHV* had the highest carriage. Patients with AB/KP infections in which the virulence gene was detected had lower Albumin (ALB) and hemoglobin (HGB), higher blood glucose (GLU), higher white blood cell (WBC) and neutrophil (NEU), and higher interleukin 6 (IL-6) and procalcitonin (PCT), compared with patients with AB/KP infections in which the virulence gene was not detected ($P < 0.05$). Patients with KP infections in which virulence genes were detected had higher GLU, higher WBC and NEU, and higher IL-6 and PCT compared with patients with AB infections in which virulence genes were detected ($P < 0.05$). Patients with KP infection without detectable virulence genes had lower HGB and higher WBC and NEU compared with patients with AB infection without detectable virulence genes ($P < 0.05$).

Conclusion: The mechanism of virulence of AB and KP is mainly related to affecting bacterial biofilm formation and iron uptake; patients with AB and KP infections in which virulence genes were detected were more likely to be resistant to penicillins, cephalosporins, and carbapenems, among others; patients with pneumoniae infections of KP appeared to be more severe than patients with pneumoniae infections of AB.

Keywords: *Acinetobacter baumannii*, *Klebsiella pneumoniae*, virulence genes, resistance genes

Introduction

Lower respiratory tract infections are clinically categorized based on acquisition settings, with community-acquired pneumonia (CAP) and hospital-acquired pneumonia (HAP) representing distinct entities in terms of etiology, risk factors, and antimicrobial resistance patterns.[1] HAP is predominantly associated with multi-drug resistant (MDR) pathogens, including carbapenem-resistant *Acinetobacter baumannii* (AB) and extended-spectrum β-lactamase-producing *Klebsiella pneumoniae* (KP).[2] While CAP typically involves pathogens like *streptococcus pneumoniae* and *Haemophilus influenzae*,

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MDR pathogens such as *AB* and *KP* are increasingly implicated in severe CAP cases, particularly among immunocompromised hosts or those with chronic comorbidities.[3] AB and KP, the two common and important conditional pathogens, are increasingly becoming a focus of clinical attention.

KP is a common gram-negative bacillus and one of the most common opportunistic pathogens, according to the virulence characteristics, KP can be classified into classical KP (cKP) and hypervirulent KP (hvKP).[4] cKP primarily affects immunocompromised individuals and is linked to hospital-associated infections. In contrast, hvKp tends to infect healthy populations through community exposure, with higher incidence among diabetic patients and those with gastrointestinal diseases, hvKp exhibits greater virulence than cKP and frequently triggers systemic invasive infections.[5] AB has a low nutritional requirement, strong resistance, can survive for a long time in the natural environment, has strong adhesion, and is also one of the common opportunistic pathogen, which can easily cause nosocomial infections in critically ill patients, such as mechanical ventilation-associated pneumonia, bloodstream infections, endocarditis, skin and soft-tissue infections, urinary tract infections, and meningitis.[6]

The degree of virulence of AB and KP is related to virulence factors, which are a number of specific and non-specific protein-like substances produced by the bacteria during colonization and infection in the host body, and these virulence factors play key roles in cytotoxicity, cellular adhesion, biofilm formation, bacterial resistance, serum resistance, iron uptake, and interactions with other bacteria.[7] The widespread dissemination of drug-resistant genes has promoted the emergence of MDR in pathogenic bacteria, and the rapid growth of antimicrobial resistance in AB and KP has undoubtedly posed a great challenge to healthcare workers and made clinical treatment more problematic,[8,9] and the relationship between virulence and infection and drug resistance has also attracted attention.

In this study, we collected lower respiratory tract specimens from patients with lung infections in the intensive care unit (ICU) of Affiliated Hospital of Guizhou Medical University in December 2023 and analyzed the relationship between the distribution of virulence genes and resistance genes and clinical features of lower respiratory tract infections with AB and KP.

Objects and Methods

Research Population

Patients with pneumonia in the ICU of the Affiliated Hospital of Guizhou Medical University in December 2023 were selected as study subjects. Inclusion criteria: 1. hospitalized patients with radiologically and clinically confirmed pneumonia caused by AB or KP, verified by microbial identification from lower respiratory tract specimens; 2. availability of complete clinical, imaging, and laboratory data; 3. informed consent provided. Exclusion criteria: cases with incomplete clinical data or ambiguous microbial results (eg, possible colonization, contamination, or polymicrobial infections). The study was approved by the Ethics Committee of Guizhou Medical University Hospital according to the Declaration of Helsinki, and all patients gave informed consent. A total of 32 patients with AB pneumonia and 22 patients with KP infection pneumonia were included. In this study, 92.5% (n = 50) of enrolled patients met CAP diagnostic criteria (symptom onset <48 hours post-admission), while 7.5% (n = 4) were classified as HAP cases.

Clinical Data Collection

Clinical data were collected from the patients, including clinical parameters on the day of specimen delivery or within 24 hours, including albumin (ALB) and hemoglobin (HGB), blood glucose (GLU), white blood cell (WBC), neutrophil percentage (NEUT%), neutrophil count (NEUT#), interleukin 6 (IL-6) and procalcitonin (PCT), the clinical data are presented in supplementary Table 1.

Detection of Virulence Genes and Resistance Genes

Lower respiratory tract specimens were collected through endotracheal aspirates (ETA) from mechanically ventilated patients or bronchoalveolar lavage fluid (BALF) from non-ventilated patients following standardized protocols. All specimens were processed within 2 hours of collection to minimize pre-analytical variability, the samples were packaged in DNase-free and RNase-free sterile cryogenic vials and tested by tNGS by Sanway Clinical Laboratories Inc. Adopt

Full-Auto Ultra-Micro Nucleic Acid Extraction Technology to complete the extraction of gene fragments at ultra-micro level (Yeast genome DNA extraction kit). Full-AutoIntelligent high-fidelity library construction technology is used to complete the gene library construction (Pathogenic microorganisms targeted gene detection library building kit-296 kinds). SWEseq multiplex amplicon sequencing technology is used to greatly improve the specificity and accuracy (FASTASeq 300 gene sequencer). Real quantitative detection of pathogens is realized by introducing quantitative internal reference, obtaining the real copy number of each detected pathogen in the sample and accurately typing the pathogen (FASTASeq300 Sequencing kit V1.0 FCM-D SE075-D). This method can detect 296 core pathogens, 1281 resistance genes and 207 virulence genes.[10]

Statistical Analysis

SPSS 23.0 software was used for data processing and analysis, the detection rate and the composition ratio were expressed as percentages (%), one-way ANOVA was used for comparison between groups and LSD-*T* test was used for multiple comparisons of clinical indicators between groups, and $P < 0.05$ was considered as statistically significant difference.

Results

Detection of AB Virulence Genes

In 32 patients with AB pneumonia, AB virulence genes were detected in 22 patients, and a total of 9 virulence genes were detected, the composition of which is shown in Figure 1B. *adeG* and *adeF* had the highest detection rate of 62.5%, followed by *lpxD* of 56.25%, *lpsB, lpsC*, and *lpsD* with a detection rate of 50%, *csuD* with a detection rate of 43.75%, and *lpxL* with a detection rate of 18.75%, and the detection rates are shown in Figure 1A.

Detection of KP Virulence Genes

In 22 patients with KP pneumonia, KP virulence genes were detected in 12 patients, and a total of 10 virulence genes were detected, the composition of which is shown in Figure 2B. *ybtE* had the highest detection rate of 54.54%, followed by *ybtP* of 45.45%, *ybtT, allB* of 27.27%, *ybtQ, allR*, and *allC* of 18.18%, *clbD*, the *clbB* and *clbM* at 9.09%, and the detection rates are shown in Figure 2A.

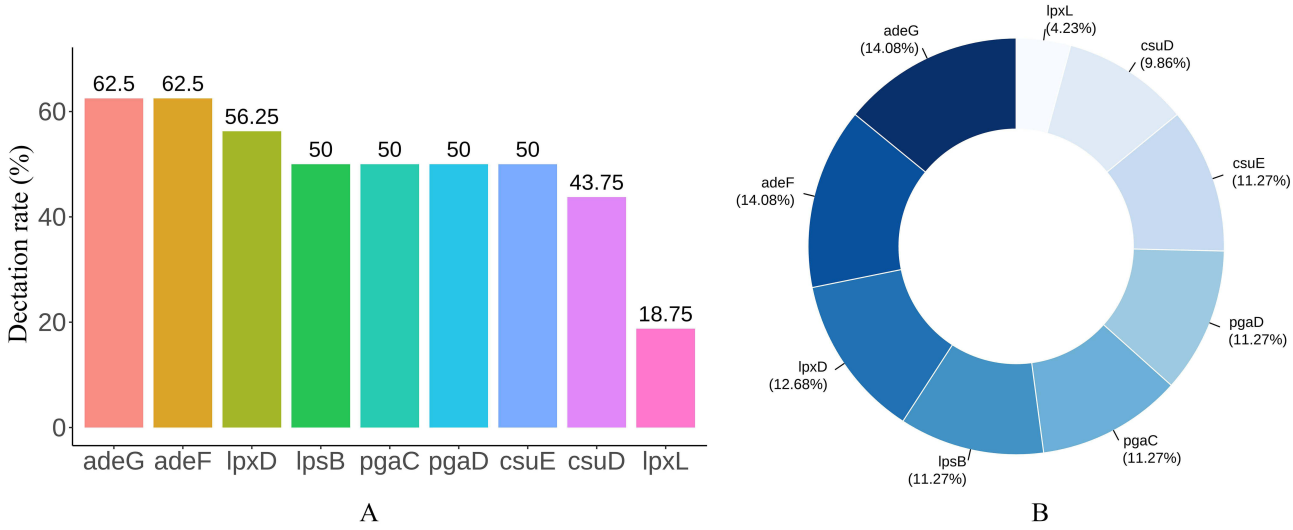

Figure 1 Detection of AB virulence genes. **A**: the detection rate; **B**: the composition ratio.

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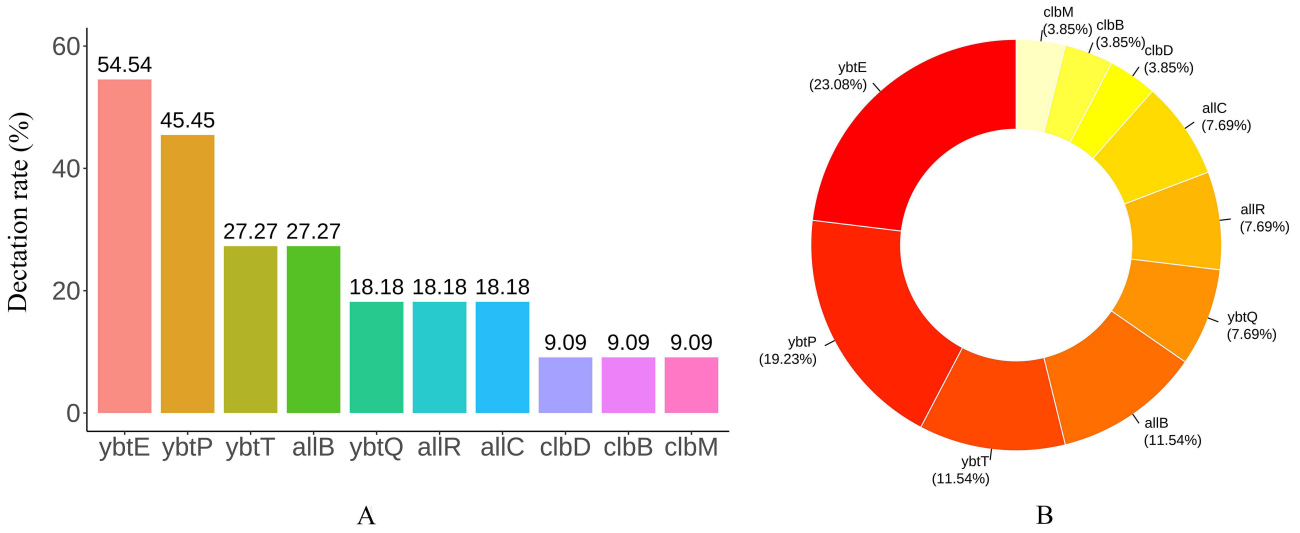

Figure 2 Detection of KP virulence genes. **A**: the detection rate; **B**: the composition ratio.

Detection of Resistance Genes in AB Carrying Virulence Genes

In 10 patients with AB infections in which virulence genes were not detected, no resistance genes were detected, and in 22 patients in which virulence genes were detected, resistance genes were detected in 20 of them, and the distribution of different virulence genes carrying resistance genes is shown in Table 1, with *OXA23* having the highest rate of detection followed by *TEM* and *ADE*, and the others being *ACR, EMR*, to*lC, AmpC*, and *tetW*.

Detection of Resistance Genes in KP Carrying Virulence Genes

Resistance genes were not detected in 10 patients with KP infections in which virulence genes were not detected, and resistance genes were detected in 10 of the 12 patients in which virulence genes were detected. The distribution of different virulence genes in carrying resistance genes is shown in Table 2, with the highest detection rates for *KPC, TEM* and *SHV*, followed by *tetW* and *cmlA*.

Comparison of Indicators of Nutritional Status in Patients with AB and KP Infections

Patients with AB infection carrying the virulence gene had lower ALB and HGB and higher GLU compared to patients with AB infection not carrying the virulence gene ($P < 0.05$); patients with KP infection carrying the virulence gene had lower ALB and HGB and higher GLU compared to patients with KP infection not carrying the virulence gene ($P < 0.05$); patients with KP infection carrying the virulence gene had higher GLU compared to patients with KP infection carrying

Table 1 Detection of Resistance Genes in AB Carrying Virulence Genes (N)

Virulence Gene	Drug Resistance Gene							
	OXA23	***TEM***	***ADE***	***ACR***	***EMR***	***tolC***	***AmpC***	***tetW***
adeG (N=20)	16	14	6	2	2	2	2	2
adeF (N=20)	16	14	6	2	2	2	2	2
lpxD (N=18)	16	14	6	2	2	2	2	2
lpsB (N=16)	16	14	6	2	2	2	2	2
pgaC (N=16)	16	14	6	2	2	2	2	2
pgaD (N=16)	16	14	6	2	2	2	2	2
csuE (N=16)	16	14	6	2	2	2	2	2
csuD (N=14)	14	14	6	2	2	2	2	2
lpxL (N=6)	6	4	2	2	2	2	2	2

Table 2 Detection of Resistance Genes in KP Carrying Virulence Genes (N)

Virulence Gene	Drug Resistance Gene				
	KPC	TEM	SHV	tetW	cmlA
ybtE (N=12)	8	8	8	2	2
ybtP (N=10)	8	8	8	2	2
ybtT (N=6)	6	6	6	0	2
allB (N=6)	4	4	4	2	0
ybtQ (N=4)	4	4	4	0	0
allR (N=4)	4	4	4	2	0
allC (N=4)	4	4	4	2	0
clbD (N=2)	0	0	0	2	0
clbB (N=2)	0	0	0	2	0
clbM (N=2)	0	0	0	2	0

the virulence gene had higher GLU compared to patients with AB infection ($P < 0.05$); patients with KP infection not carrying the virulence gene had lower HGB compared to patients with AB infection not carrying the virulence gene ($P < 0.05$), as shown in Figure 3 and Table 3.

Comparison of Inflammatory Markers in Patients with AB and KP Infections

WBC and NEU were higher in patients with AB infections carrying the virulence gene compared to patients with AB infections not carrying the virulence gene ($P < 0.05$); WBC and NEU were higher in patients with KP infections carrying the virulence gene compared to patients with KP infections not carrying the virulence gene ($P < 0.05$); WBC and NEU were higher in patients with KP infections carrying the virulence gene compared to AB infection patients had higher WBC and NEU compared to patients with KP infection that did not carry the virulence gene ($P < 0.05$); patients with KP infection that carried the virulence gene compared to patients with AB infection that did not carry the virulence gene had higher WBC and NEU ($P < 0.05$), as shown in Figure 4 and Table 3.

Comparison of Infection Severity Indicators in Patients with AB and KP Infections

IL-6 and PCT were higher in patients with AB infections carrying the virulence gene compared to patients with AB infections not carrying the virulence gene ($P < 0.05$); IL-6 and PCT were higher in patients with KP infections carrying

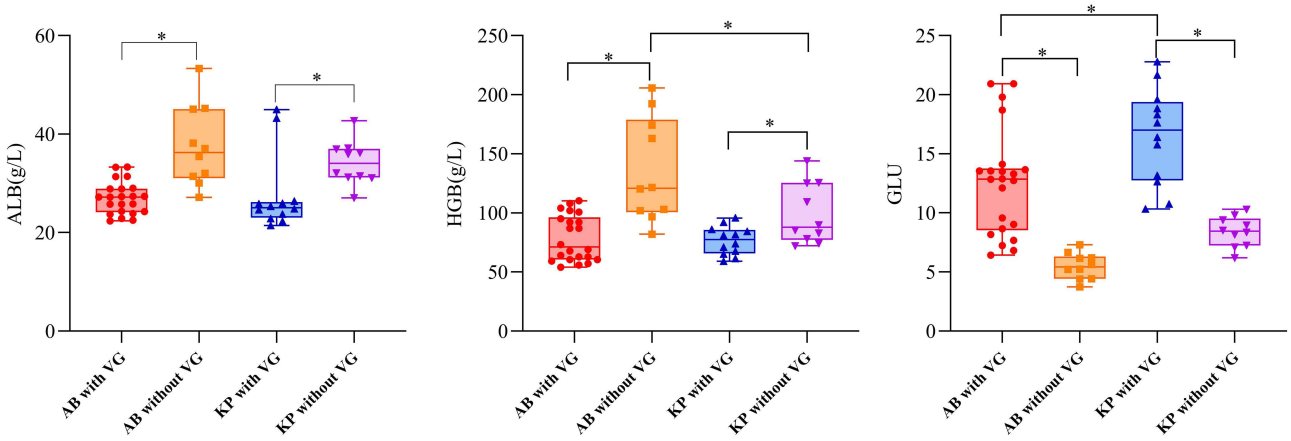

Figure 3 Comparison of indicators of nutritional status in patients with AB and KP infections.
Note: * $P<0.05$.
Abbreviations, AB, *Acinetobacter baumannii*; KP, *Klebsiella pneumoniae*; VG, virulence gene; ALB, albumin; HGB, hemoglobin; GLU, blood glucose.

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Table 3 Comparison of Clinical Indicators in Patients with AB and KP Infections

	AB with VG	AB without VG	KP with VG	KP without VG	F	P
ALB	27.00±3.29	37.50±8.17	27.60±7.86	34.21±4.43	9.876	0.000
HGB	78.43±19.38	136.10±43.97	76.52±12.01	98.8±25.45	13.662	0.000
GLU	12.56±0.75	5.49±1.11	16.47±1.01	8.41±1.11	20.682	0.000
WBC	15.67±0.95	10.73±1.41	21.76±1.28	17.08±1.41	68.810	0.000
NEUT#	13.07±4.99	8.76±2.67	19.83±3.51	14.85±5.57	235.798	0.000
NEUT%	92.32±2.07	85.33±3.08	97.31±2.81	90.96±3.08	2.792	0.050
IL-6	234.23±205.56	43.82±32.60	1945.45±265.37	185.934±290.70	11.514	0.000
PCT	10.13±11.56	2.39±4.64	58.29±35.69	5.14±3.88	24.225	0.000

Abbreviations: AB, *Acinetobacter baumannii*; KP, *klebsiella pneumoniae*; VG, virulence gene; ALB, albumin; HGB, hemoglobin; GLU, blood glucose; WBC, white blood cell; NEUT%, neutrophil percentage; NEUT#, neutrophil count; IL-6, interleukin 6; PCT, procalcitonin.

the virulence gene compared to patients with KP infections not carrying the virulence gene ($P < 0.05$); IL-6 and PCT were higher in patients with KP infections carrying the virulence gene compared to patients with AB infections carrying the virulence gene compared to patients with AB infections carrying the virulence gene ($P < 0.05$); IL-6 and PCT were higher in patients with KP infection carrying the virulence gene compared to patients with AB infection carrying the virulence gene ($P<0.05$); there was no significant difference in IL-6 and PCT between patients with KP infection carrying the virulence gene and patients with AB infection not carrying the virulence gene, as shown in Figure 5 and Table 3.

Discussion

Lung infections are a serious threat to human life and health, and rapid and accurate pathogen detection results can help clinical diagnosis and treatment, and improve patient prognosis.[11] The mNGS technology has been widely used for pathogen diagnosis of many infectious diseases including respiratory infections. tNGS has a microbial detection rate comparable to that of metagenomic next-generation sequencing (mNGS) for specimens of lower respiratory tract infections, and is able to detect pathogens, virulence genes, and resistance genes at the same time in a single assay, which is of greater health economic value compared to the costly testing of mNGS.[12] The tNGS technique is more sensitive in the detection of respiratory pathogens compared to the culture method, and the diagnosis of lower respiratory tract infections by the tNGS technique can provide an accurate guide to the treatment strategy of the patient.[13]

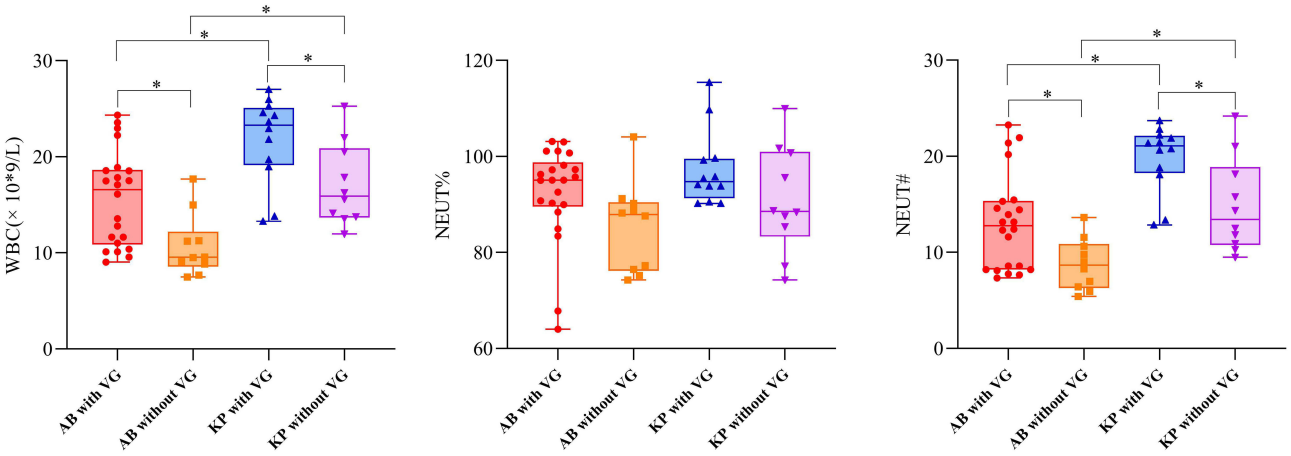

Figure 4 Comparison of inflammatory markers in patients with AB and KP infections.
Note: * $P<0.05$.
Abbreviations: AB, *Acinetobacter baumannii*; KP, *Klebsiella pneumoniae*; VG, virulence gene; WBC, white blood cell; NEUT%, neutrophil percentage; NEUT#, neutrophil count.

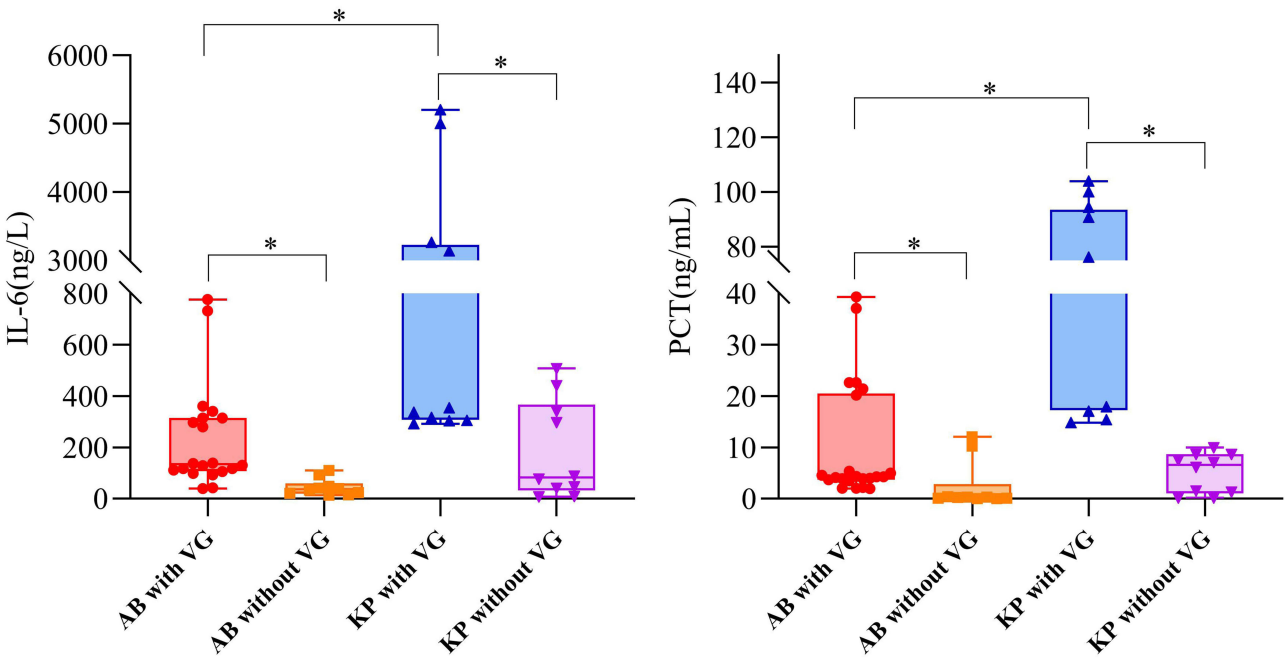

Figure 5 Comparison of infection severity indicators in patients with AB and KP infections.
Note: * *P*<0.05.
Abbreviations: AB, *Acinetobacter baumannii*; KP, *Klebsiella pneumoniae*; VG, virulence gene; IL-6, interleukin 6; PCT, procalcitonin.

Our study showed that the virulence genes *adeG* and *adeF*, which are genes involved in biofilm formation in AB and play an important role in the synthesis and translocation of self-induced molecules during biofilm formation, were detected most frequently in AB-infected patients with pneumonia.[14] The next highest detection rates were for the *lpxD, lpsB* and *lpxL* genes, which are virulence genes that are AB LPS genes associated with immune escape, evading the host immune response and triggering an inflammatory response in the host.[15] *pgaC* and *pgaD* are *PNAG* (poly-β-1,6-N-acetyl-D-glucosamine) genes associated with biofilm formation in AB and are essential for biofilm formation.[16] *csuE* and *csuD* are bacteriophage genes associated with biofilm formation in AB, which play a role in the initial stages of biofilm formation by enabling bacterial cells to adhere to abiotic surfaces and begin to form microcolonies before the biofilm structure is fully developed.[17] The results of the current study showed that *ybtE, ybtP* and *ybtT* virulence genes were detected at the highest rate in patients with pneumonia infected with KP, and *ybt* genes are genes associated with iron uptake function in KP.[18] *allB, allR* and *allC* are trophic factor genes of KP associated with Allantoin utilization, which provides a nitrogen source to increase the virulence of KP at certain sites of infection.[19] *clbD, clbB* and *clbM* are toxin genes of KP associated with colibactin, which induces DNA damage.[20]

Meanwhile, the results of the current study showed that the detection of resistance genes was higher in patients with AB with detected virulence genes and KP with detected virulence genes. More significant were the detections of *OXA*-23, *KPC, TEM* and *SHV*. Carrying *OXA*-23 and *KPC* genes can be resistant to penicillins, cephalosporins, and carbapenems.[21,22] Carrying *TEM* and *SHV* genes can develop resistance to penicillins, cephalosporins, but their activity can be inhibited by β-lactamase inhibitors.[23] Carrying the *ADE* gene can develop resistance to multiple drugs through the multidrug efflux pump.[24] Carrying the *tetW* gene can result in resistance to tetracyclines.[25] Carrying the *cmlA* gene can lead to resistance to chloramphenicol drugs.[26] Iron is essential for bacterial growth, and iron carriers can provide iron to bacteria in low iron environments with a very high iron affinity to promote bacterial growth and reproduction. HvKP produces more iron carriers than cKP and has a 6 to 10-fold increase in iron carrier activity,[8] hvKP produces four types of iron carriers: enterobacteriacein, yersiniacein, salmonellin, and aerobacteriacein. Yersinobactin is widespread in KP, but is more common in hvKP, and studies have shown that yersinobactin is significantly associated with an increased risk of invasive infections (bacteremia, liver abscess, etc). The *ybt* gene encoding yersinobactin is present on the FIBK plasmid.[27] The FIBK plasmid is widespread and highly stable in KP and many FIBK plasmids have acquired AMR

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transposons, allowing for virtually unobstructed polymerization of AMR with virulence genes, which can greatly contribute to drug resistance in hvKP strains.[28]

Biomarkers have been used to assist in categorizing diagnoses, determining the duration of antibiotic therapy, and determining the prognosis of patients with pneumonia, and a number of biomarkers have been validated to determine the ability to risk-stratify patients with pneumonia as an adjunct to clinical scoring systems.[29] Routine blood test is one of the commonly used clinical tests, which can be used to observe and analyze the changes in patients' conditions through the changes in indicators and assist patients in the determination of infectious diseases, in which the WBC count and the percentage of NEU belong to the common inflammatory indicators.[30]

Serum ALB is the major multifunctional protein, which accounts for 55–60% of all plasma proteins and responds to the nutritional status of the body, energy supply, changes in the hormonal milieu, and systemic inflammatory responses.[31] ALB is an independent predictor of morbidity and mortality in a wide range of diseases and is associated with systemic inflammation; the more severe the disease, the lower the ALB concentration, and hypoalbuminemia is associated with higher mortality.[32] HGB is also the main component of protein in the human body, and anemia is mainly defined by a decrease in HGB, and any degree of anemia can be used as an independent risk factor leading to illness, death, or debilitation in the elderly.[33] Pulmonary sensory disease as a consumptive disease, after the onset of the disease causes patients to lose body mass, wasting, which inhibits the function of the body's bone marrow hematopoietic system, causing anemia, patients with pulmonary infections have a corresponding increase in the secretion of inflammatory factors, the activity of the macrophage system is enhanced, and the destruction of erythrocytes is increased, which results in infectious anemia, and a lower hemoglobin content will make the oxygen-carrying capacity of erythrocytes decrease, affecting the lower HGB content will reduce the oxygen carrying capacity of erythrocytes, affecting the oxygen supply of the body, and the lung tissue in a state of hypoxia will aggravate the symptoms of lung infection.[34]

Diabetes mellitus, especially those with poor glycemic control, is a high-risk group for hvKP infection, which is characterized by increasing incidence, rapid progression, and high morbidity and mortality year by year.[35] The mechanisms of susceptibility to hvKP in diabetic patients include both host and microbial aspects, specifically the disruption of the mucosal barrier in diabetic patients, the increased translocation of colonizing bacteria, immune abnormalities, high titer bacteremia and microthrombosis, and the high glucose environment affects hvKP carbon source metabolism and iron metabolism, thus enhancing the virulence and escape ability of hvKP.[36]

IL-6 is a soluble mediator with pleiotropic effects on inflammation, immune response, and hematopoiesis, and it induces acute phase protein synthesis in hepatocytes while inhibiting albumin production, IL-6 expression is produced in response to eg infection and tissue damage, and IL-6 and TNF-α expression levels are associated with early death in patients with pneumonia.[37] Studies have shown that IL-6 levels are significantly increased in patients with pneumonia, and changes in IL-6 levels in patients with severe pneumonia can be used to assess the severity of the disease in patients with pneumonia and to determine the prognosis of the patients.[38] PCT is the most commonly used biomarker in the diagnosis and treatment of pneumonia, and serum PCT levels can be used as a diagnostic and prognostic indicator of pneumonia.[39] Studies have shown that PCT level at admission is a better biomarker for the assessment of pneumonia severity and prognosis compared with WBC and pneumonia levels, and the level of PCT expression in the blood is positively correlated with the progression of severe pneumonia, and the level of PCT changes with the severity of the disease.[40] PCT can not only identify bacterial and nonbacterial infectious pneumonia but also the application of antibiotics and the timing of discontinuation of antibiotics PCT can be used to identify bacterial infections from non-bacterial infections, and the timing of antibiotic application and discontinuation can also be determined according to PCT expression levels.[41]

The results of the current study showed that AB/KP with detected virulence genes had lower ALB and HGB, higher GLU, WBC, NEU, IL-6, and PCT compared to AB/KP with no virulence genes detected. KP with detected virulence gene had higher GLU, WBC, NEU, IL-6 and PCT compared to AB with detected virulence gene. KP without detected virulence gene had lower HGB, higher WBC and NEU compared to AB without detected virulence gene. The findings suggest that patients with KP-infected pneumonia appear to be more severe than those with AB-infected pneumonia.

The predominance of CAP cases (92.5%) in our cohort highlights an emerging clinical challenge: severe community-onset infections caused by traditionally "nosocomial" pathogens like AB and KP. This aligns with global reports of AB

and KP causing fatal CAP in diabetic patients and immunocompromised individuals.[42,43] However, the limited HAP sample size precludes direct comparisons of virulence/resistance gene profiles between CAP and HAP subgroups-a key limitation. Previous studies have shown that virulence genes and resistance genes differ in AB and KP infections in patients with CAP and HAP, such divergence likely stems from differing selection pressures, hospital environments favor resistance gene accumulation, while community settings may select for hypervirulence.[44,45]

Limitation

Limitations of this study were that the majority of our study participants were patients with CAP and fewer patients with HAP, and our current study design did not include longitudinal follow-up of mortality endpoints. Future multicenter studies stratifying CAP/HAP cohorts are urgently needed to clarify these context-dependent mechanisms and propose mortality correlation analyses as a priority for future longitudinal studies.

Conclusion

Studies of virulence genes, resistance genes, and the relationship with clinical indicators may increase the understanding of the mechanisms of resistance and virulence of the bacteria involved, as well as provide new ideas and approaches for the future study of inter-bacterial and bacteria-host interactions. Clinicians managing severe CAP should remain vigilant, particularly in regions with high AB/KP community prevalence.

Data Sharing Statement

The datasets used during the current study are available from the corresponding author on reasonable request.

Ethics Approval and Consent to Participate

The study was submitted to The Ethics Committee of Affiliated Hospital of Guizhou Medical University.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no competing interests in this work.

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