JIDC | THE JOURNAL OF INFECTION IN DEVELOPING COUNTRIES

Original Article

The role of syndromic testing in pneumonia diagnosis: a comparison with culture methods

Ade Dharmawan[1], Pusparini[2]

[1] Department of Clinical Microbiology, Faculty of Medicine and Health Sciences, Krida Wacana Christian University, Jakarta, Indonesia
[2] Department of Clinical Pathology, Faculty of Medicine, Universitas Trisakti, Jakarta, Indonesia

Abstract

Introduction: Pneumonia is an infection in the pulmonary tissue that is caused by bacteria, viruses, fungi, or parasites. Based on Indonesian Health Survey data for 2023, the prevalence of pneumonia was 10.8%. The rapid detection of pathogens accompanied by their antibiotic sensitivity pattern is crucial to obtain relevant outcomes. The gold standard test by bacterial culture needs approximately 72 hours. In order to speed this up, the PCR-based test was developed, such as syndromic testing.

Methodology: The present study was a retrospective study conducted from May 2021 to July 2024, using total sampling, on BAL and sputum specimens, with as inclusion criteria of patients aged ≥ 18 years diagnosed with pneumonia on the basis of bacterial culture and the pneumonia panel test.

Results: A total of 147 specimens were collected, with a 66.7% predominance of males and a mean age of 67.24 ± 18.23 years. There was 72.11% correspondence between the two tests, with the proportion of pathogens being 67.35% from the pneumonia panel test and 55.1% from culture. The distribution of pathogens in both tests was dominated by *K. pneumoniae*, whereas the most frequent antibiotic resistance genes were CTX-M and IMP. The antibiotics that may still be of choice for the therapy of Gram-negative bacteria are tigecycline and amikacin, while for *P. aeruginosa,* the antibiotic of choice is piperacillin-tazobactam.

Conclusions: The pneumonia panel test provides higher positivity rates, faster results, and detects resistance genes, but lacks coverage for fungi and *S. maltophilia*.

Key words: Culture; pneumonia panel; syndromic testing.

J Infect Dev Ctries 2026; 20(1):62-69. doi:10.3855/jidc.21072

(Received 14 November 2024 – Accepted 21 July 2025)

Introduction

Pneumonia is an acute respiratory tract infection affecting the pulmonary tissues, which may be caused by bacteria, viruses, fungi, or parasites. Pneumonia is frequently the cause of an individual becoming hospitalized and is one of the principal causes of morbidity and mortality throughout the world. Based on the Indonesian Health Survey (*Survei Kesehatan Indonesia*) data for 2023, the prevalence of pneumonia in Indonesia was found to be 10.8%, with the highest prevalence of 12.6% at ≥ 75 years. A study in Mongolia on patients ≥ 18 years old found an incidence of hospitalized pneumonia patients of 17.65% in the year 2018 [1,2].

The rapid detection of pathogens accompanied by their antibiotic sensitivity pattern is crucial to the administration of appropriate therapy, such that it may produce relevant clinical outcomes. Delay in the administration of antibiotics may result in prolonged hospitalization and increased mortality rate. The current gold standard for the microbiological diagnosis of pneumonia is the culture method, which needs approximately 72 hours to identify bacteria and their resistance pattern. Apart from this, conventional methods are frequently also less sensitive and are capable of detecting only 30 - 50% of pathogens in patients with a diagnosis of pneumonia. This may be influenced by prior empirical antibiotic administration as well as by poor pre-analytical preparation for fastidious bacteria requiring special conditions. Therefore, there is a need for rapid and reliable tests for pneumonia [3–5].

To overcome these limitations, several tests based on the polymerase chain reaction (PCR) have been developed to detect viral and bacterial pathogens and antibiotic resistance genes. This has the potential to revolutionize therapies by directly providing information to physicians about the existing pathogens and their probable antibiotic sensitivity by detecting genotypic resistance markers. Several studies have shown the superior diagnostic accuracy of the PCR-based platforms to detect bacterial pathogens in sputum as compared with standard culture. The BioFire FilmArray® pneumonia panel (BioFire Diagnostics

LLC, Salt Lake City, UT, USA) constitutes a multiplex PCR test that can detect 15 typical bacteria, 3 atypical bacteria, 8 respiratory viruses, and 7 antibiotic resistance markers. This test gives semi-quantitative results for 15 bacteria within approximately 1 hour and was approved by the FDA (Food and Drug Administration) in 2018 [6–9]. The present study was conducted to compare pneumonia panel syndromic testing with bacterial culture in sputum and broncho-alveolar lavage (BAL) specimens.

Methodology

This was a retrospective study at a private hospital in Jakarta from May 2021 – July 2024. The samples used were sputum and BAL specimens from patients with a diagnosis of pneumonia. The specimens were collected by total sampling, which obtained 147 specimens from sputum (100 specimens) and BAL (47 specimens). The applied inclusion criteria were, among others, patients aged ≥ 18 years with a diagnosis of pneumonia, who underwent pneumonia panel syndromic testing and resistance cultures in parallel. The exclusion criteria comprised samples for follow-up tests or treatment evaluations and poor-quality sputum. This study obtained ethical clearance from the Research Ethics Committee, Faculty of Medicine and Health Science UKRIDA under No. 1861/SLKE/IM/UKKW/FKIK/KEPK/X/2024.

Culture Tests

Cultures were conducted according to standard operating procedures for laboratories. Sputum quality was evaluated using American Society for Microbiology (ASM) criteria by Gram staining of the samples with an epithelial cell count of < 10/lpf or a leukocyte/SEC ratio of ≥ 10 and > 5 microbes per field at 100× magnification. All specimens meeting the requirements were then inoculated in blood agar, chocolate agar, and MacConkey agar (Oxoid, Thermofisher Scientific, Massachusetts, USA) for bacterial identification and sensitivity testing using the Vitex®-2 system (bioMérieux, Marcy l'Étoile, France).

Table 1. Distribution of Pneumonia Panel and Culture Results in BAL Specimens (n = 47) and Sputum Specimens (n = 100).

Pneumonia panel	Culture	
	Pathogens +	Pathogens -
BAL (47 specimens)		
Pathogens +	29	5
Negative	4	9
Sputum (100 specimens)		
Pathogens +	44	21
Negative	4	31

BioFire FilmArray® Pneumonia panel

Based on the manufacturer's instructions, the pneumonia panel test was done using the included flocked swabs for sputum and BAL specimens. The swabbed samples were mixed with sample buffer and injected into the pneumonia panel cartridge, which was then inserted into the FilmArray instrument for analysis. The BioFire FilmArray® pneumonia panel is a closed-pouch system that performs PCR multiplex nested extraction of nucleic acids and final analysis. The panel tests 15 typical bacterial pathogens, consisting of 11 Gram-negative species (*Acinetobacter calcoaceticus-baumannii* complex, *Enterobacter cloacae, Escherichia coli, Haemophilus influenzae, Klebsiella aerogenes, Klebsiella oxytoca, Klebsiella*

Table 2. Correspondence vs. noncorrespondence between pneumonia panel and culture test results for BAL and sputum specimens.

No.	Pneumonia Panel	Culture
BAL specimens		
1	Negative	*Candida tropicalis*
2	Negative	*Candida albicans*
3	*Pseudomonas aeruginosa*	*Candida albicans*
4	*Haemophilus influenzae*	Negative
5	*Haemophilus influenzae*	Negative
6	*Human Rhinovirus/Enterovirus*	Negative
7	*Influenza A*	Negative
8	*Coronavirus*	*Klebsiella pneumoniae*
9	Negative	*Raoultella planticolae*
10	*S. aureus, H. influenzae*	*Klebsiella pneumoniae*
11	Negative	*Rhizobium radiobacter*
12	*Acinetobacter baumannii*	Negative
13	*Coronavirus*	*Klebsiella pneumoniae*
Sputum specimens		
1	*S. aureus*	Negative
2	Negative	*C. albicans*
3	Negative	*S. aureus*
4	*H. influenzae, M. catarrhalis*	Negative
5	*K. pneumoniae*	Negative
6	*S. pneumoniae*	Negative
7	*S. aureus*	Negative
8	*A. baumannii*	*E. faecium*
9	*A. baumannii, Parainfluenza viruses*	Negative
10	*Human Rhinovirus/Enterovirus*	Negative
11	*Human metapneumovirus*	Negative
12	*Parainfluenza Viruses*	Negative
13	*H. influenzae, Adenovirus, Human Rhinovirus/Enterovirus*	Negative
14	Negative	*E. coli*
15	*A. baumannii*	*S. maltophilia*
16	*M. pneumoniae, Human Rhinovirus/Enterovirus, Influenza A*	Negative
17	Negative	*C. albicans*
18	*Human Rhinovirus/Enterovirus*	*S. aureus*
19	*H. influenzae, Parainfluenza viruses*	*C. glabrata*
20	*Human Rhinovirus/Enterovirus*	Negative
21	*Influenza A*	Negative
22	*Influenza A*	Negative
23	*H. influenzae*	Negative
24	*Respiratory Syncytial Viruses*	Negative
25	*H. influenzae*	*S. maltophilia*
26	*Influenza A*	Negative
27	*M. catarrhalis, S. agalactiae*	Negative
28	*Coronavirus*	Negative

pneumoniae, Moraxella catarrhalis, Proteus spp., Pseudomonas aeruginosa, Serratia marcescens), and four Gram-positive species (*Staphylococcus aureus, Streptococcus agalactiae, Streptococcus pneumoniae, Streptococcus pyogenes*), three atypical bacterial pathogens (*Chlamydia pneumoniae, Legionella pneumophila, Mycoplasma pneumoniae*), nine viral pathogens (Adenovirus, Human metapneumovirus, Human rhinovirus/Enterovirus, Influenza A, Influenza B, Parainfluenza viruses, Respiratory Syncytial viruses), and seven antibiotic resistance genes (MecA/C and MREJ, CTX-M, NDM, IMP, OXA-48 like, KPC and VIM). The results for typical bacteria are reported semi-quantitatively, giving the estimated amount of bacterial nucleic acids, with bins allowing the detection of around 10^4, 10^5, 10^6, or $\geq 10^7$ copies/mL [3].

Results

The total collected sample was 147 patients, with a 66.7% predominance of males in comparison with 33.3% females. The age range of the patients in this study was 18–98 years, with a mean age of 67.24 ± 18.23. The collected specimens consisted of 47 BAL and 100 sputum specimens. The results of the pneumonia panel and culture tests may be seen in Table 1. The number of corresponding vs noncorresponding pathogens that were found in the BAL specimens was 34 corresponding and 13 noncorresponding pathogens between these two tests (Table 2). In both tests, among 100 sputum specimens, 72 were corresponding specimens and 28 specimens were noncorresponding (Table 2).

In the pneumonia panel test results, there were 147 specimens, comprising 99 pathogen-positive specimens (67.35%) with 52 specimens yielding a single pathogen, 33 specimens yielding 2 pathogens, and 14 specimens yielding > 2 pathogens, while in 48 specimens (32.65%) no pathogens were found. The 3 most frequent pathogens were *K. pneumoniae* at 21.25%, and *A. baumannii* and *P. aeruginosa* at 13.13% each (Table 3).

In the culture test results of 147 sputum and BAL specimens, 81 specimens (55.1%) yielded pathogens, while 66 specimens (44.9%) did not. These negative results indicate either no bacterial growth observed in culture or the growth of *Streptococcus viridans* with bacterial counts below the diagnostic threshold for BAL specimens (< 10,000 CFU/mL). Of the 81 pathogen-yielding specimens, there were 78 specimens with a single pathogen, whereas 3 specimens yielded 2 pathogens (bacteria and fungi). In the study results, there were 3 of the most frequent pathogens according to culture tests, namely *K. pneumoniae* at 28.57%, *P. aeruginosa* at 17.86% and *S. aureus* at 14.29% (Table 3).

Table 3. Distribution of pneumonia-causing pathogens from pneumonia panel and culture tests.

Pathogen	Pneumonia panel		Culture	
	n	%	n	%
K. pneumoniae	34	21.25	24	28.57
A baumannii	21	13.13	11	13.10
P. aeruginosa	21	13.13	15	17.86
S. aureus	17	10.63	12	14.29
E. coli	11	6.88	8	9.52
H. influenzae	10	6.25	0	0
Human rhinovirus/enterovirus	10	6.25	-	-
Influenza A	8	5.00	-	-
S. agalactiae	5	3.13	0	0
C. albicans	-	-	5	5.95
Coronavirus	4	2.50	-	-
E. cloacae	3	1.88	0	0
Parainfluenza viruses	3	1.88	-	-
M. catarrhalis	2	1.25	0	0
M. pneumoniae	2	1.25	0	0
S. pneumoniae	2	1.25	0	0
Human metapneumovirus	2	1.25	-	-
C. glabrata	-	-	2	2.38
S. maltophilia	-	-	2	2.38
K. aerogenes	1	0.63	0	0
S. marcescens	1	0.63	0	0
Proteus spp.	1	0.63	0	0
Adenovirus	1	0.63	-	-
Respiratory Syncytial Viruses	1	0.63	-	-
E. faecium	-	-	1	1.19
Raoultella planticolae	-	-	1	1.19
Rhizobium radiobacter	-	-	1	1.19
C. parapsilosis	-	-	1	1.19
C. tropicalis	-	-	1	1.19
Total	160	100	84	100

From the distribution of pathogens, it was found that 47 specimens had resistance genes that were detected with the pneumonia panel, consisting of 46 resistance genes of Gram-negative bacteria and 1 resistance gene of Gram-positive bacteria (Table 4), with 23 specimens having a single resistance gene, 17 specimens having 2 resistance genes, and 7 specimens having > 2 resistance genes.

Based on Tables 5 and 6, the sensitivity test results for Gram-negative bacteria show that *K. pneumoniae, A. baumannii,* and *E. coli* still had a high degree of sensitivity to amikacin and tigecycline, while for *P. aeruginosa,* the antibiotics were amikacin and piperacillin-tazobactam. For Gram-positive bacteria, both *S. aureus* and *E. faecium* were still 100% sensitive to vancomycin, linezolid, and tigecycline.

Table 4. Distribution of Resistance Genes in Gram-Negative and Gram-Positive Bacteria.

Resistance Gene	n	%
Gram Negative		
CTX-M	30	37.97
IMP	22	27.85
NDM	17	21.52
OXA-48-like	8	10.13
KPC	1	1.27
Gram Positive		
MecA/C and MREJ	1	1.27
Total	79	100

Discussion

In this study, the majority of the patients were males at 66.7% and the mean age of the total sample was 67.24 years. These results were similar to the results of the study conducted by Gong *et al.,* who found that the majority of patients with pneumonia were males at 70.6%, with a mean age of 64 ± 20 years [10]. The risk factor causing males to be more susceptible to the occurrence of pneumonia is that males smoke more than females. Smoking causes a person to be more susceptible to bacterial infection, because smoking causes changes in the structure of the bronchial mucosal epithelium, including loss of cilia, enlargement of the mucosal glands, increased number of goblet cells, and disturbance of several important functions of the innate and adaptive immune systems, that may support the presence and distribution of microbes in the bronchial tract. Other factors that play a role are hormones. Estrogens in females function as protection against pneumonia by increasing the ability of the body to eliminate bacteria, reducing inflammation in the lungs, and increasing the survival level. In an in vitro experiment, pulmonary macrophages of female rats and women showed a higher effectiveness in killing accumulated bacteria, caused by activation of nitrogen oxide-3 (NOS3) synthase by estrogens. This contributes to increasing the ability of macrophages in the killing of bacteria, accelerating bacterial cleansing, and increasing host survival in cases of primary and secondary pneumonia (after influenza infection) [11]. In the pneumonia panel test the total number of positive specimens reaches 99/147 (67.35%), whereas in culture tests the total number of positive specimens is 81/147 (55.10%). This may be seen from the fact that the number of positives from using the pneumonia panel is higher than in the culture method. These results are similar to those of a study conducted by Zacharioudakis

Table 5. Sensitivity Pattern of Gram-Negative bacteria from culture tests.

Gram-Negative Bacteria	Number of isolates	AMK %S	GEN %S	AMP %S	ETP %S	MEM %S	CAZ %S	CRO %S	FEP %S	ATM %S	SAM %S	TZP %S	SXT %S	CIP %S	LVX %S	TGC %S
Klebsiella pneumoniae	24	83	63	IR	58	58	46	46	54	46	42	50	46	50		100
Pseudomonas aeruginosa	15	80	60	0	IR	60	60	IR	47	60	IR	80	IR	47		IR
Acinetobacter baumannii	11	73	36	IR		36	0	0	9	IR	55	9	55	9		100
Escherichia coli	8	88	38	0	88	88	25	25	25	25	25	25	25	25		100
Stenotrophomonas maltophilia	2	IR	IR	IR	IR	IR		IR		IR	IR	IR	100		100	
Raoultella planticolae	1	100	100	0		100	100	100	100	100	100	100		100		100
Rhizobium radiobacter	1	100	100	0		100	100	100	100	100	100	100	100	100	100	100

AMK: Amikacin; GEN: Gentamicin; AMP: Ampicillin; ETP: Ertapenem; MEM: Meropenem; CAZ: Ceftazidime; CRO: Ceftriaxone; FEP: Cefepime; ATM: Aztreonam; SAM: Ampicillin-Sulbactam; TZP: Piperacillin-Tazobactam; SXT: Trimethoprim-Sulfomethoxazole; CIP: Ciprofloxacin; LVX: Levofloxacin; TGC: Tigecycline; IR: Intrinsic Resistant).

Table 6. Sensitivity Pattern of Gram-Positive bacteria from culture tests.

Gram-Positive Bacteria	Number of isolates	GEN %S	AMP %S	AMC %S	SXT %S	CIP %S	LVX %S	TCY %S	PEN %S	OXA %S	VAN %S	ERY %S	LNZ %S	MFX %S	CLI %S	TGC %S
Staphylococcus aureus	12	83	0	83	83	67	75	67	33	83	100	67	100	75	83	100
Enterococcus faecium	1	0	0	0	IR	0	0	0	0		100	0	100		IR	100

AMC: Amoxicillin-Clavulanate; TCY: Tetracycline; PEN: Penicillin; OXA: Oxacillin; VAN: Vancomycin; ERY: Erythromycin; LNZ: Linezolid; MFX: Moxifloxacin; CLI: Clindamycin).

et al., who found that the pneumonia panel may detect a total number of pathogens of 74.3%, higher than with the culture test, which reaches 41.4% [12]. Similar reports were obtained in the study conducted by Yoo *et al*., who obtained higher positive results with the pneumonia panel as compared with culture (59.7% vs 37.3%) [13]. These results were obtained because the pneumonia panel constitutes a polymerase chain reaction (PCR)-based test that can detect both live and dead bacteria and extracellular bacterial DNA [14]. For pathogens that are found with the pneumonia panel and culture, when at least one identical pathogen is found, this study found a correspondence of 72.11% between both tests. These results are not very different from the study conducted by Lee *et al*., who obtained a correspondence of 79% between both tests [15]. Another study conducted by Westhuyzen *et al.* also found a correspondence of 80% between the pneumonia panel and culture test [16]. In our study, it was found that culture was unable to detect viruses and fastidious bacteria, such as *H. influenzae, S. pneumoniae, M. catarrhalis,* and *M. pneumoniae*, when compared with the pneumonia panel that can detect such bacteria. This may have been caused by the small number of bacteria or by prior administration of antibiotics. These results are similar to those of Zacharioudakis *et al*., who were unable to detect fastidious bacteria with the culture test [12]. Fastidious bacteria have difficulty growing in routine culture conditions, and their growth may be inhibited by the growth of normal flora, such that they lose their viability [4,16]. On the other hand, the limitation of the pneumonia panel is that it cannot detect fungi and pathogenic bacteria such as *Stenotrophomonas maltophilia*. The present study found *Candida sp.* and *S. maltophilia* in the proportions of 6.12% and 1.36%, respectively. There are a sufficient number of fungi that cause pneumonia in Indonesia, especially in patients with severe pneumonia. The study conducted by Singh *et al.* in Cipto Mangunkusumo Hospital (RSCM) found that the proportion of pneumonia caused by fungi was 5.97% [17]. Similar results were found by the study conducted by Webber *et al.,* who found that among pathogens causing pneumonia 13% were fungi and 2% *S. maltophilia* [14]. Other studies conducted by Jitmuang *et al.* and Layanto *et al.* found relatively high prevalences of *S. maltophilia* of 16.7% and 25.31%, respectively [18,19]. The absence of *S. maltophilia* as a target in the pneumonia panel may be a problem, because this bacterium frequently constitutes a pathogen in cases of Hospital Associated Pneumonia (HAP) and Ventilator Associated Pneumonia (VAP), while this bacterium has intrinsic resistance against various antibiotics [18]. *S. maltophilia* may cause significant morbidity and mortality. The study by Guerci *et al.* found mortality figures of 49.7% as a result of infection by *S. maltophilia* among pneumonia patients in intensive care units [20].

Discrepancies were observed between the pneumonia panel results and conventional culture methods in pathogen detection. While most undetected pathogens consisted of viruses and fastidious bacteria, the pneumonia panel successfully identified several non-fastidious bacterial species - including *Staphylococcus aureus*, *Klebsiella pneumoniae*, and *Acinetobacter baumannii* - that failed to grow in culture. This discrepancy may be attributed to prior antibiotic administration before specimen collection, representing a notable study limitation, as we could not differentiate between patients who had or had not received empirical antibiotics. These findings corroborate previous studies by Yoo *et al.,* who similarly reported pneumonia panel detection of non-fastidious organisms (*Serratia marcescens*, *Klebsiella oxytoca*, and *Proteus* spp.) with negative culture results. Comparable results were demonstrated in Webber *et al.*'s study, which identified additional non-fastidious species (*S. aureus*, *E. cloacae*, *S. agalactiae*, and *Proteus* spp.) detected exclusively by the pneumonia panel. Webber's research further established that specimen type and bacterial load serve as significant predictors for pneumonia panel detection. The observed enhanced sensitivity of PCR-based methods stems from their capacity to detect both viable and non-viable bacteria along with extracellular bacterial DNA, representing a key advantage over traditional culture techniques [13,14]

In the present study, we found that the most frequent cause of pneumonia was *K. pneumoniae* at 21.25% using the pneumonia panel and 28.57% using the culture test. Other pathogens that were also frequently found were other Gram-negative bacteria, such as *P. aeruginosa* and *A. baumannii,* both at 13.13%, whereas the most frequent viruses were human rhinovirus/enterovirus that contributed a prevalence of 6.25%. These results are also similar to the study conducted in Bandung by Soeroto *et al*., who found that the 3 most frequent pathogens found in patients with pneumonia were *K. pneumoniae*, *A. baumannii,* and *P. aeruginosa* at 23.9%, 19.4%, and 18.1%, respectively [21]. Similar results were also found in several studies that were conducted in Indonesia, among others, the study by Susanto *et al.,* conducted at Dr. Soedarsono Hospital in Pasuruan, found the most frequent pathogen

to be *K. pneumoniae* at 25.4%, followed by *A. baumannii* at 15.3% [11]. The study conducted at Budhi Asih Hospital in Jakarta by Dharmawan *et al.* found that the most numerous pathogens causing community-acquired pneumonia (CAP) were *K. pneumoniae* at 29.6%, followed by *A. baumannii* at 10.2% [5]. In the results found by the study of Carugati *et al*., the most numerous pathogens causing pneumonia were *S. pneumoniae* at 8.2%, followed by Gram-negative bacteria such as *P. aeruginosa* and *K. pneumoniae*. A meta-analysis conducted by Shoar *et al.* also concluded that the most common pathogen causing pneumonia was *S. pneumoniae* [22,23]. These differences in etiology may have been caused by regional differences; as had been mentioned previously, the majority of pathogens in Indonesia are Gram-negative bacteria. This may have been influenced by colonization of the nasopharynx by bacteria that mostly have the potential to become pneumonia-causing pathogens. The study conducted by Farida *et al.* in Semarang found a prevalence of 15% in *K. pneumoniae* colonizing the nasopharynx of adults, whereas other Gram-negative bacteria had a prevalence of 19%. This is associated with the relatively poor hygiene of foods and drinks [24]. In this study, we identified *Raoultella planticola* and *Rhizobium radiobacter*, which are rarely pathogenic. However, we decided to report both isolates as clinically significant pathogens based on the following considerations: (1) BAL cultures from both patients showed pure growth of a single isolate, (2) bacterial counts exceeded the diagnostic threshold (> 10,000 CFU/mL), and (3) clinical correlation supported a diagnosis of pneumonia in both cases.

This study found that the prevalence of the ESBL CTX-M gene was 37.97%, followed by the carbapenemase IMP gene at 27.85%, NDM at 21.52% and OXA-48-like at 10.13%. Similar results were found in the study of Jitmuang *et al.*, where the most frequent resistance genes detected were CTX-M at 42.9%, whereas our finding for carbapenemase was slightly different from their study, which found that the most numerous genes were OXA-48-like at 25%, followed by NDM at 14.3%. Of the 6 CTX-M genes that were detected singly with corresponding bacteria found between culture and pneumonia panel, all had phenotypic resistance to 3rd generation cephalosporins, such that there was a 100% correspondence between genotypes and phenotypes. In our study, of the 28 carbapenemase genes that were detected in corresponding bacteria between culture and pneumonia panel, there were 26 corresponding genes between bacteria with ≥ 1 carbapenemase gene (IMP, NDM, KPC, OXA-48-like) and carbapenem-resistant phenotypes. However, 2 were different because although they also had ≥ 1 carbapenemase gene, they were, by resistance testing, still sensitive to the carbapenem antibiotics group, such that there was a genotypic-phenotypic correspondence of 92.86%. These results were also similar to those of the study conducted by Gong *et al.*, who found consistency between the detected genotypes of the resistance genes and the phenotypic resistance, with a correspondence of 85%. These results may have been caused by several factors, among other things, heteroresistant microorganisms, lack of expression of the aforementioned carbapenemase gene, mixed populations, and loss of plasmids carrying these resistance genes, but this needs to be replicated for both genotypic and phenotypic confirmation, using different methods if any [25]. This constitutes one of the limitations of this study, because in this study, no replicate testing was conducted, such that the causes of these inconsistencies in results could not be definitely confirmed.

Among the sensitivity patterns that were determined phenotypically on the antibiotics that were excellent against *K. pneumonia*e, were amikacin at 83% and tigecycline at 100%. The sensitivity of *A. baumannii* and other bacteria for tigecycline was also 100%, whereas the sensitivity of *A. baumannii* for amikacin was 73%. On the other hand, in the case of *P. aeruginosa,* the antibiotics for which its sensitivity was excellent were amikacin and piperacillin-tazobactam, for both of which the sensitivity was 80%. The study results of Sitompul *et al.* showed that the sensitivities of *K. pneumoniae*, *A. baumannii*, and *P. aeruginosa* for amikacin were 93%, 67%, and 63%, respectively [26]. The study conducted by Djordjevic *et al.* found that the antibiotic for which *P. aeruginosa* was the most sensitive was piperacillin-tazobactam, with a sensitivity of 72.8% in cases of HAP [27]. Tigecycline has been approved by the FDA (Food and Drug Administration) of the United States for the treatment of infections complicated skin and skin structure infections, complicated intra-abdominal infections, and community pneumonia. A systematic review showed that the use of tigecycline combinations may significantly reduce the 30-day mortality rates in comparison with the use of monotherapy against carbapenem-resistant *Enterobacterales*. The review showed that combinations with meropenem in cases of carbapenemase-producing *K. pneumoniae* may increase the survival rates. Another study that was conducted by Zhang *et al.* recommends combinations of tigecycline

and amikacin for carbapenem-resistant *K. pneumoniae*. The study of Zhou *et al.* also showed that tigecycline constitutes one of the potential choices for treating HAP caused by multidrug-resistant *A. baumannii* [28–30]. The limitation of this study is that it was conducted retrospectively, such that it is unknown whether or not the tested specimens had received antibiotics before the test was conducted or whether or not the patients had taken antibiotics on their own.

Conclusions

The pneumonia panel test may increase the positivity figures when compared with culture (67.35% vs 55.10%). Other advantages of this test are its ability to detect resistance genes, which can be used to adjust the administration of antibiotics, and the shorter time needed to obtain results. However, the limitation of the pneumonia panel test is that there are as yet no fungi and *S. maltophilia* as targets that have sufficient potential to become causative pathogens of pneumonia. In this study, the prevalence of bacteria with resistance genes was 47/99 pathogen-containing specimens or 47.47%. From the phenotypic tests, it was found that the best choice of antibiotics for Gram-negative bacteria in this study was tigecycline and amikacin, except for *P. aeruginosa,* because it has an intrinsic resistance to tigecycline.

Author Contributions

Dharmawan A was involved in the planning and supervision of the work and in the design of the tables. Dharmawan A and Pusparini performed the analysis and drafted the manuscript. Pusparini aided in interpreting the results and worked on the manuscript. Both authors discussed the results and commented on the manuscript.

Corresponding author

Ade Dharmawan, MD,
Clinical Microbiologist
Faculty of Medicine and Health Sciences,
Krida Wacana Christian University
Jakarta, Indonesia
Tel: +6281513266809
Email: ade.dharmawan@ukrida.ac.id

Conflict of interest

No conflict of interest is declared.

References

1. Health Development Policy Agency (2023) Indonesia Health Survey**.** Ministry of Health, Republic of Indonesia. Available: https://www.badankebijakan.kemkes.go.id/en/hasil-ski-2023/. Accessed:1.11.2025. [Article in Indonesian].
2. Fagerli K, Ulziibayar M, Suuri B, Luvsantseren D, Narangerel D, Batsaikhan P, Tsolmon B, Gessner BD, Dunne EM, Grobler AC, Nguyen CD, Mungun T, Mulholland EK, von Mollendorf C (2023) Epidemiology of pneumonia in hospitalized adults ≥ 18 years old in four districts of Ulaanbaatar, Mongolia, 2015–2019. Lancet Regional Health - West Pacific 30: 100591. doi: 10.1016/j.lanwpc.2022.100591.
3. Yoo IY, Huh K, Shim HJ, Yun SA, Chung YN, Kang OK, Huh HJ, Lee NY (2020) Evaluation of the BioFire FilmArray Pneumonia Panel for rapid detection of respiratory bacterial pathogens and antibiotic resistance genes in sputum and endotracheal aspirate specimens. Int J Infect Dis 95: 326-331. doi: 10.1016/j.ijid.2020.03.024.
4. Gastli N, Loubinoux J, Daragon M, Lavigne JP, Saint-Sardos P, Pailhoriès H, Lemarié C, Benmansour H, d'Humières C, Broutin L, Dauwalder O, Levy M, Auger G, Kernéis S, Cattoir V, French FA-PP study group (2021) Multicentric evaluation of bioFire filmarray pneumonia panel for rapid bacteriological documentation of pneumonia. Clin Microbiol Infect 27: 1308-1314. doi: 10.1016/j.cmi.2020.11.014.
5. Dharmawan A, Karuniawati A, Sudarmono PP, Lestari DC, Rumende CM (2020) Epithelial cells count and the ratio of leukocytes and epithelial cells as the criteria to determine qualified specimen for community-acquired pneumonia (CAP)-causing pathogens identification. Indones Biomed J 12: 34-39. doi: 10.18585/INABJ.V12I1.873.
6. Debbagh F, Houmich T Ben, Guennouni M, Sahraoui H, Hanchi AL, Mouaffak Y, Younous S, Soraa N (2023) Evaluation PCR panel of the FilmArray® pneumonia plus for pathogen detection of ventilator-associated pneumonia in children and its impact on therapeutic management. Iran J Microbiol 15: 19-26. doi: 10.18502/ijm.v15i1.11914.
7. Caméléna F, Moy AC, Dudoignon E, Poncin T, Deniau B, Guillemet L, Le Goff J, Budoo M, Benyamina M, Chaussard M, Coutrot M, Lafaurie M, Plaud B, Mebazaa A, Depret F, Berçot B (2021) Performance of a multiplex polymerase chain reaction panel for identifying bacterial pathogens causing pneumonia in critically ill patients with COVID-19. Diagn Microbiol Infect Dis 99: 115183. doi: 10.1016/j.diagmicrobio.2020.115183.
8. Poole S, Clark TW (2020) Rapid syndromic molecular testing in pneumonia: The current landscape and future potential. J Infect 80: 1-7. doi: 10.1016/j.jinf.2019.11.021.
9. Monard C, Pehlivan J, Auger G, Alviset S, Tran Dinh A, Duquaire P, Gastli N, D'Humières C, Maamar A, Boibieux A, Baldeyrou M, Loubinoux J, Dauwalder O, Cattoir V, Armand-Lefèvre L, Kernéis S, ADAPT study group (2020) Multicenter evaluation of a syndromic rapid multiplex PCR test for early adaptation of antimicrobial therapy in adult patients with pneumonia. Crit Care. 24: 1-11. doi: 10.1186/s13054-020-03114-y.
10. Gong J, Yang J, Liu L, Chen X, Yang G, He Y, Sun R (2024) Evaluation and clinical practice of pathogens and antimicrobial resistance genes of biofire filmarray pneumonia panel in lower respiratory tract infections. Infection 52: 545-555. doi: 10.1007/s15010-023-02144-2.
11. Susanto F, Purnamawati CE, Riasari D (2020) 2018-2019 Antimicrobial resistance pattern in bacterial pneumonia at Dr.

Soedarsono Hospital, Pasuruan. Malang Respir J 2: 41-48. doi: 10.21776/ub.mrj.2020.001.01.2.
12. Zacharioudakis IM, Zervou FN, Dubrovskaya Y, Inglima K, See B, Aguero-Rosenfeld M (2021) Evaluation of a multiplex PCR panel for the microbiological diagnosis of pneumonia in hospitalized patients: experience from an academic medical center. Int J Infect Dis 104: 354-360. doi: 10.1016/j.ijid.2021.01.004.
13. Yoo IY, Seok HS, Kwon JA, Lee J, Jo S, Kim SY, Park YJ (2023) Evaluation of the biofire® filmarray® pneumonia panel with conventional bacterial culture in conjunction with leukocyte esterase test. Diagnostics 13. doi: 10.3390/diagnostics13111847.
14. Webber DM, Wallace MA, Burnham CAD, Anderson NW (2020) Evaluation of the biofire filmarray pneumonia panel for detection of viral and bacterial pathogens in lower respiratory tract specimens in the setting of a tertiary care academic medical center. J Clin Microbiol 58. doi: 10.1128/JCM.00343-20.
15. Lee SH, Ruan SY, Pan SC, Lee TF, Chien JY, Hsueh PR (2019) Performance of a multiplex PCR pneumonia panel for the identification of respiratory pathogens and the main determinants of resistance from the lower respiratory tract specimens of adult patients in intensive care units. J Microbiol Immunol Infect 52: 920-928. doi: 10.1016/j.jmii.2019.10.009.
16. Van Der Westhuyzen M, Samodien N, Brink AJ, Moodley C (2023) Utility of the BioFire®FilmArray®Pneumonia Panel plus assay for syndromic testing of lower respiratory tract infections in a low/middle-income setting. JAC-Antimicrobial Resist 5: 1-8. doi: 10.1093/jacamr/dlac139.
17. Singh G, Fauzi NB, Masse SF, Aditianingsih D, Sugiarto A, Loho T, Wulandari D (2022) Microbial pattern and antibiotic resistance of severe pneumonia in a national referral hospital in Indonesia. Preprints: 1-10. doi: 10.20944/preprints202203.0072.v1.
18. Jitmuang A, Puttinad S, Hemvimol S, Pansasiri S, Horthongkham N (2022) A multiplex pneumonia panel for diagnosis of hospital-acquired and ventilator-associated pneumonia in the era of emerging antimicrobial resistance. Front Cell Infect Microbiol 12: 1-13. doi: 10.3389/fcimb.2022.977320.
19. Layanto N, Dharmawan A (2022) Infection and antimicrobial susceptibility pattern of *Stenotrophomonas maltophilia* in the ICU of Hospital X. J Kedokt Meditek 28: 24-29. doi: 10.36452/jkdoktmeditek.v28i1.2310.
20. Guerci P, Bellut H, Mokhtari M, Gaudefroy J, Mongardon N, Charpentier C, Louis G, Tashk P, Dubost C, Ledochowski S, Kimmoun A, Godet T, Pottecher J, Lalot JM, Novy E, Hajage D, Bouglé A, Constantin JM, Perbet S (2019) Outcomes of stenotrophomonas maltophilia hospital-acquired pneumonia in intensive care unit: A nationwide retrospective study. Crit Care 23: 1-13. doi: 10.1186/s13054-019-2649-5.
21. Soeroto AY, Tarmidi IK, Darmawan G, Laurus G, Santoso P (2021) Factors associated with multidrug-resistant pathogens in community-acquired pneumonia patients hospitalized in a provincial teaching hospital in indonesia. Shiraz E Med J 22: 1-6. doi: 10.5812/semj.99779.
22. Carugati M, Aliberti S, Sotgiu G, Blasi F, Gori A, Menendez R, Encheva M, Gallego M, Leuschner P, Ruiz-Buitrago S, Battaglia S, Fantini R, Pascual-Guardia S, Marin-Corral J, Restrepo MI (2020) Bacterial etiology of community-acquired pneumonia in immunocompetent hospitalized patients and appropriateness of empirical treatment recommendations: an international point-prevalence study. Eur J Clin Microbiol Infect Dis 39: 1513-1525. doi: 10.1007/s10096-020-03870-3.
23. Shoar S, Musher D (2020) 1498. Etiology of Community-acquired pneumonia in adults: a systematic review. Open Forum Infect Dis 7: S751-S751. doi: 10.1093/ofid/ofaa439.1679.
24. Farida H, Severin JA, Gasem MH, Keuter M, Van Den Broek P, Hermans PWM, Wahyono H, Verbrugh HA (2013) Nasopharyngeal carriage of *Klebsiella pneumoniae* and other Gram-negative bacilli in pneumonia-prone age groups in Semarang, Indonesia. J Clin Microbiol 51: 1614-1616. doi: 10.1128/JCM.00589-13.
25. Yee R, Bard JD, Simner PJ (2021) The genotype-to-phenotype dilemma: How should laboratories approach discordant susceptibility results? J Clin Microbiol 59: 1-12. doi: 10.1128/JCM.00138-20.
26. Sitompul PA, Indriani R, Rusli A, Sundari T, Rosamarlina R, Hartono TS, Maemun S, Syahril M, Rudiatmoko DR, Setiawaty V (2022) Antimicrobial sensitivity pattern from hospitalized pneumonia patients in national referral infectious disease hospital in Indonesia. Adv Med 1-5. doi: 10.1155/2022/3455948.
27. Djordjevic ZM, Folic MM, Jankovic SM (2017) Distribution and antibiotic susceptibility of pathogens isolated from adults with hospital-acquired and ventilator-associated pneumonia in intensive care unit. J Infect Public Health 10: 740-744. doi: 10.1016/j.jiph.2016.11.016.
28. Ni W, Han Y, Liu J, Wei C, Zhao J, Cui J, Wang R, Liu Y (2016) Tigecycline treatment for carbapenem-resistant enterobacteriaceae infections: A systematic review and meta-analysis. Med 95: 1-10. doi: 10.1097/MD.0000000000003126.
29. Zhang J, Yu L, Fu Y, Zhao Y, Wang Y, Zhao J, Guo Y, Li C, Zhang X (2019) Tigecycline in combination with other antibiotics against clinical isolates of carbapenem-resistant *Klebsiella pneumoniae* in vitro. Ann Palliat Med 8: 522-631. doi: 10.21037/apm.2019.09.11.
30. Zhou Y, Chen X, Xu P, Zhu Y, Wang K, Xiang D, Wang F, Banh HL (2019) Clinical experience with tigecycline in the treatment of hospital-acquired pneumonia caused by multidrug-resistant Acinetobacter baumannii. BMC Pharmacol Toxicol 20: 1-8. doi: 10.1186/s40360-019-0300-3.