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Antibiotic Resistance and Detection of Qnr Genes in isolated Uropathogenic Bacteria from Patients with Urinary Catheters

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Abstract Antibiotic resistance in bacteria is a serious global public health problem. This study aimed to characterize antibiotic resistance genes in patients with urinary catheters. The study involved bacterial strain collection, antibiotic susceptibility testing, and molecular characterization for the detection of genes mediated by fluoroquinolone resistance. A total of 81 strains were collected. Strain distribution showed that Staphylococcus aureus was the most prevalent species (44.44%, n=36). Resistance profiling showed that coagulase-negative Staphylococcus strains (n=12) and Acinetobacter baumannii strains (n=5) expressed 100% resistance to cefoxitin and all tested beta-lactam antibiotics, respectively. Acinetobacter baumannii was resistant to ciprofloxacin (100%, n=5). However, amikacin showed greater activity against Pseudomonas aeruginosa (n=28) and Acinetobacter baumannii (n=5) strains, with 25% and 0% activity, respectively. Molecular gene characterization identified the qnr A, qnr B, and qnr S genes. The strains exhibited a high diversity of resistance genes, with 12.5%, 77.5%, and 60% observed in qnr A, qnr B, and qnr S, respectively. Co-expression of fluoroquinolone resistance genes was demonstrated in 30 strains. This study highlights the high prevalence of antibiotic-resistant bacteria and underscores the crucial role of microbiological and molecular surveillance of bacteria. The results highlight the need for rigorous antibiotic management to prevent the spread of resistant strains.

Keywords: *Antibiotic resistance, uropathogens, urinary catheter, co-expression*

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1. Introduction

Antibiotic resistance is a major threat to global public health today. It is defined as the ability of bacteria to survive and multiply despite the presence of antibiotics, drugs designed to inhibit or eliminate them. This phenomenon, naturally present in bacterial evolution, has been greatly amplified by the excessive and often inappropriate use of antibiotics in human and veterinary medicine, as well as in agriculture [1].

Among antibiotics are fluoroquinolones. They represent an important class of antibiotics used to treat a wide range of bacterial infections, including urinary, respiratory, and gastrointestinal infections. However, resistance to fluoroquinolones expressed by certain bacteria has developed rapidly, compromising their clinical effectiveness. These antibiotics target bacterial enzymes essential for DNA replication, namely DNA gyrase and topoisomerase IV. Point mutations in the genes encoding these enzymes, particularly gyrA and parC, reduce the bactericidal effect of these drugs [2]. These mutations in quinolone-determining regions (QRDRs) are the main resistance markers observed in many bacterial species.

Furthermore, resistance can also result from increased activity of membrane efflux systems, which actively expel fluoroquinolones from the bacterial cell [3]. In addition, alterations in membrane permeability can limit the entry of fluoroquinolones into the cell, contributing to resistance [4]. The clinical impact of these markers is significant. A French study reported a notable increase in fluoroquinolone resistance in Escherichia coli isolated from urinary tract infections between 2015 and 2019, reflecting the spread of QRDR mutations and efflux mechanisms [5]. This trend underscores the importance of monitoring resistance markers to adapt therapeutic strategies and limit the spread of resistant strains.

Fluoroquinolone resistance, mediated by qnr genes, is a growing public health problem in West Africa. A study conducted in Nigeria found that 93.3% of fluoroquinolone-resistant Escherichia coli isolates carried at least one qnr resistance gene, with rates of 33.3% for qnrA and 60% for qnrB, respectively. No qnrS genes were detected in this study [6].

In Gabon, a study demonstrated high resistance to quinolones in enteric pathogens isolated from the stool of young children, with resistance rates of 100% to ofloxacin and 97.3% to ciprofloxacin. The qnrB and qnrS genes were detected in 13.5% and 13.5% of the strains, respectively, while the qnrA gene was not identified [7]. In Côte d'Ivoire, several studies have highlighted the presence of these resistance genes. Research conducted in Abidjan detected the qnrD (36.7%), qnrB (36.7%), qnrS (13.3%), and qnrA (6.7%) genes in Gram-negative bacilli. Notably, this study was the first to identify the qnrD gene in certain Ivorian bacterial strains [8].

Thus, antibiotic resistance leads to a significant increase in morbidity and mortality rates, lengthens hospital stays, and raises healthcare costs. Furthermore, it limits available treatment options, rendering some treatments ineffective, which poses a real challenge to healthcare systems worldwide [4].

Faced with this crisis, comprehensive strategies are essential. These include more rational use of antibiotics, the development of new drugs, and the strengthening of hygiene and infection control measures. The objective of this study is to characterize the qnr A, B, and S genes in uropathogenic bacteria isolated from urinary catheter users.

2. Materials and Methods

2.1. Collection of Uropathogenic Strains

The bacteria were collected from the Biocollection of the National Reference Center for Antibiotics (CNR) at the Pasteur Institute of Côte d'Ivoire (IPCI). A total of 81 bacterial strains, consisting of Staphylococcus aureus (36), Pseudomonas aeruginosa (28), coagulase-negative Staphylococcus (12), and Acinetobacter baumannii (5), were isolated from the urine of patients with urinary catheters during 2022. These isolates were stored at -80°C in a 1% glycerol-enriched brain broth at the IPCI's Biological Resource Center (CeReB). The bacterial strain identifiers were determined based on information related to the hospital department (nephrology, outpatient, neurology, intensive care, pediatrics, pulmonology, emergency), the patient's age, and sex.

2.2. Revivification of Bacterial Strains

Using a Pasteur pipette, the strains were collected and cultured in nutrient broth, Heart and Brain Broth (BCC), and then incubated at 37°C for 24 h. Next, they were successively inoculated onto the selective medium (Chapman, Cetrimide, and Eosin Methylene Blue (EMB) agar) contained in Petri dishes. The dishes were then incubated at 37°C for 24 h to obtain young bacterial colonies, which were then subcultured onto ordinary agar for antibiotic susceptibility testing.

2.3. Determination of the Phenotype of Collected Strains to Antibiotics

The method of scattering antibiotic-impregnated discs onto Müeller-Hinton agar was used to evaluate the susceptibility of the strains according to the recommendations of the Antibiogram Committee of the French Society for Microbiology [9]. The antibiotic-impregnated discs used are listed in Table 1.

Table 1. List of tested antibiotic-impregnated discs

Families of antibiotics	Antibiotics tested	Abbreviations	Disk charges (µg)
Beta-lactams	Ticarcillin	TIC	75
	Cefepime	FEP	30
	Ceftazidime	CAZ	10
	Cefoxitin	FOX	30
	Imipenem	IMP	10
	Meropeneme	MEM	10
	Ticarcillin/Clavulanic Acid	TCC	75/10
	Aztreonam	ATM	30
Fluoroquinolones	Ciprofloxacin	CIP	5
	Norfloxacin	NOR	5
	Moxifloxacin	MOX	5
Aminosides	Gentamicin	GEN	10
	Amikacin	AKN	30
Macrolides	Erythromycin	ERY	15
Lincosamides	Clindamycin	CMN	2

2.4. Detection of Qnr Genes by PCR

DNA extraction from the collected strains and the reference strains Escherichia coli ATCC 29522 and Klebsiella pneumoniae ATCC 70603 was performed using alkaline lysis with phenolization. Conventional polymerase chain reaction (PCR) was used to detect fluoroquinolone resistance genes (qnr A, B, and S). Specific primer pairs listed in Table 2 were used to amplify the genes. PCR amplification was performed in a 25 µL volume using a thermocycler (Perkin® Elmer Gen Amp Lapplied Biosystems 9700). The amplification conditions consisted of an initial DNA denaturation step for 5 min at 95°C. This step was followed by 35 amplification cycles, including denaturation at 95°C for 1 min, hybridization at 56°C for 1 min, elongation at 72°C for 1 min 30 s, and a final elongation step at 72°C for 10 min. The reaction medium consisted of 12.5 µL of Master Mix, 1 µL of forward and antisense primers, and 6.5 µL of ultrapure water. To each well of the PCR plate, pre-filled with the mixture, 5 µL of bacterial DNA was added. Another reaction mixture without DNA was used as the negative control. The amplified products were analyzed by electrophoresis on 1.5% agarose gel (Invitrogen) stained with ethidium bromide. The reading was taken on an ultraviolet (UV) plate (Gel Doc).

Table 2. List of primers used for the detection of fluoroquinolone resistance genes

Gene name	Primer	5'-3' primer sequences	Amplicons (pb)	References
qnr A	F R	GATAAAGTTTTTCAGCAAGAGG ATCCAGATCGGCAAAGGTT	542	Touati *et al*., 2008
qnr B	F R	GACAGAAACAGGTTCACCG GT CAAGACGTTCCAGGAGCAA CG	594	Touati *et al*., 2008
qnr S	F R	GACGTGCTAACTTGCGTGAT AACACCTCGACTTAAGTCTGA	388	Seyed *et al*., 2014

3. Results

3.1. Distribution of Collected Strains According to Hospital Departments

Analysis of the results showed that the nephrology department had the highest isolation rate at 60.49%, followed by the outpatient department (13.58%). The lowest bacterial rate (1.23%) was recorded in the surgery and intensive care departments (χ^2 = 51.87; p = 0.000). The complete dataset is shown in Figure 1.

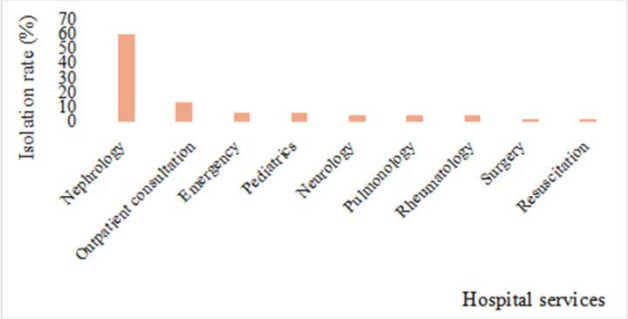


Figure 1. Distribution of collected strains according to hospital departments

3.2. Distribution of Strains According to Sex and Age of Patients

Of the 81 bacterial strains, 69 were isolated from males (85%) and 12 from females (15%), for a sex ratio of 5.75. Analysis revealed a predominance of strains isolated from males compared to females (χ^2 = 24.60; p = 0.000). Regarding age, the analysis showed a significant difference in isolation rates across age groups (χ^2 = 14.29; p = 0.003). The most represented age group was that of elderly patients aged 51 to 73 years (46%), followed by the 36-50 year age group (25%). The lowest rate of isolation (9%) was represented by individuals aged 0 to 18 years (Figure 2).

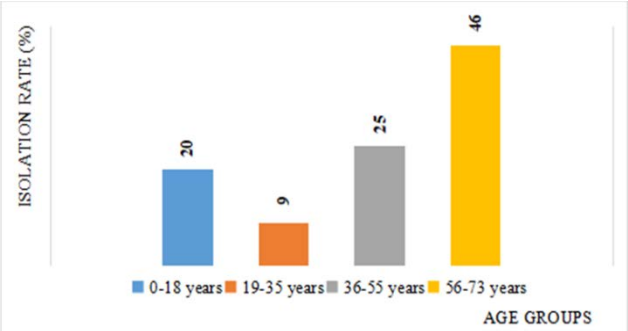
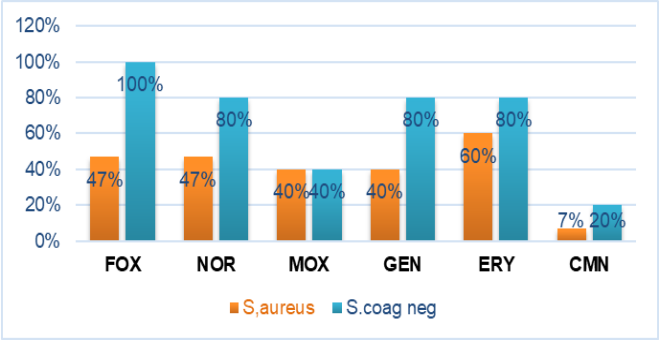


Figure 2. Distribution of collected strains according to age class

3.3. Antibiotic Resistance Profile of Collected Strains

Resistance of Staphylococcus aureus and coagulase-negative Staphylococcus to cefoxitin, erythromycin, and clindamycin

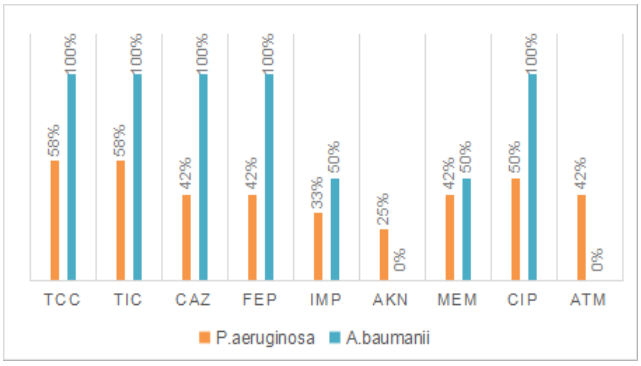
Cefoxitin was the only beta-lactam molecule tested in these species. Of the 36 Staphylococcus aureus strains tested, 17 were resistant, representing a resistance rate of 47.22%. In contrast, all 12 coagulase-negative Staphylococcus strains tested were completely resistant (100%). In this study, the results showed that in the presence of gentamicin and erythromycin, both Staphylococcus aureus and coagulase-negative Staphylococcus exhibited a low resistance rate (40%). Furthermore, coagulase-negative Staphylococcus showed resistance rates of 80% and 20% to erythromycin and clindamycin, respectively (Figure 3).



FOX: cefoxitin; NOR: norfloxacin; MOX: moxifloxacin; GEN: gentamicin ERY: erythromicin CMN: clindamicin

Figure 3. Resistance rates of staphylococci to antibiotics

Resistance of Pseudomonas aeruginosa and Acinetobacter baumannii to beta-lactams



TCC: ticarcillin (clavulanic acid); TIC: ticarcillin; CAZ: ceftazidime; FEP: cefepin; IMP: imipenem; AKN: amikacin; MEN: meropenem; CIP: ciprofloxacin; ATM: aztreonam

Figure 4. Resistance rate of non-fermenting Gram-negative bacilli to antibiotics

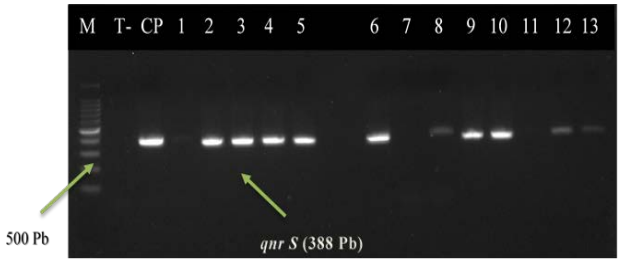
A total of 33 strains, including 5 *Acinetobacter baumannii* and 28 *Pseudomonas aeruginosa*, were tested against beta-lactams. Each of these species showed different resistance phenotypes in the presence of the antibiotics used. *Acinetobacter baumannii* exhibited high resistance rates (100%) to almost all the beta-lactams tested, except for imipenem, to which the bacteria were partially resistant (50%). As for Pseudomonas aeruginosa, the results showed a resistance rate of 58% against ticarcillin + clavulanic acid and ticarcillin alone, compared to 42% for cefepime, ceftazidime, imipenem, and meropenem (Figure 4).

Germ resistance to fluoroquinolones

Resistance rates of 47% and 60% for norfloxacin and moxifloxacin were observed in the presence of Staphylococcus aureus. In contrast, coagulase-negative staphylococci showed a high resistance rate of 80% to these antibiotics. Regarding Acinetobacter baumannii and Pseudomonas aeruginosa strains, resistance rates of 100% and 50% were observed against ciprofloxacin (Figure 4).

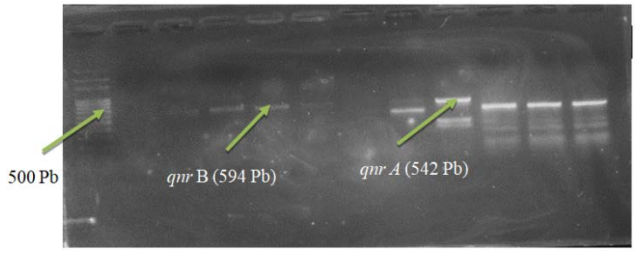
3.4. Genotypes of the Studied Strains

Fluoroquinolone Resistance Genes Detected in the Tested Strains



Lane M: molecular weight marker (Solis Biodyne 100 Pb), Lane CP: positive control, Lane T-: negative control, Lanes 7 and 11: negative samples, Lanes 1, 2, 3, 4, 5, 6, 8, 9, 10, 12, 13: positive samples.

Figure 5. Electrophoretic profiles on 1.5% agarose gel of the qnr S gene detected in the tested strains



Lane M: molecular weight marker (Solis Biodyne 100 Pb), Lane CP: positive control, Lane T-: negative control, Lane 5: negative samples, Lanes 1, 2, 3, 4, 6, 7: positive samples for qnr B, Lanes 6, 8, 9, 10: positive samples for qnr A.

Figure 6. Electrophoretic profiles on 1.5% agarose gel showing multiplex PCR for the detection of the qnr A and qnr B genes

A total of 40 strains, consisting of *Staphylococcus aureus* (n=17), *Pseudomonas aeruginosa* (n=13), coagulase-negative Staphylococcus (n=8), and Acinetobacter baumannii (n=2), phenotypically resistant to ciprofloxacin, norfloxacin, and moxifloxacin, were selected for molecular analysis. Analysis of the results revealed that resistance genes were present at varying levels. Specifically, the qnrB and qnrS genes were predominant, with levels of 80% and 62.5%, respectively, in 32 and 25 strains. The qnrA gene was present in 5 strains at a level of 12.5%. Figure 5 and Figure 6 present the electrophoretic profiles of the genes analyzed.

Simultaneous Expression of Fluoroquinolone Resistance Genes

Agarose gel electrophoresis revealed the simultaneous expression (co-expression) of fluoroquinolone resistance genes. Indeed, the *Acinetobacter baumannii* strains expressed the different types of co-expression: qnr A-B, qnr B-S, and qnr A-B-S, with levels of 50% (n=1), 100% (n=2), and 50% (n=1), respectively. However, *Staphylococcus aureus*, coagulase-negative *Staphylococcus*, and *Pseudomonas aeruginosa* exhibited only one type of co-expression, qnr B-S. Table 3 presents all the results.

Table 3. Distribution of fluoroquinolone resistance genes detected in the tested strains

Souches (N)	Genes N (%)			Co-expression N (%)		
	qnr A	*qnr B*	*qnr S*	*qnr A-B*	*qnr B-S*	*qnr A-B-S*
S. aureus (17)	3(17,64)	16 (94,11)	8 (47,06)	0	7 (41,17)	0
S. coag neg (08)	0	4 (50)	4 (50)	0	4 (50)	0
P. aeruginosa (13)	0	10 (76,92)	11 (84,61)	0	7 (53,84)	0
A. baumanii (02)	2 (100)	2 (100)	2 (100)	1 (50)	2 (100)	1 (50)
Total (40)	5	32	25	1	20	1

S. aureus: Staphylococcus aureus ; S. coag neg: coagulase negative Staphylococcus; P. aeroginosa: Pseudomonas aeroginosa ; A. baumanii: Acinetobacter baumanii

4. Discussion

A total of 81 bacterial strains, including Staphylococcus aureus, Pseudomonas aeruginosa, coagulase-negative staphylococci, and Acinetobacter baumannii, were isolated from the urine of patients with urinary catheters. Of these strains, Staphylococcus aureus was predominant, with an isolation rate of 44.44%. This bacterial profile is consistent with several studies conducted in hospital settings, which report a strong involvement of Staphylococcus aureus and Pseudomonas aeruginosa in catheter-associated urinary tract infections, as well as the notable presence of coagulase-negative staphylococci, often underestimated but relevant in this type of infection [10].

The isolation of these strains highlights the bacterial diversity involved in urinary device-associated infections. The predominance of Staphylococcus aureus in this study is a significant finding. This bacterium is well known for its ability to colonize medical devices and form biofilms, which promotes infection persistence and resistance to antimicrobial treatments [11].

The presence of Pseudomonas aeruginosa, coagulase-negative staphylococci, and Acinetobacter baumannii also underscores the complexity of catheter-related urinary tract infections, as these microorganisms are frequently implicated in nosocomial infections. Pseudomonas aeruginosa is particularly recognized for its multiple

mechanisms of antibiotic resistance and its ability to survive in hospital environments, according to Poole [12]. Similarly, Acinetobacter baumannii is an emerging opportunistic pathogen, often associated with multidrug-resistant infections, particularly in immunocompromised patients or those with medical devices [13]. However, one study highlighted that enterobacteria, particularly Escherichia coli and Klebsiella pneumoniae, were the major etiological agents of urinary tract infections in these patients [14].

The analysis revealed a predominance of infection in men (85%) compared to 15% in women. This prevalence corroborates that of Sanou et al. [14], who, in their study of 47 patients with catheters, found that 44 were male, representing 93.6% of the sample. This predominance could be explained by several factors such as age, comorbidities, and care practices. For example, in men over 50, the incidence of urinary tract infections increases due to prostatic hypertrophy and the use of catheters, which may explain a higher prevalence in this population [15].

The results indicated that the coagulase-negative Staphylococcus (CNS) strains tested were completely resistant (100%) to cefoxitin. This rate is higher than that reported by Martins et al. [16]. These authors reported that 47% of CNS isolates were resistant to cefoxitin, with a prevalence of 47% methicillin resistance. The total resistance of these strains observed in this study is a strong indicator of the likely presence of resistance mechanisms mediated by the mecA gene, characteristic of methicillin-resistant Staphylococcus strains [17]. Furthermore, according to Becker et al. [18], high resistance to beta-lactams complicates clinical management and antibiotic therapy.

Acinetobacter baumannii isolates exhibited high resistance rates (100%) to almost all of the beta-lactams tested. This result differs from that obtained in a study conducted at the Mohammed V Military Teaching Hospital in Rabat, Morocco. It reported a high resistance to ticarcillin of 72.3% [19].

Analysis of the results obtained showed that the strains of Acinetobacter baumannii and Pseudomonas aeruginosa expressed resistance rates of 100% and 50%, respectively, to ciprofloxacin. Several scientific studies have observed high resistance rates of these bacteria to ciprofloxacin, although the percentages vary according to geographical contexts and the periods studied. Indeed, a retrospective study conducted over seven years in a burn intensive care unit reported that the resistance of Pseudomonas aeruginosa to ciprofloxacin was 42.9% in 2018, while that of Acinetobacter baumannii was 90.5% during the same period [20]. In Tunisia, Mellouli et al. [21] found that Acinetobacter baumannii resistance to ciprofloxacin was 93.5%, with colistin resistance of 1.8%. These variations can be attributed to factors such as local antibiotic use practices, infection control protocols, and differences in data collection and analysis methods.

Regarding the detection of qnr genes in this study, analysis of the results shows that resistance genes were present at varying rates. Specifically, qnr genes B and S were predominant, with rates of 80% and 62.5%, respectively, in 32 and 25 strains. qnr A was present in 5 strains at a rate of 12.5%. The variable presence of qnr resistance genes in the studied strains reflects the diversity of genetic mechanisms involved in quinolone resistance, particularly to ciprofloxacin. The qnr genes encode proteins that protect DNA gyrase and topoisomerase IV, the main targets of quinolones, thus reducing the efficacy of these antibiotics [22].

In this study, the predominance of the qnrB (80%) and qnrS (62.5%) genes is consistent with several reports that have also identified these genes as the most frequent in clinical strains of Pseudomonas aeruginosa and Acinetobacter baumannii. For example, a systematic review published by Cattoir and Nordmann [23] highlights that the qnrB and qnrS genes are often associated with high quinolone resistance in Enterobacteriaceae and non-fermenting bacteria. The lower presence of the qnrA gene (12.5%) confirms that this variant is less frequent in these species, although it is also an important factor in plasmid-mediated quinolone resistance [24]. This variable distribution of qnr genes underscores the importance of molecular surveillance for better understanding region-specific resistance profiles and adapting therapeutic strategies.

The results showed that Acinetobacter baumannii strains expressed different types of co-expression: qnr A-B, qnr B-S, and qnr A-B-S, with levels of 50%, 100%, and 50%, respectively. The results indicating co-expression of qnr A-B, qnr B-S, and qnr A-B-S genes in Acinetobacter baumannii strains demonstrate the complexity and multiplicity of the genetic mechanisms responsible for quinolone resistance. The co-presence of several qnr genes in the same strain can amplify protection against quinolones by providing more robust resistance that is potentially more difficult to overcome by antibiotic treatments [22]. The high co-expression rate of the qnr B-S type (100%) suggests that this combination could be particularly advantageous for bacterial survival under antibiotic pressure, which is consistent with previous observations where the qnrB and qnrS genes are frequently detected together in mobile plasmids, thus facilitating their dissemination between bacteria [23].

Although less frequent, qnr A-B (50%) and qnr A-B-S (50%) co-expressions indicate significant genetic diversity that could contribute to varied resistance profiles. Such diversity can complicate the clinical management of infections, as it can lead to different levels of resistance and response to treatments [24].

5. Conclusion

The presence of qnr genes detected in the pathogens in this study suggests the susceptibility of these genes to dissemination via horizontal gene transfer mechanisms. The Acinetobacter baumannii strains expressed different types of co-expression. The resistance profile showed that coagulase-negative Staphylococcus and Acinetobacter baumannii strains expressed 100% resistance in the presence of cefoxitin, respectively. However, amikacin showed greater activity against Pseudomonas aeruginosa (n=28) and Acinetobacter baumannii strains. The information on antibiotic resistance obtained in this study highlights the need for rigorous antibiotic management to prevent the spread of resistant strains.

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Authors Contribution

Eric Joel Tahou and Kouadio Fernique Konan carried out sample collection and processing, data collection, laboratory analyses, and interpretation. Bertin Tiecoura performed data analysis, statistical analyses, results visualization, and literature reviews. Assanvo Simon-Pierre N'guetta and Kouadio Nathalie Guessennd provided critical feedback on the manuscript and supervised this study. All authors contributed to the drafting, review, and approval of the final version of the manuscript.

Competing Interests

The authors have no competing interests.

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