Active surveillance of adult healthcare-associated infections in intensive care units: resistance and molecular profile in an upper middle-income country

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Abstract

Objective: This study aimed to characterize epidemiological and molecular profile of Healthcare-associated infections [HAI] in 21 intensive care units (ICU) in a city in Colombia.

Methods: Descriptive study of prevalence. Adult patients were screened in 21 ICUs for HAIs: VAP, CLABSI, CAUTI and/or SSI. Microbiological and genotypic identification was performed.

Results: Prevalence of HAIs was 41.4% (CI 36.9-45.9). VAP 15.8% (CI 12.7-19.4); CLABSI, 13.5% (CI 10.6-16.9); CAUTI, 7.7% (CI 5.5-10.5); and SSI, 4.4% (CI 2.7-6.6). Gram-negative bacteria (71.7%) predominated (P. aeruginosa (19.1%), K. pneumoniae (13.4%) and E. coli (13%)). Pseudomonas spp. 20-30% were resistant to carbapenems and ≥ 10% to aztreonam, 3rd- and 4th-generation cephalosporins, and β-lactamase inhibitors. In VAP and CLABSI, 30% of Staphylococcus aureus were resistant to oxacillin. In CAUTI, Staphylococcus epidermidis exhibited 100% resistance. In P aeruginosa resistance gene were blaTEM, blaSHV, and blaCTX-M (15-32%), KPC (5.7%), and oxacillinase blaoXA-48 (1.8%) and blaoXA-1-40-30 (20-50%). In E. coli, genes qnrB, qnrS and qnrD were identified. In CLABSI, ermc-type (16.7%), aph(2)’If (7.7%) and ant(4)’-Ia (7.7%) were identified in Staphylococcus aureus.

Conclusions: VAP and CLABSI predominate in ICUs evaluated in Colombia due to resistant gram-negative bacteria by ESBL-type resistance genes plasmids, efflux pumps hindering the therapeutic approach.

Keywords: Molecular Epidemiology; Infection Control; genotyping, Colombia.
Introduction

The Surveillance of Healthcare-Associated Infections [HAI] in Infection Control guarantees safety in patient care. HAIs are defined as pathologies resulting from healthcare that are absent in hospital admission.\(^1\)\(^2\)

Epidemiology is complex, as there is heterogeneity in the quality of information and the knowledge gap in surveillance policy in low- and middle-income countries. Patients hospitalized in Intensive Care Units (ICUs) in developed countries are affected by at least one episode of HAI, with high incidence ranging from 5.7 to 19.1% with frequencies of 29% in Surgical Site Infections [SSIs] and 24% Urinary Tract Infections [UTIs] and 19% Bloodstream Infections [BSIs], 14.8% hospital pneumonia and the remaining 13.1% other infections.\(^3\)

The etiology is sometimes complex to establish; HAIs are reported predominantly in ICUs related to gram-negative bacteria, such as *Pseudomonas aeruginosa* and *Escherichia coli*, and in gram-positive, coagulase-negative *Staphylococcus* coli, and in *Staphylococcus aureus*. Regarding antimicrobial resistance, *Staphylococcus aureus* resistant to oxacillin (MRSA), resistance to 3rd generation cephalosporins in *E. coli* (16%), *Klebsiella spp.* (40%) and *Enterobacter spp.* (34%); and resistance to carbapenems such as *Klebsiella spp.* (15%), *P. aeruginosa* (26%) and *A. baumannii* (64%) predominate.\(^4\)\(^5\) Resistance genes have been molecularly characterized, such as those related to Extended-Spectrum ß-lactamases [ESBLs]. Aminoglycoside Modifying Enzymes [AMEs], Plasmid-Mediated Quinolone Resistance [PMQR] or mutations in the *gyrA* gene.\(^6\)\(^7\)

The following study aimed to characterize the molecular profile of HAIs in 21 adult intensive care units in Cartagena de Indias, Colombia.

Material and methods

**Study design**

Research supported by the health authority of Cartagena in the case of a descriptive study of prevalence. Twenty-one adult care ICUs were monitored for: Ventilator-Associated Pneumonia [VAP], Central Line-Associated Bloodstream Infection [CLABSI]; Catheter-Associated Urinary Tract Infection [CAUTI] and/or Surgical Site Infection [SSI]. Over 10 months, 481 adult patients with a diagnosis of HAI were included according to the criteria by the Centers for Disease Control and Prevention of the United States [CDC] and the National Institute of Health of Colombia [INS]. A biological sample was available for analysis. All patients and/or their families signed an informed consent to participate.

**Phenotypic characterization and antimicrobial susceptibility profile**

For phenotypic identification and susceptibility tests [sensitivity and minimum inhibitory concentrations-MIC-] the automated standardized method of the MicroScan4 analyzer [Beckman Coulter\(^*\)] following CLSI [Clinical and Laboratory Standards Institute] principles were performed.\(^8\)

**Genotypic characterization**

The DNA was obtained using protocols standardized by the UNIMOL (Unidad Investigacion Molecular) laboratory and using the commercial kit for genomic DNA, Wizard\(^*\) [Promega\(^*\)] according to the manufacturer’s recommendations. For molecular characterization, the endpoint Polymerase Chain Reaction [PCR] technique was used (see Table 1). According to the susceptibility profile, resistance genes against pharmacological groups of clinical interest were chosen. (Table 1) and sequenced using the Sanger technique and analyzed using BLAST online software.\(^9\)

**Statistical analysis**

The collected variables were analyzed according to their nature using descriptive statistics. SPSS IBM\(^*\) was used for all analyses with a two-tailed significance level of 0.05.

Results

**Prevalence of HAIs in Cartagena, Colombia**

Over 10 months, 481 patients were screened, 282 were excluded (did not meet the inclusion criteria), the reasons for exclusion were not meeting the CDC criteria to define the IAAS cases in other cases information was incomplete; finally 199 patients were monitored, of which 90.5% (180) had a single HAI. A homogeneous distribution was observed in relation to sex (50.3% men). On average, the patients were 59.6 years old (SD 19.5 years old). The time elapsed between admission to the ICU and infection (HAI) in 50% of the population was 4 days (IQR 2-9), for CLABSI 50% of the patients had this infection at 5 days (IQR 2-13). Satisfactory recovery was observed in 78.3% of patients, and 12.6% died, with HAI being the final cause of death in 92% of cases. The event with the highest proportion of deaths was VAP (17.1%) (Table 2).

During the follow-up time, the prevalence of HAI was 41.4% (CI 36.9-45.9), VAP was the most prevalent at 15.8% (CI 12.7-19.4) followed by CLABSI 13.5% (CI 10.6-16.9), CAUTI 7.7% (CI 5.5-10.5) and SSI with 4.4% (CI 2.7 - 6.6) (Table 2).

**Phenotypic characterization of microorganisms associated with HAI**

Regarding the etiology, 14.7% of the cases of HAI presented polymicrobial etiology. In 9.4% of the samples, no germ was isolated. The distribution of microorganisms had a large predominance of gram-negative bacteria (71.7%), among which *Pseudomonas aeruginosa* (19.1%), *Klebsiella pneumoniae* (13.4%) and *Escherichia coli* (13%) were the most identified. Within the group of gram-positive (13.1%), *Staphylococcus aureus* (4.3%), *Staphylococcus epidermidis* (1.7%), and *Staphylococcus hominis* (1.7%) were the most identified. Regarding fungi (5.5%), *Candida famata* (2%) and *Candida albicans* (1.3%) were the most identified (Table 3). *Pseudomonas aeruginosa* was more frequent in VAP (24.3%), CLABSI (18%) and SSI (15.4%), while in CAUTI, *Escherichia coli* was found at 18.2% frequency.
Table 1. Resistance genes and primers used for the genotyping of microorganisms isolated in patients with HAI in Cartagena, Colombia.

| GEN | SEQUENCES (5’ – 3’) | Primer size (bp) | Annealing Temp. (°C) | Resistance/Reference Phenotype |
|-----|---------------------|------------------|----------------------|------------------------------|
| ermA, ermC | *(a)* | ermA F: AAG CGG TAA ACC CCT CTG A  
ermA R: TTCT GCA AAT CCC TTC TCA AC  
ermC F: AAT CTT CAA TTC CTG CAT GT  
ermC R: TCTA TCG TGG AAT ACG GGT TG | ermA 190bp; Ta: 55  
ermC 299bp; Ta: 55 | Erythromycin - Clindamycin  
ermA 24  
ermC 24 |
| mecA | *(b)* | mecA F: AAA ATC GAT GGT AAA GGT TGG C  
mecA R: AGT TCT GCA GTA CCG GAT TG | mecA 532bp; Ta: 55 | ß-lactams  
mecA 24 |
| qnrA, qnrB, qnrS, qnrC, qnrD, aac(6’)-Ib-cr, qepA, oqxA, oqxB | *(a and c)* | qnrA F: AGAGGATTTCTCACGCCAGG  
qnrA R: TGCCAGGCACAGATCTTGAC | qnrA 580bp; Ta: 54ºC  
qnrB 264bp; Ta: 54ºC  
qnrC 307bp; Ta: 55ºC  
qnrD 581bp; Ta: 57ºC  
aac(6’)-Ib-cr 519bp  
qepA 199bp; Ta: 63ºC  
oqxA 392bp Ta: 68ºC  
oqxB 512bp Ta: 70ºC | Quinolones  
qnrA 35  
qnrB 35  
qnrC 35  
qnrD 35  
aac(6’)-Ib-cr 35  
qepA 35  
oqxA 35  
oqxB 35 |
| aac[3]-Ia, aac[6’]-Ib, aph[2’]If, aac[6’]-Ie/aph[2’]Ia, ant[4’]-Ia | *(a and c)* | aac[3]-Ia F: ATGGGCATCATTCGCCAGG  
aac[3]-Ia R: TGGCGCCAGCAGATCTTGAC | aac[3]-Ia 484bp; Ta: 60ºC  
aac[6’]-Ib 524bp; Ta: 58ºC  
aph[2’]If 420bp; Ta: 50ºC  
ant[4’]-Ia 1106bp  
ant[4’]-Ia 134bp; Ta: 58ºC | Aminoglycosides  
aac[3]-Ia 36  
aac[6’]-Ib 36  
aph[2’]If 36  
aac[6’]-Ie/aph[2’]Ia 37  
ant[4’]-Ia 38 |
| TEM, SHV, KPC, CTX-M, MTSO, OXA 48, OXA 23 | *(c)* | TEM F: GCC GAA CCC CTA TTT G  
TEM R: ACC AAT GTA TAA TCA CTG AG | TEM 1017bp; Ta: 55 | ß-lactams  
TEM 25  
SHV 26  
KPC 27  
CTX-M 28  
MTSO 29  
OXA 48 36  
OXA 23 36 |
| TEM, SHV, KPC, CTX-M, MTSO, OXA 48, OXA 23 | *(c)* | SHV F: TTA TCT CCC TGT TAG CCA C  
SHV R: GAT TGG CTG ATG TCT CGC GG | SHV 795bp; Ta: 60  
KPC 798bp; Ta: 55  
MultiTSO 564bp; Ta: 60  
OXAs 48 281bp; Ta: 60  
OXAs 23 64bp; Ta: 52 | ß-lactams  
TEM 1017bp; Ta: 55  
SHV 795bp; Ta: 60  
KPC 798bp; Ta: 55  
MultiTSO 564bp; Ta: 60  
OXAs 48 281bp; Ta: 60  
OXAs 23 64bp; Ta: 52 |

*a* Used in Gram (+) resistant microorganisms. *b* Resistant S. aureus. *c* Gram (-) resistant microorganisms.
**Resistance profiles associated with HAI**

The antibiotic groups of interest evaluated in gram-negative bacteria (Table 4), for *Pseudomonas aeruginosa* (the most prevalent) revealed elevated resistance values of 20-30% against carbapenems; ≥10% against aztreonam, 3rd and 4th-generation cephalosporins, and β-lactamase inhibitors. *P. aeruginosa* exhibited its greatest resistance in VAP, presenting some degree of resistance against all the antibiotics tested, with values of 30-45% against carbapenems and colistin. *Klebsiella pneumoniae* presented resistance of 40% against ampicillin/sulbactam; in VAP this resistance reached 50%, followed by 46.2% against meropenem and up to 37% against other β-lactams. In CLABSI and CAUTI, *K. pneumoniae* maintained resistance percentages of 15-50% against the majority of β-lactams tested. *Escherichia coli* exhibited its greatest resistance against ampicillin/sulbactam, with 41% general resistance and >40% resistance in VAP and CLABSI. The resistance against quinolones in *E. coli* were higher in CLABSI (47.6%). In *Staphylococcus aureus* its resistance against carbapenems; ≥10% against aztreonam, 3rd and 4th-generation cephalosporins, and β-lactamase inhibitors. *P. aeruginosa, K. pneumoniae, E. coli* were identified in values that ranged between 15-32% as well as KPC carbapenemases, which were found at a 5.7% frequency (Tables 6 and 7).

Oxacillinase type OXA-48 was recognized exclusively in *P. aeruginosa* (1.8%). In contrast, OXA-23 was identified in both *P. aeruginosa* (8.8%) and *E. coli* (2.6%), OXA-1, OXA-4 and OXA-30 were found with values of 20-50% in the gram-negatives evaluated.

PMQR resistance (quinolones) related to *qnrB, qnrS and qnrD* genes was detected in greater proportions in resistant *E. coli* strains. The *aac(6’)-Ib-cr* gene of the PMQR type was found exclusively in *P. aeruginosa* (8.8%). Genes coding for *qepA* and *qepB* efflux pumps were identified in *P. aeruginosa, K pneumoniae* and *E. coli* (Table 6).

**Genotyping of microorganisms isolated in patients with HAI in Cartagena, Colombia**

In gram-negative strains, resistance genes associated with extended spectrum β-lactamases (ESBL) were identified in the most prevalent strains (*P. aeruginosa, K. pneumoniae, E. coli*). In *P. aeruginosa*, genes associated with ESBL such as TEM, SHV, CTX-M were identified in values that ranged between 15-32% as well as KPC carbapenemases, which were found at a 5.7% frequency (Tables 6 and 7).

**Table 2.** Sociodemographic characteristics of the population monitored for HAI in Cartagena, Colombia.

| Characteristic | Patients | VAPb | CLABSIb | CAUTIb | SSIc |
|---------------|----------|------|---------|--------|------|
| Sample distribution: N (%) | 199 | 189 | 176 | 173 | 157 |
| Number of monitored patients | 218 | 200 | 196 | 193 | 177 |
| Number of monitored events | 100 (50.3%) | 95 (46.6%) | 98 (49.7%) | 97 (49.7%) | 89 (56.8%) |
| Male | 100 (50.3%) | 95 (46.6%) | 98 (49.7%) | 97 (49.7%) | 89 (56.8%) |
| Female | 99 (49.7%) | 104 (53.4%) | 98 (49.7%) | 96 (49.6%) | 88 (53.2%) |
| AGE: (mean in years and SD) | 59.6 (19.5) | 58.9 (20.7) | 59.7 (18.7) | 58.9 (21.1) | 62.6 (14.4) |
| Patient in isolation: N (%) | 8 (4.2%) | 6 (3.2%) | 0 | 2 (5.4%) | 0 |
| YES | 189 (95%) | 180 (90%) | 0 | 34 (91.9%) | 20 (93.2%) |
| NO | 2 (0.8) | 2 (1%) | 60 (100%) | 1 (4.8%) | 1 (4.8%) |
| Type of ICU N (%) | 162 (81.4%) | 156 (78.3%) | 60 (100%) | 24 (66.7%) | 16 (76.2%) |
| Intensive | 37 (18.6%) | 22 (11.7%) | 0 | 6 (27.3%) | 5 (23.8%) |
| Intermediate | 70 (92.1%) | 64 (90.7%) | 60 (100%) | 11 (44.2%) | 6 (25.0%) |
| Time spent in ICU until diagnosis of HAI (days) - median [IQR] | 4 (2-9) | 4 (2-7) | 5 (2-13) | 4 (2-8) | 3 (0-12) |
| Outcome: N (%) | 25 (12.6%) | 23 (11.9%) | 5 (10.2%) | 5 (10.2%) | 1 (4.8%) |
| Death | 156 (78.3%) | 145 (72.8%) | 52 (11.7%) | 30 (66.7%) | 18 (81.8%) |
| Recovery | 18 (9%) | 18 (9%) | 7 (15.6%) | 2 (4.4%) | 2 (9.5%) |
| NS | 12 (93.3%) | 11 (91.7%) | 5 (83.3%) | 5 (100%) | 0 |
| NS | 1 (7.7%) | 1 (8.3%) | 1 (16.7%) | 0 | 1 (4.8%) |
| NS | 5 (83.3%) | 5 (41.6%) | 5 (83.3%) | 0 | 1 (4.8%) |
| Prevalence % (95% CI) | 41.4 (36.4-46.5) | 15.8 (12.7-19.4) | 13.5 (10.6-16.9) | 7.7 (5.5-10.5) | 4.4 (2.7-6.6) |
| Number of Events per hospitalization: N (%) | 180 (90.5%) | 160 (85.6%) | 60 (92.3%) | 31 (83.8%) | 20 (95.2%) |
| 1 event | 19 (9.5%) | 20 (10.4%) | 7 (7.7%) | 6 (16.2%) | 1 (4.8%) |
| > 1 event | 161 (88.6%) | 140 (73.5%) | 53 (86.4%) | 25 (76.5%) | 19 (95.2%) |
| Diagnosis at admission: N (%) | 84 (42.2%) | 80 (40.2%) | 31 (51.7%) | 13 (35.1%) | 7 (33.3%) |
| Infectious Medical Diagnosis | 33 (43.4%) | 35 (46.1%) | 13 (47.7%) | 7 (33.3%) | 3 (14.3%) |
| Non-Infectious Medical Diagnosis | 33 (16.6%) | 7 (13.8%) | 9 (13.8%) | 6 (16.2%) | 2 (7.7%) |
| Major Surgery | 2 (1%) | 1 (1.3%) | 1 (1.3%) | 1 (2.7%) | - |
| No data | 2 (1%) | 1 (0.5%) | 1 (1.3%) | 1 (2.7%) | - |

* Ventilator-Associated Pneumonia. b Central Line-Associated Bloodstream Infection. c Catheter-Associated Urinary Tract Infection. d Surgical Site Infection.
In *Staphylococcus aureus*, macrolide resistance related to genes encoding erm was found in 16.7% in CLABSI. Regarding aminoglycosides, 
aph2’Ia and ant4’-Ia were identified in 7.7%. In CLABSI resistance to aminoglycosides was associated with the presence of 
aph2’Ia (25%), aac[6’]-Ie/aph2’Ia (25%) and ant4’-Ia (25%) (Table 7).

### Discussion

This work allowing for the first time in Cartagena a phenotypic and genotypic characterization of the HAIIs in all the adult ICUs found that the most prevalent HAIIs were VAP and CLABSI. Other national and international studies also describe VAP and CLABSI as the most frequent. Since permanent notification of HAI began in Colombia (2013) CLABSI and VAP are the most frequent. International series highlight as the most affected population those approximately 60 years of age, with infection times of +/− 6 days.

This study establishes that 71.7% of the isolates in HAI are gram-negative bacteria (*P. aeruginosa* in VAP, *K. pneumoniae* and *E. coli* in CLABSI and CAUTI) and 13.1% gram-positive bacteria (*S. aureus* in VAP and CLABSI). This behavior is not far from what is reported worldwide. Data from the SIVIGILA surveillance have showed the predominance of these pathogens in national isolates, and in the current updates. In the USA, *E. coli* and *S. aureus* are the most frequent, although *P. aeruginosa* and *K. pneumoniae* have also been described. In Europe, *P. aeruginosa* leads the headlines in VAP, followed by coagulase-negative *Staphylococcus* in CLABSI and *E. coli* in CAUTI in the same way as the present study. Literature indicates these pathogens are the most reported and factors like population > 60 years, extended hospital stays, insertion and duration of invasive devices, antibiotic pressure and the breakdown of aseptic measures are associated with HAIIs by these microorganisms.

In the resistance patterns VAP exhibited the highest percentages of resistance, especially in *P. aeruginosa* against to carbapenems, quinolones and colistin. This result is associated with worse outcomes in this type of patient, data are consistent with those reported by surveillance systems such as SENTRY, ECDC and RELAVRA, generating a public health alarm affecting the outcome of these patients.

The genotypic profile showed a wide presence of ESBL associated genes of Ambler class A (*blaTEM, blaSHV, blaCTX-M, blaOXA-2*), and D (*blaOXA-1, blaOXA-4, blaOXA-23, blaOXA-48*), which have been identified in nosocomial enterobacteria, as in our case, in South America since 1987, in Chile, Buenos Aires and since 2002 in Colombia, where they are all widely distributed in the national territory. A profile that drew attention was the simultaneous presence of *blaTEM, blaSHV, blaCTX-M, blaOXA-1, blaOXA-2*, and *blaOXA-23* in strains of *K. pneumoniae* and *P. aeruginosa* in VAP and CLABSI, confirming the epidemiological behavior of these resistance markers and the poor therapeutic response compromising the outcome of the patients.

In quinolones draws attention the highest proportion of *qnrS* (12.9%), *qnrD* (15.4%), *aac[6’]-Ib-cr* (23.1%) genes in *E. coli,*
decreasing the activity of ciprofloxacin and norfloxacin, and against quinolones, posing an unfortunate scenario for this type of drug so useful in these infections.\

It is concluded that in the monitored population VAP and CLABSI are the most prevalent HAIs whose most frequent etiology is gram-negative bacteria with a resistance profile against most of the antibiotics tested, corroborated by the presence of resistance genes hindering the therapeutic approach and prognosis and generating an alert to the interventions derived from this characterization.

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**Ethical disclosures**

Protection of human and animal subjects. The authors declare that no experiments were performed on humans or animals for this investigation.
Table 6. Distribution of the resistance genes to β-lactams, quinolones and aminoglycosides in gram-negative isolates from patients with HAI in Cartagena, Colombia.

| Organism/gene       | %          | VAP% | CLABSI% | CAUTI% | SSI% |
|---------------------|------------|------|---------|--------|------|
| P. aeruginosa (n=57) |            |      |         |        |      |
| TEM                 | 31.6       | 19.2 | 45      | 42.9   | 25   |
| SHV                 | 28.1       | 15.4 | 45      | 28.6   | 25   |
| CTX-M               | 14.4       | 15.4 | 10      | 28.6   | 25   |
| KPC                 | 5.7        | 3.9  | 10      | 0      |      |
| MTSO                | 22.8       | 15.4 | 35      | 14.3   | 25   |
| OXA-48              | 1.8        | 3.9  | 0       | 0      |      |
| OXA-23              | 8.8        | 11.5 | 10      | 0      |      |
| qnrB                | 0          | 0    | -       | -      | -    |
| qnrS                | 1.8        | 33.3 | -       | -      | -    |
| qnrD                | 1.8        | 50   | -       | -      | -    |
| aac(6’)-Ib-cr       | 0          | 0    | -       | -      | -    |
| oqxA                | 1.8        | 50   | -       | -      | -    |
| oqxB                | 1.8        | 100  | -       | -      | -    |
| aac(6’)-Ib          | 0          | -    | 0       | -      | -    |
| aph(2’)-Ilf         | 1.8        | 0    | 0       | 14.3   | -    |
| aac(6’)-Ie/aph(2’)-Ia | 0       | -    | 0       | -      | -    |
| ant(4’)-Ia          | 0          | -    | -       | 0      | -    |
| K. pneumoniae       |            |      |         |        |      |
| (n=40)              |            |      |         |        |      |
| TEM                 | 57.5       | 71.4 | 55.6    | 33.3   | 50   |
| SHV                 | 37.5       | 50   | 33.3    | 33.3   | 0    |
| CTX-M               | 42.5       | 50   | 38.9    | 33.3   | 50   |
| KPC                 | 32.5       | 35.7 | 27.8    | 33.3   | 50   |
| MTSO                | 50         | 57.1 | 55.6    | 16.7   | 50   |
| OXA-48              | 0          | 0    | 0       | 0      |      |
| OXA-23              | 0          | 0    | 0       | 0      |      |
| qnrB                | 2.5        | 0    | 5.6     | -      | -    |
| qnrS                | 0          | -    | 0       | -      | -    |
| qnrD                | 0          | 0    | -       | -      | -    |
| aac(6’)-Ib-cr       | 2.5        | -    | 5.6     | -      | -    |
| oqxA                | 2.5        | -    | 5.6     | -      | -    |
| oqxB                | 7.5        | 14.3 | 5.7     | -      | -    |
| aac(6’)-Ib          | 7.5        | 7.1  | 11.1    | -      | -    |
| aph(2’)-Ilf         | 5          | 0    | 11.1    | -      | -    |
| aac(6’)-Ie/aph(2’)-Ia | 10      | 14.3 | 11.1    | -      | -    |

Table 7. Distribution of resistance genes to erythromycin, clindamycin, β-lactams, aminoglycosides, and quinolones in isolates of S. aureus and S. epidermidis in patients with HAI.

| Organism/gene       | %          | VAP% | CLABSI% | CAUTI% | SSI% |
|---------------------|------------|------|---------|--------|------|
| S. aureus (n=13)    |            |      |         |        |      |
| ermA                | 0          | 0    | 0       | 0      | 0    |
| ermC                | 7.7        | -    | 16.7    | 0      | 0    |
| mecA                | 0          | 0    | 0       | 0      | 0    |
| aac(3’)-Ia          | -          | -    | -       | -      | -    |
| aph(2’)-Ifl         | 7.7        | 14.3 | -       | -      | -    |
| aac(6’)-Ie/aph(2’)-Ia | 0      | -    | 0       | -      | -    |
| ant(4’)-Ia          | 7.7        | 14.3 | 0       | 0      | -    |
| qnrS                | 0          | -    | 0       | -      | -    |
| qnrD                | -          | -    | -       | -      | -    |
| aac(6’)-Ib-cr       | -          | -    | -       | -      | -    |
| qepA                | 0          | -    | 0       | -      | -    |
| oqxA                | -          | -    | -       | -      | -    |
| oqxB                | -          | -    | -       | -      | -    |
| S. epidermidis (n = 5) |          |      |         |        |      |
| ermA                | 0          | 0    | 0       | 0      | -    |
| ermC                | 40         | -    | 25      | 100    | -    |
| mecA                | 0          | 0    | 0       | 0      | 0    |
| aac(3’)-Ia          | 0          | -    | 0       | -      | -    |
| aph(2’)-Ifl         | 20         | -    | 25      | -      | -    |
| aac(6’)-Ie/aph(2’)-Ia | 20      | -    | 25      | -      | -    |
| ant(4’)-Ia          | 20         | -    | 25      | -      | -    |
| qnrS                | 40         | -    | 25      | 100    | -    |
| qnrD                | 20         | -    | 0       | 100    | -    |
| aac(6’)-Ib-cr       | 20         | -    | 0       | 100    | -    |
| qepA                | 40         | -    | 25      | 100    | -    |
| oqxA                | 40         | -    | 25      | 100    | -    |
| oqxB                | 40         | -    | 25      | 100    | -    |

0 Resistance gene not detected. - Gene not tested.  VAP: Ventilator-Associated Pneumonia.  CLABSI: Central Line-Associated Bloodstream Infection.  CAUTI: Catheter-Associated Urinary Tract Infection.  SSI: Surgical Site Infection.

Right to privacy and informed consent. The authors declare that no data that enables identification of the patients appears in this article.

Conflict of interest. The authors declare that the revision was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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