We aimed to investigate the antibiotic resistance and virulence markers in Gram negative bacilli (GNB) and Gram positives coccus (GPC), strains recently isolated from the hospital environment and from patients with surgical wound infections in order to obtain epidemiologically relevant data. The strains identification was performed with the automated miniApi system. The resistance phenotypes were established using disk diffusion (CLSI, 2017). 61 strains were screened for the production of enzymatic soluble virulence factors: hemolysins, amylase, caseinase, aesculin hydrolysis, DNA-ase, lipase, gelatinase and lecithinase, which give microorganisms the ability to colonize and disseminate in the host. Multiplex PCR reactions were performed for the detection of carbapenemases, aminoglycoside-resistant determinants (AME's), quinolone and tetracycline resistance in GNR and SCCmec cassette type in Staphylococcus aureus strains and carbapenemases and ESBLs producers proved to be positive for the majority of the tested soluble virulence factors, proving the pathogenic potential of these strains. In S. aureus isolates the molecular analysis showed that 60% of the isolates were MRSA and the molecular analysis revealed the presence of the SCCmec cassette type mec IV a and III types. Our data suggest the hypothesis according to which nosocomial origin of the strains can be explained by multiple drug resistance and virulence determinants.

Keywords: resistance, virulence, nosocomial infections

Antimicrobial resistance (AMR) represent a growing public health which consist in the capacity of the microorganisms to survive exposure to antibiotic treatment [1]. Infections caused by multidrug resistant (MDR) and virulent Gram-positive and Gram negative bacteria are very common in hospital settings but recently there have been described that are involved also in community environments [2]. The bacteria included in ESKAPE acronym (Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa, and Enterobacter spp.) are presently causing most of the nosocomial infections in hospital settings [3-5]. There are several intrinsinc factors for e.g. point mutation, gene amplification and extrinsic factors like horizontal transfer of resistant gene by mobile genetic elements such as transposons, integrons or plasmids responsible for the development of AMR. The excessive use of antibiotics is strongly related to the widespread of antibiotic resistant bacteria, especially in Intensive Care Units (ICUs) all over the world, which result in increasing the mortality rates [6]. Several factors affect the risk of nosocomial infections, including underlying disease, severity of illness, length of ICU stay, and usage of invasive devices and procedures.

Experimental part

Bacterial strains and phenotypic analysis

The study included 61 recently isolated (Sept-Dec 2017) belonging to GNR (Escherichia coli (n = 15); A. baumannii (n = 20)) and GPC (S. aureus (n = 26)). The hospital strains were identified using Api 20 E/Api 20 NE/ API Staff system and confirmed by VITEK2 automatic system.

The antibiotic susceptibility was determined by Kirby-Bauer standard disk diffusion method [using the antibiotics recommended by CLSI, 2017, 2018 for E. coli us following: meropenem (MEM), imipenem (IMP), ertapenem (ETP), cefazolin (CFZ) cephalotin (CEF) ceftriaxon (CTX), cefpodoxim (CFP) cefuroxime (CMX), cefoxitin (FOX), ceftazidim (CAZ), aztreonam (ATM), cepefime (FEP), amoxicillin-clavulanic acid (AMC), piperacillin-tazobactam (PIP-TZP), ciprofloxacin (CIP), levofloxacine (LEV)
gentamycin (GEN), amikacin (AMK), kanamycin (KAN), nitrofurantoin (NIT), trimethoprim-sulfamethoxazole (SXT), tetracycline (TET), chloramphenicol (CHL) and colistin] and quality control was performed with E. coli ATCC 25922, for A. baumannii a reference strain of Pseudomonas aeruginosa - ATCC 27853 and for S. aureus penicillin (PEN), oxacillin (OXA), vancomycin (VAN), gentamycin (GEN), tetracycline (TET), ciprofloxacin (CIP), nitrofurantoin (NIT), trimethoprim-sulfamethoxazole (SXT), chloramphenicol (CHL), rifampin (RIF), linezolid (LZD), clindamycin (CLI) and azithromycin (AZM) using as reference strains S. aureus ATCC 25923 and by automated methods (Vitek II).

**Evaluation of the soluble enzymatic factors**

The virulence phenotypes were investigated by performing enzymatic tests for the expression of the following soluble virulence factors in overnight culture: haemolysins, DN-ase, pore forming toxins (lecithinase, lipase), proteases (caseinase, gelatinase), amylase and aesculin hydrolysis. Detection of haemolysin production was performed by spotting the fresh cultures on 5% sheep blood agar medium and incubation at 37°C for 24h. The colourless area around the culture revealed the presence of haemolysis activity. For DNA-ase test, the hydrolysis of DNA in the agar by bacterial DNA-ase activity reduces the agar pH. Positive result if appear a clear zone around growth area. For lipase production the strains were spotted on 1% Tween 80 agar as a substrate and followed by incubation at 37°C for 24 h and an opaque zone around the spot revealed the positive reaction; for lecithinase production, the cultures were spotted into 2.5% yolk agar and incubated at 37°C for 24 h. A clear zone around the spot indicated the presence of lecithinase production. The protease activity (caseinase and gelatinase) was determined using 15% soluble casein agar, respectively 3% gelatine as substrate. The strains were spotted and after incubation at 37°C for 24 h, a white precipitate surrounding the growth indicated casein proteolysis, and colourless area around culture due to the gelatin hydrolysis, indicated the positive reaction forgelatinase. Amylase was detected using agar with 1% starch and hydrolysis was revealed after adding Lugol’s solution (yellow ring around the culture, while the rest of the plate will be blue). For the aesculin hydrolysis the medium containing Fe³⁺ citrate was used and inoculated by spotting, then incubated for 24h at 37°C temperature. A black precipitate around culture due to esculetin released under the action of beta-galactosidase was considered positive reaction.

**Molecular analysis**

Genetic support of ARGs and virulence in GNR

The genetic support of the resistance (carbapenemases, ESBLs, quinolones aminoglycosides and tetracyclines) and virulence in GNR strains (table 1, 2, 3, 4) was investigated by simplex and multiplex PCR, using a reaction mix of 20µL (PCR Master Mix 2x, Thermo Scientific) containing 1µl of bacterial DNA extracted using the alkaline extraction method (table 1).

**Screening of S. aureus resistance and virulence genes by PCR.**

The genotypic characterization of the SCCmec cassette types present in the analysed strains was performed using PCR methods (simplex and multiplex) in order to elucidate the structure of these genetic elements and to obtain the relevant epidemiological data. Two reactions were performed using the multiplex PCR with five and four pairs of specific primers respectively for the various sequences of the SCCmec cassette. Their classification and parameters used to conduct the reactions followed the protocol developed by Miheirico et al. [21] and Zhang et al. [22]. The detection of the specific virulence genes was performed by three simplex PCR and three multiplex PCR assays according with previous published protocols [23].

The detection of the specific virulence genes was performed by three simplex PCR and three multiplex PCR assays (table 5). The information obtained was used to compare the prevalence of specific resistance and virulence genes amongst nosocomial strains isolated from the hospital settings.

In order to achieve samples in PCR reaction, was used PCR thermal Bio-Rad.

| The gene | Concentration | Final volume |
|----------|---------------|--------------|
| **primer** | MgCl₂ | dNTP | DNA Taq-pol | Reaction buffer | DNA |
| blaTEM | 0.5µM | 1.2mM | 2µM | 0.2U | 1x | 10x | 20µl |
| blaCTX | | | | | |
| blaKON | | | | | |
| blaOXA | | | | | |
| blaGIM | | | | | |
| MecA | | | | | |
| MecB | | | | | |
| QmrA | | | | | |
| gryB | | | | | |
| GayE | | | | | |
| accA | | | | | |
| tetA | | | | | |
| tetB | | | | | |
| tetC | | | | | |
| tetD | | | | | |

**Table 1**

THE COMPOSITION OF THE REACTION MIX
### Table 2
**PRIMERS SEQUENCES USED IN SIMPLEX AND MULTIPLEX PCR ASSAYS FOR CARBAPENEM RESISTANCE GENES**

| The gene | Primer | Nucleotide sequence | Amplification size and Tm | References |
|----------|--------|---------------------|--------------------------|------------|
| *blaOXA-41* | OXA-F | CGGTGGTATAGGATAGGACAC | 438 | 52°C | [7, 8] |
|           | OXA-R | CATCAAGTCTAACCACCCACCG |            |            |            |
| *blaNDM* | NDM-F | GGTCTGCGATCCTGCTTTC | 621 | 52°C |            |
|           | NDM-R | CGGAATGCTCATCAAGGATAC |            |            |            |
| *blaTEM* | TEM-F | ATGAGTTTCAACATTTTCG | 861 | 59°C | [9] |
|           | TEM-R | TTACCAATCTCATTAAATCG |            |            |            |
| *blaSIV* | SIV-F | GCTCTCAGAAGAGGTGTGA | 888 | 58°C | [10] |
|           | SIV-R | TTAACGTCGTGCAATCTCG |            |            |            |
| *blaVIM* | VIM-F | CGGTGGTGACGAAGTGTGC | 730 | 59°C | [11] |
|           | VIM-R | CGGCGGGAGGATAGGGATAC |            |            |            |
| *blaOXA-23* | OXA-23F | ATGACTTATACTATTTTGTTC | 301 | 52°C | [12] |
|           | OXA-23R | TGTCAGAAGCTCAAATTAATA |            |            |            |
| *blaOXA-24* | OXA-24F | GTACATAATCAAGGATGGA | 245 | 52°C |            |
|           | OXA-24R | CCCCCTTAAACGTAATTTTG |            |            |            |

### Table 3
**PRIMERS SEQUENCES USED IN SIMPLEX AND MULTIPLEX PCR ASSAYS FOR QUINOLONE, AMINOGLYCOSIDES AND TETRACYCLINE GENES**

| QuinA-R | AGA CCA TTT TCT ACC CCA GG |
| QuinB-R | TGC CAG GCA CAG ATC TNG AC |
| QuinC-R | GMG ATH GAA ATT CGC CAC TG |
| QuinD-R | TTT GCC YTY CGC CAG TCG AA |
| QuinE-R | TCT AAA CCG TCG AGT TCG CGG |
| Gyrf-B-F | GCGCGACAGTGCGCCCGCGCA |
| Gyrf-B-R | CGCAGGAAGAGATCTCATCAAC |
| ParE-F | CGCGGTGTTGCTCCTCGGGCGTGATGAAGGA |
| ParE-E | TCCAGGGCGTCTAGATGTCCCCGCA |
| acc-3-la-F | ATGCCACATCATTGGCCCA |
| acc-3-la-R | TCCGCGCTTGAACAGGATTTG |
| sapB-F | ATGGGACAACACGCGGAGGTCGC |
| sapB-R | TTAGCCCGCAATATGCGGAC |
| tetA | GCGCGATCTGGTICACTCCG |
| tetB | TACCTGAAATTTATCTCTGG |
| tetC | GCCCGATATCCGTATCTCCG |
| tetD | GGAATATCCTCCCCGAGGGC |

### Table 4
**PRIMERS SEQUENCES USED IN SIMPLEX AND MULTIPLEX PCR ASSAYS FOR VIRULENCE GENES IN GNR ISOLATES**

| The gene | Primer | Amplification size and Tm | References |
|----------|--------|--------------------------|------------|
| hlyA | ACAACAGGATATGAGACTGT | 1,177 bp | 60°C | 116 | [18] |
| sI1a/E | CGAGAGAATATATCAAAAAACTGGA | 408 bp | 60°C | 19 | [19] |
| popC2 | GAGCAAGTGGACTGAAACCTGGG | 328 bp | 63°C | 19 | [19] |
| fimH | TGC AGA AGG GAT AAG CCG TGG | 508 bp | 63°C | [17] |
| cwa | GCA GIC ACC TGG CCT CCG GTA | | | |
| evf | GAATTTTGCATTATTGCTGATG | 541 bp | 40°C | 20 | [20] |
| cexA | GCCCTTGATGGGATGGCCCGCA | 65°C | 494 bp | Designed by Chifferia |
| bpaA | F. CAAATGGCTCCGGCCTTGGT | 65°C | 324bp | Designed by Chifferia |
| eae | CAGGCGTAAACAGAAAGATGTAAT | 32°C-1 M | 397bp | Designed by Chifferia |
Results and discussions
Phenotypic results of the distribution of resistance profiles in analysed E. coli isolates have shown that the majority of the strains were resistant to IMP, CAZ and FEP (73.33%), followed by MEM and AMK (66.66%). 53.33% of the strains were resistant to cephalosporin's (CTX, CXM), PIP-TZP and CIP. A low percentage of E. coli strains were resistant to ATM and ETP (20%) (fig. 1). A. baumannii antibiotic resistance profiles revealed a high level of

| Table 4 |
| CONTINUED |

| Gene | Primers | Nucleotide sequence | Amplification program |
|------|---------|---------------------|-----------------------|
| AggR | CGAAGTATACACAAAGGAAAGGTA | 56°C, 640bp | Designed by Chifriuc |
| EAggE | CGGCGAAAATCTGTATATTT | 56°C, 630bp | Designed by Chifriuc |
| ompA | GGTTCGCTGATCGTGAAT | 531 | [13] |
| ampA | AGCAAGCTGATATACCCGATCC | 451 | |

| Table 5 |

NUCLEOTIDE SEQUENCES OF PRIMERS USED IN THE IDENTIFICATION OF THE VIRULENCE GENES

| Gene | Primers | Nucleotide sequence | Amplification program |
|------|---------|---------------------|-----------------------|
| bop | BBP-1 BBP-2 | AACTACATCTTCTTCAATTCCTGCTTCACTCAG ATGCCTTGAAATAAACAACCA TAC | 94°C, 5 min 25 94°C, 1 min 55°C, 1 min 72°C, 1 min 72°C, 10 min |
| ebpS | EBP-1 EBP-2 | CATCCAGAAGCAATTGGAACAC AGAAGGTGTACCTGATCATGTTATC | 94°C, 5 min 25 94°C, 1 min 55°C, 1 min 72°C, 1 min 72°C, 10 min |
| fnbB | FNBB-1 FNBB-2 | GTACACGGTAAATGTCGAA TTGATCTC GAAGGTGATAGGATGTA TATGTC | 94°C, 5 min 25 94°C, 1 min 55°C, 1 min 72°C, 1 min 72°C, 10 min |
| fliB | FIB-1 FIB-2 | GCTACACTACATTGCCCTTAAACGACG CACCATTCGGTTTGAC | 94°C, 5 min 25 94°C, 1 min 55°C, 1 min 72°C, 1 min 72°C, 10 min |
| cifA | CLFA-1 CLFA-2 | ATGGCCGACTGCTATCATGCT | 94°C, 5 min 25 94°C, 1 min 55°C, 1 min 72°C, 1 min 72°C, 10 min |
| cifB | CLFB-1 CLFB-2 | ACATCAATATATATAGCCGG GCAAC TTTGCAACTTACCTGTTTCGC AC | 94°C, 5 min 25 94°C, 1 min 55°C, 1 min 72°C, 1 min 72°C, 10 min |
| fnbA | forward reverse | CTAGGAAAATATAGCTTCGTTGTTAAGCAATGGGC | 94°C, 5 min 25 94°C, 1 min 55°C, 1 min 72°C, 1 min 72°C, 10 min |
| cma | forward reverse | AGIGGCTACGTTACATTGC CAACTGTTAATGGTCCA | 94°C, 5 min 25 94°C, 1 min 55°C, 1 min 72°C, 1 min 72°C, 10 min |
| coag | CI C2 | CGAGAAGAAAGAGCTTACAAGAA GAAAGAAAACCACCTCAACTAC AGT | 94°C, 5 min 40 94°C, 30 sec 55°C, 30sec. 72°C, 1 min 72°C, 5 min |
| luk-PV | luk-PV-1 luk-PV-2 | ATCAAGTCGATATGGCTTGGACAGATCCCA GCTCAGTGATGGTTAGA GCAAACGC | 95°C, 5 min 30 95°C, 30 sec 55°C, 2 min. 72°C, 1 min 72°C, 10 min |
| hig | higC higB | GCCAACCCGTTATGAAGA GATGC CCATGACCAGTACAACCGGA T | |
| tst | tst1 tst2 | CATTACAAAGCCATATAT AAAGC CATTATTATTTTCAATTAC CACCGC | 94°C, 5 min 30 94°C, 30 sec 58°C, 30 sec 72°C, 2 min 72°C, 3 min |
resistance to MEM, AMK, GEN and SXT (85% of the isolates); quinolones (84% were resistant to CIP and LEV) followed by IMP (80%), TET (70%) and CAZ (68%). The lowest level of resistance was founded to cephalosporin’s (fig. 1).

The distribution of carbapenemases in GNR rods have shown the presence of carbapenemases OXA-48 (70% of the strains) and TEM (10%) in E. coli (); OXA-23 and OXA-24 in A. baumannii strains (53% respectively 47%). Previously, Bonin et al., in 2011 revealed the co-expression of bla<sub>OXA-23</sub> and bla<sub>OXA-24</sub> in A. baumannii isolated from patients in Timișoara, Arad and Resita [24]. More recently Gheorghe et al., in 2015 demonstrated the presence of bla<sub>OXA-23</sub> in A. baumannii isolated from patients in Bucharest, Romania [25]. In 2016 Georgescu et al., demonstrated first time the presence of one variant of bla<sub>OXA-48</sub> in A. baumannii isolated from chronic leg ulcer samples [26]. In 2017 Dahdouh et al., revealed also a high prevalence of blaOXA-24 with an increased of virulence factors production in carbapenem resistant Acinetobacter baumannii (CRAB) isolated between 2009 and 2013 from a Spanish hospital (Madrid) [27]. Similar to our results, Pournaras et al., in 2017 have shown that the OXA-58 was the predominant carbapenemase revealed among CRAB isolated from 11 hospitals between 2000 and 2009 in Greece [28].

The distribution of virulence factors of the analysed strains has revealed that the majority (86.88%) were positive for lipase, followed by lecithinase.

### Table 6

| Strain Code/no | Activin hydrolisis | DN-ase | Lipase | Caseinase | Lecithinase | Gelatinase | Amylase | Hemolysin |
|----------------|--------------------|--------|--------|-----------|------------|-----------|---------|-----------|
| 1 E.coli       | +                  | +      | +      | +         | -          | +         | α       |           |
| 2 E.coli       | +                  | -      | +      | +         | +          | -         | +       | γ         |
| 3 E.coli       | -                  | -      | +      | -         | -          | +         | +       | β         |
| 4 E.coli       | +                  | -      | +      | -         | +          | -         | +       | α         |
| 5 E.coli       | -                  | -      | +      | -         | -          | +         | +       | β         |
| 6 E.coli       | +                  | -      | +      | -         | -          | -         | -       | -         |
| 7 E.coli       | +                  | +      | +      | +         | -          | -         | -       | -         |
| 8 E.coli       | -                  | -      | +      | -         | -          | -         | -       | -         |
| 9 E.coli       | +                  | -      | +      | -         | -          | -         | -       | -         |
| 10 E.coli      | -                  | -      | +      | -         | -          | +         | -       | β         |
| 11 E.coli      | +                  | -      | +      | +         | -          | -         | -       | γ         |
| 12 E.coli      | +                  | +      | +      | -         | -          | -         | -       | α         |
| 13 E.coli      | +                  | -      | +      | -         | -          | -         | α       |           |
| 14 E.coli      | +                  | -      | +      | -         | -          | -         | -       | -         |
| 15 E.coli      | +                  | +      | +      | -         | -          | -         | -       | α         |
| 16 E.coli      | -                  | +      | +      | +         | -          | -         | +       | α         |
| 17 E.coli      | -                  | +      | +      | -         | -          | +         | +       | α         |
| 18 E.coli      | -                  | +      | +      | -         | -          | +         | +       | α         |
| 19 E.coli      | -                  | -      | +      | -         | -          | +         | γ       |           |
an enzyme involved in dissemination of the infections, (62.29% of the isolates) aesculin hydrolysis (57.37%), caseinase - a protease that contribute to tissue degradation (47.54%) and DN-ase (40.98%) (table 6). A low percentage of investigated strains produced gelatinase which revealed the production of proteases with large-spectrum proteolytic activity. Similar to this study, Gheorghe et al., in 2017 revealed a high percentage of S. aureus isolated from acneiform reactions pustule and periungual lesions were positive for lecithinase, lipase and caseinase [31].

**Table 6**

| 20 Aabc | - | + | + | - | - | - | + | γ |
|----------|---|---|---|---|---|---|---|---|
| 21 Aabc | - | + | + | - | - | - | + | γ |
| 22 Aabc | - | - | + | - | - | - | + | γ |
| 23 Aabc | + | - | + | - | - | - | + | α |
| 24 Aabc | + | - | + | - | - | - | + | α |
| 25 Aabc | + | - | + | - | - | - | + | α |
| 26 Aabc | - | + | + | - | - | - | - | γ |
| 27 Aabc | - | + | + | - | - | - | - | γ |
| 28 Aabc | - | - | - | - | - | - | - | γ |
| 29 Aabc | - | - | - | - | - | - | - | α |
| 30 Aabc | - | + | + | - | - | - | - | α |
| 31 Aabc | - | + | + | - | - | - | - | α |
| 32 Aabc | - | + | + | - | - | - | - | α |
| 33 Aabc | - | - | + | - | - | - | - | α |
| 34 Aabc | - | - | + | - | - | - | - | α |
| 35 Aabc | - | + | + | - | - | - | - | γ |
| 36 S. aureus | + | + | + | - | + | + | - | γ |
| 37 S. aureus | - | + | + | + | + | + | - | α |
| 38 S. aureus | + | - | + | - | + | + | - | γ |
| 39 S. aureus | + | + | + | - | + | + | - | γ |
| 40 S. aureus | + | - | - | + | + | - | - | α |
| 41 S. aureus | + | + | - | + | + | - | - | γ |
| 42 S. aureus | + | - | + | + | + | - | + | β |
| 43 S. aureus | + | - | + | - | + | + | - | β |
| 44 S. aureus | - | + | + | + | - | - | - | α |
| 45 S. aureus | + | - | + | + | + | - | - | α |
| 46 S. aureus | + | - | + | - | + | - | - | α |
| 47 S. aureus | + | + | - | - | + | - | - | β |
| 48 S. aureus | + | - | + | + | - | - | - | β |
| 49 S. aureus | + | - | - | - | + | - | + | α |
| 50 S. aureus | + | - | + | + | - | - | - | β |
| 51 S. aureus | + | - | - | - | + | - | - | β |
| 52 S. aureus | + | + | + | + | + | - | - | γ |
| 53 S. aureus | + | + | + | + | - | - | - | α |
| 54 S. aureus | + | + | + | + | - | - | - | γ |
| 55 S. aureus | + | + | - | - | + | - | - | γ |
| 56 S. aureus | + | + | + | + | + | - | - | γ |
| 57 S. aureus | + | + | + | + | + | - | + | α |
| 58 S. aureus | - | - | + | + | + | - | - | γ |
| 59 S. aureus | + | - | + | + | + | - | - | α |
| 60 S. aureus | + | - | + | + | + | - | - | β |
| 61 S. aureus | + | + | + | - | + | - | - | α |

*E. coli* virulence factors are represented by adhesins [P fimbriae (papG), type 1 fimbriae (fimH), S fimbriae (sfa) and A fimbriae (afa)]; toxins, such as hemolysin A (hlyA) and cytotoxic necrotizing factor 1 (cnf1); iron uptake, such as aerobactin (aer); protectins, such as serum resistance (traT); and others, such as pathogenicity-associated islands (PAIs) and Tir-containing protein of *E. coli* (tcpc) [32-34]. Regarding virulence markers in *E. coli* isolates our study revealed the presence of fimH gene (66% of the strains); sfaDE (46.66%) and cnf1 (33.33%). In Romania, Mladin et...
al., in 2009 revealed the presence of fimH (in high percentage) and cnf1 genes in E. coli nosocomial strains isolated from the Neuropyschiatric Clinical Hospital of Craiova, during December 2006 - November 2007 [35].

Opposite to us, Chelariu et al., in 2017 demonstrated a low percentage of this virulence markers in Enterobacteriaceae strains isolated from stool samples in patients with metabolic syndrome [36]. Mitache et al., revealed the co-expression of several virulence markers: fimH, papC, sfαDE and cnf1 in Enterobacteriaceae isolated from hospital surfaces after decontamination with quaternary ammonium compounds, triclosan and iodine disinfectants in Public Health Diagnostic and Research Laboratory from Bucharest in 2017 [30].

In A. baumannii, the investigated isolates are equipped with not only enzymatic resistance mechanisms, but also the ompA biofilm-producing virulence factor (66.66% of the analysed strains). Similar to our study, Handal et al., in 2017 have demonstrated a high percentages of A. baumannii isolates positive for OmpA gene [15].

The molecular analysis of selected virulence genes in S. aureus isolates showed that 40% were positives for clfA gene, 35% for clfB gene and a lower percentages for fib and hlg genes (15% and 7% respectively). These results regarding the presence of the clfA, clfB, fib and hlg genes highlight the importance of the adherence stage in the development of the invasive infections determined by S. aureus regardless of the infectious sources. Very closer percentages were demonstrated by Gheorghe et al., in 2017 in S. aureus strains isolated in 2016 from acneiform reactions pustule and periungual lesions in patients with cutaneous drug adverse reactions in Bucharest, Romania [31].

Conclusions

The obtained data revealed that the isolated strains harbour multiple drug resistance and virulence determinants, raising the need for the implementation of screening and intervention measures for the prevention of infections with MDR and virulent strains occurred in hospitalized patients.

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