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Surveillance of antimicrobial resistance among clinical isolates recovered from a tertiary care hospital in Al Qassim, Saudi Arabia

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Abstract

Background: The emergences of antimicrobial-resistances have become an important issue in global healthcares. Limitations in surveying hinder the actual estimates of resistance in many countries.

The aim: the present study was designed to retrospectically survey antimicrobial susceptibility for resistance profiling of dominant pathogens in a tertiary-care center in Buraidah, Saudi Arabia from January-2011 to December-2011.

Materials and Methods: the design was cross-sectional and spanned records of a 1000 bacterial non-related isolates. Antibiograms were based on the 2012 Clinical and Laboratory Standards Institute guidelines.

Results showed that *Staphylococcus aureus, Acinetobacter baumannii, Klebsiella pneumoniae, Pseudomonas aeruginosa, Staphylococcus epidermidis, and Escherichia coli*, were the most resistant. All isolates of *S. aureus*, *S. epidermidis*, and *Staphylococcus haemolyticus,* were resistant to penicillin (100%), and oxacillin with 52%, 75%, and 82%, respectively. Interestingly, an increasing trend of resistance-pattern was seen for the three species against gentamicin 26%, 50%, 68% ciprofloxacin 22%, 50%, 68%, tetracycline 30%, 44%, 27%, erythromycin 26%, 64%, 73%, and clindamycin 20%, 47%, 50% suggesting potential between- species transfer of resistances. *Acinetobacter baumannii* was resistances to all antibiotics tested including ciprofloxacin (90%), ceftazidime (89%), cefepime (67%), Trimethoprim/sulfamethoxazole (66%), amikacin (63%), gentamicin (51%), tetracycline (43%), piperacillin-tazobactam (42%), and imipenem (9%). A similar pattern was seen by *P. aeruginosa*. Furthermore, a typical pattern of resistance in K. pneumoniae carbapenemase–producing organisms was observed.

Conclusion: we have shown staphylococci, *Acinetobacter baumannii, Pseudomonas aeruginosa*, and enteric bacteria were the most resistant species in this region.

Key Words: Antimicrobial-Resistance, nosocomial-pathogens, AST-surveillance program, infection control

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Introduction

The recent emergences of nosocomial multidrug resistant bacteria have been significant healthcare and economics issues around the globe. [1, 2, 3] For example, a recent mulitcenter study has shown that preventing a single case of surgical infection due to methicillin resistant *Staphylococcus aureus* (MRSA) could save the hospital as much as $60,000. [4] Furthermore; in Canada, direct cost due to MRSA infection averaged $82 million in 2004 and was proposed to reach $129 million by 2010. [5] Similarly, the development of antimicrobial resistance among gram negative bacteria created difficulties in treating infected patients in hospitals. [6] For many years the beta-lactam antibiotics (broad spectrum penicillins and cephalosporins) have been the main therapeutic options for the treatment of enterobacterial infections. However, the widely reported emergences of extended spectrum beta-lactamase (ESBL)-producing strains have limited the use of these drugs. The genes for ESBL resistance are encoded on freely transmissible genetic elements, greatly increasing the risk of spread of resistance to other organisms. In recent years there have been several reports on the rise of carbapenem-resistant bacteria. [7] These bacterial species included *K. pneumoniae, E. coli,* and *S. marcescens* which showed resistant to multiple classes of antibiotics, including carbapenems, cephalosporins, fluoroquinolones, and aminoglycosides. [8] Thus, due to the rapid global spread of resistances resulting in significant losses, several initiatives have been made to implement monitoring programs of which surveillance is one of the most important. [9] A successful example is the European Antibiotic Resistance Surveillance System that has been in place since the year 2000. [10]

Scheduled screening and assessment of antimicrobial susceptibility patterns in hospitals have been successful in uncovering some crucial factors on how some bacterial strains rapidly develop pan-resistance. For instance, due to consistent reporting over the last 26 years, a number of factors have been identified that contributed to the increased resistance in nosocomial and community-acquired pathogens including ESBL strains in Europe. These included the overuse of antibiotics in humans and animals, hospital cross-infection, human migration, and changes in the food chain. [10]

In Canada, an integrated action plan showed the commonly used drug prescription rates in the country. [11] Thus, a strong evidence exists from population genetics that the development of new resistance is an outcome of antibiotic selective pressure. For example, correlation of outpatient antibiotic use with prevalence of penicillin-nonsusceptible *Streptococcus pneumoniae*, macrolide-resistant *S. pneumoniae*, and macrolide-resistant *S. pyogenes* in 20 countries have shown that streptococcal resistance is directly associated with antibiotic selection pressure on a national level. [12] In addition, development of rapid resistances has also been found to occur through novel mechanism(s). For instance, how *Acinetobacter baumannii* rapidly developed broad resistance has been quite elusive. [10,13] Recent studies suggested that this species has novel abilities to survive in a diverse range of environments due to genomic plasticities and elaborate resistance gene transfer mechanisms that occur through the release of outer member vesicles or other horizontal means. [14, 15] In the aforementioned studies two carbapenem-resistant clinical strains of *A. baumannii* (AbH12O-A2 and AbH12O-CU3) expressing the plasmid-borne bla(OXA-24) gene (plasmids pMMA2 and pMMCU3, respectively) were used to demonstrate that *A. baumannii* releases outer membrane vesicles (OMVs) during *in vitro* growth. These OMVs harbored the bla(OXA-24) gene. The incubation of these OMVs with the carbapenem-susceptible *A. baumannii* ATCC 17978 host strain yielded full resistance to carbapenems indicating that clinical isolates of *A. baumannii* may release OMVs as a mechanism of horizontal gene transfer whereby carbapenem resistance genes are delivered to surrounding *A. baumannii* bacterial isolates. In addition, *A. baumanni* and *P. aeruginosa* are well known for their intrinsic (chromosomally encoded and not horizontally transferred) resistances to a wide range of drugs. These two species can induce extraordinary resistance mechanisms against any antimicrobial agent and are becoming resistant to all commercial drugs. [16, 17, 18, 19] For these reasons, these two pathogens are capable of initiating successful infections that

frequently lead to increased mortality rates in health care systems. [20, 21] Thus, it has been widely accepted that successful measures to prevent antibiotic resistance should include scheduled surveillance programs. In this study, one-year surveillance was carried out to determine the most common antibiotics and resistant staphylococcal and gram negative bacterial species circulating in the region.

Materials and Methods

Although this study only analyzes retrospective data from bacterial isolates, and hence, would be **exempted** from criteria under Human Subject Research, ethical approval for this project was obtained from the Ethical Committee of the Ministry of Health, General Health Affairs Directorate, Training, Medical Education and Research, Al Qassim Province, No 687/44/45 and No 688/44/45 to fully comply with the request to include the details of ethical clearance.

We aimed to determine the antimicrobial resistance patterns of most commonly isolated bacterial pathogens regardless of the specimen type or infection site. Antimicrobial surveillance was conducted utilizing in-patient microbiology laboratory records for one year (January to December 2011), from King Fahad Specialist Hospital, Buraidah which is a 540-bed tertiary care center. This hospital serves patients from all socioeconomic strata within Buraidah and the surrounding regions in Al-Qassim province which has a population base of approximately one million. Clinical specimens from patients are routinely submitted to the microbiology laboratory and antimicrobial susceptibility testing results are processed and recorded. Pathogens were identified by using routine standard bacteriological methods and ID and susceptibility testing was done using automated MicroScan following standard recommendations, followed by disc diffusion testing against oxacillin and susceptibility to other non beta lactams as possible indicators for CA-MRSA. Interpretations were based on the 2012 Clinical and Laboratory Standards Institute (CLSI) guidelines. [22]

Isolates of bacterial species were tested against 20 antimicrobials prescribed for gram positive and enteric pathogens. The following antimicrobials belonging to indicated broad classifications were tested: Beta lactam penicillins[Penicillin(PEN), Oxacillin(OXA), Amoxicillin /Clavulanate(AMC), Ampicillin (AMP), (uridopenicillin piperacillin-tazobactam) (TZP)]; Beta lactam Cephalosporin [Cefuroxine (CXM), Cefotaxime (CTX), Ceftazidime (CAZ), Cefepime (FEP)]; Carbapenems [Imipenem (IPM)]; Aminoglycosides [Amikacin (AMK), Gentamycin (GEN)]; Fluroquinolone [Ciprofloxacin (CIP)]; sulphonamides[Trimethoprim/sulfamethoxazole (SXT)]; Tetracyclines[Tetracycline (TET)]; Macrolide [Erythromycin (ERY)]; lincosamide [Clindamycin (CLI)]; Nitrofurantoin [Nitrofuran (NIT)]; Glycopeptide[Vancomycin (VAN)]; and synthetic oxazolidinone drugs [Linezolid (LND)].

For these 20 antimicrobials (see table) tested against different groups of bacteria, the standard MIC breakpoints and interpretations are indicated in brackets: Gram positives *S. aureus* NCTC12973/ ATCC29213: Amoxicillin/Clavulanate (AMC, <=4/2 = Susceptible); Ampicillin (AMP, >8 beta lactamase positive); Ciprofloxacin (CIP, <=1 = Susceptible); Clindamycin (CLI, 0.5 = Susceptible); Erythromycin (ERY,<=0.5 = Susceptible); Gentamycin (GEN, <=1 = Susceptible); Imipenem (IPM, <=4 = Susceptible); Linezolid (LND, <=2 = Susceptible); Nitrofuran (NIT, <=32); Oxacillin (OXA, 1 = Susceptible), Penicillin (PEN, >8 = beta lactamase positive); Tetracycline (TET, <=4 = Susceptible); Trimethoprim/sulfamethoxazole (SXT, <=2/38 = Susceptilbe); Vancomycin (VAN, 1 = Susceptible). *E. coli* NCTC12241/ ATCC26922 and enteric Gram negatives: Amikacin (AMK, <=16 = Susceptible); Amoxicillin/Clavulanate (AMC, 16/8 = Intermediate); Ampicillin (AMP,> 16 Resistant); Cefepime (FEP, <=8 = Susceptible); Cefotaxime (CTX, <=2 = Susceptible); Ceftazidime (CAZ, <=1 = Susceptible); Cefuroxime (CXM, <=4 = Susceptible); Ciprofloxacin (CIP, <=1 = Susceptible); Gentamycin (GEN, <=4 = Susceptible); Imipenem (IPM, <=4 = Susceptible); nitrofuran (NIT, <=32); piperacillin-tazobactam (TZP, <16 = Susceptible); Tetracycline (TET, <=4 = Susceptible); Trimethoprim/sulfamethoxazole (SXT, <=2/38 = Susceptible). *Pseudomonas aeruginosa* ATCC27853 and Gram negatives: Amikacin (AMK, <=16 = Susceptible); Amoxicillin/Clavulanate (AMC,<16/8);

Ampicillin (AMP,> 16); Cefepime (FEP, <=8 = Susceptible); Cefotaxime (CTX, 16 = Intermediate); Ceftazidime (CAZ, 4 = Susceptible); Cefuroxime (CXM, >16); Ciprofloxacin (CIP, <=1 = Susceptible); Gentamycin (GEN, <=4 = Susceptible); Imipenem (IPM, <=8 = Resistant); nitrofuran (NIT, >64); piperacillin-tazobactam (TZP, <16 = Susceptible); Tetracycline (TET, >8); Trimethoprim/sulfamethoxazole (SXT, <=2/38). The isolates selected for study were confined to unrelated first isolates from different patients; multiple isolates from the same patient were excluded. Eight hundred and forty eight isolates recovered from Al –Qassim hospitals in 2010 were tested against the commonly used antibiotics.

Results

Although emphasis was placed on determining the rates of resistances of common Gram positive cocci and enteric and Gram negative bacterial pathogens (Table 1), we have also determined antimicrobial susceptibilities of these species (Table 2).

Multidrug Resistant Staphylococci

As indicated in Table 1, staphylococcal species *(S. aureus, Staphylococcus epidermidis*, and *Staphylococcus haemolyticus*) reported high levels of resistance to beta-lactam antibiotics. Isolates belonging to these three species were all resistant to penicillin with 100%; only six and one isolates of the former two species respectively were intermediate. However, while 52%, 75%, and 82% of the isolates, respectively, were resistant to oxacillin, 26%, 51%, 68% of them, respectively, were resistant to gentamicin (Table 1). *S. aureus* reported lesser resistance rates of 22%, 30%, 26%, and 20% to ciprofloxacin, tetracycline, erythromycin, and clindamycin, respectively, than the other two staphylococcal species which mostly showed over 50% resistance to these drugs (Table 1, Table 2). Vancomycin and linezolid were the most effective antibiotics against the three species. In addition, a few isolates in each species were also resistant to trimethoprim/sulfamethoxazole.

Gram Negative Bacteria

Acinetobacter baumanii reported resistance to all of the nine drugs tested ranging from only 11 isolates (9%) against imipenem to 54 (42%) isolates against piperacillin-tazobactam and 90% to ciprofloxacin. Similarly, multidrug resistant *P. aeruginosa* isolates recorded 21% resistance to the carbapenem (imipenem), 41% to ceftazidime, and 30% to cefepime, as well as 19% resistance to piperacillin-tazobactam. In addition, significant resistances were reported against gentamicin (27%) and ciprofloxacin (26%). As shown in Tables 1 and 2 the aminoglycoside amikacin and the sulphonamide combination drug trimethoprim/sulfamethoxazole and to some extent the beta lactam uridopenicillin piperacillin-tazobactam, as well as the carbapenem imipenem, were effective against *P. aeruginosa.*

Enteric Bacteria

Klebsiella pneumonia isolates were resistant to the following antibiotics; amoxicillin 46(38%), ampicillin *119(99%)* ceftazidime 50(42%), cefepime 60(50%), cefotaxime 80(67%), cefuroxine 86(72%), amikacin 11(9%), gentamicin 40(33%), ciprofloxacin 39(33%), Trimethoprim/sulfamethoxazole 76(63%), and tetracycline 29(24%) (Table). Similar to *P. aeruginosa, K. pneumoniae* isolates also showed lower resistances to imipenem and amikacin, where only 5 and 11 isolates, respectively, were resistant. Furthermore, a similar resistance pattern was also found in almost all tested *E. coli* isolates. However, larger number of isolates were resistant to ampicillin 85(97%), tetracycline 49(56%), and Trimethoprim/sulfamethoxazole 62(70%). *E. coli* isolates were also resistant to the rest of tested drugs ranging from 16% to 39%. Reports on *Proteus mirabilis* showed similar resistance pattern to that of *E. coli* for amoxicillin 18(27%), piperacillin-tazobactam 3(4%), ceftazidime 8(12%), cefepime 13(19%), ciprofloxacin 27(40%), and Trimethoprim/sulfamethoxazole 49(73%). While 22% and 28% of *P. mirabilis* isolates were resistant to amikacin and ampicillin respectively, much higher resistances were reported for the remaining drugs namely, 42% each for cefotaxime and gentamicin, 58% and 60% for tetracycline and cefuroxine, respectively. Similar to other enteric bacteria, the carbapenem and the combination drug piperacillin-tazobactam were the most effective against *Pr. mirabilits.* Enterobacter isolates

showed low levels of resistance to tested drugs except for a slightly higher number of resistances against ampicillin and amoxicillin. No vancomycin resistance was reported in this study. However, all of the 16 isolates of *S. marcescens* were resistant to amoxicillin and ampicillin while low levels of resistances were reported for the other drugs.

Discussion

In this study, we have surveyed the antimicrobial resistance patterns of the most frequently isolated pathogenic bacteria from a major hospital in Al Qassim region of Saudi Arabia. We are well aware that resistance patterns may differ in hospitals at different geographic regions; however, we found no significant variations in antimicrobials used at Al Qassim hospitals. For this reason, we focused on this major tertiary care center where specimens from other hospitals are also routinely submitted for bacteriological analysis. For *S. aureus*, the high levels of resistance to beta lactam antibiotics penicillin (100%) and oxacillin (54%) (Tables 1) with susceptibility to non beta lactams indicated a typical pattern of CA-MRSA (Table 2). The prevalence of CA-MRSA in global hospitals has been discussed widely. [23] Usually, CA-MRSA strains show susceptibility to other drugs such as erythromycin, tetracyclines, and trimethoprim-sulfamethoxazole; however, in the recent years, non–beta lactam resistances in community clones of MRSA, especially USA300, have been reported. [24, 25, 26]

The two staphylococcal species, *S. epidermidis*, and *S. haemolyticus,* recorded similar resistance patterns to that of *S. aureus* for penicillin and oxacillin and were both susceptible to the last resorts vancomycin and linezolid (Table 2). A recent report suggested that specific *SCCmec* IV subtypes among these species mediate between species transfer of beta lactam resistance. [27] Nevertheless, unlike *S. aureus*, much higher rates of resistance to other antibiotics such as gentamicin, ciprofloxacin, trimethoprim/sulfamethoxazole, tetracycline, erythromycin, and clindamycin were recorded among the two staphylococcal species. The evolution of *S. epidermidis* into significant healthcare associated pathogen has been attributed to the potential recombination events and the acquisition of mobile genetic elements. [28]This would further explain the role of *S. epidermidis* as potential reservoir for drug resistance among nosocomial pathogens. [29] Furthermore, staphylococcal species with similar antibiogram patterns have been increasingly co-isolated from positive blood cultures. [30] Thus, regional surveys of antimicrobial susceptibility patterns might also provide clues to possible sources of species-specific resistances and therapies as has been successfully applied to different regions. [31, 26]

Among gram negative species, *A. baumannii* recorded one of the highest rates of resistances to all antibiotics tested with nearly 100% to many of them (Table 1). Although carbapenem resistance was lower (Table 2), there is a potential risk for rapid resistance transfer because the organism has many elaborative mechanisms. [14] Due to this, significant attention from the public, scientific, and medical communities has been given to this species. [15] Similarly, high rates of multidrug resistant *P. aeruginosa* isolates were recorded in 2011 with 21% imipenem, 41% ceftazidime, and 30% to cefepime. In addition, significantly higher number of isolates was resistant to gentamicin (27%) and ciprofloxacin (26%) (Table). However, ampicillin, amikacin, and trimethoprim-sulfamethoxazole were effective against *P. aeruginosa* (Table 2). Low levels of resistances were reported against the last resort piperacillin-tazobactam. Thus, *A. baumanii* and *P. aerugenosa* constituted significant risks in this region, consistent with many reports in different regions. [16, 17, 18] Based on the suggested terminologies, the isolates of *A. baumanii* and *P. aeruginosa* reported in this study could be described as multidrug resistant. [32]

The emergence and spread of carbapenem-resistant enteric bacteria have been a major clinical and public health challenge. [7] In this study, the prevalence of imipenem resistance among *K. pneumoniae* isolates that were simultaneously resistant to other antibiotics is consistent with Bratu et al.'s finding that *K. pneumoniae* carbapenemase–producing organisms such as *K. pneumoniae, E. coli,* and *S. marcescens* were typically resistant to multiple classes of antibiotics, including carbapenems, cephalosporins, fluoroquinolones, and aminoglycoside. [8] These clones were initially reported in 2001, and subsequently have been reported from at

least 10 countries in four continents. [33] In addition, coliforms resistance to impenem with increased resistance to other drugs such as ampicillin, tetracycline, and trimethoprim/sulfamethoxazole represented a similar concern, in agreement with Bratu et al.'s finding. [8] Resistance rates of enterococci and serratia were generally low, and no vancomycin resistant enterococcus (VRE) was reported in this study (Tables 1 and 2). However, Khan et al., (2008) [34] reported that 33 of the 34 VRE isolated from two large tertiary-care hospitals in Riyadh region (Saudi Arabia) belonged to the global clonal complex (CC17).

Conclusion

Thus, in this study we have assessed antimicrobial resistances against 20 antimicrobials in a major hospital during 2011. While penicillin and methicillin resistant profiles of staphylococci were dominant, vancomycin and linezolid were still effective. Of significant concern was the increased rate of multidrug resistance *A. baumanii* and *P. aeruginosa*. Although there was an increasing risk for potential multidrug resistances such as carbapenem resistance among enterobacteriaceae, there still remained a safer range of therapeutic options for this group, except for the narrow range of options for *K. pneumoniae.*

Conflict of interest

The authors declare that they have no conflict of interest

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Table 1. Resistance rates against antibiotics used in King Fahad Specialist Hospital, Buraidah, Al-Qassim, Saudi Arabia

*The numbers in brackets indicate percentages of resistant isolates;-*** Susceptibility not determined/not available.

		Antibiotics tested																			
Bacterial spp	#isolate	PEN	OXA	AMC	IPM	AMP	TZP	CXM	CTX	CAZ	FEP	AMK	GEN	CIP	SXT	TET	ERY	CLI	NIT	VAN	LND
S. aureus	157	151 (100)*	81 (52)	-**	-	-	-	-	-	-	-	-	40 (26)	35 (22)	12 (8)	47 (30)	41 (26)	32 (20)	-	2 (1)	1
S. epidermidis	90	89 (100)	67 (75)	-	-	-	-	-	-	-	-	-	45 (50)	45 (50)	5 (6)	40 (44)	58 (64)	42 (47)	-	-	5 (6)
S. haemolyticus	22	22 (100)	18 (82)	-	-	-	-	-	-	-	-	-	15 (68)	15 (68)	-	6 (27)	16 (73)	11 (50)	-	-	-
A. baumannii	128	-	-	-	11 (9)	-	54 (42)	-	-	114 (89)	86 (67)	80 (63)	65 (51)	115 (90)	84 (66)	55 (43)	-	-	-	-	-
P. aeruginosa	111	-	-	-	23 (21)	1	21 (19)	-	-	45 (41)	33 (30)	11 (10)	30 (27)	29 (26)	2 (2)	-	-	-	-	-	-
K. pneumoniae	120	-	-	46 (38)	5 (4)	119 (99)	27 (23)	86 (72)	80 (67)	50 (42)	60 (50)	11 (9)	40 (33)	39 (33)	76 (63)	29 (24)	-	-	-	-	-
E. coli	88	-	-	18 (20)	-	85 (97)	3 (3)	34 (39)	32 (36)	14 (16)	23 (26)	1	15 (17)	25 (28)	62 (70)	49 (56)	-	-	-	-	-
P. mirabilis	67	-	-	18 (27)	2 (3)	19 (28)	3 (4)	40 (60)	28 (42)	8 (12)	13 (19)	15 (22)	28 (42)	27 (40)	49 (73)	39 (58)	-	-	-	-	-
E. cloacae	33	-	-	14 (42)	-	18 (55)	2 (6)	7 (21)	5 (15)	4 (12)	-	1 (3)	2 (6)	3 (9)	-	-	-	-	1	-	-
E. fecalis	6											4									2
S. mascescence	16	-	-	16 (100)	-	16 (100)	1 (6)	11 (69)	2 (13)	1 (6)	1 (6)	1 (6)	2 (13)	2 (13)	-	7 (44)	-	-	-	-	-
Total # of isolates	848																				

Table 2. Susceptibility and resistance patterns against antibiotics used in King Fahad Specialist Hospital, Buraidah, Al-Qassim, Saudi Arabia

	#isolate	Action	Antibiotics tested																			
Drugs / Bacteria			PEN	OXA	AMC	IPM	AMP	TZP	CXM	CTX	CAZ	FEP	AMK	GEN	CIP	SXT	TET	ERY	CLI	NIT	VAN	LND
Staphylococcus aureus	157	S		75	-	-	-	-	-	-	-	-	-	88	93	11	88	85	85	-	151	119
		R	151	81	-	-	-	-	-	-	-	-	-	40	35	12	47	41	32	-	2	1
		I	6	1										29	29	134	22	31	40		4	37
Staphylococcus epidermidis	90	S	0	18	-	-	-	-	-	-	-	-	-	25	32	5	45	19	36	-	89	84
		R	89	67	-	-	-	-	-	-	-	-	-	45	45	5	40	58	42	-	-	5
		I	1	5										20	13	80	5	13	12		1	1
Staphylococcus haemolyticus	22	S	-	3	-	-	-	-	-	-	-	-	-	6	7	2	16	2	11	-	22	21
		R	22	18	-	-	-	-	-	-	-	-	-	15	15	-	6	16	11	-	-	-
		I		1										1		20		4				1
Acinetobacter baumanni	128	S	-	-	-	108	-	6	-	-	2	10	23	52	6	32	52	-	-	-	-	-
		R	-	-	-	11	-	54	-	-	114	86	80	65	115	84	55	-	-	-	-	-
		I				9		68			12	32	25	11	7	12	21					
Pseudomonas aeruginosa	111	S	-	-	-	81	-	60	-	-	55	39	85	68	76	-	-	-	-	-	-	-
		R	-	-	-	23	1	21	-	-	45	33	11	30	29	2	-	-	-	-	-	-
		I				7	-	30			11	39	15	13	6	-						
Klebsiella pneumoniae	120	S	-	-	38	110	-	58	27	28	14	28	98	68	54	36	66	-	-	11	-	-
		R	-	-	46	5	119	27	86	80	50	60	11	40	39	76	29	-	-	-	-	-
		I			36	5	-	35	7	12	56	32	11		27	8	25			-		
E. coli	88	S	-	-	44	86	3	70	51	47	34	44	76	70	44	24	8	-	-	13	-	-
		R	-	-	18	-	85	3	34	32	14	23	1	15	25	62	49	-	-	-	-	-
		I			26	2		15	3	9	40	21	11	3	19	2	31			-		
Proteus mirabilis	67	S	-	-	37	61	16	61	21	24	14	27	52	27	36	15	2	-	-	-	-	-
		R	-	-	18	2	19	3	40	28	8	13	15	28	27	49	39	-	-	-	-	-
		I			12	4	32	3	6	15	45	27		12	4	3	26					
Enterobacter cloacae	33	S	-	-	3	18	-	14	10	12	5	18	13	16	14	18	18	-	-	1	-	-
		R	-	-	14	-	18	2	7	5	4	-	1	2	3	-	-	-	-	1	-	-
		I			16	-	15	17	16		-	15	19	15	16	-	-			-		
Enterobacter fecalis	16	S											12								16	14
		I											4									2
Serratia mascescence	16	S	-	-	-	16	-	12	3	11	3	9	11	14	13	16	2	-	-	-	-	-
		R	-	-	16	-	16	1	11	2	1	1	1	2	2	-	7	-	-	-	-	-
Total	848	I						3	2	3	-	6	4		1		-					

Surveillance of antimicrobial resistance among clinical isolates