Antibiotic Susceptibility Pattern of *Acinetobacter* Species Isolated in Clinical Specimens from the University College Hospital, Ibadan, Nigeria

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Summary

INTRODUCTION

***Acinetobacter* has emerged as important pathogen in hospital associated infections (HAI) with increasing antimicrobial resistance ability, morbidity and mortality. Because the antibiotic susceptibility of *Acinetobacter* species differs significantly among countries and even units of same hospital, local surveillance for resistance pattern is important. We determined the antibiotic susceptibility pattern of clinical isolates of *Acinetobacter* species.**

MATERIALS AND METHODS

***Acinetobacter* isolates from different clinical specimens between January and December 2016 were identified using Microbact 20E and subjected to antibiotic susceptibility using disc diffusion method. The level of drug resistance was categorised accordingly.**

RESULTS

Thirty-seven isolates among infected patients with mean age 35.63 years ± 22.78, male: female 1.5:1. *Acinetobacter baumannii* caused 26 (70.3%) of the infections, especially among surgical patients. Fifteen (40.5%) from blood and nine (24.3%) from wound biopsy (swab). Susceptibility of *A. baumannii* to Meropenem and Levofloxacin was 61.5%, and 69.2% respectively but the susceptibility of *Acinetobacter haemolyticus* and *Acinetobacter iwoffii was* 100% to Ampicillin-sulbactam, Quinolones, Meropenem, and Piperacillin/Tazobactam, and 88.9%-

100.0% to Aminoglycosides. Ten (27.0%) and 5 (13.5%) *A. baumanni* identified as MDR and XDR respectively.

CONCLUSION

MDR and XDR Acinetobacter isolates are present in University College Hospital (UCH). Infection control practices should be strengthened to prevent further spread of resistant strains.

Keywords*: Antimicrobial Resistance, Antibiotic Susceptibility Patterns, Acinetobacter, MDR, XDR,*

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Introduction

Acinetobacter baumannii is an aerobic, pleomorphic and non-motile, gram-negative bacillus that has emerged as an opportunistic pathogen that can lead to serious hospital associated infections (HAIs), with high incidence among immune-compromised individuals; particularly those who have experienced a prolonged hospital stay or in critical care units [1, 2]. Its pathogenic potential includes the ability to adhere to surfaces, formation of biofilms, antimicrobial resistance and ability to acquire genetic material from unrelated genera, thus making it a versatile and difficult pathogen to control and eliminate [3, 4, 5].

The role of environmental contamination in the transmission of HAI in general and in *A. baumannii* infections in particular is well recognized [6, 7]. *Acinetobacter* species do not have fastidious growth requirements and this explains their ability to persist in either moist or dry conditions in the hospital environment, thereby contributing to transmission [7]. *Acinetobacter baumannii* is the most clinically significant species, isolated and implicated in both community-acquired and HA infections especially in critical care facilities [8]. This organism specifically targets moist tissues such as mucous membranes or areas of the skin that are exposed, either through accident or injury and easily lead to necrotizing process, followed by bacteraemia and if left untreated, can lead to septicaemia and death, although other organisms may contribute to the outcome as they may enhance spread to the blood stream [9, 10].

Other notable sites of infection or colonization include the respiratory tract, blood, pleural fluid, urinary tract, surgical wounds, the central nervous system, skin and eyes [11]. The initial infection is reported to rapidly spread to the blood stream by which it is associated with high mortality ranging between 40% and 60% [12].

In the respiratory tract, patients may acquire ventilator associated pneumonia (VAP) therefore constituting a threat to patients who require mechanical ventilation, because *A. baumannii* has the ability to form biofilms on the surface of endotracheal tubes; this most likely accounts for the relatively high levels of colonization in the lower part of the respiratory tract [13]. It has also been shown to form biofilms on abiotic surfaces, such as glass and equipment used in intensive care units, and/or on biotic surfaces such as epithelial cells [4, 14]. Patients that utilize artificial devices such as catheters, sutures, ventilators and those who have undergone dialysis or antimicrobial therapy

within a period of 90 days are also found to be at risk of developing *A. baumannii* infections [8]. However, community associated pneumonia caused by *Acinetobacter* was reported in Australia and Asia in about 10% of community residents with excessive alcohol consumption [3].

Acinetobacter bloodstream infection was reported as having the third highest crude mortality rate in the intensive care unit (ICU), exceeded only by *Pseudomonas aeruginosa* and *Candida* spp infections [15]. Other sites where *Acinetobacter* has been well documented include burn units, neuro-surgical units [16, 17] with the mortality rate as high as 70%; although there may be other contributory factors. *Acinetobacter* accounted for 1.3% of all monomicrobial bloodstream infections in a seven-year review in United States especially in ICU-acquired blood stream infection [15].

Acinetobacter baumannii was first isolated from the soil by Beijerinck, a Dutch microbiologist in 1911 with a different name but the genus *Acinetobacter* was widely accepted following the study by Baumann and colleagues in 1968 [18]. The genus *Acinetobacter*, as currently defined, comprises gram-negative, strictly aerobic, non-fermenting, non-fastidious, non-motile, catalase-positive, and oxidase-negative bacteria with 26 named species and nine genomic species [19].

In the 1970s *A. baumannii* was thought to have been susceptible to most antibiotics, but currently, this organism appears to be resistant to most first-line antibiotics [20]. However, it has become one of the most common and serious multi-drug resistant (MDR) pathogens which are given the acronym **"**ESKAPE,**"** representing *Enterococcus faecium, Staphylococcus aureus, Klebsiella pneumoniae, Acinetobacter baumannii, Pseudomonas aeruginosa* and *Enterobacter* spp. [21]. The most important mechanism of resistance is the overexpression of AmpC cephalosporinase and resistance to extended spectrum cephalosporin, which is intrinsically linked to the presence of ISAba1 [22]. Cefepime and carbapenems, however, appear to be stable in response to these enzymes [23]. Antimicrobial resistance has been recognized as one of the three most important problems facing human health [24].

A. baumannii also possess an intrinsic class D oxacillinase belonging to the OXA-51-like group of enzymes that constitutes over 40 sequence variants as reported by Alsultan [25]. The ubiquitous nature of OXA-51-like genes in *A. baumannii* has led to this gene becoming an important genetic marker in identification of the organism to the species level as the enzymes are able to hydrolyze penicillins (benzylpenicillin, ampicillin, ticarcillin and piperacillin) and could also weakly hydrolyse carbapenems (imipenem and meropenem). The blaOXA-23, blaOXA-40 and blaOXA-58-like lineage genes encode the production of oxacillinases that is most common enzymatic mode of carbapenem resistance [22].

Outbreaks of carbapenem-resistant *A. baumannii* were observed and reported in a hospital in New York City as far back as 1991 and 1992 [26]. This observation was made during the time imipenem was used to manage an outbreak of infections due to ESBL-producing *Klebsiella pneumoniae*. A more recent survey of 76 centers in the United States showed that only 60.2% of *A. baumannii* were susceptible to imipenem [27]. Griffith, *et al*. in 2006 and Petersen *et al* 2011 in separate studies suggest that multidrug resistant *A. baumannii* is not ubiquitous but likely acquired nosocomially by providing evidences from studies in which strains isolated from the skin of patients entering the hospital were found to be different from those isolated from clinical specimens of patients with established infection from *A.*

baumannii [27, 28]. Infections caused by *A. baumannii* and other *Acinetobacter* species are becoming better recognized in Nigerian hospitals with improved laboratory methods of diagnosis, and also in other parts of Africa with prevalence ranging from 8.5% to 14% [29, 30, 31]

The public health importance of multidrug resistant organisms cannot be overlooked in low resource settings like Nigeria, with poor health funding and weak health systems. The struggle with multidrug resistant organisms will be an additional huge burden on already weak health systems.

The optimal treatment for *A. baumannii*, especially HAIs resulting from multiple resistant strains, is yet to be established, coupled with the fact that the resistance pattern is not uniform across institutions. It is therefore a clinical necessity to put in place well designed procedures or protocol to help guide clinicians on decisions regarding the current best therapeutic practice. An assessment of specimen sources from which *Acinetobacter* species can be isolated is necessary to understand the spectrum of diseases in which it is implicated in our setting and also determine the antibiotic susceptibility profiles of recovered isolates of *Acinetobacter* to serve as a guide in the choice of empirical antibiotics, hence the reasons for this study.

Materials and Methods

All *Acinetobacter* isolates recovered from different clinical specimens sent for microbiological analysis between January and December 2016 at the University College Hospital (UCH), Ibadan were included for further analysis. The hospital is an 800-bed capacity teaching hospital, offering tertiary level of health care.

The choice of culture media for the isolation was based on standard operating procedure (SOP) depending on the type of specimen sent, while initial biochemical analysis used the conventional methods of gram reaction, indole, citrate, urease and oxidase tests. The suspected *Acinetobacter* isolates were identified to species level using Microbact (OXOID Microbact™ identification kits Microbact™ GNB 24E, Oxoid Ltd Wade Road, Basingstoke, Hants, RG24 8PW, UK) [32] and subsequently confirmed with Vitek 2.

Antimicrobial susceptibility testing of the isolates was performed by the disk agar diffusion method following the recommendation by Clinical Laboratory and Standards Institute CLSI [33]

The level of drug resistance was categorised, according to the joint committee of European centre for disease control (ECDC) and Centre for disease control and prevention of the US (CDC), as multi-drug resistance (MDR), extensively drug resistance (XDR) and Pan-drug resistance (PDR) and the various categories defined accordingly to allow uniformity of definition. MDR was defined as acquired non-susceptibility to at least one agent in three or more antimicrobial categories, XDR was defined as non-susceptibility to at least one agent in all but two or fewer antimicrobial categories (i.e. bacterial isolates remain susceptible to only one or two categories) and PDR was defined as non-susceptibility to all agents in all antimicrobial categories [7, 34].

Data was analysed and presented by proportions and the isolates were also analysed along the units of the hospital where they were isolated from, the socio-demography of patients and sites of infections.

Results

During the study period, a total of 87 *Acinetobacter* species were isolated conventionally, 43 were confirmed with Microbact but only 37 were confirmed by Vitek 2 and referred for further analysis.

A. baumannii was the main species responsible for 26 (70.3%) of the infections, followed by *A. haemolyticus* ten (27.0%) and the remaining one (2.7%) was caused by *A. iwoffii*. The mean age of patients infected with *Acinetobacter* spp. was 35.6 years (standard deviation ± 22.8, range 1-73 years). The gender (male: female) ratio was 1.5:1(Table I presented at the end of the article summarizes these findings). Also, majority of the *Acinetobacter* spp. were isolated from blood samples (15, 40.5%), and wound biopsy/swab samples (9, 24.3%), other sources include sputum (5, 13.5%), urine (2, 5.4%) while others (6, 16.2%) were recovered from bronchial washings, tracheal and pleural aspirates (Table II).

Considering the hospital site distribution of all *Acinetobacter* isolates, *A. baumannii* was found to be the most common isolates among patients in all the hospital units with highest prevalence of 30.8% each found in surgical and sedical units. Multiple species were observed in all the units except in neurology ward where only *A. baumannii* was isolated while *A. baumannii* was isolated from all the wards, except from the neonatal ward. *A. haemolyticus* were found in only 7 of the 19 hospital units where positive samples were received, while *A. iwoffii* was only isolated from the medical outpatient unit (MOP) and from sputum specimen. All the three *Acinetobacter* species (13.5%) occurred in the medical outpatient unit (MOP) and both *Acinetobacter baumannii* (5.4%) and *A. haemolyticus* (8.1 %,) were found in the Geriatric ward. However, majority (88.8%) of the *Acinetobacter* in the paediatric units is of the *haemolyticus* species (Figure 1 presented at the end of the article).

The antimicrobial susceptibility profiles of the pathogens and the variation across the various specimens are shown in Table III and figure 2. Of the specimens that tested positive for *Acinetobacter baumannii*, 61.5%, and 69.2% were susceptible to Meropenem and Levofloxacin respectively, while 76.5% and 53.8% are resistant to Imipenem and Ciprofloxacin respectively. Susceptibility rates were high for aminoglycosides (75.0%–80.9%) and Ampicillin+Sulbactam (81.8%). Majority of *Acinetobacter haemolyticus* were 100% susceptible to Quinolones, Ampicillin+Sulbactam, Meropenem and Piperacillin+Tazobactam but 88.9%-100.0% susceptible to aminoglycosides. Additionally, high and equal susceptibility rates (100.0%) for Aminoglycosides, Ceftriaxone, and Ciprofloxacin by *Acinetobacter iwoffii* were also observed in this study.

Considering the level of drug resistance among the isolates, 40.5% of all the isolates were resistant to the various antibiotics, but only *Acinetobacter baumannii* exhibited multidrug resistance characteristics with ten (27.0%) and five (13.5%) identified as MDR and XDR respectively, although none was PDR. In addition, multidrug resistant isolates were most commonly obtained from wound biopsy/wound swab four (40.0%), while XDR isolates were commonly obtained from both blood and wound biopsy specimen ten (40.0%). Tables 4 and 5 show the distribution of MDR among specimens and across the major units in the hospital.

Discussion

Isolation of *A. baumannii* in a hospital environment, pose a significant risk, particularly in ICU and critical care wards where patients are chronically ill and mostly immune-compromised, with prolonged hospital stay. Thus group represent a high-risk group for *A. baumannii* infection. Over 70% of the isolates from this study were *A. baumannii* with majority being isolated from blood stream and wound biopsy. Patients from the ICU had isolates from both blood stream and tracheal aspirates and bronchial washings more commonly. This is in agreement with previous documentations [2, 8].

Pathogenicity linked to Biofilm formation has been well documented [5, 6, 14]. Although we did not test for biofilm-forming strains, isolates from tracheal aspirates and patients with urinary catheters are likely to be of such strains and also judging by the level of exposure to aminoglycosides which almost always accompany antibiotic combination therapy in our center. Although, Rodrıguez-Bano *et al*, [14] in 2008 found that the pathogenicity of non-biofilm forming strains was not significantly different from biofilm forming strains, the biofilm-forming isolates are also less frequently resistant to Imipenem and Ciprofloxacin. The levels of resistance to Imipenem and Ciprofloxacin among *A. baumannii* in our study also support the assumption that biofilm-forming strains are most likely responsible for those infections.

Outbreaks in hospitals have been linked to health care professionals with colonized hands and poor personal hygiene who act as opportunist carriers of an epidemic strain. Other contributory factors to outbreak are contaminated ventilators or respiratory care equipment [13]. All the isolates from the ICU are *A. baumanni* and are from blood, pleural fluid and tracheal aspirate and exhibited highest resistance. Lack of mutations conveying fluoroquinolone resistance in genes, parC and gyrA in resistant *Acinetobacter baumannii* was used to explain the low resistance to fluoroquinolones like Ciprofloxacin [35]. However, the level of resistance to fluoroquinolones in the current study ranged from 30.8-53.8%, and judging by the frequency of use of fluoroquinolones in the UCH, it is most likely that there may be mutations in other genes other than parC and gyrA, that confer resistance to fluoroquinolones or possibly there may be other mechanisms of resistance to fluoroquinolones.

Tayabali *et al* [35] also demonstrated that *A. baumanni* was associated with reduced capacity of bacterial elimination from the host; this may partially explain why it is more virulent in immune-compromised individuals and isolated more commonly from surgical wounds, burn patients and ICU patients in this study.

A study in Sudan, reported high levels of resistance to several antibiotics with most of *A. baumannii* isolates where 92% were resistant to Cefepime, 96% to Ceftazidime, 99% to Ceftriaxone, 100% to Cefuroxime, 100% to Cephalexin, 92% to Gentamicin, 81% to Amikacin, 91% to Ciprofloxacin, 98% to Amoxiclav, 89% to Meropenem, 95% to Aztreonam and 37% to Colistin [36].

In a study by Odewale *et al* [31] in another hospital in the same state as ours, there was high resistance to carbapenems, aminoglycosides, quinolones and cephalosporins and the only antibiotic to which *A. baumannii* was 100% susceptible was Colistin. This is quite different from our findings and further confirms that susceptibility pattern cannot be generalized even in a region.

Another study in India by Sheth and colleagues in 2012 [37] reported susceptibility

among *Acinetobacter baumanni* where isolates were still highly susceptible to carbapenems, aminoglycosides and quinolones but poorly susceptible to the third generation cephalosporins and ampicillin/sulbactam.

The *A. baumannii* in our study showed appreciable susceptibility to ampicillin-sulbactam. The Indian study is comparable to the findings of our study except that the level of resistance to cephalosporins, fluoroquinolones and carbapenems observed in the current study has practically rendered them ineffective and therefore not recommended for empirical use.

Although, Colistin was not tested in our study, being the only antibiotic with appreciable susceptibility levels, as reported from other studies, is an indication that there is a limited choice of empirical antibiotics when multidrug resistant *A. baumanni* is isolated. Our study observed variability in susceptibility profiles across the sources of infections when we examined all the *Acinetobacter* species. Aminoglycosides susceptibility rates range from 50.0% to 86.7% across all the specimens collected, susceptibility rate of 22.2% to 100% to quinolones, while 60% was the highest susceptibility rate to cephalosporins among *Acinetobacter* species from most specimens (Figure 2).

In an industry-supported surveillance report (MYSTIC) from 48 European hospitals for the period 2002– 2004, just 73.1% of *A. baumannii* isolates were susceptible to Meropenem and 69.8% were susceptible to Imipenem and susceptibility to other antibiotics was also very low, with 32.4%, 34.0% and 47.6% being susceptible to Ceftazidime, Ciprofloxacin and Gentamicin, respectively [38]. Considering the time interval of the above study to the current study, it is obvious that there is worsening of the resistance pattern. The current study observed variability in susceptibility profiles across the sources of infections when we examined all the *Acinetobacter* species while higher susceptibility to aminoglycosides is still maintained (Figure 2). Overall, susceptibility rates for Aminoglycosides ranged from 50.0% to 100%, Meropenem from 50.0% to 100% and Ampicillin+Sulbactam, commonly employed in the paediatric units were from 80% to 100%. Sulbactam, among the beta-lactamase inhibitors, has been reported to possess the greatest intrinsic bactericidal activity against *A. baumannii* isolates, with about 90% of seriously ill patients on mechanical ventilation demonstrating clinical improvement [37, 39]. In fact a study identified treatment with ampicillin-sulbactam as the only statistically significant variable associated with reduced mortality in patients with MDR *A. baumannii* blood stream infection [40]. Thus the finding in our study, which is coming over a decade after, is corroborating this earlier finding. However, for other species of *Acinetobacter*, higher susceptibility rates of 86.7% and 100% to Gentamicin were observed among blood and sputum isolates respectively, 100% susceptibility rate from sputum and wound swab/biopsy to Amikacin and susceptibility rate of 100% to Meropenem was observed from respiratory tract isolates while an equal susceptibility rate (100%) of *Acinetobacter* isolates obtained from all sources except blood was seen with Ampicillin+Sulbactam. Thus, implying that they are more unlikely to be responsible for multidrug resistance infection. It is therefore important to determine the actual species responsible for infection before selecting the most appropriate antibiotics for treatment.

In the literature, various terms have been used to describe the extent of resistance of *A. baumannii* to antibiotics. Multidrug Resistant *A. baumannii* (MDR-AB) is used to describe the

isolates which are resistant to at least three classes of antibiotics including penicillins, cephalosporins, fluroquinolones and aminoglycosides, while the term Extreme Drug Resistant (XDR) is used when the isolates are resistant to the three above mentioned families plus carbapenems. Finally Pandrug Resistant (PDR), which is used to describe the *A. baumannii*, which are XDR with resistance to polymyxins. Employing the same definitions we found ten (27.0%) and five (13.5%) *A. baumanni* identified as MDR and XDR respectively, with four (40%) each of the MDR being from blood and wound and majority of XDR being from blood, wound and urinary tract isolates.

The significance of multidrug resistance *Acinetobacter* infection is the difficulty faced in managing such cases and the tendency to cause clonal dissemination and high mortality [39]. The use of combination therapy such as Carbapenem-Sulbactam, and Colistin-Rifampin, were suggested, however, there is no categorical recommendation of which combination is preferred [39, 40], while, mortality is considerably higher in MDR cases [2, 40].

Limitations

The findings of the resistance pattern from one study cannot be generalized as the antibiotic disc susceptibility test results vary from one hospital to another depending on the hospital environment, antibiotic use or policy, and infection control practices.

We could not determine the genetic basis of the resistance observed among the isolates due to non-availability of molecular diagnostic facility.

Conclusion

Three different species of *Acinetobacter* were found in hospital associated infections (HAI), *A. haemolyticus* with better susceptibility is more common in paediatric units while the most common was *A. baumanni*, which was found in all major units of the hospital and showed both MDR and XDR patterns.

Recommendations

Adequate control measures in terms of infection prevention practices especially hand washing should be strengthened to prevent further spread of the resistant strains. We will also recommend that antibiotic policy should be strictly adhered to so as to prevent a situation of PDR with attendant grave consequences.

Clinical Microbiology Laboratory should also be actively involved in providing surveillance for MDR *A. baumannii*, especially for patients colonized with multidrug (MDR)- or extensively drug-resistant (XDR) *A. baumannii* for infection control purposes.

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Appendix

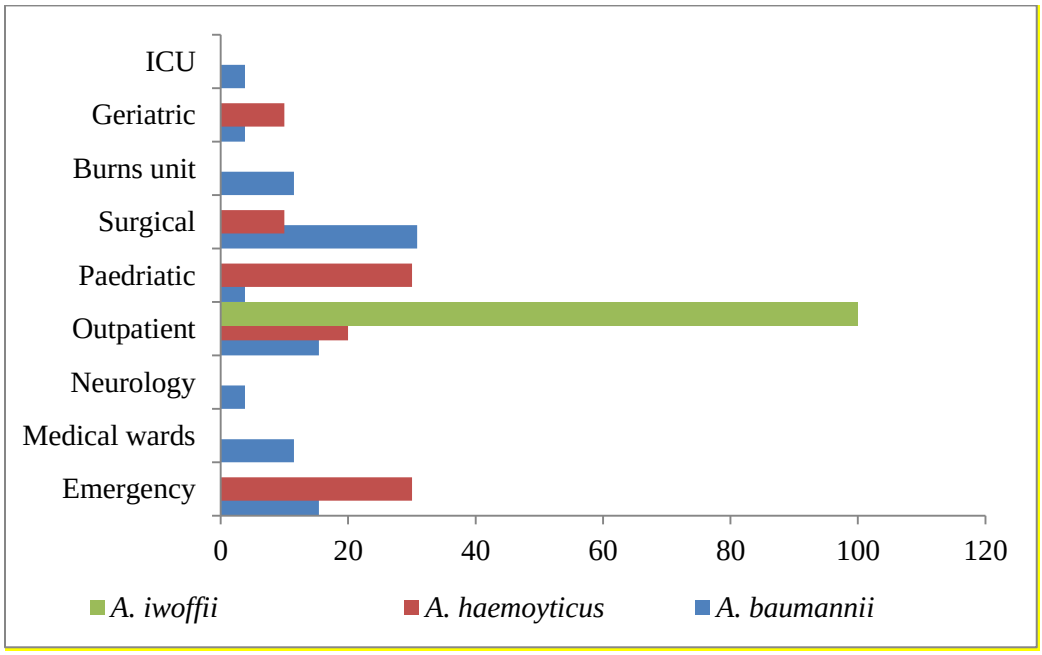
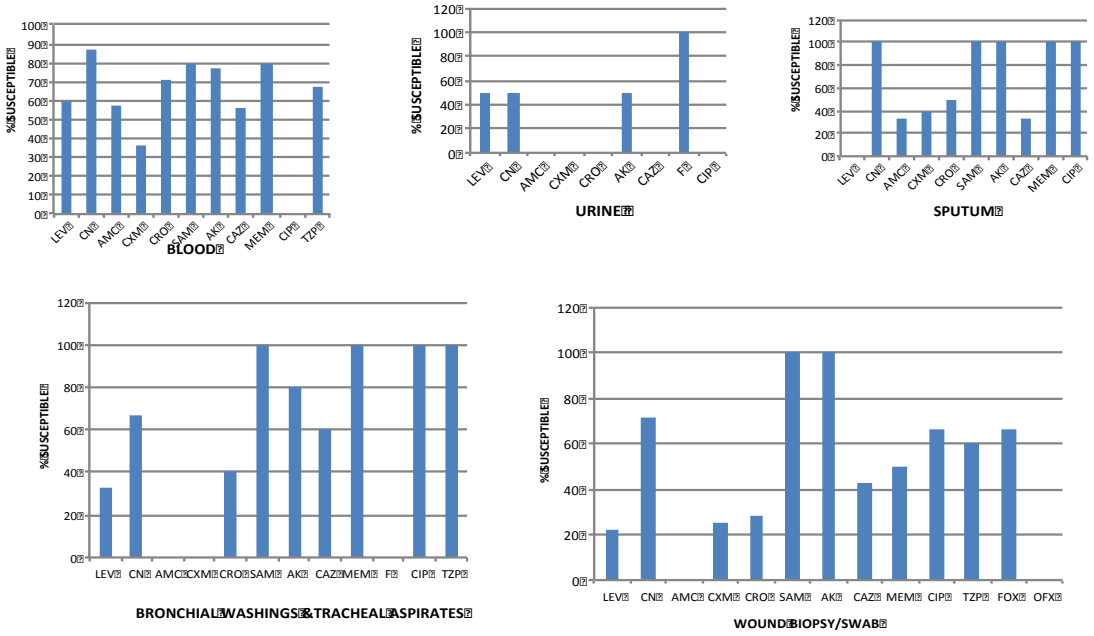


Figure 1: Distribution of Acinetobacter Species According to Major Hospital Units



KEY: LEV-Levofloxacin, CN-Gentamicin, CRO- Ceftriaxone, SAM- Ampicillin+sulbactam, AMC-Amoxicillin, AK- Amikacin, CAZ- Ceftazidime, MEM- Meropenem, F- Nitrofurantoin, CIP- Ciprofloxacin, TZP- Ampicillin+Tazobactam, FOX- Cefoxitin, CXM- Cefuroxime, OFX-Ofloxacin

Figure 2: Variation in Antibiotic Susceptibility of Acinetobacter across 5 Different Sources

Table 1: Prevalence of Resistance in Relation to Age and Sex (N = 37)

Variable	Number examined	Number resistant (%)	P value
Age			
<35	17	9(52.9)	p = 0.157 χ2=2.006
>35	20	6(30.0)	
Sex			
Male	22	6 (60.0)	p = 0.47 χ2=3.963
Female	15	9 (27.3)	
Total	37	15 (40.5)	

Table 2: Distribution of Acinetobacter Spp. Based on Various Clinical Samples (N=37)

Acinetobacter spp	Types of sample					
	Blood	Wound biopsy/swab	Sputum	Urine	Others*	Total
A. baumannii	08	08	02	02	06	26(70.3)
A. haemolyticus	07	01	02	-	-	10(27.0)
A. iwoffii	-	-	01	-	-	1(2.7)
Total	15 (40.5)	9 (24.3)	5(13.5)	2(5.4)	6(16.2)	37(100.0)

Others (*Peritoneal aspirate, Tracheal aspirate and Bronchial washing)

Table 3: Susceptibility Pattern of Acinetobacter Species from UCH, Ibadan

Antibiotics	*A. baumanni* N=26		*A. haemolyticus* N=10		*A. iwofii* N=1	
	Tested	Susceptible N (%)	Tested	Susceptible N (%)	Tested	Susceptible N (%)
Aminoglycosides						
Gentamicin	24	18 (75.0)	9	8 (88.9)	1	1 (100.0)
Amikacin	21	17 (80.9)	8	7 (87.5)	1	1 (100.0)
Cephalosporins						
Cefuroxime	13	2 (15.4)	8	5 (62.5)	1	0 (0.0)
Cefoxitin (FOX)	2	1 (50.0)	1	1 (100.0)	NT	-
Ceftriaxone	15	4 (26.7)	8	6 (75.0)	1	1 (100.0)
Ceftazidime	17	8 (47.1)	8	4 (50.0)	NT	-
Penicillin/Pen combinations						
Amoxicillin	14	3 (21.4)	2	2 (100.0)	1	0 (0.0)
Ampicillin /Sulbactam	11	9 (81.8)	5	5 (100.0)	NT	-
Piperacillin /Tazobactam	6	3 (50.0)	3	3 (100.0)	NT	-
Quinolones						
Ciprofloxacin	13	6 (46.2)	3	3 (100.0)	1	1 (100.0)
Ofloxacin	1	0 (0.0)	NT	-	NT	-
Levofloxacin	13	9 (69.2)	5	5 (100.0)	NT	-
Carbapenems						
Meropenem	13	8 (61.5)	4	4 (100.0)	NT	-
Imipenem	17	4 (23.5)	8	7 (87.5)	1	1 (100.0)

NT= Not Tested

Table 4: The occurrence of MDR, XDR, PDR and S of Acinetobacter **Species** *in Different Clinical Samples*

Sample	MDR	XDR	S	PDR
Blood	1(10.0)	2 (40.0)	12 (54.5)	0 (0.0)
Wound biopsy/swab	4(40.0)	2(40.0)	3 (13.6)	0 (0.0)
Urine	1(10.0)	1(10.0)	0 (0.0)	0 (0.0)
Sputum	1(10.0)	0(0.0)	4 (18.2)	0 (0.0)
Others*	3(30.0)	(0.0)	3 (13.6)	0 (0.0)
Total	10 (27.0)	5 (13.5)	22 (59.5)	0(0.0)

KEY: MDR: Multidrug resistant, XDR: Extensively drug resistant, PDR: Pan-drug resistant, S: Susceptible, Others* (Peritoneal aspirate, Tracheal aspirate and Bronchial washing)

Table 5: The occurrence of MDR, XDR, PDR and S of Acinetobacter **Species** *in Different Hospital Units*

Departments	MDR	XDR	S	PDR
Burns unit	3(100.0)	0 (0.0)	0(0.0)	0(0.0)
Emergency unit	2(28.6)	1(14.3)	4(57.1)	0(0.0)
Geriatric	1(0.0)	1(50.0)	0(50.0)	0(0.0)
ICU	1(100.0)	0(0.0)	0(0.0)	0(0.0)
Medical ward	0(0.0)	0(0.0)	3(100.0)	0(0.0)
Neurology	0(0.0)	0(0.0)	1(100.0)	0(0.0)
Outpatients	2(28.6)	0(0.0)	5(71.4)	0(0.0)
Paediatric	1(25.0)	0(0.0)	3(75.0)	0(0.0)
Surgical	1(11.1)	3 (33.3)	5(55.6)	0(0.0)