

Prevalence of *Acinetobacter baumannii* in Diyala governorate from Different Clinical Source

Abstract

Acinetobacter baumannii bacteria from various clinical sources (Blood, urine, CSF, Ear swab, burn swab, wound swab, sputum and vaginal swab) were isolated from patients attending to Baquba Teaching Hospital and Al-Betool Teaching Hospital. A total of (27) isolates of *A. baumannii* bacteria out of (200) samples were isolated from various clinical sources and the maximum isolates were obtained from blood samples 10(37.03%), urine samples 4(14.8%), CSF samples 4 (14.81%), burn swab (14.81%), sputum swab 2(7.4%), wound swab 1(3.7) , Ear swab 1(3.7) and vaginal swab 1(3.7). All isolates were tested for their resistance to (23) different antibiotics and the results showed that resistant to Amoxicillin, Amoxicillin/Clavulanic acid and Trimethoprim were (96.2%) but lesser to Ticarcillin, Ticarcillin / Clavulanic Acid, Piperacillin/Tazobactam, Ceftriaxone, Imipenem, Ciprofloxacin and Levofloxacin were 88.8%.

Keywords: *Acinetobacter baumannii*, AST and Vitek 2 system.

Introduction

Acinetobacter baumannii is a Gram-negative bacterium, aerobic and non-motile rod. This opportunistic bacterium has emerged as a significant concern in human health, responsible for a variety of infections, including pneumonia, meningitis, septicemia, and urinary tract infections. It possesses several virulence factors, such as the ability to form biofilms, adhere to surfaces, invade host cells, acquire iron, and induce host cell death [1]. *A. baumannii* easily attaches to both biological and non-biological surfaces, where it can form biofilms. This characteristic is significant for many pathogens as it aids in the colonization of prosthetic materials and plays a role in drug resistance and evasion of the host immune system in vivo [2]. *A. baumannii* has developed resistance to many of the most effective antimicrobial agents, leading to a significant increase in morbidity and mortality rates, particularly in intensive care units across various countries [3]. *Acinetobacter baumannii* is a significant multidrug-resistant (MDR) opportunistic pathogen commonly associated with nosocomial infections, largely due to its remarkable ability to develop resistance to various groups of antibiotics. Multiple mechanisms contribute to the acquisition of this multidrug resistance [4]. Resistance in *Acinetobacter baumannii* strains exhibiting the MDR phenotype is attributed to a diverse array of genes responsible for antibiotic resistance, encompassing both intrinsic and acquired mechanisms [5]. There are three mechanisms of antibiotic resistance in *A. baumannii*: First, resistance can arise by reducing membrane permeability or increasing antibiotic efflux, thereby preventing access to the target. Additionally, bacteria may employ genetic changes or post-translational modifications to protect the drug target. Finally, antibiotics can be directly inactivated through hydrolysis or modification processes [6]. This pathogen has developed multidrug resistance (MDR) in recent years, mainly due to the widespread overuse of antibiotics and inadequate antibiotic management. Prolonged hospital stays, use of catheters, and mechanical ventilation are associated with MDR isolates, and invasive infections are more likely to occur in immunocompromised individuals and those who are critically ill [7].

Materials and Methods

In the current study, 27 *A. baumannii* isolates were employed. Samples were gathered between January and July of 2024. Blood, urine, CSF, burns, wounds, blood cultures, and sputum were among the items that were taken from four Baghdad hospitals. All obtained samples, with the exception of blood, were grown immediately on blood agar and MacConkey agar and incubated for twenty-four hours at 37°C. Blood samples are cultured immediately on blood agar and MacConkey agar and then incubated for further 24 hours at 37°C. The blood specimen must be placed in blood culture bottles that include nutrients that encourage the growth of aerobes bacteria. All bacterial isolates were tested by Vitek 2 system. Bacterial identification based on the biochemical reactions between the bacterial suspension and the culture media existed in the GN-ID Cards loaded manually into the VITEK system. According to the manufacturer's instructions (BioMerieux, France), the following steps are performed automatically in the software of the system. The susceptibility of *Acinetobacter baumannii* isolates against 22 antibiotics were determined by Vitek 2 system by using Antibiotics Susceptibility Gram-negative (AST-GN).

Results

In this study a total of (200) samples were obtained from from (Blood, urine, CSF, Ear swab, burn swab, wound swab, sputum and vaginal swab as shown in table (1).

Table (1): Distribution of samples and number of *Acinetobacter baumannii* isolates

<i>Acinetobacter baumannii</i> bacteria (27 isolated)	Type of specimen	Total specimen	Number of isolates	%
	blood	30	10	37.03
	Urine	55	4	14.81
	CSF	15	4	14.81
	Ear swab	5	1	3.7
	Brun swab	30	4	14.81
	Wound swab	45	1	3.7
	Sputum swab	15	2	7.4
	vaginal swab	5	1	3.7
Total	200	27	100	

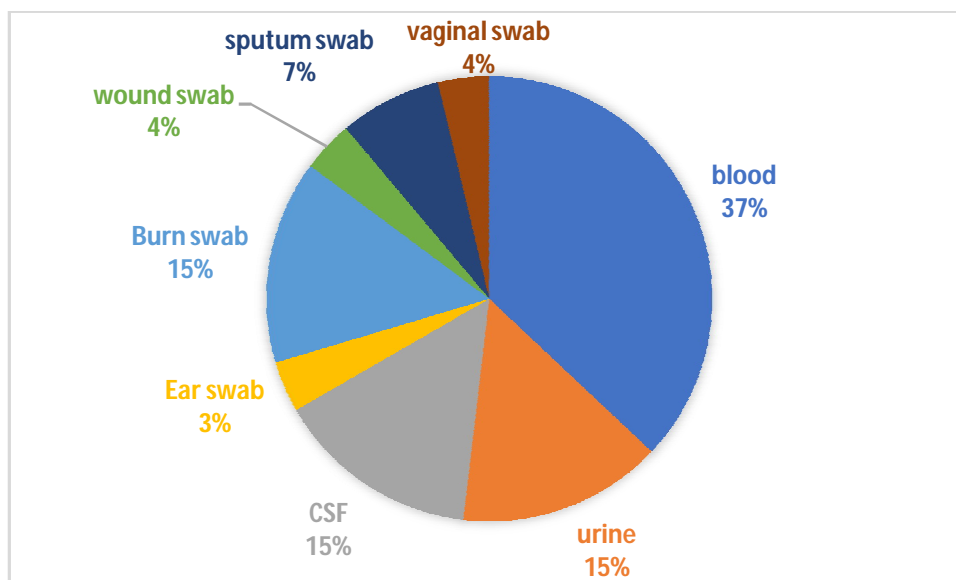


Figure (1): Acinetobacter baumannii isolate distribution

In this study most of *A. baumannii* isolates were highly resistant to the tested antibiotics as shown in table (2) and Figure (2).

Table (2): AST test on *A. baumannii* isolates

AB	<i>Acinetobacter baumannii</i> bacteria (27 isolated)	
	S(100%)	R(100%)
Amoxicillin	1(3.7)	26(96.2)
Amoxicillin/Clavulanic acid	1(3.7)	26(96.2)
Ticarcillin	3(11.1)	24(88.8)
Ticarcillin / Clavulanic Acid	3(11.1)	24(88.8)
Piperacillin	6(22.2)	19(70.3)
Piperacillin/Tazobactam	3(11.1)	24(88.8)
Cefuroxime	2(7.4)	25(92.5)
Cefixime	2(7.4)	25(92.5)
Cefotaxime	2(7.4)	25(92.5)
Ceftriaxone	3(11.1)	24(88.8)
Ceftazidime	2(7.4)	25(92.5)
Cefepime	4(14.8)	23(85.1)
Imipenem	4(14.8)	24(88.8)
Meropenem	4(14.8)	23(85.1)
Gentamicin	5(18.5)	22(81.4)
Ciprofloxacin	3(11.1)	24(88.8)
Levofloxacin	3(11.1)	24(88.8)

Tigecycline	11(40.7)	16(59.2)
Minocycline	9(33.3)	18(66.6)
Colistin	9(33.3)	18(66.6)
Tobramycin	6(22.2)	21(77.7)
Trimethoprim	1(3.7)	26(96.2)
Trimethoprim/Sulfamethoxazole	10(37.03)	17(62.9)

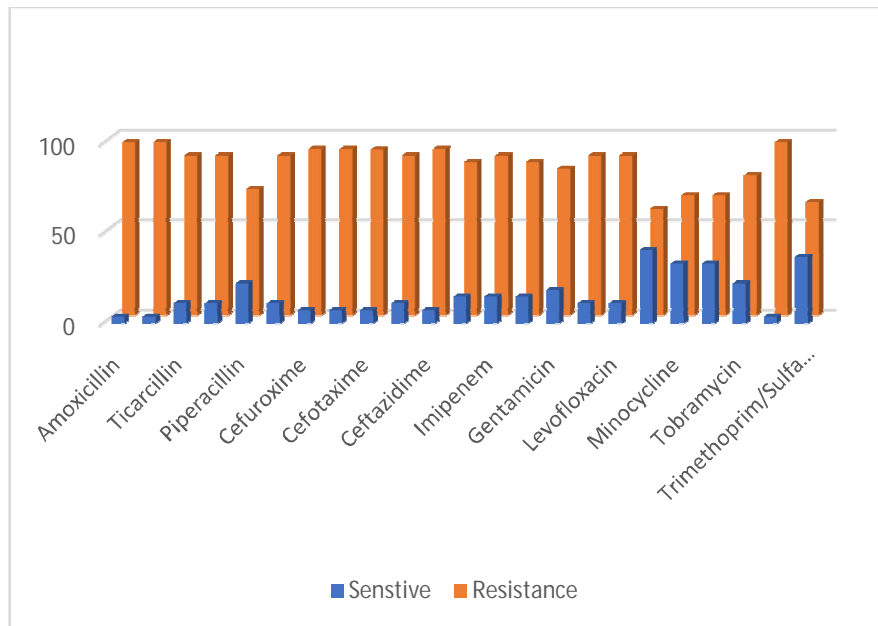


Figure (2):.AST test on *A. baumannii* isolates

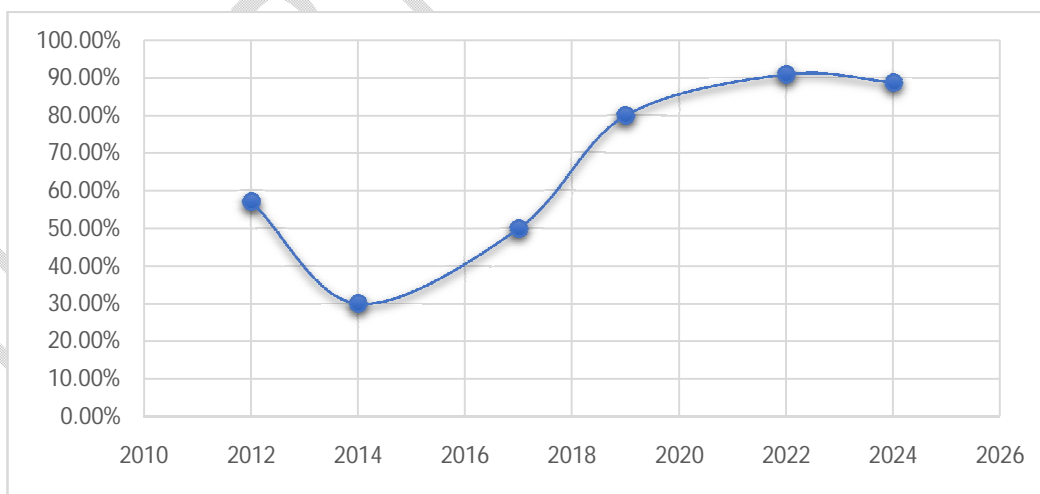


Figure (3): Increase Imipenem resistance of *A. baumannii* clinical isolates in Iraq

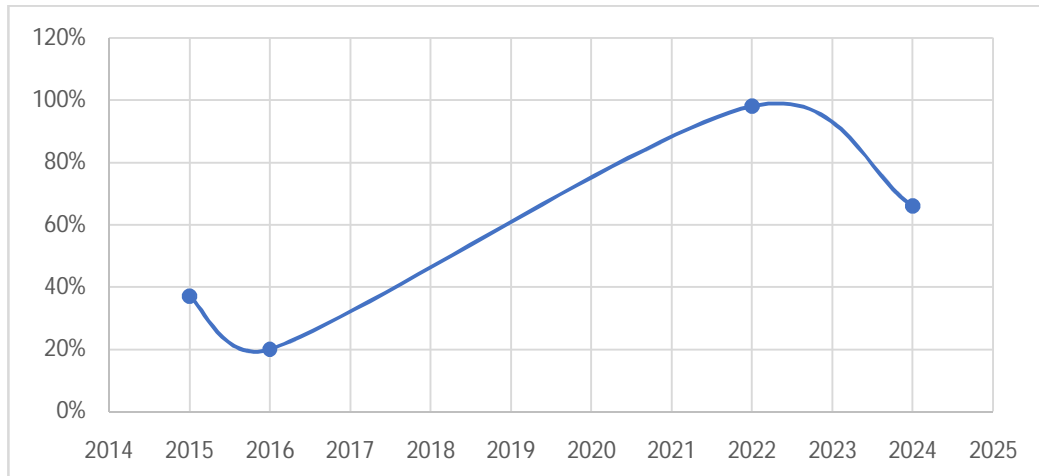


Figure (4): Increase Colistin resistance of *A. baumannii* clinical isolates in Iraq

Discussion

The results shown in table (1) appeared that from the total 27 isolates 10(37.03%) from blood, 4(14.81%) isolated from Urine, CSF, Brun ,2(7.4%) from sputum respectively. The lowest number and percentage of *A.baumannii* was Wound, Ear, vaginal swabs 1(3.7%). According to this study, blood is where this bacterium most commonly causes infections. The results of this study agree with previous study [8]and this result differed with what the researcher reached [9]. urine is often only detected when an indwelling urinary catheter is present. While it is infrequently linked to pyelonephritis and urosepsis, it is seldom invasive and typically just affects the lower urinary tract[10,11]. This result was compared to the result obtained by [12,13] and [14] have isolated *Acinetobacter baumannii* from urine rate reaching to (16,10% and 11.5%)respectively. The results of this study shows that *A. baumannii* bacteria can be isolated from burn and this result agreed withprevious study [15], who has isolated these bacteria from burn samples at rate of (17%).Around the world, burns are a serious public health problem and one of the most prevalent and destructive types of trauma[15,16]. The presence of an ample amount of oxygen in the respiratory tract, especially the lower part of it, encourages bacteria to settle and invade the region because it is a forced wind [17]. A bacteriological study stated that more than 60% of hospital-acquired pneumonia infections are caused by Negative bacteria including *Acinetobacter baumannii* the reason for this may be attributed to their ability to blind adhesion to epithelial cells in the respiratory tract as a result of inhibitory substances such as bacteriocins, was as the presence of mucous matter [18].

Because *A. baumannii* may form biofilms in the skin and cause infections in soft tissues, it is present in wounds and occlusive dressings [7]. Because many bacterial species have particular receptors for these chemicals, bacteria can readily colonize the surfaces of burn wounds.The reason for the difference in the isolation ratio is due to the number of samples taken the time of collection of the samples, the environment from which the samples were isolated, the health conditions in which the patients lived, and the length of time they stayed in Hospitalization, indiscriminate use of antibiotics excessively and the difference in the number of samples taken for a study and variance.Numerous dangerous nosocomial infections, including as urinary tract

infections, meningitis, wound infections, bloodstream infections, and ventilator-associated pneumonia, can be caused by *A. baumannii*. individuals in the high-risk groups include individuals who are mechanically ventilated, have indwelling foreign devices, are debilitating patients, and are hospitalized to intensive care units (ICUs) [19][20].

Resistance to broad-spectrum antimicrobial agents in *A. baumannii* is now an emerging issue globally. In the present study, Tigecycline was found the most effective antimicrobial against isolated *A. baumannii* strains, with a 40.7% sensitivity rate, followed by trimethoprim-sulfamethoxazole by 37.03%, Minocycline and Colistin with a 33.3% while showed a high resistance rate to Amoxicillin, Amoxicillin/Clavulanic acid and Trimethoprim by (96.2%), Cefuroxime, Cefixime, Cefotaxime and Ceftazidime by (92.5%), Ticarcillin, Ticarcillin / Clavulanic Acid, Piperacillin/Tazobactam, Ceftriaxone, Ciprofloxacin and Levofloxacin (88.8%), and Cefepime, Imipenem, Meropenem, Gentamicin, Piperacillin, Tobramycin which exhibited the low level of antimicrobials effectiveness against *A. baumannii* strains.

The results of the current study demonstrated that the highest resistance to almost all β -lactam antibiotic classes under study was as follows cephalosporins (Amoxicillin, Amoxicillin/Clavulanic acid, Cefuroxime, Cefixime, Cefotaxime, Ceftriaxone, Ceftazidime, Cefepime, Piperacillin, Oxacillin) These findings were close to local studies related to *A. baumannii* isolates by [21] [22], who found that (100%) of *A. baumannii* isolates in Iraqi hospital environment resisted Oxacillin, Ceftazidime and Cefepime. Another study in Iran hospital by [23] found (100% resistance rate to (Cefoxitin, Cefoxitin, Oxacillin, Ceftazidime, Cefepime). The three main types of resistance mechanisms are: (1) enzymes that deactivate antimicrobials; (2) bacterial targets are less accessible; or (3) mutations that alter targets or cellular activities. Regarding the first group, a variety of β -lactamases found in *Acinetobacter* species hydrolyze and provide resistance to cephalosporins, carbapenems, and penicillins. Chromosome-encoded AmpC cephalosporinases provide resistance to broad-spectrum cephalosporins [24].

As the isolates of bacteria *A. baumannii* resistance to Gentamicin in the percentage (81.4%). The results are consistent with [25], which was reached by the resistance rate (62%). While the resistance to isolates of the antibiotic Imipenem (88.8%), Meropenem (85.1%). This close with the results of the current study, which gave a 100% resistance to this antibiotic.

The results of Ciprofloxacin and Levofloxacin are agreed with [26] research, which was reached by the resistance rate (85%), and also agreed with [27] research. The resistance rate was (80%), Another local study revealed that *A. baumannii* clinical isolates were 100% resistant to Ciprofloxacin [28]. resistance of *A. baumannii* for Tobramycin, were (77.7%) consistent with the study of [29]. that showed 100% resistance for Tobramycin.

Because of its low rates of resistance, tigecycline could be the treatment of choice for managing infections at this facility. Although The results of resistance to trimethoprim /sulphamethoxazole also had a low resistance rate in our present study, were close to the results reached by the researcher in the research [27] and also close to the results of the research of [30] who recorded that the *Acinetobacter baumannii* resist to a wide range of antibiotic including trimethoprim / sulphamethoxazole in the percentage (81%). fewer patients were treated by this antibiotic, as its side effects of nephrotoxicity and neurotoxicity [31].

These bacteria are resistant to nearly all antibiotics for a variety of reasons. Among these, the most significant one is the existence of efflux pump families [32]. There are several families of efflux pumps in *Acinetobacter baumannii*, but the most significant family is the existence of Resistance. These families allow us to exclude antibiotics, heavy metals, and a host of other substances. The three pump groups that make up the nodulation cell division (RND) family are each in charge of resisting a particular kind of antibiotics. Clinical isolates of *A. baumannii* were reported to be 37% resistant to colistin in a previous investigation conducted in Sudan in 2015 (33). Previous study in Iraq in 2016 found that *A. baumannii* clinical isolates were 20% resistant to colistin (34). Previous study has reported that *A. baumannii* from clinical isolates were 96% resistant to colistin (35). Colistin-resistant *A. baumannii* may become resistant to colistin due to modifications of the outer membrane which may increase the permeability to other cell wall antimicrobial agents. Previous studies have reported that colistin-resistant *A. baumannii* strains were more susceptible to other antimicrobial agents than colistin-susceptible strain (36).

Previous study in Iraq in 2012 shown that resistance to Imipenem was 57.1% (37). Previous study in Iraq in 2014 shown that low resistance to Imipenem 30% (38). Other study also in Iraq shown that One (10%) isolate was observed to be imipenem and meropenem resistant (39). Iraqi study in 2017 found that *A. baumannii* clinical isolates were 68.7% and 50% resistant to Cefotaxime and Imipenem respectively (40). *A. baumannii* clinical isolates in previous study were 68.7% and 90.9% resistant to Imipenem (41,42).

In present study appeared to have resistance to Cefotaxime (92.5%) compare to previous study shown that all isolates appeared to have resistance to Cefotaxime (60%) (Jabur, 2014). Previous study have reported that most isolates of *A. baumannii* are considered to be resistant to Cefotaxime in rate reach to (80%) and (60%) respectively (43,44). Other previous study found that (12.2%) of isolates were resistant to Cefotaxime (45).

Conclusion

A. baumannii has demonstrated a high level of resistance to the majority of tested antibiotics, earning it the reputation of being the most therapeutically significant *Acinetobacter* species globally. It has become a significant nosocomial opportunistic pathogen in hospital infection epidemics.

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