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REVIEWOF ACINETOBACTER BAUMANNII: TAXONOMY, EPIDEMIOLOGY, PATHOGENICITY, AND ANTIMICROBIAL RESISTANCE

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Abstract

Genus Acinetobacter are wide spread in nature , and there are at least 25 different types of them .In the medical field Acinetobacter baumannii is one of the most important species of this genus. It is an opportunistic pathogen and have many virulence factors that make the bacteria capable of causing many hospital-acquired diseases leads to nosocomial outbreak attendant with arise in death rates . This bacteria has the ability to be resistant to many antibiotics and the emergence of high levels of multi-drug resistance A. baumannii has made it priority health issue and is considered a serious threat to health care facilities , public health and the elderly which requires a tremendous effort to stop this escalation.

Keywords: Acinetobacter Baumanni, Multi-Drug Resistance, Hospital-Acquired Infections.

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1.Introduction

The genus Acinetobacter was first isolated from soil in 1911 by scientist Beijerinck who called Micrococcus calcoaceticus [1,2] . During the 1940s ,the last century it was referred to known as Acinetobacter [3] . The genus Acinetobacter includes species which are gram-negative coccobacilli , non-motile ,strictly aerobic and non-oxidase production , while catalase is produced [4] . On usual laboratory media, it can grow easily and found in humid environments including ponds, waste water , moist soil , water treatment plant , fish farms and sea water [5] . Acinetobacter are wide spread in nature and there are at least 25 different types of them [6] . During the past two decade , clinical importance of A. baumannii isolates were increased due to the injuries caused among the wounded American soldiers during the war in Iraq and Afghanistan and high ability to acquire resistance to all antibiotics which are currently used , as well as their ability to stay for a long time in the hospital environment which lead to their rapid spread and infection outbreaks [7,8] . Also , A. baumannii is responsible for many hospital acquired infections including Pneumonia were associated with the artificial respiration system in patients were confined to hospital intensive care units (ICUs) . It can infect the skin, wounds , the respiratory System and digestive system in human [9,10] . A. baumannii is characterized by having many virulence factors that contribute to its pathogenicity[11]. This review aimed to focus on the role of Acinetobacter bauomannii which is considered one of the most important pathogens that cause infectious disease in hospital around the world and multi-drug resistant that made it apriority health issue.

Acinetobacte baumannii

Medically, it is the most important species of the genus Acinetobacter . The species was first isolated from soil and water in (1968) by scientist Baumann , as well as from food samples such as meat and vegetables . It was also isolated from various human pathological samples such as blood , sputum , pleural fluid and urine[12] . A. baumannii appears as gram-negative coccobacilli are non-motile , aerobic , opportunistic pathogens that mainly attack hospitalized patients they have the ability to resist and survive for a long time throughout hospitals enhances their ability to spread [13] . Biochemical characteristics of Acinetobacter baumannii shown in table (1) . This type can survive for a long time in dry conditions , for example it lasts 7days on dry washed clothes , 6 days on dry filter paper , 3days on Formica, 7days on glass and more than 25 days on cotton . It has the ability to grow at a temperature of44°c and this is what distinguishes it from other species [14].

Table 1 : Biochemical characteristics of Acinetobacter baumannii

Tests	Results
Morphology	Coccobacilli
Motility	Non motile
Hemolysis	-ve
Growth at 37°C , 44°C	+ve
Production of Oxidase	-ve
Production of Catalase	+ve
Utilization of citrate	+ve
Hydrolysis of gelatin	-ve
Oxidative-Fermentative(Glucose, Dextrose)	+ve
Arginine	+ve
Malonate	+ve
Production of urease	-ve
Reduction of nitrate	-ve
Production of Indole	-ve
Fermentation of lactose	-ve

(-ve : Negative , +ve Positive)

Current Taxonomy State

According to Bergey's Manual of Systematic Bacteriology were categorized the Acinetobacter genus with the Neisseriaceae family [15],and one species Acinetobacter calcoaceticus . The names of the species have persisted great taxonomic modifications over the years because of the superior knowledge of molecular strategies of the genetic makeup of this set of pathogens[16] . Recent classification of Acinetobacter which has accepted by bacterial taxonomists is shown in table 2[17,18].

Table 2 :Recent classification of Acinetobacter baumannii

Classification	
Domain	Bacteria
Phylum	Proteobacteria
Class	Gammaproteobacteria
Order	Pseudomonadales
Family	Moraxellaceae
Genus	Acinetobacter
Species	Acinetobacter bauomannii

Epidemiology of Acinetobacter baumannii

The epidemiology of Acinetobacter baumannii is a wide spread which includes infection related to wars , tropical environments , and hospital – acquired infections [19] . It can habitat soil and water , and other probably reservoirs are include lice and food animal [20] . In human , high colonization rates of the skin , throat, wounds, respiratory tract and gastrointestinal tracts of various degree of importance documented in numerous outbreaks [21,22] . In particular outbreaks among patients in the intensive care unit (ICU). In addition, many reports have been indicating cause of out breaks and nosocomial infections which are involve ventilator - associated pneumoniae , meningitis, septicemia , bacteremia, urinary tract infections and endocarditis [23,24] . Most epidemiological studies have been described the prevalence of infection with multi-drug resistance A. baumannii more than 21% of all hospital infection in various regions of the world, including North America , China ,Brazil , Japan and Europe [25] . Several cases have been reported from hospitals in the middle East such as United Arab Emirates , Saudia Arabia ,Bahrain and Lebanon ,which indicated prevalence of infection by A.baumannii from 19.1% in 2006 to 90% in 2015 [26,27] . Community acquired pneumonia infection have been reported in Asia and Australia especially during the wet season [28,29] . It is not known what causes ahigh rates of infection with Acinetobacter in a particular geographical area, however this may due to the variation in temperature and humidity that affect the colonized bacteria [30] .

Pathogenicity

A. baumannii is an opportunistic bacterium that has attracted the attention of specialists may due to its high ability to develop resistance against many antibiotics (Multiple Drug Resistance (MDR) and it has the potential to acquire genetic material from different races as well as its biofilm formation and its great ability to adhere to surface [31].The cause of pathogenicity of A. baumannii is due to its possession of many virulence factors(Figure1) including (curlifiber , cytotoxic , necrotizing factor , siderophores and aerobactin) , in addition to its production of colicin (v) , gelatinase , capsule and possession of polysaccharides and the formation of pellicle assay . Also it produces some types of enzymes , including lipolytic enzyme (lipase) and proteolytic enzyme (protease) [32] . Diseases caused by A. baumannii are wide spread and in different area of the human body with varying severity among the infected . These infections include : pneumonia , endocarditis , meningitis , wound and burn injuries , peritonitis , osteomyelitis , arthritis and bacteremia [33,34] . It can also colonize the skin , the nervous system and the eyes, and cause soft tissue infections [35,36] and poses a major risk to hospitalize patients especially those in the Intensive Care Unit (ICU) . Those who are

immunocompromised and spended long time in the hospital and these are exposed to infection with bacteria through hospital equipment or through the care workers by direct contact with the infected patient , and this bacteria is contaminated in different parts of the hospital environment , it was found on curtains , throat examination blades , door handles , wipers , keys, patient lift equipment , as well as a medical equipment . They need to pay attention to disinfecting common equipment , especially catheter and respiratory equipment [37,38].

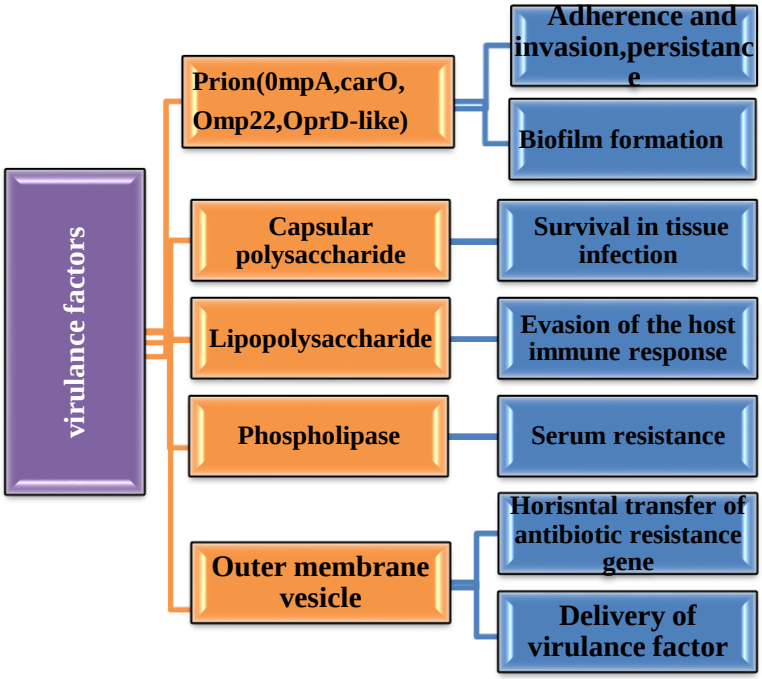

Figure 1:Virulance factor of Acinetobacter baumannii

Resistance of A. baumannii to antibiotics

A. baumannii have spread throughout the world due to their ability to survive in various environmental conditions and to acquire new genetic factors as a result of : first – mobile genetic elements such as transferable plasmid and intergons . Second – the inherited modification of the autonomous genes by mutations that change the target of the antagonists , which in turn change the self – resistance [39,40] . The presence of such motile genetic elements are concern and acquisition of genes can trigger drastic changes in resistance . Additionally , the ability of A. baumannii to confer on plasmids carrying anti – resistance traits are likely to spread among other types of bacteria [41,42] . The bacteria exhibit self – resistance to various antigens due to the decrease of the permeability in the outer membrane and flow pumps . The bacteria have become resistance to all commonly antibiotics including ,B-lactams , Quinolones , polymyxins , Aminoglycosides , Tetracyclines , Chloramphenicol and Glycylcyclines [43,44] . The resistance of A. baumannii to beta lactams includes several mechanisms (A): Enzymatic mechanisms or the production of antigen degradation enzymes . The main mechanisms in resistance to beta– lactam lies in their composition of broad spectrum beta lactamases ,which are encoded by genes that carried on a plasmid or chromosome . The AMPC – B – lactamases is one of the chromosomal enzymes that degrade broad – spectrum cephalosporins , except for cefepime [45,46] . AS well as the enzyme Oxacillinases (OXA), which are characterized by their effectiveness of analyzing anti-carbapenems . As for the enzymes containing minerals , they degrade all B- lactams except for Aztreonam which are often encoded by mobile genetic elements that can pass easily between bacteria [47] . (B): Non – enzymatic mechanisms including changing the permeability of the membrane either by losing or decreasing outer membrane proteins (OMPs) , or by increasing the flow pumps which leads to prevent the entry of the antagonist or enhance its expulsion to the outside [48] . The sequences of penicillin binding proteins (PBPs) were varied , as changes occur in the expression levels of normal PBPs . Studies have indicated that there are several differences in the expression pattern of the binding proteins between A. baumannii susceptible bacteria and anti – carbapenem resistant and that decrease in antagonist affinity which is associated with increased expression of these proteins [49] . A. baumannii resists Quinolones by modulating the

antibody binding site where mutation occur in genes encoding the DNA gyrase or one of the targeted topoisomerases and the bacteria also have flow pumps that cat on the extracellular antagonist tunnel [50] . As for the resistance of A. baumannii to aminoglycoside it can by changing the flow pumps or by expressing the modulating enzymes , especially Acetyltransferases , Nucleotidyl transferases and phosphotransferases . To produce these enzymes , the encode chromosomal or plasmid genes or paired with inegrons and jumpers genes [51,52] . These enzymes modify the hydroxyl or amine group present within these antibiotics and thus reduce the affinity for the target site . Bacteria possess the gene responsible for the 16s rRNA methylation that prevents significant resistance to most Aminoglycosides such as Amikacin , Tobramycin and Gentamicin [53,54] while the mechanisms of bacterial resistance to Tetracycline are modulated the target site on the ribosomal subunit (30s), due to presence of the gene which is carried on the conjugated plasmide which were encoded for the protein that provides protection of the ribosomal subunit of the antibiotic , as well as the possession of a gene that encodes flow systems for this antibiotic [55,56] . As for the mechanism of resistance to polymyxins , it reduced the net negative charge of the outer membrane of the bacteria and thus reduced the affinity between the antibiotic and the surface at the bacteria , as indicated by [57,58] , as for the mechanism of action of polymyxins is by stable electrical interactions between the positive charges of polymyxins groups and the negatively charged phosphate groups on Lipid A in lipopolysaccharide (LPS) and resistance can occur as a result in inhibition of certain genes responsible for the biosynthesis of the membrane of lipid A or the elimination completely [59]. Also [60]was indicated that alterations to lipid A with the introduction of phosphor-ethanolamine were presented in all isolates of A. baumannii anti-colistin resistance.

Conclusion

Acinetobacter baumannii was considered one of the most important pathogen that cause infectious diseases in hospitals around the world . The emergence of high levels of multi-drug resistant A. baumannii made them a priority health issue, especially in hospital and nursing homes for children and the elderly which are requires further research and development to discover new antibiotics to reduce deaths from resistant infections around the world . It is also necessary to improve programs to prevent and limit the spread of infection with this pathogens in hospitals.

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