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Detection of Carbapenem Resistant Acinetobacter: From Clinical Samples

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ABSTRACT

Acinetobacter has appeared from an organism of uncertain pathogenecity towards an infectious agent. Among nonfermenting bacterium A. baumannii is the second-most-commonly-isolated organisms in human. The fast intensify of their resistance to antibiotics, especially global emergence and extend of Acinetobacter strains resistant to carbapenem more restricted the therapeutic alternatives. In this study we evaluate the % of resistivity of Acinetobacter against carbapenem antibiotics at Jinnah University for Women, Karachi. Total 439 isolates of Acinetobacter were collected from different clinical samples of hospitalized patients from January to December 2013, identified by standard microbiological methods. Antibiograms were done on Mueller-Hinton agar plates with disk diffusion method (Kirby Bauer method). Disc tested: Meropenem (10 µg/disk). The results were interpreted according to the Guidelines of the Clinical and Laboratory Standards Institute. (CLSI). Among 439 samples, 300 (68.3%) samples were resistant to Meropenem and the remaining that is 139 (31.7%) showed sensitivity to the drugs. In underdeveloped countries including Pakistan the contentment of multidrug resistance and their dissemination in Acinetobacter species is not a simple task. While multiple drug resistance is increasing in this pathogen, and carbapenem conflict is quickly spreading which may become a major threat in future. So in Pakistan needs detail and organized data about carbapenem resistant Acinetobacter in order to understand the existence of Acinetobacter in our community and to manage almost certainly outbreaks because we have less information according to resistance trends of Acenetobacter.

Keywords: Antibiogram, Carbapenem, Resistant, Outbreaks.

INTRODUCTION

Species of genus Acinetobacter are gram-negative belongs to the class Gammaproteobacteria. Species are non-motile, coccobacili in shape appear in pairs, oxidase-negative (Peleg *et al*., 2008). Acinetobacter consist of 27 authentically named and 11 unknown (genomic) species. Species names, including: *A. baumannii, A. calcoaceticus, A. haemolyticus, A. johnsonii, A. junii,* and *A. lwoffii* (Manchanda *et al*., 2010). Widely dispersed in environment they can be alive on dry and moist surfaces, as well as in hospital surroundings (Maragakis and Perl, 2008). Patients in the intensive care unit (ICU), including burn patients, trauma patients, and patients requiring mechanical ventilation are mainly affected by *Acinetobacter baumannii* (Bassetti *et al*., 2008). Also some strains have been found from foodstuffs, water, and associate with low occurrence of allergies. Several species of Acinetobacter can cause serious infections in immunocompromised patients (Peleg *et al*., 2008). They also show comparatively wide scale of antibiotic resistance (Towner, 2009). Infections together with skin and wound added complication as well as bacteremia, and meningitis (Choi *et al*., 2008). The bacteria have the ability to colonize in medical utensils e.g. catheters (Doughari

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et al., 2011). Plasmid-borne markers which made the Acinetobacter resistant to various antibiotics are capable of transferring to new pathogenic bacteria through horizontal gene transfer (Juni, 1978). 'MDR *Acinetobacter spp.*' defined as the isolate resistant to at least three classes of antimicrobial agents — all penicillins and cephalosporin (including inhibitor combinations), fluroquinolones, and amino glycosides. 'XDR *Acinetobacter spp.*' that is resistant to the three classes of antimicrobials described above (MDR) and shall also be resistant to carbapenems (Kurcik-Trajkovska, 2009).

The clinical isolates of *Acinetobacter spp.* were generally susceptible to gentamicin, minocycline, nalidixic acid, ampicillin, or carbenicillin, singly or in a combination therapy, throughout the early 1970s (Bergogne-Bérézin and Towner, 1996). But, since 1975, growing resistance started to appear in almost all groups of drugs including the first and second generation cephalosporins. Primarily, they retained at least partial susceptibility against the third and fourth generation cephalosporins, fluoroquinolones, semi synthetic aminoglycosides, and carbapenems, with nearly 100% isolates holding susceptibility to imipenem (Vila *et al*., 2007). However, during late 1980s and 1990s, worldwide emergence and spread of Acinetobacter strains resistant to imipenem further limited the therapeutic alternatives. By the late 1990s, the only useful agents that fight many severe infections caused by *Acinetobacter sp*. were carbapenems (Cunha, 2013). Moreover, the therapeutic options are decreasing due to the emergence of carbapenem resistance in the strains of *A. baumannii* (SPL, 2011). Various mechanisms have been found to be accountable for the resistance to carbapenems in *A. baumannii* (CUH, 2013). The resistivity mechanismsusually fall into 3 categories: 1. antimicrobial-inactivating enzymes, 2. reduced access to bacterial targets, or 3. mutations that change targets or cellular functions (Camp and Tatum, 2010). Treatment is difficult for healthcare-acquired infection caused by *A. baumannii* resistant to imipenem (Smith *et al*., 2007). Carbapenems still represent the treatment of choice. A. baumannii is competent to grow at different temperature and pH because it does not have fastidious growth requirements. The versatile organism exploits a variety of both carbon and energy sources. These qualities explain the ability of Acinetobacter species to persist in either moist or dry conditions in the hospital environment, thereby contributing to transmission. This hardi- ness, combined with its intrinsic resistance to many antimicrobial agents, contributes to spread in the hospital setting.

Figure 1: Outbreaks of Acinetobacter in United States between 2002 and 2007 (Chuang *et al*., 2011).

MATERIALS AND METHODS

Setting: Department of Microbiology, Jinnah University for Women Karachi.

Duration of study: From January 2013to December 2013

Sampling technique: Non-duplicate consecutive sampling.

Inclusion Criteria: All Acinetobacter colonies isolated from different clinical samples of patients.

Exclusion Criteria: Sample showing no growth or growth of gram positive bacteria, growth of gram negative bacteria other than Acinetobacter and yeast. Repeat and duplicate samples from the same patient were also being excluded.

Study design: Descriptive study.

Data collection: All clinical samples were collected in sterilized container according to samples from patients of different hospitals and platted right after the collection. Identification will be taken by standard microbiological methods. Inoculation of clinical samples was done on standard media such as sheep blood agar (SBA) Mackonkey agar and Chocolate Agar. Antibiograms were done on Mueller-Hinton agar plates with disk diffusion method according to Kirby Bauer method. Disc tested: Meropenem (10 µg/disk) (Oxoid Ltd., England) The results was interpreted according to the Guidelines of the Clinical and Laboratory Standards Institute (CLSI).

RESULTS

Table I: Susceptibity pattern of *Acinetobacter sp.* from different samples.

S.No	Sample	Total no. of samples	Susceptible to Meropenem	Resistant to Meropenem
1	Tracheal aspirates	195	41	154(78.9%)
2	Blood	34	18	16 (47 %)
3	Urine	30	14	16(53.3 %)
4	Pus and swabs	101	53	48(52.4 %)
5	Sputum	41	13	27 (65.5 %)
6	Fluid	38	4	34 (89.7 %)

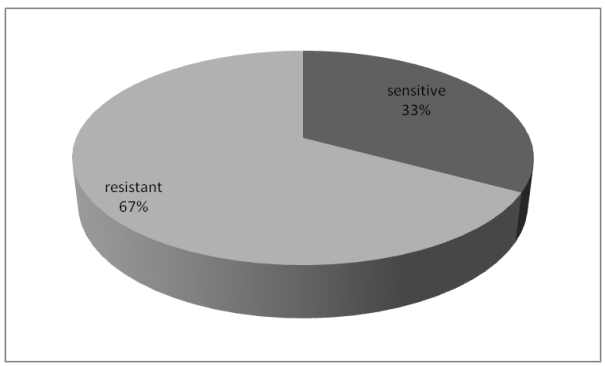

Figure 2: Sensitivity & resistivity % of meropenem of total samples.

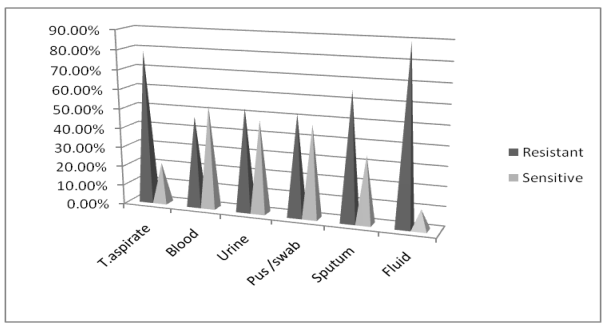

Figure 3: Sensitivity & resistivity % of meropenem among different clinical samples.

DISCUSSION

We have performed our studies on 439 isolates. We hereby observed the sensitivity and resistivity pattern of Meropenem on different samples including pus, urine, tracheal aspirates, sputum, blood and body fluids which were loaded with Acinetobacter. We observed that among 439 samples, 295(67%) samples were resistant to Meropenem the remaining i.e. 144(33%) showed sensitivity to the drug. Out of 439 samples 195 were tracheal aspirates, 34 were blood samples, 30 were urine, 101 were pus and swabs, 41 were sputum samples and 38 were fluid samples.

This increased resistivity of Acinetobacter is considered as an important health problem due to considerable clinical impact of this resistivity on the management of health care associated infections. In intensive care unit, the serious concern of high morbidity and high mortality rates of A.baumanii is their nosocomial outbreaks. Clinical threats scored by acinetobacter are its adaptation to the environment, versatile metabolism and its ability to develop resistance against antibiotics that are used in clinical settings. This danger has caused an noticeable and alarming decline in the available chemotherapeutic resources, that includes drugs like, carbapenem antibiotics that inhibits peptidoglycan biosynthesis that was considered the first-rate standard for Acinetobacter treatment until recently (Guerrero *et al*., 2010).

Infections due to Acinetobacter frequently involve organ systems that have a high fluid content (eg, respiratory tract, CSF, peritoneal fluid, urinary tract), manifesting as nosocomial pneumonia, associated with continuous ambulatory peritoneal dialysis (CAPD), or catheter-associated bacteruria. The presence of Acinetobacter isolates in respiratory secretions in incubated patients nearly always represents colonization. Acinetobacter pneumonia occur in outbreaks and are usually associated with colonized respiratory-support equipment or fluids. Nosocomial meningitis may occur in colonized neurosurgical patients with external ventricular

drainage tubes.

A. baumannii is a multiresistant aerobic gram-negative bacillus sensitive to relatively few antibiotics. Multidrug-resistant Acinetobacter is not a new or emerging phenomenon, but *A. baumannii* has always been an organism inherently resistant to multiple antibiotics.

Since the past decade, antimicrobial resistance among *Acinetobacter sp.* is a rising concern. The species are equipped with extensive antimicrobial resistance due to the presence of the porin channels, efflux mechanisms and the non static behaviour of the bacteria in hot and humid conditions (Davies and Rubin, 2007).

CONCLUSION

In underdeveloped countries including Pakistan , india and Bangladesh the contentment of multidrug resistance and their dissemination in *Acinetobacter sp.* is not a simple task. While multiple drug resistance is increasing in this pathogen, and carbapenem conflict is quickly spreading which become a major threats in future because patient-to-patient transmission in hospitals through contaminated hands of healthcare workers and fomites is the main factors which increases the spreading of MDR Acenetobacter and cure should be followed. The increased resistivity of the pattern of the organism has become a serious threat for health in Pakistan.

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