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Antibiotic resistance patterns of *Acinetobacter* species isolated in King Hussein Medical Center, Jordan

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Acinetobacter species is aerobic, Gram-negative coccobacilli, oxidase-negative, catalase positive, nonfermenting bacteria. Though widely prevalent in nature and generally regarded as commensals of human skin, respiratory and genitourinary tracts colonize the skin up to 25% and up to 7% in the pharynx of healthy adults.[1]

In 1986, taxonomy of the genus *Acinetobacter* was changed extensively by Bouvet and Grimont,[2] who outlined 12 different species by DNA-DNA-hybridization, including the named species *Acinetobacter Baumanii* (*A. Baumanii*), *Acinetobacter Calcoaceticus* (*A. Calcoaceticus*), *Acinetobacter Haemolyticus* (*A. Haemolyticus*), *Acinetobacter Johnsonii* (*A. Johnsonii*), *Acinetobacter Junii* (*A. Junii*) and *Acinetobacter Lwoffii* (*A. Lwoffii*) and 6 unnamed genomic species. Most of *A. Baumannii* and all *Acinetobacter* species strains 3 and 10 represent organisms that were formerly classified as *Acinetobacter Anitratus* (*A. Anitratus),* whereas all *A. Junii, A. Lwoffii* and *Acinetobacter* species strains 11 were formerly classified as *A. Lwoffii*.[3] These species are often multiresistant to antibiotics, meaning that therapy and infection control are complicated.[4] *Acinetobacter* species now known to be responsible for a wide range of nosocomial infections, including bacteremia, secondary meningitis, urinary tract infections, pneumonia, tracheobronchitis, endocarditis, wound infections and surgical site infections. *Acinetobacter* infections are most frequently associated with the use of a ventilator, urinary tract catheter or other invasive device. In the United States of America, among the intensive care unit (ICU) patients, during the period 1987-1996, reported cases of nosocomial *Acinetobacter* infections were 3447, the average rate of infection being significantly higher during summer than in winter.[4]

The aim of this study was to determine antibiotic resistance rates of *Acinetobacter* species strains isolated from patients in order to give information to clinicians when empiric therapy is necessary. A total of 133 consecutive, non duplicate *Acinetobacter* species isolates were studied over a period of 18 months, between July 2000 and December 2001 from various clinical materials at King Hussein Medical Center, Amman, Jordan, which is a 800 bed hospital. Duplicated isolates from the same infective episode in the same patients were excluded. According to the instructions provided by the manufacture, Vitek-1 system (Bio Merieux, France) were used for identification and studying the susceptibility of isolates, using V 1306 vitek GNI (Gram-negative identification) card for identification of the isolates, and V 4313 vitek GNS-528 (Gram-negative susceptibility) cards were used for studying the susceptibility of *Acinetobacter* isolates, and for the susceptibility testing of the isolates from urine using V 4525 Vitek GNS-203. *Echerichia coli* ATTCC 25922, *Staphylococcus aureus* ATTCC 25923 and *Pseudomonas aeruginosa* ATTCC 27853 were used as quality control organisms.

During the period of 18 months, a total of 133 *Acinetobacter* species isolated from clinical specimens were tested for antimicrobial susceptibility, among these isolates were 38 (28.57%) were from urine, 31 (23.31%) from blood, 54 (40.60%) from wounds, 7 (5.26%) from sputum, and 3 (2.55%) from other specimens. *Acinetobacter Calcoaceticus Biotype Anitratus* was the most common *Acinetobacter* species isolated with 130 isolates (97.74%), followed by *A. Lowffii* with 2 isolates (1.50%) and *A. Calcoaceticus-Bumanii* complex with 1 isolate (0.75%). The results of activities of antimicrobial agents against the *Acinetobacter* isolates are shown in **Table 1**. The most active antimicrobials for the isolates from urine was minocycline, while for the isolates from other specimens were the imipenem, ticarcillin/clavulanate, ceftazidime and netilmicin. An important feature of *Acinetobacter* species, is their intrinsic resistance to multiple antibiotics. Recently, reported surveys have demonstrated high rates of resistance to aminoglycosides, cephalosporins, quinolones, penicillins, monobactams, and imipenem, often in excess of 50%, among clinical isolates of *Acinetobacter*. Antimicrobial treatment of the infections due to highly resistant *Acinetobacter* strains can lead to treatment failure, and is associated with an increased risk of death.[5]

Table 1 - Resistance rates to the antimicrobial agents.

Antibiotic	Blood isolates (%)	Wounds and other (%)	All isolates (%)	Antibiotics	Urine isolates (%)
Amikacin	(23.53)	(44.83)	(34.18)	Amoxicillin/CA	(65.1)
Aztreonam	(58.82)	(86.21)	(72.51)	Ampicillin	(88.5)
Cefepime	(41.18)	(79.31)	(60.24)	Carbenicillin	(28.5)
Cefsulodin	-	-	-	Cefazolin	(9.14)
Ceftazidime	(29.41)	(13.79)	(21.60)	Ceftriaxone	(5.14)
Ciprofloxacin	(29.41)	(72.41)	(50.91)	Cefuroxime/sodium	(9.14)
Gentamicin	(58.82)	(82.76)	(70.79)	Cephalothin	(9.14)
Imipenem	(5.88)	(6.90)	(6.39)	Ciprofloxacin	(51.43)
Netilmicin	(41.18)	(20.69)	(30.93)	Gentamicin	(62.86)
Pefloxacin	(41.18)	(72.41)	(56.80)	Minocycline	-
Piperacillin	(64.71)	(79.31)	(72.01)	Nalidixic acid	(51.43)
Piperacillin/tazobactam	(17.65)	(44.83)	(31.24)	Nitrofurantoin	(100)
Ticarcillin	(41.18)	(48.28)	(44.73)	Ofloxacin	(51.43)
Ticarcillin/clavulanate	(23.53)	(13.79)	(18.66)	Ticaracillin/clavulanate	(1.14)
Tobramycin	(41.18)	(37.93)	(39.55)	Tobramycin	(45.1)
Trimethoprim/sulfamethoxazole	(58.82)	(72.41)	(65.61)	Trimethoprim/sulfamethoxazole	(65.1)

In our study, wounds were the most common site of isolation 40.60%, due to high number of specimens from the burn unit and surgical ICU. The results of antimicrobial susceptibility, for the *Acinetobacter* strains, which were isolated from urine, showed high percentage of susceptibility to minocycline (100%), to ticarcillin/CA (82.86%), to carbenicillin (71.43%), and to tobramycin (54.29%). By contrast, the other 14 antibiotics which tested in this group, showed high percentage of resistance (>50%), for the *Acinetobacter* strains which isolated from clinical specimens other than urine, and among the cephalosporins, which *Acinetobacter* bacteria are naturally resistant due to the production of cephalosporinase,[5] the ceftazidime showed the percentage of resistance of (21.60%) and for cefepime (60.24%). The aminoglycosides in our study was 39.55% of the isolates, and were resistance to tobramycin, 70.79% to gentamicin, 34.18% to amikacin, and 30.93% to netilmicin. Thus, the presence of aminoglycoside-modifying enzymes is responsible for the resistance of *Acinetobacter* strains to a great number of aminoglycosides.[6] Trimethoprim-sulfamethoxazole in our study showed percentage of resistance of 65% for *Acinetobacter* isolates from urine and other specimens. Among quinolones, 50.91% of the isolates were resistance to ciprofloxacin, 56.80% to pefloxacin, and 51.43% of the *Acinetobacter* isolates from urine were resistance to ofloxacin. In β -lactam group of antibiotics, imipenem was the most active antibiotics for all isolates, with 6.39% resistance rate, followed by ticarcillin/clavulanate with 18.66%, ceftazidime 21.60%, and piperacillin /tazobactam 31.24%, while aztreonam 72.51%, piperacillin 72.01% and cefepime 60.24% showed high percentage of resistances, while in European hospitals,[7] imipenem 31%, ceftazidime 35% and piperacillin/tazobactam 34%.

In conclusion, the susceptibility rates of *Acinetobacter* species vary widely, geographically and with time, although comparison between studies is difficult, one explanation for this is the variation in methods of susceptibility testing and in breakpoints to determine sensitivity, and in the present study and other studies, imipenem remained one of the most active antibiotics against *Acinetobacter* bacteria.

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Anti-*Toxoplasma gondii* antibodies in patients infected with hepatitis B virus

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Toxoplasma gondii (*T. gondii*) is closely related to other Coccidia and has certain similarities to malarial parasites. The parasites were first discovered on a North African rodent called the *Ctenodactylus gundii*, hence, the species was named as *gondii*. Although serologic evidence indicates a high rate of human exposure to the organism, the disease itself is relatively rare. *Toxoplasma gondii* can infect many vertebrates as well as humans, but the definitive host is the house cat and other members of the Felidae family. This organism is an obligate intracellular parasite, which are found in humans in 2 forms. The actively proliferating trophozoites or tachyzoites are usually seen in early, more acute phases of infection. The resting forms or tissue cysts are primarily found in muscle and brain, probably as a result of the host immune response.[1] *Toxoplasma* infections can be acquired postnatally and are categorized into 4 groups: (a) lymphadenitis, fever, headache and myalgia, with a possibility of splenomegaly and a brief erythematous rash (b) typhus-like exanthematous form with myocarditis, meningoencephalitis atypical pneumonia and possible death (c) retinochoroiditis, which may be severe, requiring enucleation (d) central nervous system involvement, which is usually fatal.[2] *Toxoplasma gondii* is transmitted parenterally, flourish in states of immunosuppression and most of *Toxoplasma* infections are asymptomatic. The large number of people who are serologically positive for *T. gondii* suggest that the majority of infections are benign, with most people exhibiting few (cold or light case of flu) or no symptoms. In a small percentage of cases, symptoms may range from mild to severe results.[2]

In Turkey, hepatitis B virus (HBV) is still a serious health problem. The prevalence of HBV carriage is 2-10%. Hepatitis B represents syndromes of hepatocellular necrosis, inflammatory and regenerative responses associated with little or no liver disease or with acute hepatitis. Patients with HBV demonstrate various cellular and humoral immunity disorders. Immunosuppression seems to increase HBV replication.[3] It may be thought that toxoplasmosis may lead to more frequent and more severe diseases in patients with HBV and may change the course of the disease. Therefore, we planned this study to determine the seroprevalence of anti-*T. gondii* antibodies in patients infected with HBV.

One hundred patients (57 males and 43 females; mean age: 46.5 ± 10.2) with HBV were selected and followed up by Ege University Medical Faculty, Gastroenterology department. All selected patients had positive hepatitis B surface antigen (HBs Ag) and they have been followed for at least 6 months for HBV. We also selected 50 healthy volunteer blood donors as control group (31 males and 19 females; mean age: 40 ± 6.7). Blood samples were taken from the brachial vein of all patients under sterile conditions. The sera were separated after centrifugation at 1000 x rounds per minute for 10 minutes and stored at –20 ^{o}C until the analysis.