

Spectrum and Antibiotic Resistance of Uropathogens Isolated from Hospital and Community Patients with Urinary Tract Infections in Two Large Hospitals in Kuwait

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Key Words

Urinary tract infection · Uropathogens · Antibiotic resistance

Abstract

Objectives: To determine the spectrum of microbial etiology and antibiotic resistance pattern of the uropathogens that cause urinary tract infections in 2 large teaching hospitals in Kuwait over a period of 1 year. **Materials and Methods:** The Vitek identification card system was used to identify the uropathogens. Susceptibility of the isolates against 18 antibiotics was performed by the microbroth dilution method using the Vitek automated system. In addition, gram-positive bacteria were tested in parallel by the disk diffusion technique. **Results:** The six overall most common isolates were: *Escherichia coli*, accounting for 47% of isolates in both hospitals, followed by *Candida* spp. (10.8%), *Klebsiella pneumoniae* (9.6%), *Streptococcus agalactiae* (GBS; 9.5%), *Enterococcus faecalis* (4.2%) and *Pseudomonas aeruginosa* (4.1%). Amikacin provided the widest coverage amongst all the antibiotics tested followed by ciprofloxacin, gentamicin and piperacillin-tazobactam. For the gram-negatives, high resistance (26–63%) to the β -lactam antibiotics was noted, especially to ampicillin, amoxicillin-clavulanic

acid, cephalothin and cefuroxime. Resistance to trimethoprim-sulfamethoxazole was also high. None of the enterococci was resistant to the glycopeptides, but 38–60% of the *Staphylococcus haemolyticus* were resistant to vancomycin or teicoplanin. **Conclusion:** These data show the high level of antimicrobial resistance amongst the uropathogens causing urinary tract infection in the two hospitals studied.

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Introduction

Urinary tract infection (UTI) is a very common infection both in the community and hospital patients and ranks high amongst the most common reasons that compel a patient to seek medical attention [1]. Acute UTIs are associated with substantial morbidity and problems of recurrent infections. Studies have shown that approximately 25% of women with first UTI will have another attack within 6 months [2]. A mounting body of evidence indicate that UTI is responsible for a large proportion of antibiotic consumption in and out of the hospital. No wonder that the infection has a large socio-economic impact and contributes to the emergence of antibiotic resistance in the hospital and the community [3, 4].

To optimize the use of empiric antibiotic therapy for UTI, it is important for clinicians to have a working knowledge of the etiological agents and the susceptibility patterns of UTI pathogens in their population. Since uropathogens and their susceptibility may vary from time to time and from one institution to another, each hospital or institution must have its own evaluation for such infection so that any change in the causative agents and susceptibility pattern can be detected and managed accordingly. Such evaluation is also useful to detect the spread of infection from one patient to another in the hospital as well as to assist in the setting up of an antibiotic policy.

Several studies in the past have shown that *Escherichia coli* is the most common etiological agent of UTI in hospital and community patients and that hospital-acquired UTI (HA-UTI), in particular, is characteristically associated with a higher prevalence of enterococci and coagulase-negative staphylococci (CNS) [5–9]. Of interest is the report of many recent studies from USA and Europe that showed increasing antibiotic resistance among uropathogenic *E. coli* to ampicillin, trimethoprim and sulfonamides [6, 7, 9, 10]. Also a previous report from Kuwait reported high resistance rate to ampicillin by this organism [11]. Importantly, the majority of acute UTIs are caused by strains of these uropathogens [12]. The apparent shift in the etiological agents of different infections encountered in the last decade has not been assessed in UTI in this part of the world. It is noteworthy that no comparative information has emerged concerning this important infection and the problem of antibiotic resistance amongst its uropathogens encountered in different hospitals in the Gulf region, especially in Kuwait with its diverse population.

This study was designed to determine the microbial etiological agents of UTI, as well as their antimicrobial susceptibility, amongst the inpatients and outpatients of a maternity and a general hospital in Kuwait.

Materials and Methods

This study was conducted in the Departments of Microbiology of two large teaching hospitals (Mubarak Al-Kabeer Hospital, MKH, and Maternity Hospital, MH) attached to the Medical School of Kuwait University. MKH is a 500-bedded tertiary hospital situated adjacent to the Medical School with a general intensive care unit (ICU), a pediatric ICU, a busy urology unit, renal dialysis and kidney transplant units. MH, a specialized 400-bedded hospital, dealing with obstetric and gynecological patients as well as neonates, is 18 km away. It also has an adult ICU and a special

care baby unit as well as neonatal ICU. A total of 37,203 and 14,359 records of urinary uropathogens and their antibiograms from MKH and MH, respectively, for the study period of January to December 2002, were analyzed. Urine specimens with bacterial growth of $\geq 10^5$ colony-forming unit/ml were regarded as significant bacteriuria. The materials were divided into 2 groups. One group comprised urine samples from patients admitted to the hospitals (HA-UTI) and the second group comprised samples from polyclinics and outpatient clinics attached to the hospitals (community-acquired UTI; CA-UTI).

Antibiotic susceptibility testing was performed for all significant gram-negative isolates by an automated Vitek machine (bioMérieux, Marcy-l'Étoile, France). All gram-positive bacteria were tested manually by Kirby-Bauer method. The results were interpreted according to the recommendation of the National Committee for Clinical Laboratory Standards [13]. Susceptibility testing for yeasts was not done. Identification and susceptibility testing were carried out by the same methods in both laboratories. In order to eliminate duplication, only one species per patient was included in the analysis. The materials were not stratified according to age or clinical diagnosis.

Reference strains of *E. coli*, ATCC 25922, and *Pseudomonas aeruginosa*, ATCC 27853, were used as controls for the gram-negative bacteria and were included in all daily runs. *Staphylococcus aureus*, ATCC 25923, and *Enterococcus faecalis*, ATCC 929212, were used as gram-positive control strains.

Results

Of the 37,203 urine samples from MKH, 3,594 (9.7%) and of 14,359 from MH 1,102 (7.7%) had significant bacteriuria. In order to eliminate duplicated results, only one species per patient was included in this analysis. The materials were not stratified according to age or clinical diagnosis.

The top six uropathogens causing UTI isolated from both hospitals were *E. coli* (47%), *Candida* spp. (10.8%), *Klebsiella pneumoniae* (9.6), *Streptococcus agalactiae* (GBS; 9.5%), *E. faecalis* (4.2%) and *P. aeruginosa* (4.1%).

The distribution of gram-negative, gram-positive bacteria and yeast isolated from the clinical samples is shown in table 1. These isolates from both hospital laboratories represented clinically significant pathogens. As shown in this table, *E. coli* was the predominant pathogen isolated from urine samples from HA-UTI and CA-UTI patients in both hospitals. In MKH, the top five most common organisms causing UTI in hospitalized patients were, in the descending order, the following: *E. coli* (35%), *Candida* spp. (19.8%), *K. pneumoniae* (9.9%), *P. aeruginosa* (9.8%) and *E. faecalis* (5.2%). In the CA-UTI patients, they were: *E. coli* (56.7%), *S. agalactiae* (group B streptococci; GBS) (10.4%), *K. pneumoniae* (8.5%), *E. faecalis*

Table 1. Microbial uropathogens isolated from urine of patients with UTIs in MKH and MH

| Microorganism | Number (%) of micro-organisms isolated from UTI | | | |
|------------------------------------|---|--------------|-------------|------------|
| | MKH patients | | MH patients | |
| | community | hospital | community | hospital |
| Gram-negative bacteria | | | | |
| <i>E. coli</i> | 1,389 (56.7) | 401 (35) | 146 (46.2) | 268 (34.1) |
| <i>K. pneumoniae</i> | 209 (8.5) | 113 (9.9) | 33 (10.4) | 97 (12.3) |
| <i>P. aeruginosa</i> | 68 (2.8) | 112 (9.8) | 3 (0.9) | 10 (1.3) |
| <i>Acinetobacter calcoaceticus</i> | 26 (1.1) | 40 (3.5) | 1 (0.3) | 11 (1.4) |
| <i>P. mirabilis</i> | 49 (2.0) | 14 (1.2) | 4 (1.3) | 2 (0.3) |
| <i>Enterobacter</i> spp. | 42 (1.7) | 48 (4.2) | 5 (1.6) | 11 (1.4) |
| <i>Citrobacter</i> spp. | 10 (0.4) | 14 (1.2) | 4 (1.3) | 2 (0.3) |
| Miscellaneous GNB | 56 (2.3) | 25 (2.2) | 0 | 32 (4) |
| Gram-positive bacteria | | | | |
| <i>S. agalactiae</i> (GBS) | 255 (10.4) | 28 (2.4) | 38 (12) | 126 (16.0) |
| <i>E. faecalis</i> | 95 (3.9) | 59 (5.2) | 8 (2.5) | 33 (4.2) |
| <i>S. aureus</i> | 32 (1.3) | 11 (1) | 2 (0.6) | 4 (0.5) |
| <i>S. epidermidis</i> | 25 (1) | 8 (0.7) | 0 | 0 |
| <i>S. haemolyticus</i> | 21 (0.9) | 5 (0.4) | 0 | 0 |
| <i>S. saprophyticus</i> | 74 (3) | 26 (2.3) | 6 (1.9) | 0 |
| Miscellaneous GPB | 38 (1.5) | 14 (1.2) | 9 (2.8) | 26 (3.3) |
| Yeasts | | | | |
| <i>C. albicans</i> | 22 (0.9) | 63 (5.5) | 45 (14.2) | 135 (17.2) |
| <i>Candida</i> spp. | 38 (1.6) | 164 (14.3) | 12 (3.8) | 29 (3.7) |
| Total | 2,449 | 1,145 | 316 | 786 |

(3.9%) and *Staphylococcus saprophyticus* (3%). Among the HA-UTI patients at the MH, the commonest organisms were: *E. coli* (34.1%), *Candida* spp. (20.9%) of which, *Candida albicans* was 17.2%, GBS (16%), *K. pneumoniae* (12.3%), *E. faecalis* (4.2%) and *S. saprophyticus* (3.3%). Those responsible for UTI in the community were: *E. coli* (46.2%), *Candida* spp. (18%), GBS (12%) and *K. pneumoniae* (10.4%).

Susceptibility Data

Of the antimicrobial agents tested at MKH (table 2), only amikacin, cefotaxime, ciprofloxacin and nitrofurantoin showed excellent activities against the *E. coli* isolates. Only 2 and 1%, 2 and 5%, 5 and 6% and 5 and 6% of the *E. coli* isolates from the CA-UTI and HA-UTI cases were resistant to amikacin, cefotaxime, ciprofloxacin and nitrofurantoin, respectively. Resistance rates by these isolates, from both CA-UTI and HA-UTI to ampicillin (63 and 76%, respectively), amoxicillin-clavulanic acid (amoxiclav) (45 and 53%), trimethoprim-sulfamethoxazole (SXT) (51 and 44%), piperacillin (46 and 62%) and nalidixic acid (16 and 25%) were very high. With the ex-

ception of *Proteus mirabilis*, the resistance rates of all the other gram-negative bacteria to the four first-line antibiotics (ampicillin, amoxiclav, SXT and nitrofurantoin) were also far above the acceptable 10% level of resistance.

At MH, the resistance rates of the gram-negative bacteria were relatively lower than the rates at MKH. Table 3 shows the antibiotic resistance rates of the gram-negative bacterial isolates. A large proportion of the *E. coli* isolates from the MH and MKH cases were also resistant to ampicillin (53 and 63%, respectively), amoxiclav (26 and 26%), SXT (42 and 37%) and piperacillin (32 and 52%). However, only 4 and 3% of *E. coli* isolates from these two settings, respectively, were resistant to nitrofurantoin.

In general, β -lactam antibiotics, e.g. ampicillin and amoxicillin-clavulanic acid, were less active than the 2nd and 3rd generation cephalosporins against *E. coli* from both hospitals. In MH isolates of *E. coli* resistance rates to cefotaxime, cefuroxime and cephalothin were 3, 20 and 9%, respectively, whereas in the MKH isolates, 8, 18 and 17%, respectively, were resistant.

Table 2. Percentage of resistant gram-negative uropathogens isolated from outpatients vs. inpatients with UTIs in MKH

| Bacteria | CA/HA | Percentage of CA vs. HA bacteria resistant to: | | | | | | | | | | | | | |
|-------------------------|-----------|--|-------|-------|-------|-------|-------|-------|-------|-------|-------|--------|-------|-------|-------|
| | | AN | AUG | AMP | CF | CTX | CXM | CIP | SXT | GN | NA | NIT | NOR | PIP | TZP |
| <i>E. coli</i> | 1,389/401 | 2/1 | 45/53 | 63/76 | 33/42 | 2/5 | 8/14 | 5/6 | 51/44 | 4/4 | 16/25 | 5/6 | 8/21 | 46/62 | 10/8 |
| <i>K. pneumoniae</i> | 209/113 | 2/1 | 25/35 | 99/98 | 26/26 | 1/8 | 1/17 | 20/9 | 22/33 | 2/4 | 10/14 | 46/65 | 3/9 | 33/80 | 0/41 |
| <i>P. aeruginosa</i> | 68/112 | 10/12 | – | – | – | – | – | 0/22 | – | 14/24 | 50/24 | –/– | 21/22 | 3/17 | 0/10 |
| <i>A. calcoaceticus</i> | 26/40 | 31/54 | 24/62 | 88/92 | 96/99 | 36/58 | 80/72 | 27/51 | 19/34 | 54/44 | 25/44 | 96/100 | 33/58 | 35/58 | 19/34 |
| <i>P. mirabilis</i> | 49/14 | 0/7 | 4/7 | 37/43 | 12/43 | 25/20 | 82/64 | 12/3 | 31/12 | 0/9 | 7/29 | –/– | 11/12 | 25/21 | 0/7 |
| <i>Enterobacter</i> sp. | 42/48 | 1/18 | 85/96 | 96/96 | 98/96 | 14/25 | 33/67 | 4/35 | 17/29 | 0/31 | 24/35 | 54/72 | 16/19 | 13/63 | 50/46 |
| <i>Citrobacter</i> | 10/14 | 13/7 | 75/67 | 90/98 | 67/97 | 14/22 | 55/36 | 42/7 | 18/14 | 11/7 | 46/22 | 9/14 | 48/33 | 25/29 | 40/30 |

CA = Community acquired; HA = hospital acquired; AN = amikacin; AUG = amoxicillin/clavulanic acid; AMP = ampicillin; CF = cephalothin; CTX = cefotaxime; CXM = cefuroxime; CIP = ciprofloxacin; SXT = cotrimoxazole; GN = gentamicin; NA = nalidixic acid; NIT = nitrofurantoin; NOR = norfloxacin; PIP = piperacillin; TZP = piperacillin/tazobactam.

Table 3. Percentage of outpatients vs. inpatients with UTI gram-negative pathogens resistant to antibiotics in MH

| Bacteria | CA/HA | Percentage of CA vs. HA urinary bacterial isolates resistant to: | | | | | | | | | | | | | |
|--------------------------|---------|--|-------|---------|---------|-------|---------|-----|-------|------|-------|---------|-------|------|--|
| | | AN | AUG | AMP | CF | CTX | CXM | CIP | SXT | GN | NA | NIT | PIP | TZP | |
| <i>E. coli</i> | 146/268 | 0/5 | 26/26 | 53/63 | 9/17 | 3/8 | 20/18 | 8/7 | 42/37 | 4/4 | 11/20 | 4/3 | 32/52 | 10/8 | |
| <i>K. pneumoniae</i> | 33/97 | 0/0 | 3/20 | 100/100 | 0/7 | 0/0 | 3/4 | 4/0 | 16/12 | 2/4 | 0/0 | 59/100 | 32/30 | 0/0 | |
| <i>P. aeruginosa</i> | 3/10 | 0/0 | – | – | – | – | – | 0/0 | – | 0/30 | – | – | 0/0 | 0/0 | |
| <i>P. mirabilis</i> | 4/2 | 0/0 | 0/50 | 0/33 | 0/0 | 0/0 | 0/0 | 0/0 | 33/45 | 0/0 | 0/0 | 100/100 | 0/0 | 0/0 | |
| <i>A. calcoaceticus</i> | 1/11 | –/0 | –/0 | –/100 | –/55 | –/46 | –/46 | –/0 | –/9 | –/0 | –/100 | –/27 | –/36 | –/0 | |
| <i>Enterobacter</i> spp. | 5/11 | 0/27 | 0/0 | 100/100 | 60/100 | 20/55 | 20/46 | 0/0 | 0/0 | 0/18 | 0/0 | 0/36 | 0/36 | 0/27 | |
| <i>Citrobacter</i> spp. | 4/2 | 0/0 | 0/0 | 100/100 | 100/100 | 0/0 | 100/100 | 0/0 | 0/0 | 0/0 | 0/0 | 0/0 | 0/0 | 0/0 | |

CA = Community acquired; HA = hospital acquired; AN = amikacin; AUG = amoxicillin/clavulanic acid; AMP = ampicillin; CF = cephalothin; CTX = cefotaxime; CXM = cefuroxime; CIP = ciprofloxacin; SXT = cotrimoxazole; GN = gentamicin; NA = nalidixic acid; NIT = nitrofurantoin; PIP = piperacillin; TZP = piperacillin/tazobactam.

Table 4. Percentage of resistant gram-positive uropathogens isolated from outpatients vs. inpatients with UTI in MKH and MH

| Bacteria | CA/HA | Percentage of CA vs. HA uropathogens resistant to: | | | | | | | | |
|-------------------------|--------|--|-------|-------|-------|-------|---------|-------|-------|-------|
| | | AMP | CF | CC | CLOX | SXT | P | VAN | TEIC | NIT |
| MKH | | | | | | | | | | |
| <i>S. agalactiae</i> | 255/28 | 0/0 | – | – | – | – | 0/0 | 0/0 | 0/0 | 0/0 |
| <i>E. faecalis</i> | 95/59 | 2/26 | – | – | – | – | 26/30 | 0/0 | 0/0 | 1/0 |
| <i>S. aureus</i> | 32/11 | – | 14/18 | 12/33 | 0/18 | 7/36 | 98/99 | 0/0 | 0/0 | 0/0 |
| <i>S. epidermidis</i> | 25/8 | – | 13/19 | 20/25 | 52/66 | 37/41 | 89/78 | 0/0 | 0/0 | 25/16 |
| <i>S. haemolyticus</i> | 21/5 | – | 33/80 | 19/40 | 14/20 | 44/33 | 76/80 | 38/40 | 47/60 | 17/21 |
| <i>S. saprophyticus</i> | 74/26 | – | 46/50 | 0/0 | 46/50 | 8/50 | 85/100 | 0/0 | 0/0 | 15/0 |
| MH | | | | | | | | | | |
| <i>S. agalactiae</i> | 38/126 | 0/0 | 0/0 | 16/4 | –/– | 27/35 | 0/0 | 0/0 | 0/0 | 0/0 |
| <i>E. faecalis</i> | 8/33 | 0/13 | –/– | –/– | –/– | 33/55 | 13/91 | 0/0 | 0/0 | 38/49 |
| <i>S. aureus</i> | 2/4 | 100/100 | 0/0 | 0/0 | 0/0 | 0/25 | 100/100 | 0/0 | 0/0 | 0/0 |
| <i>S. saprophyticus</i> | 6/0 | 100/– | 0/– | 0/– | 0/– | 100/– | 100/– | 0/– | 0/– | 0/– |

AMP = Ampicillin; CF = cephalothin; CC = clindamycin; CLOX = cloxacillin; SXT = cotrimoxazole; P = penicillin; VAN = vancomycin; TEIC = teicoplanin; NIT = nitrofurantoin.

Ciprofloxacin activity against *E. coli* was fairly good; 8 and 7% of the CA-UTI and HA-UTI isolates, respectively, were resistant. *P. aeruginosa* isolates were very sensitive; none was resistant to amikacin, ciprofloxacin and piperacillin.

The resistance rates of the gram-positive bacterial isolates to the commonly tested antibiotics in MKH are shown in table 4. *E. faecalis* had high resistance rates of 26 and 30% in the CA-UTI and HA-UTI cases, respectively, to penicillin. The CNS exhibited high level of resistance to cephalothin, clindamycin, cloxacillin and penicillin.

As shown in table 4, none of the gram-positive bacteria isolated from UTI cases at MH were resistant to vancomycin or teicoplanin. All the GBS were sensitive to penicillin, and the entire *E. faecalis* isolates were sensitive to the glycopeptides. Cloxacillin resistance in *S. aureus* was observed more often in MKH than in MH. None of the *S. aureus* isolates from MH was resistant to cloxacillin. The majority of both CA-UTI and HA-UTI isolates of *Staphylococcus epidermidis* exhibited high resistance rates to ampicillin (80 and 93%), cephalothin (60 and 59%), cefuroxime (40 and 52%), clindamycin (60 and 59%) and SXT (40 and 60%).

Discussion

Our study indicates that *E. coli* is still the most common cause of UTI in the community and hospital settings in Kuwait. Although this finding is similar to a previous study in Kuwait by Helin and Araj in 1986 [11], there is a difference in its prevalence rate, which indicates a decline in the proportion of patients with *E. coli* UTI. Findings similar to ours have been reported in Poland [14], India [15], North America [8, 16], Italy [17], Norway [18] and UK [19]. However, some investigators have shown that the percentage of *E. coli* as a causative agent of UTI is slowly declining, being replaced by other members of the Enterobacteriaceae and enterococci [20]. In addition, the etiological profile of UTI in the present study shows considerable shift from a previous study, similar to ours, reported from Kuwait in 1986 [11]. In this study, *Klebsiella/Enterobacter* species and *Pseudomonas* species were second (18%) and third (10%), respectively, in the order of ranking. However, in our study, *Candida* spp., including *C. albicans*, were the second most common cause of HA-UTI among MKH patients and both HA-UTI and CA-UTI patients of MH. *S. agalactiae* (GBS) was third in the order of ranking in the same settings in both hos-

pitals. These findings may signal a gradual departure from published records of prevalent etiological agents of UTI. The high prevalence of yeast as a cause of UTI may be related to the type of patients, advances in surgical, medical management of the patients as well as excessive use of broad-spectrum antibiotics, high prevalence of diabetes mellitus and obesity in the Kuwaiti population. Other studies have also reported increasing prevalence of UTI caused by *Candida* spp. A previous report from India [15] indicated that *Candida* species were the fourth common cause of UTI in their institution. This is supported by the findings in the 2001 ESCMID Study Group Report on a European perspective study on nosocomial UTI, where *Candida* spp. was the third most commonly isolated micro-organisms from 522 patients [21].

Although CNS and enterococci are common causes of UTI in hospitalized patients in some centers [14, 16, 18], they were not prominent causative agents in our study. Enterococci, in this study, are the fifth commonest etiological agent in hospitalized patients in both hospitals, while CNS was the least common agent. The prevalence rate of enterococci in Kuwait remains unchanged almost two decades after the observation published earlier [11] which showed that enterococci ranked fifth among the pathogens that caused UTI. *S. saprophyticus* was not a prominent member of the CNS causing UTI in our study; this is in contrast to a report by Nicolle et al. [22] where 95% of all CNS cultured from urine of symptomatic female outpatients were *S. saprophyticus*.

The high prevalence rate of GBS in CA-UTI and HA-UTI at MH and community setting of MKH is an interesting finding worthy of note. Although the ESCMID Study Group Report [21] demonstrated a 1.6% prevalence rate of GBS in UTI among patients in the non-EU countries, there is scarcely any other report that has published such high prevalence rate of GBS in UTI as observed in our study. The explanation for this observation may be related to the high prevalence (25%) of diabetes mellitus among the Kuwaiti population. In a previous study, 36% of patients with UTI had diabetes mellitus [23] and this disease is a known predisposing factor for GBS infection in adults. Besides, previous experience shows that there is high vaginal colonization rate of pregnant and non-pregnant individuals by GBS in Kuwait [24], and this observation correlates with high prevalence of GBS in UTI [unpubl. obs.].

We are well aware of difficulties in comparing the susceptibility results where different sensitivity methods have been used in various studies. However, we will nonetheless compare our results with those published in other

parts of the world because the methods employed in this study, Vitek and Kirby-Bauer, correlate well, with the recommended methods. The data presented in this report highlight the problem of bacterial resistance among uropathogens in both hospitalized and community patients with UTI, and is consistent with reports from other centres [15].

In the present study, the high resistance rate of *E. coli* and other Enterobacteriaceae to ampicillin is alarming. For example, almost three quarters and two thirds of *E. coli* from hospitalized patients in MKH and MH, respectively, were resistant to ampicillin. This is much higher than the findings reported by other researchers in Italy (36%) [17], UK (23, 44 and 50%) [19, 25], USA (43 and 43%) [8, 16], Canada (33%) [8] and Norway (25%) [18]. However, a resistance rate of 87%, which is higher than ours, has been reported in India [15].

The Enterobacteriaceae, particularly among the hospitalized patients, in our hospitals exhibited relatively higher resistance rates in general to amoxicillin-clavulanic acid and SXT than those that have been reported elsewhere [16]. In our study and another one [18], nitrofurantoin and amikacin remained very active drugs against most *E. coli* isolates.

As reported in a previous study from Kuwait [11], there is an increase in the resistance rates of *E. coli* against some antibiotics like ampicillin, gentamicin and nalidixic acid. In general, the level of resistance by *E. coli* and non-*E. coli* uropathogens against SXT, a drug that is recommended as first-line therapy by the IDSA treatment guidelines [26], is discomfoting and cannot be recommended for empirical use in UTI in our country. Resis-

tance to fluoroquinolone (ciprofloxacin) in this study is within an acceptable range (<8%) for the most common uropathogens in both hospitals. The alternative drugs that have been recommended by the IDSA for empirical treatment of patients with uncomplicated UTI include fluoroquinolones and nitrofurantoin. The results of our study therefore support the use of either of these drugs as a reasonable choice for empiric therapy in uncomplicated UTI, particularly in the community setting.

In contrast to SXT and ampicillin, nitrofurantoin demonstrated the best and most consistent activity against gram-positive isolates in both hospitals. This should reinforce the recommendation for its use in UTI.

Conclusion

Apart from *E. coli*, the spectrum of the etiological agents of UTIs in MKH and MH and the order of frequency are different when compared with other geographical regions of the world. The bacteria causing UTIs in these hospitals are highly resistant to some of the antibiotics recommended for empiric use in the therapy of UTI in the western countries, but nitrofurantoin and fluoroquinolones remain relatively active against most uropathogens.

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