

Antibiotic resistance of *Enterococcus faecalis* and *Enterococcus faecium* isolated from urinary tract infections in Zagazig University hospitals

Fathy M. Serry, Eman M. Elmasry, Wael A.-H. Heagazy, Safaa A. Abdel-Karim

Department of Microbiology and Immunology, Faculty of Pharmacy, Zagazig University, Egypt

ABSTRACT

This study aims to investigate the resistance profile of enterococci to commonly used antibiotics to provide correct treatment of urinary tract infections and to stop the continual emergence of highly resistant species of bacteria. Three hundred and twenty five (325) urine samples were collected from patients in Urinary Tract (UT) Department and Outpatient urinary tract clinic of Zagazig University Hospitals in Zagazig city, Sharkia, Egypt. One hundred and twenty seven enterococcal isolates (39.1%) were isolated from urine samples. Seventy (55.1%) of enterococcal isolates were *Enterococcus faecalis* and fifty seven (44.9%) of enterococcal isolates were *Enterococcus faecium*. All the isolates are identified and the sensitivity to a number of antibiotics was determined by disc diffusion method.

Using standard breakpoint sensitivity values: the highest percentages of resistance of enterococcal isolates were found for penicillin G, rifampin, erythromycin, doxycycline, ampicillin, lincomycin and amoxicillin. The moderate percentages of resistance were found for ciprofloxacin, azithromycin, clarithromycin and spiramycin, gentamicin and clindamycin, whereas the lowest percentages of resistance were seen for and chloramphenicol, vancomycin, teicoplanin, sulphamethoxazole/trimethoprim and imipenem.

These values are seemed to be higher than wide world values. This is probably due to the variation in the bacterial sensitivity pattern over time and between different geographical districts, misusing of antibiotics and not continuing the antibiotic therapy for sufficient period of time. In conclusion, this high resistance rate represents a dangerous alarm that necessitates the search for new therapeutic options.

INTRODUCTION

Enterococci are gram-positive, catalase-negative, non-spore forming, facultative anaerobic bacteria, which usually inhabit the alimentary tract of humans in addition to being isolated from environmental and animal sources (Fisher and Phillips, 2009).

For many past years, it was believed that enterococci were harmless to humans and unimportant medically. Since the 1980s, enterococci have been identified as an important cause of nosocomial infections, generally ranking as the third or fourth most prevalent genus among nosocomial pathogens (Hoberman and Wald, 1997; Delanghe *et al.*,

2000). In the last decade, enterococci have been reported as a cause of urinary tract infections which are one of the most common infectious diseases (Fisher and Phillips, 2009). Nearly 10% of people will experience a UTI during their lifetime (Hoberman and Wald, 1997; Delanghe *et al.*, 2000). Eighty-five to 90% of enterococcal infections are due to *Enterococcus faecalis* and c. 10% to *Enterococcus faecium* (Leclercq, 2009).

Antibiotic resistance may vary among different bacterial species, but it is created by only few mechanisms: (i) Antibiotic inactivation – direct inactivation of the active antibiotic molecule (Wright, 2005); (ii)

Target modification – alteration of the sensitivity to the antibiotic by modification of the target (Lambert, 2005); (iii) Efflux pumps and outer membrane (OM) permeability changes – reduction of the concentration of drug without modification of the compound itself (Kumar and Schweizer, 2005); or (iv) Target bypass – some bacteria become refractory to specific antibiotics by bypassing the inactivation of a given enzyme. There is an amazing diversity of antibiotic resistance mechanisms within each of these four categories and a single bacterial strain may possess several types of resistance mechanisms. Which of these mechanisms prevails depends on the nature of the antibiotic, its target site, and the bacterial species and whether it is mediated by a resistance plasmid or by a chromosomal mutation (Dzidic *et al.*, 2008).

The dramatic increase in antibiotic resistance of Enterococcus species worldwide highlights the need for a greater understanding of this genus, including its resistance (Fisher and Phillips, 2009). Treatment of UTI is based on information determined from the antimicrobial resistance pattern of the urinary pathogens which includes enterococci (Farajnia *et al.*, 2009). Area-specific monitoring studies aimed to gain knowledge about the type of pathogens responsible for UTIs must be performed and the knowledge of bacterial resistance patterns may help the clinicians to choose the correct empirical drug useful for the treatment of such nosocomial infections (Hryniewicz *et al.*, 2001).

The aim of this study was the investigation of the resistance profile of enterococci to commonly used antibiotics in patients from the Zagazig university hospitals to provide correct treatment of urinary tract infections and to stop the continual emergence of highly resistant strains of bacteria.

MATERIALS and METHODS:

Bacterial isolates

Urine samples from 325 patients of Urinary Tract Department and outpatient clinic of Urinary Tracts of Zagazig University Hospitals were collected in the period from March 2013 to January 2014. Only one specimen per patient was collected. Samples were collected from clean-catch midstream fresh urine in sterile plastic jars. Samples were immediately transported to the microbiological laboratory at Faculty of Pharmacy, where they were immediately processed according to Winn and Koneman (2006).

Urine samples were centrifuged at 10,000 rpm for 15 minutes, supernatants were discarded and sediments were spread over the surface of tryptone soya agar (Lab M, Limited Lancashire, United Kingdom), blood Agar and m-enterococcus agar (Oxoid, Hampshire, England) plates. The plates were incubated at 37°C for 18 hours, and for 48 hours in negative cases. A specimen was considered positive for UTI if leukocytes per high-power field were observed on microscopic examination of the urine.

Identification of enterococci

Bacterial isolates were picked from agar plates and presumptively identified based on colony morphology, biochemical characters on cultured media and microscopic examination of gram stained films according to standard microbiological techniques (Winn and Koneman, 2006).

Bacterial identification was based on the corresponding laboratory tests: gram stain, catalase test, and growth in 6.5 % sodium chloride broth, growth at 45°C and 10°C, growth and hemolysis pattern on blood agar, growth on Mac-conkey agar (Oxoid, Hampshire, England). The identification of the species of enterococci was achieved by arabinose fermentation broth test which gives

positive result with *Enterococcus faecium* (yellow color due to arabinose fermentation) and gives negative result with *Enterococcus faecalis* (red color).

Susceptibility testing

Antimicrobial susceptibility of isolates to different antimicrobials was tested by the disk diffusion method according to the Clinical Laboratory Standards Institute (CLSI, 2012) guidelines, using Mueller Hinton agar medium (Lab M, Limited Lancashire, United Kingdom). Four separate colonies of each isolate were transferred to 5 ml of Mueller Hinton broth (Lab M, Limited Lancashire, United Kingdom) and incubated at 37°C to turbidity approximately equivalent to 0.5 McFarland turbidity standards. The broth cultures were shaken well, further diluted 1: 200 in broth to obtain inoculum density between 10⁵ and 10⁶ CFU/mL. A sterile cotton swab was dipped into the bacterial suspension and the excess liquid was removed by rotating the swab several times against the inside wall of the tube above the fluid level. The surface of a dried Mueller Hinton agar plate was streaked with inoculating swab in different directions. The inoculated plates were left on a flat level surface undisturbed for 3-5 minutes. The antibiotic disks were placed on the inoculated plates by using fine pointed forceps and lightly pressed into the agar with the forceps. The antibiotic disks used were Penicillin (P, 10µg), Amoxicillin (AX, 20µg), Ampicillin (AMP, 10µg), Ciprofloxacin (CIP, 5µg), Gentamicin (CN, 10µg), Imipenem (IPM, 10µg), Sulfamethoxazole/ Trimethoprim (SXT, 1.25/23.27µg), Vancomycin (VA, 30µg), Teicoplanin (TEC, 30µg), Doxycycline (DO, 30µg), Clarithromycin (CLR, 15µg), Azithromycin (AZM, 15µg),

Erythromycin (E, 15µg), Clindamycin (DA, 2µg), Lincomycin (L, 2µg), Spiramycin (SP, 100µg), rifampin (RA, 5µg) and Chloramphenicol (C, 30µg) and were supplied from Oxoid, Hampshire, England. The disks were arranged at 15 mm from edge of the Petri dish and 30 mm from each other. The plates were incubated inverted at 37°C for 18 hr. Plates were examined and diameters of the complete inhibition zones were measured in mm, and interpreted as sensitive (S), intermediate (I) and resistant (R) according to CLSI (2013).

RESULTS:

Isolation and identification of bacteria

Out of the 325 collected urine samples, 127 (39.1%) of patients had significant bacteriuria with enterococci. Out of 127 clinical enterococcal isolates, 70 (55.1%) were identified as *Enterococcus faecalis* whereas, 57 (44.9%) isolates were identified as *Enterococcus faecium*. The isolates were catalase negative, grow at 10°C and 45°C, and grow in 6.5% NaCl broth, show alpha or Gamma hemolysis on blood agar and lactose fermentor on Mac-Conkey agar. *Enterococcus faecium* ferment arabinose sugar producing yellow color while *Enterococcus faecalis* doesn't ferment arabinose.

Antimicrobial susceptibility

The rates of resistance of isolated *Enterococcus faecium* and *Enterococcus faecalis* to a panel of antibiotics, including penicillins, carbapenems, glycopeptides, aminoglycosides, macrolides, phenicols, anasamycins, tetracyclines and quinolones are shown in table (1).

Table (1): Antibiotic resistance pattern of *Enterococcus faecium* isolates and *Enterococcus faecalis* to various antimicrobial chemotherapeutic agents

Antibiotic	Number and percentage of resistant isolates					
	<i>Enterococcus faecium</i> n (57)		<i>Enterococcus faecalis</i> n (70)		Total <i>enterococci</i> n (127)	
	N	%	N	%	N	%
Penicillin G	49	86.0	68	97.1	117	92.1
Amoxicillin	46	80.7	32	45.7	78	61.4
Ampicillin	39	68.4	49	70.0	88	69.3
Vancomycin	9	15.8	4	5.7	13	10.2
Teicoplanin	9	15.8	4	5.7	13	10.2
Imipenem	14	24.6	9	12.9	23	18.1
Erythromycin	35	61.4	55	78.6	90	70.9
Azithromycin	27	47.4	41	58.6	68	53.5
Clarithromycin	26	45.6	39	55.7	65	51.2
Spiramycin	25	43.9	40	57.1	65	51.2
Lincomycin	37	64.9	50	71.4	87	68.5
Clindamycin	19	33.3	34	48.6	53	41.7
Chloramphenicol	13	22.8	25	35.7	38	29.9
Gentamicin	25	43.9	31	44.3	56	44.1
Rifampin	48	84.2	58	82.9	106	83.5
Sulphamethoxazole/trimethoprim	4	7.0	15	21.4	19	15.0
Doxycycline	37	64.9	52	74.3	89	70.0
Ciprofloxacin	33	57.9	38	54.3	71	55.9

DISCUSSION:

In our study, enterococcal isolates obtained from 325 urine specimens were 127 (39.1%) isolates including 70 (21.5%) *Enterococcus faecalis* isolates and 57 (17.5%) *Enterococcus faecium* isolates of total collected urine specimens. This prevalence rate of enterococcal isolates (39.1%) is higher than that of Karlowsky *et al.* (2011) and Swaminathan and Alangaden (2010) who reported enterococcal rates of (10%) and (15%) respectively. This current rate of enterococcal isolates is lower than that of Andrews *et al.* (1999) and Arias *et al.* (2003) who reported prevalence rates of (60%) and

(42.8%) respectively.

The present prevalence rate of *Enterococcus faecalis* isolates (21.5%) and *Enterococcus faecium* isolates (17.5%) differs from that of Arias *et al.* (2003) and Swaminathan and Alangaden (2010) who reported *E. faecalis* rates of (33%) and (4%) respectively and *E. faecium* rates of (22%) and (6%) respectively. The current prevalence rate of *E. faecalis* isolates (21.5%) is lower than that of Andrews *et al.* (1999) and Desai *et al.* (2001) who reported *E. faecalis* prevalence rates of (90.6%) and (40.74%) respectively.

In this study, Out of 127 enterococcal isolates, 67 isolates (52.8%) were considered

as multidrug resistant (MDR) as they were resistant to at least three different classes of antimicrobials according to Magiorakos *et al.* (2012). Forty MDR isolates were resistant to 3 classes of antimicrobials, 20 isolates were resistant to 4 classes of antimicrobials, 10 isolates were resistant to 5 classes of antimicrobials and one isolate was resistant to 6 classes of antimicrobials. The present rate of MDR isolates (52.8%) is lower than that of Hasan *et al.* (2007) who reported MDR rate of (77.8%).

In our study, the highest percentages of resistance of enterococcal isolates were found for penicillin G (92.1%), rifampin (83.5%), erythromycin (70.9%), doxycycline (70.0%), ampicillin (69.3%), lincomycin (68.5%) and amoxicillin (61.4%). The moderate percentages of resistance were found for ciprofloxacin (55.9%), azithromycin (53.5%), clarithromycin and spiramycin (51.2%), gentamicin (44.1%), clindamycin (41.7%), and chloramphenicol (29.9%) whereas the lowest percentages of resistance were seen for vancomycin and teicoplanin (10.2%), sulphamethoxazole / trimethoprim (15%) and imipenem (18.1%).

The present study have shown lower resistance rate of enterococcal isolates to vancomycin (10.2%) than that of Mylotte and Tayara (2000) and Pourakbari *et al.* (2012) who reported resistance rates of (25.8%) and (20.8%) respectively. Our rate (10.2%) is higher than that of McDonald *et al.* (2004) and Yakar *et al.* (2010) who reported resistance rates of (2%) and (0%) respectively.

Our study have shown higher resistance rate of enterococcal isolates to teicoplanin (10.2%) than that of Giacometti *et al.* (2000), Arias *et al.* (2003) and Yakar *et al.* (2010) who reported resistance rates of (2.1%), (5.9%) and (0%) respectively. Our rate (10.2%) is lower than that of Pourakbari *et al.* (2012) and Sharifi *et al.* (2013) who reported resistance rates of (15.3%) and (18.6%) respectively.

This investigation have shown lower

resistance rate of enterococcal isolates to gentamicin (44.1%) than that of Lee *et al.* (2011) and Pourakbari *et al.* (2012) who reported resistance rates of (45%) and (73.6%) respectively. Our rate (44.1%) is higher than that of Das *et al.* (2006) and Farajnia *et al.* (2009) who reported resistance rates of (14.2%) and (37.5%) respectively.

This study have shown lower resistance rate of enterococcal isolates to ciprofloxacin (55.9%) than that of Hasan *et al.* (2007) and Sharifi *et al.* (2013) who reported resistance rates of (84.2%) and (65.4%) respectively. Our rate (55.9%) is higher than that of Yakar *et al.* (2010) and Lee *et al.* (2011) who reported resistance rates of (50%) and (45%) respectively. The current study have shown higher resistance rate of enterococcal isolates to penicillin G (92.1%) than that of Hasan *et al.* (2007) and Sharifi *et al.* (2013) who reported resistance rates of (84.1%), (86.9%) and (68.6%) respectively. Our rate of penicillin G resistance (92.1%) is lower than that of Hasan *et al.* (2007) who reported resistance rate of (98.8%).

This study have shown lower resistance rate of enterococcal isolates to lincomycin (68.5%) than that of Hasan *et al.* (2007) who reported resistance rate of (86.1%).

Our results have shown lower resistance rate of enterococcal isolates to rifampin (83.5%) than that of Sharifi *et al.* (2013) who reported resistance rate of (86.2%). Our rate (83.5%) is higher than that of Arias *et al.* (2003) who reported resistance rate of (45.4%).

The present study have shown higher resistance rate of enterococcal isolates to amoxicillin (61.4%) than that of Barret *et al.* (1999) who reported resistance rate of (48.3%).

This study have shown higher resistance rate of enterococcal isolates to ampicillin (69.3%) than that of Lee *et al.* (2010) and Sharifi *et al.* (2013) who reported resistance rates of (5%) and (28.2%)

respectively. Our rate (69.3%) is lower than that of Lee *et al.* (2010) and Pourakbari *et al.* (2012) who reported resistance rates of (74.1%) and (52.7%) respectively.

The current study have shown higher resistance rate of enterococcal isolates to chloramphenicol (29.9%) than that of Arias *et al.* (2003) and Zhanel *et al.* (2001) who reported resistance rates of (9.2%) and (4.3%) respectively. Our rate (29.9%) is lower than that of Pourakbari *et al.* (2012) who reported resistance rate of (56%).

Our results have shown lower resistance rates of enterococcal isolates to erythromycin (70.9%) than that of Pourakbari *et al.* (2012) and Sharifi *et al.* (2013) who reported resistance rates of (97.8%) and (73.9%) respectively.

The present study have shown lower resistance rates of enterococcal isolates to imipenem (18.1%) than that of Sharifi *et al.* (2013) and Pourakbari *et al.* (2012) who reported resistance rates of (27.7%) and (56%) respectively. Our rate (18.1%) is higher than that of Giacometti *et al.* (2000) and Yakar *et al.* (2010) who both reported resistance rate of (0%).

Our study have shown higher resistance rate of enterococcal isolates to doxycycline (70%) than that of Zhanel *et al.* (2001) who reported resistance rate of (30.1%). Our rate (70%) is lower than that of Bayram and Balci (2006) who reported resistance rate of (84.1%).

This study have shown lower resistance rate of enterococcal isolates to sulphamethoxazole / trimethoprim (15%) than that of Karlowsky *et al.* (2011) and Pourakbari *et al.* (2012) who reported resistance rates of (84%) and (86.8%) respectively. Our rate of sulphamethoxazole / trimethoprim resistance (15%) is higher than that of Yakar *et al.* (2010) who reported resistance rates of (50%).

The present study have shown lower resistance rate of enterococcal isolates to clindamycin (41.7%) than that of Bayram and Balci (2006) and Pourakbari *et al.* (2012) who

reported resistance rates of (86.4%) and (84.6%) respectively.

This study have shown higher resistance rate of enterococcal isolates to clarithromycin (51.2%) than that of Giacometti *et al.* (2000) who reported resistance rate of (37.5%). Our rate of clarithromycin resistance (51.2%) is lower than that of Lim *et al.* (2002) who reported resistance rate of (85%).

Our results have shown lower resistance rate of enterococcal isolates to azithromycin (53.5%) than that of Lim *et al.* (2002) who reported resistance rate of (90%).

The current study have shown lower resistance rate of enterococcal isolates to spiramycin (51.2%) than that of Lim *et al.* (2002) and Hande *et al.* (2015) who reported resistance rate of (85.5%) and (100%).

Conclusions

The present study shows that the enterococcal resistance to commonly used antibiotics is highly increased which represents a dangerous alarm and considerable therapeutic challenge. This remarked resistance is probably due to the variation in the bacterial sensitivity pattern that varies over time and between different geographical districts, misusing of antibiotics and not continuing the antibiotic therapy for sufficient period of time which interrupts the full eradication of bacterial infection and misusing of antibiotics. Unfortunately, all these causes have increased the epidemics of antimicrobial resistance worldwide and the resistance in some species has developed to the level that no clinically available treatment is effective. So, previously mentioned causes necessitate the search for new therapeutic options to overcome this continual increase in bacterial resistance.

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مقاومة بكتيريا الانتيروكوكس فاشيام والانتيروكوكس فيكالييس للمضادات الحيوية والمعزولة من عدوى المسالك البولية في مستشفيات جامعة الزقازيق

فتحي محمد سري، إيمان محمود المصري، وائل محمد علي ، صفاء عبدالعال عبد الكريم
قسم الأحياء الدقيقة والمناعة، كلية الصيدلة، جامعة الزقازيق، مصر

تهدف هذه الدراسة إلى فحص الصورة العامة لمقاومة بكتيريا الانتيروكوكس للمضادات الحيوية الشائعة الاستخدام لتقديم العلاج الصحيح لعدوى المسالك البولية ووقف الظهور المستمر لأنواع شديدة المقاومة من البكتيريا. تم جمع ثلاثمائة وخمس وعشرون عينة بول من المرضى في قسم المسالك البولية وعيادات المسالك البولية الخارجية بمستشفيات جامعة الزقازيق بمدينة الزقازيق، الشرقية، مصر. تم فصل مائة وسبعة وعشرين عذلة من بكتيريا الانتيروكوكس (بنسبة ٣٩,١%) من عينات البول. سبعة وخمسون عذلة من بكتيريا الانتيروكوكس كانت تنتمي لبكتيريا الانتيروكوكس فاشيام (بنسبة ٤٤,٩%) وسبعون عذلة كانت تنتمي لبكتيريا الانتيروكوكس فيكالييس (بنسبة ٥٥,١%). كل العزلات تم تحديد مقاومتها لمجموعة من الأنواع المختلفة من المضادات الحيوية باستخدام طريقة انتشار المضاد الحيوي من القرص. باستخدام قيم الحساسية القياسية الثابتة: وجد أن أعلى نسب مقاومة لعزلات الانتيروكوكس كانت للمضاد الحيوي بنسلين جى، ريفامبين، اريثروميسين، دوكسيسيكلين، أمبيسلين، لينكومايسين وأموكسيسيلين. وجد أن النسب المعتدلة لمقاومة بكتيريا الانتيروكوكس كانت للمضاد الحيوي سيبروفلوكساسين، أزيثروميسين، كلاريثروميسين وسبيراميسين، جنتاميسين والكلينداميسين، في حين ان أدنى نسبة مئوية لمقاومة بكتيريا الانتيروكوكس كانت للمضاد الحيوي كلورامفينيكول، فانكومايسين، تيكوبلانين، السلفاميثوكسازول / ترايميثوبريم وإيميبينيم.

ويبدو أن هذه القيم لبكتيريا الانتيروكوكس أعلى من قيم المقاومة في جميع أنحاء العالم. وربما يعود ذلك إلى نمط حساسية البكتيريا الذى يختلف مع مرور الوقت وبين المناطق الجغرافية المختلفة. وفي هذا المعدل العالي لمقاومة بكتيريا الانتيروكوكس يمثل إنذار خطير يستدعي البحث عن خيارات علاجية جديدة.