

Comparison of Clinical and Laboratory Standards Institute and European Committee on Antimicrobial Susceptibility Testing Breakpoints for β -Lactams in *Enterobacteriaceae* Producing Extended-Spectrum β -Lactamases and/or Plasmid-Mediated AmpC β -Lactamases

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Background: In 2010, the Clinical and Laboratory Standards Institute (CLSI) revised breakpoints for cephalosporins and carbapenems and indicated that extended-spectrum β -lactamase (ESBL) testing is no longer necessary for *Enterobacteriaceae*. We compared the results of the CLSI 2010 and the European Committee on Antimicrobial Susceptibility Testing (EUCAST) MIC breakpoints for *Enterobacteriaceae* producing ESBL and/or plasmid-mediated AmpC β -lactamase (PABL). **Methods:** A total of 94 well-characterized clinical isolates of *Escherichia coli*, *Klebsiella oxytoca*, *Klebsiella pneumoniae*, *Proteus mirabilis*, *Salmonella* spp., *Shigella* spp., *Citrobacter freundii*, *Enterobacter aerogenes*, *Enterobacter cloacae*, and *Serratia marcescens* were analyzed. Of them, 57 were ESBL producers, 24 were PABL producers, and 13 were ESBL plus PABL co-producers. Broth microdilution MIC tests were performed for cefotaxime, ceftazidime, aztreonam, cefepime, and imipenem.

Results: Among the 94 isolates containing ESBL and/or PABL, the number of isolates that were susceptible to cefotaxime, ceftazidime, aztreonam, cefepime, and imipenem according to the CLSI 2010 vs. the EUCAST breakpoints were 4 (4.3%) vs. 4 (4.3%); 26 (27.7%) vs. 8 (8.5%); 37 (39.4%) vs. 14 (14.9%); 71 (75.5%) vs. 31 (33.0%); and 76 (80.9%) vs. 90 (95.7%), respectively. Of the 18 isolates that were not susceptible to imipenem according to the CLSI 2010 breakpoints, 13 isolates (72.2%) were *P. mirabilis*.

Conclusion: The CLSI 2010 MIC breakpoints without tests to detect ESBL and/or PABL for *Enterobacteriaceae* could be unreliable. Thus, special tests for ESBLs and AmpC β -lactamases are required to detect the resistance mechanisms involved. (**Korean J Clin Microbiol 2011;14:24-29**)

Key Words: CLSI, EUCAST, *Enterobacteriaceae*, Breakpoint

INTRODUCTION

Extended-spectrum β -lactamases (ESBLs) hydrolyze penicillins, cephalosporins (except cephamycins), and aztreonam and which are usually inhibited by clavulanate, sulbactam, and tazobactam [1]. AmpC β -lactamases preferentially hydrolyze cephalosporins (except fourth generation cephalosporins) and resist inhibition by clavulanate, sulbactam, and tazobactam [2]. Since ESBL and plasmid-mediated AmpC β -lactamase (PABL) genes are transmissible, it is important that ESBLs and PABLs be tested for in *Enterobacteriaceae* in hospital and long-term care facility

patient population where ESBLs and PABLs are encountered [3].

The Clinical and Laboratory Standards Institute (CLSI) recommended by 2009, as follows. Some strains of *Klebsiella* spp. and *Escherichia coli* producing ESBLs will show MICs above the normal susceptible population but below the standard breakpoints for certain extended-spectrum cephalosporins or aztreonam. Such strains should be screened for potential ESBL production by using ESBL tests. For all confirmed ESBL-producing strains, the test interpretation should be reported as resistant all penicillins, cephalosporins, and aztreonam [4]. However, in January and June 2010, CLSI revised breakpoints of some parenteral cephalosporins (e.g., cefazolin, cefotaxime, ceftriaxone, ceftazidime, and ceftizoxime), aztreonam, and carbapenems (e.g., doripenem, ertapenem, imipenem, and meropenem) for *Enterobacteriaceae*. When using the new interpretive criteria, routine ESBL testing is no longer necessary before reporting results. It is no longer necessary to edit results for cephalosporins, aztreonam, or penicillins from susceptible to resistant [5,6]. European Committee on Antimicrobial Suscepti-

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bility Testing (EUCAST) breakpoints for *Enterobacteriaceae* differ from CLSI, but the recommendation of EUCAST on ESBL detection is similar to the CLSI 2010 guidelines. The purpose of this study was to compare the results of CLSI 2010 and EUCAST MIC breakpoints for *Enterobacteriaceae* producing ESBL and/or PABL in Korea. Eventually, the necessity and availability of the ESBL and PABL detection tests were also analyzed.

MATERIALS AND METHODS

1. Bacterial strains

A total of 94 well-characterized clinical isolates of *Escherichia coli* (n=24), *Klebsiella oxytoca* (n=4), *Klebsiella pneumoniae* (n=24), *Proteus mirabilis* (n=17), *Salmonella* spp. (n=2), *Shigella* spp. (n=3), *Citrobacter freundii* (n=4), *Enterobacter aerogenes* (n=4), *Enterobacter cloacae* (n=8), and *Serratia marcescens* (n=4) were analyzed: 57 were ESBL producers, 24 were PABL producers, and 13 were ESBL plus PABL co-producers.

Seventy-five isolates had been previously characterized by appropriate biochemical, phenotypic, and molecular procedures to determine their types of β -lactamase production [7-12]. Six isolates including an TEM-8-producing *E. coli*, an SHV-2-producing *K. pneumoniae*, two TEM-52-producing *P. mirabilis*, and two CTX-M-14-producing *P. mirabilis* were obtained from Dr. Kyungwon Lee (Yonsei University College of Medicine, Seoul, Korea). The remaining 13 *P. mirabilis* collected from in 12 hospitals in Korea during 2007 were included in this study. Searches for genes coding for the ESBLs and PABLs were performed by PCR amplification and direct sequencing described previously [13-15].

2. Broth microdilution MIC testing

Mueller-Hinton Broth media containing twofold dilutions of cefotaxime, ceftazidime, aztreonam, cefepime, and imipenem at concentration ranging from 0.25 to 512 μ g/mL, were prepared and placed in 96-well microplate. A bacterial suspension was inoculated into each well, according to the recommendation of CLSI in document M7-A8 [16]. *E. coli* ATCC 25922 was inoculated in each set of tests for quality control. The MIC results were interpreted by old and CLSI 2010 breakpoints [4-6] and EUCAST breakpoints [17] (Table 1).

RESULTS

Among 94 isolates containing ESBL and/or PABL, the number of isolates which were susceptible by CLSI 2010 vs. EUCAST breakpoints against cefotaxime, ceftazidime, aztreonam, cefepime, and imipenem were 4 (4.3%) vs. 4 (4.3%), 26 (27.7%) vs. 8 (8.5%), 37 (39.4%) vs. 14 (14.9%), 71 (75.5%) vs. 31 (33.0%), and 76 (80.9%) vs. 90 (95.7%), respectively. The number of isolates which were resistant by CLSI 2010 vs. EUCAST against cefotaxime, ceftazidime, aztreonam, cefepime, and imipenem were 89 (94.7%) vs. 89 (94.7%), 62 (66.0%) vs. 62 (66.0%), 42

Table 1. MIC interpretive standards of CLSI and EUCAST in *Enterobacteriaceae*

Antimicrobial agent	MIC interpretive standard (μ g/mL): susceptible/intermediate/resistant		
	CLSI		EUCAST 2010
	2009	2010	
Cefazolin	$\leq 8/16/\geq 32$	$\leq 1/2/\geq 4$	–
Cefotaxime	$\leq 8/16 \sim 32/\geq 64$	$\leq 1/2/\geq 4$	$\leq 1/2/\geq 4$
Ceftriaxone	$\leq 8/16 \sim 32/\geq 64$	$\leq 1/2/\geq 4$	$\leq 1/2/\geq 4$
Ceftizoxime	$\leq 8/16 \sim 32/\geq 64$	$\leq 1/2/\geq 4$	–
Ceftazidime	$\leq 8/16/\geq 32$	$\leq 4/8/\geq 16$	$\leq 2/4 \sim 8/\geq 16$
Aztreonam	$\leq 8/16/\geq 32$	$\leq 4/8/\geq 16$	$\leq 1/2 \sim 8/\geq 16$
Cefepime	$\leq 8/16/\geq 32$	$\leq 8/16/\geq 32$	$\leq 1/2 \sim 8/\geq 16$
Doripenem	–	$\leq 1/2/\geq 4$	$\leq 1/2/\geq 4$
Ertapenem	$\leq 2/4/\geq 8$	$\leq 0.25/0.5/\geq 1$	$\leq 0.5/1/\geq 2$
Imipenem	$\leq 4/8/\geq 16$	$\leq 1/2/\geq 4$	$\leq 2/4 \sim 8/\geq 16$
Meropenem	$\leq 4/8/\geq 16$	$\leq 1/2/\geq 4$	$\leq 2/4 \sim 8/\geq 16$

(44.7%) vs. 42 (44.7%), 13 (13.8%) vs. 23 (24.5%), and 4 (4.3%) vs. 0 (0%), respectively.

Of the 18 isolates which were non-susceptible by CLSI 2010 breakpoints against imipenem, 13 isolates (77.2%: 11 isolates were intermediate and two isolates were resistant) were *P. mirabilis*. The remaining five imipenem-non-susceptible isolates, three isolates (*E. coli* co-producing CTX-M-15 plus DHA-1, *K. pneumoniae* producing GES-5, and *E. aerogenes* producing TEM-52) were imipenem-intermediate and two isolates (*K. oxytoca* co-producing SHV-12 plus DHA-1 and *E. aerogenes* producing CTX-M-14) were imipenem-resistant (Table 2).

DISCUSSION

According to the CLSI, when using the CLSI 2010 breakpoints, it is not necessary to perform ESBL screen and confirmatory tests when reporting results to guide management of patients' therapy [5]. It now recommended that these results be reported without changing the cephalosporin susceptible result to resistant because studies indicate that MIC is the best predictor of treatment outcome of infections caused by β -lactamase-producing *Enterobacteriaceae*. This study showed that only four (4.3%) of the 94 isolates producing ESBL and/or PABL were susceptible to cefotaxime using the CLSI 2010 (or EUCAST) breakpoints. The data suggest that almost all *Enterobacteriaceae* harboring ESBLs and/or PABLs will be detected using the CLSI 2010 (or EUCAST) cefotaxime susceptible breakpoint, because they will test as intermediate or resistant to the agent. However, many of the isolates producing ESBLs and/or PABLs were susceptible to ceftazidime (26 isolates, 27.7%), aztreonam (37 isolates, 39.4%), and cefepime (71 isolates, 75.5%) by using the CLSI 2010 breakpoints. The isolates of susceptible to ceftazidime, aztreonam, and cefepime by using the CLSI 2010 breakpoints were more than that by using the EUCAST breakpoints. Especially, too many isolates producing ESBLs and/or PABLs showed susceptible to cefe-

Table 2. Results of antimicrobial susceptibility testing by the CLSI 2010 and the EUCAST interpretation criteria for isolates containing ESBL and/or plasmid-mediated AmpC β -lactamase

Organism	β -Lactamase (No. of isolates)	Reference	No. of isolates susceptible (resistant) by CLSI/EUCAS				
			CTX	CAZ	ATM	FEP	IPM
<i>E. coli</i>	SHV-12 (2)	7	0/0 (2/2)	0/0 (2/2)	0/0 (2/2)	2/1 (0/0)	2/2 (0/0)
	TEM-8 (1)	K. Lee	1/1 (0/0)	0/0 (1/1)	1/0 (0/0)	1/1 (0/0)	1/1 (0/0)
	CTX-M-3 (2)	7	0/0 (2/2)	1/0 (0/0)	0/0 (1/1)	0/0 (2/2)	2/2 (0/0)
	CTX-M-14 (4)	7	0/0 (4/4)	4/1 (0/0)	1/0 (0/0)	1/0 (0/3)	4/4 (0/0)
	CTX-M-15 (1)	7	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	1/1 (0/0)
	DHA-1 (2)	7	0/0 (2/2)	0/0 (2/2)	1/0 (1/1)	2/2 (0/0)	2/2 (0/0)
	CMY-1 (3)	7	0/0 (3/3)	1/0 (2/2)	1/0 (2/2)	3/1 (0/0)	3/3 (0/0)
	CMY-2 (2)	7	0/0 (2/2)	0/0 (2/2)	0/0 (1/1)	2/2 (0/0)	2/2 (0/0)
	SHV-12 plus DHA-1 (1)	8	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	1/1 (0/0)	1/1 (0/0)
	CTX-M-14 plus DHA-1 (2)	8	0/0 (2/2)	2/0 (0/0)	2/0 (0/0)	2/0 (0/0)	2/2 (0/0)
	CTX-M-14 plus CMY-2 (1)	8	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	1/1 (0/0)
	CTX-M-15 plus DHA-1 (2)	8	0/0 (2/2)	0/0 (1/1)	0/0 (2/2)	0/0 (1/2)	1/2 (0/0)
CTX-M-15 plus CMY-10 (1)	8	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	1/1 (0/0)	
Subtotal (24)			1/1 (23/23)	8/1 (14/14)	6/0 (13/13)	14/8 (6/10)	23/24 (0/0)
<i>K. oxytoca</i>	DHA-1 (3)	9	1/1 (1/1)	0/0 (1/1)	2/2 (0/0)	3/3 (0/0)	3/3 (0/0)
	SHV-12 plus DHA-1 (1)	9	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	0/0 (1/0)
	Subtotal (4)		1/1 (2/2)	0/0 (2/2)	2/2 (1/1)	3/3 (1/1)	3/3 (1/0)
<i>K. pneumoniae</i>	SHV-2 (1)	K. Lee	0/0 (1/1)	0/0 (1/1)	0/0 (0/0)	1/0 (0/0)	1/1 (0/0)
	SHV-2a (2)	9	0/0 (2/2)	0/0 (2/2)	0/0 (1/1)	2/0 (0/0)	2/2 (0/0)
	SHV-5 (1)	9	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	1/1 (0/0)	1/1 (0/0)
	SHV-12 (2)	9	0/0 (2/2)	1/0 (1/1)	0/0 (1/1)	1/0 (0/1)	2/2 (0/0)
	TEM-52 (1)	9	0/0 (1/1)	0/0 (1/1)	1/0 (0/0)	1/0 (0/0)	1/1 (0/0)
	CTX-M-9 (1)	9	0/0 (1/1)	1/0 (0/0)	1/0 (0/0)	1/0 (0/0)	1/1 (0/0)
	CTX-M-14 (3)	9	0/0 (3/3)	3/0 (0/0)	1/0 (0/0)	3/0 (0/0)	3/3 (0/0)
	GES-5 (2)	9	0/0 (2/2)	0/0 (2/2)	0/0 (2/2)	0/0 (1/2)	1/2 (0/0)
	DHA-1 (4)	9	1/1 (3/3)	0/0 (4/4)	2/0 (2/2)	4/4 (0/0)	4/4 (0/0)
	CMY-1 (3)	9	0/0 (3/3)	1/0 (2/2)	3/0 (0/0)	3/2 (0/0)	3/3 (0/0)
	SHV-12 plus DHA-1 (4)	9	0/0 (4/4)	0/0 (4/4)	0/0 (4/4)	3/2 (0/1)	4/4 (0/0)
	Subtotal (24)			1/1 (23/23)	6/0 (18/18)	8/0 (11/11)	20/9 (1/4)
<i>P. mirabilis</i>	TEM-52 (2)	K. Lee	0/0 (2/2)	1/0 (1/1)	2/1 (0/0)	2/1 (0/0)	0/2 (0/0)
	CTX-M-12 (3)	This study	0/0 (3/3)	1/1 (2/2)	2/1 (0/0)	2/0 (1/1)	1/3 (0/0)
	CTX-M-14 (2)	K. Lee	0/0 (2/2)	2/2 (0/0)	2/2 (0/0)	2/0 (0/0)	1/2 (0/0)
	CTX-M-15 (3)	This study	0/0 (3/3)	3/1 (0/0)	3/1 (0/0)	1/0 (1/2)	1/3 (0/0)
	DHA-1 (2)	This study	1/1 (1/1)	1/1 (1/1)	2/2 (0/0)	2/2 (0/0)	0/0 (2/0)
	CMY-2 (4)	This study	0/0 (4/4)	0/0 (2/2)	4/4 (0/0)	4/4 (0/0)	0/4 (0/0)
	SHV-12 plus CTX-M-14 plus DHA-1 (1)	This study	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	0/0 (0/1)	1/1 (0/0)
	Subtotal (17)			1/1 (16/16)	8/5 (7/7)	15/11 (1/1)	13/7 (2/4)
<i>Salmonella</i>	CTX-M-15 (1)	10	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	1/1 (0/0)
	CMY-2 (1)	9	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	1/0 (0/0)	1/1 (0/0)
Subtotal (2)			0/0 (2/2)	0/0 (2/2)	0/0 (2/2)	1/0 (1/1)	2/2 (0/0)
<i>Shigella</i>	CTX-M-14 (3)	11	0/0 (3/3)	3/1 (0/0)	3/0 (0/0)	3/0 (0/0)	3/3 (0/0)
<i>C. freundii</i>	SHV-12 (3)	12	0/0 (3/3)	0/0 (3/3)	0/0 (3/3)	3/2 (0/0)	3/3 (0/0)
	TEM-52 (1)	12	0/0 (1/1)	0/0 (1/1)	1/0 (0/0)	1/0 (0/0)	1/1 (0/0)
Subtotal (4)			0/0 (4/4)	0/0 (4/4)	1/0 (3/3)	4/2 (0/0)	4/4 (0/0)
<i>E. aerogenes</i>	SHV-12 (1)	12	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	1/0 (0/0)	1/1 (0/0)
	TEM-52 (2)	12	0/0 (2/2)	0/0 (2/2)	0/0 (1/1)	2/0 (0/0)	1/2 (0/0)
	CTX-M-14 (1)	12	0/0 (1/1)	1/1 (0/0)	1/1 (0/0)	0/0 (0/1)	0/0 (1/0)
Subtotal (4)			0/0 (4/4)	1/1 (3/3)	1/1 (2/2)	3/0 (0/1)	2/3 (1/0)
<i>E. cloacae</i>	SHV-12 (3)	12	0/0 (3/3)	0/0 (3/3)	0/0 (3/3)	3/0 (0/0)	3/3 (0/0)
	CTX-M-3 (1)	12	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	1/1 (0/0)
	CTX-M-9 (1)	12	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	1/1 (0/0)	1/1 (0/0)
	SHV-12 plus CTX-M-9 (3)	12	0/0 (3/3)	0/0 (3/3)	0/0 (3/3)	2/0 (1/1)	3/3 (0/0)
Subtotal (8)			0/0 (8/8)	0/0 (8/8)	0/0 (8/8)	6/1 (2/2)	8/8 (0/0)
<i>S. marcescens</i>	SHV-12 (1)	12	0/0 (1/1)	0/0 (1/1)	0/0 (1/1)	1/0 (0/0)	1/1 (0/0)
	TEM-52 (3)	12	0/0 (3/3)	0/0 (3/3)	1/0 (0/0)	3/1 (0/0)	3/3 (0/0)
	Subtotal (4)			0/0 (4/4)	0/0 (4/4)	1/0 (1/1)	4/1 (0/0)
Total (94)			4/4 (89/89)	26/8 (62/62)	37/14 (42/42)	71/31 (13/23)	76/90 (4/0)

Abbreviations: CTX, cefotaxime; CAZ, ceftazidime; ATM, aztreonam; FEP, cefepime; IPM, imipenem.

pime by using the CLSI 2010 because the CLSI 2010 breakpoint of cefepime (susceptible MIC criteria using CLSI 2010 vs. EUCAST, $\leq 8 \mu\text{g/mL}$ vs. $\leq 1 \mu\text{g/mL}$) was not lowered.

A number of investigations have reported an association between poor clinical response and serious infections arising from ESBL- or PABL-producing bacteria. Bloodstream infections caused by ESBL-producing strains of *K. pneumoniae* represent a serious clinical problem associated with high mortality rate [18]. When the treatment response was assessed 72 h after antimicrobial therapy, the treatment failure rates were 51.9% in patients with bacteremia due to PABL-producing *K. pneumoniae*. Of the 13 patients with bacteremia due to DHA-1-producing *K. pneumoniae*, nine patients had received imipenem and remaining four patients had received extended-spectrum cephalosporins. All patients who had received extended-spectrum cephalosporins died. Of nine patients had received imipenem, seven were cured [19]. Failure to use an antibiotic against ESBL-producing *K. pneumoniae* was associated extremely high mortality. Use of carbapenem was associated with a significantly lower mortality than was use of other antibiotics (e. g., cephalosporins and β -lactam/ β -lactamase inhibitor combinations) active *in vitro* [20]. In contrast, clinical success was similar between patients with ESBL and non-ESBL-producing isolates. The proportion of successes for patients with infecting isolates manifesting MIC results of 1, 2, 4, and $8 \mu\text{g/mL}$ was 73%, 75%, 33%, and 14%, respectively. These data support the contention that for *Enterobacteriaceae* infection, the MIC value is more predictive outcome than ESBL production [21]. It is still controversial whether it is safe to classify isolates with MIC values below the CLSI 2010 (or EUCAST) clinical breakpoint as susceptible to the drug in question unless a specific ESBL and/or AmpC screening test has been performed. The controversy is difficult to resolve. In Korea, most of the clinical microbiology laboratories have been currently using the CLSI guideline for antimicrobial susceptibility testing. Therefore, the new CLSI MIC breakpoints without tests to detect ESBL and/or AmpC β -lactamase for *Enterobacteriaceae* could be unreliable and dangerous yet. Special tests for ESBLs and/or AmpC β -lactamases are required to detect the resistance mechanisms involved.

In this study, the 13 (76.5%) and 2 (11.8%) of 17 *P. mirabilis* isolates were non-susceptible to imipenem by the CLSI 2010 and EUCAST breakpoint, respectively. *Proteus* and *Morganella* are poor target for imipenem [17]. *P. mirabilis* tend to higher than meropenem and doripenem MICs [5]. However, a lot of discrepancy between the imipenem susceptibility results by using CLSI and EUCAST for *P. mirabilis*, further study is needed. An SHV-12 plus DHA-1 co-producing *K. oxytoca* and an CTX-M-14-producing *E. aerogenes* also showed resistant to imipenem by the CLSI 2010 (each of MICs were $4 \mu\text{g/mL}$). The two isolates showed negative results on modified Hodge test and EDTA-sodium mercaptoacetic acid double-disk synergy test for screening of carbapenemase and metallo- β -lactamase, respectively (data not shown). Porin loss may have reduced susceptibility to imipenem [22,23].

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=국문초록=

Extended-Spectrum β -Lactamase 및 Plasmid-Mediated AmpC β -Lactamases 생성 *Enterobacteriaceae*의 β -Lactam제에 대한 Clinical and Laboratory Standards Institute와 European Committee on Antimicrobial Susceptibility Testing의 감수성 기준 비교

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배경: 2010년에 Clinical and Laboratory Standards Institute (CLSI)에서는 장내세균(*Enterobacteriaceae*)에 대한 cephalosporin 제와 carbapenem제의 감수성 기준을 변경하면서 이제는 extended-spectrum β -lactamase (ESBL) 검사를 하지 않아도 된다고 하였다. 이에 저자들은 ESBL 및 plasmid-mediated AmpC β -lactamase (PABL)를 생성 장내세균을 대상으로 새로운 CLSI 및 European Committee on Antimicrobial Susceptibility Testing (EUCAST)의 MIC 감수성기준을 적용한 결과를 비교하고자 하였다.

방법: 총 94주의 *Escherichia coli*, *Klebsiella oxytoca*, *Klebsiella pneumoniae*, *Proteus mirabilis*, *Salmonella* spp., *Shigella* spp., *Citrobacter freundii*, *Enterobacter aerogenes*, *Enterobacter cloacae*, *Serratia marcescens*를 대상으로 하였고, 57주가 ESBL 생성균주, 24주가 PABL 생성균주, 13주가 ESBL+PABL 동시생성균주였다. 액체배지 미량희석법으로 cefotaxime, ceftazidime, aztreonam, cefepime, imipenem에 대한 MIC를 측정하였다.

결과: 94주의 ESBL 및 PABL 생성균주 중, CLSI 2010 및 EUCAST 기준에 감수성인 균주수는 cefotaxime, ceftazidime, aztreonam, cefepime, imipenem에 대하여 각각 4 (4.3%) 및 4 (4.3%), 26 (27.7%) 및 8 (8.5%), 37 (39.4%) 및 14 (14.9%), 71 (75.5%) 및 31 (33.0%), 76 (80.9%) 및 90주(95.7%)였다. CLSI 2010 기준으로 imipenem 비감수성을 보인 18주 중 13주 (72.2%)가 *P. mirabilis*이었다.

결론: 장내세균에 새로운 CLSI 2010의 MIC 기준을 적용하면서 ESBL 및 PABL 검출을 위한 검사를 하지 않는 것은 유용하지 않을 수 있다. 따라서 ESBL 및 AmpC β -lactamase 등을 검출할 수 있는 검사가 필요할 것으로 생각된다. [대한임상미생물학회지 2011;14:24-29]

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