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A STUDY OF COLONIZATION WITH ESBL AND AMPC E COLI IN GUT OF PATIENTS OF TERTIARY CARE HOSPITAL, AHMEDABAD

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ABSTRACT

Background: Higher prevalence of ESBL (Extended spectrum beta-lacamase)-producing *E. coli* in fecal carriage has been reported in the nosocomial setting than in the community due to high levels of antibiotic consumption. This study is to know the prevalence of ESBL producing *E.coli* in stool samples of Hospitalized patient with non GIT complains.

Material & Methods: A total 300 Stool samples of the patients with non GIT complains admitted in General Hospital, Sola, Ahmadabad were collected for study between December 2012 to March 2013. All samples were tested for routine stool microscopy as well as culture and sensitivity according to NCCLS guideline.

Result: Of 100 samples showing E.coli out of 300 samples, 43% show ESBLs producing E.coli and Amp C producing E.coli show in 26% cases. While 31% was normal E.coli without ESBLs B-lactamase and AmpC B lactamase. Commonest age group is 0-15 years.

Conclusion: Our normal gut flora is highly replaced by the ESBLs/AmpC producing *E.coli*, though they are non pathogenic organism. A threatening epidemiological problem is the dissemination of ESBL-producing organisms to healthy people in the community, which might depend on the frequency of ESBL fecal carriage as well as on the presence of ESBL producing organisms in the food chain.

Key words: ESBL, E.coli, AmpC

INTRODUCTION

Multidrug resistance is increasingly seen in many Gram-negative bacteria as a result of widespread use of various antibiotics.[1,2]. Extended spectrum β-Lactamase (ESBLs) are enzymes that commonly mediate resistance to β-Lactam antimicrobial drugs in Gram-negative bacteria [3] and are most commonly found in Escherichia coli and Klebsiella pneumoniae. [1, 2]

ESBL-producing organisms have been widely reported in many countries. [4] These enzymes are typically plasmid-mediated. .[4] ESBLs are often encoded by genes located on large plasmids and these also carry genes for resistance to other antimicrobial agents such as Aminoglycosides, Trimethoprim, Sulphonamides, Tetracycline, Chloramphenicol and Fluoroquinolones.[4] They are not active against cephamycins or carbapenems, and are highly susceptible in vitro to inhibition by lactamase inhibitors, such as clavulanic acid [5].

Higher prevalence of ESBL-producing *E. coli* in fecal carriage has been reported in the nosocomial setting than in the community. [6] Patients of medical units with high levels of antibiotic consumption have also shown higher rates of ESBL colonization. [7] Patients with community infections and members of their households represent a reservoir for ESBL producers, increasing the dispersal of resistance in healthy people.[7] Rates of colonization by extended-

spectrum-β-Lactamase (ESBL)-producing organisms have increased dramatically worldwide.[8, 9]

The intestinal tract provides an important reservoir for antibiotic-resistant gram-negative bacilli, including Enterobactericae species. [10] Fecal shedding onto patients' skin and environmental surfaces contributes to nosocomial transmission of antibiotic resistant gram-negative pathogens.[10] Finally, the intestinal tract provides an important site for transfer of genes conferring antibiotic resistance. Selective pressure exerted by antibiotics plays a crucial role in the emergence and dissemination of antibiotic- resistant microorganisms.[10]

This study is to know the prevalence of ESBL and AmpC producing *E.coli* in stool samples of hospitalized patient with non GI complains.

MATERIALS AND METHOD

A total 300 Stool samples of the patients with non GIT complains admitted in General Hospital, Sola, Ahmedabad were collected for study between December 2012 to March 2013.

All samples were tested for routine stool microscopy as well as culture on MacConkey agar, [11, 12] XLD agar plates[11, 12] and incubated at 37^0C for 24 hr. Samples those are negative for pus cell, RBC, and show Lactose fermenting pink colony on MacConkey agar and yellow colony on XLD agar was subjected for various biochemical tests like Motility, Indole, Citrate, Methyl red and Vogus Proskauer test. [11, 12, 13]

All E coli which were isolated from normal routine microscopy were subjected for antibiotic sensitivity test was performed by disc diffusion test according to NCCLS guideline.[14] Gram negative panel consisting of Ampicillin (AMP), Ampicillin+SulbactumAS (20/10), Co-trimoxazole (COT) (30ug), Chloramphenicol (CH) (30ug), Ciprofloxacin (CIP) (5ug), Tetracycline (TE) (30ug), Ofloxacin (OF) (5ug), Amikacin (AK) (30ug), Gentamicin (GM) (10ug), Cefoxitin (CX) (30ug), Cefotaxime (CTX) (30ug), Ceftazidime (CAZ) (30ug) and Ceftazidime+Clavulanicacid (CAC) drugs were tested for E.coli. ESBL Producing E.coli was indicated by increase 5 mm zone size with ceftazidime +clavulanic acid than plain Ceftazidime. AmpC B-lactamase E.coli is indicated when E.coli is resistant to B Lactam+ B Lactam inhibitors means resistant to Ceftazidime + Clavulanic acid along with other B-Lactam drugs. [13, 14]

RESULTS

Of the 300 stool samples, 100 samples show normal flora of E coli.(In which microscopy was normal).Among this 100 samples ,43% show ESBLs producing E.coli and Amp C producing E.coli shown in 26% cases. While 31% was normal E.coli without ESBLs B lactamase and AmpC B lactamase. (**Table 1)**

Table-1: Prevalence of ESBL and Ampc in E.coli

Non Pathogenic *E.coli*	100 nos.
ESBL producing *E.coli*	43(43.0%)
Ampc producing *E.coli*	26(26.0%)
Normal *E.coli* (Without ESBL/Ampc producing β lactamase)	31(31.0%)

Table-2: Age wise Distribution among ESBL/ AmpC producing *E.coli*

Age in years	ESBL(n=43)	AmpC(n=26)
0-15	*22* (51)	15(58)
16-30	*05* (12)	04(15)
31-45	05(12)	01(4)
45 above	11(25)	06(23)

Table-3: Resistance to other Anti-microbial in ESBL/AmpC producing E.coli

Antimicrobial	ESBLs E.coli(n=43)	AmpC E.coli(n=26)
COT	35(81)	24(92)
CH	10(23)	11(42)
TE	35(81)	25(96)
CIP	38(83)	25(96)
OF	19(41)	20(77)
GM	25(58)	18(69)
AK	1(2)	2(51)

Among ESBLs E.coli, 75 %(32) male and 25 %(11) were female and AmpC E coli 47 %(12) male and 53 %(14) were female.

ESBL and AmpC β lactamase producing *E.coli* colonization is more found in age group between 0-15 years. **(Table 2)**

ESBL and AmpC β lactamase producing *E.coli* also resistance to other antibiotic as shown in **Table 3**

DISCUSSION

In the study, a total 300 stool samples from the hospitalized patients were processed for detection of ESBLs/ AmpC producing *E.coli.*

E.coli is normal flora of gut as well as it is important pathogenic bacteria causing gastrointestinal infection mainly diarrhea. So stool routine microscopy as well as macroscopic appearance is very important to rule out either sample has pathogenic *E.coli* or non pathogenic *E.coli.*

If stool appearance was normal, OBT negative, No pus cells, No RBC, then it should consider as non pathogenic *E.coli* in stool culture.

In our study we found non pathogenic *E.coli* in 100 samples out of 300 total samples.

Out of 100 samples, 43% were ESBL *E.coli* correlate with Quiang et al 42%[15] and 26% were AmpC producing *E.coli* while 31% were without β lactamase producing *E.coli.* Incidence of β lactamase producing(ESBL/AmpC) *E.coli* 69% which is very well correlated with Arazazu Valvarde et al. study whose result was 70%[16]

ESBL producing *E.coli* was come out in male patient more as compare to female patient compare with Quiang et al 42%[15 and] AmpC *E.coli* is common in female.

ESBL and AmpC β lactamase producing *E.coli* colonization is more found in age group between 0-15 years because more pediatric stool samples are received in our laboratory.

CONCLUSION

Our normal gut flora is highly replaced by the ESBLs/AmpC producing *E.coli,* though they are non pathogenic organism. Higher use of antibiotic is one of the commonest factors for ESBLs/AmpC *E.coli*.

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