Original Research Article

Phenotypic Detection of AmpC beta-lactamase producing *Escherichia coli* among Patients in Hospital Wards

ABSTRACT

Background and Objectives:

AmpC beta-lactamase producing *Escherichia coli* have emerged in hospital environments as important health issue that are of global health concern as drug-resistant pathogenic bacteria that evolve into strains that are resistant to many classes of antibiotics. However, awareness of the prevalence of AmpC β-lactamase producing microorganisms such as *E. coli* could be very valuable for achieving more accurate epidemiological results, as well as controlling their spread in different hospital ward.

Methodology:

A total of four hundred (400) clinical samples comprising of two hundred and seventy-nine (279) urine samples and one hundred and Twenty-one (121) wound samples were collected from patients in different ward at AE-FUTHA. The clinical samples were analyze using standard microbiological culture and identification of *Escherichia coli.* Detection of phenotypic AmpC β-lactamases production was performed using Cefoxitin-Cloxacillin Double-Disk Synergy Test (CC-DDST). Antibiotic susceptibility studies of AmpC β-lactamases producing *Escherichia coli* was determined using the Kirby–Bauer disk diffusion method and the results were construed using the Clinical Laboratory Standard Institute (CLSI) zone diameter breakpoints.

Result: Phenotypic AmpC β-lactamases producing *E. coli* accounted overall prevalence rate of 94.1 %. The frequency of AmpC β-lactamases producing *E. coli* in urine samples were 57(93.4 %) comprising of high occurrence rate in Surgical ward 15(100 %) followed by medical ward 24(88.9 %) but also accounted for overall prevalence rate of 38(95.0 %) in wound samples consisting of 9(100 %) from children ward and medical ward 10(100 %) followed by Orthopedic ward 13(86.7 %) with the least prevalence rate. The AmpC β-lactamases producing *E. coli* exhibited high percentage of resistance within the range of 50-100% against ceftriaxone, ceftazidime, cefepime, azetronam, Trimethoprim-Sulfamethoxazole Ticarcillin-clavulanic acid but were susceptible to ofloxacin 70.0 %, Imipenem 83.3 % and amikacin 100%.

Conclusion:

The findings of this study is a proof of the occurrence of AmpC beta-lactamase producing *Escherichia coli* among patients admitted in hospital ward and there's urgent need for controlling and managing the development of MDR genotype strain. Moreover, to prevent the spread of resistance among AmpC beta-lactamase producing *Escherichia coli* to other strain and improve the effectiveness of antibiotics, it is suggested to establish a precise schedule for antibiotic use in each region based on their antibiotic resistance pattern.

Keyword: *AmpC beta-lactamase producing, Escherichia coli, Patients*

1. INTRODUCTION

AmpC β-lactamase production is one of the mechanisms of resistance to β-lactam antibiotics in Gram negative bacteria such as *Escherichia coli* conferring resistance to a wide variety of β-lactam antibiotics including 7-α-methoxy cephalosporins (cefoxitin or cefotetan), oxyimino cephalosporins (cefotaxime, ceftazidime, ceftriaxone), monobactam (aztreonam) and are not inhibited by clavulanic acid [1]. AmpC-producing *E. coli* have emerged in hospital environments as important health issue that are of global health concern as drug-resistant disease-causing pathogenic bacteria that evolve into strains that are resistant to many classes of antibiotics [2, 3, 4, 5]. In recent time, AmpC β-lactamases producing *E. coli* are considered clinically important bacteria as the may cause diarrhea, extra-intestinal disorders in humans and also confer antimicrobial resistance to the narrow-spectrum, expanded-spectrum and the broad-spectrum cephalosporins and are emerging challenge facing the health sector across the globe

especially in the area of infection control and prevention. Treatment options are severely limited because AmpC is often associated with other multiple resistance genes, such as those resistances to quinolones as well as other β-lactamase genes [1, 6]. The economic cost of antimicrobial resistance goes beyond the morbidity and mortality associated with the medical condition caused by the bacteria harboring multidrug resistant genes; it also includes the loss of efficacy or ineffectiveness of some available antimicrobial drugs [7]. In most developing countries like Nigeria, the detection of AmpC β-lactamase enzymes responsible for the truncated action of antimicrobial agent is still ill-detected in our hospitals because routine antibiogram studies performed in most of our hospitals is almost ineffective in detecting these bacteria strain. Therefore, determining the prevalence of pathogenic *E. coli* possessing AmpC β-lactamases especially among patients admitted in the hospital ward coupled with proper susceptibility testing of these organisms is critical to reducing the possible risks associated with infections due to AmpC β-lactamases.

2. MATERIALS AND METHODS

2.1 Study Area

This study was carried out at Alex Ekwueme - Federal University Teaching Hospital, Abakaliki (AE-FUTHA). AE-FUTHA is in Abakaliki town, the capital city of Ebonyi State. It is a public tertiary health care facility with equipment and capacity to deliver all manner of health services with more than 2,000 beds and intensive care units (ICUs) for children and adults. As a tertiary training institution, AE-FUTHA draws its patients within and outside the state. It is rated a center of excellence for its quality health services delivery. It is located in 6.32^{o}N latitude and 8.12^{o}E and longitude and is situated at an elevation of 117 meters above sea level. Abakaliki is populated and inhabited by indigenes and people from other parts of Nigeria. Ebonyi State shares border with Benue State to the north, Enugu State to the west, Imo and Abia to the south and Cross River to the east. The climate is characterized by a hot dry period which stretches from November-April, while the rainy season is from May-October. The maximum temperature during dry season is 37.6 ^{o}C while the minimum temperature is 27.1 ^{o}C [8]. The major occupations of people in Abakaliki are farming and trading, there are also civil servants and students and all these people engage in a busy life activity.

2.2 Sample collection and Isolation of *Escherichia coli*

A total of four hundred (400) clinical samples comprising of two hundred and seventy-nine (279) urine samples and one hundred and Twenty-one (121) wound samples were collected from patients in different ward at AE-FUTHA. After instruction on procedure to collect urine specimen by a trained hospital personnel about 20 ml of a non-repetitive clean catch morning mid-stream urine samples, were collected from patients using sterile screw-capped, wide-mouth container. Single mini-tip culture swab sticks were used for collection of wound swab samples. All sample containers were labeled with the unique sample number; date and time of collection.

The collected samples were analyzed for the presence of *E. coli* by inoculating a loopful of each urine sample and the wound swab sample into a separate tube of sterile nutrient broth (Lifesave Biotech, USA) and incubated at 37 °C for 24 h. After overnight incubation, a loopful of the turbid broth culture was aseptically seeded by streaking on sterile solidified Eosin Methylene Blue agar (Lifesave Biotech, USA) and was incubated at 37 °C for 24h. *E. coli* from positive cultures was identified by their characteristic appearance (color, consistency, shape) on the differential media. Each greenish metallic sheen colonies was subculture on sterilized solidified nutrient agar (Lifesave Biotech, USA) and incubated at 37 °C for 24 h for Gram staining reaction, motility test and biochemical profiling, using standard procedures. Biochemical tests such as indole production, sugar fermentation, citrate utilization, methyl red test, voges-proskaeur test, urease test and oxidase test were carried out while further confirmation through VITEK 2 System (bioMerieux, France) was used to identify and confirm *E. coli* isolates [9, 10].

2.3 Phenotypic Detection of AmpC β-lactamase

Bacterial strains that produce AmpC β-lactamase are resistant to the cephamycins but susceptible to the fourth generations cephalosporin, cefepime [11]. All test isolates were standardized according to Peter *et al.* [12] and were subjected to the antimicrobial activity of cefoxitin disk (30 µg) on aseptically streaked MH agar plates. Each plate was incubated at 30 ^{o}C for 18 h [11]. Inhibition zones was interpreted according to CLSI criteria. Cefoxitin-cloxacillin double-disk synergy test (CC-DDST) was performed according to the method described by Ejikeugwu *et al*. [11]. Single disks containing 30 µg of cefoxitin was placed 20 mm away from a disk containing 20 µg of cloxacillin on MH agar plates already inoculated with the test bacteria (equivalent to 0.5 McFarland turbidity standards). Each plate was incubated at 30 ^{o}C for 18 h. Inhibition zones was interpreted according to CLSI criteria.

A difference of 4 mm in the cefoxitin-cloxacillin inhibition zones minus the cefoxitin disk used alone was indicative of AmpC β-lactamase production phenotypically.

2.4 Antimicrobial sensitivity testing

Antibiotic sensitivity testing was carried out on all phenotypic AmpC β-lactamase producing isolates of *E. coli* by employing Kirby Bauer disk diffusion method using sterilized Mueller-Hinton agar in accordance with the guideline of the Clinical Laboratory Standards Institute (CLSI) [13]. Bacteria suspension of the test isolates was prepared using 0.5 MacFarland's standards and seeded on solidified Mueller Hinton agar. The plates were allowed to pre-diffuse for 5 minutes. Thereafter the following antibiotic disc: Amikacin (15 µg), Azetronam (30 µg), Ceftazidime (30 µg), Ceftriaxone (30 µg), Cefepime (30 µg), Cefotaxime (30 µg), Ofloxacin (5 µg), Imipenem (30 µg), Trimethoprim-Sulfamethoxazole (25 µg), Ticarcillin-clavulanic acid (85 µg) were impregnated on the inoculated Mueller-Hinton agar plates and incubated at 37^0C for 18-24 hours. After which, the diameters of zones of inhibition produced by each of the antibiotic disc were measured and the results interpreted in accordance with the criteria of Clinical Laboratory Standards Institute [13, 14].

3. RESULT

3.1 Distribution of AmpC β-lactamases producing *E. coli* from patient according to wards

The frequency distribution of bacteria was more in Surgical ward 36(53.7 %) followed by medical ward 34(36.6 %) and orthopedic ward 10(24.4 %). *E. coli* isolated from urine were highly predominant in medical ward 27(29.0 %) followed by 10(24.4 %) and 15(22.4 %) from Orthopedic and Surgical ward respectively. The frequency of bacteria isolation from wound samples were high in orthopedic ward 15(45.5 %) followed by 9(52.9 %) in Children ward. The frequency of *E. coli* was 9(52.9 %), 15(45.5 %), 6(26.1 %), 10(20.8 %) from Children, Orthopedic, Surgical and Medical ward respectively. The frequency of AmpC β-lactamases producing *E. coli* in urine samples were 57(93.4 %) comprising of high occurrence rate in Surgical ward 15(100 %) followed by medical ward 24(88.9 %) but also accounted for overall prevalence rate of 38(95.0 %) in wound samples consisting of 9(100 %) from children ward and medical ward 10(100 %) followed by Orthopedic ward 13(86.7 %) with the least prevalence rate as presented in Table 1.

3.2 Antibiotic susceptibility profile of AmpC β-lactamases producing *E. coli* from urine samples of patients according to hospital wards

AmpC β-lactamases producing *E. coli* from patients at medical ward were highly resistant to Azetronam 100 %, Ceftazidime 100 %, Ceftriaxone 100 %, Cefepime 100 %, Cefotaxime 100 %, Trimethoprim-Sulfamethoxazole 100 % and Ticarcillin-clavulanic acid 100 % but were susceptible to Imipenem 100 %, Amikacin 83.3 % and Ofloxacin 79.2 %. AmpC β-lactamases producing *E. coli* from patients in surgical ward were highly resistant 100 %, 100 %, 100 %, 100 %, 100 %, 93.3 % and 86.7 % to Azetronam, Ceftazidime, Cefotaxime, Trimethoprim-Sulfamethoxazole, Ticarcillin-clavulanic acid, Cefepime and Ceftriaxone respectively but were susceptible to Amikacin 100 %, Imipenem 100 % and Ofloxacin 86.7 %. Isolate recovered from urine samples in children ward exhibit 100 %, 100 %, 100 %, 88.9 %, 77.8 %, 66.7 %, 55.6 %, 11.1 % resistance to Azetronam, Trimethoprim-Sulfamethoxazole, Ticarcillin-clavulanic acid, Ceftriaxone, Cefotaxime, Ceftazidime, Cefepime and Amikacin respectively but 100 % susceptible to ofloxacin and imipenem. AmpC β-lactamases producing *E. coli* from patients at orthopedic ward were extremely resistant to Ceftazidime 100 %, Ceftriaxone 100 %, Cefepime 100 %, Trimethoprim-Sulfamethoxazole 100 %, Ticarcillin-clavulanic acid 100 % but were susceptible to Ofloxacin 77.8 %, Amikacin 100 % and imipenem 100 % (Table 2).

3.3 Antibiotic susceptibility profile of AmpC β-lactamases producing *E. coli* from wound samples of patient according to different hospital wards.

From medical ward, AmpC β-lactamases producing *E. coli* exhibit 100 % resistant to Azetronam, Ceftriaxone, Cefepime, Trimethoprim-Sulfamethoxazole and Ticarcillin-clavulanic but 10 %, 40% and 100% susceptible to Cefotaxime, Ceftazidime and Imipenem respectively. From surgical ward, majority of the isolates were 83.3 %, 100 %, and 16.7 % resistant to Ceftriaxone, Ticarcillin-clavulanic acid, and imipenem respectively. From Children ward, the isolates were susceptible to impenem 88.9 %, Amikacin 77.8 %, ofloxacin 55.6 %, Ceftriaxone 33.3 %. AmpC β-lactamases producing *E. coli* from patients in orthopedic ward were 100 % resistant to Azetronam, Ceftazidime, Ceftriaxone, Cefepime, Cefataxime, Trimethoprim-Sulfamethoxazole and Ticarcillin-clavulanic acid but were susceptible to Amikacin 76.9 %, ofloxacin 61.5 % and Imipenem 100 % (Table 3).

Table 1: Distribution of AmpC β-lactamases producing *E. coli* from patient at Alex Ekwueme - Federal University Teaching Hospital (AE-FUTHA) according to wards

Clinical Sample	**Wards**	**No. of sample**	***E. coli* (%)**	**AmpC β-lactamases**	
				Positive (%)	**Negative (%)**
Urine	Medical	93	27(29.0)	24(88.9)	3(11.1)
	Surgical	67	15(22.4)	15(100)	0(0.0)
	Children	78	9(11.5)	9(100)	0(0.0)
	Orthopedic	41	10(24.4)	9(90.0)	1(10.0)
Sub-total		**279**	**61(21.9)**	**57(93.4)**	**4(6.6)**
Wound swab	Medical	48	10(20.8)	10(100)	0(0.0)
	Surgical	23	6(26.1)	6(100)	0(0.0)
	Children	17	9(52.9)	9(100)	0(0.0)
	Orthopedic	33	15(45.5)	13(86.7)	2(13.3)
Sub-total		**121**	**40(33.1)**	**38(95.0)**	**2(5.0)**
Overall total		**400**	**101(25.3)**	**95(94.1)**	**6(5.9)**

Table 2: Antibiotic susceptibility profile of AmpC β-lactamases producing *E. coli* from urine samples of patients at Alex Ekwueme - Federal University Teaching Hospital (AE-FUTHA) according to hospital wards

Ward	**Medical**		**Surgical**		**Children**		**Orthopedic**	
	E. coli (n=24)		***E. coli* (n=15)**		***E. coli* (n=9)**		***E. coli* (n=9)**	
Antibiotics (µg)	**R (%)**	**S (%)**	**R (%)**	**S (%)**	**R (%)**	**S (%)**	**R (%)**	**S (%)**
Amikacin (15)	4(16.7)	20(83.3)	0(0.0)	15(100)	1(11.1)	8(88.9)	0(0.0)	9(100)
Azetronam (30)	24(100)	0(0.0)	15(100)	0(0.0)	9(100)	0(0.0)	8(88.9)	1(11.1)
Ceftazidime (30)	24(100)	0(0.0)	15(100)	0(0.0)	6(66.7)	3(33.3)	9(100)	0(0.0)
Ceftriaxone (30)	24(100)	0(0.0)	13(86.7)	2(13.3)	8(88.9)	1(11.1)	9(100)	0(0.0)
Cefepime (30)	24(100)	0(0.0)	14(93.3)	1(6.7)	5(55.6)	4(44.4)	9(100)	0(0.0)
Cefotaxime (30)	24(100)	0(0.0)	15(100)	0(0.0)	7(77.8)	2(22.2)	9(100)	0(0.0)
Ofloxacin (5)	5(20.8)	19(79.2)	2(13.3)	13(86.7)	0(0.0)	9(100)	2(22.2)	7(77.8)
Imipenem (30)	0(0.0)	24(100)	0(0.0)	15(100)	0(0.0)	9(100)	0(0.0)	9(100)
Trimethoprim-Sulfamethoxazole (25)	24(100)	0(0.0)	15(100)	0(0.0)	9(100)	0(0.0)	9(100)	0(0.0)
Ticarcillin-clavulanic acid (85)	24(100)	0(0.0)	15(100)	0(0.0)	9(100)	0(0.0)	9(100)	0(0.0)

Key: R-Resistance, S- Susceptible, n=Number of isolates

Table 3: Antibiotic susceptibility profile of AmpC β-lactamases producing *E. coli* from wound samples of patient at Alex Ekwueme - Federal University Teaching Hospital (AE-FUTHA) according to different hospital wards.

Ward	**Medical**		**Surgical**		**Children**		**Orthopedic**	
	E. coli (n=10)		***E. coli* (n=6)**		***E. coli* (n=9)**		***E. coli* (n=13)**	
Antibiotics (µg)	**R (%)**	**S (%)**	**R (%)**	**S (%)**	**R (%)**	**S (%)**	**R (%)**	**S (%)**
Amikacin (15)	2(20)	8(80)	1(16.7)	5(83.3)	2(22.2)	7(77.8)	3(23.1)	10 (76.9)
Azetronam (30)	10(100)	0(0.0)	6(100)	0(0.0)	9(100)	0(0.0)	13(100)	0(0.0)
Ceftazidime (30)	6(60)	4(40)	6(100)	0(0.0)	9(100)	0(0.0)	13(100)	0(0.0)
Ceftriaxone (30)	10(100)	0(0.0)	5(83.3)	1(16.7)	6(66.7)	3(33.3)	13(100)	0(0.0)
Cefepime (30)	10(100)	0(0.0)	6(100)	0(0.0)	9(100)	0(0.0)	13(100)	0(0.0)
Cefotaxime (30)	9(90)	1(10)	6(100)	0(0.0)	9(100)	0(0.0)	13(100)	0(0.0)
Ofloxacin (5)	3(30)	7(70)	2(33.3)	4(66.7)	4(44.4)	5(55.6)	5(38.5)	8(61.5)
Imipenem (30)	0(0.0)	10(100)	0(0.0)	6(100)	1(11.1)	8(88.9)	0(0.0)	13(100)
Trimethoprim-Sulfamethoxazole (25)	10(100)	0(0.0)	6(100)	0(0.0)	9(100)	0(0.0)	13(100)	0(0.0)
Ticarcillin-clavulanic acid (85)	10(100)	0(0.0)	6(100)	0(0.0)	9(100)	0(0.0)	13(100)	0(0.0)

Key: R-Resistance, S- Susceptible, n=Number of isolates

4. DISCUSSIONS

AmpC β-lactamases production was detected with high prevalence rate of 94.1 % from *E. coli* isolates in this study but was higher than earlier findings in a Pakistan, 7.9% [15] and 12.5% reported in Abakaliki, Nigeria from animal sample [11] and other studies in Iran and Canada [2, 16]. In Nigeria and beyond, there is a paucity of studies on AmpC β-lactamases producing bacteria from clinical origin. Although limited information's exist for possible comparison but observed disparity on these valuable studies might be due to variation in patient type and guidelines of AmpC detection methods. Phenotypic AmpC detection methods have different guidelines and the use of variable guidelines could result on variability in the magnitude of AmpC production for different studies. Moreover, bacterial species that are susceptible for acquiring AmpC genes are different in different parts of the world.

In the present study, all bacterial isolates showed high level of resistance to Ceftazidime, Ceftriaxone, Cefoxitin, Cefuroxime, Cefotaxime ranging from 60.0-100%. In contrast with other studies; Najjuka *et al.* [17] stated that 73% of cefoxitin resistant and pAmpC positive isolates were susceptible to third-generation cephalosporins. Also 26.0 % (10/38) has been reported from Switzerland, for pAmpC producing isolates that were susceptible to third-generation cephalosporins [18] while in Northern Europe 100 % of isolates exhibiting resistance to third-generation cephalosporins carried pAmpC genes [19]. Also, Robatjazi *et al.* [2] demonstrated high prevalence rate for resistance to certain antibiotics, such as cefuroxime and cefotaxime. In another study, antimicrobial resistance pattern revealed that AmpC producing *E. coli* were highly resistant to ceftazidime, cefotaxime, cefuroxime, cefixime, ceftriaxone and cefoxitin (100% each) [20]. This is similar with reported trend of AmpC *E. coli* isolates resistant to cefoxitin (91.7%), ceftriaxone (83.3%), ceftazidime (87.5%) has been reported in Abakaliki, Southeastern, Nigeria [11]. In Southern India, AmpC, Gram-negative bacterial isolates from human immunodeficiency virus infected patients revealed that 87.3% and 83.2% of the bacterial isolates were resistant to 3GC such as cefotaxime and ceftazidime, respectively, the resistance rate of bacterial isolates to cefoxitin was 56.6% [21]. One such study from Korea reported that all of the AmpC producing *E. coli* were resistant to ceftazidime, cefotaxime, ceftriaxone and cefoxitin (100% each) [22]. AmpC producers seem susceptible to cephalosporins *in-vitro* but when cephalosporins are used *in vivo*, they result in treatment failure [23, 24]. Therefore, cephalosporins should not be recommended in treating infections caused by AmpC producing bacteria in the study area. Overall, findings of this authors suggest that antibiotic susceptibility testing of enterobacteria in most countries may yield false results for third-generation cephalosporins e.g, ceftriaxone, cefotaxime and ceftazidime. Given that bacterial isolates are not routinely tested for AmpC β-lactamase production, region specific protocols guided by surveillance data are necessary. This differences in this result might be linked to misuse of cephalosporin antibiotic by patients without physician prescription which results in the development of antibiotic resistance in *E. coli* from patient in this setting.

Azetronam and Trimethoprim-Sulfamethoxazole 100% resistant was common in all hospital wards. Similar study has reported AmpC *E. coli* 65.7% resistant against trimethoprim/sulfamethoxazole [25]. Ibrahim *et al.* [3] also found AmpC *β*-lactamase-producing GNB showed high resistance rates to trimethoprim/sulfamethoxazole (90.1%), and aztreonam (81.2 %). Also another study 92% of bacterial isolates were resistant to aztreonam, 87% to trimethoprim (Rameshkumar *et al.*, 2021). This observation indicates exposure to sub-lethal dose of this antibiotic by the patient over time. In order to overcome β-lactamase-mediated resistance, β-lactamase inhibitors such as clavulanate, sulbactam, and tazobactam have been developed and applied in clinical practice, and found to greatly increase the efficacy of β-lactams in combination therapies. But in recent time the failure or stability of β-lactamase inhibitors is uncertain, Additionally, beta-lactamase inhibitor (Ticarcillin-clavulanic acid in this study) resistance could be used as a pointer or rationale to the actual level of *in vitro* resistance to other beta-lactamase inhibitor i.e., the resistance to any antimicrobial agent in the beta-lactamase inhibitor class has an impact on the resistance of other agents within this drug class. However, the high level of resistant across ward in our study could also be due to other possible mechanisms like efflux pump or loss of porin by the strain.

Also, the high trend of resistant to antimicrobial agent in this study may be linked to overexpression of the AmpC β-lactamases which are encoded on plasmid of *E. coli*. AmpC β-lactamases are clinical important enzymes and also known to be transferred to organisms lacking or poorly expressing chromosomal AmpC genes such as *E. coli*. β-lactamases producing *E. coli* is one of the currently priority pathogens for intervention and response. *E. coli* strains are easily spreading, efficient at acquiring and disseminating resistance plasmids and production of different types of β-lactam genetic determinant. The presence of AmpC β-lactamases enzyme in *E. coli* in this study provides opportunity for the horizontal transmission of these enzymes from one organism to another. AmpC β-lactamases producing *E. coli* were 70.0 %, 83.3 % and 100% sensitive to ofloxacin, Imipenem and Amikacin. The effectiveness of this antimicrobial agent to AmpC β-lactamases producing isolate in this study substantiate reported in few

literature [26, 27, 28]. Thus, for better management of AmpC β-lactamases producing strain infection, microbial classification of infection as well as drug sensitivity test of organism recovered are essential for making appropriate decision of antimicrobials that will effectively eradicate the pathogen. Ofloxacin, Imipenem and Amikacin are alternatives that can be used as first line chemotherapy for this pathogen However, this will depend on local resistance proportions, which this study have provided at the moment.

5. CONCLUSION

This study highlighted the prevalence of AmpC β-lactamases producing *E. coli* from different wards in the hospital. Therefore, the result of this study may be proof of the urgent need for controlling and managing the development of MDR genotype strain. Moreover, the reported high level of antibiotic resistant accentuate the need that antibiotic stewardship procedures should be applied to limit the illogical use of antibiotics in the study settings. Thus, with the effectiveness of ofloxacin, imipenem and amikacin, the treatment of AmpC β-lactamases producing isolates cases are deemed or advise to be guided by antibiotics susceptibility testing. Further, studies are required to detect the clonal spread of subtype AmpC β-lactamases and other genetic determinant of ARG in bacteria.

CONSENT

All authors declare that written informed consent was obtained from the patient or care-giver of the patient before collection of sample

ETHICAL CLEARANCE

The study was carried out after obtaining approval from the Ethical Review Committee of Ebonyi State Ministry of Health, Abakaliki with approval no: SMOH/ERC/042/21.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

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